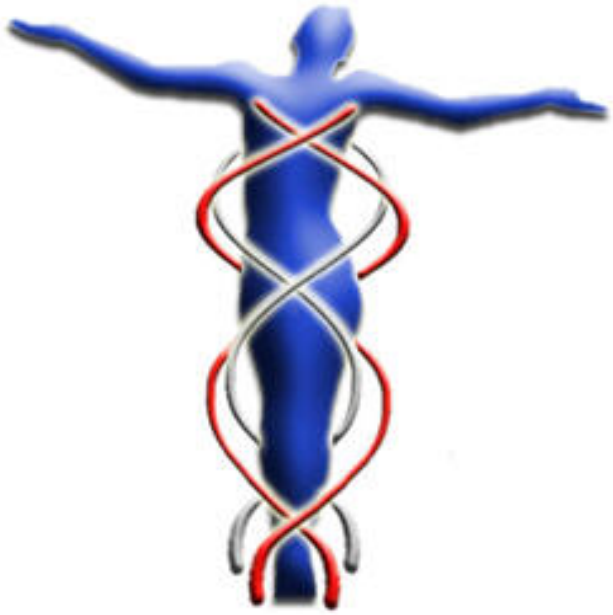
ΕΜΜΑΝΟΥΗΛ Β. ΟΙΚΟΝΟΜΟΥ

ΕΠΙΚΟΥΡΟΣ ΚΑΘΗΓΗΤΗΣ
ΚΛΙΝΙΚΗΣ ΒΙΟΧΗΜΕΙΑΣ
ΙΑΤΡΙΚΗΣ ΣΧΟΛΗΣ ΠΑΝΕΠΙΣΤΗΜΙΟΥ ΑΘΗΝΩΝ

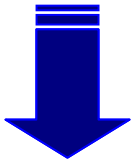
ΒΙΟΓΡΑΦΙΚΟ ΣΗΜΕΙΩΜΑ ΥΠΟΨΗΦΙΟΥ

Για Μονομοποίηση

Στη Βαθμίδα του ΕΠΙΚΟΥΡΟΥ ΚΑΘΗΓΗΤΗ



ΑΘΗΝΑ 2014



ΠΕΡΙΕΧΟΜΕΝΑ ΕΠΙΣΥΝΑΠΤΟΜΕΝΟΥ CD

1. **ΗΛΕΚΤΡΟΝΙΚΗ ΜΟΡΦΗ ΒΙΟΓΡΑΦΙΚΟΥ ΣΗΜΕΙΩΜΑΤΟΣ**
2. **ΞΕΝΟΓΛΩΣΣΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΤΟΥ SCI (Ξ.Δ. SCI) ΣΕ ΜΟΡΦΗ ΑΡΧΕΙΟΥ PDF Ή WORD**
3. **ΞΕΝΟΓΛΩΣΣΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΕΚΤΟΣ SCI (Ξ.Δ. εκτός SCI) ΣΕ ΜΟΡΦΗ ΑΡΧΕΙΟΥ PDF Ή WORD**
4. **ΕΛΛΗΝΙΚΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ (Ε.Δ.) ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ ΣΕ ΜΟΡΦΗ ΑΡΧΕΙΟΥ WORD**
5. **ΕΛΛΗΝΙΚΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΠΑΝΕΛΛΗΝΙΩΝ ΣΥΝΕΔΡΙΩΝ (Ε.Δ. σε Π.Ε.Ε.Π.Σ.) ΣΕ ΜΟΡΦΗ ΑΡΧΕΙΟΥ WORD**

ΠΕΡΙΕΧΟΜΕΝΑ

ΑΝΑΛΥΤΙΚΟ ΒΙΟΓΡΑΦΙΚΟ ΣΗΜΕΙΩΜΑ

1. ΠΡΟΣΩΠΙΚΑ ΣΤΟΙΧΕΙΑ

ΟΝΟΜΑΤΕΠΩΝΥΜΟ	:	ΟΙΚΟΝΟΜΟΥ ΕΜΜΑΝΟΥΗΛ
ΕΘΝΙΚΟΤΗΤΑ	:	ΕΛΛΗΝΙΚΗ
ΔΙΕΥΘΥΝΣΗ ΚΑΤΟΙΚΙΑΣ	:	ΚΑΔΜΟΥ 8 & ΑΓΑΜΕΜΝΩΝΟΣ 24 – ΝΤΡΑΦΙ ΠΙΚΕΡΜΙΟΥ ΡΑΦΗΝΑΣ Τ.Κ. 19009 – Τ.Θ. 2589
ΤΗΛΕΦΩΝΟ ΟΙΚΙΑΣ	:	210-6002540
ΤΗΛΕΦΩΝΑ ΕΡΓΑΣΙΑΣ	:	210-7286259, 210-6421309
ΚΙΝΗΤΟ ΤΗΛΕΦΩΝΟ	:	6944-281173
FAX	:	210-6084476
e-MAIL	:	eveconom@otenet.gr, eveconom@med.uoa.gr
ΗΜΕΡΟΜΗΝΙΑ ΓΕΝΝΗΣΕΩΣ	:	30 ΙΑΝΟΥΑΡΙΟΥ 1962
ΤΟΠΟΣ ΓΕΝΝΗΣΕΩΣ	:	ΑΘΗΝΑ, ΕΛΛΑΔΑ
ΟΙΚΟΓΕΝΕΙΑΚΗ ΚΑΤΑΣΤΑΣΗ	:	ΑΓΑΜΟΣ

2. ΠΑΡΟΥΣΑ ΘΕΣΗ

Επίκουρος Καθηγητής επί θητεία Κλινικής Χημείας – Φαρμακευτικής Βιοχημείας Ιατρική Σχολή Πανεπιστημίου Αθηνών Τομέας Υγείας Μητέρας-Παιδιού - Β' Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική, ΑΡΕΤΑΙΕΙΟ Πανεπιστημιακό Νοσοκομείο Αθηνών - Εργαστήριο Θεραπευτικής Εξατομίκευσης (Πρόγραμμα ΕΛΚΕ 70/3/9503)

3. ΣΤΡΑΤΙΩΤΙΚΗ ΘΗΤΕΙΑ

Απολυτήριο Πολεμικού Ναυτικού Ναύτης Νοσοκόμος – Ναυτική Βάση Νότιου Ευβοϊκού
04.03.1991 – 04.03.1992

4. ΕΚΠΑΙΔΕΥΣΗ – ΜΕΤΕΚΠΑΙΔΕΥΣΗ - ΔΙΠΛΩΜΑΤΑ

Α. ΕΓΚΥΚΛΙΟΣ ΕΚΠΑΙΔΕΥΣΗ	Λύκειο Κρανιδίου Αργολίδας, Ιούνιος 1979, Βαθμός Αποφοίτησης : ΑΡΙΣΤΑ (19 και 2/13)
Β. ΠΡΟΠΤΥΧΙΑΚΗ ΕΚΠΑΙΔΕΥΣΗ	Εθνικό και Καποδιστριακό Πανεπιστήμιο της Αθήνας, Πτυχίο Φαρμακευτικού Τμήματος της Σχολής Επιστημών Υγείας, Ιούνιος 1985. Βαθμός Αποφοίτησης: ΑΡΙΣΤΑ (8,63).
Γ. ΑΔΕΙΑ ΑΣΚΗΣΕΩΣ ΕΠΑΓΓΕΛΜΑΤΟΣ	Άδεια Ασκήσεως του Επαγγέλματος της Φαρμακευτικής, Υπουργείο Δημόσιας Υγείας, Οκτώβριος 1992.
Δ. ΜΕΤΑΠΤΥΧΙΑΚΗ ΕΚΠΑΙΔΕΥΣΗ	

1. **Εθνικό και Καποδιστριακό Πανεπιστήμιο της Αθήνας. Τμήμα Φαρμακευτικής Τεχνολογίας και Ραδιο-φαρμακευτικής, Διδακτορικό Δίπλωμα, Ιούνιος 1993. Αντικείμενο Διδακτορικής Διατριβής : "Ραδιοανοσοαναλύσεις για τον Προσδιορισμό της Διαιθυλο- στιλβοιστρόλης στον Ορό και Αξιολόγηση Αυτών". Βαθμός Διδακτορικής Διατριβής :ΑΡΙΣΤΑ.**

2. **Ινστιτούτο Πυρηνικής Ιατρικής, Kern Forschoungsanlage, Juelich, Γερμανία. Μεταδιδακτορική Εκπαίδευση στο Τμήμα Ραδιοφαρμάκων-Ραδιοδιαγνωστικών Προϊόντων-Ραδιοανοσοαναλύσεων, 1989-1991.**
Ειδική Εκπαίδευση σε Ανοσοδιαγνωστικές Τεχνικές (RIA, IRMA, ELISA, FIA, Chemiluminescence Immunoassays).
Βεβαίωση από το αντίστοιχο Ινστιτούτο.

3. **Κέντρο Πυρηνικών Ερευνών "ΔΗΜΟΚΡΙΤΟΣ", Πρόγραμμα Μετα-πτυχιακών Μαθημάτων στα ακόλουθα αντικείμενα (1985 - 1987) : Φασματοσκοπία NMR, Κρυσταλλο- γραφία, Βιομετρία, Βιοχημεία, Νευροχημεία, Ραδιοανοσοχημεία, Ελεγχος Ραδιοφαρμακευτικών Σκευασμάτων, Ειδικά Θέματα IN VITRO και IN VIVO Ραδιοφαρμακευτικών Σκευασμάτων, Θεωρία και Γλώσσες Ηλεκτρονικών Υπολογιστών (BASIC).**

Ε. ΞΕΝΕΣ ΓΛΩΣΣΕΣ

1. Αγγλικά (Proficiency Cambridge)
2. Γερμανικά (Mittelstufe Goethe)

5. ΔΙΔΑΚΤΙΚΟ ΕΡΓΟ

Α. ΠΡΟΠΤΥΧΙΑΚΟ ΕΚΠΑΙΔΕΥΤΙΚΟ ΕΡΓΟ

1. ΜΑΘΗΜΑΤΑ

1.1. "*Θεραπεία Ορμονικής Υποκατάστασης και Καρδιαγγειακή Νόσος – Ο Ρόλος του Ατομικού Παράγοντα*"
Μάθημα στους 6/ετείς τριμηνίτες φοιτητές της Ιατρικής Σχολής
Σε όλες τις ομάδες των ακαδημαϊκών περιόδων 2003-2014

1.2. **"Θρόμβωση : Παθοφυσιολογία – Αντιμετώπιση"**
Μάθημα στους ειδικευομένους χειρουργούς, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, 15-02-2011

Β. ΜΕΤΑΠΤΥΧΙΑΚΟ ΕΚΠΑΙΔΕΥΤΙΚΟ ΕΡΓΟ

1. ΜΑΘΗΜΑΤΑ

1.1. *"Μεθοδολογία Εργαστηριακής Ιατρικής Ερευνας" (4 ώρες)*
Διετές Μεταπτυχιακό Πρόγραμμα «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολής Πανεπιστήμιου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών, ακαδημαϊκό έτος 2007 – 2008 : 29.11.2007, ακαδημαϊκό έτος 2008 – 2009 : 29.10.2008, ακαδημαϊκό έτος 2009 – 2010 : 26.11.2009, ακαδημαϊκό έτος 2010 – 2011 : 01.12.2010, ακαδημαϊκό έτος 2011 – 2012 : 24.11.2011, ακαδημαϊκό έτος 2012 – 2013 : 28.11.2012, ακαδημαϊκό έτος 2013 – 2014 : 6.11.2013

1.2. **"***ΟΡΜΟΝΕΣ : Βιοσύνθεση – Απελευθέρωση – Μεταφορά – Δράση – Μεταβολισμός –*

Μηχανισμοί Ελέγχου" (2 - 4 ώρες)
Διετές Μεταπτυχιακό Πρόγραμμα «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολής Πανεπιστήμίου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών, ακαδημαϊκό έτος 2007 – 2008 : 12.12.2007 (4 ώρες), ακαδημαϊκό έτος 2008 – 2009 : 30.10.2008 (2 ώρες), ακαδημαϊκό έτος 2009 – 2010 : 02.12.2009 (2 ώρες), ακαδημαϊκό έτος 2010 – 2011 : 02.12.2010 (2 ώρες), ακαδημαϊκό έτος 2011 – 2012 : 30.11.2011 (2 ώρες), ακαδημαϊκό έτος 2012 – 2013 : 29.11.2012 (2 ώρες), ακαδημαϊκό έτος 2013 – 2014 : 7.11.2013 (2 ώρες)

1.3. *"ΕΡΓΑΣΤΗΡΙΑΚΕΣ ΑΣΚΗΣΕΙΣ – Βιοχημική Μεθοδολογία"*
Διετές Μεταπτυχιακό Πρόγραμμα «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολής Πανεπιστήμίου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών, ακαδημαϊκό έτος 2007 – 2008 : 17.01.2008 και 23.01.2008, ακαδημαϊκό έτος 2008 – 2009 : 23.01.2009, 26.01.2009, 30.01.2009, 02.02.2009, 09.02.2009, 16.02.2009, 27.02.2009, 09.03.2009, 27.04.2009

1.4. "*Δυναμικές Δοκιμασίες Ορμονών στη Γυναικεία Αναπαραγωγή" (4 ώρες)*
Διετές Μεταπτυχιακό Πρόγραμμα «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολής Πανεπιστήμίου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών, ακαδημαϊκό έτος 2007 – 2008 : 20.12.2007, ακαδημαϊκό έτος 2008 – 2009 : 05.11.2008, ακαδημαϊκό έτος 2009 – 2010 : 03.12.2009, ακαδημαϊκό έτος 2010 – 2011 : 08.12.2010

1.5. "*Καθορισμός και Διαφοροποίηση Φύλου στην Ενδομήτριο Ζωή" (2 ώρες)*
Διετές Μεταπτυχιακό Πρόγραμμα «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολής Πανεπιστήμίου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών, ακαδημαϊκό έτος 2007 – 2008 : 16.01.20087, ακαδημαϊκό έτος 2008 – 2009 : 13.11.2008, ακαδημαϊκό έτος 2009 – 2010 : 16,12.2009, ακαδημαϊκό έτος 2010 – 2011 : 16.12.2010

1.6. "Κλινική Σημασία Ορμονικών Μετρήσεων – Το Εργαστήριο στην Κλινική Ενδοκρινολογία"
Πρόγραμμα Μεταπτυχιακού Μαθήματος Ενδοκρινολογίας Ιατρικής Σχολής Πανεπιστημίου Αθηνών : 01.02.2005 – 24.05.2005

1.7. "*Μεθοδολογία Εργαστηριακής Ιατρικής Έρευνας*"
Πρόγραμμα Μαθημάτων Κορμού Α' Εξαμήνου Ακαδημαϊκού Ετους 2003-2004 ΠΜΣ "Κλινική Ιατρική, Εργαστηριακή Ιατρική, Προληπτική και Κοινωνική Ιατρική" :19.04.2004 – 16.07.2004

2. ΣΥΜΒΟΛΗ ΣΤΗΝ ΕΚΠΟΝΗΣΗ ΔΙΔΑΚΤΟΡΙΚΩΝ ΔΙΑΤΡΙΒΩΝ

2.1 ΟΛΟΚΛΗΡΩΜΕΝΕΣ ΔΙΔΑΚΤΟΡΙΚΕΣ ΔΙΑΤΡΙΒΕΣ

2.1.1. *"Επίδραση της ορμονικής θεραπείας στο καρδιαγγειακό σύστημα των φαινοτυπικά θηλέων ατόμων με αμιγή ή γονιδιακή δυσγενεσία τύπου ΧΥ ή με διαταραχές διαφοροποίησης του φύλου με καρυότυπο ΧΥ"*
Διδακτορική Διατριβή του **Μαιευτήρος-Γυναικολόγου Ιατρού κου Παντελή Τσίμαρη**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 5385/07.02.2005) : Ε. Δεληγεώργλου, Γ. Μαστοράκος, **Ε. Οικονόμου**
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ. 8084/23.07.2014) : Γ.Κρεατσάς, Δ. Λουτράδης, Δ. Κασσάνος, Ν. Βιτωράτος, Ε. Δεληγεώργλου, Γ. Μαστοράκος, **Ε. Οικονόμου**
Συμβολή : **Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής**

2.1.2. ***"Διερεύνηση της σχέσης των κυκλοφορούντων καρκινικών κυττάρων, δεικτών***

αποπτωτικής ομοιοστασίας και γενετικής ετερογένειας παραγόντων που την επηρεάζουν, στον καρκλινο μαστού – Κλινική Σημασία"
Διδακτορική Διατριβή της ειδικευομένης Χειρουργού Ιατρού κας **Αννέζας Γιαλούρου**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ.Πρωτ. 8215/21.04.2008): Ε. Παπαλάμπρου, Ι. Ψυχογιός, **Ε. Οικονόμου**
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ. 6521/05.06.2014) : Ε. Παπαλάμπρου, Δ. Βώρος, Κ. Γεννατάς, Ι. Βασιλείου, Α. Κονδή – Παφίτη, Ι. Ψυχογιός, **Ε. Οικονόμου**
Συμβολή : **Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής**

2.1.3. "Η επίδραση της τεριπαρατίδης στο οστικό μεταβολισμό οστεοπορωτικών μετεμμηνοπαυσιακών γυναικών"
Διδακτορική Διατριβή του Ιατρού κου **Εμμανουήλ Λογοθέτη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ.1605/27.01.2014) : Γ.Κρεατσάς, Ν. Βιττωράτος, Γ. Χριστοδουλάκος, Ε. Λαμπρινουδάκη, Α. Αντωνίου, **Ε. Οικονόμου**, Γ. Καππαρό.
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη - Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής**

2.1.4. "Συγκριτική μελέτη του ενδοκυττάριου μονοπατιού μεταγωγής σήματος mTOR σε δείγματα φυσιολογικού ιστού, προκαρκινωματωδών αλλοιώσεων και καρκίνου του τραχήλου της μήτρας"
Διδακτορική Διατριβή του Μαιευτήρα Χειρουργού Γυναικολόγου Ιατρού κου **Αριστείδη Ασημομύτη**
Επιβλέπουσα Τριμελής Εξεταστική Επιτροπή: Ε. Πατσούρης, Γ. Ρασιδάκης, **Ε. Οικονόμου**
Συμβολή : **Συμβουλευτική Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ. 1313/21.01.2014) : Ε. Πατσούρης, Ε. Αγαπητός, Σ, Τσελένη-Μπαλαφούτα, Α. Λάζαρης, Γ. Ρασιδάκης, **Ε. Οικονόμου**, Α. Νόννη
Συμβολή : Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής

2.1.5. "Βιοχημική και γενετική προσέγγιση της απόκρισης της νευροορμονικής ομοιοστασίας και του οστικού μεταβολισμού στη διαδερμική χορήγηση ορμονικής θεραπείας σε γυναίκες με ψυχογενή ανορεξία "
Διδακτορική Διατριβή Μαιευτήρος-Γυναικολόγου Ιατρού κας **Ευγενίας Στεργιώτη**
Συμβολή (ως μέλος της επιβλέπουσας τριμελούς επιτροπής): **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ.8921/27.05.2013) : Γ.Κρεατσάς, Δ. Λουτράδης, Δ. Μπότσης, Ε. Δεληγεώργλου, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**, Α. Τσίτσικα.
Συμβολή : **Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής**

2.1.6. *"Η επίδραση της ορμονικής θεραπείας και της ραλοξιφαίνης στην ενδοθηλιακή λειτουργικότητα του αρτηριακού δικτύου και σε επιλεγμένους δείκτες απόπτωσης»*
Διδακτορική Διατριβή Μαιευτήρος-Γυναικολόγου Ιατρού κας **Ζωής Σιάσου**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 10247/24.06.2004): Γ. Χριστοδουλάκος, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ.7003/27.08.2013) : Γ.Κρεατσάς, Γ. Χριστοδουλάκος, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**, Κ.Πανουλής, Χ. Συριστατίδης, Π. Παππά.
Συμβολή : **Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής**

2.1.7. "Διερεύνηση της σχέσης μονονουκλεοτιδικών πολυμορφισμών των γονιδίων που κωδικοποιούν ένζυμα μεταβολισμού του φυλικού οξέος με δομικούς και

λειτουργικούς αρτηριακούς δείκτες μετεμμηνοπαυσιακών γυναικών:αλληλεπίδραση με τα αγγειοκινητικά, ψυχολογικά και ψυχοσωματικά συμπτώματα της εμμηνόπαυσης"
Διδακτορική Διατριβή του Ιατρού κου **Βασιλείου Γρηγορίου**
Συμβολή (ως μέλος της επιβλέπουσας τριμελούς επιτροπής): **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ.2227/20.11.2012) : Γ.Κρεατσάς, Ν. Βιττωράτος, Ν. Βλάχος, Ε. Κουσκούνη, **Ε. Οικονόμου**, Ε. Λαμπρινουδάκη, Ζ. Ηλιοδρομίτη.
Συμβολή : **Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής**

2.1.8. "Κυτταρογενετική και μοριακή μελέτη καρκίνου ωοθηκών"
Διδακτορική Διατριβή του **Ιατρού κου Χρήστου Γ. Αραβίδη**
Επιβλέπουσα Τριμελής Εξεταστική Επιτροπή (Αρ.Πρωτ. 11046/12-7-2005) : Α. Πανανή, Κ. Γεννατάς, , Ε. Οικονόμου
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ. 6810/21.03.2011) : Γ. Κρεατσάς, Α. Αντσακλής, Α. Καλπίνη-Μαύρου, Α. Πανανή, Κ. Γεννατάς, Σ. Κίτσιου-Τζέλη, **Ε. Οικονόμου**
Συμβολή : Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής

2.1.9. *"Βιοχημικοί δείκτες «επαγώμενης» αντιοξειδωτικής κυτταρικής προστασίας και διαταραχής της λειτουργίας του αγγειακού ενδοθηλίου στη συστηματική κυκλοφορία εγκύων γυναικών κατά τη διάγνωση της προεκλαμψίας και μετά το πέρας της κύησης"*
Διδακτορική Διατριβή της **Ειδικευομένης Μαιευτήρος-Γυναικολόγου Ιατρού κας Αικατερίνης Παπακωνσταντίνου**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 6374/08.03.2006) : Ν. Βιτωράτος, Ο. Γρηγορίου, **Ε. Οικονόμου**
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ. ----/10.11.2010) : Ν. Βιτωράτος, Ο. Γρηγορίου, **Ε. Οικονόμου,** Δ. Λουτράδης, Δ. Μπότσης, Χ. Χρέλιας, Κ. Πανουλής
Συμβολή : Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής

2.1.10. *"Η επίδραση της ορμονικής θεραπείας και της τιβολόνης στον αθηρωματογόνο χημειοτακτισμό των λευκών αιμοσφαιρίων του περιφερικού αίματος"*
Διδακτορική Διατριβή της **Ιατρού κας Σοφίας Βλάχου**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 10493/29.06.2005) : Γ. Χριστοδουλάκος, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη**
Επταμελής Εξεταστική Επιτροπή (Αρ.Πρωτ. 4415/15-1-2010) : Γ. Χριστοδουλάκος, Σ. Κονιδάρης, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου,** Κ. Πανουλής, Λ. Αραβαντινός, Ν. Βραχνής
Συμβολή : **Τελική Αξιολόγηση ως μέλος της Επταμελούς Εξεταστικής Επιτροπής**

2.1.11. *"Βιοχημικοί δείκτες «επαγώμενης» αντιοξειδωτικής κυτταρικής προστασίας και διαταραχής της λειτουργίας του αγγειακού ενδοθηλίου στη συστηματική κυκλοφορία εγκύων γυναικών κατά τη διάγνωση της προεκλαμψίας και μετά το πέρας της κύησης"*
Διδακτορική Διατριβή του **Ειδικευομένου Μαιευτήρος-Γυναικολόγου κου Γεωργίου Καλαμπαλίκη**
Επταμελής Επιτροπή Αξιολόγησης (Αρ. Πρωτ. 9818/12.07.2006) : Α. Αντσακλής, Ε. Πατσούρης, Ε. Διακομανώλης, Α. Μαλαμίτση-Puchner, Σ. Δενδρινός, Α. Νταλαμάγκα, **Ε. Οικονόμου**
Συμβολή : **Καθοδήγηση Στατιστικής Επεξεργασίας Αποτελεσμάτων, Συμβολή στην Εξαγωγή Συμπερασμάτων, Συνολική Αξιολόγηση**

2.1.12. "*Βιοχημική προσέγγιση της αγγειακής επαναδιάταξης ως αποτέλεσμα του μεταβολισμού*

της εξωκυττάριας ουσίας σε υπερτασικούς ασθενείς"
Διδακτορική Διατριβή της **Καρδιολόγου Ιατρού κας Αλεξάνδρας Ζερβουδάκη**, Αθήνα 2004.
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη - Αξιολόγηση Αποτελεσμάτων - Συγγραφή Περιλήψεων - Αρθρων που δημοσιεύθηκαν σε Διεθνή και Ελληνικά Περιοδικά**

2.1.13. "*Νευρογενείς αγγειοδραστικοί δείκτες λειτουργικών βλαβών νευρικών κυττάρων του εγκεφάλου στη συστηματική κυκλοφορία υπερτασικών ασθενών. Επίδραση αντιυπερτασικής αγωγής*"
Διδακτορική Διατριβή του **Παθολόγου Ιατρού κου Ιωάννη Σ. Ελευσινιώτη**, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αθήνα 2002.
Συμβολή : **Θεματοδότηση - Εργαστηριακή Υποστήριξη - Αξιολόγηση Αποτελεσμάτων - Συγγραφή Περιλήψεων - Αρθρων που δημοσιεύθηκαν σε Διεθνή και Ελληνικά Περιοδικά**

2.1.14. "*Επίδραση της οξείας υπογλώσσιας χορήγησης αναστολέα του μετατρεπτικού ενζύμου της αγγειοτασίνης στο κολπικό νατριουρητικό πεπτίδιο. Βιοχημικές και αιμοδυναμικές συσχετίσεις*"
Διδακτορική Διατριβή του **Παθολόγου Ιατρού κου Ευάγγελου Δημητρέλλου**, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αθήνα 2002.
Συμβολή : **Εργαστηριακή Υποστήριξη - Αξιολόγηση Αποτελεσμάτων - Στατιστική Επεξεργασία**

2.1.15. "*Ο ρόλος των προφλεγμονωδών κυτταροκινών, της ενεργοποίησης των αιμοπεταλίων και της παραγωγής θρομβίνης στη χρονία σταθερή στηθάγχη. Επίδραση της χορήγησης ασπιρίνης στη μυοκαρδιακή ισχαιμία στη διάρκεια των καθημερινών δραστηριοτήτων*"
Διδακτορική Διατριβή του **Καρδιολόγου Ιατρού κου Ιγνάτιου Γ. Οικονομίδη**, Ιατρική Σχολή Παν. Αθηνών, Αθήνα 2000.
Συμβολή : **Εργαστηριακή Υποστήριξη - Αξιολόγηση Αποτελεσμάτων - Στατιστική Επεξεργασία - Συγγραφή Περιλήψεων και Αρθρων που δημοσιεύθηκαν σε Διεθνή και Ελληνικά Περιοδικά**

2.1.16. "*Η κλινική σημασία των δεικτών αποδόμησης του κολλαγόνου σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Διδακτορική Διατριβή του **Ειδικευόμενου Καρδιολόγου Ιατρού κου Δημητρίου Π. Παπαδόπουλου**, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αθήνα 2000.
Συμβολή : **Εργαστηριακή Υποστήριξη - Αξιολόγηση Αποτελεσμάτων - Στατιστική Επεξεργασία - Συγγραφή Περιλήψεων και Αρθρων που δημοσιεύθηκαν σε Διεθνή και Ελληνικά Περιοδικά**

2.2 ΔΙΔΑΚΤΟΡΙΚΕΣ ΔΙΑΤΡΙΒΕΣ ΣΕ ΕΞΕΛΙΞΗ

2.2.1. **"Γονιδιακοί πολυμορφισμοι σε γυναίκες με συνδρομο πολυκυστικών ωοθηκών και δυσλιπιδαιμία"**
Διδακτορική Διατριβή **της Ειδικευόμενης Ιατρού Γυναικολόγου κας Χριστίνας Τζούμα**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 7192/10.05.12) : Ε. Δεληγεώργολου (επιβλέπων), **Ε. Οικονόμου**, Ε. Λαμπρινουδάκη
Συμβολή : Εργαστηριακή Υποστήριξη

2.2.2. **"Συσχέτιση των γδΤ κυττάρων του περιφερικού αίματοσε αυτόματες αποβολές σε γυναίκες με χλαμυδιακές λοιμώξεις"**
Διδακτορική Διατριβή **Ιατρού Γυναικολόγου κου Ιερώνυμου Βοσκάκη**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 3584/28.12.11) : Σ. Δενδρινός, **Ε. Οικονόμου**, Ε. Δεληγεώργολου (επιβλέπων)

Συμβολή : Εργαστηριακή Υποστήριξη

2.2.3. *"***Διερεύνηση της γενετικής ετερογένειας των γονιδίων τεσσάρων αιμοπεταλιακών γλυκοπρωτεϊνικλων υποδοχέων και διακυτταρικλων συνδλέσμων σε γυναίκες με επαναλαμβανόμενες αποβολές ή αποτυχίες εξωσωματικής γονιμοποίησης και εμβρυομεταφοράς"**
Διδακτορική Διατριβή του Ειδικευόμενου Ιατρού Μαιευτικής-Γυναικολογίας κου Νικόλαου Βλαχάδη
Επιβλέπουσα Τριμελής Επιτροπή (Αρ.Πρωτ. 9606/05.07.2011) : **Ε. Οικονόμου** (επιβλέπων), Ν. Βιτωράτος, Ε. Κουσκούνη
Συμβολή : **Θεματοδότηση – Εργαστηριακή Υποστήριξη**

2.2.4. *"**Λειτουργικότητα αιμοπεταλίων και γενετική ετερογένεια γονιδίων αιμοπεταλικών υποδοχέων στις καθέξιν αποβολές και στις επαναλαμβανόμενες αποτυχημένες I.V.F.**"*
Διδακτορική Διατριβή του **Μοριακού Βιολόγου-Γενετιστή κου Βασιλείου Τσαμαδιά**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ.Πρωτ. 8239/23.04.2009) : Γ. Κρεατσάς, Ε. Κουσκούνη (επιβλέπουσα), **Ε. Οικονόμου**
Συμβολή : Θεματοδότηση – Εργαστηριακή Υποστήριξη

2.2.5. *"**Ανατομικοί δείκτες του περιφερικού αρτηριακού δικτύου και χημειοτακτισμός στην προεκλαμψία και στον σακχαρώδη διαβήτη κύησης**"*
Διδακτορική Διατριβή της **Ειδικευόμενης Μαιευτήρος – Γυναικολόγου κας Χριστίνας Βάτζου**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ.Πρωτ. 10474/29.06.2008 : Ν. Βιτωράτος, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**
Συμβολή : **Εργαστηριακή Υποστήριξη**

2.2.6. *"Διερεύνηση της σχέσης της γενετικής βάσης της ανοσολογικής απάντησης στην ανάπτυξη και στη σταδιοποίηση της ενδομητριώσεως και στη διασπορά στη συτηματική κυκλοφορία της ενδοπυελικής φλεγμονής που τη συνοδεύει"*
Διδακτορική Διατριβή του **Ειδικευόμενου Μαιευτήρος – Γυναικολόγου κου Κωνσταντίνου Κοντοράβδη**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ.Πρωτ. 10726/25.07.2007 : Γ. Κρεατσάς, **Ε. Οικονόμου**, Ν. Βιτωράτος
Συμβολή : Θεματοδότηση - Εργαστηριακή Υποστήριξη

2.2.7. *"Βιοχημική διερεύνηση της επίδρασης ενδομορφινών της συστηματικής κυκλοφορίας στην αποπτωτική ομοιοστασία και στο αγγειακό ενδοθήλιο γυναικών με ενδοπυελική ενδομητρίωση"*
Διδακτορική Διατριβή του **Ειδικευομένου Μαιευτήρος-Γυναικολόγου Ιατρού κου Αθανασίου Γκέκα**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 7826/13.04.2006) : Α. Κοντοράβδης, **Ε. Οικονόμου,** Ν. Βλάχος
Συμβολή : Θεματοδότηση - Εργαστηριακή Υποστήριξη

2.2.8. *"Η επίδραση της ενδομήτριας καθυστέρησης ανάπτυξης στον οστικό μεταβολισμό και στην επίτευξη της κορυφαίας οστικής μάζας στην εφηβεία σε αρουραίους WHISTAR. Πειραματική Μελέτη"*
Διδακτορική Διατριβή του **Ειδικευομένου Μαιευτήρος-Γυναικολόγου Ιατρού κου Γεωργίου Βάγγου**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 10470/29.06.2005) : Γ. Χριστοδουλάκος, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**
Συμβολή : Εργαστηριακή Υποστήριξη

2.2.9. **"Οξειδωτικό stress και ποιότητα σπέρματος στην ουρογεννητική λοίμωξη"**

Διδακτορική Διατριβή **Ιατρού Ουρολόγου κου Βασιλείου Σκουτέρη**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ.Πρωτ.10547/29.06.2005) : Ο. Γρηγορίου, Ε. Κουσκούνη, **Ε. Οικονόμου**
Συμβολή : Θεματοδότηση - Εργαστηριακή Υποστήριξη

2.2.10. "*Βιοχημική διερεύνηση της επίπτωσης της προεκλαμπτικής κύησης στην αναστολή της οστεοκλαστικής δραστηριότητας της εγκύου*"
Διδακτορική Διατριβή **Εδικευομένου Ιατρού Γυναικολόγου κου Χαλίλ-Ηλία Μπαμπαμέτο**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 7153/21.01.2005) : Κ. Παπαδιάς, Ν. Βιτωράτος, **Ε. Οικονόμου**
Συμβολή : Θεματοδότηση - Εργαστηριακή Υποστήριξη

2.2.11. "*Βιοχημική διερεύνηση δεικτών φλεγμονώδους αντίδρασης στη συστηματική κυκλοφορία γυναικών με ενδοπυελική ενδομητρίωση*"
Διδακτορική Διατριβή **Ειδικευομένου Ιατρού Γυναικολόγου κου Βασιλείου Κελλάρη**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 5348/07.02.2005) : Γ. Χριστοδουλάκος, Ε. Κουσκούνη, **Ε. Οικονόμου**
Συμβολή : Θεματοδότηση - Εργαστηριακή Υποστήριξη

2.2.12. *"Βιοχημικοί δείκτες πλακουντιακής ισχαιμίας στην εγκατεστημένη προεκλαμψία και στη πρώτη περίοδο λοχείας"*
Διδακτορική Διατριβή του **Ειδικευομένου Μαιευτήρος-Γυναικολόγου Ιατρού κου Δημητρίου Χριστάκη**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 5385/07.02.2005) : Ν. Βιτωράτος, Ε. Κουσκούνη, **Ε. Οικονόμου**
Συμβολή : Θεματοδότηση - Εργαστηριακή Υποστήριξη

2.2.13. "*Ταυτοποίηση της στεφανιογραφικά επιβεβαιωμένης στεφανιαίας νόσου από την επαγωγή κυτταροπροστατευτικών ορολογικών παραγόντων και τις μεταβολές υπερηχοκαρδιογραφικών δεικτών κατά τη δοκιμασία φόρτισης με δοβουταμίνη*"
Διδακτορική Διατριβή **Ιατρού κου Χρήστου Κοκκινάκη**
Επιβλέπουσα Τριμελής Επιτροπή (Αρ. Πρωτ. 11062/15.07.2004) : Χ. Στεφανάδης, Χ. Πίτσαβος, **Ε. Οικονόμου**
Συμβολή : Θεματοδότηση - Εργαστηριακή Υποστήριξη

6. ΕΡΓΑΣΤΗΡΙΑΚΟ ΕΡΓΟ

6.1. Επίκουρος Καθηγητής επί θητεία Κλινικής Χημείας - Φαρμακευτικής Βιοχημείας, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Τομέας Υγείας Μητέρας Παιδιού, Β' Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική, Αρεταίειο Νοσοκομείο Αθηνών, Εργαστήριο Γενετικής Θεραπευτικής Εξατομίκευσης , Μαϊος 2009 - σήμερα

6.2. Επιστημονικός Συντονιστής Εργαστηρίου Γενετικής Θεραπευτικής Εξατομίκευσης, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Νοέμβριος 2007 - σήμερα

6.3. Λέκτορας Κλινικής Χημείας - Φαρμακευτικής Βιοχημείας, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Τομέας Υγείας Μητέρας Παιδιού, 2η Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική, Αρεταίειο Νοσοκομείο Αθηνών, Ορμονολογικό Εργαστήριο, Δεκέμβριος 2003 - 2008

6.4. Επιστημονικός Υπεύθυνος Εργαστηρίου Ραδιοϊσοτοπικών Αναλύσεων του Τμήματος Πυρηνικής Ιατρικής του Θεραπευτηρίου "ΥΓΕΙΑ", Φεβρουάριος 1998 - σήμερα

6.5. Νοσοκομειακός Φαρμακοποιός, Επιμελητής Γ' ΕΣΥ, Ιπποκράτειο Γενικό Νοσοκομείο

Αθηνών, Οκτώβριος 1995 - Νοέμβριος 2003.

6.6. Επιστημονικός Συνεργάτης (περιστασιακά) του Διαβητολογικού Κέντρου της Πρώτης Παιδιατρικής Κλινικής του Νοσοκομείου Παίδων "Π. και Α. ΚΥΡΙΑΚΟΥ", Οκτώβριος 1993 - Ιούνιος 1994

6.7. Επιστημονικός Συνεργάτης του Ερευνητικού Ανοσολογικού-Βιοχημικού Εργαστηρίου της Πανεπιστημιακής Καρδιολογικής Κλινικής του Ιπποκρατείου Γενικού Νοσοκομείου της Αθήνας, Φεβρουάριος 1992 – Νοέμβριος 2003

6.8. Επιστημονικός Συνεργάτης (περιστασιακά) του τμήματος Νεογνολογίας του Νοσοκομείου "ΑΛΕΞΑΝΔΡΑ", Φεβρουάριος 1990 - Μάρτιος 1994

7. ΝΟΣΟΚΟΜΕΙΑΚΕΣ ΚΑΙ ΠΑΝΕΠΙΣΤΗΜΙΑΚΕΣ ΘΕΣΕΙΣ

7.1. ΝΟΣΟΚΟΜΕΙΑΚΕΣ ΘΕΣΕΙΣ

7.1.1 Εργαστήριο Ραδιοϊσοτοπικών Αναλύσεων του Τμήματος Πυρηνικής Ιατρικής του Θεραπευτηρίου "ΥΓΕΙΑ", Επιστημονικός Υπεύθυνος, Φεβρουάριος 1998 - σήμερα.

7.1.2 Φαρμακευτική Υπηρεσία Ιπποκρατείου Γενικού Νοσοκομείου Αθηνών, Επιμελητής Γ', Οκτώβριος 1995 - Νοέμβριος 2003

7.1.3 Ανοσολογικό-Βιοχημικό Εργαστήριο της Πανεπιστημιακής Καρδιολογικής Κλινικής του Ιπποκρατείου Γενικού Νοσοκομείου της Αθήνας, Επιστημονικός Συνεργάτης, Φεβρουάριος 1992 – Δεκέμβριος 2003.

7.1.4 Διαβητολογικό Κέντρο της Πρώτης Παιδιατρικής Κλινικής του Νοσοκομείου Παίδων "Π.και Α. ΚΥΡΙΑΚΟΥ", Επιστημονικός Συνεργάτης, Οκτώβριος 1993 - Ιούνιος 1994.

7.1.5 Τμήμα Νεογνολογίας του Νοσοκομείου "ΑΛΕΞΑΝΔΡΑ", Επιστημονικός Συνεργάτης, Φεβρουάριος 1990 - Μάρτιος 1994.

7.2 ΠΑΝΕΠΙΣΤΗΜΙΑΚΕΣ ΘΕΣΕΙΣ

7.2.1. Εργαστήριο Γενετικής Θεραπευτικής Εξατομίκευσης, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Επιστημονικός Συντονιστής, Νοέμβριος 2007 - σήμερα

7.2.2. Ορμονολογικό Εργαστήριο Αρεταίειου Πανεπιστημιακού Νοσοκομείου Αθηνών, Β' Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική Ιατρικής Σχολής Πανεπιστημίου Αθηνών, Λέκτορας Κλινικής Χημείας – Φαρμακευτικής Βιοχημείας, Δεκέμβριος 2003 – 2008

8. ΕΡΕΥΝΗΤΙΚΟ ΕΡΓΟ

8.1. ΕΡΕΥΝΗΤΙΚΑ ΕΡΓΑΣΤΗΡΙΑ

8.1.1. Ερευνητικό Τμήμα Εργαστηρίου Γενετικής Θεραπευτικής Εξατομίκευσης, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Νοέμβριος 2007 – σήμερα

8.1.2. Ερευνητικό Τμήμα Ορμονολογικού Εργαστηρίου Β' Πανεπιστημιακής Μαιευτικής και Γυναικολογικής Κλινικής, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Δεκέμβριος 2003 – 2008

8.1.3. Ερευνητικό Τμήμα Εργαστηρίου Βιοχημείας – Ανοσολογίας Α' Πανεπιστημιακής Καρδιολογικής Κλινικής, Ιπποκράτειο Νοσοκομείο Αθηνών, Φεβρουάριος 1992 – Δεκέμβριος 2003

8.1.4. Ερευνητικό Τμήμα Ινστιτούτου Ραδιοδιαγνωστικών Προϊόντων – Ραδιοϊσοτόπων, Εργαστήριο Ραδιοϊσοτοπικών Ανοσοναλύσεων, Κέντρο Πυρηνικών Ερευνών «ΔΗΜΟΚΡΙΤΟΣ», 1985 – 1989

8.2. ΕΡΕΥΝΗΤΙΚΑ ΠΡΟΓΡΑΜΜΑΤΑ

8.2.1. ΔΙΕΘΝΗ ΕΡΕΥΝΗΤΙΚΑ ΠΡΟΓΡΑΜΜΑΤΑ

1. **"***Circulating biochemical parameters of endothelial dysfunction and apoptosis after renal artery stenting: implications in arterial wall injury***"**
Brountzos EN, Kelekis NL, Malagari K, **Economou E**, Kelekis AA
Research Grant from Cardiovascular and Interventional Radiological Society of Europe

8.2.2. ΕΛΛΗΝΙΚΑ ΕΡΕΥΝΗΤΙΚΑ ΠΡΟΓΡΑΜΜΑΤΑ

1. "*ΚΑΡΚΙΝΙΚΑ ΚΥΤΤΑΡΑ ΜΑΣΤΟΥ ΣΤΗ ΣΥΣΤΗΜΑΤΙΚΗ ΚΥΚΛΟΦΟΡΙΑ - Εντοπισμός του υποπληθυσμού με μεταστατικό δυναμικό και διερεύνηση της σχέσης της παρουσίας τους με κυκλοφορούντες δείκτες αποπτωτικής και νεοαγγειογενετικής ομοιοστασίας*"
Εμμανουήλ Β. Οικονόμου, Ευαγγελία Κουσκούνη
Ερευνητικό Πρόγραμμα "ΚΕΝΤΡΟΥ ΕΛΕΓΧΟΥ ΚΑΙ ΠΡΟΛΗΨΗΣ ΝΟΣΗΜΑΤΩΝ", Εγκριση από την Επιστημονική Επιτροπή (13.03.2008), Υπογραφή Συμβάσεως (0.1.09.2008)

2. "*ΚΑΡΚΙΝΙΚΑ ΚΥΤΤΑΡΑ ΜΑΣΤΟΥ ΣΤΗ ΣΥΣΤΗΜΑΤΙΚΗ ΚΥΚΛΟΦΟΡΙΑ - Εντοπισμός του υποπληθυσμού με μεταστατικό δυναμικό και διερεύνηση της σχέσης της παρουσίας τους με κυκλοφορούντες δείκτες αποπτωτικής και νεοαγγειογενετικής ομοιοστασίας*"
Εμμανουήλ Β. Οικονόμου, Ευαγγελία Κουσκούνη
Ερευνητικό Πρόγραμμα "ΚΕΝΤΡΟΥ ΕΛΕΓΧΟΥ ΚΑΙ ΠΡΟΛΗΨΗΣ ΝΟΣΗΜΑΤΩΝ", Εγκριση από την Επιστημονική Επιτροπή (13.03.2008), Υπογραφή Συμβάσεως (0.1.09.2008)

3. "*ΓΕΝΕΤΙΚΗ ΒΑΣΗ ΤΗΣ ΕΝΔΟΜΗΤΡΙΩΣΕΩΣ – Διερεύνση της σχέσης της γενετικής βάσης της αθηρωσκλήρυνσης, του οξειδωτικού stress και της ανοσολογικής απάντησης στην ανάπτυξη της νόσου και στη διασπορά στη συστηματική κυκλοφορία της συνοδού ενδοπεριτοναϊκής φλεγμονής*"
Ευαγγελία Κουσκούνη, **Εμμανουήλ Β. Οικονόμου**
Ερευνητικό Πρόγραμμα "ΚΕΝΤΡΟΥ ΕΛΕΓΧΟΥ ΚΑΙ ΠΡΟΛΗΨΗΣ ΝΟΣΗΜΑΤΩΝ", Αρ. Πρωτ. Εγκ. 1460/4-5-2007

4. "*Ταυτοποίηση της στεφανιογραφικά επιβεβαιωμένης στεφανιαίας νόσου από την επαγωγή κυτταροπροστατευτικών ορολογικών παραγόντων και τις μεταβολες υπερηχοκαρδιογραφικών δεικτών κατά τη δοκιμασία φόρτισης με δοβουταμίνη"* Χρήστος Πίτσαβος, **Εμμανουήλ Β. Οικονόμου**
Ερευνητικό Πρόγραμμα "ΚΑΠΟΔΙΣΤΡΙΑΣ", Αρ. Πρωτ. Εγκ. 1219/13-4-2007,
Αριθ. Προγράμματος 70/4/9151

5. "*Βιοχημικοί δείκτες επαγωμένης αντιοξειδωτικής κυτταρικής προστασίας και διαταραχής της λειτουργίας του αγγειακού ενδοθηλίου στη συστηματική κυκλοφορία εγκύων γυναικών κατά τη διάγνωση της προεκλαμψίας και μετά το πέρας της κυήσεως*"
Εμμανουήλ Β. Οικονόμου
Ερευνητικό Πρόγραμμα "ΚΑΠΟΔΙΣΤΡΙΑΣ", Αρ. Πρωτ. Εγκ. 1219/13-4-2007,

Αριθ. Προγράμματος 70/4/8143

6. "*Μελέτη της επίδρασης της ορμονικής κατάστασης των λιπιδαιμικών γυναικών στην αντιλιπιδαιμική θεραπεία με στατίνες*"
Κωνσταντίνος Παπαδιάς, **Εμμανουήλ Β. Οικονόμου**
Ερευνητικό Πρόγραμμα "ΚΑΠΟΔΙΣΤΡΙΑΣ", Αρ. Πρωτ. Εγκ. 1219/13-4-2007, Αριθ. Προγράμματος 70/4/8181

7. "*Βιοχημική διερεύνηση της επίδρασης των ενδομορφινών της συστηματικής κυκλοφορίας στην αποπτωτική ομοιοστασία και στο αγγειακό ενδοθήλιο γυναικών με ενδοπυελική ενδομητρίωση*"
Αντώνιος Κοντοράβδης, **Εμμανουήλ Β. Οικονόμου**
Ερευνητικό Πρόγραμμα "ΚΑΠΟΔΙΣΤΡΙΑΣ", Αρ. Πρωτ. Εγκ. 1219/13-4-2007, Αριθ. Προγράμματος 70/4/9010

8. "*Βιοχημική διερεύνση της επίπτωσης της προεκλαμπτικής κυήσεως στην αναστολή της οστεοκλαστικής δραστηριότητας της εγκύου*"
Νικόλαος Βιτωράτος, **Εμμανουήλ Β. Οικονόμου**
Ερευνητικό Πρόγραμμα "ΚΑΠΟΔΙΣΤΡΙΑΣ", Αρ. Πρωτ. Εγκ. 1219/13-4-2007, Αριθ. Προγράμματος 70/4/9004

9. "*Βιοχημικοί δείκτες ψευδοαγγειογένεσης, αγγειογένεσης και αγγειοποίησης στο πρώτο και στο δεύτερο τρίμηνο της προεκλαμπτικής κύησης*"
Εμμανουήλ Β. Οικονόμου, Δημήτριος Ρίζος, Δημήτριος Χασιάκος
Ερευνητικό Πρόγραμμα "ΚΑΠΟΔΙΣΤΡΙΑΣ", Αρ. Πρωτ. Εγκ. 1664/28-6-2005

10. "*Βιοχημικοί δείκτες πλακουντιακής ισχαιμίας στην εγκατεστημένη προεκλαμψία και στην πρώτη περίοδο λοχείας*"
Οικονόμου Εμμανουήλ, Βιτωράτος Νικόλαος, Κουσκούνη Ευαγγελία, Παπαδιάς Κωνσταντίνος
Επιχορήγηση Ερευνας από την Ιατρική Σχολή του Πανεπιστημίου Αθηνών, Αρ. Πρωτ. 10241/23-6-2005

11. "*Μελέτη της Επίδρασης της Ορμονικής Κατάστασης των Δυσλιπιδαιμικών Γυναικών στην Αντιλιπιδαιμική Θεραπεία με Στατίνες*"
Παπαδιάς Κωνσταντινίνος, **Οικονόμου Εμμανουήλ**, Κουσκούνη Ευαγγελία, Γρηγορίου Οδυσσέας
Επιχορήγηση Ερευνας από την Ιατρική Σχολή του Πανεπιστημίου Αθηνών, Αρ. Πρωτ. 10241/23-6-2005

12. **"***Διερεύνηση πιθανής θυμο-εξαρτώμενης διέγερσης λευκοκυττάρων του αίματος ως ένδειξη συστηματικής ανοσολογικής και φλεγμονώδους αντίδρασης στην ενδοπυελική ενδομητρίωση***"**
Οικονόμου ΕΒ, Κουσκούνη ΕΕ, Γρηγορίου Ο, Βιτωράτος Ν
Επιχορήγηση Έρευνας από την Ιατρική Σχολή του Πανεπιστημίου Αθηνών, Αρ. Πρωτοκολ. 10996/14-07-2004

13. **"***Βιοχημική διερεύνηση της επίπτωσης της χρονίας, συστηματικής άσηπτης φλεγμονώδους αντίδρασης της μητέρας στον οστικό μεταβολισμό της στην προεκλαμπτική κύηση***"**
Γρηγορίου Ο**, Οικονόμου ΕΒ,** Κουσκούνη ΕΕ, Βιτωράτος Ν
Επιχορήγηση Έρευνας από την Ιατρική Σχολή του Πανεπιστημίου Αθηνών, Αρ. Πρωτοκολ. 10996/14-07-2004

14. **"***Αγγειογενετικοί και αντιαγγειογενετικοί παράγοντες σε νεογνά με ενδομήτρια καθυστέρηση ανάπτυξης***»**

Μαλαμίτση-Πούχνερ Αριάδνη, **Οικονόμου Εμμανουήλ,** Μπούτσικου Θεοδώρα, Κουσκούνη Ευαγγελία
Επιχορήγηση Έρευνας από την Ιατρική Σχολή του Πανεπιστημίου Αθηνών, Αρ. Πρωτοκολ. 10996/14-07-2004

8.3. **ΠΕΡΙΓΡΑΦΗ ΕΡΕΥΝΗΤΙΚΟΥ ΕΡΓΟΥ**

8.3.1. Διαμόρφωση Ερευνητικής Ιδέας – Προτάσεως

8.3.2. Βιβλιογραφική Ενημέρωση

8.3.3. Επιλογή Είδους και Τύπου Ερευνητικής Μεθοδολογίας

8.3.4. Διαμόρφωση Ερευνητικού Ερωτηματολογίου

8.3.5. Επιλογή Ομάδας Μελέτης

8.3.6. Επιλογή Βιολογικου Δείγματος / Αναλυτικής Μεθοδολογίας

8.3.7. Ανάλυση Δειγμάτων

8.3.8. Αναλυτική Αξιολόγηση Αποτελεσμάτων

8.3.9. Βιολογική Αξιολόγηση Αποτελεσμάτων

8.3.10. Ποιοτικός Ελεγχος Αποτελεσμάτων

8.3.11. Στατιστική Επεξεργασία Αποτελεσμάτων

8.3.12. Ερμηνεία Αποτελεσμάτων

8.3.13. Συγγραφή Περιλήψεων

8.3.14. Συγγραφή Άρθρων που δημοσιεύθηκαν σε Διεθνή Περιοδικά

8.3.15. Διασπορά Αποτελεσμάτων (Διαλέξεις-Εισηγήσεις-Δημοσιεύσεις)

9. ΒΡΑΒΕΙΑ – ΔΙΑΚΡΙΣΕΙΣ – ΥΠΟΤΡΟΦΙΕΣ

9.1. 7^{ο} Πανελλήνιο Συνέδριο Παγκρεατολογίας, CROWNE PLAZA HOTEL, Αθήνα, 3-5 Απριλίου 2009
ΒΡΑΒΕΙΟ ΕΛΕΥΘΕΡΗΣ ΑΝΑΚΟΙΝΩΣΗΣ με τίτλο «Ιστολογική και λειτουργική διερεύνηση παγκρεατικής λειτουργίας σε πειραματικό μοντέλο ηπατεκτομής και ισχαιμίας-επαναιμάτωσης ήπατος»
Κ. Νάστος, Ν. Αρκαδόπουλος, Γ. Δευτερέβος, Ν. Παπουτσιδάκης, Α. Κόνδη-Παφίτη, Γ. Φραγκουλίδης, **Ε. Οικονόμου**, Ι. Ανδρεάδου, Τ. Νομικός, Γ. Κωστοπαναγιώτου, Β. Σμυρνιώτης

9.2. 10ο Πανελλήνιο Ηπατολογικό Συνέδριο, 26-29 Απριλίου, Hilton, Αθήνα, 2007
1ο ΒΡΑΒΕΙΟ ΚΑΛΥΤΕΡΗΣ ΕΛΕΥΘΕΡΗΣ ΑΝΑΚΟΙΝΩΣΗΣ με τίτλο «Αντιμετώπιση οξειδωτικών βλαβών κατά τις μείζονες ηπατεκτομές με χρήση δεσφεροξαμίνης. Προοπτική τυχαιοποιημένη μελέτη».
Ν. Αρκαδόπουλος, **Ε. Οικονόμου**, Κ. Θεοδωράκη, Γ. Βασιληκώστας, Κ. Καραπάνος, Α. Παφίτη, Ε. Κουσκούνη, Ι. Βασιλείου, Δ. Βώρος, Β. Σμυρνιώτης

9.3. Admission to **Membership of the "International Order of Merit"**, 10 June 2007

9.4. Announcement, Recognition and Inclusion by the International Biographical Association, Cambridge, England, in "Outstanding Scientists of the 21st Century – Inaugural Edition-", March 2007

9.5. Election as **"Life Fellow of the International Biographical Association"** Headquarters in England, March 2007

9.6. Selection by American Biographical Institute as a "Great Mind of the 21st Century", 23 February 2007

9.7. Election by the International Biographical Association, Cambridge, England, as "International Health Professional of the Year 2007", 16 January 2007

9.8. Ανεξάρτητη Επιλογή Δημοσίευσης Βιογραφικού Σημειώματος στη Νέα 6η Εκδοση (2006-2007) της Αρχαιότερης Επετηρίδας του Κόσμου **"Marquis Who's Who in Medicine and Healthcare"**

9.9. Ινστιτούτο Πυρηνικής Ιατρικής, Υποτροφία Μεταδιδακτορικών Σπουδών, Kern Forschungsanlage, Juliech, Γερμανία, 1989 - 1991. Βεβαίωση από το αντίστοιχο Ινστιτούτο

9.10. Ελληνική Επιτροπή Ατομικής και Πυρηνικής Ενέργειας, Υποτροφία Μεταπτυχιακών Σπουδών, κατόπιν εξετάσεων, με σκοπό της εκπόνηση Διδακτορικής Διατριβής στο Εργαστήριο Ραδιοανοσοχημείας του Ερευνητικού Κέντρου Πυρηνικών Ερευνών "ΔΗΜΟΚΡΙΤΟΣ", 1985 - 1989.

9.11. Ιδρυμα Κρατικών Υποτροφιών, Υποτροφίες για τα Ακαδημαϊκά Ετη 1980 - 1981, 1981-1982, 1982 - 1983, 1983 - 1984.

10. ΜΕΛΟΣ ΕΠΙΣΤΗΜΟΝΙΚΩΝ ΕΤΑΙΡΕΙΩΝ

10.1. European Society of Cardiology (F.E.S.C.) (κατόπιν επιλογής), 2004 - σήμερα

10.2. American Association for Clinical Chemistry (F.A.A.C.C.) (κατόπιν επιλογής), 2001 - σήμερα

11. ΣΥΝΤΑΚΤΙΚΟ ΕΡΓΟ

11.1. ΜΕΛΟΣ ΣΥΝΤΑΚΤΙΚΗΣ ΕΠΙΤΡΟΠΗΣ ΠΕΡΙΟΔΙΚΩΝ

11.1.1. Μόνιμο Μέλος της Ομάδας Διεθνών Κριτών (κατόπιν επιλογής) του περιοδικού "Medical Science Monitor", Τελική Επιλογή : 16.08.2005.

11.2. ΣΥΜΒΟΥΛΟΣ ΣΥΝΤΑΚΤΙΚΗΣ ΕΠΙΤΡΟΠΗΣ ΕΠΙΣΤΗΜΟΝΙΚΩΝ ΠΕΡΙΟΔΙΚΩΝ

11.2.1. *"Opposing Roles of Leptin and Ghrelin in the Equine Corpus Luteum Regulation: an in vitro study"*
Αποστολή προς κρίση 18.05.2014 για το περιοδικό **" Mediators of Inflammation "**

11.2.2. *"Etanercept and pregnancy loss in lipopolysaccharide-induced endotoxemic rats"*
Αποστολή προς κρίση 16.4.2014 για το περιοδικό **" Mediators of Inflammation "**

11.2.3. **"***Up-regulation of chemokine receptor CXCR4 and caspase-dependent apoptosis genes can synergistically favour activation of endothelial cells exposed to low shear stress*"
Αποστολή προς κρίση 01.04.2013 για το περιοδικό **"Journal of Cellular Physiology"**

11.2.4. *"Decidual endothelial cells exhibit a distinguishing profile of angiogenetic factors, chemokines and adhesion molecules"*
Αποστολή προς κρίση 25.11.2013 για το περιοδικό **" Mediators of Inflammation "**

11.2.5. **"***Mannose-binding lectin deficiency is associated with myocardial infarction: the HUNT2 Study in Norway*"
Αποστολή προς κρίση 12.02.2012 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.6. " Endothelial Progenitor Cell Biology in Cardiovascular Diseases – Role of Reactive Oxygen Species and Inflammation "
Αποστολή προς κρίση 12.02.12 για το περιοδικό "**Mediators of Inflammation**"

11.2.7. **"**Endothelial dysfunction in autoimmune diseases"
Αποστολή προς κρίση 19.10.2011 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.8. **"***Aortic Arch Inflammation in Psoriasis is Associated with HDL Particle Concentration*"
Αποστολή προς κρίση 15.07.2011 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.9. **"***Different pathophysiology of microparticle shedding in patients with acute coronary syndrome and stable angina*"
Αποστολή προς κρίση 10.04.2011 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.10. **"***Interleukin-6 in the prediction of cardiovascular events in diabetes patients: results from the ESTHER study*"
Αποστολή προς κρίση 30.11.2010 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.11. **"***B<beta>-Fibrinogen gene promoter A-455 allele associates with severe coronary artery stenosis in victims of sudden prehospital death*"
Αποστολή προς κρίση 26.10.2010 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.12. **"***Plasma haptoglobin level is associated with long-term mortality according to haptoglobin phenotype in non-diabetic acute myocardial infarctions*"
Αποστολή προς κρίση 02.09.2010 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.13. **"***Mean Platelet Volume and Prevalence of Peripheral Artery Disease, the National Health and Nutrition Examination Survey, 1999 to 2004*"
Αποστολή προς κρίση 25.06.2010 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.14. "Circulating Lymphotoxin Beta Receptor and Atherosclerosis: Observations from the Dallas Heart Study"
Αποστολή προς ΔΕΥΤΕΡΗ κρίση 27.04.2010 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.15. **"***Circulating Lymphotoxin Beta Receptor and Atherosclerosis: Observations from the Dallas Heart Study"*
Αποστολή προς κρίση 15.02.2010 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.16. **"***The influence of persistent pathogens on circulating levels of inflammatory markers: The Multi-Ethnic Study of Atherosclerosis"*
Αποστολή προς κρίση 02.10.2009 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.17. *"Effects of pravastatin and rosuvastatin on thw generation of adiponectin in thw visceral adipose tissue in patients with coronary artery disease"*
Αποστολή προς κρίση 09.08.2009 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.18. "*Associations of four novel chemokines with multiple atherosclerosis phenotypes in a large population based sample: results from the Dallas Heart Study*"
Αποστολή προς κρίση 28/3/2009 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.19. "*Women with endometriosis showed a diminished synthesis of IL-2 and IFN-γ bt T-lymphocytes in comparison with women without endometriosis: after antioxidant supplementation an improvement in both cytokines was observed*".

Αποστολή προς κρίση 13-3-2009 για το περιοδικό "**Mediators of Inflammation**"

11.2.20. "*Serum amyloid A induction of cytokines in monocytes/macrophages and lymphocytes*".
Αποστολή προς ΔΕΥΤΕΡΗ κρίση 23/2/2009 για το περιοδικό "**ATHEROSCLEROSIS**"

11.2.21. "*Serum amyloid A induction of cytokines in monocytes/macrophages and lymphocytes*".
Αποστολή προς ΤΡΙΤΗ κρίση 15/1/2009 για το περιοδικό "**ATHEROSCLEROSIS"**

11.2.22. "*Usefulness of haptoglobin and serum amyloid A proteins as biomarkers for atherothrombotic ischemic stroke diagnosis confirmation*".
Αποστολή προς κρίση 14/12/2008 για το περιοδικό "**ATHEROSCLEROSIS**"

11.2.23. "*Usefulness of haptoglobin and serum amyloid A proteins as biomarkers for ischemic stroke subtype classification*"
Αποστολή προς ΔΕΥΤΕΡΗ κρίση 09.12.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.24. *"Circulating sTWEAK improves the prediction of coronary artery disease "*
Αποστολή προς ΤΡΙΤΗ κρίση 07.12.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.25. *"Lower Levels of ADAMTS13 are Associated with cardiovascular disease in young patients" has been submitted to be considered for publication in this journal"*
Αποστολή προς κρίση 30.11.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.26. *"Circulating sTWEAK improves the prediction of coronary artery disease "*
Αποστολή προς ΔΕΥΤΕΡΗ κρίση 23.11.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.27. *"Activation of diacylglycerol-protein kinase C and inositol triphosphate –Ca2+ signal pathway induced by OX40-OX40L ligand interaction in human umbilical vein endothelial cells"*
Αποστολή προς ΔΕΥΤΕΡΗ κρίση 16.11.2008 για το περιοδικό **"Mediators of Inflammation "**

11.2.28. *"The up-regulation of monocyte chemoattractant proteinh-1 (MCP-1) in endothelial cells maintained under low folate stress is mediated by the p38 MAPK pathway"*
Αποστολή προς ΤΡΙΤΗ κρίση 14.11.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.29. **"***Serum amyloid A is independently associated with metabolic risk factors but not with early atherosclerosis: The cardiovascular Risk in Young Fiins Study"*
Αποστολή προς κρίση 13.11.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.30. **"***Activation of blood coagulation and platelets in patients with aortic stenosis is independent from vascular atherosclerotic burden"*
Αποστολή προς κρίση 23.10.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.31. "*Usefulness of haptoglobin and serum amyloid A proteins as biomarkers for ischemic stroke subtype classification*"
Αποστολή προς κρίση 22.10.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.32. *"The up-regulation of monocyte chemoattractant protein-1 (MCP-1) in endothelial cells maintained under low folate stress is mediated by the p38 MAPK pathway"*
Αποστολή προς ΔΕΥΤΕΡΗ κρίση 03.10.2008 για το περιοδικό

"ATHEROSCLEROSIS"

11.2.33. "*Activation of diacylglycerol-protein kinase C and inositol triphosphate –Ca2+ signal pathway induced by OX40-OX40L ligand interaction in human umbilical vein endothelial cells*"
Αποστολή προς κρίση 25.09.2008 για το περιοδικό **"Mediators of Inflammation "**

11.2.34. **"***Circulating sTWEAK measurement improves the prediction of coronary artery disease"*
Αποστολή προς κρίση 30.07.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.35. *"Circulating sTWEAK improves the prediction of coronary artery disease "*
Αποστολή προς κρίση 29.06.2008 για το περιοδικό **"ATHEROSCLEROSIS"**

11.2.36. **"***The up-regulation of monocyte chemoattractant proteinh-1 (MCP-1) in endothelial cells maintained under low folate stress is mediated by the p38 MAPK pathway"*
*A*ποστολή προς κρίση 09.06.2008 για το περιοδικό **"ATHEROSCLEROSIS".**

11.2.37. "*Correlation between serum erythropoietin and plasma endothelin-1 during the menstrual cycle*"
Αποστολή προς κρίση 22.04.2008 για το περιοδικό "**Mediators of Inflammation**"

11.2.38. "*The influence of ibuprofen on aortal expression of the MCP-1 gene in rabbits with experimental atherosclerosis*"
Αποστολή προς κρίση 08.11.2004 για το περιοδικό "**Biochemical Pharmacology**"

11.2.39. "*Παθογενετικοί μηχανισμοί των συγγενών καρδιακών ανωμαλιών*"
Αποστολή προς κρίση 18.02.1999 για το περιοδικό "**Ελληνική Καρδιολογική Επιθεώρηση**".

11.2.40. "*Προσδιορισμός των διαλυτών μορφών των κυτταρικών προσκολλητικών μορίων σε υπερτασικούς ασθενείς με ή χωρίς σημαντική υπερλιπιδαιμία*"
Αποστολή προς κρίση 04.09.1998 για το περιοδικό "**Ελληνική Καρδιολογική Επιθεώρηση**"

11.2.41. "*Μελέτη της έκκρισης PAF-ακετυλοϋδρολάσης από αιμοπετάλια ασθενών που υποβάλλονται σε αγγειοπλαστική στεφανιαίων κατά την ενεργοποίηση τους με θρομβίνη*"
Αποστολή προς κρίση 04.08.1998 για το περιοδικό "**Ελληνική Καρδιολογική Επιθεώρηση**"

11.2.42. "*Παράγοντες της φλεγμονής στο οξύ έμφραγμα του μυοκαρδίου*" Αποστολή προς κρίση 08.05.1998 για το περιοδικό "**Ελληνική Καρδιολογική Επιθεώρηση**".

11.2.43. "*Εκτίμηση των επιπέδων των διαλυτών μορφών των αποπτωτικών υποδοχέων Fas και TNFRI στη συμφορητική καρδιακή ανεπάρκεια*"
Αποστολή προς κρίση 14.01.1998 για το περιοδικό "**Ελληνική Καρδιολογική Επιθεώρηση**"

11.2.44. "*Ελεύθερες ρίζες οξυγόνου και ο ρόλος τους στη γήρανση του ανθρώπου*"
Αποστολή προς κρίση 16.09.1997 για το περιοδικό "**Αρχεία Ελληνικής Ιατρικής**"

11.2.45. "*Λιπιδική υπεροξείδωση και ενζυμική διέγερση σε καλλιέργεια καρδιακών κυττάρων νεογέννητων επίμυων μετά από χορήγηση νταουνομυκίνης*"
Αποστολή προς κρίση 31.07.1996 για το περιοδικό "**Ελληνική Καρδιολογική Επιθεώρηση**"

11.3. ΜΟΝΟΓΡΑΦΙΕΣ – ΚΕΦΑΛΑΙΑ ΣΕ ΒΙΒΛΙΑ

11.3.1. "Νεότερα δεδομένα από τη μοριακή βιολογία της στεφανιαίας νόσου : Αθηρωσκλήρυνση και Αυτοανοσία"
Εμμανουήλ Β. Οικονόμου
Καρδιολογικά Θέματα 2001, 383-408, 2001.
Κεφάλαιο στον ειδικό τόμο με τα θέματα του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών κατά το Ακαδημαϊκό Ετος 2000-2001.

11.3.2. "Υπέρταση και ασυμπτωματικές βλάβες στον εγκέφαλο"
Εμμανουήλ Β. Οικονόμου
Καρδιολογικά Θέματα 1999 : 794-811, 1999.
Κεφάλαιο στον ειδικό τόμο με τα θέματα του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών κατά το Ακαδημαϊκό Ετος 1998-1999.

11.3.3. "Κυτταροχυμικοί και νευρορμονικοί μηχανισμοί στην παθοφυσιολογία της καρδιακής ανεπάρκειας"
Ε.Β. Οικονόμου, Π.Κ. Τούτουζας.
Καρδιολογικά Θέματα 1998 : 115-133, 1998.
Κεφάλαιο στον ειδικό τόμο με τα θέματα του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών κατά το Ακαδημαϊκό Ετος 1997-1998

11.3.4. "Ισχαιμική προετοιμασία του μυοκαρδίου (ischaemic preconditioning). Ανασκόπηση των γενικών χαρακτηριστικών και της μοριακής βάσης του φαινομένου"
Ε.Β. Οικονόμου, Π.Κ. Τούτουζας
Καρδιολογικά Θέματα 1997 : 309-324, 1997.
Κεφάλαιο στον ειδικό τόμο με τα θέματα του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών κατά το Ακαδημαϊκό Ετος 1996-1997.

11.3.5. "Ρόλος και in vitro εκτίμηση της λειτουργίας του ενδοθηλίου στην Καρδιολογία"
Ε.Β. Οικονόμου
Καρδιολογικά Θέματα 1995 : 493-513, 1995.
Κεφάλαιο στον ειδικό τόμο με τα θέματα του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών κατά το Ακαδημαϊκό Ετος 1994-1995.

11.3.6. "Λειτουργία των ενδοθηλιακών κυττάρων των αγγείων στην αρτηριακή υπέρταση και στο σακχαρώδη διαβήτη. Ρόλος της ομοιόστασης του ελεύθερου ενδοκυττάριου ασβεστίου στα κύτταρα αυτά".
Ε.Β. Οικονόμου
Καρδιολογικά Θέματα 1995 : 515-537, 1995.
Κεφάλαιο στον ειδικό τόμο με τα θέματα του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών κατά το Ακαδημαϊκό Ετος 1994-1995.

11.3.7. "Ο ρόλος του ενδοθηλίου των στεφανιαίων αγγείων στη στεφανιαία νόσο"
Ε.Β. Οικονόμου
Καρδιολογικά Θέματα 1994 : 483-504, 1994.
Κεφάλαιο στον ειδικό τόμο με τα θέματα του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών κατά το Ακαδημαϊκό Ετος 1993-1994.

11.3.8. "Ραδιοανοσοαναλύσεις για τον προσδιορισμό της διαιθυλοστιλβοιστρόλης στον ορό και αξιολόγηση αυτών"
Εμμανουήλ Β. Οικονόμου
Διδακτορική Διατριβή, Ιούνιος 1993

11.3.9. "Η Βιοχημεία των Ελευθέρων Ριζών Οξυγόνου"
Εμμανουήλ Β. Οικονόμου
"Ελεύθερες Ρίζες Οξυγόνου", σελίδες 9-45,1993.
Κεφάλαιο στον ειδικό τόμο με τα θέματα Δορυφορικού Συμποσίου που πραγματοποιήθηκε στα πλαίσια του 19ου Πανελληνίου Ιατρικού Συνεδρίου, 4 - 8 Μαϊου, Αθήνα 1993.

12. ΟΡΓΑΝΩΤΙΚΟ ΚΑΙ ΔΙΟΙΚΗΤΙΚΟ ΕΡΓΟ

12.1. Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών – **Εμπνευστής και Δημιουργός Προτύπου Εργαστηρίου Γενετικής Θεραπευτικής Εξατομίκευσης** – Κλινική Νοσοειδική Φαρμακογενωμική, Νοέμβριος 2007 - σήμερα

Οργάνωση, Κοστολόγηση, Δρομολόγηση, Διαχειριστική Ένταξη στις Νοσοκομειακές Διαδικασίες, Ενταξη και Εφαρμογή στην Εργαστηριακή Πρακτική εξετάσεων:

12.1.1.
- Ελεγχος Γενετικής Θρομβοφιλίας [Μοριακά Προσιοριζόμενη Γενετική Θρομβοφιλία (Παράγοντας V (Leiden) G1691A, Προθρομβίνη II G20210A, MTHFR A1298C, MTHFR C677T, PAI-1 5G/4G, PAI-1, γλυκοπρωτεϊνη IIB/IIIA (GpIIB/IIIA)]
- Βιοχημικά Προσδιοριζόμενη Γενετική Θρομβοφιλία [Ανεπάρκειες Πρωτεϊνών C, S, free S, Z, Αντιθρομβίνης III)
- Έλεγχος Δραστικότητας Αιμοπεταλίων (Διαφορική Διάγνωση Φαρμακογενούς και Οικογενούς Διαταραχής Δραστικότητας Αιμοπεταλίων, Ελεγχος Αποκρισιμότητας στην αντιθρομβωτική Θεραπεία Αντιαιμοπεταλιακού τύπου)]

Αριθμητική και Θεραπευτική Αλγοριθμοποίηση του Αποτελέσματος (ένα αριθμοποιημένο αποτέλεσμα για το σύνολο των αποτελεσμάτων θρομβοφιλίας προς ευχερή εξατομίκευση της φαρμακευτικής θεραπείας και προσέγγισης του ασθενούς από το θεράποντα ιατρό – Διάκριση Ανεπαρκειών τύπων I, II και III προς αποτελεσματικότερη θεραπευτική αντιμετώπιση

12.1.2.
- Ελεγχος Γενετικής Αιμορροφιλίας – Βιοχημικά Προσδιοριζόμενες Ανεπάρκειες Παραγόντων II, V, VII, VIII, IX, X, XI, XII, XIII, vW

Αριθμητική και Θεραπευτική Αλγοριθμοποίηση του Αποτελέσματος (ένα αριθμοποιημένο αποτέλεσμα για το σύνολο των αποτελεσμάτων από αιμορροφιλίας από ευχερή εξατομίκευση από φαρμακευτικής θεραπείας και προσέγγιση του ασθενούς από το θεράποντα ιατρό

12.1.3.
- Ελεγχος Επίκτητης Θρομβοφιλίας – Βιοχημικός Προσδιορισμός Παρουσίας Αντιπηκτικών Λύκου (αδρός ποιοτικός έλεγχος παρουσίας αντισωμάτων και επιβεβαιωτικός έλεγχος), IgG, IgM και IgA αντισώματα έναντι καρδιολιπίνης, IgG και IgM αντισώματα έναντι φωσφατιδυλοσερίνης και έναντι β2-γλυκοπρωτεϊνης καθώς και ολικά αντισώματα έναντι της φωσφατιδυλοαιθανολαμίνης

12.1.4. Ελεγχος Φαινοτυπικής Θρομβοφιλίας – Βιοχημικός έλεγχος APTT, PT, TT, INR, πλασμινογόνο, ινωδογόνο, D-Dimmers, APC-R

12.1.5. Ποσοτικος Προσδιορισμος Αναστολης Παραγοντα Xa Επι Χορηγησεως Ακλασματοποιητης Ηπαρινης (UFH) - Ηπαρινης Χαμηλου Μοριακου βαρους (LMWH) ή Δια του στόματος χορήγησης αναστολέα

12.1.6. Ελεγχος δραστικότητας αιμοπεταλίων – Απόκριση στην αντιαιμοπεταλιακή θεραπεία με ακετυλοσαλικυλικό οξύ ή κλοπιδογρέλη

12.1.7. Ελεγχος Γενετικής Οστεοπορώσεως - Αριθμητική και Θεραπευτική Αλγοριθμοποίηση του Αποτελέσματος (ένα αριθμοποιημένο αποτέλεσμα για το σύνολο των αποτελεσμάτων θρομβοφιλίας προς ευχερή εξατομίκευση της φαρμακευτικής θεραπείας και προσέγγισης του ασθενούς από το θεράποντα ιατρό

12.1.8. Ελεγχος Γενετικής Αθηρωσκλήρυνσης - Αριθμητική και Θεραπευτική Αλγοριθμοποίηση του Αποτελέσματος (ένα αριθμοποιημένο αποτέλεσμα για το σύνολο των αποτελεσμάτων θρομβοφιλίας προς ευχερή εξατομίκευση της φαρμακευτικής θεραπείας και προσέγγισης του ασθενούς από το θεράποντα ιατρό

12.1.9. Ελεγχος Γενετικής Βάσης Αζωοσπερμικού Παράγοντα – 22 μικροελλείψεις – Γενετική βάση ανδρικής υπογονιμότητας – Έλεγχος Διαταραχών Σπερματογένεσης – Καθέξιν Αποβολές

12.1.10. Ελεγχος Υπολειματικής Νόσου σε συμπαγείς καρκίνους Μαστού, Πνεύμονα, Παχέος Εντέρου, Προστάτη, Ωοθηκών – Προσδιορισμός κυκλοφορούντων καρκινικών κυτάρων σε συγκεντρώσεις έως και 2 κύτταρα ανά 5 ml αίματος – Μοριακή Ταυτοποίηση – Ημιποσοτικοποίηση Αποτελέσματος – Προσδιορισμός φαρμακευτικής Θεραπείας από τη Μοριακή Ταυτότητα της Υπολειματικής Νόσου – Παρακολούθηση Αλλαγής Μοριακής Ταυτότητας κατά τη Διάρκεια της Φαρμακευτικής Θεραπευτικής Αντιμετωπίσεως (Εντοπισμός Χημειοαντοχής) – Αξιολόγηση Ανάγκης Επικουρικής Φαρμακευτικής Θεραπείας – Αξιολόγηση Αποκρισιμότητας στη Φαρμακευτική Θεραπεία

12.1.11. Ανοσοφαινοτύπηση – Θρομβοφαινοτύπηση - Θρομβοανοσοφαινοτύπηση – Μελέτη έκφρασης γλυκοπρωτεϊνικών υποδοχέων και μορίων προσκόλλησης στην επιφάνεια της κυτταροπλασματικής μεμβράνης των αιμοπεταλίων καθώς και μορίων προσκόλλησης ή μορίων ταυτοποίησης ή μορίων διέγερσης στην επιφάνεια της κυτταροπλασματικής μεμβράνης των λευκοκυττάρων και των υποπληθυσμών τους

12.1.12. Γενετικός έλεγχος Δυσανεξίας στα Σάκχαρα – Γενετικός έλεγχος 6 πολυμορφισμών για τη διαπίστωση γενετικής διαταραχής στην πέψη των σακχάρων σε νεογνά

12.1.13. Ελεγχος Εμβρυομητρικής Αιμορραγίας

12.1.14. Ελεγχος Λειτουργικης Βασής Αντιστασης Στη Θεραπεια Με Κλοπιδογρελη

12.1.15. Ελεγχος Μεταβολισμού Θειοπουρινών (Γενετικός και Βιοχημικός)

12.1.16. Γενετικος Ελεγχος Τυποποίησης HLA (Class I – Class II)

12.1.17. Ελεγχος Παρούσιας Γονιδίων Αναλόγων Των Ανοσοσφαρινών Υποδοχέων Φυσικών Κυτταροκτόνων (KIR)

12.2. **Member of Scientific Commitee,** The 9th Athens Congress on Women's Helath and Disease "From Puberty to Menopause", Athens Hilton, August 28-30, 2014, Greece

12.3. **Μέλος της Επιστημονικής Επιτροπής του** 6ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 4-5 Απριλίου 2014, Ξενοδοχείο Royal Olympic, Αθήνα

12.4. **Member of Local Advisory Committee**, 8th Congress of Women's Health & Disease, Kos, September 1 – 3, 2011, Greece

12.5. **Μέλος της Οργανωτικής Επιτροπής του** 5ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 1-2 Απριλίου 2011, Ξενοδοχείο Stratos Vasilikos, Αθήνα

12.6. **Μέλος της Οργανωτικής Επιτροπής του** 2ου Πανελληνίου Συνεδρίου Παιδικής και Εφηβικής Γυναικολογίας, 17 – 18 Σεπτεμβρίου 2010, Αίγλη Ζαππείου, Αθήνα

12.7. **Co-chairman of the Lecture "Endometriosis**: From pathophysiology to treatment" by F. Petraglia in the "7th Athens Congress on Women's Health and Disease", September 11 – 13, 2008, Athens, Greece"

12.8. **Member of the Scientific Committee of the "7th Athens Congress on Women's Health and Disease**", September 11 – 13, 2008, Athens, Greece

12.9. **Μέλος Οργανωτικής Επιτροπής 1ου Πανελληνίου Συνεδρίου Κλιμακτηρίου και Εμμηνόπαυσης**, 18 – 19 Απριλίου 2008, Μέγαρο Μουσικής Αθηνών, Αθήνα

12.10. Ορμονολογικό Εργαστήριο Β' Πανεπιστημιακής Μαιευτικής και Γυναικολογικής Κλινικής, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Δεκέμβριος 2003 - 2008 – **Επιστημονική Υποστήριξη προϋπαρχόντων εξετάσεων**

- Ελεγχος θυρεοειδικής λειτουργίας, ορμονικός έλεγχος καταμμήνιου κύκλου, ορμονικός έλεγχος εμμηνόπαυσης, έλεγχος κυήσεως, παρακολούθηση επιπέδων ανοσοκατασταλτικών φαρμάκων – Συμβολή στον Αναλυτικό και Βιολογικό Ποιοτικό Ελεγχο, Συμβολή στην Αξιολόγηση Αποτελεσμάτων, Συμβολή στη Μηχανογραφική Υποστήριξη συστημάτων διαχείρισης αποτελεσμάτων – Αναδιαμόρφωση Τιμών Αναφοράς
- Ορμονολογικό Εργαστήριο Β' Πανεπιστημιακής Μαιευτικής και Γυναικολογικής Κλινικής, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Δεκέμβριος 2003 - 2008 - **Οργάνωση, Κοστολόγηση, Δρομολόγηση, Διαχειριστική Ένταξη στις Νοσοκομειακές Διαδικασίες, Ενταξη και Εφαρμογή στην Εργαστηριακή Πρακτική ΝΕΩΝ εξετάσεων**:

1. Διεγχειρητικός Προσδιορισμός Παραθορμόνης – Υπολογισμός Βιολογικού Χρόνου Υποδιπλασιαμού της Ορμόνης – Εργαστηριακή Αξιολόγηση Αποτελεσματικότητας Εξαιρέσεως Παραθυρεοειδικών Αδενωμάτων και έκτοπης Παραγωγής Παραθορμόνης

2. Εργαστηριακός Ελεγχος Οστικού Μεταβολισμού – Βιοχημικοί Δείκτες Οστικής Αποδόμησης και Αναδόμησης (C τελοπεπτίδιο του κολλαγόνου I, 25-υδροξυ-βιταμίνη D (D3), N-τελοπεπτίδιο του προκολλαγόνου I (P1NP), Παραθορμόνη (PTH), Αμινοτελικό και Διάμεσο τμήμα Μορίου Οστεοκαλσίνης (N-MID-Osteocalcin)-Βιοχημικός Έλεγχος Εμμηνοπαυσιακής και Φαρμακογενούς (γλυκοκορτικοειδή, αναστολείς αρωματάσης) Οστεοπορώσεως.

3. Εργαστηριακή Διαφορική Διάγνωση Υπερπρολακτιναιμίας και Μακροπρολακτιναιμίας – Απομόνωση Μακροπρολακτίνης από τα βιολογικά υγρά (καταβύθιση) – Εξορθολογισμός θεραπευτικής αντιμετωπίσεως

4. Προσδιορισμός Κλάσματος Ελεύθερης Κορτιζόλης στα ούρα – Απομόνωση από γλυκουρονίδια

12.11. **Επιστημονικός Συντονιστής Α' Κύκλου Μαθημάτων** (40 ώρες μαθημάτων, προαιρετική εργαστηρική εκπαίδευση, 9 διδάσκοντες) **Μεταπτυχιακού Προγράμματος «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολή του Πανεπιστημίου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών,** 29.11.2007 – 16.01.2008, 22.10.2008 – 20.11.2008

12.12. **Μέλος Επιστημονικής Επιτροπής 5ου Πανελληνίου Συνεδρίου Κλινικής Χημείας**, 18– 21 Νοεμβρίου, Αθήνα, Ελλάδα, 2004

13.ΣΥΜΜΕΤΟΧΗ ΣΕ ΕΠΙΣΤΗΜΟΝΙΚΕΣ ΣΥΝΑΝΤΗΣΕΙΣ

13.1. ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ

13.1.1. ΚΛΙΝΙΚΗ ΧΗΜΕΙΑ – ΦΑΡΜΑΚΕΥΤΙΚΗ ΒΙΟΧΗΜΕΙΑ

1. "*Effects of intravenous infusion of desferrioxamine on pro- and anti-inflammatory selected cytokines balance in warm hepatic ischemia-reperfusion injury of patients undergoing major liver resection performed under selective vascular exclusion*"
Economou EV, Kouskouni E, Smymiotis V, et al.
59th Annual Meeting of American Association of Clinical Chemistry, July 15 – 19, San Diego, CA, USA, 2007

2. **"***Attenuated and delayed elevation of serum ischemia modified albumin by intravenous infusion of desferrioxamine in hepatic warm ischemia/reperfusion injury"*
Economou EV, Efstratiou V, Smyrniotis V, et al.
59th Annual Meeting of American Association of Clinical Chemistry, July 15 – 19, San Diego, CA, USA, 2007

3. **"***Protection from inteleukin-1 beta converting enzyme (caspase-1)-related apoptotic cell death by intravenous infusion of desferrioxamine in hepatic warm ischemia/reperfusion injury"*
Economou EV, Loginidis I, Smyrniotis V, et al.
59th Annual Meeting of American Association of Clinical Chemistry, July 15 – 19, San Diego, CA, USA, 2007

4. "*Modest homocysteinemia does not contribute to chronic low-grade inflammation in childhood and adolescence obesity*"
E. V. Economou, A. Malamitsi-Puchner, E. Kouskouni, I. Magaziotou-Elefsinioti, D. Damianaki-Uranou, C. I. Stefanadis, G. Creatsas
56th Annual Meeting of the American Association for Clinical Chemistry, July 25-29, Los Angeles, USA, 2004

5. "*Lack of a homeostatic association between modest homocysteinemia and leukocyte transendothelial migration in childhood and adolescence obesity*"
E. V. Economou, A. Malamitsi-Puchner, E. Kouskouni, I. Magaziotou-Elefsinioti, D. Damianaki-Uranou, C. I. Stefanadis, G. Creatsas
56th Annual Meeting of the American Association for Clinical Chemistry, July 25-29, Los Angeles, USA, 2004

6. "*Adaptive angiogenesis fails to counteract elevated endothelial leukocyte adhesiveness after percutaneous transluminal coronary angioplasty*"
E. V. Economou, E. Kouskouni, M.G. Toutouza, G. Creatsas, C. Stefanadis
56th Annual Meeting of the American Association for Clinical Chemistry, July 25-29, Los Angeles, USA, 2004

7. "*Different patterns of alterations in circulating matrix metalloproteinases 2 and 9 after a severe acute myocardial infarction*"
E. V. Economou, E. Kouskouni, C.P.Pitsavos, G. Creatsas, C. Stefanadis
56th Annual Meeting of the American Association for Clinical Chemistry, July 25-29, Los Angeles, USA, 2004

8. "*Coincidental elevation of circulating amyloid beta 42 and the peripheral inflammatory response after a severe acute myocardial infarction*"
E. V. Economou, E. Kouskouni, C.P.Pitsavos, A. Masourou, G. Creatsas, C. Stefanadis
56th Annual Meeting of the American Association for Clinical Chemistry, July

25-29, Los Angeles, USA, 2004

9. *"The influence of calcium antagonists on plasma levels of endothelin 1-21 and arachidonic acid metabolites in chronically hypertensive patients"*
E. Economou, G. Vyssoulis, K. Giannakopoulou, M. Toutouza, P. Toutouzas
46th National Meeting of the American Association for Clinical Chemistry, July 17 - 21, New Orlean, USA, 1994

10. "*Plasma endothelin 1-21 levels in fetuses at 18-24 weeks of gestation*"
E. Economou, A. Malamitsi-Puchner, A. Antsaklis, D. Aravantinos
46th National Meeting of the American Association for Clinical Chemistry, July 17 - 21, New Orlean, USA, 1994

11. "*A comparison of five curve-fitting procedures in testosterone radioimmunoassay*"
E.V. Economou, Th. Siatra-Papastaikoudi, A. Foundos
53rd World Congress of Pharmacy and Pharmaceutical Sciences of the International Pharmaceutical Federation, September 5 - 10, Tokyo, Japan, 1993

12. "*A 125I-radioligand assay for serum diethylstilbestrol (DES)*"
G. P. Evangelatos, **E. V. Economou**, E. Livaniou, D. S. Ithakissios
43rd National Meeting of the American Association for Clinical Chemistry, July 28 - August 1, Washington DC, USA, 1991

13. "*Radioimmunoassay methodology for the estimation of diethylstilbestrol (DES) in serum of patients with advanced prostatic cancer*"
E. V. Economou, G. P. Evangelatos, T. Siatra-Papastaikoudi, D. S. Ithakissios
The 51st Pharmaceutical World Congress, of the International Pharmaceutical Federation, 1 to 6 of September, Washington DC, USA, 1991

14. "Direct radioimmunoassay for diethylstilbestrol (DES) in serum of patients with prostatic cancer"
E. V. Economou, S. E. Kakabakos, G. P. Evangelatos, D. S. Ithakissios
The 50th Pharmaceutical World Congress of the International Pharmaceutical Federation, 3 to 7 of September, Istanbul, Turkey, 1990

13.1.2. ΝΕΦΡΟΛΟΓΙΑ – ΒΙΟΛΟΓΙΑ ΝΕΦΡΙΚΩΝ ΑΡΤΗΡΙΩΝ

1. "*The angiotensin converting enzyme inhibitor ramipril increases total antioxidant capacity in haemodialysis patients*"
Dimitriadis GD, Chouliaras IC, **Economou EV**, Theodoridis TG, Galea VTH, Chouliaras GL, Metaxatos GL, Cokkinou VD, Hadjiconstantinou VE.
World Congress of Nephrology, 2005

2. "*The angiotensin converting enzyme inhibitor ramipril reduces serum CRP levels in hemodialysis patients*"
George Dimitriadis, Ioannis Chouliaras, **Emanuel Economou**, Paraskevi Dagounaki, Gerge Tsagalis, Theofanis Apostolou, Vasilios Margelos, George Metaxatos, Valsamakis Hadjiconstantinou
World Congress of Nephrology, June 8-12, 2003, Berlin, Germany

3. "*The anti-inflammatory effects of atorvastatin in hemodialysis patients*"
I. Chouliaras, G. Dimitriadis, **E. Economou**, P. Dagounaki, L. Sinodinos, G. Koutroubas, Chr. Christodoulidou, N. Nikolopoulou, V. Hadjiconstantinou
World Congress of Nephrology, June 8-12, 2003, Berlin, Germany

13.1.3. ΜΑΙΕΥΤΙΚΗ - ΓΥΝΑΙΚΟΛΟΓΙΑ

1. *"Improvement of endothelial function in 46,XY DSD patients after 6 months of*

hormone therapy"
Tsimaris Pandelis, Deligeoroglou Efthimios, Athanasopoulos Nikolaos, **Economou Emmanuel**, Stamatelopoulos Kimon, Rizos Demetrios, Papamichael Christos, Lambrinoudaki Irene, Mastorakos George,Creatsas George
The 9th Athens Congress on Women's Health and Disease, "From Puberty to Menopause" Athens Hilton, August 28-30, 2014

2. *"The effect of ERS1-XBAL polymorphism on bone density of adolescent girls with anorexia nervosa"*
Evgenia Stergioti, Efthimios Deligeoroglou, Konstantinos D. Dimopoulos, Vasileios Karountzos, Artemis Tsitsika, **Emmanouil Economou**, George Creatsas
The 9th Athens Congress on Women's Health and Disease, "From Puberty to Menopause" Athens Hilton, August 28-30, 2014

3. *"Genetic heterogeneity of Pecam-1 and P-Selectin genes are associated with in vitro fertilization – embryotransfer failure"*
Vlachadis N, Vrachnis N, Kouskouni E, Vitoratos N, **Economou E.**
The 9th Athens Congress on Women's Health and Disease, "From Puberty to Menopause" Athens Hilton, August 28-30, 2014

4. *"IVF-ET Failure is associated with Genetic Heterogeneity of Platelet Glycoproteins Ia and IIIa"*
Vlachadis N, Tsamadias V, Kouskouni E, Vitoratos N, Creatsas G, **Economou E**
10th Congress of European Society of Gynecology, Bruxelles, September 18-21, 2013

5. *"Genetic heterogeneity of platelets' glycoprotein receptors Ia and IIIa is associated with platelet function in women with recurrent miscarriages"*
Tsamadias V, Vlachadis N, Kouskouni E, Creatsas G, **Economou E**
10th Congress of European Society of Gynecology, Bruxelles, September 18-21, 2013

6. *"IVF – Failure is associated with genetic heterogeneity of platelet glycoproteins and cell adhesion molecules"*
Tsamadias Vasilios, Vlachadis Nikolaos, Papakonstantinou Emmanuel, Kouskouni Evangelia, **Economou Emanuel**
8th Congress of Women's Health & Disease, Kos, September 1 – 3, 2011, Greece

7. *"Increased risk for spontaneous miscarriages in women with genetic heterogeneity of platelet glycoproteins and cell adhesion molecules"*
Tsamadias Vasilios, Vlachadis Nikolaos, Papakonstantinou Emmanuel, Kouskouni Evangelia, **Economou Emanuel**
8th Congress of Women's Health & Disease, Kos, September 1 – 3, 2011, Greece

8. "*IL-β midtrimester amniotic fluid concentrations are positively associated with preterm delivery*"
K. Puchner, C. Iavazzo, D. Gourgiotis, M. Boutsikou, S. Baka, D. Hassiakos, E. Kouskouni, **E. Economou**, K. Tassis, G. Creatsas
The XXII European Congress of Perinatal Medicine, May 26-29, 2010, Granada, Spain

9. "*Second trimester amniotic fluid IL-10 and IL-18 concentrations cannot be used as predictors of preterm delivery*"
K. Puchner, C. Iavazzo, D. Gourgiotis, M. Boutsikou, S. Baka, D. Hassiakos, E. Kouskouni, **E. Economou**, K. Tassis, G. Creatsas
The XXII European Congress of Perinatal Medicine, May 26-29, 2010, Granada, Spain

10. "*Can apoptotic molecules during genetic amniocentesis be used to predict preterm delivery?*"
K. Puchner, C. Iavazzo, D. Gourgiotis, M. Boutsikou, S. Baka, D. Hassiakos, E.

Kouskouni, **E. Economou**, K. Tassis, G. Creatsas
The XXII European Congress of Perinatal Medicine, May 26-29, 2010, Granada, Spain

11. "*Measurable serum markers of oxidative stress response in women with endometriosis*"
A. Augoulea, Lambrinoudaki I., **E. Economou**, G. Kaparos, A. Kontoravdis, C. Papadias, G. Cristodoulakos, G. Creatsas
The 7th Athens Congress on Women's Health and Disease, 11 – 13 September, 2008, Athens, Greece

12. "*Does electromagnetic radiation from cell phones influence sperm motility in humans?*"
D. Tzanakaki, S. Baka, **E. Economou**, D. Hassiakos, S. Konidaris
The 7th Athens Congress on Women's Health and Disease, 11 – 13 September, 2008, Athens, Greece

13. "*Endogenous sex hormones and risk factors for atherosclerosis in healthy greek postmenopausal women*"
Vlachou S., Lambrinoudaki I., Christodoulakos G., **Economou E**., Argeitis J., Creatsas M., Kouskouni E., Rizos D.
The 7th Athens Congress on Women's Health and Disease, 11 – 13 September, 2008, Athens, Greece
XVI Meeting of Balkan Clinical Laboratory Federation and 7th Hellenic Congress of Clinical Chemistry, 16-18 October, 2008, Athens, Greece

14. "E-cadherin expression in cervical epithelial cells of postmenopausal women: association with hormone therapy, tibolone and raloxifene"
V. Sioulas, S. Vlachou, G. Chrostodoulakos, I. Lambrinoudaki, E. Politi, **E. Economou**, T. Sergentanis, C. Panoulis, A. Augoulea, A. Alexandrou, G. Creatsas.
12th World Congress on the Menopause, May 19 – 23, 2008, Madrid, Spain

15. *"Healthy postmenopousal women with higher index of androgeneicity exhibit a pro-atherogenic risk profile"*
Lambrinoudaki I., Christodoulakos G., Vlachou S., Rizos D., **Economou E**., Argeitis J., Creatsa M., Kouskouni E., Augoulea A., Botsis D.
41st Annual Meeting of the European Society for Clinical Investogation, 17 – 20 April, 2007, Uppsala, Sweeden

16. "*Effect of hormone therapy, tibolone and raloxifene on the circulating markers of chemotaxis, MCP-1 and RANTES in postmenopausal women*"
Christodoulakos G., Lambrinoudaki I., Karaflou M., Vlachou S., **Economou E**., Papadias C., Vitoratos N., Panoulis C., Kouskouni E., Augoulea A., Creatsas G**.**
41st Annual Meeting of the European Society for Clinical Investigation, 17 – 20 April, 2007, Uppsala, Sweeden

17. "*The effect of hormone therapy, tibolone and raloxifene on serum markers of chemotaxis*"
Christodoulakos G., Lambrinoudaki I., **Economou E.**, Papadias C., Vitoratos N., Panoulis C., Kouskouni E., Vlachou S., Creatsas G.
XVIII FIGO World Congress of Gynecology and Obstetrics, 5 – 10 November, KualaLumpur, Malaysia, 2006

18. "*Effect of hormone therapy and tibolone on plasma index of atherosclerosis"*
G. Christodoulakos, I. Lambrinoudaki, **E. Economou**, C. Panoulis, E. Kouskouni, A. Augoulea, G. Creatsas
The 6th Athens Congress on Women's Health and Disease, September 23-25, Athens, Greece, 2005

13.1.4. ΠΑΙΔΙΑΤΡΙΚΗ - ΝΕΟΓΝΟΛΟΓΙΑ

1. **"***Perinatal changes of neurotrophins in intrauterine growth restricted and appropriate for gestational age term offspring and their mothers***"**
K.E. Nikolaou, A. Malamitsi-Puchner, **E. Economou**, M. Boutsikou, T. Boutsikou, K.P. Puchner, M. Kyriakakou, D. Hassiakos
International Congress of the European Academy of Paediatrics, October 7-10, Barcelona, Spain, 2006

2. *"Circulating neurotrophin levels in the perinatal period of intrauterine growth restricted fetuses and neonates at term"*
Malamitsi-Puchner A., Nikolaou KE, **Economou E**, Boutsikou M, Boutsikou T, Kyriakakou M, Puchner KP, Hassiakos D.
32nd Annual Meeting of the Swiss, Austrian and German Society of Neonatalogy and Pediatric Intensive Medicine, May 18-20, Vienna, Austria, 2006

3. *"The impact of intrauterine growth restriction on circulating levels of angiogenic factors and their possible role as catch-up growth predictors"*
A. Malamitsi-Puchner, T. Boutsikou, **E. Economou**, E. Makrakis, K.E. Nikolaou, D. Hasiakos
13th European Workshop on Neonatology, October 12 – 15, Freiburg, Germany, 2005

4. "*Expression of angiopoietin-2 and endostatin in intrauterine growth restriction during the perinatal period*"
Ar. Malamitsi-Puchner, T. Boutsikou, **E. Economou**, Z. Iliodromiti, E. Kouskouni, D. Hasiakos
International Congress of the European Society for Pediatric Research, September 19-22, Stockholm, Sweeden, 2004

5. "*Perinatal changes of BDNF in pre- and fullterm neonates*"
A.Malamitsi-Puchner, **E. Economou**, O. Rigopoulou, T. Boutsikou
International Congress of the European Society for Pediatric Research, October, 2003

6. "*Circulating monocyte and T-lymphocyte specific chemokines are elevated in overweight adolescents*"
I Magaziotou, D. Damianaki, I. Elefsiniotis, **E. Economou**, A. Mariolis, C. Stefanadis, P. Toutouzas
39th Annual Meeting of European Society for Pediatric Research, 17 -19 September, Brussels, Belgium, 2000

7. "*Plasma levels of total homocysteine are elevated in overweight adolescents*"
I Magaziotou, D. Damianaki, **E. Economou**, I. Elefsiniotis, A. Mariolis, C. Stefanadis, P. Toutouzas
39th Annual Meeting of European Society for Pediatric Research, 17 - 19 September, Brussels, Belgium, 2000

8. *"Endothelin (ET) 1-21 plasma concentration in children and adolescents with IDDM as marker of endothelial dysfunction"*
A. Malamitsi-Puchner, **E. Economou**, F. Karachaliou, D. Delis, K. Kassiou, C. S. Bartsocas
20th Annual Meeting of the International Society for Pediatric and Adolescent Diabetes, November 2 - 6, Atami, Japan, 1994

9. "*Endothelin (ET) 1-21 plasma levels in the first and fourth day of life in healthy and ill premature infants*"

Ariadne Malamitsi-Puchner, Theodore Efstathopoulos, Zoe Hadjistamatiou, **Emmanuel Economou**, Sophia Sevastiadou, Demetrios Nicolopoulos
Pediatric Week, June 29 - July 6, Rotterdam, Holland,1994

10. "*Lipid peroxide plasma levels in normal fullterm and premature neonates*"
A. Malamitsi-Puchner, S. Sevastiadou, T. Efstathopoulos, **E. Economou**, Z. Hadzistamatiou, D. Nicolopoulos
14th European Congress of Perinatal Medicine, June 5 - 8, Helsinki, Finland,1994

11. "*Endothelin 1-21 plasma levels in fetuses of 18 - 24 weeks*"
A. Malamitsi-Puchner, A. Antsaklis, **E. Economou**, N. Papantoniou, S. Mesogitis, N. Koutra, D. Aravantinos
14th European Congress of Perinatal Medicine, June 5 - 8, Helsinki, Finland,1994

12. "*Postnatal changes of lipid peroxide levels : possible indirect index of gradually increased oxygenation?*"
A. Malamitsi-Puchner, **E. Economou**, T. Efstathopoulos, S. Sevastiadou, D. Nicolopoulos
4th European Workshop on Neonatology, October 21 - 23, Corfu Island, Greece, 1993

13. "*Endothelin-1,2 levels in the first and fourth day postpartum in normal fullterm and premature neonates*"
A. Malamitsi-Puchner, **E. V. Economou**, S. Sevastiadou, T. Efstathopoulos, D. Nicolopoulos
2nd World Congress of Perinatal Medicine, September 19 - 24, Roma, Italy, 1993

14. "*Evidence that vitamin E may reduce the levels of prostacyclin and thromboxane in the blood of premature neonates*"
Ariadne Malamitsi-Puchner, **Emmanuel Economou**, Eugenia Papathoma, Costas Papas
An International Symposium on Progress and Controversies in Neonatal / Perinatal Medicine, September 5 - 6, Volos, Greece, 1992

15. "*Influence of vitamin E on the levels of prostacycline and thromboxane in the blood of premature neonates*"
Ariadne Malamitsi-Puchner, **Emmanuel Economou**, Eugenia Papathoma, Costas Papas
VI International Berlin Symposium "Research in Perinatal Medicine", June 5 to 8, Berlin, Germany, 1991

13.1.5. ΚΑΡΔΙΟΛΟΓΙΑ - ΑΓΓΕΙΟΛΟΓΙΑ

1. *"The association between pre-hypertension status and oxidative stress markers related to atherosclerotic disease; the ATTICA study"*
Chrysohoou C, Pitsavos C, Panagiotakos DB, et al.
XXVIIIth Congress of the European Society of Cardiology, 2 – 6 September, Barcelona – Spain, 2007

2. *"Increased inflammatory and coagulant activity in patients with spontaneous echocardiographic contrast in the thoracic aorta*"
Aggeli C, Giannopoulos G, **Economou E**, et al.
XXVIIIth Congress of the European Society of Cardiology, 2 – 6 September, Barcelona – Spain, 2007

3. **"***Long-term fish consumption offers cardiovascular protection in healthy people due to its anti-arrhythmic and anti-inflammatory properties; The Attica study."*
Chrysohoou C, Pitsavos C, Skoumas J, Krinos X, Papademetriou L, Kambaxis M, Nikolaou V, Panagiotakos D, **Economou M**, Stefanadis C
56th Annual Scientific Meeting of the American College of Cardiology**,** March 24-27, New Orleans, Louisiana, USA, 2007

4. *"Association of spontaneous echocardiographic contrast in the thoracic aorta with increased systemic inflammatory and coagulant activity"*
Aggeli C, Giannopoulos G, **Economou E**, et al.
84th Scientific Session of the American Heart Association, November 12-15, Chicago, IL, 2006

5. "*Similar kinetic profile in serum levels of heparin-binding growth factors bFGF and HGF in acute myocardial infarction patients: the double peak hypothesis*"
K.I. Kapetanios, C.E. Pitsavos, S. Kastelanos, G.P. Vyssoulis, **E.V. Economou**, M.G. Toutouza, C.I. Stefanadis
XXVIth Congress of the European Society of Cardiology, 28 August – 1 September, Munich – Germany, 2004

6. "*Concerted action of various angiogenic factors during acute myocardial infarction in patients : the three lines of defense*"
Konstantinos I. Kapetanios, Christos E. Pitsavos, Stamatis Kastelanos, Grigorios P. Vyssoulis, Demosthenes B. Panagiotakos, **Emanuel V. Economou**, Marina G. Toutouza, Christodoulos I. Stefanadis, Pavlos K. Toutouzas
52nd Annual Scientific Session of American College of Cardiology, March 30 - April 2, Chicago, USA, 2003

7. "*The clinical significance of circulating levels of granulocyte/macrophage-colony stimulating factor in acute myocardial infarction patients*"
Konstantinos I. Kapetanios, Christos E. Pitsavos, Stamatis Kastelanos, Grigorios P. Vyssoulis, Demosthenes B. Panagiotakos, **Emanuel V. Economou**, Marina G. Toutouza, Christodoulos I. Stefanadis, Pavlos K. Toutouzas
52nd Annual Scientific Session of American College of Cardiology, March 30 - April 2, Chicago, USA, 2003

8. "*Matrix metalloproteinases activity in acute myocardial infarction patients. Do their activity contribute to post-myocardial infarction dilation?*"
D. Papadopoulos, **E.V. Economou**, K.I. Kapetanios, P.K. Toutouzas
XXIVth Congress of the European Society of Cardiology, 31 August - 4 September, Berlin, Germany, 2002

9. "*Circulating vasoactive amyloid beta ptotein ending at 42 amino acid is elevated in severe acute myocardial infarction*"
E.V. Economou, C.P. Pitsavos, A. Masourou, A. Zervoudaki, M.G. Toutouza, K.I. Kapetanios, C.I. Stefanadis, P.K. Toutouzas
XXIVth Congress of the European Society of Cardiology, 31 August - 4 September, Berlin, Germany, 2002

10. "*Clinical significance of matrix metalloproteinases activity in acute myocardial infarction patients*"
D. Papadopoulos, K.I. Kapetanios, **E.V. Economou**, P.K. Toutouzas
XXIVth Congress of the European Society of Cardiology, 31 August - 4 September, Berlin, Germany, 2002

11. "*Circulating matrix metalloproteinase 2 and tissue inhibitor of metalloproteinases 1*

*after severe and minor acute myocardial in*farction"
E.V. Economou, C.P. Pitsavos, A. Masourou, A. Zervoudaki, M.G. Toutouza, K.I. Kapetanios, C.I. Stefanadis, P.K. Toutouzas
XXIVth Congress of the European Society of Cardiology, 31 August - 4 September, Berlin, Germany, 2002

12. "*Coincidental fluctuation of circulating amyloid beta 42 and tissue inhibitor of metalloproteinases 1 after a severe acute myocardial in*farction"
E.V. Economou, C.E. Pitsavos, A. Masourou, A. Zervoudaki, K.I. Kapetanios, M.G. Toutouza, C.I. Stefanadis, P.K. Toutouzas
XXIVth Congress of the European Society of Cardiology, 31 August - 4 September, Berlin, Germany, 2002

13. "*Effects of antihypertensive monotherapy on plasma levels of pituitary adenylate cyclase activating polypeptide-38 in hypertensive subjects*"
J. Elefsiniotis, C. Tsioufis, **E. Economou**, M. Toutouza, C. Stefanadis, P. Toutouzas
XXIVth Congress of the European Society of Cardiology, 31 August - 4 September, Berlin,Germany, 2002

14. "*A beta chemokine (MCP-1)-induced antigen-independent cellular and not a humoral immune response can be stimulated after stent percutaneous transluminal coronary angioplasty*"
Emanuel V. Economou, Christos Pitsavos, Anastasia Katinioti, Konstantinos Tedolouris, Athanasios Trikas, Marina Toutouza, Christodoulos Stefanadis, Pavlos Toutouzas
51st Annual Scientific Session of American College of Cardiology, March 17 - 20, Atlanda, Georgia, USA, 2002

15. "*Serum levels of interleukin-1beta converting enzyme/caspase-1 can be used to detect and quantify apoptotic process in patients with coronary artery disease undergoing percutaneous transluminal coronary angioplasty*"
E. Economou, C. Pitsavos, A. Katinioti, A. Trikas, K. Kapetanios, C. Stefanadis, M. Toutouza, P. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000
51st Annual Scientific Session of American College of Cardiology, March 17 - 20, Atlanda, Georgia, USA, 2002

16. *"Depressed plasma levels of active extracellular matrix metalloproteinases 2 and 9 in patients with essential mild to moderate hypertension"*
E. Economou, C. Pitsavos, C. Stefanadis, A. Zervoudaki, K. Tsioufis, M. Toutouza, P. Toutouzas
50th Annual Scientific Session of American College of Cardiology, March 18 – 21 Orlando, Florida, USA, 2001
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000
XIIth International Symposium on Atherosclerosis, June 25 - 29, Stockholm, Sweeden, 2000

17. "*Cholesterol blood levels and collagenolysis in patients with acute myocardial infarction*"
D.P. Papadopoulos, **E.V. Economou**, K.I. Kapetanios, P.K. Toutouza
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5 Stockholm, Sweeden, 2001

18. "*Silent ischaemic episodes in patients with chronic stable angina are associated with increased thrombin generation during daily life activities*"
I. Ikonomidis, F. Andreotti, C. Pitsavos, C. Stefanadis, **E. Economou**, M. Marinou, P. Nihoyannopoulos, P. Toutouzas
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5 Stockholm, Sweeden, 2001

19. "*Dissociation between the clustering of newer risk factors and estrogen-induced improvement in aortic elasticity in hypertensive postmenopausal women*"
K. Tsioufis, K. Tzioumis, C. Stefanadis, M. Toutouza, I. Kallikazaros, I. Elefsiniotis, **E. Ekonomou**, C. Pitsavos, E. Tsiamis, P. Toutouzas
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5 Stockholm, Sweeden, 2001

20. "*Effect of angiotensin converting enzyme inhibitor on collagenolytic enzymes activity in patients with acute myocardial infarction*"
D.P. Papadopoulos, **E.V. Economou**, K.I. Kapetanios, P.K. Toutouzas
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5 Stockholm, Sweeden, 2001

21. "*Increased levels of macrophage colony stimulating factor and C-reactive protein predict worse prognosis in patients with chronic stable angina. A six-year follow-up study*"
I. Ikonomidis, D. Tsiapras, F. Andreotti, C. Pitsavos, C. Stefanadis, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
74th Scientific Session of the American Heart Association, November 11 - 14, Anaheim, California, USA, 2001
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5 Stockholm, Sweeden, 2001

22. "*Heart fatty acid binding protein : the advent of unique marker for the early detection of acute myocardial infarction patients*"
K.I. Kapetanios, C.E. Pitsavos, K. Vasiliadou, M.G. Toutouza, **E.V. Economou**, C.I. Stefanadis, P.K. Toutouzas
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5 Stockholm, Sweeden, 2001

23. "*Time coincidence in alterations of circulating amyloid A and matrix metalloproteinases 2 and 9 may increase amyloid A amyloidogenic potential after a severe acute myocardial infarction*"
E.V. Economou, C.E. Pitsavos, A. Masourou, K.I. Kapetanios, A. Zervoudaki, M.G. Toutouza, C.I. Stefanadis, P.K. Toutouzas
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5 Stockholm, Sweeden, 2001

24. "*Stent-percutaneous transluminal coronary angioplasty triggers an antigen-independent cellular immune response*"
E.V. Economou, C.E. Pitsavos, A. Katinioti, K. Tedolouris, M.G. Toutouza, C.I. Stefanadis, P.K. Toutouzas
XXIIIrd Congress of the European Society of Cardiology, September 1 - 5, Stockholm, Sweeden, 2001

25. "*Dissociation between plasma levels of homocysteine, insulin and leptin with estrogen-induced improvement in aortic elasticity in hypertensive postmenopausal women*"
K. Tzioumis, C. Tsioufis, M. Toutouza, **E. Ekonomou**, A. Michaelidis, I.

Kallikazaros, E. Tsiamis, C. Stefanadis, P. Pitsavos
11th European Meeting on Hypertension, Milan, Italy, June 15 - 16, 2001

26. "*The effect of antihypertensive monotherapy on plasma levels of pituitary adenylate cyclase activating polypeptide-38 in patients with mild to moderate essential hypertension*"
J. Elefsiniotis, **E. Economou**, C. Tsioufis, D. Panagiotakos, M. Toutouza, G. Vyssoulis, P. Toutouzas
11th European Meeting on Hypertension, Milan, Italy, June 15 - 16, 2001

27. "*Hepatocyte growth factor : potential role of a novel vascular modulator in acute myocardial infarction patients*"
Konstantinos I.Kapetanios, Christos E. Pitsavos, **Emmanuel V. Economou**, Marina G. Toutouza, Christodoulos I. Stefanadis, Pavlos K. Toutouzas
50th Annual Scientific Session of American College of Cardiology, March 18 – 21, Orlando, Florida, USA, 2001

28. "*Matrix metalloproteinase-2 levels and features of atherosclerotic plaque vulnerability as detected by intracoronary ultrasound*"
M. Vavouranakis, K. Toutouzas, V. Vulcevic, C. Chrysochou, D. Markou, **E. Economou**, C. Stefanadis, K. Tsioumis, N. Marinakis, P. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000
50th Annual Scientific Session of American College of Cardiology, March 18 – 21 Orlando, Florida, USA, 2001

29. *"Silent ischaemia during daily life is related to increased thrombin generation in patients with chronic stable angina"*
Ignatios Ikonomidis, Felicitta Andreotti, Christodoulos Stefanadis, Christos Pitsavos, **Emanuel V. Economou**, Pavlos Toutouzas, Petros Nihoyannopoulos
50th Annual Scientific Session of American College of Cardiology, March 18 – 21, Orlando, Florida, USA, 2001
74th Scientific Session of the American Heart Association, November 11 - 14, Anaheim, California, USA, 1996

30. *"The effect of a dihydropyridine and a non dihydropyridine calcium antagonist on plasma levels of active extracellular matrix metalloproteinases 2 and 9 in patients with essential mild to moderate hypertension"*
E. Economou, C. Pitsavos, C. Stefanadis, A. Zervoudaki, K. Tsioufis, M. Toutouza, P. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000
XIIth International Symposium on Atherosclerosis, June 25 - 29, Stockholm, Sweeden, 2000

31. "*Six months follow-up of the circulating basic fibroblast growth factor in patients with coronary artery disease undergoing percutaneous transluminal coronary angioplasty*"
A. Katinioti, **E. Economou,** C. Pitsavos, C. Stefanadis, A. Trikas, M. Toutouza, K. Kapetanios, P. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000
49th Annual Scientific Session of American College of Cardiology, March 12 – 15 Anaheim, California, USA, 2000

32. "*Plasma levels of plasminogen activator inhibitor 1 are elevated while plasma levels of von Willebrand factor and soluble E-selectin remain unaffected in patients with coronary* artery disease undergoing percutaneous transluminal coronary angioplasty"
E. Economou, A. Katinioti, C. Pitsavos, K. Vasiliadou, K. Kapetanios, C. Stefanadis, M. Toutouza, P. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000

33. *"Serum levels of beta chemokine RANTES and acute phase protein amyloid A can be both used to recognize and measure the severity of inflammatory response in coronary artery disease"*
E. Economou, C. Pitsavos, A. Katinioti, A. Trikas, K. Kapetanios, C. Stefanadis, M. Toutouza, P. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000

34. "*Soluble P-selectin : a novel and earlier prognostic indicator compared to the convetional cardiac markers in acute myocardial infarction patients*"
K.I. Kapetanios, C.E. Pitsavos, K. Vasiliadou, M.G. Toutouza, **E.V. Economou**, C.I. Stefanadis, P.K. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000

35. *"Serial changes in serum levels of soluble P-selectin in acute myocardial infarction patients : effects of non-invasive coronary recanalisation therapy"*
K.I. Kapetanios, C.E. Pitsavos, M.G. Toutouza, K. Vasiliadou, **E.V. Economou**, C.I. Stefanadis, P.K. Toutouzas
XXIInd Congress of the European Society of Cardiology, August 26 - 30, Amsterdam, The Netherlands, 2000

36. "*Plasma levels of total homocysteine are elevated in overweight children and adolescents*"
E. Economou, M. Toutouza, C. Pitsavos, C. Stefanadis, J. Magaziotou-Elefsinioti, J. Elefsiniotis, D. Uranou, P. Toutouzas
XIIth International Symposium on Atherosclerosis, June 25 - 29, Stockholm, Sweeden, 2000

37. "*Percutaneous transluminal coronary angioplasty results in stimulation of monocyte- but not eosinophil-specific chemotaxis*"
Emanuel V. Economou, Anastasia A. Katinioti, Christodoulos I. Stefanadis, Christos P. Pitsavos, Athanasios G. Trikas, Marina G. Toutouza, Pavlos K. Toutouzas
49th Annual Scientific Session of American College of Cardiology, March 12 – 15, Anaheim, California, USA, 2000

38. "*Heat production of atherosclerotic plaques and inflammation assessed by the acute phase proteins in acute coronary syndromes*"
Christodoulos Stefanadis, Leonidas Diamandopoulos, John Dernellis, **Emanuel Economou**, Eleftherios Tsiamis, Konstantinos Toutouzas, Charalambos Vlachopoulos, Pavlos Toutouzas
49th Annual Scientific Session of American College of Cardiology, March 12 – 15, Anaheim, California, USA, 2000

39. "*The clinical implication of circulating levels of angiogenin in acute myocardial*

infarction patients"
K.I Kapetanios, **E.V. Economou**, C.I. Stefanadis, C.E. Pitsavos, D.M. Farmakis, K. Vasiliadou, M.G. Toutouza, P.K. Toutouzas
XXIst Congress of the European Society of Cardiology, August 28 - September 1, Barcelona, Spain,1999

40. "*Effects of cigarette smoking on proinflammatory cytokines and platelet activation in patients with stable angina*"
I. Ikonomidis, F. Andreotti, C. Stefanadis, C. Pitsavos, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
XXIst Congress of the European Society of Cardiology, August 28 - September 1, Barcelona, Spain,1999

41. "*Aspirin reduces procoagulant activity and thrombin generation in patients with stable angina*"
I. Ikonomidis, F. Andreotti, S. Loizou, C. Stefanadis, C. Pitsavos, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
XXIst Congress of the European Society of Cardiology, August 28 - September 1, Barcelona, Spain,1999

42. "*Beneficial effect of alcohol on the elastic properties of the aorta*"
C. Vlachopoulos, C. Stefanadis, N. Giatrakos, **E. Economou**, E. Tsiamis, C. Spiliopoulou, A. Koutselinis, P. Toutouzas
XXIst Congress of the European Society of Cardiology, August 28 - September 1, Barcelona, Spain,1999

43. "*Biochemical changes involved in the mechanism of neurocardiogenic syncope. Observations during tilt table testing*"
K. Gatzoulis, A. Theopistou, **E. Economou**, S. Sideris, K. Avgeropoulou, J. Gialafos, P. Toutouzas
XXIst Congress of the European Society of Cardiology, August 28 - September 1, Barcelona, Spain,1999

44. "*Basic fibroblast growth factor, a potent promoter of angiogenesis, is a useful predictor of the progression of ventricular remodelling in acute myocardial infarction patients*"
K.I. Kapetanios, **E.V. Economou**, C.I. Stefanadis, C.E. Pitsavos, D.M. Farmakis, K. Vasiliadou, M.G. Toutouza, P.K. Toutouzas
XXIst Congress of the European Society of Cardiology, August 28 - September 1, Barcelona, Spain,1999

45. "*The significance of free insulin-like growth factor-1 activation in the process of remodelling in patients with acute myocardial infarction*"
K.I. Kapetanios, **E.V. Economou**, C.I. Stefanadis, C.E. Pitsavos, D.M. Farmakis, K. Vasiliadou, M.G. Toutouza, P.K. Toutouzas
XXIst Congress of the European Society of Cardiology, August 28 – September 1, Barcelona, Spain,1999

46. "*Aspirin reduces transient ischaemia in stable angina by reducing increased cytokine plasma levels and platelet activation*"
I. Ikonomidis, F. Andreotti, C. Stefanadis, C. Pitsavos, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
XXIst Congress of the European Society of Cardiology, August 28 – September 1, Barcelona, Spain,1999

47. "*Aspirin reduces C-reactive protein plasma levels in patients with stable angina by reducing cytokine plasma levels*"
Ignatios Ikonomidis, Felicita Andreotti, Christodoulos Stefanadis, Christos Pitsavos, **Emanouel Economou**, Pavlos Toutouzas, Petros Nihoyannopoulos
48th Annual Scientific Session of American College of Cardiology, March 7 - 10New Orleans, Louisiana, USA, 1999

48. "*Aspirin reduces daily life ischaemia in stable angina by reducing increased cytokine levels and platelet activation*"
Ignatios Ikonomidis, Felicita Andreotti, Christodoulos Stefanadis, Christos Pitsavos, **Emanouel Economou**, Pavlos Toutouzas, Petros Nihoyannopoulos
48th Annual Scientific Session of American College of Cardiology, March 7 - 10New Orleans, Louisiana, USA, 1999

49. "*Extracellular superoxide dismoutase level is implicated in excercise-induced myocardial preconditioning*"
Andreas P. Michaelidis, **Emmanouil V. Oikonomou**, Christos K. Seferlis, Zoi O. Psomadaki, Polychronis E. Dilaveris, Dimitris J. Richter, George K. Andrikopoulos, Christodoulos I. Stefanadis, Pavlos K. Toutouzas
48th Annual Scientific Session of American College of Cardiology, March 7 - 10New Orleans, Louisiana, USA, 1999

50. *"Serum amyloid A levels in patients with mild to moderate hypertension, before and after treatment with a calcium antagonist"*
E. Economou, C. Pitsavos, C. Stefanadis, A. Trikas, M. Toutouza, J. Elefsiniotis, K. Tsioufis, K. Kapetanios, P. Toutouzas
48th Annual Scientific Session of American College of Cardiology, March 7 - 10, New Orleans, Louisiana, USA, 1999
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

51. *"Serum levels of antiphosphatidylserine antibodies in patients with mild to moderate hypertension, before and after treatment with a calcium antagonist"*
E. Economou, C. Pitsavos, C. Stefanadis, M. Toutouza, A. Trikas, K. Tsioufis, K. Kapetanios, P. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

52. "*Plasma levels of thrombus precursor protein in patients with mild to moderate hypertension treated by a calcium antagonist*"
E. Economou, C. Pitsavos, C. Stefanadis, M. Toutouza, K. Tsioufis, A. Trikas, K. Kapetanios, P. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

53. "*Platelet activation induced by increased cytokine plasma levels, is related to daily life ischemia in chronic stable angina*"
I. Ikonomidis, F Andreotti, C. Stefanadis, C. Pitsavos, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

54. "*Soluble Fas is associated to free insulin-like growth factor but not to basic fibroblast growth factor in plasma of patients at the terminal stage of chronic heart failure*"
E. Economou, D. Farmakis, C. Stefanadis, C. Pitsavos, M. Toutouza, K.

Kapetanios, P. Papadopoulos, P. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

55. "*Important angiogenic factors as markers of severity of chronic heart failure*"
E. Economou, C. Stefanadis, C. Pitsavos, M. Toutouza, D. Farmakis, K. Kapetanios, P. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

56. "*Angiogenin, a potent inducer of angiogenesis, found to be increased in acute myocardial infarction patients*"
K.I. Kapetanios, C.I. Stefanadis, C.E. Pitsavos, P.D. Papadopoulos, **E.V. Economou**, D.M. Farmakis, M.G. Toutouza, D.P. Papadopoulos, P.K. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

57. "Elevated circulating levels of the soluble form of Fas/APO-1, an important cofactor to the activation of apoptosis, in chronic heart failure"
E. Economou, D. Farmakis, C. Stefanadis, C. Pitsavos, M. Toutouza, K. Kapetanios, P. Papadopoulos, P. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

58. *"Low-dose aspirin reduces elevated C-reactive protein plasma levels in patients with chronic stable angina by reducing cytokine plasma levels : a randomized, placebo controlled, cross-over trial"*
I. Ikonomidis, F. Andreotti, C. Stefanadis, C. Pitsavos, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

59. "*Plasma levels of free insulin-like growth factor-1 were elevated, while plasma levels of vascular endothelial growth factor remained unaffected in acute myocardial infarction patients*"
P.D. Papadopoulos, C.I. Stefanadis, C.E. Pitsavos, K.I. Kapetanios, **E.V. Economou**, D.M. Farmakis, D.P. Papadopoulos, M.G. Toutouza, P.K. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

60. "*Plasma levels of basic fibroblast growth factor developed a double-peak increase in acute myocardial infarction patients*"
K.I. Kapetanios, C.I. Stefanadis, C.E. Pitsavos, P.D. Papadopoulos, **E.V. Economou**, D.M. Farmakis, M.G. Toutouza, D.P. Papadopoulos, P.K. Toutouzas
XXth Congress of the European Society of Cardiology, August 22 - 26, Vienna, Austria, 1998

61. "*Correlation between markers of fibrillar collagen degradation and myocardial damage in patients with acute myocardial infarction*"
D.P. Papadopoulos, P.D. Papadopoulos, **E.V. Economou**, K.I. Kapetanios, D.M. Farmakis, C.I. Stefanadis, P.K. Toutouzas
XIIIth World Congress of Cardiology, April 26 - 30, Rio de Janeiro, Brazil, 1998

62. "*A double-peak increase of basic fibroblast growth factor was found in acute myocardial infarction patients*"
K.I. Kapetanios, P.D. Papadopoulos, E.V. Economou, M.G. Toutouza, D.M. Farmakis, D.P. Papadopoulos, C.I. Stefanadis, P.K. Toutouzas

XIIIth World Congress of Cardiology, April 26 - 30, Rio de Janeiro, Brazil, 1998

63. *"Increased cytokine levels and thrombin generation in stable angina are reduced by aspirin. A randomized, placebo-controlled, cross-over trial"*
Ignatios Ikonomidis, Felicita Andreotti, Christodoulos Stefanadis, Christos Pitsavos, **Emanouil Economou**, Pavlos Toutouzas
47th Annual Scientific Session of American College of Cardiology, March 29 - April 1, Atlanta, Georgia, USA, 1998

64. "*Daily life ischemia in chronic stable angina is related to platelet activation induced by increased cytokine plasma levels*"
Ignatios Ikonomidis, Felicita Andreotti, Christodoulos Stefanadis, Christos Pitsavos, **Emanouil Economou**, Pavlos Toutouzas
47th Annual Scientific Session of American College of Cardiology, March 29 - April 1, Atlanta, Georgia, USA, 1998

65. "Plasma levels of soluble E-selectin in patients with mild to moderate hypertension treated by a calcium antagonist or a I1-imidazoline agonist"
Emanuel V. Economou, Christos P. Pitsavos, Christodoulos I. Stefanadis, Marina G. Toutouza, Athanasios G. Trikas, Gregory P. Vyssoulis, Konstantinos K. Kapetanios, Ekaterini E. Giannakopoulou, Areti A. Livanou, Pavlos K. Toutouzas
47th Annual Scientific Session of American College of Cardiology, March 29 - April 1, Atlanta, Georgia, USA, 1998

66. "*Low dose asprin reduces elevated C-reactive protein plasma levels in patients with chronic stable angina by reducing cytokine plasma levels*"
Ignatios Ikonomidis, Crist Stefanades, **Emanuel Economou**, Cris Pitsavos, Felicita Andreotti, Pablo Tutuzas, Petros Nihoyannopoulos
71st Scientific Session of the American Heart Association, November 8 - 11, Dallas, Texas, USA, 1998

67. *"Regulation of collagen degradation in patients with acute myocardial infarction. Preliminary data"*
D.P. Papadopoulos, P.D. Papadopoulos, **E.V. Economou**, D.M. Farmakis, M.G. Toutouza, K.I. Kapetanios, P.K. Toutouzas
1st International Congress on Coronary Artery Disease-From Prevention to Intervention, September 21 - 24, Prague, Czech Republic, 1997

68. *"Plasma levels of soluble E-selectin are elevated while plasma levels of von Willebrand factor and plasminogen activator inhibitor-1 remain unaffected in patients with mild hypertension"*
E. Economou, K. Vasiliadou, A. Trikas, M. Toutouza, A. Giannakopoulou, G. Vyssoulis, A. Livanou, C. Pitsavos, C. Stefanadis, P. Toutouzas
XIXth Congress of the European Society of Cardiology, August 24 - 28, Stockholm, Sweeden, 1997

69. *"Pathophysiological correlates of increased plasma insulin-like growth factor-1 levels in patients with chronic heart failure"*
E. Economou, M. Toutouza, D. Farmakis, P. Papadopoulos, C. Pitsavos, C. Stefanadis, P. Toutouzas
XIXth Congress of the European Society of Cardiology, August 24 - 28, Stockholm, Sweeden, 1997

70. *"Thrombin generation and cytokine levels are increased in patients with chronic stable angina but only cytokine levels are associated to transient myocardial ischaemia"*
I. Ikonomidis, F. Andreotti, C. Stefanadis, C. Pitsavos, **E. Economou**, P. Toutouzas,

P. Nihoyannopoulos
XIXth Congress of the European Society of Cardiology, August 24 - 28, Stockholm, Sweeden, 1997

71. *"Aspirin reduces thrombin generation and cytokine levels in patients with chronic stable angina. A new therapeutic action of aspirin"*
I. Ikonomidis, F. Andreotti, C. Stefanadis, C. Pitsavos, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
XIXth Congress of the European Society of Cardiology, August 24 - 28, Stockholm, Sweeden, 1997

72. "*Angiogenic factors basic fibroblast growth factor and angiogenin in advanced heart failure patients*"
D.M. Farmakis, P.D. Papadopoulos, **E.V. Economou**, M.G. Toutouza, K.I. Kapetanios, T.A. Argyriou, P.K. Toutouzas
46th Annual Scientific Session of American College of Cardiology, March 16 -19, Anaheim, California, USA, 1997
XVIIIth Congress of the European Society of Cardiology, August 25 - 29, Birmingham, United Kingdom, 1996

73. *"Severity of coronary artery disease is associated to macrophage colony stimulating factor, interleukin-1b and -6 plasma levels"*
I. Ikonomidis, **E. Economou**, C. Pitsavos, C. Stefanadis, P. Toutouzas
69th Scientific Session of the American Heart Association, November 10 - 13, New Orleans, Lousiana, USA, 1996
XVIIIth Congress of the European Society of Cardiology, August 25 - 29, Birmingham, United Kingdom, 1996

74. "*Aspirin reduces macrophage colony stimulating factor plasma levels in patients with coronary artery disease. A new therapeutic role of aspirin*"
I. Ikonomidis, O. Cole, **E. Economou**, S. Loizou, C. Pitsavos, P. Toutouzas, P. Nihoyannopoulos
69th Scientific Session of the American Heart Association, November 10 - 13, New Orleans, Lousiana, USA, 1996
XVIIIth Congress of the European Society of Cardiology, August 25 - 29, Birmingham, United Kingdom, 1996

75. "*Increased cytokine plasma levels in stable angina are related to transient myocardial ischaemia by enhancing platelet activation*"
Ignatios Ikonomidis, Felicita Andreotti, Chris I. Stefanadis, Christos Pitsavos, Olotoun Cole, Margarita Marinou, **Emanouil Economou**, Paul K. Toutouzas, Petros Nihoyannopoulos
70th Scientific Session of the American Heart Association, November 9 - 12, Orlando, Florida, USA, 1996

76. "*The role of tumor necrosis factor-a and -b on heart failure patients*"
P.D. Papadopoulos, D.M. Farmakis, **E.V. Economou**, M.G. Toutouza, K.I. Kapetanios, D.P. Papadopoulos, P.K. Toutouzas
9th Annual Meeting of Mediterranean Association of Cardiology and Cardiac Surgery, October 20 - 23, Tel Aviv, Israel, 1996

77. "*Insulin-like growth factor-1 in congestive heart failure patients*"
P.D. Papadopoulos, D.M. Farmakis, **E.V. Economou**, M.G. Toutouza, K.I. Kapetanios, T.A. Argyriou, P.K. Toutouzas
XVIIIth Congress of the European Society of Cardiology, August 25 - 29, Birmingham, United Kingdom, 1996

78. "*Activated transforming growth factor β2 is depressed and basic fibroblast growth factor is increased in heart failure patients*"
D. Farmakis, P. Papadopoulos, **E. Economou**, M. Toutouza, T. Argyriou, K. Kapetanios, I. Bosinakou, D. Papadopoulos, P. Toutouzas
8th Annual Meeting of Mediterranean Association of Cardiology and Cardiac Surgery, October 12 - 15, Limassol, Cyprus, 1995

79. "*Influence of calcium antagonists on plasma levels of basic fibroblast growth factor in relation to plasma levels of some arachidonic acid metabolites in patients with mild hypertension*"
E.V. Economou, M. Toutouza, G. Vyssoulis, K. Giannakopoulou, P. Toutouzas
XVIIth Congress of the European Society of Cardiology, August 20 - 24, Amsterdam, The Netherlands, 1995

80. *Serum endothelin changes with calcium antagonist antihypertensive treatment*"
Vyssoulis G., **Economou E**., Giannakopoulou K., Karpanou E., Toutouzas P.
7th Annual Meeting of the Mediterranean Association of Cardiology and Cardiac Surgery, October 5 - 8, Hammamet, Tunisia,1994

13.2. ΕΛΛΗΝΙΚΑ ΣΥΝΕΔΡΙΑ

13.2.1. ΚΛΙΝΙΚΗ ΧΗΜΕΙΑ – ΦΑΡΜΑΚΕΥΤΙΚΗ ΒΙΟΧΗΜΕΙΑ

1. "*Εξατομικευμένη Θεραπεία*"
Ε. ΟΙΚΟΝΟΜΟΥ
Πρόσκληση Διάλεξης στα Πλαίσια των Μηνιαίων Επιστημονικών Συναντήσεων της Ελληνικής Μικροβιολογικής Εταιρείας, 21 Μαϊου, Αίθουσα "Ι. Παπαβασιλείου" Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αθήνα 2014

2. "*Θρομβοφιλία – Σύγχρονη εργαστηριακή προσέγγιση διάγνωσης και θεραπείας*"
Ε. ΟΙΚΟΝΟΜΟΥ
Εισήγηση στα πλαίσια της 1ης Πανελλήνιας Επιστημονικής Διημερίδας Εφαρμοσμένης Εργαστηριακής Ιατρικής, Αθήνα, Royal Olympic Hotel, 11-12 Φεβρουαρίου 2012

3. "*ΦΑΡΜΑΚΟΓΕΝΩΜΙΚΗ: ΣΥΓΧΡΟΝΗ ΠΡΟΚΛΗΣΗ ΣΥΜΒΟΛΗΣ ΤΟΥ ΕΡΓΑΣΤΗΡΙΟΥ ΣΤΗΝ ΕΞΑΤΟΜΙΚΕΥΜΕΝΗ ΘΕΡΑΠΕΥΤΙΚΗ* "
Ε. ΟΙΚΟΝΟΜΟΥ
Εισήγηση σε δορυφορικό συμπόσιο με θέμα "Η Μοριακή Διάγνωση στην Εφαρμογή της Εξατομικευμένης Θεραπείας" στα πλαίσια συνεδρίου με θέμα "ΣΥΓΧΡΟΝΟ ΚΛΙΝΙΚΟ ΕΡΓΑΣΤΗΡΙΟ 2009", Αθήνα, Divani Caravel Hotel, 22-24 Οκτωβρίου 2009

4. "*Θεραπευτική Εξατομίκευση – Η σύγχρονη πρόκληση της συμβολής του εργαστηρίου στη θεραπευτική προσέγγιση"*
Εμμανουήλ Β. Οικονόμου
Πρόσκληση Διάλεξης στα Πλαίσια των Μηνιαίων Επιστημονικών Συναντήσεων της Ελληνικής Μικροβιολογικής Εταιρείας, 19 Φεβρουαρίου, Αμφιθέατρο ΝΙΜΤΣ, Αθήνα, 2008

5. "*Από τη Φαρμακογενετική στη Φαρμακογενωμική : Διέξοδος στην Εξατομίκευση της Φαρμακευτικής Θεραπείας;*"
Εμμανουήλ Β. Οικονόμου
5ο Πανελλήνιο Συνέδριο Κλινικής Χημείας, 18-21 Νοεμβρίου, Αθήνα, 2004

6. "*Ραδιοανοσοαναλυτικός προσδιορισμός της Διαιθυλοστιλβοιστρόλης απευθείας σε ορό ασθενών με καρκίνο του προστάτη*"

Ε.Β. Οικονόμου, Σ.Η. Κακαμπάκος, Γ.Π. Ευαγγελάτος, Δ.Σ. Ιθακήσιος
5ο Πανελλήνιο Φαρμακευτικό Συνέδριο, Αθήνα, 26 - 28 Μαίου, 1990

7. "*Ραδιοανοσοαναλυτικός προσδιορισμός της Διαιθυλοστιλβοιστρόλης (DES) σε βιολογικά υγρά και σε ιστικά εκχυλίσματα. Υπάρχουσες Μέθοδοι-Προβλήματα-Προσανατολισμοί*"
Ε.Β. Οικονόμου, Γ.Π. Ευαγγελάτος, Θ. Σιάτρα-Παπασταϊκούδη, Δ.Σ. Ιθακήσιος
4ο Πανελλήνιο Φαρμακευτικό Συνέδριο, Αθήνα, 21 - 23 Μαίου, 1988

8. "*Μελέτη της επίδρασης χρωστικών στο σύστημα ραδιοανοσοανάλυσης για τη μέτρηση της θυροξίνης*"
Ε.Β. Οικονόμου, Σ.Η. Κακαμπάκος, Ε. Λιβανίου, Κ. Σωτηριάδης-Βλάχος, Γ.Π. Ευαγγελάτος
3ο Πανελλήνιο Φαρμακευτικό Συνέδριο, Αθήνα, 31 Μαίου έως 2 Ιουνίου, 1986

9. "*Συγκριτική μελέτη των μεθόδων χλωραμίνης-Τ, γαλακτοϋπεροξειδάσης και ιωδογόνου για την επισήμανση της ανθρώπινης ωχρινοποιητικής ορμόνης με* ^{125}I"
Σ.Η. Κακαμπάκος, **Ε.Β. Οικονόμου**, Ε. Λιβανίου, Κ. Σωτηριάδης-Βλάχος, Γ.Π. Ευαγγελάτος
3ο Πανελλήνιο Φαρμακευτικό Συνέδριο, Αθήνα, 31 Μαίου έως 2 Ιουνίου, 1986

13.2.2. ΧΕΙΡΟΥΡΓΙΚΗ

1. "*Εργαστηριακή προσέγγιση ελέγχου θρομβοφιλίας – Πότε και Γιατί*"
Ε. ΟΙΚΟΝΟΜΟΥ
Εκπαιδευτική Αγγειολογική Ημερίδα – Κατευθυντήριες οδηγίες στη διάγνωση και θεραπεία αρτηριακής υπέρτασης, σακχαρώδους διαβήτη, δισλιπιδαιμίας, αρτηριακής και φλεβικής θρόμβωσης, θρομβοφιλίας και διακοπής του καπνίσματος. Εισήγηση στα πλαίσια στρογγυλού τραπεζιού με θέμα "Κατευθυντήριες οδηγίες στη διάγνωση και στη θεραπεία θρομβοφιλίας" – 22 Οκτωβρίου 2011, Μεγάλο Αμφιθέατρο του ΓΝΝΘΑ "Η ΣΩΤΗΡΙΑ"

2. "*Ιστολογική και λειτουργική διερεύνηση παγκρεατικής λειτουργίας σε πειραματικό μοντέλο ηπατεκτομής και ισχαιμίας-επαναιμάτωσης ήπατος*"
Κ. Νάστος, Ν. Αρκαδόπουλος, Γ. Δευτερέβος, Ν. Παπουτσιδάκης, Α. Κόνδη-Παφίτη, Γ. Φραγκουλίδης, **Ε. Οικονόμου**, Ι. Ανδρεάδου, Τ. Νομικός, Γ. Κωστοπαναγιώτου, Β. Σμερνιώτης
7ο Πανελλήνιο Συνέδριο Παγκρεατολογίας, CROWNE PLAZA HOTEL, Αθήνα, 3-5 Απριλίου 2009 - ΒΡΑΒΕΙΟ ΕΛΕΥΘΕΡΗΣ ΑΝΑΚΟΙΝΩΣΗΣ

3. "Εργαστηριακή προσέγγιση ομάδων υψηλού κινδύνου φλεβικών θρομβοεμβολικών επεισοδίων»
Εμμανουήλ Β. Οικονόμου
Συμμετοχή σε Στρογγυλό Τραπέζι με θέμα "ΠΡΟΛΗΨΗ ΕΝ ΤΩ ΒΑΘΕΙ ΦΛΕΒΙΚΩΝ ΘΡΟΜΒΩΣΕΩΝ» στα πλαίσια του Μετεκπαιδευτικού Προγράμματος της Β' Πανεπιστημιακής Χειρουργικής Κλινικής του Πανεπιστημίου Αθηνών, 20 Νοεμβρίου, Αμφιθέατρο «ΠΑΠΑΔΗΜΗΤΡΙΟΥ», Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, 2008

4. «*Προστασία από τις βλάβες ισχαιμίας/επαναιμάτωσης κατά τις μείζονες ηπατεκτομές με χρήση δεσφεροξαμίνης*»
Αρκαδόπουλος Ν., **Οικονόμου Ε**., Θεοδωράκη Κ., Καραπάνος Κ., Βασιληκώστας Γ., Παφίτη Α., Κουσκούνη Ε., Βώρος Δ., Βασιλείου Ι., Σμυρνιώτης Β.
4ο Ετήσιο Συνέδριο Ιατρικής Σχολής, 1-2 Ιουνίου, Αίγλη Ζαππείου, 2007

5. «*Αντιμετώπιση οξειδωτικών βλαβών κατά τις μείζονες ηπατεκτομές με χρήση*

δεσφεροξαμίνης. Προοπτική τυχαιοποιημένη μελέτη»
Ν. Αρκαδόπουλος, **Ε. Οικονόμου**, Κ. Θεοδωράκη, Γ. Βασιληκώστας, Κ. Καραπάνος, Α. Παφίτη, Ε. Κουσκούνη, Ι. Βασιλείου, Δ. Βώρος, Β. Σμυρνιώτης
10ο Πανελλήνιο Ηπατολογικό Συνέδριο, 26-29 Απριλίου, Hilton, Αθήνα, 2007
1ο ΒΡΑΒΕΙΟ ΚΑΛΥΤΕΡΗΣ ΕΛΕΥΘΕΡΗΣ ΑΝΑΚΟΙΝΩΣΗΣ

6. «*Μπορεί η διεγχειρητική ταχεία μέτρηση της παραθορμόνης να βελτιώσει τα αποτελέσματα της παραθυρεοειδεκτομής*»
Μαρίνης Α, **Οικονόμου Ε**., Βασιλικώστας Γ., Σταφυλά Β., Κυριαζή Μ., Τσιαντούλα Π., Κωνσταντινίδης Χ., Κουσκούνη Ε., Βασιλείου Ι.
25ο Πανελλήνιο Συνέδριο Χειρουργικής – Διεθνές Χειρουργικό Forum 2006, 22-26 Νοεμβρίου, Αθήνα, 2006

7. «*Χηλική δέσμευση του σιδήρου για πρόληψη βλαβών ισχαιμίας-επαναιμάτωσης κατά τις μείζονες ηπατεκτομές. Προοπτική τυχαιοποιημένη μελέτη*»
Ν. Αρκαδόπουλος**, Ε.Οικονόμου,** Κ. Θεοδωράκη, Γ. Βασιληκώστας, Κ. Καραπάνος, Μ. Φράγκου, Ι. Κόντης, Ι. Βασιλείου, Δ. Βώρος, Π. Δημακάκος, Β Σμυρνιώτης
25ο Πανελλήνιο Συνέδριο Χειρουργικής – Διεθνές Χειρουργικό Forum 2006, 22-26 Νοεμβρίου, Αθήνα, 2006

8. *«Διεγχειρητική μέτρηση παραθορμόνης: ένα εργαστηριακό εργαλείο για την εκτίμηση της επιτυχούς χειρουργικής αντιμετώπισης του υπερπαραθυρεοειδισμού»*
Δ. Ρίζος, **Ε. Οικονόμου**, Ε. Κουσκούνη, Α. Μαρίνης, Ι. Βασιλείου
6ο Πανελλήνιο Συνέδριο Κλινικής Χημείας, 9 – 11 Νοεμβρίου, Συνεδριακό Κέντρο «ΔΑΪΣ», Αθήνα, 2006

13.2.3. ΝΕΟΓΝΟΛΟΓΙΑ – ΠΑΙΔΙΑΤΡΙΚΗ

1. *«Πρωτότυπη, ανεξάρτητη της ομοκυστεϊναιμίας, χρόνα συστηματική φλεγμονώδης αντίδραση χαμηλής εντάσεως σε παχύσαρκα παιδιά προεφηβικής ηλικίας»*
Εμμανουήλ Β. Οικονόμου, Αριάδνη Β. Μαλαμίτση-Puchner, Χρήστος Πίτσαβος, Ευαγγελία Κουσκούνη, Ιωάννα Μαγαζιώτου-Ελευσινιώτη, Γεώργιος Κ. Κρεατσάς
3ο Ετήσιο Επιστημονικό Συνέδριο Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, Αθήνα, 2006

2. *«Επίπεδα νευροτροφινών σε έμβρυα και νεογνά με ενδομήτρια καθυστέρηση ανάπτυξης»*
Κ.Ε. Νικολάου, Α. Μαλαμίτση-Puchner, **Ε. Οικονόμου**, Μ. Μπούτσικου, Θ. Μπούτσικου, Μ. Κυριακάκου, Κ.Ρ. Puchner, Δ. Χασιάκος
3ο Ετήσιο Επιστημονικό Συνέδριο Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, Αθήνα, 2006

3. "*Περιγεννητικές μεταβολές του εγκεφαλικού νευροτροφικού παράγοντα (BDNF) σε πρόωρα και τελειόμηνα νεογνά*"
Α. Μαλαμίτση-Πούχνερ, **Ε. Οικονόμου**, Ο. Ρηγοπούλου, Θ. Μπούτσικου
1ο Συνέδριο Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 4-6 Ιουνίου, Αθήνα, 2004

4. "*Επίπεδα ενδοθηλίνης 1-21 σε παιδιά και εφήβους με ινσουλινοεξαρτώμενο σακχαρώδη διαβήτη (ΙΕΣΔ)*"
Αριάδνη Μαλαμίτση-Puchner, **Ε. Οικονόμου**, Φενέλη Καραχάλιου, Δ. Δελής, Κορίνα Κασσιού, Χ.Σ. Μπαρτσόκας
32ο Πανελλήνιο Παιδιατρικό Συνέδριο, Κέρκυρα, 17 - 19 Ιουνίου 1994

5. "*Επίπεδα ενδοθηλίνης 1-21 (ΕΤ 1-21) στο πρώτο και τέταρτο 24ωρο της ζωής υγιών*

και πασχόντων προώρων νεογνών"
Αριάδνη Μαλαμίτση-Puchner, Θ. Ευσταθόπουλος, Ζωή Χατζησταματίου, **Ε. Οικονόμου**, Σοφία Σεβαστιάδου, Δ. Νικολόπουλος
32ο Πανελλήνιο Παιδιατρικό Συνέδριο, Κέρκυρα, 17 - 19 Ιουνίου 1994

6. "*Επίπεδα των πολυακόρεστων λιπαρών οξέων στο πλάσμα τελειομήνων και προώρων νεογνών*"
Α. Μαλαμίτση-Puchner, Σ. Σεβαστιάδου, Θ. Ευσταθόπουλος, **Ε. Οικονόμου**, Ζ. Χατζησταματίου, Δ. Νικολόπουλος
8ο Πανελλήνιο Συνέδριο Περιγεννητικής Ιατρικής, Αθήνα, 16 - 17 Απριλίου 1994

7. "*Επίπεδα ενδοθηλίνης (ΕΤ) 1-21 στο πλάσμα φυσιολογικών εμβρύων 18 - 24 εβδομάδων*"
Α. Μαλαμίτση-Puchner, Α. Αντσακλής, **Ε. Οικονόμου**, Ν. Παπαντωνίου, Σ. Μεσογίτης, Ν. Κούτρα, Σ. Παπαχαρίτωνος, Γ. Μπίτσιου , Δ. Αραβαντινός
8ο Πανελλήνιο Συνέδριο Περιγεννητικής Ιατρικής, Αθήνα, 16 - 17 Απριλίου 1994

8. "*Επίπεδα ενδοθηλινών-1,2 το 1ο και 4ο 24ωρο της ζωής σε φυσιολογικά τελειόμηνα νεογνά*"
Αριάδνη Μαλαμίτση-Puchner, **Ε. Οικονόμου**, Σοφία Σεβαστιάδου, Θ. Ευσταθόπουλος, Α. Νικολόπουλος
31ο Πανελλήνιο Παιδιατρικό Συνέδριο, Κασσάνδρα Χαλκιδικής, 4 - 6 Ιουνίου 1993

9. "*Επίδραση της βιταμίνης Ε στα επίπεδα της προστακυκλίνης και της θρομβοξάνης στο αίμα προώρων νεογνών*"
Αριάδνη Μαλαμίτση-Puchner, **Εμμανουήλ Β. Οικονόμου**, Εγενία Παπαθωμά, Κωνσταντίνος Παπάς
29ο Πανελλήνιο Παιδιατρικό Συνέδριο, Ηράκλειο Κρήτης, 25 - 26 Μαίου, 1991

13.2.4. ΕΝΔΟΚΡΙΝΟΛΟΓΙΑ - ΜΕΤΑΒΟΛΙΣΜΟΣ

1. "*Αυξημένα επίπεδα χυμοκινών MCP-1 και RANTES στον ορό παχύσαρκων παιδιών*"
Ι. Μαγαζιώτου, Δ. Δαμιανάκη, **Ε. Οικονόμου**, Ι. Ελευσινιώτης, Μ. Τούτουζα, Χ. Στεφανάδης, Π. Τούτουζας
27ο Πανελλήνιο Συνέδριο Ενδοκρινολογίας και Μεταβολισμού, Μάρτιος 23 - 25, Λευκωσία, Κύπρος, 2000

2. "*Αυξημένα επίπεδα χυμοκινών MCP-1 και RANTES στον ορό παχύσαρκων παιδιών*"
Ι. Μαγαζιώτου, Δ. Δαμιανάκη, **Ε. Οικονόμου**, Ι. Ελευσινιώτης, Μ. Τούτουζα, Χ. Στεφανάδης, Π. Τούτουζας
27ο Πανελλήνιο Συνέδριο Ενδοκρινολογίας και Μεταβολισμού, Μάρτιος 23 - 25, Λευκωσία, Κύπρος, 2000

13.2.5. ΜΑΙΕΥΤΙΚΗ - ΓΥΝΑΙΚΟΛΟΓΙΑ

1. "Ο συνδυαστικός κίνδυνος αποτυχημένης εμβρυομεταφοράς μετά απο εξωσωματική γονιμοποίηση (*IVF-ET FAILURE)* της γενετικής ετερογένειας των αιμοπεταλιακών γλυκοπρωτεϊνικών υποδοχέων Ια και ΙΙΙΑ "
Ν. Βλαχάδης, Β. Τσαμαδιάς, Ν. Βραχνής, **Ε. Οικονόμου**
Ελεύθερη ανακοίνωση στα πλαίσια του 40ου ΕΤΗΣΙΟΥ Πανελληνίου Ιατρικού Συνεδρίου, 14-17 Μαϊου 2014

2. "*Ανοσολογία και ART*"

Ε. ΟΙΚΟΝΟΜΟΥ
Εισήγηση στα πλαίσια στρογγυλής τραπέζης με θέμα «Γονιμότητα και ειδικές καταστάσεις» του 6ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 4-5 Απριλίου 2014, Ξενοδοχείο Royal Olympic, Αθήνα

3. "Η έκφραση των παραγόντων CD40, CD40L και ADAM8 σε γυναίκες με ενδομητρίωνση"
Σ.Στεργιώτης, Α. Αυγουλέα, Ε. Νιέερη, **Ε. Οικονόμου,** Γ. Καπαρός, Α. Παλαιολόγου, Λ. Αραβαντινός, Κ. Πανουλής, Γ. Κρεατσάς
Ελεύθερη ανακοίνωση στα πλαίσια του 6ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 4-5 Απριλίου 2014, Ξενοδοχείο Royal Olympic, Αθήνα

4. "Ο πολυμορφισμός C677T μεταβάλει την επίδραση της ορμονικής θεραπείας υποκατάστασης σε μετεμμηνοπαυσιακές γυναίκες "
Δ. Παπαδημητρίου, **Ε. Οικονόμου**, Γ. Καπαρός, Δ. Ρίζος, Κ. Πανουλής, Ε. Δεληγεώργλου, Α. Αλεξάνδρου, Α. Αυγουλέα, Μ. Αποστολάκης, Μ. Κρεατσά, Ε. Κουσκούνη, Ε. Λαμπρινουδάκη
Ελεύθερη ανακοίνωση στα πλαίσια του 6ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 4-5 Απριλίου 2014, Ξενοδοχείο Royal Olympic, Αθήνα

5. "Η γενετική ετερογένεια του αιμοπεταλιακού γλυκοπρωτεΐνικού υποδοχέα Ια σχετίζεται με την αποτυχία εμβρυομεταφοράς μετά απο εξωσωματική γονιμοποίηση (IVF-ET FAILURE)"
Ν. Βλαχάδης, Β. Τσαμαδιάς, Ε. Κουσκούνη, **Ε. Οικονόμου**
Ελεύθερη ανακοίνωση στα πλαίσια του 39ου ΕΤΗΣΙΟΥ Πανελληνίου Ιατρικού Συνεδρίου, Πέμπτη 23 Μαϊου 2013

6. "Η γενετική ετερογένεια των αιμοπεταλιακών γλυκοπρωτεϊνικών υποδοχέων Ια και IIIα σχετίζεται με την λειτουργικότητα των αιμοπεταλίων σε γυναίκες με αποβολές "
Β. Τσαμαδιάς, Ν. Βλαχάδης, Ε. Κουσκούνη, **Ε. Οικονόμου**
Ελεύθερη ανακοίνωση στα πλαίσια του 39ου ΕΤΗΣΙΟΥ Πανελληνίου Ιατρικού Συνεδρίου, Πέμπτη 23 Μαϊου 2013

7. "Φαρμακολογία της φαρμακευτικής αντισύλληψης"
Ε. ΟΙΚΟΝΟΜΟΥ
Εισήγηση στα πλαίσια της ημερίδας «Ημέρες Ενδοκρινολογίας της Γυναίκας» με θέμα «Ορμονικά Αντισυλληπτικά», Σάββατο 21 Μαϊου 2011

8. "*Παθοφυσιολογική και θεραπευτική συσχέτιση αθηρωσκλήρυνσης και οστεοπόρωσης*"
Ε. ΟΙΚΟΝΟΜΟΥ
Εισήγηση στα πλαίσια του 5ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 1-2 Απριλίου 2011, Ξενοδοχείο Stratos Vasilikos, Αθήνα

9. *"Πως επηρεάζει η ηλεκτρομαγνητική ακτινοβολία των κινητών τηλεφώνων την ανδρική γονιμότητα;***"**
Τζανακάκη Δ., Μπάκα Σ., **Οικονόμου Ε**., Χασιάκος Δ., Κονιδάρης Σ.
11^{ο} Πανελλήνιο Συνέδριο Μαιευτικής και Γυναικολογίας, Ξενοδοχείο HILTON, 28-31 Μαϊου 2009

10. *«Οι επιπτώσεις της μείωσης των οιστρογόνων κατά την εμμηνόπαυση στο καρδιαγγειακό σύστημα της γυναίκας»*
Εμμανουήλ Β. Οικονόμου
4^{ο} Πανελλήνιο Συνέδριο Κλιμακτηρίου και Εμμηνόπαυσης, Μέγαρο Μουσικής Αθηνών, 18-19 Απριλίου 2008 Εισήγηση στα Πλαίσια Στρογγυλού Τραπεζιού με θέμα «Εμμηνόπαυση και Καρδιαγγειακό Σύστημα»

11. *«Η επίδραση της ορμονικής θεραπείας, της τιβολόνης και της ραλοξιφαίνηςστους*

χημειοτακτικούς παράγοντες MCP-1 και RANTES»
Βλάχου Σ., Γκαλάπη Φ., Καλλίγερου Ε., Παπαδημητρίου Δ., Λμαπρινουδάκη Ε. **Οικονόμου Ε**., Αυγουλέα Α., Πανουλής Κ., Αλεξάνδρου Α., Κουσκούνη Ε., Κρεατσάς Γ.
4ο Πανελλήνιο Συνέδριο Κλιμακτηρίου και Εμμηνόπαυσης, Μέγαρο Μουσικής Αθηνών, 18-19 Απριλίου 2008

12. *«Η έκφραση της Ε-καντχερίνης σε τραχηλικά επιθηλιακά κύτταρα μετεμμηνοαπυσιακών γυναικών: συσχέτιση με την ορμονική θεραπεία, την τιβολόνη και την ραλοξιφαίνη»*
Β. Σιούλας, Σ. Βλάχου, Α. Αλεξάνδρου, Α. Πολίτη, Ε. Κουτσελίνη, **Ε. Οικονόμου**, Θ.
4ο Πανελλήνιο Συνέδριο Κλιμακτηρίου και Εμμηνόπαυσης, Μέγαρο Μουσικής Αθηνών, 18-19 Απριλίου 2008

13. *«Επίδραση της ορμονικής θεραπείας και της τιβολόνης στα επίπεδα των λιπιδίων, των λιποπρωτεϊνλων και στον αθηρωματικό δείκτη πλάσματος»*
Δ. Παπαδημητρίου, Σ. Βλάχου, Φ. Γκαλάπη, **Ε. Οικονόμου**, Κ. Παπαδιάς, Κ.Πανουλής, Ε. Κουσκούνη, Α. Αλεξάνδρου, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Γ. Κρεατσάς
4ο Πανελλήνιο Συνέδριο Κλιμακτηρίου και Εμμηνόπαυσης, Μέγαρο Μουσικής Αθηνών, 18-19 Απριλίου 2008

14. "Θεραπευτική εξατομίκευση – Η σύγχρονη πρόκληση της συμβολής του εργαστηρίου στη θεραπευτική προσέγγιση"
Εμμανουήλ Β. Οικονόμου
Μηνιαία Επιστημονική Συγκέντρωση της Ελληνικής Μικροβιολογικής Εταιρείας, Αμφιθέατρο Νοσοκομείου ΝΙΜΙΤΣ, 19 Φεβρουαρίου 2008

15. *«Οι αθηρωματογόνες λιποκυτοκίνες και οι δείκτες κυτταρικής απόπτωσης στον ορό επηρεάζονται διαφορετικά από τη θεραπεία σε υγιείς μετεμμηνοπαυσιακές γυναίκες»*
Βλάχου Σ., Χριστοδουλάκος Γ., Λαμπρινουδάκη Ε., **Οικονόμου Ε**., Καλλίγερου Ε., Γκαλάπη Φ., Κρεατσά Μ., Σιάσου Ζ., Πανουλής Κ., Παπαδιάς Κ.
4ο Ετήσιο Συνέδριο Ιατρικής Σχολής, 1-2 Ιουνίου, Αίγλη Ζαππείου, 2007

16. *«Λεπτίνη-Γκρελίνη: Η διαφορετική επίδραση που τους ασκούν η ορμονική θεραπεία και η ραλοξιφαίνη»*
Βλάχου Σ., Λαμπρινουδάκη Ε., Χριστοδουλάκος Γ., **Οικονόμου Ε**., Πανουλής Κ., Καλλίγερου Ε., Γκαλάπη Φ., Αλεξάνδρου Α., Κουσκούνη Ε., Κρεατσάς Γ.
4ο Ετήσιο Συνέδριο Ιατρικής Σχολής, 1-2 Ιουνίου, Αίγλη Ζαππείου, 2007

17. «*Ενδογενείς ορμόνες του φύλου και παράγοντες κινδύνου για αθηρωσκληρυνση σε υγιείς μετεμμηνοπαυσιακές γυναίκες»*
Β. Μπουρνιά, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Δ. Ρίζος, **Ε. Οικονόμου**, Ι. Αργείτης, Σ. Βλάχου, Μ. Κρεατσά, Ε. Κουσκούνη, Δ. Μπότσης
3ο Ετήσιο Επιστημονικό Συνέδριο Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, Αθήνα, 2006

18. *«Επίδραση της ορμονικής θεραπείας, της τιβολόνης και της ραλοξιφαίνης στην απόπτωση και στις λιποκυτοκίνες ρεζιστίνη και αδιπονεκτίνη του ορού»*
Ζ. Σιάσου, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**, Ε. Κουσκούνη, Κ. Πανουλής, Σ. Λάππα, Σ. Βλάχου, Μ. Κρεατσά, Ε. Καλλιγέρου, Γ. Χριστοδουλάκος
3ο Ετήσιο Επιστημονικό Συνέδριο Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, Αθήνα, 2006

19. "*Η δράση της ορμονικής θεραπείας και της ραλοξιφαίνης στις μεταλλοπρωτεϊνάσες 2 και 2 του ορού μετεμμηνοπαυσιακών γυναικών*»

Πανουλής Κ, Χριστοδουλάκος Γ, Λαμπρινουδάκη Ε, **Οικονόμου Ε**, Αυγουλέα Α., Κρεατσά Μ., Μπότσης Δ., Δενδρινός Σ.
3^0 Πανελλήνιο Συνέδριο Κλιμακτηρίου και Εμμηνόπαυσης, 4-6 Μαρτίου 2005, Αθήνα, 2005

20. "*Η δράση της ορμονικής θεραπείας και της ραλοξιφαίνης στη VE-καντχερίνη του ορού μετεμμηνοπαυσιακών γυναικών*»
Κρεατσά Μ., Χριστοδουλάκος Γ., Λαμπρινουδάκη Ε., **Οικονόμου Ε.**, Παπαδιάς Κ., Πανουλής Κ., Αυγουλέα Α., Κρεατσάς Γ.
3^0 Πανελλήνιο Συνέδριο Κλιμακτηρίου και Εμμηνόπαυσης, 4-6 Μαρτίου 2005, Αθήνα, 2005

21. "*Η επίδραση της χορήγησης ορμονικής αντισύλληψης στον οστικό μεταβολισμό θηλέων πειραματοζώων αναπαραγωγικής ηλικίας – Πειραματική Μελέτη*"
Μ. Ελευθεριάδης, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Ο. Γρηγορίου, **Ε. Οικονόμου**, Ε. Αντωνίου, Ε. Κουσκούνη, Δ. Περρέα, Ι. Δοντά, Π. Ράπτου, Γ. Λυρίτης, Γ. Κρεατσάς
1^o Συνέδριο Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 4-6 Ιουνίου, Αθήνα, 2004

22. "*Επίπεδα ενδοθηλινών (ΕΤ 1-21) πλάσματος πριν και μετά από οιστρογονική θεραπεία υποκατάστασης σε πρωτοπαθώς αμηνορροϊκές έφηβες*"
Γ. Κρεατσάς, Α. Μαλαμίτση-Puchner, Ε. Χασάν, **Ε. Οικονόμου**, Δ. Αραβαντινός
7^o Πανελλήνιο Συνέδριο Μαιευτικής και Γυναικολογίας, Μάϊος 14 - 17, Ηράκλειο Κρήτης, 1997

13.2.6. ΚΑΡΔΙΟΛΟΓΙΑ – ΒΙΟΛΟΓΙΑ ΑΓΓΕΙΩΝ

1. *«Οξειδωτικό stress και αθηρωματική νόσος»*
Εμμανουήλ Β. Οικονόμου
Πανελλήνιο Συνέδριο Αθηρωσκλήρυνσης, 29 Νοεμβρίου – 2 Δεκεμβρίου, Ξενοδοχείο CARAVEL-DIVANI, Αθήνα, 2006
Εισήγηση μετά από πρόσκληση στα πλαίσια στρογγύλης τράπεζας με θέμα «ΠΑΘΟΦΥΣΙΟΛΟΓΙΑ ΤΗΣ ΑΘΗΡΩΣΚΛΗΡΥΝΣΗΣ»

2. "*Κλινική σημασία των δεικτών αποδόμησης του κολλαγόνου σε ασθενείς με οξύ έμφραγμα του μυοκάρδιου*"
Δ.Π. Παπαδόπουλος, Κ.Ι. Καπετάνιος, **Ε.Β. Οικονόμου**, Π.Κ. Τούτουζας
23^o Πανελλήνιο Συνέδριο Καρδιολογίας, 30 Οκτωβρίου - 2 Νοεμβρίου, Αθήνα, 2002

3. "*Νεότερα δεδομένα από τη μοριακή βιολογία της στεφανιαίας νόσου : Αθηροσκλήρυνση και Αυτοανοσία*"
Εμμανουήλ Β. Οικονόμου
Εισήγηση στα πλαίσια του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών για το Ακαδημαϊκό Ετος 2000 - 2001, Ιούνιος 2001, 26 Ιουνίου 2001

4. "*Νευροπροστατευτική επίδραση αντιυπερτασικής αγωγής*"
Ι.Σ. Ελευσινιώτης, **Ε. Οικονόμου**, Κ. Τσιούφης, Δ. Παναγιωτάκος, Χ. Πίτσαβος, Χ. Στεφανάδης, Π. Τούτουζας
21^o Πανελλήνιο Συνέδριο Καρδιολογίας, Νοέμβριος 2 - 4, Αθήνα, 2000

5. "*Υπέρταση και ασυμπτωματικές βλάβες στον εγκέφαλο*"
Εμμανουήλ Β. Οικονόμου

Εισήγηση στα πλαίσια του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών για το Ακαδημαϊκό Ετος 1998 - 1999, 1 Ιουνίου 1999

6. "*Η μείωση των επιπέδων της C-αντιδρώσας πρωτεϊνης σε ασθενείς με σταθερή στηθάγχη από τη χορήγηση ασπιρίνης οφείλεται στη μείωση της παραγωγής κυτταροκινών. Μια τυχαιοποιημένη, διπλά τυφλή, ελεγχόμενη από placebo κλινική μελέτη*"
Ι. Οικονομίδης, **Ε. Οικονόμου**, Χ. Πίτσαβος, Χ. Στεφανάδης, Μ. Μαρίνου, Π. Τούτουζας, Π. Νιχογιαννόπουλος
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

7. "*Η διαλυτή μορφή Fas συνδέεται με την ελεύθερη μορφή του ανάλογου της ινσουλίνης αυξητικού παράγοντα τύπου 1 αλλά όχι και με το βασικό αυξητικό παράγοντα των ινοβλαστών στο πλάσμα ασθενών με χρονία καρδιακή ανεπάρκεια τελικών σταδίων*"
Ε. Οικονόμου, Δ. Φαρμάκης, Χ. Στεφανάδης, Χ. Πίτσαβος, Μ. Τούτουζα, Κ. Καπετάνιος, Π. Παπαδόπουλος, Π. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

8. "*Σύγκριση των αυξητικών παραγόντων fIGF-1 και VEGF σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Κ.Ι. Καπετάνιος, Χ.Ι. Στεφανάδης, Χ.Η. Πίτσαβος, **Ε.Β. Οικονόμου**, Δ.Μ. Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

9. "*Συσχέτιση των δεικτών αποδόμησης του κολλαγόνου με τη μυοκαρδιακή βλάβη σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Δ.Π. Παπαδόπουλος, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Κ.Ι. Καπετάνιος, Δ.Μ. Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

10. "*Συσχέτιση δεικτών κολλαγονολυτικής δραστηριότητας και μυοκαρδιακής βλάβης σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Δ.Π. Παπαδόπουλος, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Κ.Ι. Καπετάνιος, Δ.Μ. Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

11. "*Συσχετίσεις της λεπτίνης σε δυσλιπιδαιμικούς ασθενείς*"
Δ.Μ. Φαρμάκης, Χ.Ε. Πίτσαβος, Ι.Ν. Σκούμας, Χ.Σ. Τσελίκα, Κ.Ι. Καπετάνιος, Μ.Γ. Τούτουζα, **Ε.Β. Οικονόμου**, Δ. Μάρκου, Χ.Ι. Στεφανάδης, Π.Κ. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

12. "*Χρονικές μεταβολές του αυξημένου βασικού ινωδοβλαστικού αυξητικού παράγοντα σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Κ.Ι. Καπετάνιος, Χ.Ι. Στεφανάδης, Χ.Η. Πίτσαβος, **Ε.Β. Οικονόμου**, Δ.Μ. Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

13. "*Η αυξημένη αγγειογενίνη ως δείκτης αγγειογένεσης σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Κ.Ι. Καπετάνιος, Χ.Ι. Στεφανάδης, Χ.Η. Πίτσαβος, **Ε.Β. Οικονόμου**, Δ.Μ. Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

14. "*Επίπεδα αμυλοειδούς Α στον ορό ασθενών με ήπια έως μέτρια υπέρταση, πριν και μετά από αγωγή με ένα αναστολέα των διαύλων ασβεστίου*"

Ε. Οικονόμου, Χ. Πίτσαβος, Χ. Στεφανάδης, Α. Τρίκας, Μ. Τούτουζα, Ι. Ελευσινιώτης, Κ. Τσιούφης, Κ. Καπετάνιος, Π. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

15. "*Επίπεδα αντισωμάτων έναντι της φωσφατιδυλοσερίνης στον ορό ασθενών με ήπια έως μέτρια υπέρταση, πριν και μετά από αγωγή με ένα αναστολέα των διαύλων ασβεστίου*"
Ε. Οικονόμου, Χ. Πίτσαβος, Χ. Στεφανάδης,, Μ. Τούτουζα, Α. Τρίκας, Κ. Τσιούφης, Κ. Καπετάνιος, Π. Τούτουζας
19ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 22 - 24, Αθήνα, 1998

16. "*Κυτταροχυμικοί και νευρορμονικοί μηχανισμοί στην παθοφυσιολογία της καρδιακής ανεπάρκειας*"
Ε. Β. Οικονόμου, Π.Κ. Τούτουζας
Εισήγηση στα πλαίσια του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών για το Ακαδημαϊκό Ετος 1997 - 1998, 26 Μαϊου 1998

17. "*Βιοχημικοί δείκτες αποδόμησης του κολλαγόνου στο οξύ έμφραγμα του μυοκαρδίου*"
Δ.Π. Παπαδόπουλος, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Δ.Μ. Φαρμάκης, Μ.Γ. Τούτουζα, Κ.Ι. Καπετάνιος, Π.Κ. Τούτουζας
18ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 23 - 25, Θεσσαλονίκη, Ελλάδα, 1997

18. "*Η αύξηση των επιπέδων των κυκλοφορούντων κυτοκινών συσχετίζεται με την έκταση της στεφανιαίας νόσου*"
Ι. Οικονομίδης, **Ε. Οικονόμου**, Χ. Πίτσαβος, Χ. Στεφανάδης, Μ. Μαρίνου, Π. Τούτουζας, Π. Νιχογιαννόπουλος
18ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 23 - 25, Θεσσαλονίκη, Ελλάδα, 1997

19. "*Η μείωση της καθημερινής ισχαιμίας από την ασπιρίνη σε ασθενείς με σταθερή στηθάγχη οφείλεται στη μείωση παραγωγής κυτοκινών και ενεργοποίησης αιμοπεταλίων*"
Ι. Οικονομίδης, **Ε. Οικονόμου**, Χ. Πίτσαβος, Χ. Στεφανάδης, Μ. Μαρίνου, Π. Τούτουζας, Π. Νιχογιαννόπουλος
18ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 23 - 25, Θεσσαλονίκη, Ελλάδα, 1997

20. "*Τα επίπεδα της υπεροξειδικής Mn-δισμουτάσης του πλάσματος ενέχονται στην προκαλούμενη κατά την άσκηση ισχαιμική προετοιμασία του μυοκαρδίου*"
Α. Μιχαηλίδης, **Μ. Οικονόμου**, Χ. Σεφερλής, Ζ. Ψωμαδάκη, Ι. Νικαλέξης, Γ. Κατσιμακλής, Ι. Αντωνίου, Π. Τούτουζας
18ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 23 - 25, Θεσσαλονίκη, Ελλάδα, 1997

21. "*Ισχαιμική προετοιμασία του μυοκαρδίου (ischaemic preconditioning). Ανασκόπηση των γενικών χαρακτηριστικών και της μοριακής βάσης του φαινομένου*"
Ε.Β. Οικονόμου, Π.Κ. Τούτουζας
Εισήγηση στα πλαίσια του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών για το Ακαδημαϊκό Ετος 1996 - 1997, 11 Φεβρουαρίου 1997

22. "*Ο ενεργοποιημένος transforming growth factor β2 ελλατώνεται και ο basic fibroblast growth factor αυξάνει σε ασθενείς με καρδιακή ανεπάρκεια*"
Δ.Μ. Φαρμάκης, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Μ.Γ. Τούτουζα, Θ.Α.

Αργυρίου, Κ.Ι. Καπετάνιος, Ε. Μποσινάκου, Δ.Π. Παπαδόπουλος, Π.Κ. Τούτουζας
16ο Πανελλήνιο Συνέδριο Καρδιολογίας, Οκτώβριος 5 - 7, Ρόδος, 1995

23. "Ρόλος και in vitro εκτίμηση της λειτουργίας του ενδοθηλίου στην Καρδιολογία"
Εμμανουήλ Β. Οικονόμου
Εισήγηση Δορυφορικού Συμποσίου με θέμα "Ο ΚΑΘΟΡΙΣΤΙΚΟΣ ΡΟΛΟΣ ΤΟΥ ΕΝΔΟΘΗΛΙΟΥ ΣΤΗ ΦΥΣΙΟΛΟΓΙΑ ΚΑΙ ΠΑΘΟΛΟΓΙΑ ΤΟΥ ΚΑΡΔΙΑΓΓΕΙΑΚΟΥ ΣΥΣΤΗΜΑΤΟΣ", στα πλαίσια του 10ου Διεθνούς Συνεδρίου Κλινικής Καρδιολογίας, 28 Απριλίου 1995

24. "*Λειτουργία των ενδοθηλιακών κυττάρων των αγγείων στην αρτηριακή υπέρταση και στο σακχαρώδη διαβήτη. Ρόλος της ομοιόστασης του ελεύθερου ενδοκυττάριου ασβεστίου στα κύτταρα αυτά*"
Εμμανουήλ Β. Οικονόμου
Εισήγηση στα πλαίσια του Μετεκπαιδευτικού Προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών για το Ακαδημαϊκό Ετος 1994 - 1995, 21 Φεβρουαρίου 1995

25. "*Επίδραση αντιυπερτασικής αγωγής με αναστολέα ασβεστίου σε αγγειοδραστικές ουσίες που εκκρίνονται από το αγγειακό ενδοθήλιο και ρυθμίζουν τον αγγειακό τόνο*"
Α.Ε. Γιαννακοπούλου, **Ε.Β. Οικονόμου**, Γ.Π. Βυσσούλης, Ε.Α. Καρπάνου, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
15ο Πανελλήνιο Καρδιολογικό Συνέδριο, Αθήνα, Νοέμβριος, 1994

26. "*Η βιοχημεία της λειτουργίας του ενδοθηλίου στη στεφανιαία νόσο*"
Εμμανουήλ Β. Οικονόμου
Εισήγηση στα πλαίσια του μετεκπαιδευτικού προγράμματος της Καρδιολογικής Κλινικής του Πανεπιστημίου Αθηνών, 28 Ιουνίου 1994

27. "*Επίδραση της αντιυπερτασικής αγωγής με αναστολέα ασβεστίου στην ενδοθηλίνη ορού*"
Α.Ε. Γιαννακοπούλου, **Ε.Β. Οικονόμου**, Γ.Π. Βυσσούλης, Μ.Γ. Τούτουζα, Χ.Σ. Τσελίκα, Π.Κ. Τούτουζας
4ο Πανελλήνιο Συνέδριο Αρτηριακής Υπέρτασης, Θεσσαλονίκη, 18 - 20 Νοεμβρίου, 1993

28. "*Ρόλος των ελευθέρων ριζών οξυγόνου στην καταστολή της μυοκαρδιακής λειτουργίας μετά από στεφανιαίες επεμβάσεις*"
Λ. Χατζηνικολάου, **Ε.Β. Οικονόμου**, Σ. Συμινελάκης, Μ. Κραβαρίτου, Ι. Παπαϊωάννου, Π.Κ. Τούτουζας
14ο Πανελλήνιο Καρδιολογικό Συνέδριο, Αθήνα 28 - 30 Οκτωβρίου 1993

29. "*Η βιοχημεία των ελευθέρων ριζών οξυγόνου*"
Εμμανουήλ Β. Οικονόμου
19ο Ετήσιο Πανελλήνιο Ιατρικό Συνέδριο, 4 - 8 Μαϊου, Αθήνα, 1993 Εισήγηση σε Επιστημονική Συνάντηση της Ιατρικής Εταιρείας Τρικάλων με θέμα "ΕΛΕΥΘΕΡΕΣ ΡΙΖΕΣ ΟΞΥΓΟΝΟΥ", 12 Ιουνίου 1993
Εισήγηση σε Δορυφορικό Συμπόσιο με θέμα "Η ΣΗΜΑΣΙΑ ΤΩΝ ΕΛΕΥΘΕΡΩΝ ΡΙΖΩΝ ΟΞΥΓΟΝΟΥ ΣΤΗΝ ΙΣΧΑΙΜΙΑ ΤΟΥ ΜΥΟΚΑΡΔΙΟΥ - Η ΠΡΟΚΛΗΣΗ ΣΤΗ ΔΕΚΑΕΤΙΑ ΤΟΥ '90", στα πλαίσια της 6ης Ετήσιας Συνάντησης της Μεσογειακής Εταιρείας Καρδιολογίας και Καρδιοχειρουργικής, Κέρκυρα, 29 Σεπτεμβρίου 1993

14. ΕΡΕΥΝΗΤΙΚΟ ΣΥΓΓΡΑΦΙΚΟ ΕΡΓΟ

14.1. ΞΕΝΟΓΛΩΣΣΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ

14.1.1. ΞΕΝΟΓΛΩΣΣΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΠΕΡΙΟΔΙΚΑ ΤΟΥ SCI (Π.Ξ.Δ. SCI)

1.	ΔΗΜΟΣΙΕΥΜΕΝΕΣ	:	66
2.	ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ	:	0
	ΣΥΝΟΛΟ	:	66

ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ

1.	Καρδιολογία - Αγγειολογία	:	14
2.	Γυναικολογία	:	29
3.	Νεογνολογία – Παιδιατρική	:	16
4.	Χειρουργική	:	3
5.	Ψυχιατρική	:	1
6.	Ραδιοφαρμακολογία	:	3

ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ

1.	Πειραματικές Εργασίες	:	3
2.	Κλινικοεργαστηριακές Μελέτες	:	63

ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ

1ος Συγγραφέας σε	:	6
2ος Συγγραφέας σε	:	13
3ος Συγγραφέας σε	:	17
Τελευταίος Συγγραφέας	:	6
Άλλη Θέση	:	24

ΣΥΝΤΕΛΕΣΤΗΣ ΑΠΗΧΗΣΗΣ (IMPACT FACTOR, I.F.) (2013)

Συνολικός	:	295,54
Συνολικός (εκτός Letters to the Editor)	:	166,38
Μέσος Ορος Συνολικού	:	4,41
Προσωπικός	:	190,25
Μέσος Ορος Προσωπικού	:	2,84

ΒΙΒΛΙΟΓΡΑΦΙΚΕΣ ΑΝΑΦΟΡΕΣ

Από SCI Expanded	:	1518
Από Βιβλία	:	42
Αυτοαναφορές	:	22
Διατριβές	:	82
Συνέδρια	:	27
Ηλεκτρονικός τύπος – Λοιπά έντυπα	:	19
Πατέντες	:	4
Συνολικός Αριθμός Βιβλιογραφικών Αναφορών	:	1692
Συνολικός Αριθμός Βιβλιογραφικών Αναφορών (εκτός αυτοαναφορών)	:	1670
Προσωπικός Συνολικός Αριθμός	:	594,90
Προσωπικός Συνολικός Αριθμός (εκτός αυτοαναφορών)	:	584,75
h-index	:	20
i-10 index	:	32

"ΠΡΟΣΩΠΙΚΟΣ" : ΔΙΟΡΘΩΜΕΝΟΣ ΜΕ ΒΑΣΗ ΤΗ ΘΕΣΗ ΤΟΥ ΣΥΓΓΡΑΦΕΩΣ

Συντελεστής ανά θέση	1η και τελευταία θέση	2η θέση	3η θέση	άλλη θέση
	1	0.5	0.33	0.25

14.1.2. ΚΑΤΑΤΟΛΟΣ ΠΛΗΡΩΝ ΞΕΝΟΓΛΩΣΣΩΝ ΔΗΜΟΣΙΕΥΣΕΩΝ ΣΕ ΠΕΡΙΟΔΙΚΑ ΤΟΥ SCI

1. TSIMARIS P, DELIGEOROGLOU E, ATHANASOPOULOS N, **ECONOMOU E**, STAMATELOPOULOS K, RIZOS D, PAPAMICHAEL C, LAMBRINOUDAKI I, MASTORAKOS G, CREATSAS G.
The effect of hormone therapy on biochemical and ultrasound parameters associated with atherosclerosis in 46,XY DSD individuals with female phenotype.
(2014) Gynecol Endocrinol 9:1-5

Θεματολογία	**:**	**Γυναικολογία (Εφηβική)**
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**20**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**1,303**
Προσωπικός Συντελ. Απήχησης	**:**	**0,306**

2. VLACHADIS N, VRACHNIS N, **ECONOMOU E**.
Variant GADL1 and response to lithium in bipolar I disorder. (Letter to the Editor)
(***2014) N Engl J Med. 2014 May 8;370(19):1856***

Θεματολογία	**:**	**Ψυχιατρική**
Μεθοδολογία	**:**	**Φαρμακογενετική**
Αριθ. Ατόμων	**:**	**294**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**51,658**
Προσωπικός Συντελ. Απήχησης	**:**	**51,658**

3. VLACHADIS, N., VRACHNIS, N., **ECONOMOU E**., SIRISTATIDIS, C.
Zooming in on the definition of 'recurrent implantation failure'. (Letter to the Editor)
(2014) Reproductive BioMedicine Online, 2014;29(1):144-5

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**Σχολιασμός άρθρου ανασκόπησης**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**2,675**
Προσωπικός Συντελ. Απήχησης	**:**	**0,883**

4. VLACHADIS, N., VRACHNIS, N., **ECONOMOU, E**.
Fertility treatments and multiple births in the United States (Letter to the Editor)
(2014) New England Journal of Medicine, 370 (11), pp. 1069-1070

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Φαρμακολογία**
Αριθ. Ατόμων	**:**	**Σχολιασμός επιδημιολογικής μελέτης**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**51,658**

Προσωπικός Συντελ. Απήχησης	**:**	**51,658**

5. VLACHADIS, N., TSAMADIAS, V**., ECONOMOU, E**.
Aspirin to improve IVF unexplained implantation rates: Time for an individualized approach (Letter to the Editor)
(2014) Reproductive BioMedicine Online, 28 (1), p. 133

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Φαρμακολογία**
Αριθ. Ατόμων	**:**	**234**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**2,675**
Προσωπικός Συντελ. Απήχησης	**:**	**2,675**

6. VLACHADIS, N., TSAMADIAS, V., **ECONOMOU, E**.
Current concepts of optimal gestational age for delivery based on gestational age associated risks of fetal and neonatal death (Letter to the Editor)
(2013) American Journal of Obstetrics and Gynecology, 209 (6), p. 595

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Παθοφυσιολογία**
Αριθ. Ατόμων	**:**	**Σχολιασμός επιδημιολογικής μελέτης**
Αναφορές	**:**	**1**
Προσωπικός Αριθμός Αναφορών	**:**	**1**
Συντελ. Απήχησης	**:**	**3,877**
Προσωπικός Συντελ. Απήχησης	**:**	**3,877**

7. VLACHADIS, N., TSAMADIAS, V., **ECONOMOU, E**.
Association between miscarriage and future maternal cardiovascular disease. (Letter to the Editor)
(2013) Heart, 99 (22), p. 1706

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Παθοφυσιολογία**
Αριθ. Ατόμων	**:**	**Σχολιασμός επιδημιολογικής μελέτης**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**5,014**
Προσωπικός Συντελ. Απήχησης	**:**	**5,014**

8. VLACHADIS, N., TSAMADIAS, V., **ECONOMOU, E.**
Statins in pregnancy: Safety and perspectives of therapeutic applications (Letter to the Editor)
(2013) BJOG: An International Journal of Obstetrics and Gynaecology, 120 (11), pp. 1439-1440

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Φαρμακολογία**
Αριθ. Ατόμων	**:**	**Σχολιασμός πολυκεντικής προοπτικής μελέτης**
Αναφορές	**:**	**2**
Προσωπικός Αριθμός Αναφορών	**:**	**2**

Συντελ. Απήχησης	**:**	**3,760**
Προσωπικός Συντελ. Απήχησης	**:**	**3,760**

9. STERGIOTI, E., DELIGEOROGLOU, E., **ECONOMOU, E.**, TSITSIKA, A., DIMOPOULOS, K.D., DAPONTE, A., KATSIOULIS, A., CREATSAS, G.
Gene receptor polymorphism as a risk factor for BMD deterioration in adolescent girls with anorexia nervosa
(2013) Gynecological Endocrinology, 29 (7), pp. 716-719

Θεματολογία	**:**	**Γυναικολογία (Εφηβική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία – Γενετική**
Αριθ. Ατόμων	**:**	**40**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**1,303**
Προσωπικός Συντελ. Απήχησης	**:**	**0,429**

10. VARSOS P, NASTOS C, PAPOUTSIDAKIS N, KALIMERIS K, DEFTEREVOS G, NOMIKOS T, PAFITI A, FRAGULIDIS G, **ECONOMOU E**, KOSTOPANAGIOTOU G, SMYRNIOTIS V, ARKADOPOULOS N.
Desferrioxamine Attenuates Pancreatic Injury after Major Hepatectomy under Vascular Control of the Liver: Experimental Study in Pigs.
(2012) HPB Surg.;714672

Θεματολογία	**:**	**Χειρουργική**
Μεθοδολογία	**:**	**Φαρμακευτική Βιοχημεία**
Αριθ. Ατόμων (Πειραματοζώων)	**:**	**12**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**1,939**
Προσωπικός Συντελ. Απήχησης	**:**	**0,485**

11. VITORATOS, N., VLAHOS, N.F., **ECONOMOU, E.**, PANOULIS, K., CREATSAS, G.
Changes in maternal serum thioredoxin (TRX) levels after delivery in preeclamptic and normotensive pregnant women.
(2012) Hypertension in Pregnancy 31 (1) , pp. 140-146

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**27**
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**0,928**
Προσωπικός Συντελ. Απήχησης	**:**	**0,306**

12. PUCHNER K, IAVAZZO C, GOURGIOTIS D, BOUTSIKOU M, BAKA S, HASSIAKOS D, KOUSKOUNI E, **ECONOMOU E**, MALAMITSI-PUCHNER A, CREATSAS G.
The implication of second-trimester amniotic fluid TNF-alpha, cytochrome C and cell death nucleosomes in the prediction of preterm labor and/or premature rupture of membranes.
(2012) Arch Gynecol Obstet.;285(1):37-43

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**

Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**360**
Αναφορές	**:**	**8**
Προσωπικός Αριθμός Αναφορών	**:**	**2**
Συντελ. Απήχησης	**:**	**1,33**
Προσωπικός Συντελ. Απήχησης	**:**	**0,333**

13. ARKADOPOULOS N, NASTOS C, DEFTEREVOS G, KALIMERIS K, PAPOUTSIDAKIS N, ANDREADOU I, NOMIKOS T, PAFITI A, FRAGULIDIS G, **ECONOMOU E**, VARSOS P, KOSTOPANAGIOTOU G, SMYRNIOTIS V.
Pancreatic injury after major hepatectomy: a study in a porcine model.
(2012) Surg Today.;42(4):368-75

Θεματολογία	**:**	**Χειρουργική**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων (Πειραματοζώων)	**:**	**10**
Αναφορές	**:**	**2**
Προσωπικός Αριθμός Αναφορών	**:**	**0,5**
Συντελ. Απήχησης	**:**	**0,963**
Προσωπικός Συντελ. Απήχησης	**:**	**0,241**

14. PAPAKONSTANTINOU K, **ECONOMOU E**, KOUPA E, BABAMETO I, HASIAKOS D, VITORATOS N.
Antepartum and postpartum maternal plasma levels of E-selectin and VE-cadherin in preeclampsia, gestational proteinuria and gestational hypertension.
(2011) J Matern Fetal Neonatal Med.;24(8):1027-32

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**37**
Αναφορές	**:**	**2**
Προσωπικός Αριθμός Αναφορών	**:**	**1**
Συντελ. Απήχησης	**:**	**1,518**
Προσωπικός Συντελ. Απήχησης	**:**	**0,759**

15. SIOUTIS D, **ECONOMOU E**, LAMBRINOUDAKI I, TSAMADIAS V, CREATSA M, LIAPIS A.
Sp1 collagen I A1 polymorphism in women with stress urinary incontinence.
(2011) Int Urogynecol J.;22(7):835-9

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία - Γενετική**
Αριθ. Ατόμων	**:**	**45**
Αναφορές	**:**	**3**
Προσωπικός Αριθμός Αναφορών	**:**	**1,5**
Συντελ. Απήχησης	**:**	**2,169**
Προσωπικός Συντελ. Απήχησης	**:**	**1,085**

16. VITORATOS N, PAPAKONSTANTINOU K, DELIVELIOTOU A, **ECONOMOU E**, PANOULIS C, HASSIAKOS D, CREATSAS GK.
Antepartum and postpartum serum heme oxygenase-1 levels in preeclamptic and normotensive pregnant women.

(2011) In Vivo.;25(3):445-50

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**31**
Αναφορές	**:**	**1**
Προσωπικός Αριθμός Αναφορών	**:**	**0,25**
Συντελ. Απήχησης	**:**	**1,219**
Προσωπικός Συντελ. Απήχησης	**:**	**0,305**

17. PUCHNER K, IAVAZZO C, GOURGIOTIS D, BOUTSIKOU M, BAKA S, HASSIAKOS D, KOUSKOUNI E, **ECONOMOU E**, MALAMITSI-PUCHNER A, CREATSAS G.
Mid-trimester amniotic fluid interleukins (IL-1β, IL-10 and IL-18) as possible predictors of preterm delivery.
(2011) In Vivo.;25(1):141-8

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**362**
Αναφορές	**:**	**10**
Προσωπικός Αριθμός Αναφορών	**:**	**2,5**
Συντελ. Απήχησης	**:**	**1,219**
Προσωπικός Συντελ. Απήχησης	**:**	**0,305**

18. KATERINA PAPAKONSTANTINOU, **EMANOUEL ECONOMOU**, DIMITRIS HASIAKOS, NIKOLAOS VITORATOS
Antepartum and postpartum maternal plasma levels of E-selectin in pre-eclampsia, gestational proteinuria and gestational hypertension. (Letter to the Editor)
(2010) Eur J Obstet Gynecol Reprod Biol.;153(1):112-3

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**37**
Αναφορές	**:**	**2**
Προσωπικός Αριθμός Αναφορών	**:**	**1**
Συντελ. Απήχησης	**:**	**1,843**
Προσωπικός Συντελ. Απήχησης	**:**	**0,922**

19. VITORATOS N, **ECONOMOU E**, IAVAZZO C, PANOULIS K, CREATSAS G.
Maternal serum levels of TNF-alpha and IL-6 long after delivery in preeclamptic and normotensive pregnant women.
(2010) Mediators Inflamm.: ARTICLE No 908649

Θεματολογία	**:**	**Γυναικολογία (Μαιευτική)**
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**17**
Αναφορές	**:**	**20**
Προσωπικός Αριθμός Αναφορών	**:**	**10**
Συντελ. Απήχησης	**:**	**3,882**
Προσωπικός Συντελ. Απήχησης	**:**	**1,941**

20. ARKADOPOULOS N, NASTOS C, KALIMERIS K, **ECONOMOU E**, THEODORAKI K, KOUSKOUNI E, PAFITI A, KOSTOPANAGIOTOU G, SMYRNIOTIS V.
Iron chelation for amelioration of liver ischemia-reperfusion injury.
(2010) Hemoglobin. 34(3):265-77

Θεματολογία	**:**	**Χειρουργική**
Μεθοδολογία	**:**	**Φαρμακευτική Βιοχημεία**
Αριθ. Ατόμων	**:**	**12**
Αναφορές	**:**	**12**
Προσωπικός Αριθμός Αναφορών	**:**	**3**
Συντελ. Απήχησης	**:**	**0,894**
Προσωπικός Συντελ. Απήχησης	**:**	**0,224**

21. LAMBRINOUDAKI IV, AUGOULEA A, CHRISTODOULAKOS GE, **ECONOMOU EV**, KAPAROS G, KONTORAVDIS A, PAPADIAS C, CREATSAS G.
Measurable serum markers of oxidative stress response in women with endometriosis.
(2009) Fertil Steril 91 (1) :46-50

Θεματολογία	**:**	***Γυναικολογία(Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***66***
Αναφορές	**:**	**33**
Προσωπικός Αριθμός Αναφορών	**:**	**8,25**
Συντελ. Απήχησης	**:**	**4,174**
Προσωπικός Συντελ. Απήχησης	**:**	**1,044**

22. LAMBRINOUDAKI IV, KAPAROS G, AUGOULEA A, **ECONOMOU EV**, KREATSA M., PAPADIAS K.
Endometriosis and oxidative stress – serum markers? – Replay (Letter to the Editor).
(2008) Fertil Steril 89(5), 1283 – 1283

Θεματολογία	**:**	***Γυναικολογία (Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***66***
Αναφορές	**:**	**0**
Προσωπικός Αριθμός Αναφορών	**:**	**0**
Συντελ. Απήχησης	**:**	**4,174**
Προσωπικός Συντελ. Απήχησης	**:**	**1,044**

23. CHRISTODOULAKOS GE, LAMBRINOUDAKI IV, CREATSA MG, **ECONOMOU EV,** SIASOU Z, PANOULIS CP, KALLIGEROU I, PAPADIAS C.
Circulating levels of atherogenesis-associated adipocytokines and apoptotic markers are differentially influenced by hormone therapy, tibolone and raloxifene in healthy postmenopausal women.
(2008) Climacteric 11(2):155-65

Θεματολογία	**:**	***Γυναικολογία (Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	**100**
Αναφορές	**:**	**9**
Προσωπικός Αριθμός Αναφορών	**:**	**2,25**
Συντελ. Απήχησης	**:**	***1,961***
Προσωπικός Συντελ. Απήχησης	**:**	***0,490***

24. LAMBRINOUDAKI IV, CHRISTODOULAKOS GE, **ECONOMOU EV**, VLACHOU SA, PANOULIS CP, ALEXANDROU AP, KOUSKOUNI EE, CREATSAS GC.
Circulating leptin and ghrelin are differentially influenced by estrogen/progestin therapy and raloxifene.
(2008) Maturitas 20;59(1):62-71

Θεματολογία	**:**	***Γυναικολογία (Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	**88**
Αναφορές	**:**	**14**
Προσωπικός Αριθμός Αναφορών	**:**	**4,62**
Συντελ. Απήχησης	**:**	***2,844***
Προσωπικός Συντελ. Απήχησης	**:**	***0,939***

25. CHRYSOHOOU C, PANAGIOTAKOS DB, PITSAVOS C, SKOUMAS I, PAPADEMETRIOU L, **ECONOMOU M**, STEFANADIS C.
The implication of obesity on total antioxidant capacity in apparently healthy men and women: The ATTICA study.
(2007) Nutr Metab Cardiovasc Dis 17(8):590-597

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	***Κλινική Βιοχημεία***
Αριθ. Ατόμων	**:**	**3042**
Αναφορές	**:**	**46**
Προσωπικός Αριθμός Αναφορών	**:**	**11,5**
Συντελ. Απήχησης	**:**	***3,9783***
Προσωπικός Συντελ. Απήχησης	**:**	**0,995**

26. CHRYSOHOOU C, PANAGIOTAKOS DB, PITSAVOS C, SKOUMAS J, **ECONOMOU M**, PAPADIMITRIOU L, STEFANADIS C.
The association between pre-hypertension status and oxidative stress markers related to atherosclerotic disease: The ATTICA study.
(2007) Atherosclerosis 192(1):169-176

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**3042**
Αναφορές	**:**	**41**
Προσωπικός Αριθμός Αναφορών	**:**	**10,25**
Συντελ. Απήχησης	**:**	***3,706***
Προσωπικός Συντελ. Απήχησης	**:**	**0,927**

27. CHRISTODOULAKOS GE, LAMBRINOUDAKI IV, **ECONOMOU EV**, PAPADIAS C, VITORATOS N, PANOULIS CP, KOUSKOUNI EE, VLACHOU SA, CREATSAS GC.
Circulating chemoattractants RANTES, negatively related to endogenous androgens, and MCP-1 are differentially suppressed by hormone therapy and raloxifene.
(2007) Atherosclerosis 193(1):142-150

Θεματολογία	**:**	***Γυναικολογία (Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	**88**

Αναφορές	:	*17 (2 αυτοαναφορές)*
Προσωπικός Αριθμός Αναφορών	:	*5,61*
Συντελ. Απήχησης	:	*3,706*
Προσωπικός Συντελ. Απήχησης	:	*1,223*

28. MALAMITSI-PUCHNER A, NIKOLAOU KE, **ECONOMOU E**, BOUTSIKOU M, BOUTSIKOU T, KYRIAKAKOU M, PUCHNER KP, HASSIAKOS D.
Intrauterine growth restriction and circulating neurotrophin levels at term.
(2007) Early Hum Dev 83(7):465-469

Θεματολογία	:	*Παιδιατρική-Νεογνολογία*
Μεθοδολογία	:	*Κλινική Παθολογική Βιοχημεία*
Αριθ. Ατόμων	:	*120*
Αναφορές	:	*20*
Προσωπικός Αριθμός Αναφορών	:	*6,6*
Συντελ. Απήχησης	:	*2,02*
Προσωπικός Συντελ. Απήχησης	:	*0,667*

29. NIKOLAOU KE, MALAMITSI-PUCHNER A, BOUTSIKOU T, **ECONOMOU E**, BOUTSIKOU M, PUCHNER KP, BAKA S, HASSIAKOS D.
The varying patterns of neurotrophin changes in the perinatal period.
(2006) Ann N Y Acad Sci. 1092:426-433

Θεματολογία	:	*Παιδιατρική-Νεογνολογία*
Μεθοδολογία	:	*Κλινική Φυσιολογική Βιοχημεία*
Αριθ. Ατόμων	:	*30*
Αναφορές	:	*26*
Προσωπικός Αριθμός Αναφορών	:	*6,5*
Συντελ. Απήχησης	:	*4,375*
Προσωπικός Συντελ. Απήχησης	:	*1,093*

30. NICOLAOS VITORATOS, CONSTANTINOS PAPADIAS, **EMMANUEL ECONOMOU**, EVANGELOS MAKRAKIS, CONSTANTINOS PANOULIS, GEORGE CREATSAS.
Elevated circulating IL-1β and TNF-alpha, and unaltered IL-6 in first-trimester pregnancies complicated by threatened abortion with an adverse outcome.
(2006) Mediators Inflamm (4):30485

Θεματολογία	:	*Γυναικολογία (Μαιευτική)*
Μεθοδολογία	:	*Κλινική Παθολογική Βιοχημεία*
Αριθ. Ατόμων	:	*53*
Αναφορές	:	*28*
Προσωπικός Αριθμός Αναφορών	:	*9,24*
Συντελ. Απήχησης	:	*3,882*
Προσωπικός Συντελ. Απήχησης	:	*1,281*

31. IRENE LAMBRINOUDAKI, GEORGE CHRISTODOULAKOS, DEMETRIOS RIZOS, **EMMANUEL ECONOMOU**, JOHN ARGEITIS, SOFIA VLACHOU, MARIA CREATSA, EVANGELIA KOUSKOUNI, DIMITRIOS BOTSIS.
Endogenous sex hormones and risk factors for atherosclerosis in healthy greek postmenopausal women.
(2006) Eur J Endocrinol 154(6):907-916

Θεματολογία	**:**	***Γυναικολογία (Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***598***
Αναφορές	**:**	***53***
Προσωπικός Αριθμός Αναφορών	**:**	***13,25***
Συντελ. Απήχησης	**:**	***3,136***
Προσωπικός Συντελ. Απήχησης	**:**	***0,784***

32. GEORGE E. CHRISTODOULAKOS, IRENE V. LAMBRINOUDAKI, **EMMANUEL V. ECONOMOU**, CONSTANTINOS PAPADIAS, CONSTANTINOS PANOULIS, EVANGELIA KOUSKOUNI, SOFIA A. VLACHOU, GEORGE CREATSAS.
Differential effect of hormone therapy and tibolone on lipids, lipoproteins and the atherogenic index of plasma.
(2006) J Cardiovasc Pharmacol 47(4):542-548

Θεματολογία	**:**	***Γυναικολογία(Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	***519***
Αναφορές	**:**	**11 (1 αυτοαναφορά)**
Προσωπικός Αριθμός Αναφορών	**:**	***3,63***
Συντελ. Απήχησης	**:**	***2,383***
Προσωπικός Συντελ. Απήχησης	**:**	***0,786***

33. BOUTSIKOU T, MALAMITSI-PUCHNER A, **ECONOMOU E**, BOUTSIKOU M, PUCHNER KP, HASSIAKOS D.
Soluble vascular endothelial growth factor receptor-1 in intrauterine growth restricted fetuses and neonates.
(2006) Early Hum Dev 82(4):235-239

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**80**
Αναφορές	**:**	**32 (1 αυτοαναφορά)**
Προσωπικός Αριθμός Αναφορών	**:**	***10,56***
Συντελ. Απήχησης	**:**	***2,02***
Προσωπικός Συντελ. Απήχησης	**:**	***0,667***

34. MALAMITSI-PUCHNER A, BOUTSIKOU T, **ECONOMOU E**, TZONOU A, MAKRAKIS E, NIKOLAOU KE, HASSIAKOS D.
Angiopoietin-2 in the perinatal period and the role of intrauterine growth restriction.
(2006) Acta Obstet Gynecol Scand 85:45-48

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***120***
Αναφορές	**:**	***8 (1 αυτοαναφορά)***
Προσωπικός Αριθμός Αναφορών	**:**	***2,64***
Συντελ. Απήχησης	**:**	***1,85***
Προσωπικός Συντελ. Απήχησης	**:**	***0,611***

35. PITSAVOS C., PANAGIOTAKOS D.B., TZIMA N., CHRYSOHOOU C., **ECONOMOU M.**,

ZAMPELAS A., STEFANADIS C.
Adherence to the Mediterranean diet is associated with total antioxidant capacity in healthy adults: The ATTICA study.
(2005) American Journal of Clinical Nutrition, 82 (3) , pp. 694-699

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	**Κλινική Βιοχημεία**
Αριθ. Ατόμων	**:**	**3042**
Αναφορές	**:**	**137**
Προσωπικός Αριθμός Αναφορών	**:**	**34,25**
Συντελ. Απήχησης	**:**	**6,504**
Προσωπικός Συντελ. Απήχησης	**:**	**1,626**

36. **E. ECONOMOU**, A. MALAMITSI-PUCHNER, C.P. PITSAVOS, E.E. KOUSKOUNI, I. MAGAZIOTOU-ELEFSINIOTI, D. DAMIANAKI-URANOU, C.I. STEFANADIS, G. CREATSAS.
Low-grade systemic inflammation profile, unrelated to homocystenemia, in prepubertal obese children.
(2005) Mediators Inflamm 6:337-342

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***114***
Αναφορές	**:**	**12**
Προσωπικός Αριθμός Αναφορών	**:**	**12**
Συντελ. Απήχησης	**:**	***3,882***
Προσωπικός Συντελ. Απήχησης	**:**	***3,882***

37. MALAMITSI-PUCHNER A, BOUTSIKOU T, **ECONOMOU E**, SARANDAKOU A, MAKRAKIS E, HASSIAKOS D, CREATSAS G.
Vascular endothelial growth factor and placenta growth factor in intrauterine growth-restricted fetuses and neonates.
(2005) Mediators Inflamm 5:293-297

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***100***
Αναφορές	**:**	**19 (1 αυτοαναφορά)**
Προσωπικός Αριθμός Αναφορών	**:**	***6,27***
Συντελ. Απήχησης	**:**	***3,882***
Προσωπικός Συντελ. Απήχησης	**:**	***1,281***

38. VLACHOPOULOS C, AZNAOURIDIS K, ALEXOPOULOS N, **ECONOMOU E**, ANDREADOU I, STEFANADIS C.
Effect of dark chocolate on arterial function in healthy individuals.
(2005) Am J Hypertens 18(6):785-791

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	***Κλινική Βιοχημεία***
Αριθ. Ατόμων	**:**	**17**
Αναφορές	**:**	**139**
Προσωπικός Αριθμός Αναφορών	**:**	***34,75***
Συντελ. Απήχησης	**:**	***3,665***

Προσωπικός Συντελ. Απήχησης	**:**	***0,916***

39. ELEFTHERIADES MI, LAMBRINOUDAKI IV, CHRISTODOULAKOS GE, GREGORIOU OV, **ECONOMOU EV**, KOUSKOUNI EE, ANTONIOU AG, PERREA DN, DONTAS IA, RAPTOU PD, LYRITIS GP, CREATSAS GC.
Effect of oral contraceptive treatment on bone mass acquisition in skeletally immature young female rats.
(2005) Contraception 71(5):362-371

Θεματολογία	**:**	***Γυναικολογία (Μαιευτική)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	**60**
Αναφορές	**:**	**6**
Προσωπικός Αριθμός Αναφορών	**:**	***1,5***
Συντελ. Απήχησης	**:**	***3,09***
Προσωπικός Συντελ. Απήχησης	**:**	***0,773***

40. MALAMITSI-PUCHNER A, BOUTSIKOU T, **ECONOMOU E**, MAKRAKIS E, ILIODROMITI Z, KOUSKOUNI E, HASSIAKOS D.
The role of the anti-angiogenic factor endostatin in intrauterine growth restriction.
(2005) J Soc Gynecol Investig 12(3):195-197.

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**40**
Αναφορές	**:**	**11**
Προσωπικός Αριθμός Αναφορών	**:**	***3,63***
Συντελ. Απήχησης	**:**	***2,26* (I.F. 2006)**
Προσωπικός Συντελ. Απήχησης	**:**	***0,746***

41. MALAMITSI-PUCHNER A, **ECONOMOU E**, BOUTSIKOU T, NIKOLAOU KE, VRACHNIS N.
Neurotrophin-3 and FLT3 tyrosine kinase receptor in perinatal life.
(2005) Mediators Inflamm (1):53-56

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Βιοχημεία***
Αριθ. Ατόμων	**:**	**60**
Αναφορές	**:**	**9**
Προσωπικός Αριθμός Αναφορών	**:**	**4,5**
Συντελ. Απήχησης	**:**	**3,882**
Προσωπικός Συντελ. Απήχησης	**:**	**1,941**

42. CHRISTODOULAKOS GE, PANOULIS CP, LAMBRINOUDAKI IV, BOTSIS DS, DENDRINOS SG, **ECONOMOU E**, CREATSAS GC.
The effect of hormone therapy and raloxifene on serum matrix metalloproteinase-2 and 9 in postmenopausal women.
(2004) Menopause 11(3):299-305

Θεματολογία	**:**	***Γυναικολογία (Εμμηνόπαυση)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	***28***

Αναφορές	:	**17 (2 αυτοαναφορές)**
Προσωπικός Αριθμός Αναφορών	:	***4,25***
Συντελ. Απήχησης	:	***3,163***
Προσωπικός Συντελ. Απήχησης	:	***0,791***

43. CHRISTODOULAKOS G, LAMBRINOUDAKI I, PANOULIS C, PAPADIAS C, **ECONOMOU E**, CREATSAS G.
Effect of hormone therapy and raloxifene on serum VE-cadherin in postmenopausal women.
(2004) Fertil Steril 82(3):634-638

Θεματολογία	:	***Γυναικολογία (Εμμηνόπαυση)***
Μεθοδολογία	:	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	:	**28**
Αναφορές	:	**10 (3 αυτοαναφορές)**
Προσωπικός Αριθμός Αναφορών	:	***2,5***
Συντελ. Απήχησης	:	***4,174***
Προσωπικός Συντελ. Απήχησης	:	***1,044***

44. **ECONOMOU EV**, MALAMITSI-PUCHNER AV, PITSAVOS CP, KOUSKOUNI EE, MAGAZIOTOU-ELEFSINIOTI I, DAMIANAKI-URANOU D, STEFANADIS CI, CREATSAS G.
Negative association between circulating total homocysteine and proinflammatory chemokines MCP-1 and RANTES in prepubertal lean, but not in obese, children.
(2004) J Cardiovasc Pharmacol 44(3):310-315

Θεματολογία	:	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	:	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	:	***114***
Αναφορές	:	***8 (2 αυτοαναφορές)***
Προσωπικός Αριθμός Αναφορών	:	***8***
Συντελ. Απήχησης	:	***2,383***
Προσωπικός Συντελ. Απήχησης	:	***2,383***

45. PAPADOPOULOS DP, **ECONOMOU EV**, MAKRIS TK, KAPETANIOS KJ, MOYSSAKIS I, VOTTEAS VE, TOUTOUZAS PK.
Effect of angiotensin-converting enzyme inhibitor on collagenolytic enzyme activity in patients with acute myocardial infarction.
(2004) Drugs Exp Clin Res 30(2):55-65

Θεματολογία	:	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	:	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	:	**24**
Αναφορές	:	**8**
Προσωπικός Αριθμός Αναφορών	:	**4**
Συντελ. Απήχησης	:	**1,030 (I.F. 2006)**
Προσωπικός Συντελ. Απήχησης	:	**0,515**

46. MALAMITSI-PUCHNER A, SARANDAKOU A, TZIOTIS J, **ECONOMOU E**, PROTONOTARIOU E, RIGOPOULOU O.
Chemokines Rantes and interleukin-8 in the perinatal period: changes in serum concentrations.
(2004) Am J Perinatol 21(4):235- 240

Θεματολογία	:	***Παιδιατρική-Νεογνολογία***

Μεθοδολογία	**:**	***Κλινική Βιοχημεία***
Αριθ. Ατόμων	**:**	**80**
Αναφορές	**:**	**3**
Προσωπικός Αριθμός Αναφορών	**:**	**0,75**
Συντελ. Απήχησης	**:**	**1,574**
Προσωπικός Συντελ. Απήχησης	**:**	**0,519**

47. ZERVOUDAKI A, **ECONOMOU E**, PITSAVOS C, VASILIADOU K, AGGELI C, TSIOUFIS K, TOUTOUZA M, STEFANADIS, TOUTOUZAS P.
The effect of Ca2+ channel antagonists on plasma concentrations of matrix metalloproteinases-2 and -9 in essential hypertension.
(2004) Am J Hypertens 17(3):273-276

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία (Υπέρταση)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	**117**
Αναφορές	**:**	**39**
Προσωπικός Αριθμός Αναφορών	**:**	**19,5**
Συντελ. Απήχησης	**:**	**3,665**
Προσωπικός Συντελ. Απήχησης	**:**	**1,833**

48. MALAMITSI-PUCHNER A, **ECONOMOU E**, RIGOPOULOU O, BOUTSIKOU T.
Perinatal changes of brain-derived neurotrophic factor in pre- and fullterm neonates.
(2004) Early Hum Dev 76(1):17-22

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Βιοχημεία***
Αριθ. Ατόμων	**:**	**90**
Αναφορές	**:**	**21**
Προσωπικός Αριθμός Αναφορών	**:**	**10,5**
Συντελ. Απήχησης	**:**	**2,02**
Προσωπικός Συντελ. Απήχησης	**:**	**1,01**

49. ANDREAS P. MICHAELIDES, GEORGE K. ANDRIKOPOULOS, **EMMANUIL V OIKONOMOU**, ZOI D. PSOMADAKI, DIMITRIS J. RICHTER, POLYCHRONIS E. DILAVERIS, NIKOLAOS I. EXADAKTYLOS, CHRISTODOULOS I. STEFANADIS, PAVLOS K. TOUTOUZAS.
Improved myocardial performance during repetitive excercise testing. The role of extracellular superoxide dismutase activity in a model of excercise-induced myocardial preconditioning.
(2003) Am Heart J 146(1):160-167

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**50**
Αναφορές	**:**	**18**
Προσωπικός Αριθμός Αναφορών	**:**	**5,94**
Συντελ. Απήχησης	**:**	**4,497**
Προσωπικός Συντελ. Απήχησης	**:**	**1,484**

50. A. ZERVOUDAKI, **E. ECONOMOU**, C. STEFANADIS, C. PITSAVOS, K. TSIOUFIS, C. AGGELI, K. VASILIADOU, M. TOUTOUZA, P. TOUTOUZAS.

Plasma levels of active extracellular matrix metalloproteinases 2 and 9 in patients with essential hypertension before and after antihypertensive treatment.
(2003) J Hum Hypertens 17(2):119-124

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία (Υπέρταση)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	**67**
Αναφορές	**:**	**82**
Προσωπικός Αριθμός Αναφορών	**:**	**41**
Συντελ. Απήχησης	**:**	**2,818**
Προσωπικός Συντελ. Απήχησης	**:**	**1,409**

51. ANASTASIA KATINIOTI, DIMITRIS TOUSOULIS, **EMANUEL V. ECONOMOU**, CHRISTODOULOS STEFANADIS, ATHANASIOS TRIKAS, CHRISTOS PITSAVOS, COSTAS TENTOLOURIS, ARIS ANDROULAKIS, PAVLOS TOUTOUZAS.
Basic fibroblast growth factor as a predictive marker of advanced severe coronary artery disease in patients with stable angina.
(2002) Int J Cardiol 84 (2-3):195-199

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία (Επεμβατική)***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**48**
Αναφορές	**:**	**11**
Προσωπικός Αριθμός Αναφορών	**:**	**3,63**
Συντελ. Απήχησης	**:**	**5,509**
Προσωπικός Συντελ. Απήχησης	**:**	**1,818**

52. **EMANUEL V. ECONOMOU**, DIMITRIS TOUSOULIS, ANASTASIA KATINIOTI, CHRISTODOULOS STEFANADIS, ATHANASIOS TRIKAS, CHRISTOS PITSAVOS, COSTAS TENTOLOURIS, MARINA G. TOUTOUZA, PAVLOS TOUTOUZAS.
Chemokines in patients with ischaemic heart disease and the effect of coronary angioplasty.
(2001) Int J Cardiol 80 (1):55-60

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία (Επεμβατική)***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**48**
Αναφορές	**:**	**52**
Προσωπικός Αριθμός Αναφορών	**:**	**52**
Συντελ. Απήχησης	**:**	**5,509**
Προσωπικός Συντελ. Απήχησης	**:**	**5,509**

53. A. THEOPISTOU, K. GATZOULIS, **E. ECONOMOU**, C.S. SIDERIS, K. HATZOS, C. STEFANADIS, P. TOUTOUZAS.
Biochemical changes involved in the mechanism of vasovagal syncope.
(2001) Am J Cardiol 88:376-381

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία (Αρρυθμιολογία)***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**34**
Αναφορές	**:**	**16**
Προσωπικός Αριθμός Αναφορών	**:**	**5,28**

Συντελ. Απήχησης	**:**	**3,209**
Προσωπικός Συντελ. Απήχησης	**:**	**1,059**

54. DIAMANTOPOULOS E. CHARITOS D. GEORGOPOULOS V., **ECONOMOU E.**, SFAKIANAKIS M., TOUTOUZAS P., RAPTIS S.
Oxygen free radicals and the effect of a free radical scavenger in patients with intermitant claudication.
(2000) Vasc Surg 34:167-174

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	**61**
Αναφορές	**:**	**3**
Προσωπικός Αριθμός Αναφορών	**:**	**0,75**
Συντελ. Απήχησης	**:**	**0,158 (IF : 1999)**
Προσωπικός Συντελ. Απήχησης	**:**	**0,039**

55. CHRISTODOULOS STEFANADIS, LEONIDAS DIAMANTOPOULOS, JOHN DERNELLIS, **EMANUEL ECONOMOU**, ELEFTHERIOS TSIAMIS, KONSTANTINOS TOUTOUZAS, CHARALABOS VLACHOPOULOS, PAVLOS TOUTOUZAS.
Heat production of atherosclerotic plaques and inflammation assessed by the acute phase proteins in acute coronary syndromes.
(2000) J Mol Cell Cardiol 32:43-52

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**80**
Αναφορές	**:**	**114**
Προσωπικός Αριθμός Αναφορών	**:**	**28,5**
Συντελ. Απήχησης	**:**	**5,148**
Προσωπικός Συντελ. Απήχησης	**:**	**1,287**

56. IGNATIOS IKONOMIDIS, FELICITTA ANDREOTTI, **EMANOUEL ECONOMOU**, CHRISTODOULOS STEFANADIS, PAVLOS TOUTOUZAS, PETROS NIHOYANNOPOULOS.
Increased proinflammatory cytokines in patients with chronic stable angina and their reduction by aspirin.
(1999) Circulation 100:793-798

Θεματολογία	**:**	***Καρδιολογία - Αγγειολογία***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	***84***
Αναφορές	**:**	**491**
Προσωπικός Αριθμός Αναφορών	**:**	**162**
Συντελ. Απήχησης	**:**	***15,2***
Προσωπικός Συντελ. Απήχησης	**:**	***5,016***

57. A. MALAMITSI-PUCHNER, **E.ECONOMOU**, N. PAPANTONIOU, A. ANTSAKLIS, S.MESOGITIS, D. NIKOLOPOULOS.
Lipid peroxidation in healthy fetuses, preterm and fullterm neonates.
(1998) Acta Obstet Gynecol Scand 77:124-126

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Φυσιολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***73***
Αναφορές	**:**	**4**
Προσωπικός Αριθμός Αναφορών	**:**	**2**
Συντελ. Απήχησης	**:**	***1,85***
Προσωπικός Συντελ. Απήχησης	**:**	**0,925**

58. ARIADNE MALAMITSI-PUCHNER, IOANNIS E. MESSINIS, VASSILIKI SAKELLARIOU, **EMMANUEL ECONOMOU**, STYLIANOS MICHALAS.
Circulating endothelin-3 and prolactin concentrations in healthy lactating women during the early puerperium.
(1998) Eur J Endocrinol 138:181-184

Θεματολογία	**:**	***Γυναικολογία (Μαιευτική)***
Μεθοδολογία	**:**	***Κλινική Φυσιολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***50***
Αναφορές	**:**	**2**
Προσωπικός Αριθμός Αναφορών	**:**	**0,5**
Συντελ. Απήχησης	**:**	***3,136***
Προσωπικός Συντελ. Απήχησης	**:**	**0,784**

59. GEORGE C. CREATSAS, ARIADNE V. MALAMITSI-PUCHNER, ELSHEIKH A. HASSAN, **EMANUEL V. ECONOMOU**, DENIS I. ARAVANTINOS.
Endothelin plasma levels in primary amenorrheic adolescents before and after estrogen treatment.
(1996) J Soc Gynecol Invest 3:350-353

Θεματολογία	**:**	***Γυναικολογία (Εφηβική)***
Μεθοδολογία	**:**	***Φαρμακευτική Βιοχημεία***
Αριθ. Ατόμων	**:**	***25***
Αναφορές	**:**	**9 (2 αυτοαναφορές)**
Προσωπικός Αριθμός Αναφορών	**:**	**2,25**
Συντελ. Απήχησης	**:**	***2,26* (I.F. 2006)**
Προσωπικός Συντελ. Απήχησης	**:**	**0,565**

60. A. MALAMITSI-PUCHNER, **E. ECONOMOU**, K. KATSOUYANNI, F. KARACHALIOU, D. DELIS, C.S. BARTSOKAS.
Endothelin 1-21 plasma concentrations in children and adolescents with insulin-dependent diabetes mellitus.
(1996) J Pediatric Endocrinol Metab 9:463-468

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Παθολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***100***
Αναφορές	**:**	***19***
Προσωπικός Αριθμός Αναφορών	**:**	***9,5***
Συντελ. Απήχησης	**:**	***0,747***
Προσωπικός Συντελ. Απήχησης	**:**	***0,374***

61. ARIADNE MALAMITSI-PUCHNER, ARIS ANTSAKLIS, **EMMANUEL ECONOMOU**, SPIROS MESOGITIS, NICOS PAPANTONIOU, NOTA KOUTRA, DENIS

ARAVANTINOS.
Endothelin 1-21 plasma levels in fetuses of 18 - 24 weeks of gestation.
(1995) J Perinatal Med 23:321-325

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Φυσιολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***40***
Αναφορές	**:**	***9* (1 αυτοαναφορά)**
Προσωπικός Αριθμός Αναφορών	**:**	**2,97**
Συντελ. Απήχησης	**:**	***1,949***
Προσωπικός Συντελ. Απήχησης	**:**	***0,585***

62. ARIADNE MALAMITSI-PUCHNER, **EMMANUEL ECONOMOU**, THEODORE EFSTATHOPOULOS, SOFIA SEVASTIADOU, ZOE HADZISTAMATIOU, DIMITRIOS NICOLOPOULOS.
Endothelin 1-21 plasma concentrations on days 1 and 4 of life in healthy and preterm neonates.
(1995) Biol Neonate 67:317-321

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Φυσιολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	***36***
Αναφορές	**:**	**11**
Προσωπικός Αριθμός Αναφορών	**:**	**5,5**
Συντελ. Απήχησης	**:**	**1,741 (IF : 2008)**
Προσωπικός Συντελ. Απήχησης	**:**	**0,871**

63. ARIADNE MALAMITSI-PUCHNER, **EMMANUEL ECONOMOU**, SOPHIA SEVASTIADOU, THEODOROS EFSTATHOPOULOS, DIMITRIOS NICOLOPOULOS.
Endothelin 1-21 plasma levels on the first and fourth postpartum day in normal full-term neonates.
(1994) Dev Pharmacol Ther 20:195-198

Θεματολογία	**:**	***Παιδιατρική-Νεογνολογία***
Μεθοδολογία	**:**	***Κλινική Φυσιολογική Βιοχημεία***
Αριθ. Ατόμων	**:**	**20**
Αναφορές	**:**	**7**
Προσωπικός Αριθμός Αναφορών	**:**	**3,5**
Συντελ. Απήχησης	**:**	**0,313**
Προσωπικός Συντελ. Απήχησης	**:**	**0,157**

64. **E. V. ECONOMOU**, E. LIVANIOU, G. P. EVANGELATOS, D. S. ITHAKISSIOS. A.
A ^{125}I-Radioimmuno assay for Diethylstilbestrol in Serum of Patients with Prostatic Cancer treated with Stilphostrol.
(1993) Clin Chim Acta 216:81-90

Θεματολογία	**:**	***Ραδιοφαρμακολογία***
Μεθοδολογία	**:**	***Φαρμακευτική Ραδιοχημεία***
Αριθ. Ατόμων	**:**	**9**
Αναφορές	**:**	**1**
Προσωπικός Αριθμός Αναφορών	**:**	**1**
Συντελ. Απήχησης	**:**	**2,85**
Προσωπικός Συντελ. Απήχησης	**:**	**2,85**

65. **E. V. ECONOMOU**, E. LIVANIOU, G. P. EVANGELATOS, D. S. ITHAKISSIOS.
Preparation of the N-[4-hydroxy-(3-125I)-iodophenethyl]-6-(4-O-diethylstilbestryl)-hexanamide for Diethylstilbestrol (DES) Radioimmunoassays.
(1992) Steroids 57:27-31

Θεματολογία	:	*Ραδιοφαρμακολογία*
Μεθοδολογία	:	*Φαρμακευτική Ραδιοχημεία*
Αριθ. Ατόμων	:	*(Πειραματική Μελέτη)*
Αναφορές	:	*1 (αυτοαναφορά)*
Προσωπικός Αριθμός Αναφορών	:	**1**
Συντελ. Απήχησης	:	*2,803*
Προσωπικός Συντελ. Απήχησης	:	*2,803*

66. **E. V. ECONOMOU**, S. E. KAKABAKOS, G. P. EVANGELATOS, D. S. ITHAKISSIOS.
Direct Radioimmunoassay for Diethylstilbestrol (DES) in Serum.
(1990) Steroids 55:545-550

Θεματολογία	:	*Ραδιοφαρμακολογία*
Μεθοδολογία	:	*Φαρμακευτική Ραδιοχημεία*
Αριθ. Ατόμων	:	*(Πειραματική Μελέτη)*
Αναφορές	:	*1 (1 αυτοαναφορά)*
Προσωπικός Αριθμός Αναφορών	:	**1**
Συντελ. Απήχησης	:	*2,803*
Προσωπικός Συντελ. Απήχησης	:	*2,803*

14.1.3. ΞΕΝΟΓΛΩΣΣΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΠΕΡΙΟΔΙΚΑ ΕΚΤΟΣ SCI (Π.Ξ.Δ. εκτός SCI)

ΣΥΝΟΛΟ : 1. ΔΗΜΟΣΙΕΥΜΕΝΕΣ : 1
2. ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ : 0

ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ

1.	**Νεογνολογία – Παιδιατρική**		**1**

ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ

1.	**Κλινικοεργαστηριακές Μελέτες**	:	**1**

ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ

1ος Συγγραφέας σε	:	**0**
2ος Συγγραφέας σε	:	**1**
Τελευταίος Συγγραφέας	:	**0**
Άλλη Θέση	:	**0**

ΒΙΒΛΙΟΓΡΑΦΙΚΕΣ ΑΝΑΦΟΡΕΣ

Από SCI Expanded	:	**0**
Από Βιβλία	:	**0**
Αυτοαναφορές	:	**0**
ΣΥΝΟΛΟ	:	**0**

14.1.4. ΚΑΤΑΤΟΛΟΣ ΞΕΝΟΓΛΩΣΣΩΝ ΔΗΜΟΣΙΕΥΣΕΩΝ ΣΕ ΠΕΡΙΟΔΙΚΑ ΕΚΤΟΣ SCI

1. *A. MALAMITSI-PUCHNER,* ***E. V. ECONOMOU****, E. PAPATHOMA, C. PAPAS.*
Vitamine E decreases the levels of prostacycline and thromboxane in the blood of premature neonates.
(1993)Int J Feto-Maternal Med 6:47-53

Θεματολογία	**:**	Νεογνολογία
Μεθοδολογία	**:**	Κλινική Φαρμακευτική Βιοχημεία
Αριθ. Ατόμων	**:**	***25***
Αναφορές	**:**	***0***
Προσωπικός Αριθμός Αναφορών	**:**	**0**

14.1.5. ΑΝΑΛΥΤΙΚΗ ΚΑΤΑΣΤΑΣΗ ΔΙΕΘΝΩΝ ΒΙΒΛΙΟΓΡΑΦΙΚΩΝ ΑΝΑΦΟΡΩΝ ΣΕ ΞΕΝΟΓΛΩΣΣΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΠΕΡΙΟΔΙΚΑ ΕΝΤΟΣ ΚΑΙ ΕΚΤΟΣ SCI

1. (2014) Gynecol Endocrinol 9:1-5.
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

2. N Engl J Med. 2014 May 8;370(19):1856 (2014)
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

3. Reproductive BioMedicine Online, . Article in Press. (2014)
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

4. New England Journal of Medicine, 370 (11), pp. 1069-1070 (2014)
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

5. Reproductive BioMedicine Online, 28 (1), p. 133. (2014)
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

6. American Journal of Obstetrics and Gynecology, 209 (6), p. 595 (2014)
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 1
Αναφορά από:

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7. *(2013) Heart, 99 (22), p. 1706 (Letter to the Editor)*
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

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Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 2
Αναφορά από:

1. Calcaterra F, Taddeo A, Colombo E, Cappelletti M, Martinelli A, Calabrese S, Mavilio D, Cetin I, Della Bella S
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2. Reply to Letter: Statins in pregnancy: Safety and perspectives of therapeutic applications (2013) BJOG: An International Journal of Obstetrics and Gynaecology, 120 (11), pp. 1439-1440.

9. *(2013) Gynecological Endocrinology, 29 (7), pp. 716-719*
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

10. HPB Surg. 2012;2012:714672. Epub 2012 Jun 25
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

11. *Hypertension in Pregnancy* 31 (1) , pp. 140-146, 2012
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 0

12. Arch Gynecol Obstet.;285(1):37-43., 2012
Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 8
Αναφορά από:

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J Obstet Gynaecol Res. 2013 Jan;39(1):26-30.

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Placental alpha microglobulin-1 (AmniSure test) versus insulin-like growth factor binding protein-1 (Actim PROM test) for detection of premature rupture of fetal membranes
(2013) Journal of Obstetrics and Gynaecology Research, 39 (6), pp. 1129-1136.

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(2013) Iranian Red Crescent Medical Journal, 15 (10),

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Value of serum urocortin concentration in the prediction of preterm birth
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Global consequences of liver ischemia/reperfusion injury
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Desferrioxamine attenuates pancreatic injury after major hepatectomy under vascular control of the liver: Experimental study in pigs
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Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 2
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Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 10
Αναφορά από:

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Placental alpha microglobulin-1 (AmniSure test) versus insulin-like growth factor binding protein-1 (Actim PROM test) for detection of premature rupture of fetal membranes
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Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 2
Αναφορά από:

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Early pregnancy soluble E-selectin concentrations and risk of preeclampsia
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Endothelin 1-21 plasma concentrations in children and adolescents with insulin-dependent diabetes mellitus

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Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 7
Αναφορά από:

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Development of endothelin receptors in perinatal rabbit pulmonary resistance arteries
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Endothelin-1: Possible implications in pulmonary vascular disease
(1997) Heart and Lung: Journal of Acute and Critical Care, 26 (4), pp. 299-304.

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Endothelin 1-21 plasma concentrations in children and adolescents with insulin-dependent diabetes mellitus
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Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 1
Αναφορά από:

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Σύνολο Βιβλιογραφικών αναφορών δημοσίευσης : 1
Αναφορά από:

1. ECONOMOU EV, LIVANIOU E, EVANGELATOS GP, et al.
A I-125 RADIOIMMUNOASSAY FOR DIETHYLSTILBESTROL IN SERUM OF PATIENTS WITH PROSTATIC-CANCER TREATED WITH STILPHOSTROL
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A I-125 RADIOIMMUNOASSAY FOR DIETHYLSTILBESTROL IN SERUM OF PATIENTS WITH PROSTATIC-CANCER TREATED WITH STILPHOSTROL
CLIN CHIM ACTA 216 (1-2): 81-90 JUL 16 1993

14.1.6. ΞΕΝΟΓΛΩΣΣΕΣ ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ

Συνολικός Αριθμός	:	**127**
ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1. Κλινική Χημεία – Φαρμακευτική Βιοχημεία	:	**14**
2. Νεφρολογία – Βιολογία Νεφρικών Αρτηριών	:	**3**
3. Μαιευτική Γυναικολογία	:	**14**
4. Παιδιατρική - Νεογνολογία	:	**16**
5. Καρδιολογία – Αγγειολογία	:	**80**
ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
1ος Συγγραφέας ή μόνος σε	:	**38**
2ος Συγγραφέας σε	:	**18**
Τελευταίος Συγγραφέας σε	:	**4**
Άλλη Θέση	:	**60**
	:	

14.1.6.1. ΞΕΝΟΓΛΩΣΣΕΣ ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΔΙΕΘΝΗ ΠΕΡΙΟΔΙΚΑ

Συνολικός Αριθμός	:	**102**
ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1. Κλινική Χημεία – Φαρμακευτική Βιοχημεία	:	**11**
2. Νεφρολογία – Βιολογία Νεφρικών Αρτηριών	:	**3**
Μαιευτική Γυναικολογία	:	**5**
Παιδιατρική - Νεογνολογία	:	**7**
Καρδιολογία – Αγγειολογία	:	**76**
ΣΥΝΤΕΛΕΣΤΗΣ ΑΠΗΧΗΣΗΣ (IMPACT FACTOR, I.F.) (2013)		
Συνολικός	:	**1312,16**
Μέσος Ορος Συνολικού	:	**15,22**
Προσωπικός	:	**746,6**
Μέσος Ορος Προσωπικού	:	**7,32**
Συνολικός Αριθμός Βιβλιογραφικών Αναφορών	:	**10**
Συνολικός Αριθμός Βιβλιογραφικών Αναφορών (εκτός αυτοαναφορών)	:	**9**
Προσωπικός Συνολικός Αριθμός Βιβλιογραφικών Αναφορών	:	**5,58**
Προσωπικός Συνολικός Αριθμός Βιβλιογραφικών Αναφορών (εκτός Αυτοαναφορών)	:	**4,58**
ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
1ος Συγγραφέας ή μόνος σε	:	**33**
2ος Συγγραφέας σε	:	**13**
Τελευταίος Συγγραφέας σε	:	**0**
Άλλη Θέση	:	**56**

14.1.6.2. ΚΑΤΑΛΟΓΟΣ ΠΕΡΙΛΗΨΕΩΝ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΩΝ ΣΕ ΔΙΕΘΝΗ ΠΕΡΙΟΔΙΚΑ

ΚΛΙΝΙΚΗ ΧΗΜΕΙΑ – ΦΑΡΜΑΚΕΥΤΙΚΗ ΒΙΟΧΗΜΕΙΑ

1. "*Effects of intravenous infusion of desferrioxamine on pro- and anti-inflammatory selected cytokines balance in warm hepatic ischemia-reperfusion injury of patients undergoing major liver resection performed under selective vascular exclusion*"
Economou EV, Kouskouni E, Smymiotis V, et al.
Clin Chem 53 (6): A141-A141 C-122 Suppl. S JUN 2007 **IF : 7,149**

2. **"***Attenuated and delayed elevation of serum ischemia modified albumin by intravenous infusion of desferrioxamine in hepatic warm ischemia/reperfusion injury"*
Economou EV, Efstratiou V, Smyrniotis V, et al.
Clin Chem 53 (6): A81-A81 B-91 Suppl. S JUN 2007 **IF : 7,149**

3. **"***Protection from interieukin-1 beta converting enzyme (caspase-1)-related apoptotic cell death by intravenous infusion of desferrioxamine in hepatic warm ischemia/reperfusion injury"*
Economou EV, Loginidis I, Smyrniotis V, et al.
Clin Chem 53 (6): A35-A35 A112 Suppl. S JUN 2007 **IF : 7,149**

4. "*Lack of a homeostatic association between modest homocysteinemia and leukocyte transendothelial migration in childhood and adolescence obesity*"
E. V. Economou, A. Malamitsi-Puchner, E. Kouskouni, I. Magaziotou-Elefsinioti, D. Damianaki-Uranou, C. I. Stefanadis, G. Creatsas
Clin Chem : 50(S6), p. A122, abst. No D-47, 2004 **IF : 7,149**

5. "*Adaptive angiogenesis fails to counteract elevated endothelial leukocyte adhesiveness after percutaneous transluminal coronary angioplasty*"
E. V. Economou, E. Kouskouni, M.G. Toutouza, G. Creatsas, C. Stefanadis
Clin Chem : 50(S6), p. A21-22, abst. No A-72, 2004 **IF : 7,149**

6. "*Different patterns of alterations in circulating matrix metalloproteinases 2 and 9 after a severe acute myocardial infarction*"
E. V. Economou, E. Kouskouni, C.P.Pitsavos, G. Creatsas, C. Stefanadis
Clin Chem : 50(S6), p. A14, abst. No A-47, 2004 **IF : 7,149**

7. *"Modest homocysteinemia does not contribute to chronic low-grade inflammation in childhood and adolescence obesity"*
E. V. Economou, A. Malamitsi-Puchner, E. Kouskouni, I. Magaziotou-Elefsinioti, D. Damianaki-Uranou, C. I. Stefanadis, G. Creatsas
Clin Chem : 50(S6), p. A123, abst. No D-52, 2004 **IF : 7,149**

8. "*Coincidental elevation of circulating amyloid beta 42 and the peripheral inflammatory response after a severe acute myocardial infarction*"
E. V. Economou, E. Kouskouni, C.P.Pitsavos, A. Masourou, G. Creatsas, C. Stefanadis
Clin Chem : 50(S6), p. A9, abst. No A-29, 2004 **IF : 7,149**

9. "*The influence of calcium antagonists on plasma levels of endothelin 1-21 and arachidonic acid metabolites in chronically hypertensive patients*"
E. Economou, G. Vyssoulis, K. Giannakopoulou, M. Toutouza, P. Toutouzas
Clin Chem : 40(6), 1042 (P280), 1994 **IF : 7,149**

10. "*Plasma endothelin 1-21 levels in fetuses at 18-24 weeks of gestation*"
E. Economou, A. Malamitsi-Puchner, A. Antsaklis, D. Aravantinos

Clin Chem : 40(6), 1042 (P280), 1994 **IF : 7,149**

11. "*A 125I-radioligand assay for serum diethylstilbestrol (DES)*"
G. P. Evangelatos, **E. V. Economou**, E. Livaniou, D. S. Ithakissios
Clin Chem : 37(6), 1039 (P616), 1991 **IF : 7,149**

ΝΕΦΡΟΛΟΓΙΑ – ΒΙΟΛΟΓΙΑ ΝΕΦΡΙΚΩΝ ΑΡΤΗΡΙΩΝ

1. "*The angiotensin converting enzyme inhibitor ramipril increases total antioxidant capacity in haemodialysis patients*"
Dimitriadis GD, Chouliaras IC, **Economou EV**, Theodoridis TG, Galea VTH, Chouliaras GL, Metaxatos GL, Cokkinou VD, Hadjiconstantinou VE.
Nephrol Dial Transplant (Abstract Suppl.): 20, V127-V127, 2005 **IF : 3,371**

2. "*The angiotensin converting enzyme inhibitor ramipril reduces serum CRP levels in hemodialysis patients*"
George Dimitriadis, Ioannis Chouliaras, **Emanuel Economou**, Paraskevi Dagounaki, Gerge Tsagalis, Theofanis Apostolou, Vasilios Margelos, George Metaxatos, Valsamakis Hadjiconstantinou
Nephrol Dial Transpl : 18 (Abstract Suppl. 4), 721, W549, 2003 **IF : 3,371**

3. "*The anti-inflammatory effects of atorvastatin in hemodialysis patients*"
I. Chouliaras, G. Dimitriadis, **E. Economou**, P. Dagounaki, L. Sinodinos, G. Koutroubas, Chr. Christodoulidou, N. Nikolopoulou, V. Hadjiconstantinou
Nephrol Dial Transpl : 18 (Abstract Suppl. 4), 712, W517, 2003 **IF : 3,371**

ΜΑΙΕΥΤΙΚΗ - ΓΥΝΑΙΚΟΛΟΓΙΑ

1. "*Second trimester amniotic fluid IL-10 and IL-18 concentrations cannot be used as predictors of preterm delivery*"
K. Puchner, C. Iavazzo, D. Gourgiotis, M. Boutsikou, S. Baka, D. Hassiakos, E. Kouskouni, **E. Economou**, K. Tassis, G. Creatsas
The Journal of Maternal-Fetal & Neonatal Medicine, PS18, p. 108, 23 (suppl. 1), 2010 **IF : 1,518**

2. "*Can apoptotic molecules during genetic amniocentesis be used to predict preterm delivery?*"
K. Puchner, C. Iavazzo, D. Gourgiotis, M. Boutsikou, S. Baka, D. Hassiakos, E. Kouskouni, **E. Economou**, K. Tassis, G. Creatsas
The Journal of Maternal-Fetal & Neonatal Medicine, p. 629, 23 (suppl. 1), 2010 **IF : 1,518**

3. "*IL-β midtrimester amniotic fluid concentrations are positively associated with preterm delivery*"
K. Puchner, C. Iavazzo, D. Gourgiotis, M. Boutsikou, S. Baka, D. Hassiakos, E. Kouskouni, **E. Economou**, K. Tassis, G. Creatsas
The Journal of Maternal-Fetal & Neonatal Medicine, PS17, p. 108, 23 (suppl. 1), 2010 **IF : 1,518**

4. *"Healthy postmenopousal women with higher index of androgeneicity exhibit a pro-atherogenic risk profile"*
Lambrinoudaki I., Christodoulakos G., Vlachou S., Rizos D., **Economou E.**, Argeitis J., Creatsa M., Kouskouni E., Augoulea A., Botsis D.
Eur J Clin Invest 37: 44-45 136 Suppl. 1 May 2007 **IF : 3,365**

5. *"Effect of hormone therapy, tibolone and raloxifene on the circulating markers of chemotaxis, MCP-1 and RANTES in postmenopausal women"*
Christodoulakos G., Lambrinoudaki I., Karaflou M., Vlachou S., **Economou E.**, Papadias C., Vitoratos N., Panoulis C., Kouskouni E., Augoulea A., Creatsas G**.**
Eur J Clin Invest 37: 31-31 94 Suppl. 1 MAY, 2007 **IF : 3,365**

ΠΑΙΔΙΑΤΡΙΚΗ - ΝΕΟΓΝΟΛΟΓΙΑ

1. "*Expression of angiopoietin-2 and endostatin in intrauterine growth restriction during the perinatal period*"
   Ar. Malamitsi-Puchner, T. Boutsikou, **E. Economou**, Z. Iliodromiti, E. Kouskouni, D. Hasiakos
   Pediatr Res: 56(3), 174, 2004 **IF : 2,673**

2. "*Perinatal changes of BDNF in pre- and fullterm neonates*"
   A.Malamitsi-Puchner, **E. Economou**, O. Rigopoulou, T. Boutsikou
   Pediatr Res : 54(4), 583, 0353NEO, 2003 **IF : 2,673**

3. "*Plasma levels of total homocysteine are elevated in overweight adolescents*"
   I. Magaziotou, D. Damianaki, **E. Economou**, I. Elefsiniotis, A. Mariolis, C. Stefanadis, P. Toutouzas
   Horm Res (Suppl. 2) : 53, P3-465, 135, 2000 **(IF : 2,480)**

4. "*Circulating monocyte and T-lymphocyte specific chemokines are elevated in overweight adolescents*"
   I Magaziotou, D. Damianaki, I. Elefsiniotis, **E. Economou**, A. Mariolis, C. Stefanadis, P. Toutouzas
   Horm Res (Suppl. 2) : 53, P3-464, 134, 2000 **(IF : 2,480)**

5. "*Endothelin (ET) 1-21 plasma levels in the first and fourth day of life in healthy and ill premature infants*"
   Ariadne Malamitsi-Puchner, Theodore Efstathopoulos, Zoe Hadjistamatiou, **Emmanuel Economou**, Sophia Sevastiadou, Demetrios Nicolopoulos
   Pediatr Res : 36(1), P143 1994 **IF : 2,673**

6. "*Postnatal changes of lipid peroxide levels : possible indirect index of gradually increased oxygenation ?*"
   A. Malamitsi-Puchner, **E. Economou**, T. Efstathopoulos, S. Sevastiadou, D. Nicolopoulos
   Biol Neonate : 64, 182, 1993 **(IF :1,9)**
   [IF 2006]

7. "Plasma levels of total homocysteine are elevated in overweight children and adolescents"
   E. Economou, M. Toutouza, C. Pitsavos, C. Stefanadis, J. Magaziotou-Elefsinioti, J. Elefsiniotis, D. Uranou, P. Toutouzas
   Atherosclerosis : 151(1), 276 (ThP27:W31), 2000 **IF : 3,706**

ΚΑΡΔΙΟΛΟΓΙΑ - ΑΓΓΕΙΟΛΟΓΙΑ

"*Long-term fish consumption offers cardiovascular protection in healthy people due to its anti-arrhythmic and anti-inflammatory properties; The Attica study.*"
Chrysohoou C, Pitsavos C, Skoumas J, Krinos X, Papademetriou L, Kambaxis M, Nikolaou V, Panagiotakos D, **Economou M**, Stefanadis C
J Am Coll Cardiol (abstr. suppl.) 49 (9): 367A-367A Suppl. A MAR 6 2007 **IF : 14,086**

2. *"Increased inflammatory and coagulant activity in patients with spontaneous echocardiographic contrast in the thoracic aorta*"
   Aggeli C, Giannopoulos G, **Economou E**, et al.
   Eur Heart J 27: 905-905 Suppl. 1 AUG 2006 **IF : 14,097**

3. *"The association between pre-hypertension status and oxidative stress markers related to atherosclerotic disease; the ATTICA study"*
   Chrysohoou C, Pitsavos C, Panagiotakos DB, et al.
   Eur Heart J 27: 668-669 Suppl. 1 AUG 2006 **IF : 14,097**

4. *"Association of spontaneous echocardiographic contrast in the thoracic aorta with increased systemic inflammatory and coagulant activity"*
Aggeli C, Giannopoulos G, **Economou E**, et al.
Circulation 114 (18): 760-761 Suppl. S OCT 31 2006 **IF : 15,202**

5. "*Similar kinetic profile in serum levels of heparin-binding growth factors bFGF and HGF in acute myocardial infarction patients: the double peak hypothesis*"
K.I. Kapetanios, C.E. Pitsavos, S. Kastelanos, G.P. Vyssoulis, **E.V. Economou**, M.G. Toutouza, C.I. Stefanadis
Eur Heart J: 25 (Abst. Suppl.): P3493, 599, 2004 **IF : 14,097**

6. "*The clinical significance of circulating levels of granulocyte/macrophage-colony stimulating factor in acute myocardial infarction patients*"
Konstantinos I. Kapetanios, Christos E. Pitsavos, Stamatis Kastelanos, Grigorios P. Vyssoulis, Demosthenes B. Panagiotakos, **Emanuel V. Economou**, Marina G. Toutouza, Christodoulos I. Stefanadis, Pavlos K. Toutouzas
J Am Coll Cardiol (abst. suppl.) : (Suppl. A) : 41 (6), 405A (881FO-5), 2003 **IF : 14,086**

7. "*Concerted action of various angiogenic factors during acute myocardial infarction in patients : the three lines of defense*"
Konstantinos I. Kapetanios, Christos E. Pitsavos, Stamatis Kastelanos, Grigorios P. Vyssoulis, Demosthenes B. Panagiotakos, **Emanuel V. Economou**, Marina G. Toutouza, Christodoulos I. Stefanadis, Pavlos K. Toutouzas
J Am Coll Cardiol (abst. suppl.): (Suppl. A): 41 (6), 343A (1049-111), 2003 **IF : 14,086**

8. "*A beta chemokine (MCP-1)-induced antigen-independent cellular and not a humoral immune response can be stimulated after stent percutaneous transluminal coronary angioplasty*"
Emanuel V. Economou, Christos Pitsavos, Anastasia Katinioti, Konstantinos Tedolouris, Athanasios Trikas, Marina Toutouza, Christodoulos Stefanadis, Pavlos Toutouzas
J Am Coll Cardiol (abst. suppl.) : (Suppl. A) : 39 (5), 54A (1150-9), 2002 **IF : 14,086**

9. "*Effects of antihypertensive monotherapy on plasma levels of pituitary adenylate cyclase activating polypeptide-38 in hypertensive subjects*"
J. Elefsiniotis, C. Tsioufis, **E. Economou**, M. Toutouza, C. Stefanadis, P. Toutouzas
Eur Heart J (abstract suppl.) : 23, 728 (P3713), 2002 **IF : 14,097**

10. "*Coincidental fluctuation of circulating amyloid beta 42 and tissue inhibitor of metalloproteinases 1 after a severe acute myocardial infarction*"
E.V. Economou, C.E. Pitsavos, A. Masourou, A. Zervoudaki, K.I. Kapetanios, M.G. Toutouza, C.I. Stefanadis, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 23, 718 (P3674), 2002 **IF : 14,097**

11. "*Circulating matrix metalloproteinase 2 and tissue inhibitor of metalloproteinases 1 after severe and minor acute myocardial infarction*"
E.V. Economou, C.P. Pitsavos, A. Masourou, A. Zervoudaki, M.G. Toutouza, K.I. Kapetanios, C.I. Stefanadis, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 23, 469 (P2383), 2002 **IF : 14,097**

12. "*Clinical significance of matrix metalloproteinases activity in acute myocardial infarction patients*"
D. Papadopoulos, K.I. Kapetanios, **E.V. Economou**, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 23, 469 (P2382), 2002 **IF : 14,097**

13. "*Circulating vasoactive amyloid beta ptotein ending at 42 amino acid is elevated in severe acute myocardial infarction*"

E.V. Economou, C.P. Pitsavos, A. Masourou, A. Zervoudaki, M.G. Toutouza, K.I. Kapetanios, C.I. Stefanadis, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 23, 228 (P1231), 2002 **IF : 14,097**

14. "*Matrix metalloproteinases activity in acute myocardial infarction patients. Do their activity contributed to post-myocardial infarction dilation?*"
D. Papadopoulos, **E.V. Economou**, K.I. Kapetanios, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 23, 225 (P1218), 2002 **IF : 14,097**

15. "*Stent-percutaneous transluminal coronary angioplasty triggers an antigen-independent cellular immune response*"
E.V. Economou, C.E. Pitsavos, A. Katinioti, K. Tedolouris, M.G. Toutouza, C.I. Stefanadis, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 22, 283 (P1577), 2001 **IF : 14,097**

16. "*Time coincidence in alterations of circulating amyloid A and matrix metalloproteinases 2 and 9 may increase amyloid A amyloidogenic potential after a severe acute myocardial infarction*"
E.V. Economou, C.E. Pitsavos, A. Masourou, K.I. Kapetanios, A. Zervoudaki, M.G. Toutouza, C.I. Stefanadis, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 22, 526 (P2859), 2001 **IF : 14,097**

17. "*Heart fatty acid binding protein : the advent of unique marker for the early detection of acute myocardial infarction patients*"
K.I. Kapetanios, C.E. Pitsavos, K. Vasiliadou, M.G. Toutouza, **E.V. Economou**, C.I. Stefanadis, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 22, 608 (P3323), 2001 **IF : 14,097**

18. "*Increased levels of macrophage colony stimulating factor and C-reactive protein predict worse prognosis in patients with chronic stable angina. A six-year follow-up study*"
I. Ikonomidis, D. Tsiapras, F. Andreotti, C. Pitsavos, C. Stefanadis, **E. Economou**, P. Toutouzas, P. Nihoyannopoulos
Eur Heart J (abstract suppl.) : 22, 373 (P2009), 2001 **IF : 14,097**
Circulation : 104(17), II-711, No 3354, 2001 **IF : 15,202**

19. "*Effect of angiotensin converting enzyme inhibitor on collagenolytic enzymes activity in patients with acute myocardial infarction*"
D.P. Papadopoulos, **E.V. Economou**, K.I. Kapetanios, P.K. Toutouzas
Eur Heart J (abstract suppl.) : 22, 288 (P1600), 2001 **IF : 14,097**

20. "*Dissociation between the clustering of newer risk factors and estrogen-induced improvement in aortic elasticity in hypertensive postmenopausal women*"
K.Tsioufis, Tzioumis, C. Stefanadis, M. Toutouza, I. Kallikazaros, I. Elefsiniotis, **E. Ekonomou,** C. Pitsavos, E. Tsiamis, P. Toutouzas
Eur Heart J (abstract suppl.) : 22, 97 (P584), 2001 **IF : 14,097**

21. "*The effect of antihypertensive monotherapy on plasma levels of pituitary adenylate cyclase activating polypeptide-38 in patients with mild to moderate essential hypertension*"
J. Elefsiniotis, **E. Economou**, C. Tsioufis, D. Panagiotakos, M. Toutouza, G. Vyssoulis, P. Toutouzas
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Urinary thiobarbituric acid-reacting substances as potential biomarkers of intrauterine hypoxia
ARCH PEDIATR ADOLESC MED, 155 : 718-722, 2001

14.1.6.5. ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΩΝ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΔΙΕΘΝΩΝ ΣΥΝΕΔΡΙΩΝ (Proceedings ή Abstract Books)

Συνολικός Αριθμός : **28**

ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ

1. **Κλινική Χημεία – Φαρμακευτική Βιοχημεία** : **3**
2. **Μαιευτική Γυναικολογία** : **12**
3. **Παιδιατρική - Νεογνολογία** : **9**
4. **Καρδιολογία – Αγγειολογία** : **4**

ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ

1ος Συγγραφέας ή μόνος σε : **3**
2ος Συγγραφέας σε : **5**
Τελευταίος Συγγραφέας σε : **2**
Άλλη Θέση : **15**

14.1.6.6. ΚΑΤΑΛΟΓΟΣ ΠΕΡΙΛΗΨΕΩΝ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΩΝ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΔΙΕΘΝΩΝ ΣΥΝΕΔΡΙΩΝ (Proceedings ή Abstract Books)

ΚΛΙΝΙΚΗ ΧΗΜΕΙΑ – ΦΑΡΜΑΚΕΥΤΙΚΗ ΒΙΟΧΗΜΕΙΑ

1. “A comparison of five curve-fitting procedures in testosterone radioimmunoassay”

E.V. Economou, Th. Siatra-Papastaikoudi, A. Foundos
Abstracts of the **53rd World Congress of Pharmacy and Pharmaceutical Sciences of the International Pharmaceutical Federation** (published in a special supplement) : No P7-89, 82, 1993

2. "*Radioimmunoassay methodology for the estimation of diethylstilbestrol (DES) in serum of patients with advanced prostatic cancer*"
E. V. Economou, G. P. Evangelatos, T. Siatra-Papastaikoudi, D. S. Ithakissios
Abstracts of the **51st Pharmaceutical World Congress of the International Pharmaceutical Federation** (published in a special supplement): No 4p-sda-019,1991

3. "*Direct radioimmunoassay for diethylstilbestrol (DES) in serum of patients with prostatic cancer*"
E. V. Economou, S. E. Kakabakos, G. P. Evangelatos, D. S. Ithakissios
Abstracts of the **50th Pharmaceutical World Congress of the International Pharmaceutical Federation** (published in a special supplement) : No 14, CS-65,1990

ΜΑΙΕΥΤΙΚΗ - ΓΥΝΑΙΚΟΛΟΓΙΑ

1. "Improvement of endothelial function in 46,XY DSD patients after 6 months of hormone therapy"
Tsimaris Pandelis, Deligeoroglou Efthimios, Athanasopoulos Nikolaos, **Economou Emmanuel**, Stamatelopoulos Kimon, Rizos Demetrios, Papamichael Christos, Lambrinoudaki Irene, Mastorakos George,Creatsas George
The 9th Athens Congress on Women's Health and Disease, "From Puberty to Menopause"
Athens Hilton, August 28-30, 2014

2. "The effect of ERS1-XBAL polymorphism on bone density of adolescent girls with anorexia nervosa"
Evgenia Stergioti, Efthimios Deligeoroglou, Konstantinos D. Dimopoulos, Vasileios Karountzos, Artemis Tsitsika, **Emmanouil Economou**, George Creatsas
The 9th Athens Congress on Women's Health and Disease, "From Puberty to Menopause"
Athens Hilton, August 28-30, 2014

3. "Genetic heterogeneity of Pecam-1 and P-Selectin genes are associated with in vitro fertilization – embryotransfer failure"
Vlachadis N, Vrachnis N, Kouskouni E, Vitoratos N, **Economou E.**
The 9th Athens Congress on Women's Health and Disease, "From Puberty to Menopause"
Athens Hilton, August 28-30, 2014

4. "IVF-ET Failure is associated with Genetic Heterogeneity of Platelet Glycoproteins Ia and IIIa"
Vlachadis N (Speaker), Tsamadias V, Kouskouni E, Vitoratos N, Creatsas G, Economou E
10th Congress of European Society of Gynecology, Bruxelles, September 18-21, 2013

5. "Genetic heterogeneity of platelets' glycoprotein receptors Ia and IIIa is associated with platelet function in women with recurrent miscarriages"
Tsamadias V, Vlachadis N (Speaker), Kouskouni E, Creatsas G, Economou E
10th Congress of European Society of Gynecology, Bruxelles, September 18-21, 2013

6. "IVF – Failure is associated with genetic heterogeneity of platelet glycoproteins and cell adhesion molecules"
Tsamadias Vasilios M.Sc., Vlachadis Nikolaos M.D., Papakonstantinou Emmanuel M.Sc., Kouskouni Evangelia M.D., Ph.D, Economou Emanuel, Ph.D
8th Congress of Women's Health & Disease, Kos, September 1 – 3, 2011, Greece

7. "Increased risk for spontaneous miscarriages in women with genetic heterogeneity of platelet glycoproteins and cell adhesion molecules"
Tsamadias Vasilios M.Sc., Vlachadis Nikolaos M.D., Papakonstantinou Emmanuel M.Sc.,

Kouskouni Evangelia M.D., Ph.D, Economou Emanuel, Ph.D
8th Congress of Women's Health & Disease, Kos, September 1 – 3, 2011, Greece

8. "Endogenous sex hormones and risk factors for atherosclerosis in healthy greek postmenopousal women"
Vlachou S., Lambrinoudaki I., Christodoulakos G., **Economou E.**, Argeitis J., Creatsas M., Kouskouni E., Rizos D
Abstract Book of the XVI Meeting of Balkan Clinical Laboratory Federation anf 7th Hellenic Congress of Clinical Chemistry, p. 102, 16 – 18 October, 2008, Athens, Greece

9. *"Does electromagnetic radiation from cell phones influence sperm motility in humans"*
D. Tzanakaki, S. Baka, **E. Economou**, D. Hassiakos, S. Konidaris.
Abstract Book of the the 7th Athens Congress on Women's Health and Diasease, p. 217, 11 – 13 September, 2008, Athens, Greece

10. *"Measurable serum markers of oxidative stress response in women with endometriosis"*
A. Augoulea, Lambrinoudaki I., **E. Economou**, G. Kaparos, A. Kontoravdis, C. Papadias, G. Cristodoulakos, G. Creatsas.
Abstract Book of the the 7th Athens Congress on Women's Health and Diasease, p. 136 – 137, 11 – 13 September, 2008, Athens, Greece

11. "E-cadherin expression in cervical epithelial cells of postmenopausal women: association with hormone therapy, tibolone and raloxifene"
V. Sioulas, S. Vlachou, G. Chrostodoulakos, I. Lambrinoudaki, E. Politi, **E. Economou**, T. Sergentanis, C. Panoulis, A. Augoulea, A. Alexandrou, G. Creatsas.
Abstract Book of the 12th World Congress on the Menopause, May 19 – 23, 2008, Madrid, Spain

12. "*Effect of hormone therapy and tibolone on plasma index of atherosclerosis"*
G. Christodoulakos, I. Lambrinoudaki, **E. Economou**, C. Panoulis, E. Kouskouni, A. Augoulea, G. Creatsas
Abstract Book of **The 6th Athens Congrass on Women's Health and Disease**, abst. No 11, p. 65, 2005

ΠΑΙΔΙΑΤΡΙΚΗ - ΝΕΟΓΝΟΛΟΓΙΑ

1. *"Perinatal changes of neurotrophins in intrauterine growth restricted and appropriate for gestational age term offspring and their mothers"*
K.E. Nikolaou, A. Malamitsi-Puchner, **E. Economou**, M. Boutsikou, T. Boutsikou, K.P. Puchner, M. Kyriakakou, D. Hassiakos
Abstract Book of the International Congress of the European Academy of Paediatrics, October 7-10, Barcelona, Spain, 2006

2. *"Circulating neurotrophin levels in the perinatal period of intrauterine growth restricted fetuses and neonates at term***"**
Malamitsi-Puchner A., Nikolaou KE, **Economou E**, Boutsikou M, Boutsikou T, Kyriakakou M, Puchner KP, Hassiakos D.
Abstract Book of the 32nd Annual Meeting of the Swiss, Austrian and German Society of Neonatalogy and Pediatric Intensive Medicine, May 18-20, Vienna, Austria, 2006

3. *"The impact of intrauterine growth restriction on circulating levels of angiogenic factors and their possible role as catch-up growth predictors"*
A. Malamitsi-Puchner, T. Boutsikou, E. **Economou, E**. Makrakis, K.E. Nikol;aoy, D. Hasiakos
Abstract Book of the **13th European Workshop on Neonatology**, abst. No 26, p.339, 2005

4. "*Endothelin (ET) 1-21 plasma concentration in children and adolescents with IDDM as marker of endothelial dysfunction*"
A. Malamitsi-Puchner, **E. Economou**, F. Karachaliou, D. Delis, K. Kassiou, C. S. Bartsocas
Abstracts of the **20th Annual Meeting of the International Society for Pediatric and Adolescent Diabetes** (published in a special supplement) : No 0-1 (A6), 1994

5. "*Lipid peroxide plasma levels in normal fullterm and premature neonates*"
A. Malamitsi-Puchner, S. Sevastiadou, T. Efstathopoulos, **E. Economou**, Z. Hadzistamatiou, D. Nicolopoulos
Abstracts of the **14th European Congress of Perinatal Medicine** (published in a special supplement): No 122, 1994

6. "*Endothelin 1-21 plasma levels in fetuses of 18 - 24 weeks*"
A. Malamitsi-Puchner, A. Antsaklis, **E. Economou**, N. Papantoniou, S. Mesogitis, N. Koutra, D. Aravantinos
Abstracts of the **14th European Congress of Perinatal Medicine** (published in a special supplement): No 122, 1994

7. "*Endothelin-1,2 levels in the first and fourth day postpartum in normal fullterm and premature neonates*"
A. Malamitsi-Puchner, **E. V. Economou**, S. Sevastiadou, T. Efstathopoulos, D. Nicolopoulos
Abstracts (published in a special supplement): 153, 1993
Proceedings of communications and posters of the **2nd World Congress of Perinatal Medicine** (published in a special supplement): I, 629-633, 1993

8. "*Evidence that vitamin E may reduce the levels of prostacyclin and thromboxane in the blood of premature neonates*"
Ariadne Malamitsi-Puchner, **Emmanuel Economou**, Eugenia Papathoma, Costas Papas
Abstracts of the **International Symposium on Progress and Controversies in Neonatal / Perinatal Medicine** are published in special supplement.
This paper received the first prize at the "International Symposium on Progress and Controversies in Neonatal / Perinatal Medicine".
"*Influence of vitamin E on the levels of prostacycline and thromboxane in the blood of premature neonates*"
Ariadne Malamitsi-Puchner, **Emmanuel Economou**, Eugenia Papathoma, Costas Papas
Abstracts of the **VI International Berlin Symposium "Research in Perinatal Medicine"** (published in a special supplement): No 17, 1991

ΚΑΡΔΙΟΛΟΓΙΑ - ΑΓΓΕΙΟΛΟΓΙΑ

1. "*Regulation of collagen degradation in patients with acute myocardial infarction. Preliminary data*"
D.P. Papadopoulos, P.D. Papadopoulos, **E.V. Economou**, D.M. Farmakis, M.G. Toutouza, K.I. Kapetanios, P.K. Toutouzas
Abstract Book of the **1st International Congress on Coronary Artery Disease- From Prevention to Intervention**, 127, 32, 1997

2. "*The role of tumor necrosis factor-a and -b on heart failure patients*"
P.D. Papadopoulos, D.M. Farmakis, **E.V. Economou**, M.G. Toutouza, K.I. Kapetanios, D.P. Papadopoulos, P.K. Toutouzas.
Abstract Book of the **9th Annual Meeting of Mediterranean Association of Cardiology and Cardiac Surgery**, 32(10), 947, 1996

3. "*Activated transforming growth factor β2 is depressed and basic fibroblast growth factor is increased in heart failure patients*"
D. Farmakis, P. Papadopoulos, **E. Economou**, M. Toutouza, T. Argyriou, K.

Kapetanios, I. Bosinakou, D. Papadopoulos, P. Toutouzas
Abstract Book of the **8th Annual Meeting of Mediterranean Association of Cardiology and Cardiac Surgery**, 89, 1995

4. "*Serum endothelin changes with calcium antagonist antihypertensive treatment*" Vyssoulis G., **Economou E.**, Giannakopoulou K., Karpanou E., Toutouzas P. Abstracts of the 7th Annual Meeting of the Mediterranean Association of Cardiology and Cardiac Surgery (published in a special supplement): P 078,.200, 1994

14.2. ΣΥΝΟΠΤΙΚΗ ΠΑΡΟΥΣΙΑΣΗ ΞΕΝΟΓΛΩΣΣΩΝ ΔΗΜΟΣΙΕΥΣΕΩΝ

Α. ΞΕΝΟΓΛΩΣΣΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΠΕΡΙΟΔΙΚΑ

ΣΥΝΟΛΟ SCI	:	**66**
ΣΥΝΟΛΟ ΕΚΤΟΣ SCI	:	**1**

ΣΥΝΤΕΛΕΣΤΗΣ ΑΠΗΧΗΣΗΣ (IMPACT FACTOR, I.F.) (2013)

Συνολικός	:	**295,54**
Συνολικός (εκτός Letters to the Editor)	:	**166,38**
Μέσος Ορος Συνολικού	:	**4,41**
Προσωπικός	:	**190,25**
Μέσος Ορος Προσωπικού	:	**2,84**

ΒΙΒΛΙΟΓΡΑΦΙΚΕΣ ΑΝΑΦΟΡΕΣ

Συνολικός Αριθμός	:	**1692**
Συνολικός Αριθμός (εκτός αυτοαναφορών)	:	**22**
Προσωπικός Συνολικός Αριθμός	:	**594,90**
Προσωπικός Συνολικός Αριθμός (εκτός αυτοαναφορών)	:	**584,75**

Β. ΞΕΝΟΓΛΩΣΣΕΣ ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΔΙΕΘΝΗ ΠΕΡΙΟΔΙΚΑ

Συνολικός Αριθμός	:	**102**

ΣΥΝΤΕΛΕΣΤΗΣ ΑΠΗΧΗΣΗΣ (IMPACT FACTOR, I.F.) (2013)

Συνολικός	:	**1312.158**
Μέσος Ορος Συνολικού	:	**15.22**
Προσωπικός	:	**746.6**
Μέσος Ορος Προσωπικού	:	**7.32**

ΒΙΒΛΙΟΓΡΑΦΙΚΕΣ ΑΝΑΦΟΡΕΣ

Συνολικός Αριθμός	:	**10**
Συνολικός Αριθμός (εκτός αυτοαναφορών)	:	**9**
Προσωπικός Συνολικός Αριθμός	:	**5.58**
Προσωπικός Συνολικός Αριθμός (εκτός αυτοαναφορών)	:	**4.58**

14.3. ΕΛΛΗΝΙΚΕΣ ΔΗΜΟΣΙΕΥΣΕΙΣ

1.	ΔΗΜΟΣΙΕΥΜΕΝΗΣ	:	14
2.	ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ	:	0
	ΣΥΝΟΛΟ	:	14
	ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1.	Κλινική Χημεία – Φαρμακευτική Βιοχημεία	:	7
2.	Καρδιολογία – Αγγειολογία	:	7
	ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ		
1.	ΑΝΑΣΚΟΠΗΣΕΙΣ	:	4
2.	ΚΛΙΝΙΚΟΕΡΓΑΣΤΗΡΙΑΚΕΣ ΜΕΛΕΤΕΣ	:	8
3.	ΑΝΑΠΤΥΞΗ ΕΡΓΑΣΤΗΡΙΑΚΗΣ ΤΕΧΝΟΛΟΓΙΑΣ	:	2
	ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
$1^{ος}$ Συγγραφέας ή μόνος σε		:	5
$2^{ος}$ Συγγραφέας σε		:	5
Τελευταίος Συγγραφέας σε		:	0
Άλλη Θέση		:	4

14.3.1. ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ

1.	ΔΗΜΟΣΙΕΥΜΕΝΗΣ	:	8
2.	ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ	:	0
	ΣΥΝΟΛΟ	:	8
	ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1.	Κλινική Χημεία – Φαρμακευτική Βιοχημεία	:	1
2.	Καρδιολογία – Αγγειολογία	:	7
	ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ		
1.	ΑΝΑΣΚΟΠΗΣΕΙΣ	:	4
2.	ΚΛΙΝΙΚΟΕΡΓΑΣΤΗΡΙΑΚΕΣ ΜΕΛΕΤΕΣ	:	4
	ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
$1^{ος}$ Συγγραφέας ή μόνος σε		:	2
$2^{ος}$ Συγγραφέας σε		:	4
Τελευταίος Συγγραφέας σε		:	0
Άλλη Θέση		:	2

14.3.2. ΚΑΤΑΛΟΓΟΣ ΔΗΜΟΣΙΕΥΣΕΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ

1. *ΙΩΑΝΝΗΣ Σ. ΕΛΕΥΣΙΝΙΩΤΗΣ,* ***ΕΜΜΑΝΟΥΗΛ Β. ΟΙΚΟΝΟΜΟΥ****, ΧΡΗΣΤΟΣ Η. ΠΙΤΣΑΒΟΣ.*
Νευροπροστασία αντιυπερτασικής αγωγής.
Καρδιά και Αγγεία *:Μάϊος-Ιούνιος, V(3), 259-262, 2000*

Θεματολογία	:	*Καρδιολογία*
Μεθοδολογία	:	*Παθοφυσιολογία*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

2. *ΚΩΝΣΤΑΝΤΙΝΟΣ Ι. ΚΑΠΕΤΑΝΙΟΣ,* ***ΕΜΜΑΝΟΥΗΛ Β. ΟΙΚΟΝΟΜΟΥ****, ΧΡΗΣΤΟΣ Η. ΠΙΤΣΑΒΟΣ.*
Η αγγειογένεση στην αθηροσκλήρυνση : ηθικός ή φυσικός αυτουργός ; (Μέρος Β).
Καρδιά και Αγγεία *: Σεπτέμβριος-Οκτώβριος, V(5),436-447, 2000*

Θεματολογία	:	*Καρδιολογία*
Μεθοδολογία	:	*Παθοφυσιολογία*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

3. *ΚΩΝΣΤΑΝΤΙΝΟΣ Ι. ΚΑΠΕΤΑΝΙΟΣ,* ***ΕΜΜΑΝΟΥΗΛ Β. ΟΙΚΟΝΟΜΟΥ****, ΧΡΗΣΤΟΣ Η. ΠΙΤΣΑΒΟΣ.*
Η αγγειογένεση στην αθηροσκλήρυνση : ηθικός ή φυσικός αυτουργός ; (Μέρος Α).
Καρδιά και Αγγεία *: Ιούλιος-Αύγουστος, V(4), 342-351, 2000*

Θεματολογία	:	*Καρδιολογία*
Μεθοδολογία	:	*Παθοφυσιλογία*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

4. *ΚΩΝΣΤΑΝΤΙΝΟΣ ΚΑΠΕΤΑΝΙΟΣ, ΧΡΙΣΤΟΔΟΥΛΟΣ ΣΤΕΦΑΝΑΔΗΣ, ΧΡΗΣΤΟΣ ΠΙΤΣΑΒΟΣ,* ***ΕΜΜΑΝΟΥΗΛ ΟΙΚΟΝΟΜΟΥ****,ΔΗΜΟΣΘΕΝΗΣ ΠΑΝΑΓΙΩΤΑΚΟΣ, ΔΗΜΗΤΡΙΟΣ ΦΑΡΜΑΚΗΣ, ΚΑΡΜΕΝ ΒΑΣΙΛΕΙΑΔΟΥ, ΜΑΡΙΝΑ ΤΟΥΤΟΥΖΑ, ΠΑΥΛΟΣ ΤΟΥΤΟΥΖΑΣ.*
Η σημασία της ενεργοποίησης του ελεύθερου ινσουλινοειδούς αυξητικού παράγοντα -1 στη διαδικασία της αναδιαμόρφωσης σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου.
Ελληνική Καρδιολογική Επιθεώρηση *: 41, 64-74, 2000*

Θεματολογία	:	*Καρδιολογία*
Μεθοδολογία	:	*Κλινική Παθολογική Βιοχημεία*
Αριθ. Ατόμων	:	*47*

5. *ΑΙΚΑΤΕΡΙΝΗ Ε. ΓΙΑΝΝΑΚΟΠΟΥΛΟΥ,* ***ΕΜΜΑΝΟΥΗΛ Β. ΟΙΚΟΝΟΜΟΥ****, ΓΡΗΓΟΡΙΟΣ Π. ΒΥΣΣΟΥΛΗΣ, ΜΑΡΙΝΑ Γ. ΤΟΥΤΟΥΖΑ, ΠΑΥΛΟΣ Κ. ΤΟΥΤΟΥΖΑΣ.*
Μεταβολές της λειτουργικής και αιμοστατικής δραστηριότητας του αγγειακού ενδοθηλίου μετά από αντιυπερτασική αγωγή με διυδροπυριδίνες.
Ελληνική Καρδιολογική Επιθεώρηση *: 40, 20-29, 1999*

Θεματολογία	:	*Καρδιολογία*
Μεθοδολογία	:	*Κλινική Φραμακευτικη Βιοχημεία*
Αριθ. Ατόμων	:	*77*

6. ***ΕΜΜΑΝΟΥΗΛ Β. ΟΙΚΟΝΟΜΟΥ****, ΠΑΥΛΟΣ Κ. ΤΟΥΤΟΥΖΑΣ.*
Γονίδια : Ταξίδι κληρονομικότητας και προς την καρδιά.
Καρδιά και Αγγεία *: Σεπτέμβριος- Οκτώβριος, Ι(5), 402-403, 1996*

Θεματολογία	:	*Καρδιολογία*
Μεθοδολογία	:	*Κλινική Γενετική*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

7. *Α.Ε. ΓΙΑΝΝΑΚΟΠΟΥΛΟΥ, Γ.Π. ΒΥΣΣΟΥΛΗΣ,* ***Ε.Β. ΟΙΚΟΝΟΜΟΥ****, Μ.Γ. ΤΟΥΤΟΥΖΑ, Χ.Σ. ΤΣΕΛΙΚΑ, Π.Κ. ΤΟΥΤΟΥΖΑΣ.*
Είδραση αντιυπερτασικής αγωγής με αναστολέα ασβεστίου στα επίπεδα των ενδοθηλινών του πλάσματος.
Αρτηριακή Υπέρταση *: 3, 141-146, 1994*

Θεματολογία	:	*Καρδιολογία*
Μεθοδολογία	:	*Κλινική Φραμακευτικη Βιοχημεία*
Αριθ. Ατόμων	:	*25*

8. ***Ε.Β. ΟΙΚΟΝΟΜΟΥ****, Γ.Π. ΕΥΑΓΓΕΛΑΤΟΣ.*
Φθορισμοανοσοαναλύσεις (FIA). Η εναλλακτική λύση στην ευρεία εφαρμογή των ραδιοανοσοαναλύσεων (RIA).
Επιθεώρηση Κλινικής Φαρμακολογίας και Φαρμακοκινητικής*:60-70 και 115-122, 1987*

Θεματολογία	:	*Κλινική Χημεία - Βιοχημεία*
Μεθοδολογία	:	*Κλινική Αναλυτική Χημεία*
Αριθ. Ατόμων	:	*(Ανασκόπηση Μεθοδολογιών)*

14.3.3. ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΕΛΛΗΝΙΚΩΝ ΣΥΝΕΔΡΙΩΝ

1.	**ΔΗΜΟΣΙΕΥΜΕΝΗΣ**	:	**6**
2.	**ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ**	:	**0**
	ΣΥΝΟΛΟ	:	**6**

ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ

1.	**Κλινική Χημεία – Φαρμακευτική Βιοχημεία**	:	**6**

ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ

1.	**ΑΝΑΠΤΥΞΗ ΕΡΓΑΣΤΗΡΙΑΚΗΣ ΤΕΧΝΟΛΟΓΙΑΣ**	:	**2**
2.	**ΚΛΙΝΙΚΟΕΡΓΑΣΤΗΡΙΑΚΕΣ ΜΕΛΕΤΕΣ**	:	**4**

ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ

1ος Συγγραφέας ή μόνος σε	:	**3**
2ος Συγγραφέας σε	:	**1**
Τελευταίος Συγγραφέας σε	:	**0**
Άλλη Θέση	:	**2**

14.3.4. ΚΑΤΑΛΟΓΟΣ ΠΛΗΡΩΝ ΕΛΛΗΝΙΚΩΝ ΔΗΜΟΣΙΕΥΣΕΩΝ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΕΛΛΗΝΙΚΩΝ ΣΥΝΕΔΡΙΩΝ

ΚΑΤΑΛΟΓΟΣ ΠΛΗΡΩΝ ΕΛΛΗΝΙΚΩΝ ΔΗΜΟΣΙΕΥΣΕΩΝ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΕΛΛΗΝΙΚΩΝ ΣΥΝΕΔΡΙΩΝ

1. *Α. ΜΑΛΑΜΙΤΣΗ-PUCHNER, Α. ΑΝΤΣΑΚΛΗΣ,* ***Ε. ΟΙΚΟΝΟΜΟΥ****, Ν. ΠΑΠΑΝΤΩΝΙΟΥ, Σ. ΜΕΣΟΓΙΤΗΣ, Ν. ΚΟΥΤΡΑ, Σ. ΠΑΠΑΧΑΡΙΤΩΝΟΣ, Γ. ΜΠΙΤΣΙΟΥ , Δ. ΑΡΑΒΑΝΤΙΝΟΣ*
Επίπεδα ενδοθηλίνης (ΕΤ) 1-21 στο πλάσμα φυσιολογικών εμβρύων 18 - 24 εβδομάδων.
Πρακτικά 8ου Πανελληνίου Συνεδρίου Περιγεννητικής Ιατρικής (δημοσιευμένα σε ειδική έκδοση) : No 24, 113-116, 1994

Θεματολογία	:	*Κλινική Βιοχημεία*
Μεθοδολογία	:	*Φαρμακευτική Ραδιοχημεία*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

2. *Α. ΜΑΛΑΜΙΤΣΗ-PUCHNER, Σ. ΣΕΒΑΣΤΙΑΔΟΥ, Θ. ΕΥΣΤΑΘΟΠΟΥΛΟΣ,* ***Ε. ΟΙΚΟΝΟΜΟΥ****, Ζ. ΧΑΤΖΗΣΤΑΜΑΤΙΟΥ, Δ. ΝΙΚΟΛΟΠΟΥΛΟΣ*
Επίπεδα των πολυακόρεστων λιπαρών οξέων στο πλάσμα τελειομήνων και προώρων νεογνών.
Πρακτικά 8ου Πανελληνίου Συνεδρίου Περιγεννητικής Ιατρικής (δημοσιευμένα σε ειδική έκδοση) : No 14, 85-88, 1994

Θεματολογία	:	*Κλινική Βιοχημεία*
Μεθοδολογία	:	*Φαρμακευτική Ραδιοχημεία*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

3. ***Ε.Β. ΟΙΚΟΝΟΜΟΥ****, Σ.Η. ΚΑΚΑΜΠΑΚΟΣ, Γ.Π. ΕΥΑΓΓΕΛΑΤΟΣ, Δ.Σ. ΙΘΑΚΗΣΙΟΣ. Ραδιοανοσοαναλυτικός προσδιορισμός της Διαιθυλοστιλβοιστρόλης απευθείας σε ορό ασθενών με καρκίνο του προστάτη.*
Πρακτικά 5ου Πανελληνίου Φαρμακευτικού Συνεδρίου *(δημοσιευμένα σε ειδική έκδοση) : 57 - 62, 1990*

Θεματολογία	:	*Κλινική Βιοχημεία*
Μεθοδολογία	:	*Φαρμακευτική Ραδιοχημεία*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

4. ***Ε.Β. ΟΙΚΟΝΟΜΟΥ****, Γ.Π. ΕΥΑΓΓΕΛΑΤΟΣ, Θ. ΣΙΑΤΡΑ-ΠΑΠΑΣΤΑΪΚΟΥΔΗ, Δ.Σ. ΙΘΑΚΗΣΙΟΣ Ραδιοανοσοαναλυτικός προσδιορισμός της Διαιθυλοστιλβοιστρόλης (DES) σε βιολογικά υγρά και σε ιστικά εκχυλίσματα. Υπάρχουσες Μέθοδοι- Προβλήματα-Προσανατολισμοί.*
Πρακτικά 4ου Πανελληνίου Φαρμακευτικού Συνεδρίου *(δημοσιευμένα σε ειδική έκδοση) : 730-735, 1988*

Θεματολογία	:	*Κλινική Βιοχημεία*
Μεθοδολογία	:	*Φαρμακευτική Ραδιοχημεία*
Αριθ. Ατόμων	:	*(Ανασκόπηση)*

5. *Σ.Η. ΚΑΚΑΜΠΑΚΟΣ,* ***Ε.Β. ΟΙΚΟΝΟΜΟΥ****, Ε. ΛΙΒΑΝΙΟΥ, Κ. ΣΩΤΗΡΙΑΔΗΣ-ΒΛΑΧΟΣ, Γ.Π. ΕΥΑΓΓΕΛΑΤΟΣ. Συγκριτική μελέτη των μεθόδων χλωραμίνης-Τ, γαλακτοϋπεροξειδάσης και ιωδογόνου για την επισήμανση της ανθρώπινης ωχρινοποιητικής ορμόνης με* ^{125}I.
Πρακτικά 3ου Πανελληνίου Φαρμακευτικού Συνεδρίου *(δημοσιευμένα σε ειδική έκδοση) : 508-515, 1986*

Θεματολογία	:	*Κλινική Βιοχημεία*

Μεθοδολογία	:	*Ανάπτυξη Ανοσοναλύσεων*
Αριθ. Ατόμων	:	*(Ανάπτυξη Τεχνολογίας)*

6. ***Ε.Β. ΟΙΚΟΝΟΜΟΥ****, Σ.Η. ΚΑΚΑΜΠΑΚΟΣ, Ε. ΛΙΒΑΝΙΟΥ, Κ. ΣΩΤΗΡΙΑΔΗΣ-ΒΛΑΧΟΣ, Γ.Π. ΕΥΑΓΓΕΛΑΤΟΣ.*
Μελέτη της επίδρασης χρωστικών στο σύστημα ραδιοανοσοανάλυσης για τη μέτρηση της θυροξίνης.
Πρακτικά 3ου Πανελληνίου Φαρμακευτικού Συνεδρίου *(δημοσιευμένα σε ειδική έκδοση) : 500-507, 1986*

Θεματολογία	:	*Κλινική Βιοχημεία*
Μεθοδολογία	:	*Ανάπτυξη Ανοσοναλύσεων*
Αριθ. Ατόμων	:	*(Ανάπτυξη Τεχνολογίας)*

14.3.5. ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΣΥΝΕΔΡΙΑ

1.	ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ	:	21
2.	ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΕΛΛΗΝΙΚΩΝ ΣΥΝΕΔΡΙΩΝ	:	37
	ΣΥΝΟΛΟ	:	58

ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ

1.	Χειρουργική	:	4
2.	Καρδιολογία – Αγγειολογία	:	20
3.	Κλινική Χημεία – Φαρμακευτική Βιοχημεία	:	6
4.	Μαιευτική Γυναικολογία	:	17
5.	Παιδιατρική - Νεογνολογία	:	11

ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ

1.	ΑΝΑΣΚΟΠΗΣΕΙΣ	:	1
2.	ΚΛΙΝΙΚΟΕΡΓΑΣΤΗΡΙΑΚΕΣ ΜΕΛΕΤΕΣ	:	53
3.	ΑΝΑΠΤΥΞΗ ΕΡΓΑΣΤΗΡΙΑΚΗΣ ΤΕΧΝΟΛΟΓΙΑΣ	:	4

ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ

1ος Συγγραφέας ή μόνος σε	:	8
2ος Συγγραφέας σε	:	19
Τελευταίος Συγγραφέας σε	:	3
Άλλη Θέση	:	28

14.3.6. ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ

	:	
ΣΥΝΟΛΟ	:	21

ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ

1.	Χειρουργική	:	2
2.	Καρδιολογία – Αγγειολογία	:	19

ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ

1ος Συγγραφέας ή μόνος σε	:	3
2ος Συγγραφέας σε	:	9
Τελευταίος Συγγραφέας σε	:	0
Άλλη Θέση	:	9

14.3.7. ΚΑΤΑΛΟΓΟΣ ΠΕΡΙΛΗΨΕΩΝ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ

ΧΕΙΡΟΥΡΓΙΚΗ

1. «*Χηλική δέσμευση του σιδήρου για πρόληψη βλαβών ισχαιμίας-επαναιμάτωσης κατά τις μείζονες ηπατεκτομές. Προοπτική τυχαιοποιημένη μελέτη*»
Ν. Αρκαδόπουλος**, Ε.Οικονόμου,** Κ. Θεοδωράκη, Γ. Βασιληκώστας, Κ. Καραπάνος, Μ. Φράγκου, Ι. Κόντης, Ι. Βασιλείου, Δ. Βώρος, Π. Δημακάκος, Β Σμυρνιώτης
Ελληνική Χειρουργική :78 (6Ι) (συμπληρωματικό τεύχος) 111, Νο234, 2006

2. «*Μπορεί η διεγχειρητική ταχεία μέτρηση της παραθορμόνης να βελτιώσει τα αποτελέσματα της παραθυρεοειδεκτομής*»
Μαρίνης Α, **Οικονόμου Ε**., Βασιλικώστας Γ., Σταφυλά Β., Κυριαζή Μ.,Τσιαντούλα Π., Κωνσταντινίδης Χ., Κουσκούνη Ε., Βασιλείου Ι.
Ελληνική Χειρουργική :78 (6Ι) (συμπληρωματικό τεύχος) 173, No 400, 2006

ΚΑΡΔΙΟΛΟΓΙΑ - ΑΓΓΕΙΟΛΟΓΙΑ

1. "*Κλινική σημασία των δεικτών αποδόμησης του κολλαγόνου σε ασθενείς με οξύ έμφραγμα του μυοκάρδιου*"
Δ.Π. Παπαδόπουλος, Κ.Ι. Καπετάνιος, **Ε.Β. Οικονόμου**, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 43 (συμπλήρωμα Β / 2002), No 349,2002

2. "*Νευροπροστατευτική επίδραση αντιυπερτασικής αγωγής*"
Ι.Σ. Ελευσινιώτης, **Ε. Οικονόμου**, Κ. Τσιούφης, Δ. Παναγιωτάκος, Χ. Πίτσαβος, Χ.Στεφανάδης, Π. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 41 (συμπλήρωμα Β / 2000), No 151, 2000

3. *"Η μείωση των επιπέδων της C-αντιδρώσας πρωτεϊνης σε ασθενείς με σταθερή στηθάγχη από τη χορήγηση ασπιρίνης οφείλεται στη μείωση της παραγωγής κυτταροκινών. Μια τυχαιοποιημένη, διπλά τυφλή, ελεγχόμενη από placebo κλινική μελέτη"*
Ι. Οικονομίδης, **Ε. Οικονόμου**, Χ. Πίτσαβος, Χ. Στεφανάδης, Μ. Μαρίνου, Π. Τούτουζας, Π. Νιχογιαννόπουλος
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β24, 110, 1998

4. "*Η διαλυτή μορφή Fas συνδέεται με την ελεύθερη μορφή του ανάλογου της ινσουλίνης αυξητικού παράγοντα τύπου 1 αλλά όχι και με το βασικό αυξητικό παράγοντα των ινοβλαστών στο πλάσμα ασθενών με χρονία καρδιακή ανεπάρκεια τελικών σταδίων*"
Ε. Οικονόμου, Δ. Φαρμάκης, Χ. Στεφανάδης, Χ. Πίτσαβος, Μ. Τούτουζα, Κ. Καπετάνιος, Π. Παπαδόπουλος, Π. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β31, 148, 1998

5. "*Σύγκριση των αυξητικών παραγόντων fIGF-1 και VEGF σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Κ.Ι. Καπετάνιος, Χ.Ι. Στεφανάδης, Χ.Η. Πίτσαβος, **Ε.Β. Οικονόμου**, Δ.Μ.Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β34, 158,1998

6. "*Συσχέτιση των δεικτών αποδόμησης του κολλαγόνου με τη μυοκαρδιακή βλάβη σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Δ.Π. Παπαδόπουλος, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Κ.Ι. Καπετάνιος, Δ.Μ.Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β34, 160,1998

7. "*Συσχέτιση δεικτών κολλαγονολυτικής δραστηριότητας και μυοκαρδιακής βλάβης σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Δ.Π. Παπαδόπουλος, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Κ.Ι. Καπετάνιος, Δ.Μ.Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας

Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β34, 161,1998

8. "*Συσχετίσεις της λεπτίνης σε δυσλιπιδαιμικούς ασθενείς*"
Δ.Μ. Φαρμάκης, Χ.Ε. Πίτσαβος, Ι.Ν. Σκούμας, Χ.Σ. Τσελίκα, Κ.Ι. Καπετάνιος,Μ.Γ. Τούτουζα, **Ε.Β. Οικονόμου**, Δ. Μάρκου, Χ.Ι. Στεφανάδης, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β39, 183,1998

9. "*Χρονικές μεταβολές του αυξημένου βασικού ινωδοβλαστικού αυξητικού παράγοντα σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Κ.Ι. Καπετάνιος, Χ.Ι. Στεφανάδης, Χ.Η. Πίτσαβος, **Ε.Β. Οικονόμου**, Δ.Μ. Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β39, 185,1998

10. "*Η αυξημένη αγγειογενίνη ως δείκτης αγγειογένεσης σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου*"
Κ.Ι. Καπετάνιος, Χ.Ι. Στεφανάδης, Χ.Η. Πίτσαβος, **Ε.Β. Οικονόμου**, Δ.Μ.Φαρμάκης, Μ.Γ. Τούτουζα, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β40, 186,1998

11. "*Επίπεδα αμυλοειδούς Α στον ορό ασθενών με ήπια έως μέτρια υπέρταση, πριν και μετά από αγωγή με ένα αναστολέα των διαύλων ασβεστίου*"
Ε. Οικονόμου, Χ. Πίτσαβος, Χ. Στεφανάδης, Α. Τρίκας, Μ. Τούτουζα, Ι.Ελευσινιώτης, Κ. Τσιούφης, Κ. Καπετάνιος, Π. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β44, 205,1998

12. "*Επίπεδα αντισωμάτων έναντι της φωσφατιδυλοσερίνης στον ορό ασθενών με ήπια έως μέτρια υπέρταση*, πριν και μετά από αγωγή με ένα αναστολέα των διαύλων ασβεστίου"

Ε. Οικονόμου, Χ. Πίτσαβος, Χ. Στεφανάδης,, Μ. Τούτουζα, Α. Τρίκας, Κ.Τσιούφης, Κ. Καπετάνιος, Π. Τούτουζας.
Ελληνική Καρδιολογική Επιθεώρηση : 39 (συμπλήρωμα Β/1998), Β45, 206,1998

13. "*Βιοχημικοί δείκτες αποδόμησης του κολλαγόνου στο οξύ έμφραγμα του μυοκαρδίου*"
Δ.Π. Παπαδόπουλος, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Δ.Μ. Φαρμάκης, Μ.Γ.Τούτουζα, Κ.Ι. Καπετάνιος, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 38 (συμπλήρωμα), 73, Β16, 1997

14. "*Η αύξηση των επιπέδων των κυκλοφορούντων κυτοκινών συσχετίζεται με την έκταση της στεφανιαίας νόσου*"
Ι. Οικονομίδης, **Ε. Οικονόμου**, Χ. Πίτσαβος, Χ. Στεφανάδης, Μ. Μαρίνου, Π.Τούτουζας, Π. Νιχογιαννόπουλος
Ελληνική Καρδιολογική Επιθεώρηση : 38 (συμπλήρωμα), 95, Β21, 1997

15. "*Η μείωση της καθημερινής ισχαιμίας από την ασπιρίνη σε ασθενείς με σταθερή στηθάγχη οφείλεται στη μείωση παραγωγής κυτοκινών και ενεργοποίησης αιμοπεταλίων*"
Ι. Οικονομίδης, **Ε. Οικονόμου**, Χ. Πίτσαβος, Χ. Στεφανάδης, Μ. Μαρίνου, Π.

Τούτουζας, Π. Νιχογιαννόπουλος
Ελληνική Καρδιολογική Επιθεώρηση : 38 (συμπλήρωμα), 131, Β29, 1997

16. "*Τα επίπεδα της υπεροξειδικής Μη-δισμουτάσης του πλάσματος ενέχονται στην προκαλούμενη κατά την άσκηση ισχαιμική προετοιμασία του μυοκαρδίου*"
Α. Μιχαηλίδης, **Μ. Οικονόμου**, Χ. Σεφερλής, Ζ. Ψωμαδάκη, Ι. Νικαλέξης, Γ.Κατσιμακλής, Ι. Αντωνίου, Π. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 38 (συμπλήρωμα), 264, Β54, 1997

17. "*Ο ενεργοποιημένος transforming growth factor β2 ελλατώνεται και ο basic fibroblast growth factor αυξάνει σε ασθενείς με καρδιακή ανεπάρκεια*"
Δ.Μ. Φαρμάκης, Π.Δ. Παπαδόπουλος, **Ε.Β. Οικονόμου**, Μ.Γ. Τούτουζα, Θ.Α.Αργυρίου, Κ.Ι. Καπετάνιος, Ε. Μποσινάκου, Δ.Π. Παπαδόπουλος, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 36 (συμπλήρωμα), 94, Β21, 1995

18. *Επίδραση αντιυπερτασικής αγωγής με αναστολέα ασβεστίου σε αγγειοδραστικές ουσίες που εκκρίνονται από το αγγειακό ενδοθήλιο και ρυθμίζουν τον αγγειακό τόνο*"
Α.Ε. Γιαννακοπούλου, **Ε.Β. Οικονόμου**, Γ.Π. Βυσσούλης, Ε.Α. Καρπάνου, Μ.Γ.Τούτουζα, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 35Β, 1994

19. "*Ρόλος των ελευθέρων ριζών οξυγόνου στην καταστολή της μυοκαρδιακής λειτουργίας μετά από στεφανιαίες επεμβάσεις*"
Λ. Χατζηνικολάου, Ε.Β. Οικονόμου, Σ. Συμινελάκης, Μ. Κραβαρίτου, Ι. Παπαϊωάννου, Π.Κ. Τούτουζας
Ελληνική Καρδιολογική Επιθεώρηση : 173, Β42, 1993

14.3.8. ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΕΛΛΗΝΙΚΩΝ ΣΥΝΕΔΡΙΩΝ

ΣΥΝΟΛΟ : 37

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1. **Χειρουργική : 2**
2. **Καρδιολογία – Αγγειολογία : 1**
3. **Κλινική Χημεία – Φαρμακευτική Βιοχημεία : 6**
4. **Μαιευτική Γυναικολογία : 17**
5. **Παιδιατρική - Νεογνολογία : 11**

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1ος Συγγραφέας ή μόνος σε : 5
2ος Συγγραφέας σε : 10
Τελευταίος Συγγραφέας σε : 3
Άλλη Θέση : 19

14.3.9. ΚΑΤΑΛΟΓΟΣ ΠΕΡΙΛΗΨΕΩΝ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΕΛΛΗΝΙΚΩΝ ΣΥΝΕΔΡΙΩΝ

ΜΑΙΕΥΤΙΚΗ - ΓΥΝΑΙΚΟΛΟΓΙΑ

1. "Ο συνδυαστικός κίνδυνος αποτυχημένης εμβρυομεταφοράς μετά απο εξωσωματική γονιμοποίηση (*IVF-ET FAILURE)* της γενετικής ετερογένειας των αιμοπεταλιακών γλυκοπρωτεϊνικών υποδοχέων Ια και ΙΙΙΑ "
   Ν. Βλαχάδης, Β. Τσαμαδιάς, Ν. Βραχνής, **Ε. Οικονόμου**
   Ελεύθερη ανακοίνωση στα πλαίσια του 40ου ΕΤΗΣΙΟΥ Πανελληνίου Ιατρικού Συνεδρίου, 14-17 Μαϊου 2014

2. "Η έκφραση των παραγόντων CD40, CD40L και ADAM8 σε γυναίκες με ενδομητρίωνση"
   Σ.Στεργιώτης, Α. Αυγουλέα, Ε. Νιέερη, **Ε. Οικονόμου**, Γ. Καπαρός, Α. Παλαιολόγου, Λ. Αραβαντινός, Κ. Πανουλής, Γ. Κρεατσάς
   Περιλήψεις 6ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 4-5 Απριλίου 2014, Ξενοδοχείο Royal Olympic, Αθήνα

3. "Ο πολυμορφισμός C677T μεταβάλει την επίδραση της ορμονικής θεραπείας υποκατάστασης σε μετεμμηνοπαυσιακές γυναίκες "
   Δ. Παπαδημητρίου, **Ε. Οικονόμου**, Γ. Καπαρός, Δ. Ρίζος, Κ. Πανουλής, Ε. Δεληγεώργλου, Α. Αλεξάνδρου, Α. Αυγουλέα, Μ. Αποστολάκης, Μ. Κρεατσά, Ε. Κουσκούνη, Ε. Λαμπρινουδάκη
   Περιλήψεις 6ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 4-5 Απριλίου 2014, Ξενοδοχείο Royal Olympic, Αθήνα

4. "Η γενετική ετερογένεια του αιμοπεταλιακού γλυκοπρωτεΐνικού υποδοχέα Ια σχετίζεται με την αποτυχία εμβρυομεταφοράς μετά απο εξωσωματική γονιμοποίηση (IVF-ET FAILURE)"
   Ν. Βλαχάδης, Β. Τσαμαδιάς, Ε. Κουσκούνη, **Ε. Οικονόμου**
   Περιλήψεις 39ου ΕΤΗΣΙΟΥ Πανελληνίου Ιατρικού συνεδρίου, Πέμπτη 23 Μαϊου 2013

5. "Η γενετική ετερογένεια των αιμοπεταλιακών γλυκοπρωτεϊνικών υποδοχέων Ια και ΙΙΙα σχετίζεται με την λειτουργικότητα των αιμοπεταλίων σε γυναίκες με αποβολές "

Β. Τσαμαδιάς, Ν. Βλαχάδης, Ε. Κουσκούνη, **Ε. Οικονόμου**
Περιλήψεις 39ου ΕΤΗΣΙΟΥ Πανελληνίου Ιατρικού συνεδρίου, Πέμπτη 23 Μαϊου 2013

6. «Η έκφραση της Ε-καντχερίνης σε τραχηλικά επιθηλιακά κύτταρα μετεμμηνοαπυσιακών γυναικών: συσχέτιση με την ορμονική θεραπεία, την τιβολόνη και την ραλοξιφαίνη»
Β. Σιούλας, Σ. Βλάχου, Α. Αλεξάνδρου, Α. Πολίτη, Ε. Κουτσελίνη, **Ε. Οικονόμου**, Θ. Σεργεντανης, Κ. Πανουλής, Α. Αυγουλέα, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Γ. Κρεατσάς.
Περιλήψεις 4ου Πανελληνίου Συνεδρίου Κλιμακτηρίου και Εμμηνόπαυσης, Μέγαρο Μουσικής Αθηνών, 18-19 Απριλίου 2008

7. «Η έκφραση της Ε-καντχερίνης σε τραχηλικά επιθηλιακά κύτταρα μετεμμηνοαπυσιακών γυναικών: συσχέτιση με την ορμονική θεραπεία, την τιβολόνη και την ραλοξιφαίνη»
Β. Σιούλας, Σ. Βλάχου, Α. Αλεξάνδρου, Α. Πολίτη, Ε. Κουτσελίνη, **Ε. Οικονόμου**, Θ. Σεργεντανης, Κ. Πανουλής, Α. Αυγουλέα, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Γ. Κρεατσάς.
Περιλήψεις 4ου Πανελληνίου Συνεδρίου Κλιμακτηρίου και Εμμηνόπαυσης, Μέγαρο Μουσικής Αθηνών, 18-19 Απριλίου 2008

8. «Επίδραση της ορμονικής θεραπείας και της τιβολόνης στα επίπεδα των λιπιδίων, των λιποπρωτεϊνλων και στον αθηρωματικό δείκτη πλάσματος»
Δ. Παπαδημητρίου, Σ. Βλάχου, Φ. Γκαλάπη, **Ε. Οικονόμου**, Κ. Παπαδιάς, Κ.Πανουλής, Ε. Κουσκούνη, Α. Αλεξάνδρου, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Γ. Κρεατσάς
Περιλήψεις 4ου Πανελληνίου Συνεδρίου Κλιμακτηρίου και Εμμηνόπαυσης, Μέγαρο Μουσικής Αθηνών, 18-19 Απριλίου 2008

9. «Οι αθηρωματογόνες λιποκυτοκίνες και οι δείκτες κυτταρικής απόπτωσης στον ορό επηρεάζονται διαφορετικά από τη θεραπεία σε υγιείς μετεμμηνπαυσιακές γυναίκες»
Βλάχου Σ., Χριστοδουλάκος Γ., Λαμπρινουδάκη **Ε., Οικονόμου** Ε., Καλλίγερου Ε., Γκαλάπη Φ., Κρεατσά Μ., Σιάσου Ζ., Πανουλής Κ., Παπαδιάς Κ.
Περιλήψεις 4ου Ετησίου Συνεδρίου Ιατρικής Σχολής, Α168, 300, 2007

10. "Λεπτίνη-Γκρελίνη: Η διαφορετική επίδραση που τους ασκούν η ορμονική θεραπεία και η ραλοξιφαίνη"
Βλάχου Σ., Λαμπρινουδάκη Ε., Χριστοδουλάκος Γ., **Οικονόμου Ε.**, Πανουλής Κ., Καλλίγερου Ε., Γκαλάπη Φ., Αλεξάνδρου Α., Κουσκούνη Ε., Κρεατσάς Γ.
Περιλήψεις 4ου Ετησίου Συνεδρίου Ιατρικής Σχολής, Α169, 301, 2007

11. «Οι αθηρωματογόνες λιποκυτοκίνες και οι δείκτες κυτταρικής απόπτωσης στον ορό επηρεάζονται διαφορετικά από τη θεραπεία σε υγιείς μετεμμηνοπαυσιακές γυναίκες»
Βλάχου Σ., Χριστοδουλάκος Γ., Λαμπρινουδάκη Ε., **Οικονόμου Ε.**, Καλλίγερου Ε.,Γκαλάπη Φ., Κρεατσά Μ., Σιάσου Ζ., Πανουλής Κ., Παπαδιάς Κ.
Περιλήψεις 4ου Ετησίου Συνεδρίου Ιατρικής Σχολής, Α168, 300, 2007

12. "Ενδογενείς ορμόνες του φύλου και παράγοντες κινδύνου για αθηρωσκληρυνση σε υγιείς μετεμμηνοπαυσιακές γυναίκες"
Β. Μπουρνιά, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Δ. Ρίζος, **Ε. Οικονόμου**, Ι. Αργείτης, Σ. Βλάχου, Μ. Κρεατσά, Ε. Κουσκούνη, Δ. Μπότσης
Περιλήψεις 3ου Ετησίου Επιστημονικού Συνεδρίου Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, Αθήνα, 2006

13. "Επίδραση της ορμονικής θεραπείας, της τιβολόνης και της ραλοξιφαίνης στην απόπτωση και στις λιποκυτοκίνες ρεζιστίνη και αδιπονεκτίνη του ορού"
Ζ. Σιάσου, Ε. Λαμπρινουδάκη, **Ε. Οικονόμου**, Ε. Κουσκούνη, Κ. Πανουλής, Σ. Λάππα, Σ. Βλάχου, Μ. Κρεατσά, Ε. Καλλιγέρου, Γ. Χριστοδουλάκος
Περιλήψεις 3ου Ετησίου Επιστημονικού Συνεδρίου Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, Αθήνα, 2006

14. "Η δράση της ορμονικής θεραπείας και της ραλοξιφαίνης στις μεταλλοπρωτεϊνάσες 2 και 2 του ορού μετεμμηνοπαυσιακών γυναικών»
Πανουλής Κ, Χριστοδουλάκος Γ, Λαμπρινουδάκη Ε, **Οικονόμου Ε**, Αυγουλέα Α., Κρεατσά Μ., Μπότσης Δ., Δενδρινός Σ.
Περιλήψεις 3ου Πανελληνίου Συνεδρίου Κλιμακτηρίου και Εμμηνόπαυσης : σ. 36, 2005

15. "Η δράση της ορμονικής θεραπείας και της ραλοξιφαίνης στη VE-καντχερίνη του ορού μετεμμηνοπαυσιακών γυναικών»
Κρεατσά Μ., Χριστοδουλάκος Γ., Λαμπρινουδάκη Ε., **Οικονόμου Ε.**, Παπαδιάς Κ., Πανουλής Κ., Αυγουλέα Α., Κρεατσάς Γ.
Περιλήψεις 3ου Πανελληνίου Συνεδρίου Κλιμακτηρίου και Εμμηνόπαυσης : σ. 35, 2005

16. "Η επίδραση της χορήγησης ορμονικής αντισύλληψης στον οστικό μεταβολισμό θηλέων πειραματοζώων αναπαραγωγικής ηλικίας – Πειραματική Μελέτη"
Μ. Ελευθεριάδης, Ε. Λαμπρινουδάκη, Γ. Χριστοδουλάκος, Ο. Γρηγορίου, Ε. **Οικονόμου, Ε**. Αντωνίου, Ε. Κουσκούνη, Δ. Περρέα, Ι. Δοντά, Π. Ράπτου, Γ. Λυρίτης, Γ. Κρεατσάς
Περιλήψεις 1ου Ετησίου Επιστημονικού Συνεδρίου Ιατρικής Σχολής Πανεπιστημίου Αθηνών : 67, σ. 106-107, 2004

17. "Επίπεδα ενδοθηλινών (ΕΤ 1-21) πλάσματος πριν και μετά από οιστρογονική θεραπεία υποκατάστασης σε πρωτοπαθώς αμηνορροϊκές έφηβες"
Γ. Κρεατσάς, Α. Μαλαμίτση-Puchner, Ε. Χασάν, **Ε. Οικονόμου**, Δ. Αραβαντινός
Περιλήψεις 7ου Πανελληνίου Συνεδρίου Μαιευτικής και Γυναικολογίας : 41, 1997

ΧΕΙΡΟΥΡΓΙΚΗ

1. "Αντιμετώπιση οξειδωτικών βλαβών κατά τις μείζονες ηπατεκτομές με χρήση δεσφεροξαμίνης. Προοπτική τυχαιοποιημένη μελέτη"
Ν. Αρκαδόπουλος, **Ε. Οικονόμου**, Κ. Θεοδωράκη, Γ. Βασιληκώστας, Κ. Καραπάνος, Α. Παφίτη, Ε. Κουσκούνη, Ι. Βασιλείου, Δ. Βώρος, Β. Σμυρνιώτης
Περιλήψεις 10ου Πανελληνίου Ηπατολογικού Συνεδρίου, Νο21, 2007

2. "Προστασία από τις βλάβες ισχαιμίας/επαναιμάτωσης κατά τις μείζονες ηπατεκτομές με χρήση δεσφεροξαμίνης"
Αρκαδόπουλος Ν., **Οικονόμου Ε.**, Θεοδωράκη Κ., Καραπάνος Κ., Βασιληκώστας Γ., Παφίτη Α., Κουσκούνη Ε., Βώρος Δ., Βασιλείου Ι., Σμυρνιώτης Β.
Περιλήψεις 4ου Ετησίου Συνεδρίου Ιατρικής Σχολής, ΕΑ-59, 116, 2007

ΠΑΙΔΙΑΤΡΙΚΗ - ΝΕΟΓΝΟΛΟΓΙΑ

1. "Πρωτότυπη, ανεξάρτητη της ομοκυστεϊναιμίας, χρόνα συστηματική φλεγμονώδης αντίδραση χαμηλής εντάσεως σε παχύσαρκα παιδιά προεφηβικής ηλικίας"
Εμμανουήλ Β. Οικονόμου, Αριάδνη Β. Μαλαμίτση-Puchner, Χρήστος Πίτσαβος, Ευαγγελία Κουσκούνη, Ιωάννα Μαγαζιώτου-Ελευσινιώτη, Γεώργιος Κ. Κρεατσάς
Περιλήψεις 3ου Ετησίου Επιστημονικού Συνεδρίου Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, ΑΘΗΝΑ, 2006

2. "Επίπεδα νευροτροφινών σε έμβρυα και νεογνά με ενδομήτρια καθυστέρηση ανάπτυξης"
Κ.Ε. Νικολάου, Α. Μαλαμίτση-Puchner, **Ε. Οικονόμου**, Μ. Μπούτσικου, Θ. Μπούτσικου, Μ. Κυριακάκου, Κ.Ρ. Puchner, Δ. Χασιάκος
Περιλήψεις 3ου Ετησίου Επιστημονικού Συνεδρίου Ιατρικής Σχολής Πανεπιστημίου Αθηνών, 12-13 Μαϊου, Αίγλη Ζαππείου, Αθήνα, 2006

3. "Περιγεννητικές μεταβολές του εγκεφαλικού νευροτροφικού παράγοντα (BDNF) σε πρόωρα και τελειόμηνα νεογνά"
Α. Μαλαμίτση-Πούχνερ, **Ε. Οικονόμου**, Ο. Ρηγοπούλου, Θ. Μπούτσικου
Περιλήψεις 1ου Συνεδρίου Ιατρικής Σχολής Πανεπιστημίου Αθηνών : 60, σ. 101, 2004

4. "Αυξημένα επίπεδα χυμοκινών MCP-1 και RANTES στον ορό παχύσαρκων παιδιών"
Ι. Μαγαζιώτου, Δ. Δαμιανάκη, **Ε. Οικονόμου**, Ι. Ελευσινιώτης, Μ. Τούτουζα, Χ. Στεφανάδης, Π. Τούτουζας
Περιλήψεις 27ου Πανελληνίου Συνεδρίου Ενδοκρινολογίας και Μεταβολισμού : 106, 2000

5. "Αυξημένα επίπεδα ομοκυστεϊνης πλάσματος σε παχύσαρκα παιδιά"
Ι. Μαγαζιώτου, Δ. Δαμιανάκη-Ουρανού, Ι. Ελευσινιώτης, **Ε. Οικονόμου**, Μ. Τούτουζα, Χ. Στεφανάδης, Π. Τούτουζας
Περιλήψεις 27ου Πανελληνίου Συνεδρίου Ενδοκρινολογίας και Μεταβολισμού : 105, 2000

6. "Επίπεδα των πολυακόρεστων λιπαρών οξέων στο πλάσμα τελειομήνων και προώρων νεογνών"
Α. Μαλαμίτση-Puchner, Σ. Σεβαστιάδου, Θ. Ευσταθόπουλος, **Ε. Οικονόμου**, Ζ. Χατζησταματίου, Δ. Νικολόπουλος
Πρακτικά 8ου Πανελληνίου Συνεδρίου Περιγεννητικής Ιατρικής (δημοσιευμένα σε ειδική έκδοση) : No 14, 85-88, 1994

7. "Επίπεδα ενδοθηλίνης (ΕΤ) 1-21 στο πλάσμα φυσιολογικών εμβρύων 18 - 24 εβδομάδων"
Α. Μαλαμίτση-Puchner, Α. Αντσακλής, **Ε. Οικονόμου**, Ν. Παπαντωνίου, Σ. Μεσογίτης, Ν. Κούτρα, Σ. Παπαχαρίτωνος, Γ. Μπίτσιου , Δ. Αραβαντινός
Πρακτικά 8ου Πανελληνίου Συνεδρίου Περιγεννητικής Ιατρικής (δημοσιευμένα σε ειδική έκδοση) : No 24, 113-116, 1994

8. "Επίπεδα ενδοθηλίνης 1-21 σε παιδιά και εφήβους με ινσουλινο-εξαρτώμενο σακχαρώδη διαβήτη (ΙΕΣΔ)"
Αριάδνη Μαλαμίτση-Puchner, **Ε. Οικονόμου**, Φενέλη Καραχάλιου, Δ. Δελής, Κορίνα Κασσιού, Χ.Σ. Μπαρτσόκας
Περιλήψεις 32ου Πανελληνίου Παιδιατρικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) : No 73Α, 1994

9. "Επίπεδα ενδοθηλίνης 1-21 (ΕΤ 1-21) στο πρώτο και τέταρτο 24ωρο της ζωής υγιών και πασχόντων προώρων νεογνών"
Αριάδνη Μαλαμίτση-Puchner, Θ. Ευσταθόπουλος, Ζωή Χατζησταματίου, **Ε. Οικονόμου**, Σοφία Σεβαστιάδου, Δ. Νικολόπουλος
Περιλήψεις 32ου Πανελληνίου Παιδιατρικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) : No 105Α, 1994

10. "Επίπεδα ενδοθηλινών-1,2 το 1ο και 4ο 24ωρο της ζωής σε φυσιολογικά τελειόμηνα

νεογνά"
Αριάδνη Μαλαμίτση-Puchner, **Ε. Οικονόμου**, Σοφία Σεβαστιάδου, Θ. Ευσταθόπουλος, Α. Νικολόπουλος
Περιλήψεις 31ου Πανελληνίου Παιδιατρικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) : No 33, 1993

11. "Επίδραση της βιταμίνης Ε στα επίπεδα της προστακυκλίνης και της θρομβοξάνης στο αίμα προώρων νεογνών"
Αριάδνη Μαλαμίτση-Puchner, **Εμμανουήλ Β. Οικονόμου**, Εγενία Παπαθωμά, Κωνσταντίνος Παπάς
Περιλήψεις 29ου Παιδιατρικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) :
No 52ΒΑ, 1991

ΚΛΙΝΙΚΗ ΧΗΜΕΙΑ – ΦΑΡΜΑΚΕΥΤΙΚΗ ΒΙΟΧΗΜΕΙΑ

1. «Διεγχειρητική μέτρηση παραθορμόνης: ένα εργαστηριακό εργαλείο για την εκτίμηση της επιτυχούς χειρουργικής αντιμετώπισης του υπερπαραθυρεοειδισμού»
Δ. Ρίζος, **Ε. Οικονόμου**, Ε. Κουσκούνη, Α. Μαρίνης, Ι. Βασιλείου
Περιλήψεις 6ου Πανελληνίου Συνεδρίου Κλινικής Χημείας, σελ. 181, ΑΑ097, 2006

2. "Από τη Φαρμακογενετική στη Φαρμακογενωμική : Διέξοδος στην Εξατομίκευση της Φαρμακευτικής Θεραπείας;"
Εμμανουήλ Β. Οικονόμου
Περιλήψεις 5ου Πανελληνίου Συνεδρίου Κλινικής Χημείας : σ. 26-30, 2004

3. "Ραδιοανοσοαναλυτικός προσδιορισμός της Διαιθυλοστιλβοιστρόλης απευθείας σε ορό ασθενών με καρκίνο του προστάτη"
Ε.Β. Οικονόμου, Σ.Η. Κακαμπάκος, Γ.Π. Ευαγγελάτος, Δ.Σ. Ιθακήσιος
Περιλήψεις 5ου Πανελληνίου Φαρμακευτικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) : Σ5, 61, 1990

4. "Ραδιοανοσοαναλυτικός προσδιορισμός της Διαιθυλοστιλβοιστρόλης (DES) σε βιολογικά υγρά και σε ιστικά εκχυλίσματα. Υπάρχουσες Μέθοδοι-Προβλήματα-Προσανατολισμοί"
Ε.Β. Οικονόμου, Γ.Π. Ευαγγελάτος, Θ. Σιάτρα-Παπασταϊκούδη, Δ.Σ. Ιθακήσιος
Περιλήψεις 4ου Πανελληνίου Φαρμακευτικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) : Δ45, 208, 1988

5. "Συγκριτική μελέτη των μεθόδων χλωραμίνης-Τ, γαλακτοϋπεροξειδάσης και ιωδογόνου για την επισήμανση της ανθρώπινης ωχρινοποιητικής ορμόνης με 125Ι"
Σ.Η. Κακαμπάκος**, Ε.Β. Οικονόμου**, Ε. Λιβανίου, Κ. Σωτηριάδης-Βλάχος, Γ.Π. Ευαγγελάτος
Περιλήψεις 3ου Πανελληνίου Φαρμακευτικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) : 120, Κ46, 1986

6. "*Μελέτη της επίδρασης χρωστικών στο σύστημα ραδιοανοσοανάλυσης για τη μέτρηση της θυροξίνης*"
Ε.Β. Οικονόμου, Σ.Η. Κακαμπάκος, Ε. Λιβανίου, Κ. Σωτηριάδης-Βλάχος, Γ.Π. Ευαγγελάτος
Περιλήψεις 3ου Πανελληνίου Φαρμακευτικού Συνεδρίου (δημοσιευμένες σε ειδική έκδοση) : 119, Κ45, 1986

ΚΑΡΔΙΟΛΟΓΙΑ

1. "Επίδραση της αντιυπερτασικής αγωγής με αναστολέα ασβεστίου στην ενδοθηλίνη το ορού"

Α.Ε. Γιαννακοπούλου, **Ε.Β. Οικονόμου**, Γ.Π. Βυσσούλης, Μ.Γ. Τούτουζα, Χ.Σ. Τσελίκα, Π.Κ. Τούτουζας
Περιλήψεις 4ου Πανελληνίου Συνεδρίου Αρτηριακής Υπέρτασης (δημοσιευμένες σε ειδική έκδοση), No 33, 1994

ΣΥΝΟΠΤΙΚΗ ΠΑΡΟΥΣΙΑΣΗ ΕΛΛΗΝΙΚΩΝ ΔΗΜΟΣΙΕΥΣΕΩΝ

ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ

1.	**ΔΗΜΟΣΙΕΥΜΕΝΗΣ**	:	**14**
2.	**ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ**	:	**0**
	ΣΥΝΟΛΟ	:	**14**
	ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1.	**Κλινική Χημεία – Φαρμακευτική Βιοχημεία**	:	**7**
2.	**Καρδιολογία – Αγγειολογία**	:	**7**
	ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ		
1.	**ΑΝΑΣΚΟΠΗΣΕΙΣ**	:	**4**
2.	**ΚΛΙΝΙΚΟΕΡΓΑΣΤΗΡΙΑΚΕΣ ΜΕΛΕΤΕΣ**	:	**8**
3.	**ΑΝΑΠΤΥΞΗ ΕΡΓΑΣΤΗΡΙΑΚΗΣ ΤΕΧΝΟΛΟΓΙΑΣ**	:	**2**
	ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
1ος Συγγραφέας ή μόνος σε		:	**5**
2ος Συγγραφέας σε		:	**5**
Τελευταίος Συγγραφέας σε		:	**0**
Άλλη Θέση		:	**4**

ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΕΛΛΗΝΙΚΑ ΣΥΝΕΔΡΙΑ

1.	**ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ**	:	**21**
2.	**ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΕΛΛΗΝΙΚΩΝ ΣΥΝΕΔΡΙΩΝ**	:	**37**
	ΣΥΝΟΛΟ	:	**58**
	ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1.	**Χειρουργική**	:	**4**
2.	**Καρδιολογία – Αγγειολογία**	:	**20**
3.	**Κλινική Χημεία – Φαρμακευτική Βιοχημεία**	:	**6**
4.	**Μαιευτική Γυναικολογία**	:	**17**
5.	**Παιδιατρική - Νεογνολογία**	:	**11**
	ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ		
1.	**ΑΝΑΣΚΟΠΗΣΕΙΣ**	:	**1**
2.	**ΚΛΙΝΙΚΟΕΡΓΑΣΤΗΡΙΑΚΕΣ ΜΕΛΕΤΕΣ**	:	**53**
3.	**ΑΝΑΠΤΥΞΗ ΕΡΓΑΣΤΗΡΙΑΚΗΣ ΤΕΧΝΟΛΟΓΙΑΣ**	:	**4**
	ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
1ος Συγγραφέας ή μόνος σε		:	**8**
2ος Συγγραφέας σε		:	**19**
Τελευταίος Συγγραφέας σε		:	**3**
Άλλη Θέση		:	**28**

15. ΣΥΝΟΠΤΙΚΟ ΒΙΟΓΡΑΦΙΚΟ ΣΗΜΕΙΩΜΑ

ΟΝΟΜΑΤΕΠΩΝΥΜΟ	:	ΟΙΚΟΝΟΜΟΥ ΕΜΜΑΝΟΥΗΛ
ΕΘΝΙΚΟΤΗΤΑ	:	ΕΛΛΗΝΙΚΗ
ΔΙΕΥΘΥΝΣΗ ΚΑΤΟΙΚΙΑΣ	:	ΚΑΔΜΟΥ 8 & ΑΓΑΜΕΜΝΩΝΟΣ 24 – ΝΤΡΑΦΙ ΠΙΚΕΡΜΙΟΥ ΡΑΦΗΝΑΣ Τ.Κ. 19009 – Τ.Θ. 2589
ΤΗΛΕΦΩΝΟ ΟΙΚΙΑΣ	:	210-6002540
ΤΗΛΕΦΩΝΑ ΕΡΓΑΣΙΑΣ	:	210-7286259, 210-6421309
ΚΙΝΗΤΟ ΤΗΛΕΦΩΝΟ	:	6944-281173
FAX	:	210-6084476
e-MAIL	:	eveconom@otenet.gr, eveconom@med.uoa.gr
ΗΜΕΡΟΜΗΝΙΑ ΓΕΝΝΗΣΕΩΣ	:	30 ΙΑΝΟΥΑΡΙΟΥ 1962
ΤΟΠΟΣ ΓΕΝΝΗΣΕΩΣ	:	ΑΘΗΝΑ, ΕΛΛΑΔΑ
ΟΙΚΟΓΕΝΕΙΑΚΗ ΚΑΤΑΣΤΑΣΗ	:	ΑΓΑΜΟΣ
ΣΤΡΑΤΙΩΤΙΚΗ ΘΗΤΕΙΑ	:	Απολυτήριο Πολεμικού Ναυτικού Ναύτης Νοσοκόμος – Ναυτική Βάση Νότιου Ευβοϊκού 04.03.1991 – 04.03.1992
ΕΙΔΙΚΟΤΗΤΑ	:	Πτυχιούχος Φαρμακευτικής Σχολής Πανεπιστημίου Αθηνών, Φαρμακολόγος
ΣΗΜΕΡΙΝΗ ΘΕΣΗ	:	Επίκουρος Καθηγητής επί θητεία Κλινικής Χημείας – Φαρμακευτικής Βιοχημείας Ιατρική Σχολή Πανεπιστημίου Αθηνών Τομέας Υγείας Μητέρας-Παιδιού - Β' Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική, ΑΡΕΤΑΙΕΙΟ Πανεπιστημιακό Νοσοκομείο Αθηνών - Εργαστήριο Θεραπευτικής Εξατομίκευσης (Πρόγραμμα ΕΛΚΕ 70/3/9503)

ΣΥΝΟΠΤΙΚΗ ΠΑΡΟΥΣΙΑΣΗ ΣΥΝΟΛΙΚΟΥ ΕΠΙΣΤΗΜΟΝΙΚΟΥ ΕΡΓΟΥ

Α. ΕΓΚΥΚΛΙΟΣ ΕΚΠΑΙΔΕΥΣΗ

Λύκειο Κρανιδίου Αργολίδας, Ιούνιος 1979, Βαθμός Αποφοίτησης : ΑΡΙΣΤΑ (19 και 2/13)

Β. ΠΡΟΠΤΥΧΙΑΚΗ ΕΚΠΑΙΔΕΥΣΗ

Εθνικό και Καποδιστριακό Πανεπιστήμιο της Αθήνας, Πτυχίο Φαρμακευτικού Τμήματος της Σχολής Επιστημών Υγείας, Ιούνιος 1985. Βαθμός Αποφοίτησης: ΑΡΙΣΤΑ (8,63).

Γ. ΑΔΕΙΑ ΑΣΚΗΣΕΩΣ ΕΠΑΓΓΕΛΜΑΤΟΣ

Άδεια Ασκήσεως του Επαγγέλματος της Φαρμακευτικής, Υπουργείο Δημόσιας Υγείας, Οκτώβριος 1992.

Δ. ΜΕΤΑΠΤΥΧΙΑΚΗ ΕΚΠΑΙΔΕΥΣΗ

1. **Εθνικό και Καποδιστριακό Πανεπιστήμιο της Αθήνας. Τμήμα Φαρμακευτικής Τεχνολογίας και Ραδιο-φαρμακευτικής, Διδακτορικό Δίπλωμα, Ιούνιος 1993. Αντικείμενο Διδακτορικής Διατριβής : "Ραδιοανοσοαναλύσεις για τον**

Προσδιορισμό της Διαιθυλο- στιλβοιστρόλης στον Ορό και Αξιολόγηση Αυτών". Βαθμός Διδακτορικής Διατριβής :ΑΡΙΣΤΑ.

2. **Ινστιτούτο Πυρηνικής Ιατρικής, Kern Forschoungsanlage, Juelich, Γερμανία. Μεταδιδακτορική Εκπαίδευση στο Τμήμα Ραδιοφαρμάκων-Ραδιοδιαγνωστικών Προϊόντων-Ραδιοανοσοαναλύσεων, 1989-1991.**
Ειδική Εκπαίδευση σε Ανοσοδιαγνωστικές Τεχνικές (RIA, IRMA, ELISA, FIA, Chemiluminescence Immunoassays).
Βεβαίωση από το αντίστοιχο Ινστιτούτο.

3. **Κέντρο Πυρηνικών Ερευνών "ΔΗΜΟΚΡΙΤΟΣ", Πρόγραμμα Μετα-πτυχιακών Μαθημάτων στα ακόλουθα αντικείμενα (1985 - 1987) : Φασματοσκοπία NMR, Κρυσταλλο- γραφία, Βιομετρία, Βιοχημεία, Νευροχημεία, Ραδιοανοσοχημεία, Ελεγχος Ραδιοφαρμακευτικών Σκευασμάτων, Ειδικά Θέματα IN VITRO και IN VIVO Ραδιοφαρμακευτικών Σκευασμάτων, Θεωρία και Γλώσσες Ηλεκτρονικών Υπολογιστών (BASIC).**

ΔΙΔΑΚΤΙΚΟ ΕΡΓΟ

Α. ΠΡΟΠΤΥΧΙΑΚΟ ΕΚΠΑΙΔΕΥΤΙΚΟ ΕΡΓΟ

1. **ΜΑΘΗΜΑΤΑ**

1.1. "*Θεραπεία Ορμονικής Υποκατάστασης και Καρδιαγγειακή Νόσος – Ο Ρόλος του Ατομικού Παράγοντα*"
Μάθημα στους 6/ετείς τριμηνίτες φοιτητές της Ιατρικής Σχολής
Σε όλες τις ομάδες των ακαδημαϊκών περιόδων 2003-2014

1.2. **"Θρόμβωση : Παθοφυσιολογία – Αντιμετώπιση"**
Μάθημα στους ειδικευομένους χειρουργούς, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, 15-02-2011

Β. ΜΕΤΑΠΤΥΧΙΑΚΟ ΕΚΠΑΙΔΕΥΤΙΚΟ ΕΡΓΟ

1. **ΜΑΘΗΜΑΤΑ**

1.1. *"Μεθοδολογία Εργαστηριακής Ιατρικής Ερευνας"*

1.2. **"***ΟΡΜΟΝΕΣ : Βιοσύνθεση – Απελευθέρωση – Μεταφορά – Δράση – Μεταβολισμός – Μηχανισμοί Ελέγχου"***)**

1.3. *"ΕΡΓΑΣΤΗΡΙΑΚΕΣ ΑΣΚΗΣΕΙΣ – Βιοχημική Μεθοδολογία"*

1.4. "*Δυναμικές Δοκιμασίες Ορμονών στη Γυναικεία Αναπαραγωγή"*

1.5. "*Καθορισμός και Διαφοροποίηση Φύλου στην Ενδομήτριο Ζωή"*

1.6. "Κλινική Σημασία Ορμονικών Μετρήσεων – Το Εργαστήριο στην Κλινική Ενδοκρινολογία"

1.7. "*Μεθοδολογία Εργαστηριακής Ιατρικής Έρευνας*"

2. **ΣΥΜΒΟΛΗ ΣΤΗΝ ΕΚΠΟΝΗΣΗ ΔΙΔΑΚΤΟΡΙΚΩΝ ΔΙΑΤΡΙΒΩΝ**

16 **ΟΛΟΚΛΗΡΩΜΕΝΕΣ ΔΙΔΑΚΤΟΡΙΚΕΣ ΔΙΑΤΡΙΒΕΣ**

13 **Μέλος Τριμελούς Επιβλέπουσας Επιτροπής ή Μέλος Επταμελούς Εξεταστικής Επιτροπής σε 21 Διδακτορικές Διατριβές**

ΕΡΓΑΣΤΗΡΙΑΚΟ ΕΡΓΟ

1 Επίκουρος Καθηγητής επί θητεία Κλινικής Χημείας - Φαρμακευτικής Βιοχημείας, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Τομέας Υγείας Μητέρας Παιδιού, Β' Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική, Αρεταίειο Νοσοκομείο Αθηνών, Εργαστήριο Γενετικής Θεραπευτικής Εξατομίκευσης , Μαϊος 2009 - σήμερα

2 Επιστημονικός Συντονιστής Εργαστηρίου Γενετικής Θεραπευτικής Εξατομίκευσης, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Νοέμβριος 2007 - σήμερα

3 Λέκτορας Κλινικής Χημείας - Φαρμακευτικής Βιοχημείας, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Τομέας Υγείας Μητέρας Παιδιού, 2η Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική, Αρεταίειο Νοσοκομείο Αθηνών, Ορμονολογικό Εγαστήριο, Δεκέμβριος 2003 - 2008

4 Επιστημονικός Υπεύθυνος Εργαστηρίου Ραδιοϊσοτοπικών Αναλύσεων του Τμήματος Πυρηνικής Ιατρικής του Θεραπευτηρίου "ΥΓΕΙΑ", Φεβρουάριος 1998 - σήμερα

5 Νοσοκομειακός Φαρμακοποιός, Επιμελητής Γ' ΕΣΥ, Ιπποκράτειο Γενικό Νοσοκομείο Αθηνών, Οκτώβριος 1995 - Νοέμβριος 2003.

6 Επιστημονικός Συνεργάτης (περιστασιακά) του Διαβητολογικού Κέντρου της Πρώτης Παιδιατρικής Κλινικής του Νοσοκομείου Παίδων "Π. και Α. ΚΥΡΙΑΚΟΥ", Οκτώβριος 1993 - Ιούνιος 1994

7 Επιστημονικός Συνεργάτης του Ερευνητικού Ανοσολογικού-Βιοχημικού Εργαστηρίου της Πανεπιστημιακής Καρδιολογικής Κλινικής του Ιπποκρατείου Γενικού Νοσοκομείου της Αθήνας, Φεβρουάριος 1992 – Νοέμβριος 2003

8 Επιστημονικός Συνεργάτης (περιστασιακά) του τμήματος Νεογνολογίας του Νοσοκομείου "ΑΛΕΞΑΝΔΡΑ", Φεβρουάριος 1990 - Μάρτιος 1994

ΝΟΣΟΚΟΜΕΙΑΚΕΣ ΚΑΙ ΠΑΝΕΠΙΣΤΗΜΙΑΚΕΣ ΘΕΣΕΙΣ

1. ΝΟΣΟΚΟΜΕΙΑΚΕΣ ΘΕΣΕΙΣ

1.1 Εργαστήριο Ραδιοϊσοτοπικών Αναλύσεων του Τμήματος Πυρηνικής Ιατρικής του Θεραπευτηρίου "ΥΓΕΙΑ", Επιστημονικός Υπεύθυνος, Φεβρουάριος 1998 - σήμερα.

1.2 Φαρμακευτική Υπηρεσία Ιπποκρατείου Γενικού Νοσοκομείου Αθηνών, Επιμελητής Γ', Οκτώβριος 1995 - Νοέμβριος 2003

1.3 Ανοσολογικό-Βιοχημικό Εργαστήριο της Πανεπιστημιακής Καρδιολογικής Κλινικής του Ιπποκρατείου Γενικού Νοσοκομείου της Αθήνας, Επιστημονικός Συνεργάτης, Φεβρουάριος 1992 – Δεκέμβριος 2003.

1.4 Διαβητολογικό Κέντρο της Πρώτης Παιδιατρικής Κλινικής του Νοσοκομείου Παίδων "Π.και Α. ΚΥΡΙΑΚΟΥ", Επιστημονικός Συνεργάτης, Οκτώβριος 1993 - Ιούνιος 1994.

1.5 Τμήμα Νεογνολογίας του Νοσοκομείου "ΑΛΕΞΑΝΔΡΑ", Επιστημονικός Συνεργάτης, Φεβρουάριος 1990 - Μάρτιος 1994.

2. ΠΑΝΕΠΙΣΤΗΜΙΑΚΕΣ ΘΕΣΕΙΣ

2.1. Εργαστήριο Γενετικής Θεραπευτικής Εξατομίκευσης, Ιατρική Σχολή Πανεπιστημίου Αθηνών, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Επιστημονικός Συντονιστής, Νοέμβριος 2007 - σήμερα

2.2. Ορμονολογικό Εργαστήριο Αρεταίειου Πανεπιστημιακού Νοσοκομείου Αθηνών, Β'

Πανεπιστημιακή Μαιευτική και Γυναικολογική Κλινική Ιατρικής Σχολής Πανεπιστημίου Αθηνών, Λέκτορας Κλινικής Χημείας – Φαρμακευτικής Βιοχημείας, Δεκέμβριος 2003 – 2008

ΟΡΓΑΝΩΤΙΚΟ - ΔΙΟΙΚΗΤΙΚΟ ΚΑΙ ΕΥΡΥΤΕΡΟ ΕΠΙΣΤΗΜΟΝΙΚΟ ΕΡΓΟ

1. Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών – **Εμπνευστής και Δημιουργός Προτύπου Εργαστηρίου Γενετικής Θεραπευτικής Εξατομίκευσης** – Κλινική Νοσοειδική Φαρμακογενωμική, Νοέμβριος 2007 - σήμερα

   Οργάνωση, Κοστολόγηση, Δρομολόγηση, Διαχειριστική Ένταξη στις Νοσοκομειακές Διαδικασίες, Ενταξη και Εφαρμογή στην Εργαστηριακή Πρακτική εξετάσεων:

2. **Member of Scientific Commitee,** The 9th Athens Congress on Women's Helath and Disease "From Puberty to Menopause", Athens Hilton, August 28-30, 2014, Greece

3. **Μέλος της Επιστημονικής Επιτροπής του** 6ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 4-5 Απριλίου 2014, Ξενοδοχείο Royal Olympic, Αθήνα

4. **Member of Local Advisory Committee**, 8th Congress of Women's Health & Disease, Kos, September 1 – 3, 2011, Greece

5. **Μέλος της Οργανωτικής Επιτροπής του** 5ου Πανελλήνιο Συνεδρίου Κλιμακτηρίου & Εμμηνόπαυσης, 1-2 Απριλίου 2011, Ξενοδοχείο Stratos Vasilikos, Αθήνα

6. **Μέλος της Οργανωτικής Επιτροπής του** 2ου Πανελληνίου Συνεδρίου Παιδικής και Εφηβικής Γυναικολογίας, 17 – 18 Σεπτεμβρίου 2010, Αίγλη Ζαππείου, Αθήνα

7. **Co-chairman of the Lecture "Endometriosis**: From pathophysiology to treatment" by F. Petraglia in the "7th Athens Congress on Women's Health and Disease", September 11 – 13, 2008, Athens, Greece"

8. **Member of the Scientific Committee of the "7th Athens Congress on Women's Health and Disease**", September 11 – 13, 2008, Athens, Greece

9. **Μέλος Οργανωτικής Επιτροπής 1ου Πανελληνίου Συνεδρίου Κλιμακτηρίου και Εμμηνόπαυσης**, 18 – 19 Απριλίου 2008, Μέγαρο Μουσικής Αθηνών, Αθήνα

10. Ορμονολογικό Εργαστήριο Β' Πανεπιστημιακής Μαιευτικής και Γυναικολογικής Κλινικής, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Δεκέμβριος 2003 - Δεκέμβριος 2008 – **Επιστημονική Υποστήριξη προϋπαρχόντων εξετάσεων**

11. Ορμονολογικό Εργαστήριο Β' Πανεπιστημιακής Μαιευτικής και Γυναικολογικής Κλινικής, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Δεκέμβριος 2003 - Δεκέμβριος 2008 **- Οργάνωση, Κοστολόγηση, Δρομολόγηση, Διαχειριστική Ένταξη στις Νοσοκομειακές Διαδικασίες, Ενταξη και Εφαρμογή στην Εργαστηριακή Πρακτική ΝΕΩΝ εξετάσεων**

12. Επιστημονικός Συντονιστής Α' Κύκλου Μαθημάτων (40 ώρες, μαθημάτων, προαιρετική εργαστηρική εκπάιδευση, 9 διδάσκοντες) Μεταπτυχιακού Προγράμματος «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολή του Πανεπισατημίου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών, 29.11.2007 – 16.01.2008

13. Επιστημονικός Συντονιστής Α' Κύκλου Μαθημάτων (40 ώρες, μαθημάτων, προαιρετική εργαστηρική εκπάιδευση, 9 διδάσκοντες) Μεταπτυχιακού Προγράμματος «ΕΡΕΥΝΑ ΣΤΗ ΓΥΝΑΙΚΕΙΑ ΑΝΑΠΑΡΑΓΩΓΗ» της Ιατρικής Σχολή του

Πανεπιστημίου Αθηνών σε συνεργασία με το Τμήμα Μαιευτικής των Τ.Ε.Ι. Αθηνών, 22.10.2008 – 20.11.2008

ΕΡΕΥΝΗΤΙΚΟ – ΣΥΓΓΡΑΦΙΚΟ ΚΑΙ ΣΥΝΤΑΚΤΙΚΟ ΕΡΓΟ

ΣΥΝΕΡΓΑΣΙΑ ΜΕ ΕΡΕΥΝΗΤΙΚΑ ΕΡΓΑΣΤΗΡΙΑ

1. Ερευνητικό Τμήμα Εργαστηρίου Γενετικής Θεραπευτικής Εξατομίκευσης, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Ιατρική Σχολή Ππανεπιστημίου Αθηνών, Νοέμβριος 2007 – σήμερα.
2. Ερευνητικό Τμήμα Ορμονολογικού Εργαστηρίου Β' Πανεπιστημιακής Μαιευτικής και Γυναικολογικής Κλινικής, Αρεταίειο Πανεπιστημιακό Νοσοκομείο Αθηνών, Δεκέμβριος 2003 – Δεκέμβριος 2008
3. Ερευνητικό Τμήμα Εργαστηρίου Βιοχημείας – Ανοσολογίας Α' Πανεπιστημιακής Καρδιολογικής Κλινικής, Ιπποκράτειο Νοσοκομείο Αθηνών, Φεβρουάριος 1992 – Δεκέμβριος 2003
4. Ερευνητικό Τμήμα Ινστιτούτου Ραδιοδιαγνωστικών Προϊόντων – Ραδιοϊσοτόπων, Εργαστήριο Ραδιοϊσοτοπικών Ανοσοναλύσεων, Κέντρο Πυρηνικών Ερευνών «ΔΗΜΟΚΡΙΤΟΣ», 1985 – 1989

ΣΥΜΜΕΤΟΧΗ ΣΕ ΕΡΕΥΝΗΤΙΚΑ ΠΡΟΓΡΑΜΜΑΤΑ

1. Ενα Διεθνές Ερευνητικό Πρόγραμμα επιχορηγούμενο από «Cardiovascular Research and Interventional Radiological Society of Europe
2. Πέντε Ελληνικά Ερευνητικά Προγράμματα επιχορηγούμενα από την Ιατρική Σχολή Πανεπιστημίου Αθηνών
3. Εξι Ελληνικά Ερευνητικά Προγράμματα επιχορηγούμενα από προγράμματα «ΚΑΠΟΔΙΣΤΡΙΑΣ»
4. Δύο Ελληνικά Ερευνητικά Προγράμματα επιχορηγούμενα από το «ΚΕΝΤΡΟ ΕΛΕΓΧΟΥ ΚΑΙ ΠΡΟΛΗΨΗΣ ΝΟΣΗΜΑΤΩΝ (Κ.ΕΛ.Π.ΝΟ.)»

ΣΥΝΤΑΚΤΙΚΟ ΕΡΓΟ

1. Μέλος Συντακτικής Επιτροπής στο περιοδικό «Medical Science Monitor»
2. Σύμβουλος Συντακτικής Επιτροπής Επιστημονικών Περιοδικών
   - **1.** «ΕΛΛΗΝΙΚΗ ΚΑΡΔΙΟΛΟΓΙΚΗ ΕΠΙΘΕΩΡΗΣΗ» - 6 Αρθρα
   - **2.** «ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ» - 1 Αρθρο
   - **3.** «BIOCHEMICAL PHARMACOLOGY» - 1 Αρθρο
   - **4.** «MEDIATORS OF INFLAMMATION» - 8 Αρθρα
   - **5.** «ATHROSCLEROSIS» - 28 Αρθρα
   - **6.** «Journal of Cellular Physiology» - 1 Αρθρο

ΣΥΜΜΕΤΟΧΗ – ΑΝΑΚΟΙΝΩΣΕΙΣ ΣΕ ΕΠΙΣΤΗΜΟΝΙΚΕΣ ΣΥΝΑΝΤΗΣΕΙΣ

Διεθνή Συνέδρια	**:**	**130**
Πανελλήνια Συνέδρια	**:**	**79**

ΕΡΕΥΝΗΤΙΚΟ ΣΥΓΓΡΑΦΙΚΟ ΕΡΓΟ

I. ΠΛΗΡΕΙΣ ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΞΕΝΟΓΛΩΣΣΑ ΠΕΡΙΟΔΙΚΑ

1.	**ΔΗΜΟΣΙΕΥΜΕΝΕΣ**	:	**67**
2.	**ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ**	:	**0**
	ΣΥΝΟΛΟ	:	**67**

ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ

1.	**Καρδιολογία - Αγγειολογία**	:	**14**
2.	**Γυναικολογία**	:	**29**
3.	**Νεογνολογία – Παιδιατρική**	:	**17**
4.	**Χειρουργική**	:	**3**
5.	**Ψυχιατρική**	:	**1**
6.	**Ραδιοφαρμακολογία**	:	**3**

ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ

1.	**Πειραματικές Εργασίες**	:	**3**
2.	**Κλινικοεργαστηριακές Μελέτες**	:	**64**

ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ

1ος Συγγραφέας σε	:	**6**
2ος Συγγραφέας σε	:	**14**
3ος Συγγραφέας σε	:	**17**
Τελευταίος Συγγραφέας	:	**6**
Άλλη Θέση	:	**24**

ΣΥΝΤΕΛΕΣΤΗΣ ΑΠΗΧΗΣΗΣ (IMPACT FACTOR, I.F.) (2013)

Συνολικός	:	**295,54**
Συνολικός (εκτός Letter to the Editor)	:	**166,38**
Μέσος Ορος Συνολικού	:	**4,41**
Προσωπικός	:	**190,25**
Μέσος Ορος Προσωπικού	:	**2,84**

ΒΙΒΛΙΟΓΡΑΦΙΚΕΣ ΑΝΑΦΟΡΕΣ

Συνολικός Αριθμός Βιβλιογραφικών Αναφορών	:	**1692**
Συνολικός Αριθμός Βιβλιογραφικών Αναφορών (εκτός αυτοαναφορών)	:	**1670**
Προσωπικός Συνολικός Αριθμός	:	**594,90**
Προσωπικός Συνολικός Αριθμός (εκτός αυτοαναφορών)	:	**584,75**
h-index	:	**20**
i-10 index	:	**32**

"ΠΡΟΣΩΠΙΚΟΣ" : ΔΙΟΡΘΩΜΕΝΟΣ ΜΕ ΒΑΣΗ ΤΗ ΘΕΣΗ ΤΟΥ ΣΥΓΓΡΑΦΕΩΣ

Συντελεστής ανά θέση	**1η και τελευταία θέση**	**2η θέση**	**3η θέση**	**άλλη θέση**
	1	**0.5**	**0.33**	**0.25**

II. ΞΕΝΟΓΛΩΣΣΕΣ ΠΕΡΙΛΗΨΕΙΣ ΑΝΑΚΟΙΝΩΣΕΩΝ ΣΕ ΔΙΕΘΝΗ ΣΥΝΕΔΡΙΑ ΔΗΜΟΣΙΕΥΜΕΝΕΣ ΣΕ ΔΙΕΘΝΗ ΠΕΡΙΟΔΙΚΑ

Συνολικός Αριθμός		:	102
	ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1.	Κλινική Χημεία – Φαρμακευτική Βιοχημεία	:	11
2.	Νεφρολογία – Βιολογία Νεφρικών Αρτηριών	:	3
	Μαιευτική Γυναικολογία	:	5
	Παιδιατρική - Νεογνολογία	:	7
	Καρδιολογία – Αγγειολογία	:	76
	ΣΥΝΤΕΛΕΣΤΗΣ ΑΠΗΧΗΣΗΣ (IMPACT FACTOR, I.F.) (2013)		
Συνολικός		:	1312,16
Μέσος Ορος Συνολικού		:	15,22
Προσωπικός		:	746,6
Μέσος Ορος Προσωπικού		:	7,32
Συνολικός Αριθμός Βιβλιογραφικών Αναφορών		:	10
Συνολικός Αριθμός Βιβλιογραφικών Αναφορών (εκτός αυτοαναφορών)		:	9
Προσωπικός Συνολικός Αριθμός Βιβλιογραφικών Αναφορών		:	5,58
Προσωπικός Συνολικός Αριθμός Βιβλιογραφικών Αναφορών (εκτός Αυτοαναφορών)		:	4,58
	ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
1ος Συγγραφέας ή μόνος σε		:	33
2ος Συγγραφέας σε		:	13
Τελευταίος Συγγραφέας σε		:	0
Άλλη Θέση		:	56

III. ΔΗΜΟΣΙΕΥΣΕΙΣ ΣΕ ΕΛΛΗΝΙΚΑ ΠΕΡΙΟΔΙΚΑ

1.	**ΔΗΜΟΣΙΕΥΜΕΝΕΣ**	:	14
2.	**ΔΕΚΤΕΣ ΠΡΟΣ ΔΗΜΟΣΙΕΥΣΗ**	:	0
	ΣΥΝΟΛΟ	:	14
	ΘΕΜΑΤΙΚΗ ΚΑΤΑΝΟΜΗ		
1.	Κλινική Χημεία – Φαρμακευτική Βιοχημεία	:	7
2.	Καρδιολογία – Αγγειολογία	:	7
	ΕΙΔΟΣ ΔΗΜΟΣΙΕΥΣΗΣ		
1.	ΑΝΑΣΚΟΠΗΣΕΙΣ	:	4
2.	ΚΛΙΝΙΚΟΕΡΓΑΣΤΗΡΙΑΚΕΣ ΜΕΛΕΤΕΣ	:	8
3.	ΑΝΑΠΤΥΞΗ ΕΡΓΑΣΤΗΡΙΑΚΗΣ ΤΕΧΝΟΛΟΓΙΑΣ	:	2
	ΣΕΙΡΑ ΥΠΟΨΗΦΙΟΥ ΜΕΤΑΞΥ ΣΥΓΓΡΑΦΕΩΝ		
1ος Συγγραφέας ή μόνος σε		:	5
2ος Συγγραφέας σε		:	5
Τελευταίος Συγγραφέας σε		:	0
Άλλη Θέση		:	4

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ORIGINAL ARTICLE

The effect of hormone therapy on biochemical and ultrasound parameters associated with atherosclerosis in 46,XY DSD individuals with female phenotype

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Abstract

The aim of this study was to evaluate the effect of hormone therapy (HT) in the endothelial function of 46,XY disorders of sexual development (DSD) patients with female phenotype. Biochemical and ultrasound measurements were performed in 20 patients at initiation of oral 2 mg 17β-estradiol/1 mg norethisterone acetate, and after 6 months of therapy. Lipid profile, including total cholesterol (TC), LDL, HDL, triglycerides (TG) and Atherogenic Index of Plasma (AIP), as well as levels of VE-Cadherin, E-Selectin, Thrombomodulin and vWf were determined. Ultrasonographic examinations included evaluation of flow-mediated dilatation (FMD) and measurement of Carotid and Femoral Intima Media Thickness (IMT). HT raised HDL (35.4 mg/dl versus 40.1 mg/dl, $p = 0.019$) while lowering TG (166 mg/dl versus 109 mg/dl, $p = 0.026$) and AIP (0.24 versus 0.04, $p = 0.007$). No changes were noted in TC and LDL (215.7 mg/dl versus 192.25 mg/dl and 87.46 mg/dl versus 76.35 mg/dl, respectively). There was significant reduction of VE-Cadherin (4.05 ng/ml versus 2.20 ng/ml, $p = 0.002$) and E-selectin (73.98 ng/ml versus 56.73 ng/ml, $p = 0.004$). No change was observed in Thrombomodulin and vWf (11.76 ng/ml versus 13.90 ng/ml and 80.75% versus 79.55%, respectively). FMD improved significantly (5.4% versus 8.15%, $p = 0.003$), while only carotid bulb IMT decreased significantly (0.65 mm versus 0.60 mm, $p = 0.018$). Overall, HT was found to improve biochemical and ultrasound markers of endothelial function in 46,XY DSD patients with female phenotype.

Keywords

Complete androgen insensitivity syndrome, E-selectin, flow-mediated dilatation, gonadal dysgenesis, intima media thickness, swyer syndrome, VE-cadherin, vWf

History

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Introduction

46,XY disorders of sexual development (DSD) occur during the embryonic sexual differentiation process and usually lead to phenotypically female individuals with male karyotype. The most common causes include defects, either in testicular formation, or in the action of androgens. In the first case, known as XY gonadal dysgenesis or Swyer syndrome, the dysgenetic gonads, produce neither Testosterone (T) nor Anti-Müllerian Hormone (AMH), resulting in patients with female genitalia, vagina and uterus. The second case refers to Androgen Insensitivity Syndrome, and specifically to Complete Androgen Insensitivity Syndrome (CAIS), in which the gonad is functioning properly, producing both T and AMH. Due to receptor or post-receptor defect, T does not promote the differentiation of Wolffian ducts to the male genital system. Moreover, the normal production of AMH does not permit Müllerian duct development, resulting in a newborn with female external genitalia but absence of internal genital organs.

In these groups of XY females, the lack, or no action of sex hormones is associated with decreased bone mineral density (BMD), sexual dysfunction and lower overall quality of life [1]. Moreover, patients may carry a higher long-term risk of cardiovascular disease (CVD) as they lack the anti-atherogenic effect of endogenous estrogens, similarly to women with menopause, primary ovarian insufficiency (POI) or Turner syndrome (TS) [2–4].

Administration of hormone therapy (HT) until the fifth decade of life is recommended for XY DSD patients [1]. Studies in TS and POI patients receiving HT have shown a significant improvement in endothelial function [5–7]. The same effect has not been confirmed for all postmenopausal women through large RCTs, including Heart and Estrogen/Progestin Replacement Study (HERS) and Women's Health Initiative (WHI). Possible confounding factors have been considered to be

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lyzed by means of RNA sequencing with deep coverage (average, 50 million 100-bp paired-end reads, with 100 million reads per sample). This finding is especially puzzling, given the strong clinical association. An alternative possible mechanism for the pharmacogenetic association involves the kidney, in which GADL1 appears to be more abundantly expressed. Despite its name, there is no evidence that GADL1 is GAD-like in brain function. Rather, a physiologic role for GADL1 in taurine biosynthesis has been suggested, with potential relevance to kidney function.[4,5] We therefore encourage a retrospective review of kidney function and lithium levels in patients, stratified according to *GADL1* genotype, in the study by Chen et al.

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No potential conflict of interest relevant to this letter was reported.

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TO THE EDITOR: With regard to the article by Chen et al.: we calculated the odds ratio for each genotype to emphasize the strength of the associations in the combined cohorts. Thus, as compared with patients who were homozygous for the C allele, those who were heterozygous for the T allele had an odds ratio of 73.5 (95% confidence interval [CI], 35.3 to 153.3), whereas for those who were homozygous for the effective allele, the odds ratio rose to 228.7 (95% CI, 60.9 to 859.5; $P=1.7\times10^{-49}$ for trend).

Given the huge magnitude of the association between the presence of the T allele and the response to lithium therapy, we would like to ask the authors whether there was a significant difference in the minimum efficacious serum lithium level[1] between carriers and noncarriers of the "response" allele.

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DOI: 10.1056/NEJMc1401817

TO THE EDITOR: We assessed 154 Japanese patients with bipolar disorder (109 patients with bipolar I disorder and 45 with bipolar II disorder) whom we evaluated using the Alda scale (with a score of 6 to 10 indicating a good response to lithium therapy and a score of 0 to 5 indicating a poor response).[1] We genotyped rs17026688 in *GADL1*, one of the two SNPs that showed the strongest associations with a response to lithium therapy in the genomewide association study by Chen et al. This SNP showed a large effect size (odds ratio, approximately 80). As a phenotypic definition, Chen et al. examined only patients with bipolar I disorder and those with an Alda score of 0 on the first four items in criterion B. (On the Alda scale, criterion B, which is used to determine whether there is a causal relationship between clinical improvement and lithium therapy, is divided into levels B1 through B5, with each part scored as 0, 1, or 2 points.) However, as a relaxed phenotyping, we analyzed all patients with bipolar I disorder and bipolar II disorder who had an Alda score of 1 or less on items B1 through B4. We did not observe an association for any criterion (Table 1), even in the stringent phenotype analysis, as reported by Chen et al. This replication analysis did not support an association between *GADL1* variants and a response to lithium therapy in a Japanese population. Therefore, this variant is not an ideal predictor of

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LETTER

Zooming in on the definition of 'recurrent implantation failure'

To the Editor

We read with great interest the comprehensive review by Coughlan et al. (2014) on recurrent implantation failure (RIF). The authors begin their article by discussing the controversy surrounding the definition of this clinical entity.

We consider that the term 'implantation failure' is too narrow since the clinical phenomenon under consideration also includes failure to achieve a clinical pregnancy even after a successful implantation has occurred. We accordingly suggest that the terms 'embryo transfer failure after IVF' or 'IVF–embryo transfer failure', which have been used by several researchers (Catalanotti et al., 2006; Matsubayashi et al., 2008; Qublan et al., 2008) is preferable.

IVF–embryo transfer failure is the most important determinant of IVF outcome. In 2007 in the USA, 101,897 cycles were initiated using fresh nondonor eggs or embryos, resulting in 90,295 retrievals (88.6%), 82,437 embryo transfers (80.9%) and 36,079 clinical pregnancies (35.4%) which led to 29,556 live-birth deliveries (29.0%). More than half of embryo transfers after IVF failed to proceed to a clinical pregnancy (IVF–embryo transfer failure rate 56.2%). IVF failure was 64.6%, with IVF–embryo transfer failure accounting for 70.4% of the total IVF failure (CDC, 2009).

Notably, the number of embryos transferred should not be included in the definition because it is not determinative of the implantation outcome. Current evidence suggests that number of embryos transferred should be limited to no more than two, while the number of embryos transferred (one or two) has not been associated with implantation success rates. The authors used a probabilistic approach considering that every additional embryo has the same probability of successful implantation as the first. Thus, if the probability for an embryo to successfully implant is 30%, the probability of a successful implantation of at least one embryo after a double-embryo transfer, would be $1-(0.70)^2=51\%$. However, in a recent relative meta-analysis (Baruffi et al., 2009), although the double-embryo transfer produced a live birth rate of 42.5% compared with single-embryo transfer (28.4%), the implantation rate was not significantly different between the double-embryo (34.5%) and single-embryo transfer groups (34.7%). Moreover, the transfer of three or more embryos at any age should be avoided, since the transfer of three embryos increases the risk of poor perinatal outcomes without significant increase in live birth rates (Lawlor and Nelson, 2012). In 2009, in the UK, 95% of transfers were limited to one or two embryos, while in Sweden, no transfer of more than two embryos was reported whereas 70% of the cases were single-embryo transfers (Ferraretti et al., 2013).

Furthermore, although the minimal number of cycles in the definition of RIF should be limited to two consecutive failures, similarly with the recent redefinition of the recurrent pregnancy loss by the American Society for Reproductive Medicine (2013), from a clinical point of view it is implausible that one failure be considered a matter of luck whereas two or three failures constitute a disease. To quote As Penzias (2012), 'IVF failure is a problem for a couple in the singular but can be a tragedy in the plural'. Implantation rates are significantly higher for women undergoing their first embryo transfer and decline dramatically in subsequent cycles (Shapiro et al., 2001). As IVF treatment becomes more effective per cycle, it is anticipated that a greater proportion of potentially successful patients should be able to achieve pregnancy at their first attempt. Thus, an early investigation, following the first IVF–embryo transfer failure of a nulligravida woman is very likely to be beneficial.

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with intrauterine insemination before IVF is approved. The medical profession and insurance providers would be well advised to focus on non-IVF treatments, which are a major source of medical complications and increased costs associated with infertility treatments in the United States, rather than focusing on reducing twin pregnancies in association with IVF.[3-5]

Table 1. Comparison of Multiple Births Attributable to Fertility Treatments in 1998 and 2011, According to Maternal Age.

Maternal Age	Twin Births Attributable to Fertility Treatments		Triple and Higher-Order Births Attributable to Fertility Treatments	
	1998	2011	1998	2011
	percent			
<30 yr	25	32	76	70
30–34 yr	25	35	88	78
35–39 yr	31	41	87	80
≥40 yr	52	68	92	89

served among women between the ages of 30 and 34 years.

Furthermore, it would have been interesting if the authors had presented the estimated separate contributions of IVF and non-IVF procedures to twin births, as well as to triplet and higher-order births, according to maternal age. Alternatively, we suggest that the distribution of the plurality of births from natural conception was best approximated during the period from 1971 to 1973, the years with the lowest age-specific rates of multiple births during the past five decades. During this period, the effect of fertility treatments, which had recently become available, was probably negligible. Using the 1971–1973 birth rates as a reference, we estimated that by 2011, the proportion of triplet and higher-order births attributable to fertility treatments for women in all age groups was similar (76%) to the authors' estimations, but the corresponding proportion of twin births was higher (39%).

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THE AUTHORS REPLY: Our analysis showed that the increase in the rate of multiple births in the United States is attributed to three main factors: non-IVF treatments, IVF treatments, and delayed childbearing. We wholeheartedly support the advice of Gleicher et al. to focus efforts on preventing multiple births resulting from non-IVF treatments, the major source of iatrogenic multiple births in the United States. We believe that monitoring outcomes of non-IVF treatments is a critical step toward curtailing multiple births resulting from these treatments. Monitoring IVF outcomes and placing concerted attention on reducing the number of embryos transferred made it possible to reverse the trend of increasing rates of higher-order multiple births.

As much as we are encouraged by the progress, we strongly believe that it is time to intensify, and not redirect, efforts to promote healthy singleton live births as an optimal outcome of any type of fertility treatment.[1] In 2010, almost half (46%) of infants conceived through assisted reproductive technology were born in multiple gestations, primarily as twins.[2] Overall, as compared with singletons, twins are six times as likely to be preterm, nine times as likely to have a low birth weight, and five times as likely to die during the first year of life.[3]

The observation of Vlachadis et al. that the greatest effect of fertility treatments on multiple births was observed among women between the ages of 30 and 34 years is noteworthy and requires additional investigation. We hypothesize that this observation may be explained by a combination of maternal factors (e.g., women 30 to 34 years of age may have a higher risk of multiple births than do women under the age of 30 years or may seek fertility treatments more often) and treatment factors (e.g., women in that age group may have higher implantation rates than women 35 years of age or older). Since the objective of our study was to estimate the maternal age–adjusted contribution of natural conception and fertility treatments to multiple births for the entire U.S. population, we did not stratify the results according to age and made an a priori decision to use data from the 1962–1966 period because those years predated the introduction of clomiphene in the United States.

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The findings and conclusions in this letter are those of the authors and do not necessarily represent the official position of

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LETTER

Aspirin to improve IVF unexplained implantation rates: time for an individualized approach

To the Editor

The article by Akhtar et al. (2013) is the most recent in a series of relevant studies examining the effect of aspirin use on the outcome of embryo transfer after IVF in women with a history of at least one unexplained failure. Most of these reports, including the aforementioned one, did not detect any statistically significant benefit (Clark, 2013).

Two main methodological issues that arise in these studies, apart from insufficient size of groups included, are the multiplicity of possible divergence pathogenetic mechanisms leading to currently 'unexplained' IVF–embryo transfer failure and the variation in platelet response to aspirin among treated individuals (Hovens et al., 2007).

In the design of such a clinical study, researchers should confront two key questions: (i) Which is the subgroup of women expected to benefit from aspirin use? Women with inherited platelet thrombophilia, for example those who are carriers of genetic heterogeneity of platelet glycoprotein Ia (GpIa-C807T) and IIIa (GpIIIa-PlA1/PlA2), are at increased risk of implantation failure and could be considered eligible for antiplatelet treatment with aspirin (Ivanov et al., 2012); and (ii) Will these patients eventually respond to aspirin? Pre-therapeutic evaluation with laboratory tests (such as platelet function analyser, PFA-100 with a collagen/epinephrine cartridge) is important for identifying the subgroup of women most likely to respond to aspirin, and determining effective dose (Snoep et al., 2007; Reny et al., 2008).

The key message from these studies is that not all patients with unexplained implantation failure may benefit from aspirin (or heparin) use. An individualized approach to antiplatelet therapy based on pharmacogenetic profile may maximize the antithrombotic effect and lead to a clinically obvious favourable effect.

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Current concepts of optimal gestational age for delivery based on gestational age associated risks of fetal and neonatal death

TO THE EDITORS: In their excellent article, Mandujano et al[1] performed a brilliant analysis comparing fetal and neonatal mortality by week of gestation, including the risk of remaining undelivered, to determine the best gestational age of delivery.

In the low-risk cohort, the total number of deaths after 34^{+0} weeks of gestation is 16,639. The estimated minimum total number of losses that would occur (after 34^{+0} weeks) if all the remaining fetuses in the beginning of each week were delivered during this specific week is 13,669 and corresponds to 38 weeks' gestation (38^{+0} to 38^{+6}) in the low-risk cohort, the proportion of stillbirths and neonate deaths being at 53% and 47%, respectively.

These findings suggest that if all subjects were delivered by the end of the 39th week (38^{+6}), there would be 33% less intrauterine deaths with the cost of only 11% more neonatal deaths, resulting in an overall 18% reduction of total losses, whereas delivery at 39 weeks (39^{+0} to 39^{+6}) would result in only 911 more losses (14,580 vs 13,669, +7%), compared with delivery at 38 completed weeks.

The authors suggest that the best gestational age for delivery in singleton low-risk pregnancies is before 39 weeks' gestation, but this applies to this specific cohort, which is characterized by an overall double number of intrauterine deaths, in comparison with the neonatal deaths (10,863 vs 5776). In another pregnant population with a higher ratio of stillbirths to neonatal deaths (eg, high-risk pregnancies of this cohort: 1912 vs 581), the optimal gestational age for delivery would be earlier and vice versa.

A more thorough consideration of this issue possibly would be the inclusion of infant deaths associated with each gestational week in the estimation of the risk of delivery; this would lead to moving the best week for delivery later in pregnancy, probably at 39 weeks' gestation. The only undisputed finding of this study is that low-risk pregnancies should not progress beyond a specific point in the beginning of the 41st week of gestation because this would entail a continuous increase in both fetal and neonatal deaths. ■

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The authors report no conflict of interest.

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REPLY

We would like to thank Drs Vlachadis, Tsamadias, and Economou for their interest in our work and for their complimentary Letter to the Editor.[1]

We could not agree more that the reported findings in our work[2] should not be applied outside the population of the

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CORRESPONDENCE

Association between miscarriage and future maternal cardiovascular disease

To the Editor The excellent article by Oliver-Williams and coauthors[1] provides strong evidence to link miscarriages, the most prevalent major complication of pregnancy, the commonest situation in women's life, with coronary heart disease, the leading cause of death in women.

The relationship between miscarriage and cardiovascular risk should be attributed to certain risk factors shared by the two multifactorial disorders, thrombophilia holding a prominent position. Acquired thrombophilia is adequately discussed in the article. Recent attention has been focused on inherited thrombophilic factors that may predispose to pregnancy complications, including pregnancy loss. The genetic heterogeneity of several loci, including coagulation factors V (FVL-R506Q) and II (FII-G20210A), strongly associated with venous and modestly with arterial thrombosis,[2] and platelet glycoproteins Ia (GpIa-C807T) and IIIa (GpIIIa-Pl^{A1}/Pl^{A2}) which influence thrombosis exclusively in the arterial side, have been studied in women with miscarriages.[3] Clinically significant relative risks are mainly related to the accumulation of risk alleles acting synergistically.[4] The postulated pathogenetic mechanisms include abnormal placentation and reduced perfusion of the intervillous space.

Albeit the majority of miscarriages are considered to be sporadic, the important corollary of this study is that women with a history of even one miscarriage are at 50% higher risk of coronary heart disease. The authors also report a twofold risk for women with recurrent miscarriages, implying a 'dose–response relationship'. However, some women may have experienced only one fetal loss because they had only one pregnancy, but still have a higher burden of risk factors, compared with those with recurrent miscarriages. High-risk women would be expected to have an increased incidence of miscarriages in younger age (age is an established risk factor for miscarriage) or miscarry earlier in pregnancy[3] (the accumulation of risk factors contributing to an acceleration of the biological processes of the disorder). A potential explanation of the aforementioned finding could be that every fetal loss per se increases cardiovascular risk, potentially via endothelial stimulation or dysfunction, and hence these women should be considered for relevant evaluation (eg, FDA approved lipoprotein-associated phospholipase A2 and/or flow mediated dilatation).

These results support respective screening in women with at least one miscarriage to identify individual risk factors in order to provide personalised treatment for successful completion of the next pregnancy and early prevention of future cardiovascular events.

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Statins in pregnancy: safety and perspectives of therapeutic applications

Sir,

The article by Winterfeld et al.[1] adds another piece of evidence supporting the safety of statins in pregnancy. Classification of statins as pregnancy category X by the Food and Drug Administration since the beginning of the 1980s was based mainly on case reports and has remained unquestioned primarily because there was no therapeutic indication of statin use in pregnancy.[2] A pragmatic evaluation of statin safety in pregnancy is needed not only because of the growing likelihood of a pregnant woman receiving statin therapy (e.g. inherited hyperlipidaemia, increasing pregnancy rates for women over 35 years old), as the authors rightly note, but also because statins are important promising candidates for the prevention of adverse pregnancy outcomes in high-risk women.

There is a strong need for pharmacological agents for pre-eclampsia, because there is no effective prophylactic therapy and delivery remains the only definitive treatment. Certain pathogenetic and pathophysiological phenomena such as endothelial dysfunction and imbalanced immunological and inflammatory responses are parallel in pre-eclampsia and atherosclerosis. Statins have become established in the primary and secondary prevention of cardiovascular disease. Their pleiotropic beneficial effects, which extend far beyond lipid-lowering and involve endothelial function modification, immunoinflammatory responses and thrombus formation, offer a theoretical basis for using statins in the prevention of pre-eclampsia. Studies using animal models of pre-eclampsia have shown promising results.[2] Low-dose aspirin, by inhibiting the production of thromboxane A2, has been shown to reduce the risk of pre-eclampsia[3] and, moreover, a synergy between aspirin and statins in reducing cardiovascular events has been demonstrated.[4] Hence, it is plausible that a combination of aspirin with a statin initiated early (before 16 weeks) and administered throughout the pregnancy, would have increased efficacy in the prevention of pre-eclampsia. Further therapeutic applications of statins in pregnancy could also be considered, such as the potential benefits from its use (alone or in combination with aspirin) in lowering the elevated fibrinolysis inhibitor Lipoprotein (a), which appears to be a risk factor not only for pre-eclampsia, but also other obstetric complications like miscarriages or intrauterine growth restriction.

The first pilot randomised controlled trials to assess the safety of statins in pregnancy are already underway[2] and as soon as enough evidence is provided, a new group of therapeutic agents could be introduced into obstetric practice, opening new roads in the prevention of common pregnancy complications.

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Statins in pregnancy: safety and perspectives of therapeutic applications

Authors' Reply

Sir,

We thank Dr Vlachadis and colleagues for their interest in our work and their comments.[1] The results of our study, which showed no significant increase in the risk of major birth defects after statin exposure during the first trimester of pregnancy, incited them to argue in favour of the future use of statins in the prevention of pre-eclampsia and other obstetric complications.

We agree upon theoretical considerations and experimental data possibly

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ORIGINAL ARTICLE

Gene receptor polymorphism as a risk factor for BMD deterioration in adolescent girls with anorexia nervosa

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Abstract

Anorexia nervosa is a serious eating disorder that is associated with decreased bone mineral density and greater lifetime risk for fractures. This case-controlled study, analyzed single nucleotide polymorphisms of genes encoding vitamin D receptor, estrogen receptor alpha (ESR1), collagen type I and calcitonin receptor (CTR). Relationships between genotype and body mass index, cycling status and lumbar spine bone mineral density (LBMD) were determined in 40 adolescent girls with anorexia nervosa and 10 age-matched controls. The distribution of CTR-AluI genotypes differed between groups, but this polymorphism was not associated with LBMD *Z*-score. Distribution of ESR1-XbaI genotypes did not differ between groups, but the AA genotype was associated with decreased LBMD *Z*-score (≤−1) (OR = 24.79, 95% CI, 1.01–606.08). Carriers of the A allele were more likely to have decreased LBMD *Z*-scores compared with carriers of the G allele (OR = 4.12, 95% CI, 1.23–13.85, $p = 0.022$).

In conclusion, our study shows that anorexic patients with wild-type genotype ESR-XbaI receptor are in greater risk for decreased BMD in relation to those with the mutated gene. Prompt recognition of these patients is crucial because early administration of the proper therapeutic treatment may contribute to the prevention of adverse sequelae on bone metabolism.

Keywords

Adolescent, anorexia nervosa, BMD, gene polymorphism

History

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Introduction

Anorexia nervosa is a serious eating disorder of unknown etiology that presents mainly in adolescent girls and young women [1]. Clinical sequelae depend on the time the condition occurs in relation to puberty [2,3].

Low BMD is a risk for every patient with anorexia nervosa. Adolescent girls with anorexia nervosa are more likely to have low bone mineral density (BMD) than healthy girls of the same age [4], especially when the disorder appears before puberty [5]. Bone mass at the beginning of the disorder [6] and genetic factors [7] also influence this process. Because approximately 45% of the bone mineral content is acquired during puberty [8], occurrence of anorexia nervosa at this time may prevent the attainment of optimal bone mass. Accordingly, girls with anorexia nervosa have increased lifetime risk of fractures compared with girls who do not have this condition [2,9].

To test our hypothesis that one or more genes polymorphisms, involved in bone metabolism, are associated with bone density in adolescent girls with anorexia nervosa, we analyzed single nucleotide polymorphisms (SNPs) in genes encoding vitamin D receptor (VDR), estrogen receptor alpha (ESR1), calcitonin receptor (CTR) and collagen type I (COL1A1) in 40 adolescent girls with anorexia nervosa and 10 age-matched healthy controls, in relationship with body mass index (BMI), serum 17-β estradiol (E2) concentration, cycling status and lumbar BMD (LBMD).

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Materials and methods

Patients

In this pilot study, we included 40 girls (12–21 years old) with anorexia nervosa diagnosed using APA criteria (proposed DSM-V, 2012) [10] and 10 aged-matched controls (healthy girls). The study was approved by the Head of Obstetrics and Gynecology Department and the Research and Ethics Committee of our hospital. Written informed consent was obtained from all participants and their parents or legal guardians. The protocol complied with the principles laid down in the Declaration of Helsinki.

Inclusion criteria were BMI 12.5–18.5 kg/m^2 (<5th percentile for age) for cases, BMI 18.5–25 kg/m^2 (5th–85th percentile for age) for controls and ≤2 h of physical exercise per week for both groups. Exclusion criteria were organic or metabolic diseases related to bone metabolism, malnutrition, use of hormone therapy (including birth control pills) and tobacco use.

Clinical and laboratory tests

A detailed medical history was taken during the first visit, and participants completed questionnaires on demographic and

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Research Article

Desferrioxamine Attenuates Pancreatic Injury after Major Hepatectomy under Vascular Control of the Liver: Experimental Study in Pigs

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Introduction. Pancreatic injury can manifest after major hepatectomy under vascular control. The main mechanism involved seems to be remote oxidative injury due to "spillage" of reactive oxygen species and cytokines from the liver. The aim of this study is to evaluate the role of desferrioxamine in the prevention of pancreatic injury following major hepatectomy. *Methods.* Twelve Landrace pigs were subjected to a combination of major hepatectomy (70–75%), using the Pringle maneuver for 150 minutes, after constructing a porta-caval side-to-side anastomosis. The duration of reperfusion was 24 hours. Animals were randomly divided into a control group ($n = 6$) and a desferrioxamine group (DFX, $n = 6$). DFX animals were treated with continuous IV infusion of desferrioxamine 100 mg/kg. Pancreatic tissue injury, c-peptide and amylase concentrations, and pancreatic tissue oxidative markers were evaluated. *Results.* Desferrioxamine-treated animals showed decreased c-peptide levels, decreased acinar cell necrosis, and decreased tissue malondialdehyde levels 24 hours after reperfusion compared with the control group. There was no difference in portal pressure or serum amylase levels between the groups. *Conclusions.* Desferrioxamine seems to attenuate pancreatic injury after major hepatectomy under vascular control possibly by preventing and reversing production and circulation of oxidative products.

1. Introduction

Ischemia and reperfusion injury takes place during major hepatectomies due to the need for the use of vascular control techniques, as well as in liver transplantation and liver trauma. Although such maneuvers are invaluable in preventing excessive blood loss, they result in the production of cytokines and reactive oxygen species (ROS), which are responsible for induction of oxidative stress to the liver as well as to distant organs [1, 2]. Spillage of cytokines and inflammatory mediators has been shown to promote remote injury [3].

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Changes in Maternal Serum Thioredoxin (TRX) Levels after Delivery in Preeclamptic and Normotensive Pregnant Women

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Objective. To investigate changes of maternal plasma thioredoxin (TRX) levels after delivery in preeclamptic and normotensive pregnant women. *Methods*. Ten normotensive women (group A) were compared to 17 women with severe preeclampsia (group B). TRX levels were assessed in maternal plasma, immediately after delivery and 12–16 weeks postpartum. *Results*. There were no differences in plasma TRX levels between the two groups immediately antepartum ($p = 0.095$). A significant reduction in plasma TRX levels was found immediately following delivery only in normotensive group (117.76 ± 37.19 ng/mL vs. 43.45 ± 21.11 ng/mL, $p = 0.002$), but not in women with preeclampsia (80.42 ± 59.95 ng/mL vs. 53.82 ± 44.34 ng/mL, $p = 0.12$). Plasma TRX levels remained unchanged in women with preeclampsia (80.42 ± 59.95 ng/mL vs. 55.37 ± 52.23 ng/mL, $p = 0.2$) at 12–14 weeks postpartum.

Keywords Preeclampsia, TRX, Antepartum, Postpartum.

INTRODUCTION

Preeclampsia is a severe pregnancy-specific disorder with an incidence that has been reported to be approximately 5–8% (1). It is a syndrome defined by hypertension and proteinuria that also may be associated with myriad other signs and symptoms and often with subnormal fetal growth (2,3).

There is evidence that oxidative stress is increased in normal pregnancies. Serum levels of oxidative stress-related molecules have been found to be higher in pregnant than in non-pregnant women (4). However, increased serum antioxidant capacity and a gradual favoring of antioxidant activity over oxidative stress occur as normal pregnancy advances (5). In preeclampsia, several important antioxidants are markedly decreased, leading to an imbalance between the oxidants, for example, reactive oxygen species and the capacity of antioxidants to prevent oxidative damage (6,7).

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MATERNO-FETAL MEDICINE

The implication of second-trimester amniotic fluid TNF-alpha, cytochrome C and cell death nucleosomes in the prediction of preterm labor and/or premature rupture of membranes

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Abstract
Aim The multifactorial pathway leading to preterm labor possibly includes the implication of apoptosis. This study aimed to clarify the role of amniotic fluid apoptotic molecules (TNF-alpha, cytochrome C and cell death nucleosomes) at midtrimester as possible predictors of preterm labor (PTL) and/or premature rupture of membranes (PROM).
Method In this case–control study, comprising 360 women undergoing genetic amniocentesis and out of whom 38 delivered preterm and 18 out of the latter after PROM, the above apoptotic molecules were determined by ELISA. The 38 cases with PTL and 18 cases with PROM were matched for age with 38 and 18 respective controls delivering at term, and the levels of apoptotic molecules were compared.
Results Cell death nucleosome levels were found to be significantly associated with preterm delivery. Specifically, for every unit increase in nucleosomes, women were on average 0.2% more likely to deliver preterm (OR: 1.002, CI: 1.0–1.003, $p = 0.018$). In contrast, such an association was not found concerning the other two apoptotic molecules (TNF-a and Cytochrome C).
Conclusion Second-trimester amniotic fluid cell death nucleosomes' levels are significantly associated with preterm delivery and could possibly serve as predicting markers.

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Keywords Apoptosis · Cytokines · Preterm delivery

Introduction

The etiology of preterm delivery remains still unknown [1]. Several theories have been developed to explain its pathophysiology. Among them subclinical intraamniotic infection activating the cytokine signaling system [2–9], as well as the 'placental clock' theory [10–12] have been suggested. Another pathogenetic mechanism of preterm delivery implicates the pathway of apoptosis [13, 14].

Apoptosis is defined as the process of programmed cell death characterized by cell membrane changes, as well as nuclear and chromosomal DNA fragmentation [15]. Cell signals such as toxins, nitric oxide and cytokines are involved in apoptosis controlling functions (e.g. homeostasis and ischemia). Intracellular signaling is a common response to stress. As preterm labor is considered to result either from inflammation or stress processes, apoptosis could be implicated in PTL pathway. Moreover, programmed cell death of the fetal membranes might explain PROM. In this respect, it has been shown that fetal membranes from term vaginal deliveries present a zone of weakness exhibiting characteristics of apoptosis and remodeling [16]. More specifically, such a weak zone presents either paracervically in cases with artificial rupture of membranes or adjacent to the tear line in cases with spontaneous rupture of membranes showing characteristic remodeling and apoptosis findings including elevated matrix metalloproteinase 9, elevated (ADP-ribose) polymerase cleavage and decreased tissue inhibitor of matrix metalloproteinase 3 [16].

Tumor necrosis factor (TNF-alpha) is a cytokine involved in systemic inflammation and stimulation of acute

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ORIGINAL ARTICLE

Pancreatic injury after major hepatectomy: a study in a porcine model

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Abstract
Purpose The aim of this study was to investigate the pathophysiology of pancreatitis after major hepatectomy.
Methods The study used ten female pigs. Three served as sham animals (sham group) and were killed after laparotomy to obtain normal tissue samples. Seven animals were subjected to major hepatectomy (70–75%), using the Pringle maneuver for 150 min, after constructing a portacaval side-to-side anastomosis (hepatectomy group). Duration of reperfusion was 24 h.
Results Pancreatic tissue sampled 24 h after reperfusion had increased necrosis and edema in comparison to sham group and to tissue sampled at 12 h. Tissue malondialdehyde (MDA) did not differ significantly between samples at 12 and 24 h but was increased in the hepatectomy group in comparison to sham animals. Percentage increase in portal MDA content during reperfusion was greater at 12 h of reperfusion in comparison to the increase after 24 h. Portal pressure increased significantly after 12 h of reperfusion. Serum amylase and C-peptide increased during reperfusion in comparison to baseline levels.
Conclusions The findings suggest that intraoperative portal congestion is not the only cause of the development of pancreatitis after major hepatectomy. The oxidative markers suggest that reactive oxygen species produced during vascular control may be responsible as well.

Keywords Pancreatitis · Pancreatic injury · Major hepatectomy · Portacaval shunt

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Introduction

The growing need for massive resections of the liver for various causes (i.e., primary or metastatic cancer, transplantations) and the improvement of liver surgery techniques has led to aggressive surgical intervention of the organ [1, 2]. However, morbidity and mortality exceed 40 and 5%, respectively [3]. The most common postoperative complications are bleeding, abdominal sepsis, postoperative liver failure, and multiple organ dysfunction syndrome [4]. The latter two complications are thought to result from direct inflammatory damage to the remaining hepatic volume and to other organs. There is ample evidence indicating that this damage is a result of cytokine and reactive

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Antepartum and postpartum maternal plasma levels of E-selectin and VE-cadherin in preeclampsia, gestational proteinuria and gestational hypertension

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Abstract

Objective. To investigate the alterations of maternal antepartum and postpartum plasma levels of sE-selectin and VE-cadherin in normotensive pregnant women, women with preeclampsia (PE), gestational hypertension (GH), and gestational proteinuria (GP).

Methods. A total of 37 pregnant women were included in the present study; 12 with PE, 10 with GH, 5 with GP, and 10 controls. sE-selectin and VE-cadherin levels were assessed in maternal plasma at three periods; before delivery, 3–6 days after delivery, and 12–14 weeks postpartum.

Results. Women with severe preeclampsia (SPE) and GP had significantly higher plasma sE-selectin levels as compared to controls in all three periods of sampling. In the GH group, sE-selectin levels did not differ from controls. During the study, even after 12 weeks postpartum, the plasma sE-selectin levels remained unchanged in all preeclamptic groups (PE, GH, and GP). There was no difference in VE-cadherin levels between women with preeclampsia (PE, GH, and GP) and normal pregnancies.

Conclusions. We found no changes in VE-cadherin levels in preeclamptic groups. Increased antepartum and postpartum levels of sE-selectin in women with SPE and GP suggest that endothelial dysfunction may be one of the key processes in the pathogenesis of PE and the underlying mechanism, as well, that links PE with cardiovascular disease in later life. GP, also, appears to be a mild variant of PE.

Keywords: *sE-selectin, VE-cadherin, endothelial dysfunction, preeclampsia, gestational proteinuria*

Introduction

Preeclampsia (PE) is a pregnancy specific disorder of unknown etiology, which occurs in 5–7% of pregnancies and remains the major cause of maternal and fetal mortality and morbidity [1]. It is defined as new onset, persistent hypertension after 20 weeks of gestation in association with proteinuria, which progresses to a systemic hypoperfusion of multiple maternal organs [2]. However, the pathogenesis of this disorder has not been yet fully elucidated. It has been proposed that inadequate trophoblast invasion of the uterine spiral arteries is one mechanism thought to lead to placental insufficiency, which then releases factors, such as oxygen-free radicals and lipid peroxides, that adversely influence the maternal vascular endothelium [3]. Furthermore, the products of oxidative stress are intrinsically pro-inflammatory [4], leading to a more generalized inflammatory reaction, involving peripheral blood leukocytes and the clotting and complement system.

The regulation of endothelial cell contacts is of central importance for the barrier function of the blood vessel wall and for the control of leukocyte extravasation. Intercellular junctions formed by cell adhesion molecules (CAMs) are the major structural determinants of endothelial permeability and stability. Among the most currently known mechanisms involving in the stability of endothelial junctions are the vascular endothelial cadherin (VE-cadherin) [5] and the CAMs, such as selectins (E-, P- and L-selectin), integrins, and members of the immunoglobulin gene superfamily [i.e. vascular cell adhesion molecule-1 (VCAM-1) and intracellular adhesion molecule-1 (ICAM)] [6].

Soluble forms of the CAMs may be released to the circulation and increased serum levels of these molecules may indicate endothelial activation/dysfunction in preeclamptic women [7–10]. Although there are many studies for the serum levels of E-selectin in preeclamptic women, literature lacks data about E-selectin in pregnant women with gestational proteinuria (GP), which is believed to be a mild form of PE but has not been yet studied in depth [11,12]. Furthermore, the maternal plasma levels of E-selectin have never been measured again postpartum, when, as expected, the harmful effects of PE are minimal or totally absent.

As far as VE-cadherin is concerned, there are many studies demonstrating down-regulation of this important transmembrane adhesion molecule in the placentas of preeclamptic

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ORIGINAL ARTICLE

Sp1 collagen I A1 polymorphism in women with stress urinary incontinence

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Abstract
Introduction and hypothesis Stress urinary incontinence (SUI) is in part attributed to qualitative and quantitative changes in connective tissue of the urogenital tract. We examined the association of collagen type I a1 (COLIA 1) Sp1 polymorphism with the risk of SUI.
Methods Forty-five postmenopausal women suffering from urodynamically verified SUI (study group) were compared to 45 healthy volunteers (control). DNA was extracted from peripheral blood. The genotyping concerning the type 1 a1 collagen gene Sp1 polymorphism was performed with polymerase chain reaction.
Results The polymorphic T allele was overrepresented in the SUI patients (63.2% versus 36.8%, $p=0.016$). Odds ratio for SUI in women harboring the T allele was 2.19 (95% CI 1.149–4.176) compared to women with the wild-type genotype.
Conclusions The COLIA1 Sp1 polymorphism is associated with increased prevalence of stress urinary incontinence in postmenopausal women.

Keywords Collagen I A1 gene · Sp1 polymorphism · Stress urinary incontinence

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Introduction

Urinary incontinence and pelvic floor disorders are frequent problems among women, with a serious impact on health and quality of life. Urinary incontinence is the involuntary loss of urine. It affects about 30% of the female population. Among the kinds of incontinence (stress, urge, and mixed urinary incontinence), stress urinary incontinence (SUI) is the most frequent, with a prevalence of 50%. Urge and mixed type incontinence prevalence are 11% and 36%, respectively [1].

The prevalence of SUI is significantly increased after menopause, particularly in women with coexisting risk factors such as vaginal and instrumental deliveries, multiparity, obesity, and metabolic syndrome [2]. SUI can be attributed to insufficient bladder base support and neck or urethral sphincter dysfunction [3]. Continence also depends on the adequate function of the muscles of the pelvic floor as well as the connective tissue supporting structures of the paraurethral fascia and pubocervical ligaments [4]. Recent studies show an association between qualitative and quantitative changes in connective tissue and the presence of genuine SUI [5]. The main structural protein of connective tissue is type 1 collagen. The quality and organization of collagen fibers can affect the tensile strength of the pubocervical fascia which provides support to the bladder neck and base [6, 7].

Collagen type1 is a protein consisting of two a1 (I) and one a2 (I) chain {a1 (I)2a2(I)} [8]. The expression of genes encoding the collagen a1 chain (COLIA1) and a2 chain (COLIA2) is coordinated and efficiently regulated. Polymorphisms of the COLIA1 gene can affect its expression rate [9]. COLIA1 Sp1 is a single nucleotide polymorphism at the regulatory region of the COLIA 1, where guanine is substituted by thymine at the first intron of the gene. This

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Antepartum and Postpartum Serum Heme Oxygenase-1 Levels in Preeclamptic and Normotensive Pregnant Women

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Abstract. *Aim: To determine antepartum and postpartum serum heme oxygenase-1 (HO-1) levels in pre-eclamptic (PE) and normotensive pregnant women and to investigate the relationship between HO-1 levels and severity of PE. Patients and Methods: Ten normotensive women were compared to 9 women with mild PE and 12 women with severe PE. Serum HO-1 levels were measured at 30-34 gestational weeks and 12-14 weeks postpartum. Results: The severe PE group had significantly higher serum HO-1 levels antepartum compared to the mild PE and normotensive groups (5.50±1.54 vs. 3.04±0.72 ng/ml, p=0.0003, and 5.50±1.54 vs. 3.12±1.57 ng/ml, p=0.002, respectively). Serum HO-1 levels decreased significantly postpartum in the normotensive group only (3.12±1.57 vs. 2.00±0.97 ng/ml, p=0.0005). In the severe PE group, HO-1 levels antepartum were positively correlated to mean blood pressure (r=+0.79, p=0.004). Conclusion: Severe PE is associated with elevated serum HO-1 levels both antepartum and postpartum, suggesting a key role of chronic oxidative stress in the pathogenesis of PE and the endothelial dysfunction of these patients later in their life.*

Pre-eclampsia (PE) is a severe obstetric complication which usually manifests by maternal hypertension and proteinuria, progressing to a systemic hypoperfusion of multiple maternal organs and often accompanied by subnormal fetal growth (1, 2). PE contributes significantly to maternal and perinatal morbidity and mortality. Maternal and perinatal outcomes depend on the gestational age at time of the disease, severity of disease, quality of management and presence of pre-existing medical disorders (3-5).

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Key Words: Heme oxygenase-1, pre-eclampsia, mean blood pressure.

Women in normal pregnancy have an increase in oxidative stress and lipid peroxidation, when compared with non-pregnant women. However, increased serum antioxidant capacity and a gradual favoring of antioxidative activity over oxidative stress and lipid peroxidation occur as normal pregnancy advances (6). Pre-eclamptic women have an excessive increase in lipid peroxidation (7) and many studies reported that the levels of several important antioxidants are markedly increased (8-10).

Recently a new enzymatic substance, the enzyme heme oxygenase (HO-1) has been implicated as contributing factor to the antioxidant capabilities in several organ systems. The HO-1 protein is localised in placental tissue and has been shown to be involved in the maintenance of uterine quiescence throughout gestation, regulation of hemodynamic control in uterus and placenta, regulation of the apoptotic and inflammation cascades in trophoblast cells and the maintenance of a balance of the oxidant–antioxidant status within the placental tissues (11).

The potential role of HO-1 in the pathogenesis of PE has been studied, but the results of these studies are conflicting. Both an increase (12) and a reduction (13-15) of HO-1 protein expression in placentas from PE have been reported. Given that the level of HO-1 in maternal circulation has not been studied, limited knowledge exists regarding serum activation of HO-1 in preeclamptic women and whether or not this activity is altered after parturition.

The aim of the present study was to determine maternal serum HO-1 levels during pregnancy and postpartum, both in normal women, as well as in women with PE, and to investigate the relationship between maternal serum HO-1 levels and severity of PE.

Patients and Methods

Study groups. The study population consisted of 31 *primigravidas* women between 30 and 34 weeks of gestation. All women were normotensive at the booking visit and signed an informed consent form prior to inclusion into the study. Group A consisted of 10 healthy pregnant women who remained normotensive throughout their

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Mid-trimester Amniotic Fluid Interleukins (IL-1β, IL-10 and IL-18) as Possible Predictors of Preterm Delivery

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Abstract. *Aim: Strong evidence implicates chronic intra-amniotic inflammation in the etiology of preterm delivery. The purpose of this study was to determine whether amniotic fluid IL-1β, IL-10 and IL-18 concentrations in women undergoing mid-trimester amniocentesis can identify those at risk for preterm labor or preterm rupture of membranes. Patients and Methods: A case-control study was conducted to compare mid-trimester concentrations of amniotic fluid IL-1β, IL-10 and IL-18 in women delivering at term or preterm. Out of 362 women included in the study, 38 presented with preterm labor. Thirty-eight women with term delivery, matched for chronological and gestational age served as controls. Women with abnormal fetal karyotypes or major anomalies were excluded. IL-1β, IL-10 and IL-18 concentrations were determined by ELISA. Conditional logistic regression was applied in the statistical analysis. Results: IL-1β was found to be positively and significantly associated with preterm delivery. Specifically, for every unit increase in IL-1β, women were on average 7.2 (OR: 7.2, CI: 1.94-26.77, p=0.003) times more likely to deliver preterm. IL-18 levels as well as gender were significantly associated with preterm delivery. Specifically, for every unit increase in IL-18, women were on average 1% less likely to have a preterm delivery (OR: 0.99, CI: 0.98-0.99, p=0.04). On the other hand, IL-10 was not significantly associated with preterm delivery. Conclusion: Mid-trimester IL-1β concentrations are positively associated with preterm delivery. Therefore, IL-1β, determined on the occasion of mid-trimester amniocentesis could possibly serve as a marker of preterm delivery. In contrast, IL-10 and IL-18 concentrations are not elevated in mid-trimester amniotic fluid and probably cannot serve this purpose.*

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Key Words: Preterm labor, premature rupture of membranes, cytokines, prediction, interleukins, amniotic fluid.

The incidence of preterm labor varies from 7-12.8% of deliveries (1, 2) and accounts for 75% of perinatal mortality and morbidity (*e.g.* blindness, deafness, developmental delay, cerebral palsy and chronic lung disease (3, 4). Although the etiology of preterm birth is multifactorial (5), the impact of intrauterine inflammation/infection is pronounced, being implicated in nearly 30% of cases (6). Inflammation/infection-associated preterm delivery is characterized by increased amniotic fluid concentrations of various cytokines (including IL-6, TNFα, ITAC, ADAM-8, beta-defensins) (7-11).

Interleukin 1-β (IL-1β) is a 17 kDa cytokine (12), produced by macrophages, monocytes and dendritic cells as a rapid response to bacterial antigen stimuli, and is a characteristic mediator of inflammation (13). IL-10 is an 18-kDa cytokine (14), capable of inhibiting the production of cytokines by activated Th2 cells (15). IL-18 shows structural homology with the IL-1 family of cytokines (16) and is synthesized by macrophages, monocytes, keratinocytes, and epithelial cells (17).

In this study, it was hypothesized that IL-1β, IL-10 and IL-18 might be implicated in the preterm delivery pathway and therefore, they could serve as possible predictors of the former. In this respect, their amniotic fluid concentrations were determined on the occasion of genetic amniocentesis and correlated them with preterm labor and/or premature rupture of membranes.

Patients and Methods

This was a prospective matched case-control study, performed in collaboration of the Second Department of Obstetrics and Gynecology, University of Athens and the Department of Fetal Medicine of 'LITO' Maternity Hospital in Athens, during the period September 2005 - December 2006. The study population consisted of Greek women with singleton pregnancies who

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Antepartum and postpartum maternal plasma levels of E-selectin in pre-eclampsia, gestational proteinuria and gestational hypertension

Dear Editors,

Pathophysiologic features of pre-eclampsia (PE) suggest that generalized endothelial cell damage and dysfunction are the major features of the disease. E-selectin is a cell adhesion molecule and its increased expression indicates endothelial cell activation [1]. In this pilot study, we have investigated alterations of maternal antepartum and postpartum plasma levels of soluble(s) E-selectin in normotensive pregnant women and women with PE, gestational hypertension (GH) and gestational proteinuria (GP). We further sought to determine whether changes in plasma levels of sE-selectin from antepartum to postpartum would correlate with the postpartum regression of PE.

A total of 34 pregnant women were included in the present study: 12 with PE (seven with severe PE and five with mild PE), 10 with GH (five with severe GH and five with mild GH), five with GP and seven controls. sE-selectin levels were assessed in maternal plasma at three periods: before delivery, 3–6 days after delivery and 12–14 weeks postpartum. Cases complicated by chronic hypertension, chronic renal disease, autoimmune disorders, inflammatory conditions, diabetes mellitus, or taking drugs other than vitamins were not included in the study. Control patients were matched with those with PE, GH and GP for maternal age, gestational age at delivery and gestational age at blood sampling.

Statistical analysis was performed using SAS for Windows version 9.1. Although the sample sizes were relatively small, comparisons among the various groups were performed using analysis of variance (ANOVA) with the Bonferroni correction method for post hoc analysis, since the Kolmogorov–Smirnov test for normality revealed no major deviations from normality. Mixed model analysis for repeated measurements was performed to assess the evaluation of sE-selectin levels at specified times (before delivery, after delivery and 12–14 weeks postpartum).

We found that women with severe PE and GP had significantly higher plasma sE-selectin levels as compared with controls in all three periods of sampling. In the GH group, especially in women with severe GH, sE-selectin levels were also higher than in the control group, but this trend did not reach statistical significance. During the study, there was a significant reduction in the plasma sE-selectin levels of the control group from the time before delivery until 12–14 weeks postpartum, whereas in the PE, GH and GP groups there was an increase which did not reach statistical significance (Table 1).

Although there are many studies on the serum levels of E-selectin in pre-eclamptic women [1,2], data are lacking about E-selectin in pregnant women with GP, which is believed to be a mild form of PE but has not been yet studied in depth [3,4]. Furthermore, maternal plasma levels of E-selectin have never been measured postpartum, when, as expected, the harmful effects of PE are minimal or absent. These two points are the foundation of our study.

The decision to include women with GP in our study was taken after the observation that this group had worse perinatal outcomes compared with normal pregnancies (more neonates with intrauterine growth retardation). It is interesting that sE-selectin levels of women with GP were significantly higher as compared with controls, contrarily to the sE-selectin levels of hypertensive women. That may imply that women with GP are more likely to progress to PE than women with isolated hypertension, suggesting that both clinical presentations probably share a common underlying pathophysiologic mechanism [4]. We have also observed that while high blood pressure in the PE or GH group persisted for 20–30 days after delivery, proteinuria was more persistent in the PE or GP group and in some cases receded only 8–10 weeks after delivery. However, neither hypertension nor proteinuria was observed in any of the study groups 12–14 weeks postpartum.

Table 1
Plasma sE-selectin levels of women with normal pregnancies (NP), preeclampsia (PE), gestational hypertension (GH), gestational proteinuria (GP) in three periods of sampling.

Category	sE-selectin levels (ng/ml) before delivery		sE-selectin levels (ng/ml) after delivery	sE-selectin levels (ng/ml) 12–14 weeks postpartum
	N	Mean ± SD	Mean ± SD	Mean ± SD
NP	7	4.91 ± 1.79[†,*]	4.03 ± 2.01	3.33 ± 1.40
Severe PE	7	11.74 ± 2.99[*]	9.92 ± 4.95[*]	13.69 ± 6.32[*]
Mild PE	5	7.51 ± 3.49	6.19 ± 3.78	9.60 ± 4.68
Severe GH	5	8.63 ± 1.76	7.71 ± 1.51	10.92 ± 2.11
Mild GH	5	6.61 ± 3.31	6.16 ± 5.07	9.36 ± 3.93
GP	5	11.20 ± 4.95[*]	8.79 ± 3.45[*]	11.13 ± 3.98[*]

Data are presented as mean ± SD.
Comparisons among the various groups were performed using analysis of variance (ANOVA) with the Bonferroni correction method for post hoc analysis.
Differences of means of sE-selectin levels between specified periods of time (before delivery, 3–6 days after delivery and 12–14 weeks postpartum) were performed with Mixed model analysis for repeated measurements.
[†] $p = 0.03$ before delivery vs. after delivery.
[*] $p < 0.001$ before delivery vs. 12–14 weeks postpartum.
[*] $p < 0.05$ vs. normal pregnancies.

The other interesting aspect of our study was that no reduction, but rather a trend for higher plasma levels of sE-selectin, was noticed 12–14 weeks after delivery in women of all pre-eclamptic groups (PE, GH and GP), while a significant reduction was observed in normal pregnancies. This result firstly implies that endothelial dysfunction remains even after the 12th week postpartum, by which time, according to the definitions of the disease, PE is expected to resolve. Secondly, the trend for higher sE-selectin levels at the 12th week postpartum implies that the endothelial stimulation, which has been triggered during the pre-eclamptic pregnancy, progresses into endothelial dysfunction, a procedure that cannot be stopped even if the initial trigger, in our case PE, has been resolved. The endothelial dysfunction that persists might be the underlying mechanism that links PE with later cardiovascular disease (CVD) [5]. The increased levels of sE-selectin might reflect an increased sensitivity of the previously pre-eclamptic woman to develop CVD in the future, especially when other risk factors co-exist, such as obesity, abnormal lipid metabolism or insulin resistance.

We acknowledge that our conclusions are limited by the small numbers in our studies.

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Clinical Study

Maternal Serum Levels of TNF-Alpha and IL-6 Long after Delivery in Preeclamptic and Normotensive Pregnant Women

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Aim. To evaluate maternal TNF-alpha and IL-6 plasma levels in normotensive pregnant women, women with preeclampsia, and to examine the temporal changes in their levels from theantepartum to the postpartum period correlated with the regression of preeclampsia. *Method.* A prospective study was performed in the 2nd Department of Obstetrics and Gynecology, University of Athens. Blood samples were obtained: (1) antepartum at the time of clinical diagnosis of the syndrome, 2. 12-14 weeks postpartum. *Results.* No statistically significant differences were found in IL-6 levels, whereas a difference was found in TNF-alpha levels between preeclamptic and controls in antepartum period (0.80 pg/ml versus 0.60 pg/ml, P : .04). Long after delivery, TNF-alpha levels were significantly higher in preeclamptic compared to normotensive controls (0.86 pg/ml versus 0.60 pg/ml, P : .004). No difference was observed in TNF-alpha before and after delivery in both groups. No difference was noticed in IL-6 levels in women of normotensive group long after delivery compared to that before delivery. Long after delivery IL-6 levels were statistically significant higher in preeclamptic women compared to normal controls (3.53 ± 0.52 pg/ml versus 1.69 ± 0.48 pg/ml, P : .02). *Conclusion.* Preeclamptic women remain under a status of increased inflammatory stress up to 12-14 weeks postpartum despite the fact that all the other signs of preeclampsia are resolved.

1. Introduction

Although there is no systemic inflammation during pregnancy, circulating cytokines are found to be elevated in maternal plasma [1]. More specifically, normal pregnancy is characterized by local inflammatory response which leads to local production of proinflammatory cytokines that could be found in the systematic circulation. The presence of such cytokines in the systematic circulation might secondarily lead to subclinical systemic inflammatory response. In preeclampsia, a similar but exaggerated response occurs [2]. Preeclampsia is a severe pregnancy-specific disorder with an incidence which has been reported to be approximately 5–8% [3]. It is a syndrome defined by hypertension and proteinuria that also may be associated with myriad other signs and symptoms and often with subnormal fetal growth [4, 5]. The mechanisms responsible for the pathogenesis of preeclampsia have not yet been clearly identified, but reduced uterine perfusion and placental ischemia are an important initiating event in this disorder, and inflammatory cytokines are thought to link placental ischemia with cardiovascular and renal dysfunction symptoms seen in this disorder [6].

Maternal serum levels of IL-6 and TNF-alpha play a significant role in pathogenesis of preeclampsia [7]. TNF-alpha is produced by monocytes, induces apoptosis, and inhibits proliferation of trophoblast cells in preeclampsia [8]. The fact that higher circulating levels of TNF-alpha were observed in preeclampsia than in gestational hypertension suggests an association with disease severity [9]. Moreover, significantly increased soluble TNF-alpha receptors are found in the plasma of patients with preeclampsia [10]. Soluble TNF-alpha receptors bind with circulating TNF-alpha leading to a decrease of the ligand's availability [10]. At least two different views on the role of soluble receptors of TNF-a in systemic circulation have been expressed in the literature: (a) increased levels of TNF-α soluble receptors in systemic circulation may indicate either increased expression of the receptors on the cells' surface, and therefore the increased sensitivity and responsiveness of these cells to elevated levels of TNF-a, as it has been shown in the preeclamptic women

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IRON CHELATION FOR AMELIORATION OF LIVER ISCHEMIA-REPERFUSION INJURY

Nikolaos Arkadopoulos,[1] Constantinos Nastos,[1] Konstantinos Kalimeris,[2] Emmanuil Economou,[3] Kassiani Theodoraki,[4] Evangelia Kouskouni,[3] Agathi Pafiti,[5] Georgia Kostopanagiotou,[2] and Vassilios Smyrniotis[1]

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□ *Liver resections are frequently associated with significant ischemia-reperfusion (I-R) injury of the liver remnant. The aim of this study was to investigate whether deferoxamine (DFO) can ameliorate I-R injury during major hepatectomies performed under vascular exclusion of the liver in a porcine model. Twelve female domestic pigs were divided into control (n = 6) and DFO treatment (n = 6) groups and subjected to 150 min. liver ischemia followed by 70% hepatectomy and 24 hours reperfusion. Pigs in the DFO group received a continuous intravenous infusion of 100 mg/kg DFO. Liver remnant injury was evaluated by liver function tests, hepatic histology as well as serum and liver tissue malondialdehyde (MDA) concentrations. Deferoxamine-treated animals had reduced total bilirubin, γ-glutamyl transferase and ammonia levels as well as hepatocyte necrosis and oxidative injury. In a subsequent randomized clinical trial using DFO for I-R protection during major liver surgery, preliminary results revealed amelioration of hepatocellular damage, oxidative and inflammatory serum markers and apoptotic response in liver remnant biopsies.*

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Measurable serum markers of oxidative stress response in women with endometriosis

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Objective: To evaluate the hypothesis of increased systemic oxidative stress in patients with endometriosis.
Setting: Tertiary care university hospital.
Design: Cross-sectional study.
Patient(s): Sixty-six women of reproductive age undergoing laparoscopy.
Intervention(s): All women were investigated for endometriotic foci during laparoscopy. Forty-five women had laparoscopically and histologically confirmed endometriosis, and 21 women did not have endometriosis.
Main Outcome Measure(s): Four markers of oxidative stress were assessed in the serum of each patient: heat shock protein 70 (HSP70), HSP70b′, thioredoxin (TRX), and ischemia-modified albumin (IMA).
Result(s): Mean serum HSP 70b′ level was higher in patients with endometriosis compared with controls (0.178 ng/mL, SD 0.103, and 0.135 ng/mL, SD 0.014, respectively). The disease stage did not affect HSP70b′ levels. Heat shock protein 70, IMA, and TRX levels did not differ between patients with endometriosis and controls. Women with a history of arterial hypertension had higher mean IMA levels compared with women with normal blood pressure independently of the presence of endometriosis (106.7 [SD 25.4] U/mL and 85.0 [SD 11.5] U/mL, respectively).
Conclusion(s): Endometriosis is associated with increased systemic oxidative stress. The implication of increased systemic oxidative stress in disease progression or the association with other oxidative stress–related pathologic conditions needs to be addressed in further studies. (Fertil Steril® 2009;91:46–50. ©2009 by American Society for Reproductive Medicine.)

Key Words: Oxidative stress, endometriosis, heat shock proteins, ischemia-modified albumin, thioredoxin

Endometriosis is characterized by the presence and growth of endometrial tissue outside the uterine cavity and affects approximately 5% to 15% of women of reproductive age (1, 2). Oxidative stress has been implicated in the pathogenesis of endometriosis. Oxidative stress occurs when reactive oxygen species (ROS)—intermediaries of normal oxygen metabolism— are produced at a rate faster than the endogenous antioxidant defense systems can neutralize (3). Erythrocytes, macrophages, apoptotic endometrial tissue, and cell debris transplanted into the peritoneal cavity by menstrual reflux have been suggested as potential inducers of oxidative stress in endometriosis (4). Reactive oxygen species may alter morphologic and functional properties of endothelial cells, including permeability and adhesion molecule expression, thus contributing to the propagation of the inflammatory process (5).

Heat shock proteins (HSPs) are intracellular proteins induced to protect cells from various insults during periods of stress caused by infection or inflammation of other origin. Major functions of HSPs are to reverse polypeptide unfolding and to prevent protein aggregation (6). Heat shock protein 70 is the major representative of HSPs, comprising a 70 kDa polypeptide that is highly conserved throughout evolution (7). Heat shock protein 70B′ was cloned by Leung et al. (1990) as a novel HSP gene and represents a strictly stress-inducible member of the HSP family (7–9).

Thioredoxin (TRX) is a thiol oxidoreductase that regulates cellular redox status and is released from cells in response to oxidative stress (10, 11). Thioredoxin can protect against oxidative stress–induced cell injury or inflammation directly via antioxidant effects and indirectly by protein-protein interaction with signaling molecules (12). The cytosolic TRX1 and the truncated TRX80 are released from cells to the circulation and may serve as valid markers of systemic oxidative stress (13).

Ischemia-modified albumin (IMA) measured by the albumin cobalt binding test was used originally as a marker of myocardial ischemia (14). Currently, however, IMA is regarded as a marker of oxidative stress related to ischemia-reperfusion in any organ, because it is found elevated in various clinical entities associated with oxidative stress, such as systemic sclerosis (15) and ischemic stroke (16).

Although accumulating evidence exists that oxidative stress is increased locally at sites of endometrial implants, little is known about the systemic oxidative stress status in

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stress condition, involved in both endometriosis and preeclampsia.

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Reply of the Authors:

We would like to thank Dr. Montagnana and her colleagues for their kind comments on our work (1). Ischemia modified albumin (IMA) is a new promising biomarker reflecting myocardial ischemia, which increases early in the process of acute coronary events and may thus contribute to an early and accurate diagnosis (2). Beyond ischemic cardiac events, however, IMA may increase in any condition associated with ischemia. Pregnancy per se is associated with increased circulating IMA levels. First trimester circulating IMA is significantly higher compared to nonpregnant controls, possibly due to ischemia induced by trophoblast development (3). Furthermore, fetal IMA from complicated deliveries is 50% higher compared to uneventful deliveries and may increase by more than 300% in cases of severe fetal hypoxia, serving thus as a biomarker of fetal distress (4). Preeclampsia is a severe obstetric complication posing serious threats to both mother and fetus. We read with great interest the results of Dr. Montagnana and her colleagues showing that preeclamptic women have higher circulating IMA compared to normotensive women. A clinical research protocol is currently in progress in our Department evaluating among other factors circulating IMA in preeclamptic women stratified by the severity of the hypertensive disease. The aim of the protocol is to identify circulating biomarkers that may serve as early diagnostic tests predicting subsequent development of hypertensive disease in pregnancy.

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Successful pregnancy after four-step hysteroscopic technique for the treatment of atypical polypoid adenomyoma

To the Editor:

We read with interest the report by Wong et al. (1) describing the successful conservative management of a patient suffering from 5 years of primary infertility and persistent atypical polypoid adenomyoma (APA). The management proposed by the authors included a close surveillance of the APA by means of regular hysteroscopic examinations and uterine curettages (every 4–6 months) as well as the administration of the traditional Chinese herbal medication Danggui (*Angelica sinensis*), which acts as an ovulation inducer. Given the unfavorable outcome of the first pregnancy, low-dose aspirin was also administered to the patient as soon as she achieved her second pregnancy.

Recently, we have reported an innovative hysteroscopic technique for the treatment of APA in a 35-year-old infertile woman desiring pregnancy (2). This technique includes four steps, each one being characterized by a pathological analysis: [1] the removal of the APA, [2] the removal of

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Circulating levels of atherogenesis-associated adipocytokines and apoptotic markers are differentially influenced by hormone therapy, tibolone and raloxifene in healthy postmenopausal women

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Key words: HORMONE THERAPY, TIBOLONE, RALOXIFENE, ADIPOCYTOKINES, APOPTOSIS, POSTMENOPAUSE

ABSTRACT

Objective Estrogen agonist compounds may exert cardioprotective activity by modulating adipocytokine concentration and apoptosis. The objective of this study was to evaluate the effects of hormone therapy, tibolone and raloxifene on the serum adipocytokines resistin and adiponectin as well as on circulating markers of receptor-mediated apoptosis.

Design Randomized, open-label, intervention study in the Menopause Clinic of a University Hospital.

Methods One hundred healthy postmenopausal women were randomized to the following groups: conjugated equine estrogens 0.625 mg (CEE) ($n=16$); 17β-estradiol 1 mg plus norethisterone acetate 0.5 mg (E_2/NETA) ($n=15$); tibolone 2.5 mg ($n=18$); raloxifene HCl 60 mg ($n=20$); and no treatment ($n=19$). Eighty-eight women completed the 3-month study period. Main outcome measures were levels of serum adiponectin, resistin, soluble Fas and Fas ligand.

Results Levels of serum adiponectin decreased significantly in the tibolone group (baseline: 10 556.7 $\pm$ 4213.5 ng/ml; 3 months: 7856.3 $\pm$ 3450.7 ng/ml; $p=0.0001$) and increased in the CEE group (baseline: 9268.1 $\pm$ 5158 ng/ml; 3 months: 11 302.6 $\pm$ 4980.9 ng/ml; $p=0.01$). Serum resistin values increased only in the tibolone group (baseline: 2.81 $\pm$ 0.89 ng/ml; 3 months: 3.55 $\pm$ 1.31 ng/ml; $p=0.04$), while the level of Fas ligand decreased significantly in the E2/NETA (baseline: 70.4 $\pm$ 21.9 pg/ml; 3 months: 62.1 $\pm$ 18.6 pg/ml; $p=0.02$) and tibolone group (baseline: 68.2 $\pm$ 25.7 pg/ml; 3 months: 59.2 $\pm$ 21.7 pg/ml; $p=0.01$).

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Circulating leptin and ghrelin are differentially influenced by estrogen/progestin therapy and raloxifene

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Abstract

Background: Leptin and ghrelin are increasingly being recognized as cardiotropic hormones, promoting or inhibiting the atherosclerotic process, respectively. Apoptosis may be one pathway through which the actions of these hormones are mediated. Sex hormones are reported to influence the secretion and action of ghrelin and leptin.
Objective: To evaluate (1) the association of circulating ghrelin and leptin with selected markers of receptor-mediated apoptosis and (2) the effect of estrogen monotherapy, low dose estrogen–progestin therapy, tibolone and raloxifene on serum ghrelin and leptin in healthy postmenopausal women.
Methods: Eighty eight postmenopausal women aged 44–62 years were randomly allocated to daily (1) conjugated equine estrogens 0.625 mg (CEE), (2) 17β-estradiol 1 mg plus norethisterone acetate 0.5 mg (E_2/NETA), (3) tibolone 2.5 mg, (4) raloxifene HCl 60 mg or (5) no treatment. Serum markers of apoptosis sFas, Fas-ligand (Fas-L) and caspase-1 were measured at baseline. Serum leptin and ghrelin were measured at baseline and at 3 months.
Results: Body Mass Index (BMI) and estradiol levels correlated positively, while FSH correlated negatively with serum leptin (BMI: $r = 0.646$, $p = 0.005$, estradiol: $r = 0.432$, $p = 0.001$, FSH: $r = -0.401$, $p = 0.002$). Insulin levels associated positively with circulating leptin ($r = 0.394$, $p = 0.011$) and negatively with circulating ghrelin ($r = -0.401$, $p = 0.009$). Serum leptin decreased significantly in E2/NETA group (baseline: 2.882 ± 0.76 ng/ml, 3 months: 2.687 ± 0.66 ng/ml, $p = 0.043$), while it increased significantly in the raloxifene group (baseline: 2.671 ± 0.54 ng/ml, 3 months: 2.839 ± 0.47 ng/ml). Ghrelin levels decreased significantly only in the raloxifene group (baseline: 1634 ± 592 pg/ml, 3 months: 1408 ± 534 pg/ml).
Conclusion: Apoptosis may be a pathway through which leptin exerts a pro-atherogenic effect. Low dose HT may act cardioprotectively by decreasing letpin levels in healthy recently menopaused women.

Keywords: Leptin; Ghrelin; Apoptosis; Postmenopausal; Hormone therapy; Estrogen; Tibolone; Raloxifene

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The implication of obesity on total antioxidant capacity in apparently healthy men and women: The ATTICA study

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KEYWORDS
Obesity;
Antioxidant

Abstract *Background and aim:* We evaluated the association of obesity with serum total antioxidant capacity (TAC), in a population-based sample of 3042 adults. *Methods and results:* During 2001–2002 we randomly enrolled 1514 men (18–87 years old) and 1528 women (18–89 years old), from the Attica area in Greece into the study, and the sample was stratified by the age-sex distribution of the region (census 2001). Among several variables we also measured serum TAC and weight, height, waist and hip circumferences. Waist circumference greater than 102 cm for men and 88 cm for women was considered an indicator of central fat.
Methods and results: Mean waist circumference was 98 ± 13 cm in men and 84 ± 22 cm in women ($P < 0.001$), while mean hip circumference was 106 ± 28 cm in men and 103 ± 13 cm in women ($P < 0.001$). Central fat prevailed in 53% of men and 45% of women ($P < 0.001$). Male participants with central fat exhibited 5% lower TAC concentrations compared to leaner individuals (214 ± 35 vs. 226 ± 33 μmol/L, $P = 0.04$) and female participants with central fat exhibited 7% lower TAC concentrations (256 ± 38 vs. 239 ± 27 μmol/L, $P = 0.03$). Similarly, obese or overweight male participants had 6% lower TAC concentrations compared to normal weight (217 ± 33 vs. 234 ± 39 μmol/L, $P = 0.03$) and female obese or overweight participants had 10% lower TAC concentrations (226 ± 32 vs. 250 ± 30 μmol/L, $P = 0.02$) compared to the others.

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The association between pre-hypertension status and oxidative stress markers related to atherosclerotic disease: The ATTICA study

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Abstract

Background: We sought to evaluate the association between pre-hypertension status and oxidative stress markers (total antioxidant capacity (TAC) and oxidized low density lipoprotein (LDL)), in a random sample of cardiovascular disease-free adults.
Methods: The ATTICA study is a cross-sectional population-based survey that conducted in Attica region during 2001–2002. Based on a multistage and stratified random sampling, 1514 men and 1528 women (18–89 years old) were enrolled. The survey included a detailed interview; blood samples collected after 12 h of fasting and, among other clinical measurements, status of blood pressure levels was evaluated.
Results: Six hundred and fifty-three men (43%) and 535 women (35%) were defined as pre-hypertensives. Both systolic and diastolic blood pressures were inversely correlated with TAC ($p < 0.001$) and positively correlated to oxidized LDL ($p < 0.001$). Particularly, compared to normotensive subjects, pre-hypertensives had 7% lower TAC levels ($p < 0.001$) and 15% higher oxidized LDL levels ($p < 0.05$), after correcting for multiple comparisons and adjusting for age, body mass index, blood lipids, glucose, food groups consumed and other potential confounders.
Conclusions: Studying a large sample of cardiovascular disease-free adults, we revealed an association of pre-hypertension with oxidative stress markers linking to atherosclerotic process.

Keywords: Pre-hypertension; Oxidation; Atherosclerosis

1. Introduction

Several mechanisms have been suggested to describe how elevated blood pressure might confer the increased risk of coronary artery disease and stroke. Among them, structural and functional changes of the arterial wall, increased oxidative stress and accelerated atherogenesis have been implicated in hypertension [1–3]. Recently, the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure [4] suggested a new classification for borderline blood pressure levels, the "pre-hypertension". The new classification describes people with blood pressures between 120 and 139 mmHg systolic or between 80 and 89 mmHg diastolic blood pressures. This "new" category between normal blood pressure and established hypertension includes a population at high risk for developing hypertension and in which lifestyle modifications are needed [4].

The determination of antioxidative capacity is now considered as a tool in medical diagnosis and treatment of several diseases, including cardiovascular disease, cancer, diabetes mellitus and aging [5]. Total antioxidant capacity (TAC) considers the cumulative action of all the antioxidants present in plasma and body fluids and provides an integrated parameter rather than the simple sum of measurable antioxidants. There is now a wide range of evidence indicating the importance of

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Circulating chemoattractants RANTES, negatively related to endogenous androgens, and MCP-1 are differentially suppressed by hormone therapy and raloxifene

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Abstract

Background: The cardinal role of chronic inflammation in the development of atherosclerosis is increasingly being recognized. Estrogens may prevent the evolution of atherosclerosis by suppressing immune response. Furthermore, the conflicting reports on the cardiovascular effects of hormone therapy between observational and clinical trials have triggered interest on the effect of alternative therapies on the cardiovascular system.

Objective: The aim of this study was to assess the effect of estrogen, estrogen–progestin, tibolone and raloxifene therapy on circulating markers of chemotaxis in healthy postmenopausal women.

Methods: Eighty-eight postmenopausal women aged 44–62 years were randomly allocated to daily: (1) conjugated equine estrogens 0.625 mg (CEE), (2) 17β-estradiol 1 mg plus norethisterone acetate 0.5 mg (E_2/NETA), (3) tibolone 2.5 mg, (4) raloxifene HCl 60 mg or (5) no treatment. Serum monocyte chemoattractant protein-1 (MCP-1) and regulated upon activation, normal T-cell expressed and secreted (RANTES) were measured at baseline and at 3 months.

Results: Endogenous testosterone and free androgen index (FAI) correlated negatively, while SHBG correlated positively with serum RANTES (testosterone: $r = -0.27$, $p = 0.033$; FAI: $r = -0.43$, $p = 0.004$: SHBG: $r = 0.34$, $p = 0.026$). Serum MCP-1 decreased significantly in the CEE group (baseline 125.3 ± 51 pg/ml, 3 months 84.5 ± 36.1 pg/ml, $p = 0.043$), while no difference was detected between baseline and post-treatment levels in the other groups. Furthermore, a significant decrease in serum RANTES was observed at the end of 3 months only in the E2/NETA and the raloxifene group (E2/NETA baseline 8690.6 ± 3880.0 pg/ml, 3 months 6894.0 ± 1720.0 pg/ml, $p = 0.007$; raloxifene baseline 9042.4 ± 3765.6 pg/ml, 3 months 6718.1 ± 2366.2 pg/ml, $p = 0.011$).

Conclusion: Endogenous androgens may suppress chemotactic response. Postmenopausal hormone therapy and raloxifene may inhibit the expression of chemoattractant molecules and thus attenuate inflammation. The relevance of these findings in terms of clinically established caridoprotection remains to be clarified.

Keywords: MCP-1; RANTES; Estradiol; Conjugated equine estrogens (CEE); Testosterone; Postmenopausal; Atherosclerosis; Tibolone; Raloxifene

1. Introduction

Postmenopausal estrogen depletion increases the risk for cardiovascular disease (CVD). CVD is the principal cause of morbidity–mortality among postmenopausal women and is

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Intrauterine growth restriction and circulating neurotrophin levels at term

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Neurotrophins;
Intrauterine growth restriction;
Perinatal period;
Fullterm neonates;
Brain sparing effect

Abstract

Background: Intrauterine growth restricted (IUGR) fetuses are those with estimated weight < 10th customized centile, displaying signs of chronic malnutrition and hypoxia leading to brain sparing effect. Neurotrophins, [Nerve Growth Factor (NGF), Brain Derived Neurotrophic Factor (BDNF), Neurotrophin-3 (NT-3), Neurotrophin-4 (NT-4)] are important for pre- and post-natal brain development.
Aims: To investigate circulating NGF, BDNF, NT-3 and NT-4 levels in IUGR and appropriate for gestational age (AGA) fullterm fetuses and neonates (day-1 [N1] and day-4 [N4]) and in their mothers.
Study design: Prospective case control study.
Subjects: 60 mothers and their single 30 IUGR and 30 AGA fullterm fetuses and neonates.
Outcome measures: Determination, by enzyme immunoassays, of NGF, BDNF, NT-3 and NT-4 plasma levels.
Results: No statistically significant differences existed between IUGR and AGA maternal, fetal and neonatal levels of BDNF, NT-3 and NT-4. NGF was significantly higher in AGA than IUGR maternal ($p=0.007$), fetal ($p=0.01$), neonatal day 1 ($p=0.043$) and 4 ($p=0.003$) plasma, and positively correlated with the infants' centiles and birthweights. IUGR and AGA maternal neurotrophins were higher than the respective fetal and neonatal ones and no correlation with gender or delivery mode in both groups was observed.
Conclusions: In the perinatal period, circulating levels of BDNF, NT-3 and NT-4 do not differ in IUGR and AGA pregnancies, in contrast to NGF levels, which are higher in the AGA group. NGF is the only neurotrophin correlating with customized centiles and birthweights of the infants. Neurotrophin concentrations are higher in maternal plasma and do not depend on gender.

Abbreviations: AGA, appropriate for gestational age; BDNF, brain derived neurotrophic factor; IUGR, intrauterine growth restriction; MS, maternal sample; NGF, nerve growth factor; NT-3, neurotrophin-3; NT-4, neurotrophin-4; N1, neonatal day 1 sample; N4, neonatal day 4 sample; UC, umbilical cord sample.

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The Varying Patterns of Neurotrophin Changes in the Perinatal Period

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ABSTRACT: **Neurotrophins (NTs), nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), NT-3, and NT-4 are of major importance in prenatal and postnatal brain development, due to their neuroprotective action. Developmental changes alter the neuronal responsiveness to certain NTs, which subsequently are variously expressed, to properly balance their action. The following study aimed at examining the pattern of perinatal changes of the four NTs—NGF, BDNF, NT-3, and NT-4 in 30 appropriate for gestational age (AGA) full-term fetuses and neonates by determining their circulating levels at characteristic time points. This study show a gradual decrease of circulating levels of the NTs, NT-3 and NT-4 from umbilical cord (UC) to neonates day 4 (N4), while circulating levels of NGF and BDNF present the opposite pattern: an increase from UC to N4. These patterns of perinatal changes differ according to their impact on the process of neuronal development and their reaction to perinatal stress. NT3 and NT4 have been documented to act at early stages of neuronal development and to decrease after hypoxia-ischemia, while NGF and BDNF to increase. Further studies should investigate these patterns in premature or full-term infants, presenting various pathological conditions in the perinatal period.**

KEYWORDS: **neurotrophins; NT-3; BDNF; NT-4; NGF; fetus; neonate; neuronal development**

INTRODUCTION

In humans, four structurally related molecules: nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), neurotrophin-3 (NT-3) and neurotrophin-4 (NT-4) comprise the NT family,[1] a group of substances rendering neuroprotection, by reducing apoptosis and promoting survival and

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Research Communication

Elevated Circulating IL-1β and TNF-Alpha, and Unaltered IL-6 in First-Trimester Pregnancies Complicated by Threatened Abortion With an Adverse Outcome

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The purpose of the present study was to examine the profile of selected proinflammatory cytokines in maternal serum of first-trimester pregnancies complicated by threatened abortion (TACP) and its relevance to obstetric outcome. Serum levels of Th1-type cytokines interleukin-1β (IL-1β), tumor necrosis factor alpha (TNF-alpha), and Th2-type cytokine interleukin 6 (IL-6) were measured, by ELISA, in 22 women with TACP and adverse outcome at admission (group A) and compared with the corresponding levels of 31 gestational age-matched women with TACP and successful outcome at admission (group B1) and discharge (group B2) and 22 gestational age-matched women with first-trimester uncomplicated pregnancy (group C) who served as controls. Mann-Whitney U or Wilcoxon test was applied as appropriate to compare differences between groups. IL-1β and TNF-alpha were detected with significantly higher levels in group A, compared to all other groups. On the contrary, IL-6 levels were detected with no significant difference among all the other groups studied. It is concluded that in first-trimester TACP with adverse outcome, a distinct immune response, as reflected by elevated maternal IL-1β, TNF-alpha, and unaltered IL-6 levels, is relevant to a negative obstetric outcome.

INTRODUCTION

Spontaneous abortion is the loss of an intrauterine pregnancy without outside intervention before 20 weeks' gestation and can be subdivided into threatened abortion, inevitable abortion, incomplete abortion, missed abortion, septic abortion, complete abortion, and recurrent spontaneous abortion [1]. Threatened abortion refers to an intrauterine viable clinical pregnancy accompanied by an intrauterine source of painless vaginal bleeding and successful or adverse pregnancy outcome [2].

The cytokine network has been suggested to be involved with positive or negative evolution of the ongoing pregnancies [3]. Prevalence of Th2-type cytokines (secreted by T-helper 2 cells and certain antigen-presenting cells (APCs)) may be associated with successful pregnancy; whereas the dominance of Th1-type cytokines (derived from T-helper 1 cells and APCs) may be indicative of a pathological pregnancy both in experimental animals and in humans [4, 5].

IL-1β is an essential proinflammatory, Th1-type cytokine, produced by monocytes, macrophages, and epithelial cells. Its secretion leads to production of tumor necrosis factor (TNF-alpha), interferon (IFN-γ), IL-2, and IL-12 [6], uterotonic prostaglandin E_2 and/or matrix metalloproteinases by fetal membrane cells, as well as to promotion of apoptotic cell death in fetal membrane tissues [7]. Due to these properties, IL-1β may express abortogenic action. On the other side, elevated IL-1β levels may increase the likelihood of successful and complete implantation, and, during the first trimester, may also offer the fetus increased protection against microbial pathogens that were present in the uterus before the conception period, during the conception period, or in the early postconception period [8].

TNF-alpha, a Th1-type cytokine, is mainly produced by mononuclear phagocytes, natural killer cells, and antigen-stimulated T-cells. Similarly to IL-1β, TNF-alpha promotes apoptotic cell death in fetal membrane tissues [7] and activates coagulation via upregulating the novel prothrombinase, fgl2 [9]. The proinflammatory, proapoptotic, and procoagulant properties of TNF-alpha probably contribute to the widely accepted abortogenic profile of this cytokine.

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CLINICAL STUDY

Endogenous sex hormones and risk factors for atherosclerosis in healthy Greek postmenopausal women

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Abstract

Objective: To assess the association between endogenous sex hormones and risk factors for atherosclerosis in healthy postmenopausal women.
Design: Cross-sectional study in a university menopause clinic.
Methods: Serum sex hormones and lipid–lipoprotein profile, arterial pressure, homocysteine and insulin resistance, measured by the homeostasis model assessment of insulin resistance (HOMA-IR), were assessed in 598 healthy postmenopausal women not on hormone therapy.
Results: Compared with women in the lowest testosterone quartile (Q), women in the highest testosterone quartile had higher total cholesterol (Q1: 225.2±41.3 vs Q4: 246.2±38.4 mg/dl, $P<0.01$), low-density lipoprotein (LDL)-cholesterol (Q1: 146.9±37.2 vs Q4: 171.8±35.3 mg/dl, $P<0.001$), atherogenic index of plasma (AIP) (Q1: –0.224±0.238 vs Q4: –0.087±0.254, $P<0.01$), apolipoprotein B (ApoB) (Q1: 100.7±23.1 vs Q4: 113.9±23.8 mg/dl, $P<0.001$) and higher high-density lipoprotein (HDL)-cholesterol (Q1: 60.7±14.5 vs Q4: 52.9±13.0 mg/dl, $P<0.01$). Accordingly, women in the highest free androgen index (FAI) quartile had higher AIP (Q1: –0.232±0.254 vs Q4: –0.078±0.243, $P<0.001$) and ApoB (Q1: 102.4±25.5 vs Q4: 114.2±25.8 mg/dl, $P<0.01$) and lower HDL-cholesterol (Q1: 62.0±15.7 vs Q4: 51.9±11.6 mg/dl, $P<0.001$) and apolipoprotein A (Q1: 159.6±25.6 vs Q4: 147.9±24.1 mg/dl, $P<0.01$) compared with women in the lowest FAI quartile. These differences remained significant after adjustment for age, body mass index (BMI), insulin resistance and social habits. The free estrogen index (FEI) exhibited similar associations to the FAI. HOMA-IR showed an independent positive association with total testosterone (Q1: 2.00±1.36 vs Q4: 2.66±1.60, $P<0.01$), FAI (Q1: 1.70±1.12 vs Q4: 3.04±1.66, $P<0.001$) and FEI (Q1: 1.70±0.91 vs Q4: 3.08±1.77, $P<0.001$).
Conclusions: Increased androgenicity in healthy postmenopausal women is associated with an unfavorable cardiovascular risk profile. High endogenous estradiol is related to a pro-atherogenic lipid profile, an association which may, in part, be mediated by insulin resistance.

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Introduction

Cardiovascular disease (CVD) is a leading cause of death of women worldwide. While premenopausal women have a lower incidence of CVD compared with men of the same age, the incidence of the disease in women rises steeply after the age of 50 (1). The association between menopause and CVD was indicated in the Framingham cohort, where postmenopausal women had a 2–6 times greater incidence of CVD compared with premenopausal women in the same age range (2). Furthermore, premature menopause caused by bilateral oophorectomy in younger women is an established risk factor for ischemic heart disease (3). Menopause is associated with a pro-atherogenic lipid profile characterized principally by lower high-density lipoprotein-cholesterol (HDL-cholesterol), higher low-density lipoprotein-cholesterol (LDL-cholesterol) and triglyceride levels (4), central adiposity (5), increased diastolic pressure (6) and increased insulin resistance (7). Sex steroid deficiency is considered a crucial factor responsible for the menopause-associated changes in the CVD risk factor profile, and consequently for the increase in CVD risk (8).

Although the menopausal ovary does not secrete estrogens, it continues to serve as a source of androgens. To variable degrees, ovarian Δ_4 androstendione and testosterone are aromatized peripherally, mainly in the adipose tissue, to estrone and estradiol respectively, thus determining the postmenopausal endogenous estrogen milieu (9). In contrast to the established cardiovascular benefit of premenopausal sex steroids, little is known

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ORIGINAL ARTICLE

Differential Effect of Hormone Therapy and Tibolone on Lipids, Lipoproteins, and the Atherogenic Index of Plasma

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Abstract: The aim of our study was to assess the effect of various regimens and doses of hormone therapy and tibolone on the Atherogenic Index of Plasma (AIP). A total of 519 postmenopausal women attending our menopause clinic were studied in a prospective design. Women with climacteric symptoms were randomly assigned to receive 1 of the following regimens: tibolone 2.5mg, conjugated equine estrogens 0.625mg plus medroxyprogesterone acetate 5mg (CEE/MPA), 17β-estradiol 2mg plus norethisterone acetate 1mg (E_2/NETA), or 17β-estradiol 1mg plus norethisterone acetate 0.5mg (low E_2/NETA). Serum parameters were assessed at baseline and after 6 months and included total cholesterol, LDL-cholesterol, HDL-cholesterol, triglycerides, apolipoprotein A1 and apolipoprotein B. The AIP was assessed as the log (triglycerides [mmol/L]/HDL-C [mmol/L]). CEE/MPA treatment associated with lower mean LDL-C but higher mean triglyceride levels (-15.5 mg/dL $\pm$ 3.6, $P = 0.0001$; 12.6 mg/dL $\pm$ 4.8, $P = 0.01$). Furthermore, CEE/MPA treatment resulted in higher AIP levels (0.073 ± 0.021, $P = 0.001$). On the contrary, both E_2/NETA regimens and tibolone associated with lower mean triglyceride and HDL-C levels (E_2/NETA, triglycerides: -9.8 mg/dL $\pm$ 5.0, $P = 0.049$; HDL-C: -4.9 mg/dL $\pm$ 1.8, $P = 0.01$, low E_2/NETA triglycerides: -12.5 mg/dL $\pm$ 4.1, $P = 0.003$; HDL-C: -4.7mg/dL $\pm$ 1.3, $P = 0.001$; tibolone, triglycerides: -21.9mg/dL $\pm$ 2.7, $P = 0.0001$; HDL-C: -12.7 mg/dL $\pm$ 1.1, $P = 0.0001$). None of the 3 regimens had any effect on AIP. The effect of a particular regimen of hormone therapy on the lipid-lipoprotein profile differs depending on the parameter assessed. The use of unified markers such as AIP will be helpful in evaluating the overall effect of lipid-lipoprotein modulation on the cardiovascular system. In fact, the concurrent assessment of the therapy effect on both LDL-C and AIP may be more dependable in evaluating the cardiovascular impact of a given regimen.

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Key Words: hormone therapy, tibolone, atherogenic index of plasma

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In postmenopausal women estrogen deficiency has both metabolic and vascular consequences which increase the risk for cardiovascular disease (CVD). CVD is the leading cause of mortality among postmenopausal women. CVD is influenced by a cluster of factors, the balance of which determines the incidence of the disease.[1–3]

Following menopause, the beneficial effect of endogenous estrogens on lipid metabolism is lost[4] and a proatherogenic lipid-lipoprotein profile is established, which includes an increase in total cholesterol (TC), low-density lipoprotein-cholesterol (LDL-C), triglycerides (TG), and lipoprotein (a) [Lp(a)] and a minor decrease in high density lipoprotein cholesterol (HDL-C).[4–7] This proatherogenic lipid profile has been considered as contributing to the postmenopausal increase in CVD risk.[8] Observational studies have repeatedly reported that hormone therapy (HT) decreased significantly the risk for coronary artery disease (CAD).[3,9] This has been partly attributed to the HT-induced beneficial changes on the lipid-lipoprotein profile.[10] However, the clinical trials Heart and Estrogen/Progestin Replacement Study (HERS),[11] Estrogen Replacement and Atherosclerosis Study,[12] and Women's Health Initiative (WHI)[13] have failed to corroborate the results of the observational studies. Although in all 3 studies, HT associated with a significant decrease in LDL-C and an increase in HDL-C, the HERS and WHI reported a significant increase in early coronary events, whereas the ERA concluded that HT did not slow the progression of atherosclerosis. Even though the effect of HT on the vascular wall and endothelium is more important in modulating the risk for CVD compared to lipid-lipoproteins, the role of the latter should not be underestimated.[14–17] HT preparations combine different estrogens and progestins. Estrogen therapy has a more favorable effect on the lipid profile compared with estrogen-progestin.[18] However, in non-hysterectomized women, the addition of the progestin aims in preventing endometrial proliferation.[19] The type of progestin, the dose, the mode (continuous, sequential) and the route of administration may enhance, attenuate

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Soluble vascular endothelial growth factor receptor-1 in intrauterine growth restricted fetuses and neonates

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KEYWORDS
Soluble vascular endothelial growth factor receptor-1;
Angiogenesis;
Intrauterine growth restriction;
Fetus;
Neonate

Abstract

Background: Angiogenesis, a critical process for growth and development is altered in intrauterine growth restriction (IUGR). Vascular endothelial growth factor (VEGF) and its receptors VEGFR-1, soluble (s) VEGFR-1 and VEGFR-2 represent a regulatory system, essential for both physiological and pathological angiogenesis.
Aim: To study the implication of sVEGFR-1—a VEGF antagonist—in IUGR.
Study design: Prospective study.
Methods: Twenty-five IUGR and 15 appropriate for gestational age (AGA) full-term fetuses and neonates with their mothers were included in the study.
Outcome measures: sVEGFR-1 levels were determined by enzyme immunoassay in the serum of: mothers (MS), umbilical cords (UC)—representing fetal state—and neonates on day 1 (N1) and 4 (N4) of life.
Results: MS, UC, N1 and N4 sVEGFR-1 levels in IUGR were significantly higher compared to respective AGA cases ($p=0.005$, $p=0.026$, $p=0.005$ and $p=0.017$, respectively). In IUGR and AGA groups, maternal sVEGFR-1 levels were significantly higher than fetal and neonatal levels (p in all cases <0.001). The latter presented in both IUGR and AGA groups a significant decrease from UC to N4 (p in all cases <0.01). MS, N1 and N4 sVEGFR-1 levels negatively correlated with the infants' customized centiles [($r=-0.489$, $p=0.001$), ($r=-0.440$, $p=0.004$), ($r=-0.431$, $p=0.006$), respectively].
Conclusions: Higher sVEGFR-1 levels in the IUGR as compared to the AGA group possibly reflect the predominance of antiangiogenic mechanisms present in IUGR. The decrease of sVEGFR-1 levels from UC to N4 may represent ex utero initiation of growth and development and therefore, prevalence of angiogenic mechanisms.

Abbreviations: VEGF, Vascular endothelial growth factor; sVEGFR-1, Soluble vascular endothelial growth factor receptor-1; IUGR, Intrauterine growth restriction; AGA, Appropriate for gestational age; MS, Maternal serum; UC, Umbilical cord serum; N1, Day 1 neonatal serum; N4, Day 4 neonatal serum.

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ORIGINAL ARTICLE

Angiopoietin-2 in the perinatal period and the role of intrauterine growth restriction

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Abstract

Background. Angiopoietin-2, an angiogenic factor, causing destabilization and postnatal remodeling of blood vessels, is upregulated by hypoxia. We hypothesized that circulating Angiopoietin-2 levels might differ in intrauterine growth restricted and appropriate for gestational age fetuses and neonates, as the former have restricted growth and development and suffer from *in utero* hypoxia. *Methods*. This is a prospective, controlled study, including forty asymmetric, mainly due to hypertension or pre-eclampsia intrauterine growth restricted (0–9 customized centiles, corrected for gestational age, sex, maternal weight, height, ethnic group, and parity), and 20 appropriate for gestational age (42–82 customized centiles) full-term infants, as well as their mothers. Blood samples were drawn from mothers, from the doubly clamped umbilical cord (mixed arteriovenous blood, representing fetal state), and from neonates on days 1 (N1) and 4 (N4) of life (representing transition and stabilization to extrauterine life, respectively). Circulating angiopoietin-2 levels were measured by enzyme immunoassay and the statistical analysis involved *t*-test and Pearson correlation. *Results*. Angiopoietin-2 levels were significantly higher in intrauterine growth restricted cases only in N4 ($p = 0.04$). No dependence on the mode of delivery and gender was documented. *Conclusions*. These findings may suggest that intrauterine hypoxia possibly does not upregulate circulating angiopoietin-2 levels in intrauterine growth restricted fetuses and day 1 neonates; however, increased angiopoietin-2 on N4, after stabilization to extrauterine life, might signify initiation of catch-up growth-related angiogenesis and stimulation of angiogenic factors, granted that angiopoietin-2 is critically involved in postnatal vascular remodeling.

Key words: *Angiopoietin-2, intrauterine growth restriction, umbilical cord, neonate, perinatal period*

Abbreviations: *Ang-2: angiopoietin-2, IUGR: intrauterine growth restriction, AGA: appropriate for gestational age, MS: maternal serum, UC: umbilical cord serum, N1: neonatal day 1 serum, N4: neonatal day 4 serum*

Angiopoietins (Ang) play an important role in angiogenesis and are considered to be angiogenic factors. The Ang family comprises three structurally related proteins (Ang-1, 2, 3/4), which bind with similar specificity and affinity, but with opposing effects to a common endothelial cell-specific receptor tyrosine kinase, Tie2 (1–3). While Ang-1 is responsible for Tie2 activation, Ang-2 inhibits it (2).

Ang-1 possesses only weak endothelial cell mitogenic activity, causing endothelial maturation and vascular stabilization in its surrounding tissue by recruitment of pericytes and smooth muscle cells (4). On the other hand, Ang-2 is a natural antagonist of Ang-1 in endothelial cells, acting at the front of the invading vascular sprouts by blocking constitutive stabilization and maturation of vessels, allowing them to remain in a more plastic state, in which they could be more responsive to a sprouting signal (2,4–6). Thus, Ang-2 is thought to destabilize or regress blood vessels (2), permitting them to undergo remodeling (7).

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Adherence to the Mediterranean diet is associated with total antioxidant capacity in healthy adults: the ATTICA study[1–3]

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ABSTRACT

Background: Greater adherence to the Mediterranean diet has been associated with a lower incidence of cardiovascular disease and cancer.

Objective: We studied the effect of the Mediterranean diet on total antioxidant capacity (TAC) in 3042 participants who had no clinical evidence of cardiovascular disease.

Design: During 2001–2002, a random sample of 1514 men and 1528 women aged 18–89 y from the Attica area of Greece was selected. TAC was measured with an immune-diagnostic assay. Food consumption was evaluated with a validated food-frequency questionnaire, and adherence to the Mediterranean diet was assessed on the basis of a diet score that incorporated the inherent characteristics of this diet.

Results: TAC was positively correlated with diet score. The participants in the highest tertile of the diet score had, on average, 11% higher TAC levels than did the participants in the lowest tertile, even after adjustment for relevant confounders ($P < 0.01$). On the other hand, the participants in the highest tertile of the diet score had, on average, 19% lower oxidized LDL-cholesterol concentrations than did the participants in the lowest tertile ($P < 0.01$). An additional analysis showed that TAC was positively correlated with the consumption of olive oil ($\rho = 0.54$, $P = 0.002$) and of fruit and vegetables ($\rho = 0.34$ and $\rho = 0.31$, respectively; $P < 0.001$ for both), whereas it was inversely associated with the consumption of red meat ($\rho = -0.35$, $P = 0.02$).

Conclusion: Greater adherence to the Mediterranean diet is associated with elevated TAC levels and low oxidized LDL-cholesterol concentrations, which may explain the beneficial role of this diet on the cardiovascular system. *Am J Clin Nutr* 2005;82:694–9.

KEY WORDS Mediterranean diet, antioxidant capacity, oxidized LDL cholesterol, cardiovascular disease

INTRODUCTION

Several observational studies and large-scale clinical trials have provided scientific evidence that diets rich in fruit, vegetables, legumes, whole grains, fish, and low-fat dairy products are associated with a lowered incidence of various chronic diseases (1, 2). The dietary pattern that was found in the olive growing areas of the Mediterranean region (such as Greece, Spain, Italy, and France) in the late 1950s and early 1960s encompasses these dietary characteristics and has been associated with a lowered incidence of cardiovascular diseases, metabolic disorders, and several types of cancer (3–10). Many investigators have already underlined the beneficial role of this dietary pattern on lipid metabolism, blood pressure levels (1, 6, 7), and body mass index (6, 8), as well as on inflammation and coagulation processes (9).

The determination of antioxidative capacity is now considered a tool in the medical diagnosis and treatment of several diseases, including cardiovascular disease, cancer, diabetes mellitus, and aging (11). Total antioxidant capacity (TAC) considers the cumulative action of all antioxidants that are present in plasma and body fluids and provides an integrated measurement rather than the simple sum of measurable antioxidants. A wide range of evidence indicates the importance of TAC in plasma and tissues, of its modification during the development of oxidative stress, and of its feasibility as a tool for investigating the association between diet and oxidative stress (12). In addition, the oxidative conversion of LDL cholesterol to oxidized LDL cholesterol is now considered to be a key event in the initiation and acceleration of the development of the early atherosclerotic lesion, the fatty streak. Diet seems to play a fundamental role in LDL-cholesterol oxidation (13–15). In particular, high dietary intakes of β-carotene and of vitamins C and E and intakes of phenolic compounds in red wine (16) have been associated in some studies with low concentrations of oxidized LDL cholesterol. However, in most of these studies the approach has been to assess single nutrients or food items instead of dietary patterns. Recently, Martinez-Gonzalez and Estruch (17) underlined the need for randomized trials that use a whole-diet approach and not a simple antioxidant supplement to evaluate the role of the Mediterranean dietary pattern in human health. Moreover, in a recent review article, Martinez-Gonzalez and Sanchez-Villegas (5) underlined that not all components of the Mediterranean diet are protective, or at least they may not provide equal levels of protection. Thus, because food items and nutrients could have a synergistic and antagonistic effect on health outcome, the study of overall dietary patterns and not single nutrients has been suggested. In a recent

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RESEARCH COMMUNICATION

Low-Grade Systemic Inflammation Profile, Unrelated to Homocysteinemia, in Obese Children

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To investigate in prepubertal obese children (POC) the profile of chronic low-grade systemic inflammation (CLGSI) and its relation to homocysteinemia, 72 POC were evaluated for serum C-reactive protein (CRP) and amyloid A (SAA) levels, both markers of CLGSI, and plasma levels of total homocysteine (tHcy), an independent risk factor for adult atherosclerosis, in comparison to 42 prepubertal lean children (PLC). The main observations in POC were higher CRP levels compared to PLC, positive association of SAA levels to CRP levels, no association of CRP or SAA levels to tHcy levels. Thus, in POC, positively interrelated to each other, elevated CRP and unaltered SAA levels reveal a unique profile of the CLGSI, not explaining homocysteinemia-induced risk for future atherosclerosis.

INTRODUCTION

Several cardiovascular risk factors are present in obese children, among which are chronic low-grade systemic inflammation and homocysteinemia [1].

Obesity is now widely accepted as a promoter of a chronic low-grade inflammatory reaction favoring the development of atherosclerosis and cardiovascular disease [2], in both children and adolescents [3, 4] who are free of other pathological conditions. Chronic low-grade systemic inflammation, usually a persistent but more sublte than acute phase inflammatory response, can be measured by circulating C-reactive protein (CRP) and serum amyloid A (SAA) which may also have direct pro-inflammatory actions [5, 6]. They both are nonspecific acute phase reactants, primarly synthesized in liver and at least equally sensitive to reflect chronic low-grade systemic vascular inflammation [7]. Moreover, highly sensitive newer assays for CRP and SAA can now detect previously unnoticed chronic low-grade systemic inflammation [8, 9]. In obese subjects, hepatic biosynthesis and maintenance of circulating CRP and SAA is up-regulated by adipocytokines, such as interleukins 1 and 6, tumor necrosis factor A, and, for some authors, leptin, all secreted by adipose tissue [10]. However, although equally sensitive and similarly up-regulated, CRP and SAA are differentially determined in circulation. Circulating CRP levels are determined by both environmental factors and a moderate but significant degree of heritability while circulating SAA levels are determined exclusively by environmental factors [11, 12]. Moreover, several studies describe differential, population-specific, local or systemic response of CRP and SAA to chronic low-grade vascular inflammation [5, 13, 14, 15]. These variations could indicate the existence of positive and negative control mechanisms that permit, independently, the induction of CRP or SAA levels to fulfill the host-specific low-grade inflammatory response and it is likely to be of relevance to the causation of future disease.

Mild to moderate elevation of circulating total homocysteine (tHcy), commonly referred to as homocysteinemia, is generally considered to be an independent risk factor for atherosclerosis [16]. The strength of the association between an elevated tHcy and cardiovascular disease seems to vary among populations [17]. In adults, considerable evidence has been accumulated to support that the link between tHcy and atherothrombosis cannot be explained by associations of tHcy with the chronic low-grade inflammation estimated from serum CRP [13]. Confirming previous observations by others [18, 19],

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RESEARCH COMMUNICATION

Vascular Endothelial Growth Factor and Placenta Growth Factor in Intrauterine Growth-Restricted Fetuses and Neonates

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The angiogenic factors vascular endothelial growth factor (VEGF) and placenta growth factor (PlGF) are respectively up- and downregulated by hypoxia. We aimed to study circulating levels of the above factors in intrauterine growth restriction (IUGR) and to correlate their levels with the customized centiles of the infants. The study included 25 IUGR and 25 appropriate for gestational age (AGA) full-term, singleton infants and their mothers. Maternal (MS), fetal (UC), and neonatal day 1 (N1) and 4 (N4) blood was examined. MS and N1 PlGF, as well as UC VEGF levels correlated with the customized centiles of the infants ($r = 0.39$, $P = .007$, $r = 0.34$, $P = .01$, and $r = -0.41$, $P = .004$, resp). Furthermore, UC, N1, and N4 VEGF levels were higher in girls ($r = 0.36$, $P = .01$, $r = 0.33$, $P = .02$, and $r = 0.41$, $P = .005$ resp). In conclusion, positive and negative correlations of examined factors with the customized centiles of the infant could rely on placental function and intrauterine oxygen concentrations—both being usually lower in IUGR cases—while higher VEGF levels in girls should possibly be attributed to the stimulating action of estrogens.

INTRODUCTION

Vascular endothelial growth factor (VEGF), a key angiogenic factor in physiological and pathological conditions, exists in five isoforms of 121, 145, 165,189, and 206 amino acids [1]. VEGF exerts its effects by binding with high affinity to two tyrosine kinase receptors, VEGFR-1/Flt-1 [2] and VEGFR-2/KDR [3], present on endothelial cells [4]. VEGF has been demonstrated to be a potent stimulator of endothelial cell proliferation [5] and production of the plasminogen activators, required for proteolytic degradation of the extracellular matrix [6]. Both of these actions are markers of angiogenic activity. Particularly, branching angiogenesis mediated by VEGF is due to its binding to both Flt-1 and KDR receptors. The expression of VEGF and KDR is more pronounced during early gestation, particularly the first two trimesters, and decline with progression of pregnancy [7].

Placenta growth factor (PlGF) belongs to the same family and shares with VEGF 53% homology [8]. It has been shown to be a very weak stimulator of endothelial cell chemotaxis and proliferation at physiological concentrations [9, 10]. On the other hand, PlGF potentiates the action of low doses of VEGF on microvascular endothelial cells [11]. PlGF binds Flt-1 but not KDR [11] and results in nonbranching angiogenesis [12, 13]. PlGF and Flt-1 expression increases in the last trimester, towards term [14].

Oxygen is thought to be a major regulator of the balance between VEGF and PlGF function [15]. PlGF expression is stimulated under elevated PO_2 and downregulated by a low PO_2 [15, 16], whereas VEGF and its receptors are upregulated by a low PO_2 [17].

Intrauterine growth restriction (IUGR) is commonly associated with an altered placental angiogenesis, due to impaired placental oxygenation [18]. In addition, IUGR is a significant cause of infant mortality and morbidity [19]. The majority of IUGR cases present an asymmetric pattern of growth, usually associated with abnormalities in placental structure and function. The latter are responsible for deprivation of the developing fetus of sufficient oxygen and nutrients, required for his or her optimal growth [20].

In this study, we hypothesized that circulating levels of VEGF and PlGF in IUGR fetuses and neonates should differ from respective levels found in appropriate for gestational age (AGA) fetuses and neonates, as the former suffer from in utero hypoxia and present restricted growth and development. Therefore, we aimed to determine and

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Blood Vessels

Effect of Dark Chocolate on Arterial Function in Healthy Individuals

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Background: Epidemiologic studies suggest that high flavonoid intake confers a benefit on cardiovascular outcome. Endothelial function, arterial stiffness, and wave reflections are important determinants of cardiovascular performance and are predictors of cardiovascular risk.

Methods: The effect of flavonoid-rich dark chocolate (100 g) on endothelial function, aortic stiffness, wave reflections, and oxidant status were studied for 3 h in 17 young healthy volunteers according to a randomized, single-blind, sham procedure–controlled, cross-over protocol. Flow-mediated dilation (FMD) of the brachial artery, aortic augmentation index (AIx), and carotid–femoral pulse wave velocity (PWV) were used as measures of endothelial function, wave reflections, and aortic stiffness, respectively. Plasma oxidant status was evaluated with measurement of plasma malondialdehyde (MDA) and total antioxidant capacity (TAC).

Results: Chocolate led to a significant increase in resting and hyperemic brachial artery diameter throughout the study (maximum increase by 0.15 mm and 0.18 mm, respectively, $P < .001$ for both). The FMD increased significantly at 60 min (absolute increase 1.43%, $P < .05$). The AIx was significantly decreased with chocolate throughout the study (maximum absolute decrease 7.8%, $P < .001$), indicating a decrease in wave reflections, whereas PWV did not change to a significant extent. Plasma MDA and TAC did not change after chocolate, indicating no alterations in plasma oxidant status.

Conclusions: Our study shows for the first time that consumption of dark chocolate acutely decreases wave reflections, that it does not affect aortic stiffness, and that it may exert a beneficial effect on endothelial function in healthy adults. Chocolate consumption may exert a protective effect on the cardiovascular system; further studies are warranted to assess any long-term effects. Am J Hypertens 2005;18:785–791 © 2005 American Journal of Hypertension, Ltd.

Key Words: Arterial stiffness, chocolate, flavonoids, endothelial function, wave reflections, oxidant stress.

Endothelial function, elastic properties of large arteries, and magnitude and timing of wave reflections are important determinants of cardiovascular performance and have been identified as independent prognosticators of cardiovascular morbidity and mortality.[1–4]

Several epidemiologic studies suggest that regular consumption of foods and beverages rich in flavonoids is associated with a decreased risk of cardiovascular mortality, including coronary artery disease and stroke.[5] Cocoa and chocolate products have a much higher flavan-3-ol concentration and total antioxidant capacity per weight than other flavonoid-containing beverages such as red wine, green tea, and black tea.[6]

Data on the effect of chocolate on arterial function are limited.[7,8] In the present randomized, sham procedure–controlled, cross-over study, we investigated whether dark chocolate affects arterial function, using a thorough approach that integrates measures of endothelial function, aortic stiffness and wave reflections in healthy subjects. Moreover, we tested the hypothesis that any observed change on these parameters would be associated with an alteration in antioxidant status.

Methods

Study Population

We studied 17 healthy individuals (12 men and five women, mean age 28.9 years, range 24 to 32 years). All subjects were nonsmokers and nonobese (body mass index <27 kg/m^2), and they did not have diabetes, hyperlipid-

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Original research article

Effect of oral contraceptive treatment on bone mass acquisition in skeletally immature young female rats

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Abstract

The objective of the present study was to investigate the effect of oral contraceptive (OC) treatment on bone mass accrual in skeletally immature young female rats. Animals in the baseline group were killed at the beginning of the experiment and were subjected to bone density assessment by peripheral quantitative computerized tomography (pQCT). The control group was fed a base diet free of phytoestrogens, while animals in the contraceptive group received the same base diet mixed with 2.67 μg desogestrel/100 g body weight and 0.0533 μg ethinyl estradiol/100 g body weight. The duration of the treatment period was 16 weeks. Densitometric measurements by dual energy x-ray absorptiometry and serum bone markers assessment were carried out at baseline, at 8 weeks and at 16 weeks, while pQCT densitometry took place after sacrifice. All bone mineral density and bone mineral content indices measured by dual energy x-ray absorptiometry increased significantly throughout the study period in both the OC and control group. Concerning pQCT measurements, animals in both the OC and the control group had significantly higher cortical density compared with baseline (midtibia: p=.0003 and .0003, respectively). Total area and periosteal circumference were significantly higher in OC group, both in proximal (p=.003 and .003, respectively) and midtibia (p=.048 and .042, respectively) compared with baseline. Osteoprotegerin serum levels increased in both groups, and at the end of the experiment, circulating osteoprotegerin was significantly higher in the OC group compared with controls (p=.032). At the end of the experiment, carboxyl-terminal telopeptides of collagen type I levels were significantly lower in the OC-treated animals compared with controls (p=.046). Our results suggest that OC administration to skeletally immature female rats allows normal bone accrual and may even improve bone geometry. This effect may be mediated through enhanced inhibition of bone resorption.

Keywords: Bone mass accrual; Oral contraceptive; Osteoprotegerin; Wistar rat; C-telopeptides of collagen type I

1. Introduction

It is becoming increasingly appreciated that postmenopausal osteoporosis is influenced by events taking place during bone development and that a high bone mass at skeletal maturity could serve as a good predictor for lower age-related fracture risk. In adults, the bone mass present at any time in life depends on the amount achieved at maturity and that lost with aging. The magnitude of peak bone mass, achieved by early adulthood, may modulate up to 60% of the subsequent risk of osteoporosis [1]. All the above make bone development during adolescence particularly critical for establishing genetically determined peak bone mass [2–4]. Determinants of bone mass accrual include hereditary factors that account for an estimated 60% to 80% of the variability in peak bone mass [5], sex, nutrition, mechanical forces (physical activity, body mass index) and endocrine factors. Endogenous sex steroids are considered the most important endocrine regulators of bone metabolism. Estro-

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The Role of the Anti-Angiogenic Factor Endostatin in Intrauterine Growth Restriction

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OBJECTIVE: *To study the impact of intrauterine growth restriction (IUGR) on anti-angiogenesis, by determining and comparing circulating levels of the potent anti-angiogenic factor endostatin, in full-term IUGR (under the 10th customized centile) and appropriate for gestational age (AGA) fetuses, neonates, as well as their mothers, granted that IUGR implies hypoxia and endostatin is down-regulated by the latter.*

METHODS: *In 20 IUGR cases (mainly due to hypertension or preeclampsia) and 20 AGA controls we determined circulating endostatin levels, by enzyme immunoassay in the serum of mothers (MS), umbilical cords (UC–mixed arteriovenous blood)–representing the fetal state, and asymptomatic neonates on day 1 (N1) and 4 (N4) of life–signifying transition and stabilization to extrauterine life, respectively.*

RESULTS: *Endostatin levels were significantly higher in AGA than IUGR UC, N1, and N4 (P <.0000, P = .0006, P = .024, respectively). Furthermore, UC endostatin levels positively correlated with the customized centiles of the infants (Spearman correlation coefficient 0.69, P = .00001).*

CONCLUSIONS: *IUGR is characterized by lower circulating endostatin concentrations in the fetus and neonate, possibly because under lower oxygen concentrations an unbalanced state of angiogenesis stimulators versus inhibitors takes place. (J Soc Gynecol Investig 2005;12:195–7)* *Copyright © 2005 by the Society for Gynecologic Investigation.*

KEYWORDS: Endostatin, intrauterine growth restriction, preeclampsia, intrauterine hypoxia, angiogenesis.

Endostatin, a 20-kd protein, is a cleavage fragment of the C-terminal region of collagen XVIII,[1] which is localized in the basement membrane of blood vessels.[2] Additionally, endostatin is an extracellular matrix molecule.[3] It is a potent anti-angiogenic factor, implicated in the regulation of physiologic and pathologic angiogenesis.[4] Endostatin exerts its anti-angiogenic effects by inhibiting endothelial cell proliferation, migration, and tube formation,[5] while it directly induces endothelial cell apoptosis. Thus, it has been shown that endostatin treatment causes a marked reduction in the anti-apoptotic proteins bcl-2 and bcl-xl, leaving the pro-apoptotic protein bax unaffected.[6] Human microvascular endothelial cells and pericytes produce endostatin,[4] which is down-regulated by hypoxia. The same cells express the gene of vascular endothelial growth factor (VEGF), an important angiogenic factor up-regulated by hypoxia.[7,8]

Intrauterine growth restriction (IUGR) is commonly associated with altered placental angiogenesis due to impaired placental oxygenation.[9] The majority of IUGR cases present an asymmetric pattern of growth, usually associated with abnormalities in placental structure and function. The latter are responsible for deprivation of the developing fetus of sufficient oxygen and nutrients, required for optimal growth.[10] Thus, IUGR is a significant cause of infant mortality and morbidity.[11] This study was based on the hypothesis that circulating levels of endostatin should be lower in IUGR than in appropriate for gestational age (AGA) fetuses and neonates as the former suffer from in utero hypoxia. Therefore, we aimed to determine and compare circulating levels of endostatin in IUGR and AGA neonates at time points characteristic for intrauterine and extrauterine life.

SUBJECTS AND METHODS

The study was approved by the Ethics Committee of our teaching hospital and informed consent was acquired from participating mothers.

The study comprised 20 IUGR and 20 AGA full-term singleton infants and their mothers. IUGR was defined as a birth weight below the 10th customized centile. The Gestation Related Optimal Weight (GROW) computer-generated program[12,13] was used to calculate the customized centile for each pregnancy. Significant determinants of birth weight, such as gestational age, sex, maternal weight at the beginning of pregnancy, maternal height, ethnic group, and parity, were entered into the program to adjust the normal birth weight centile limits.[12]

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SHORT COMMUNICATION

Neurotrophin-3 and FLT3 Tyrosine Kinase Receptor in Perinatal Life

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Our aim is to determine—in 30 healthy full-term infants and their mothers—circulating levels of neurotrophin-3 (NT-3) (important for antenatal and postnatal brain development and implicated in the immune response) and FLT3 tyrosine kinase receptor (FLT3) (controlling hematopoiesis and found in the nervous tissue), in the fetal and neonatal life. NT-3 levels, in contrast to FLT3 ones, increased significantly on the fourth postnatal day in relation to the low levels found in the mother, fetus, and day 1 neonate ($P = .03$, respectively). Maternal and umbilical NT3 levels positively correlated with respective FLT3 levels ($P = .003$ and $P = .03$). Circulating NT-3 levels increased in early neonatal life, possibly due to exposure to various stimuli soon after birth. FLT3 levels do not seem to behave accordingly, although these two substances probably synergize.

INTRODUCTION

Neurotrophin-3 (NT-3) belongs to the neurotrophin family, which includes, among others, the nerve growth factor and the brain-derived neurotrophic factor (BDNF) [1]. Neurotrophins exert antiapoptotic activities in neurons [2] and are implicated in higher neuronal functions [3] and neurotransmitter expression [4]. Therefore, neurotrophins may play important roles in antenatal and postnatal brain development. In addition, neurotrophins are involved in the immune response [5, 6, 7, 8]. Moreover, nerve growth factor, BDNF, and NT-3 act on tyrosine kinase A, B, and C receptors, respectively. It has recently been reported that in neonatal age circulating neurotrophin levels could reflect the degree of neuronal maturity [1], since, at this age, due to the immature blood-brain barrier, neurotrophin blood levels may also represent concentrations in the CNS [9].

The FLT3 receptor (FLT3) is a member of the class III receptor tyrosine kinases [10]. Related members of this family of receptors together with their respective ligands have been shown to control numerous distinct stages of hematopoiesis [11, 12]. Recently, regions of the brain have been shown to harbor neural stem/progenitor cells that retain the capacity to proliferate and to give rise to new cells throughout the lifetime of an animal [10, 13]. Thus, they generate neurons, oligodendrocytes, astrocytes, but also retain the ability to repopulate hematopoietic systems of irradiated animals [14] and to give rise to multiple tissue types when grown in the presence of embryonic stem cells [15]. On the other hand, hematopoietic stem cells have been shown to give rise to neurons, when transplanted into mice [16, 17, 18, 19]. In this respect, many studies have confirmed the presence of FLT3 mRNA in nervous tissue [20, 21].

This study was based on the hypothesis that a possible interaction between hematopoietic and neuronal activity in the central nervous system, possibly also representing the state of maturity, might be reflected in the periphery. Thus, we aimed to determine circulating levels of NT-3 and FLT3 in full-term neonates and correlate these levels with gestational age, gender, and mode of delivery.

MATERIALS AND METHODS

The study was approved by the Ethics Committee of our teaching hospital and informed consent was obtained from participating mothers. The study included 30 healthy, infection-free, nonsmoking parturients (mean age: 25.6 ± 3.4, range: 20–40 years) and their healthy neonates appropriate for gestational age and born after single uncomplicated pregnancy and delivery. Apgar scores were ≥ 8 in the first and fifth minutes. Placentas were in all cases normal in appearance and weight. Complete blood count and C-reactive protein were within normal ranges in all newborns. All infants received breast

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The effect of hormone therapy and raloxifene on serum matrix metalloproteinase-2 and -9 in postmenopausal women

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ABSTRACT

Objective: The aim of the study was to investigate the effect of continuous-combined hormone therapy and raloxifene on the total and active forms of serum matrix metalloproteinase (MMP) -2 and -9.

Design: The study was double-blinded, with a placebo run-in period of 28 to 50 days. Twenty-eight women received either 17β-estradiol 2 mg + norethisterone acetate 1 mg (E_2/NETA) or raloxifene HCL 60 mg for a period of 6 months. Total and active forms of MMP-2 and -9 were estimated at baseline and at month 6.

Results: Total MMP-2 increased significantly in both E_2/NETA and raloxifene groups (raloxifene baseline: 278.1 ± 18.1 ng/mL; 6 months: 303.1 ± 29.9 ng/mL, $P = 0.008$) (E_2/NETA baseline: 281.9 ± 27.5 ng/mL; 6 months: 298.8 ± 12.7 ng/mL, $P = 0.025$). Similarly, both treatments increased the active MMP-2 fraction, although only the raloxifene-associated increase acquired significance (raloxifene baseline: 24.9 ± 8.6 ng/mL; 6 months: 31.6 ± 15.3ng/mL, $P = 0.045$) (E_2/NETA baseline: 21.7 ± 5.7 ng/mL; 6 months: 27.4 ± 5.8 ng/mL, $P = 0.128$). Total as well as active fractions of MMP-9 were not significantly affected by either treatment.

Conclusions: Both E_2/NETA and raloxifene increased the total and active MMP-2 serum levels. MMP-9 was not significantly affected by either regimen. Larger, long-term clinical trials are needed to elucidate the effect of HT and raloxifene on MMPs and the possible clinical implications for cardiovascular health.

Key Words: Matrix metalloproteinase – Estrogen – Hormone therapy – Raloxifene.

Atherosclerosis and its thrombotic complications contribute to morbidity and mortality among postmenopausal women.[1-5] The risk for coronary artery disease (CAD) increases after menopause, making inevitable the association of this disease state with estrogen depletion.[6]

Observational studies have suggested that long-term estrogen (ET) or estrogen-progestin (EPT) therapy administered to healthy women may decrease the risk of CAD.[2,5,7,8] However, two randomized, controlled trials evaluating the effect of a specific continuous hormone therapy (HT) regimen on primary (Women's Health Initiative)[9] and secondary (Heart and Estrogen/ Progestin Replacement Study)[10] CAD prevention did not confirm the cardiovascular benefit reported by observational studies. In fact, the Women's Health Initiative further suggested an HT-associated increase in CAD risk.[9]

Estrogens and the selective estrogen receptor modulator (SERM) raloxifene may express an antiatherogenic effect via mechanisms that modulate the lipid-lipoprotein profile,[2,11,12] homocysteine serum levels,[13,14] fibrinolysis, and carbohydrate metabolism.[2,7] Estrogens and raloxifene may promote vasodilation by

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Effect of hormone therapy and raloxifene on serum VE-cadherin in postmenopausal women

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Objective: To investigate the effect of continuous combined hormone therapy and raloxifene on serum VE-cadherin.

Design: The study was double blinded, with a placebo run–in period of 28–50 days.

Setting: University menopause clinic.

Patient(s): Twenty-eight healthy postmenopausal women devoid of climacteric complaints.

Intervention(s): Subjects were randomized to 17β-estradiol (2 mg) + norethisterone acetate (1 mg; E_2-NETA) or raloxifene hCL (60 mg) for a period of 6 months.

Main Outcome Measure(s): Serum VE-cadherin, which was estimated at baseline and at month 6.

Result(s): Serum VE-cadherin decreased significantly in both E_2-NETA and raloxifene groups (raloxifene baseline ± SD: 1.17 ± 0.44 ng/mL, 6 months: 0.82 ± 0.29 ng/mL; E_2-NETA baseline: 1.19 ± 0.47 ng/mL, 6 months: 0.92 ± 0.49 ng/mL). Percentage changes from baseline were −21.7 ± 24.3 for E_2-NETA and −26.0 ± 20.6 for raloxifene.

Conclusion(s): The effect of E_2-NETA and raloxifene suggests that these drugs may preserve interendothelial junction integrity and control vascular permeability. Although this effect may influence the progress of the atheromatous lesion, its clinical impact on coronary artery disease (CAD) remains uncertain. (Fertil Steril® 2004;82:634–8. ©2004 by American Society for Reproductive Medicine.)

Key Words: VE-cadherin, HT, estrogen, raloxifene

Atherosclerosis and its thrombotic complications are responsible for the increase in coronary artery disease (CAD)–related morbidity and mortality among postmenopausal women (1–4). Although estrogen depletion has been inevitably implicated, and although observational studies have suggested that estrogen or estrogen-progestin administration is cardioprotective (2, 5, 6), the results of two randomized clinical trials evaluating the effect of hormone therapy (HT) on primary (Women's Health Initiative) (7) and secondary (Heart and Estrogen/Progestin Replacement Study) (8) CAD prevention have questioned the efficacy of hormone therapy despite confirming estrogen's lipid-lowering effect (5).

Human vascular cadherin (VE-cad) is an endothelium-specific, calcium-dependent transmembrane glycoprotein encoded by the VE-cad gene that is exclusively present in endothelial cells (9–12). VE-cad is mainly concentrated at endothelial cell–cell contacts and is responsible for the organization and maintenance of interendothelial junctions (9). Both its extracellular and intracellular domain have been shown to interact with cytoskeleton proteins known as catenins, so as to mediate aggregation and homophilic cell–cell adhesion. VE-cad is considered to provide cohesion to the junction and to control vascular endothelial permeability (9, 11, 13, 14).

Clinical and experimental studies have suggested the presence of an immune inflammatory response throughout all stages of atherosclerosis, from its initiation to the destabilization and rupture of the plaque and thrombus formation (4, 15–17). Early in atherogenesis, systemic or local inflammatory stimuli increase cytokines (TNFα, IL-6), which activate the endothelium and cause

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ORIGINAL ARTICLE

Negative Association between Circulating Total Homocysteine and Proinflammatory Chemokines MCP-1 and RANTES in Prepubertal Lean, but Not in Obese, Children

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Abstract: This study investigated in prepubertal obese children (POC), compared with prepubertal lean children (PLC), a possible relation among plasma total homocysteine (tHcy)—an independent risk factor for future atherosclerosis—and MCP-1 and RANTES, two circulating chemokines inducing leukocyte transendothelial migration (TEM), implicated in the initial stages of the inflammatory part of the atherosclerotic process. Seventy-two POC were evaluated for circulating tHcy, MCP-1, and RANTES, and compared with 42 healthy PLC. The mean adjusted (for age, sex as well as $\log_{10}$total insulin, vitB12, folate, total cholesterol, HDL cholesterol, $\log_{10}$triglycerides, and $\log_{10}$glucose levels) differences in tHcy, MCP-1, and RANTES levels between PLC and POC were all significant [1.16 nmol/mL (P = 0.03), 26.6 pg/mL (P = 0.02), and 52.9 pg/mL (P = 0.03), respectively]. In PLC, but not in POC, tHcy levels were negatively associated with both circulating MCP-1 (B = −1.68, P = 0.007) and RANTES (B = −1.16, P = 0.01) after adjusting for age, sex, BMI, as well as $\log_{10}$total insulin, vitB12, folate, total cholesterol, HDL cholesterol, $\log_{10}$triglycerides, and $\log_{10}$glucose levels. In conclusion, in POC there is a lack, in contrast to PLC, of a possibly autoregulatory, negative association of elevated tHcy levels to increased MCP-1 and RANTES levels. This could contribute to future, homocysteine-induced atherosclerosis.

Key Words: prepubertal obesity, atherosclerosis, homocysteine, MCP-1, RANTES

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The long-term risks that arise from obesity in childhood—independently of future adult weight—are associated, among a broad range of adverse health effects, with atherosclerosis in adulthood.[1]

Mild to moderate elevation of circulating total homocysteine (tHcy), commonly referred as homocysteinemia, is generally considered an important risk factor for atherosclerosis.[2–6] However, the strength of the association between an elevated tHcy concentration and atherosclerosis seems to vary among populations. It is still unclear by which pathophysiological mechanisms tHcy may promote atherosclerosis. Plausible mechanisms include endothelial cell damage, inhibition of fibrinolysis, activation of coagulation cascade, impaired generation of nitric oxide and prostacyclin, enhanced collagen production by smooth muscle cells, and promotion of lipoprotein oxidation, as well as increased cholesterol synthesis in hepatocytes.[6–9]

Atherosclerosis is a chronic inflammatory disease. The earliest lesions of atherosclerosis, the so-called fatty streaks, which are common in infants and young children,[10] are pure inflammatory lesions, consisting only of monocyte-derived macrophages and T-lymphocytes.[11] They adhere in clusters to the endothelium, migrate over its surface, and reach the subendothelial intima by penetrating at endothelial cell junctions. There, the monocytes are converted to macrophages and ingest lipids to become foam cells.[12] The sustained increased recruitment of monocytes and T-lymphocytes into the arterial wall, called transendothelial migration (TEM), can be stimulated by chemoattractant protein-1 (MCP-1) and regulated upon activation normal T-cell expressed and secreted (RANTES), respectively.[13] Recent studies suggest that homocysteine can stimulate C-C type chemokine (eg, MCP-1 and RANTES) expression.[14]

This study investigated the hypothesis of a possible relation in POC, in comparison to PLC, among plasma tHcy and circulating proinflammatory chemokines MCP-1 and RANTES, which might reflect initial stages of the inflamma-

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EFFECT OF ANGIOTENSIN-CONVERTING ENZYME INHIBITOR ON COLLAGENOLYTIC ENZYME ACTIVITY IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Summary: *Matrix metalloproteinases and their tissue inhibitors are key enzymes degrading myocardial collagen in acute myocardial infarction (AMI). The aim of the present study was to determine whether angiotensin-converting enzyme inhibitors (ACEI) influence collagenase-1 (MMP-1) and their tissue inhibitor (TIMP-1) activity in AMI patients. Plasma levels of MMP-1, TIMP-1 and MMP-1/TIMP-1 complex were measured in 24 patients (aged 58.4 ± 13.9 years) with AMI. Thirteen patients received perindopril 4 mg/day (group A) and 11 did not (group B). Plasma samples collected on admission and at 0, 3, 6, 9, 12, 18, 24, 36 and 48 hours and on days 3, 4, 5, 7, 15 and 30 thereafter were analyzed by relevant ELISA kits. Ejection fraction (EF) was assessed by ventriculography and end-diastolic diameter (EDD) echo-study on days 6 and 30. Values of collagenolytic enzymes of group A compared with those in group B were on average lower by 34%, 18.3% and 40%, respectively. The difference in values between groups at 0 h, 3 h and 9 h was significant ($p < 0.048$). ANOVA repeated measurement analysis showed significance within subjects for MMP-1 alone ($p < 0.043$) and for MMP-1 and ACEI ($p < 0.046$), while for TIMP-1 and MMP-1/TIMP-1 complex significance was only $p < 0.0009$. Regarding EDD changes, patients in group A showed minimal or no changes (51.23 ± 1.8 mm to 51.6 ± 2.13 mm), their EF was 38.8% and infarct size was medium to large. In contrast, group B showed a trend to increase EDD (41 ± 0.78 mm to 42.33 ± 0.59 mm), their EF was 50.5% and infarct size was small to medium. In conclusion, early initiation of ACEI treatment reduces collagenolytic activity. This effect may be considered an alternative mechanism for beneficial effects on postinfarction remodeling.*

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Introduction

Matrix metalloproteinase-1 (MMP-1), their tissue inhibitor-1 (TIMP-1) and MMP-1/TIMP-1 complex are the key enzymes degrading myocardial fibrillar colla-

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Chemokines Rantes and Interleukin-8 in the Perinatal Period: Changes in Serum Concentrations

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ABSTRACT

Chemokines, a superfamily of polypeptide mediators, are a key component of immune surveillance and are implicated in the initiation of the inflammatory cascade. This study investigated whether serum concentrations of the chemokines regulated upon activation, normal T-cell expressed and presumably secreted (RANTES) and interleukin-8 (IL-8) change in the perinatal period because of the transition from intra- to extrauterine life, and compared determined values in mothers (MS) (n = 30) with those in their fetuses (UC), neonates (day of life 1 [N1] and 4 [N4]), and controls (CS) (n = 20). RANTES serum concentrations were higher in MS than in UC ($p < 0.006$), N1 ($p < 0.0001$), N4 ($p < 0.0001$), and CS ($p < 0.0001$). IL-8 serum concentrations in MS and UC, respectively, were significantly lower than in N1 ($p < 0.0002$ and $p < 0.0007$) and N4 ($p < 0.0001$ and $p < 0.0001$). Thus, after birth, neonatal serum concentrations of RANTES decrease, possibly because of elimination of the placenta (probable production site), and neonatal serum concentrations of IL-8 increase, possibly triggered by environmental antigenic stimuli to which the neonate is exposed.

KEYWORDS: RANTES, interleukin-8, perinatal period

The initiation of the inflammatory cascade is characterized by the coordinated expression of proinflammatory cytokines and chemokines. Chemokines (chemotactic cytokines) a superfamily of polypeptide mediators, are a key component of immune surveillance.[1] The relative positions of the cysteine tandem defines 4 classes of chemokines the most important of which are the C-C and C-X-C chemokines.[1] The specific effects of chemokines on their target cells are mediated by members of a family of seven transmembrane-spanning, G-protein-coupled receptors.[2]

American Journal of Perinatology, Volume 21, Number 4, 2004. Address for correspondence and reprint requests: Ariadne Malamitsi-Puchner, M.D., Second Department of Obstetrics and Gynaecology, University of Athens, 19 Soultani Str., 10682 Athens, Greece. [1]Second Department of Obstetrics and Gynaecology, University of Athens, Athens, Greece. Tel: +1(212) 584-4662. 0735-1631,p;2004,21,04,235,240,ftx,en;ajp39650x.

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Brief Communication

The Effect of Ca^{2+} Channel Antagonists on Plasma Concentrations of Matrix Metalloproteinase-2 and -9 in Essential Hypertension

Alexandra Zervoudaki, Emanuel Economou, Christos Pitsavos, Karmen Vasiliadou, Constantina Aggeli, Konstantinos Tsioufis, Marina Toutouza, Christodoulos Stefanadis, and Pavlos Toutouzas

The ability of some antihypertensive drugs to protect from vascular damage in hypertension might be partially due to their ability to control matrix metalloproteinase (MMP)–mediated extracellular matrix metabolism, which in turn may contribute to vascular remodeling. This study was designed to investigate whether treatment with felodipine or diltiazem has any effect on plasma levels of MMP-2 and MMP-9 in essential hypertensive patients. We measured plasma levels of active MMP-2 and MMP-9 in 72 hypertensive subjects and 45 controls, both before and after 6 months of treatment with felodipine (group A) or diltiazem (group B). Mean adjusted differences, before and after each treatment, for MMP-2 and MMP-9 levels were: 19.8 (P = .01) for MMP-2, 0.2 (P = .5) for MMP-9 (group A), and 1.4 (P = .4) for MMP-2, 0.2 (P = .7) for MMP-9 (group B). These findings show that MMP-2 level is raised by treatment with felodipine but not diltiazem, whereas MMP-9 is unaffected by either treatment. Am J Hypertens 2004;17:273–276 © 2004 American Journal of Hypertension, Ltd.

Key Words: Metalloproteinases, extracellular matrix, remodeling.

Arterial hypertension is usually associated with the development of vascular fibrosis. This pathologic process is characterized by structural changes in the arterial wall caused by increased deposition of extracellular matrix (ECM) components, particularly collagen, as well as alterations in ECM architecture or cell–extracellular matrix attachments.[1]

The matrix metalloproteinases (MMP) proteolytic system and the natural tissue endogenous inhibitors (TIMP) are involved in the regulation of ECM metabolism. Changes in MMP or TIMP activity may contribute to vascular remodeling in hypertensive patients by modulating ECM profile and interacting with adhesion receptors.[2] Furthermore, recent experimental and clinical studies have generated the hypothesis that treatment with certain antihypertensive drugs, which protect the vasculature and correct both arterial remodeling and endothelium dysfunction, may result in better prognosis in hypertension.[3]

We have recently shown that plasma concentrations of active MMP-2 and MMP-9 are depressed in patients with essential hypertension, which may reflect abnormal extracellular matrix metabolism.[4] Furthermore, 6 months of antihypertensive treatment with the calcium channel antagonist (CCA) amlodipine significantly increased plasma levels of active MMP-9. Previous studies have also shown diminished MMP activity in hypertensive patients. Indeed, Laviades et al[5] have shown abnormally diminished collagenase activity of MMP-1 in hypertensive individuals with left ventricular hypertrophy, a finding supported by the work of Brilla et al.[6] Moreover, 1-year treatment with the angiotensin converting enzyme inhibitor lisinopril increased MMP-1 concentrations. Recently, the proteolytic activites of MMP-9 and TIMP-1 have been suggested to be depressed in hypertensive patients but not significantly affected by short-term antihypertensive treatment with either enalapril or losartan.[7]

It is possible that some antihypertensive drugs may protect against vascular damage in hypertension, possibly exhibiting drug-specific benefits beyond arterial pressure lowering. The ability of some antihypertensive drugs to

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Perinatal changes of brain-derived neurotrophic factor in pre- and fullterm neonates

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Abstract

Background: Brain-derived neurotrophic factor (BDNF) is abundant in brain and peripheral nerves, affects normal development, growth and survival and is implicated in immune response. *Aim*: To determine in single preterm (P) and fullterm (F) neonates, circulating intra- and extrauterine levels of BDNF, supposingly reflecting their neuronal and immune maturity. *Study design*: Prospective study. *Subjects*: Thirty healthy, appropriate for gestational age (AGA) F (mean gestational age 39.2 ± 1.4 weeks), 15 healthy AGA P (29.4 ± 1.3 weeks), and their mothers. *Outcome measures*: BDNF was measured by enzyme immunoassay methods in the serum of: mothers at the first stage of labor (MS), the umbilical cord (UC) and the neonates on days 1 (N1) and 4 (N4) postpartum. *Results*: Levels of BDNF in (a) FMS did not differ from PMS, but both were significantly higher than respective (F or P) UC, N1 and N4 (p ranging from <0.01 to <0.001), (b) FUC, FN1 and FN4 were significantly higher than PUC ($p<0.001$), PN1 ($p<0.03$) and PN4 ($p<0.02$), respectively, (c) PN1 increased significantly as compared to PUC ($p<0.05$). *Conclusions*: Higher BDNF MS levels may reflect the mature nervous and immune systems of mothers. Higher BDNF levels in F than P may also be due to advanced maturity in the former. Increased BDNF levels in PN1 as compared to PUC may indicate stimulation of immune response with exposure to antigenic stimuli from the extrauterine environment. Nevertheless, this stimulation is insufficient in P, who by decreasing N4 levels are by far less protected than F.

Keywords: Brain-derived neurotrophic factor; Perinatal period; Preterm neonate; Fullterm neonate

Abbreviations: BDNF, brain-derived neurotrophic factor; F, fullterm neonate; P, preterm neonate; MS, maternal serum; UC, umbilical cord serum; N1, neonatal day 1 serum; N4, neonatal day 4 serum.

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Improved myocardial performance during repetitive exercise testing: The role of extracellular superoxide dismutase activity in a model of exercise-induced myocardial preconditioning

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Background The aim of this study was to investigate whether endogenous antioxidant defense is involved in adaptation to myocardial ischemia in patients with coronary artery disease and severe exercise-induced myocardial ischemia.

Methods Fifty patients, aged 50 to 72 years (mean, 58 ± 6 years), with positive exercise test results underwent 4 treadmill exercise tests. Thallium-201 scintigraphy was performed during the first and the fourth testing. The second, the third, and the fourth tests were performed the next day. The time interval between the second and the third test was 15 minutes, and between the third and the fourth test, the interval was 45 minutes. Extracellular superoxide dismutase activity was measured just before and at the peak of the first and the fourth exercise test.

Results The patients were divided in 2 groups according to the extent of myocardial ischemia at peak exercise of the fourth test compared with the first test. Most of the patients studied (37/50) showed improved myocardial performance during the last of the sequential exercise tests, as demonstrated with the studied exercise parameters and the extent of myocardial ischemia in thallium-scintigraphy. Extracellular superoxide dismutase activity before the last exercise test was found to be significantly increased only in the patients who had improved myocardial performance at the last of the sequential exercise tests.

Conclusion The beneficial effects of sequential episodes of exercise-induced myocardial ischemia seem to be strongly related to extracellular superoxide dismutase activity. Although there is still lack of direct evidence, our data support the theory that the favorable adaptation to repetitive exercise may represent an aspect of the clinical relevance of ischemic preconditioning in humans. (Am Heart J 2003;146:160-7.)

Ischemic preconditioning, a phenomenon related to repetitive, short periods of ischemia separated by intermittent reperfusion, has been proven to render the heart resistant to a subsequent, prolonged episode of ischemia. Myocardial protection with ischemic preconditioning has been reported to limit infarction size,[1-3] decrease the incidence of ventricular arrhythmias associated with ischemia-reperfusion,[4] and improve myocardial performance during exercise-induced repeated episodes of ischemia.[5-7]

Experimental studies have confirmed the role of ischemic preconditioning[8] in a variety of animal models, including rats, pigs, and rabbits. However, human studies do not meet the strict conditions outlined in these experimental studies. Thus, solid evidence for the occurrence, the clinical role, and the underlying mechanisms of ischemic preconditioning in humans,[9,10] is not yet available.

It has been suggested that ischemic preconditioning is a major contributor to the attenuation of ischemia observed during the second of 2 sequential periods of reversible ischemia and particularly during the second of 2 exercise tests.[5-7] Although confounding factors such as the training effect, coronary vasodilatation, and the opening of collateral vessels have also been associated with improved myocardial performance after sequential exercise tests, it is now well established that

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ORIGINAL ARTICLE

Plasma levels of active extracellular matrix metalloproteinases 2 and 9 in patients with essential hypertension before and after antihypertensive treatment

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This study was designed to test the hypothesis that plasma concentrations of matrix metalloproteinase-2 (MMP-2) and matrix metalloproteinase-9 (MMP-9), two enzymes that share similar substrate specificity (collagen type IV and V), possibly related to vascular remodelling, are altered in essential hypertension. The second aim of the study was to assess whether chronic antihypertensive treatment with the calcium channel blocker amlodipine would normalize these alterations. To test this hypothesis, we measured plasma concentrations of active MMP-2 and MMP-9 in 42 patients with never-treated essential hypertension and in 25 normotensive control subjects. Measurements were repeated after 6 months of treatment with the calcium channel blocker amlodipine. Baseline values of MMP-2 and MMP-9 were decreased ($P=0.01$ and 0.002, respectively) in hypertensive patients compared with normotensives. Hypertensive patients with systemic vascular resistances <1440 dyn s/cm^5 exhibited higher values of MMP-2 ($P=0.005$) and MMP-9 ($P=0.001$) than hypertensive patients with systemic vascular resistances >1440 dyn s/cm^5. Treated patients attained a nonsignificant increase in MMP-2 plasma concentrations, but a significant increase in MMP-9 plasma concentrations ($P=0.01$) compared to respective values before treatment. In conclusion, these findings suggest that plasma concentrations of active MMP-2 and MMP-9, mainly related to vascular extracellular matrix metabolism, are depressed in patients with essential hypertension. A 6 month treatment with amlodipine can normalize MMP-9 but not MMP-2 plasma concentrations. The hypothesis that antihypertensive treatment may modulate collagen metabolism remains to be determined by further studies.
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Keywords: metalloproteinases; extracellular matrix; vascular remodelling

Introduction

Arterial hypertension is associated with cardiac and vascular remodelling. In cardiac remodelling, the structural changes involve myocyte hypertrophy and excessive accumulation of extracellular matrix component, that is, fibrosis.[1] Fibrosis has detrimental functional consequences because it impairs myocardial stiffness and promotes arrhythmias.[2] Alterations in mechanical properties and structure in arteries have also been well documented in chronic essential hypertension.[3,4] Mechanical changes of large arteries and resistance vessels may contribute to the cardiovascular complications of hypertension.[5,6]

Two processes, vessel remodelling and/or vessel hypertrophy, are responsible for the structural changes that have been reported in large arteries as well as in resistance vessels.[7,8] Remodelling of the vessel can occur through rearrangement of the existing cellular and extracellular components of the arterial wall around a smaller vessel. Vessel hypertrophy involves vascular smooth muscle cell proliferation, hypertrophy and/or polyploidy, and deposition of extracellular matrix (ECM), which may be localized to a region or throughout the vessel wall.

The ECM is responsible for the three-dimensional spatial arrangement, necessary for the integrity and the metabolic function of the tissue in intercellular space. In hypertension, changes have been reported

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Basic fibroblast growth factor changes in response to coronary angioplasty in patients with stable angina

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Abstract

Basic fibroblast growth factor (bFGF) has been implicated in the pathogenesis of coronary atherosclerosis and in angiogenesis. We assessed the changes in serum bFGF before, immediately after, and 6 months after coronary angioplasty (PTCA). Using the ELISA methods we measured plasma bFGF in 28 patients who underwent PTCA, in 20 patients with coronary artery disease who underwent elective coronary angiography and in 28 healthy subjects. Before PTCA and coronary angiography, bFGF plasma levels were similar in both patient groups (4.4±1.0 vs. 3.3±0.5 pg/ml), but were significantly higher compared with those of the control group (0.8±0.1 pg/ml, $P<0.05$). By 24 h, 3 months and 6 months after PTCA, bFGF levels had decreased significantly in the PTCA group (3.2±0.6, 1.7±0.3 and 2.7±0.6 pg/ml, respectively, $P<0.05$). In conclusion, these findings show that bFGF levels are elevated in patients with coronary artery disease. Following PTCA, bFGF levels decreased significantly and remained stable for 6 months after the procedure. Thus, bFGF level may change in response to PTCA in patients with coronary artery disease and stable angina.

Keywords: Growth factor; Angioplasty; Coronary artery disease; Atherosclerosis

1. Introduction

In recent years a growing body of evidence has accumulated favoring a significant role of collateral circulation in patients with coronary artery disease [1]. Collateral vessel growth is induced by chemical signals from the ischemic myocardium [2]. Angiogenic growth factors are produced by cardiac tissue, are more concentrated in pericardial fluid and are increased by myocardial ischemia [3–5]. Basic fibroblast growth factor (bFGF) and vascular endothelial growth factor (VEGF) are examples of such growth factors [6–13].

Recently it has been demonstrated that bFGF mitogenic activity increases in ischemic myocardium after coronary artery ligation in the dog, and this increase parallels the increase in collateral blood flow [14]. In canine experiments it has been demonstrated that local [15,16] or systemic [17] administration of bFGF enhances collateral neovascularization, resulting in a reduction in infarct size [15].

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Chemokines in patients with ischaemic heart disease and the effect of coronary angioplasty

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Abstract

Percutaneous coronary transluminal angioplasty (PTCA) may release inflammatory mediators such as chemokines. Monocyte chemoattractant protein-1 (MCP-1) and eotaxin (EOX) are monocyte- and eosinophil-specific chemokines involved in the inflammation and pathogenesis of coronary atherosclerosis. A total of 28 patients undergoing elective PTCA, 20 coronary artery disease (CAD) patients undergoing coronary angiography and 28 healthy controls were studied. In PTCA patients before the procedure, MCP-1 plasma levels (441±64 pg/ml) were similar to those of CAD patients (430±24 pg/ml), and significantly higher compared with controls (145±17 pg/ml, $P<0.01$). MCP-1 rose significantly after 3 and 6 months following PTCA (696±89 and 876±86 pg/ml, respectively, $P<0.01$ vs. before PTCA). EOX plasma levels (155±14 pg/ml) were similar to those of CAD patients (157±14 pg/ml), but significantly higher compared with controls (83.2±10 pg/ml, $P<0.05$). EOX rose significantly 24 h (273±41 pg/ml, $P<0.05$) but not 3 months after PTCA (160±20 and 158±19 pg/ml, respectively). These findings indicate that chemokine-induced monocyte- and eosinophil-specific chemoattraction is stimulated in patients with coronary artery disease. MCP-1 levels remain significantly elevated for at least 6 months following elective PTCA, suggesting an inflammatory stimulation.

Keywords: Inflammation; Angioplasty; Monocyte; Eotaxin

1. Introduction

It is widely accepted that chronic inflammation may lead to vascular dysfunction [1–5]. This process may involve leukocyte activation and adhesion to vascular endothelium [6]. Different families of cellular adhesion molecules are expressed on the surface of vascular endothelial cells following activation by diverse stimuli and may play a role in coronary atherosclerosis [7–10].

Monocyte chemoattractant protein-1 (MCP-1) and eotaxin (EOX) are monocyte- and eosinophil-specific chemokines involved in the inflammation process and in the pathogenesis of coronary atherosclerosis [11–17]. Circulating forms of MCP-1 have been detected in plasma and their levels are elevated during inflammatory conditions [7,11]. EOX regulates eosinophil accumulation through its effect on the adhesion molecules on microvascular endothelial cells [15–17]. Balloon dilatation produces variable morphologic alterations in the coronary stenosis (usually including a larger lumen) [18,19] and may release inflammatory mediators such as chemokines. The present study tested the hypothesis that chronic inflammation may be activated with angioplasty.

The purpose of this study was to evaluate the significance of circulating soluble forms of MCP-1

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Biochemical Changes Involved in the Mechanism of Vasovagal Syncope

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Vasovagal syncope elicits one of the most powerful transient vasodilatory responses in humans. Many studies have shown an altered neurohumoral response to tilting in patients with vasovagal syncope. Vasopressin (VP) has been of particular interest, but its exact role remains unclarified, whereas the possible role of the potent vasoactive end products of arachidonic acid metabolism has not yet been addressed. We determined the changes in plasma levels of VP, thromboxane (TXA2), and prostacyclin (PGI2) in 34 syncopal patients undergoing a standardized head-up tilt-table testing protocol and compared these changes between patients with positive and negative test results. Blood samples were collected at baseline, 15 minutes in the head-up position, and at the termination of the tilt test (the induction of syncope or the completion of a negative test). Sixteen patients had a positive test result, whereas 18 completed the test without developing any syncopal symptoms. In the tilt-positive group, VP levels presented a 20-fold increase at the time of syncope when compared with baseline levels ($p = 0.0000$), without any increase at earlier stages. No change was detected at any stage in the tilt-negative patients. We did not find any difference in the levels of PGI2 at any stage in any group of patients or between the 2 groups. TXA2 levels increased significantly at 15 minutes in the upright position in both tilt-positive and tilt-negative patients. No further increase was noticed at the time of syncope in the tilt-positive group, whereas in patients with a negative test result, there was a tendency to decline at the time of the test's completion. It is concluded that although VP is markedly increased during tilt-induced vasovagal syncope, vasoactive amines such as TXA2 and PGI2 play a minor role in the vasodilatory component of the response. ©2001 by Excerpta Medica, Inc.

(Am J Cardiol 2001;88:376–381)

Many studies have shown an altered neurohumoral response to tilting in patients with vasovagal syncope.[1] With regard to potentially centrally active neurohumoral agents, endorphins,[2] serotonin,[3] nitric oxide,[4,5] and vasopressin (VP)[6–8] have been of particular interest. The humoral contributors to the efferent limb of neurally mediated syncopal events most importantly include epinephrine,[9] norepinephrine,[9] and VP.[10–12] It is evident that VP has been in the main area of interest. Its levels have been reported to increase during spontaneous and tilt-induced syncope. The results of the relative studies, however, are not entirely consistent. The exact role of VP in the pathophysiology of vasovagal syncope is not yet clarified. Endothelium on the other hand is a well-appreciated source of potent vasoactive agents. However, studies on its role in vasovagal syncope have been confined to endothelin[13,14] and nitric oxide.[4,5] We are not aware of any study investigating the role of the potent vasoactive end-products of arachidonic acid metabolism in vasovagal syncope. Our study examines the changes in plasma levels of VP, thromboxane (TXA2), and prostacyclin (PGI2) in syncopal patients undergoing a head-up tilt test and compares these changes between subjects with positive and negative test results.

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METHODS

Patients: Thirty-four consecutive patients (18 men, mean age 38 ± 21 years [range 14 to 70]) with a history of recurrent episodes of unexplained syncope suggestive of vasovagal origin were enrolled in the study. Syncope was defined as an abrupt loss of consciousness with the inability to maintain postural tone. A comprehensive physical examination, neurologic work-up, and detailed noninvasive cardiovascular examination (12-lead electrocardiogram, 2-dimensional and Doppler echocardiograms, 24-hour electrocardiographic Holter monitoring, and bilateral bedside and upright carotid sinus massage) had failed to identify the cause of syncope. The mean number of syncopal attacks per patient was 4 ± 3 (range 2 to 8), with the last episode occurring within the last 6 months.

Head-up tilt-table test: The head-up tilt-table test was performed between 09:00 and 12:00 A.M. after overnight fasting or 3 hours after a light caffeine-free meal in a quiet, slightly darkened room. Patients were not allowed to become dehydrated. All vasoactive drugs were withheld for 5 days before the tilt study. The test was performed on an electrically adjustable tilt table. When tilted, the patients were supported by belts across the waist and hip and by a foot support at the base of the tilt table. The electrocardiogram was monitored throughout the test and a single-lead electrocardiogram was obtained every 5 minutes or when symptoms occurred. A standard mercury sphygmomanometer was used to measure blood pressure every minute throughout the test. A venous catheter was

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Vascular Surgery

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Oxygen Free Radicals and the Effect of a Free Radical Scavenger in Patients with Intermittent Claudication

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ABSTRACT

Oxygen free radicals (OFRs) are implicated in tissue injury during postischemic reperfusion and play an important role in the pathogenesis of atherosclerosis. In patients with intermittent claudication the ischemia-reperfusion phenomenon could be reproduced after exercise, thus influencing the evolution of chronic peripheral arterial occlusive disease (CPAOD). The aim of this study was to investigate the behavior of OFRs and the effect of a free radical scavenger in patients with stage II_B CPAOD. Malondialdehyde (MDA), a reliable index of OFRs production, was measured in the serum of 19 patients with stage II_B CPAOD and 42 healthy controls. The blood samples were collected from a foot vein in resting condition and during reperfusion after 5 minutes of provoked ischemia. These measurements were repeated after 3 and 12 weeks of oral treatment with the free radical scavenger trimetazidine hydrochloride (60 mg daily). Statistical analysis of the findings revealed that resting MDA was significantly higher in claudicants when compared to the healthy controls (1.247 ±0.25 μmol/L vs 1.021 ±0.278 μmol/L, $p<0.005$). During postischemic reperfusion (PIR) the MDA levels were significantly

(continued on next page)

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A/A : 55

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Heat Production of Atherosclerotic Plaques and Inflammation Assessed by the Acute Phase Proteins in Acute Coronary Syndromes

Christodoulos Stefanadis, Leonidas Diamantopoulos, John Dernellis, Emanuel Economou, Eleftherios Tsiamis, Konstantinos Toutouzas, Charalambos Vlachopoulos and Pavlos Toutouzas

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C. Stefanadis, L. Diamantopoulos, J. Dernellis, E. Economou, E. Tsiamis, K. Toutouzas, C. Vlachopoulos and P. Toutouzas. Heat Production of Atherosclerotic Plaques and Inflammation Assessed by the Acute Phase Proteins in Acute Coronary Syndromes. *Journal of Molecular and Cellular Cardiology* (2000) 32, 43–52. Several studies have shown that inflammation plays an important role in the pathogenesis of coronary heart disease (CHD). Serum amyloid A (SAA) and C-reactive protein (CRP) reactants of the acute phase of inflammation, have been shown to be increased in patients with CHD. Recently *ex vivo* studies demonstrated that some types of atherosclerotic plaques show substantially warmer regions. A catheter-based technique has been developed to measure the temperature of human arteries *in vivo*. Therefore, the aim of the present study was to measure the luminal surface temperature in patients with CHD and to correlate it with the acute phase proteins in order to discriminate the role of inflammation in heat production in acute coronary syndromes.

Sixty patients were studied with CHD (20 with stable angina, 20 with unstable angina and 20 with acute myocardial infarction) and 20 sex- and age-matched controls without coronary artery disease, by measuring plasma levels of SAA, CRP, plasma lipids and intracoronary arterial luminal wall temperature. Intracoronary temperature was measured with a thermography catheter developed in our Institution: a thermistor probe with a temperature accuracy of 0.05°C, was attached at the distal end of a long 3F polyurethane shaft.

It was found that the median temperature differences at the site of the lesion from the core temperature was increased in patients with unstable angina (1.025°C) and acute myocardial infarction (2.150°C) compared with stable angina (0.300°C), $P<0.001$ for each comparison. Furthermore, stable angina has increased temperature differences compared with controls (0.200°C, $P<0.001$). There were very good correlations between CRP and SAA with the temperature ($r=0.796$, $P=0.01$ and $r=0.848$, $P=0.01$, respectively).

Local heat at the site of lesion is increased in patients with acute coronary syndromes and may arise from an aggressive inflammatory response occurring in these situations. The sensitive measurement of plaque temperature as a prognostic marker may be useful in the management of coronary heart disease.

Key Words: Acute coronary syndromes; Temperature; Inflammation; Acute phase reactants.

Introduction

Data are being accumulated at the presence of inflammation in patients with acute coronary syndromes.[1,2] Preliminary studies performed on human carotid atherosclerotic plaques suggest that unstable plaques might be detected in the future by their temperature heterogeneity,[3] which helps

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A/A : 56

Clinical Investigation and Reports

Increased Proinflammatory Cytokines in Patients With Chronic Stable Angina and Their Reduction By Aspirin

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Background—Proinflammatory cytokines released by injured endothelium facilitate interaction of endothelial cells with circulating leukocytes and thus may contribute to development and progression of atherosclerosis. We investigated whether cytokines and C-reactive protein (CRP) are indicative of myocardial ischemia or of diseased vessels and whether they are influenced by aspirin treatment in patients with chronic stable angina.

Methods and Results—Plasma macrophage colony stimulating factor (MCSF), IL-1b, IL-6, and CRP were measured in 60 stable patients after 48-hour Holter monitoring and in 24 matched controls. All patients had angiographic documentation of disease and positive exercise ECGs. Patients with ischemia on Holter monitoring (n=40) received aspirin or placebo in a 6-week, randomized, double blind, crossover trial. Blood sampling was repeated at the end of each treatment phase (3 weeks). Compared to controls, patients had more than twice median MCSF (800 versus 372 pg/mL), IL-6 (3.9 versus 1.7 pg/mL), and CRP (1.25 versus 0.23 mg/L) levels ($P<0.01$ for all comparisons). MCSF was related to ischemia on Holter monitoring ($P<0.01$), to low ischemic threshold during exercise ($P<0.01$), and together with IL-1b to number of diseased vessels ($P<0.05$). MCSF, IL-6, and CRP were all reduced after 6 weeks of aspirin treatment ($P<0.05$).

Conclusions—These findings suggest that cytokines are associated with both ischemia and anatomic extent of disease in patients with stable angina. Reduced cytokine and CRP levels by aspirin may explain part of aspirin's therapeutic action. (*Circulation*. 1999;100:793-798.)

Key Words: interleukins ■ atherosclerosis ■ coronary disease ■ ischemia ■ aspirin

Macrophage colony stimulating factor (MCSF) and IL-1b released by injured endothelium[1–5] promote the interaction of endothelial cells with circulating leukocytes[6,7] and may thus contribute to the development and progression of atherosclerosis.[6–13] MCSF induces the synthesis by endothelial cells of monocyte chemotactic protein 1, which enhances the migration of monocytes into the subendothelial layer.[7] MCSF additionally increases cholesterol uptake by macrophages[10,11] and delays their apoptosis,[12] resulting in foam cell formation, the hallmark of atherogenesis. MCSF, acting in synergy with IL-1b, induces the activation and proliferation of monocytes/macrophages.[3–6] These 2 cytokines determine a further release of cytokines from vascular cells,[3–7] including IL-6[13–15] which may be involved in smooth muscle cell proliferation.[16,17] One of the mechanisms involved in vascular cell activation and proliferation induced by MCSF[3,18] and IL-1b[19] is mediated by cyclooxygenase activity.

The plasma levels of MCSF, IL-6, and C-reactive protein (CRP) have been found elevated in patients with unstable angina[20–22] and acute myocardial infarction[22–24] but have not been carefully investigated in patients with chronic stable angina. We hypothesized that MCSF, IL-1b, and IL-6 plasma levels in patients with chronic stable coronary artery disease might be associated with the anatomic extent of disease and that aspirin administration might reduce cytokine plasma levels.

Methods

Study Population

Sixty consecutive patients (53 men, 7 women, mean age 55±5 years, range 38 to 67 years) with clinically stable angina were enrolled. The inclusion criteria were effort angina of at least 1 year's duration; exercise-induced ischemia; presence of luminal diameter stenosis >50% of 1 or more epicardial coronary arteries at angiography, performed within 1 year of enrolment; and informed consent. Exclusion criteria included ECG evidence of left ventricular hypertrophy or left bundle branch block; acute coronary events, coronary angioplasty, or surgery within the previous 6 months; cerebral vascular disease; peripheral vascular disease; diabetes mellitus; previous coronary artery bypass graft surgery; impaired renal or liver function; bleeding tendencies; allergy to aspirin; malignant or known inflammatory diseases; and age >80 years. On inclusion into the study, antiplatelet drugs were withdrawn for 2 weeks. Holter

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SHORT REPORT

Lipid peroxidation in healthy fetuses, preterm and fullterm neonates

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Key words: fetuses; fullterm neonates; lipid peroxidation; malondialdehyde serum concentration; preterm neonates

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Lipid peroxides are stable products of oxygen free radicals reaction with the polyunsaturated fatty acids of cell membranes. The latter are particularly susceptible to oxidative reactions, e.g. the rapid increase in cell oxygen tension with the onset of respiration at birth (1, 2). Nevertheless, it has not been possible to demonstrate unequivocally that hyperoxic exposure increases *in vivo* lipid peroxidation, due to insensitivity or nonspecificity of available measuring methods.

This study was based on the hypothesis that lipid peroxidation, quantitated by measurement of malondialdehyde (MDA) serum concentrations, depends on the mode of delivery and changes with time from birth in healthy fullterm and preterm neonates. The existence of a variation or a correlation between maternal and fetal MDA concentrations was investigated as well.

Material and methods

Following approval by the Hospital Ethics Committee and informed consent obtained from the parents, 20 healthy fullterm and 13 healthy preterm neonates, as well as 20 'mother- second trimester fetus' pairs, were included in the study.

All fullterms were appropriate for gestational age (AGA) born following an uncomplicated pregnancy and delivery (second stage not lasting more than 10 min) either vaginally (VD, $n=10$) or by cesarean section (CS, $n=10$) because of a previous CS. All infants were breastfed and five received supplemental formula. Preterm neonates were AGA with mean gestational age 32.6±2.5 weeks and mean birth weight 1643±401 g. Six preterms were born by VD and seven by CS because of a previous CS. All were healthy and only two required supplemental oxygen administration. Infants received maternal milk and special preterm formula. Blood was drawn from all neonates on days 1 and 4 of life.

In the 'mother- second trimester fetus' group venous blood was drawn from the mother and, immediately thereafter, fetal blood was collected by cordocentesis performed for various purposes in the 18–24 gestational week. Mean maternal age was 27.3±7.4 years and mean gestational age 21.3±1.4 weeks. Ultrasonography proved all fetuses to be AGA and failed to reveal any recognizable malformations. Placental morphology and struc-

Abbreviations:
AGA: appropriate for gestational age; VD: vaginal delivery; CS: cesarean section; MDA: malondialdehyde.

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Circulating endothelin-3 and prolactin concentrations in healthy lactating women during the early puerperium

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Abstract

Objective: To study the association between the circulating concentrations of endothelin-3 and prolactin in the early puerperium.
Design: Prospective clinical study, including twenty-five healthy puerperal women breast-feeding their healthy full-term infants.
Methods: Venous blood was drawn on day 1 and 4 post partum, and plasma endothelin-3 and serum prolactin were determined.
Results: Circulating endothelin-3 and prolactin levels on day 4 did not differ significantly from the corresponding levels on day 1. However, a significant negative correlation was found on day 4 between endothelin-3 and prolactin values ($r = -0.688$, $P < 0.001$) and an even stronger negative association existed between the net change in endothelin-3 from days 1 to 4 and the corresponding change in prolactin values ($r = -0.732$, $P < 0.001$).
Conclusions: On the fourth day post partum, lactating healthy women show negative correlation between circulating endothelin-3 and prolactin levels. Whether this indicates a role for endothelin-3 in the control of prolactin secretion in the post partum period remains to be clarified.

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Introduction

Endothelins (ETs), a family of mainly three 21-amino acid peptides (ET-1, ET-2, ET-3), synthesized by three different genes in humans, are principally known for their vasoactive and mitogenic properties (1–3). However, several recent studies indicate that ETs may also participate in neuroendocrine regulation (4). In this respect, immunoreactive ET was found in the hypothalamus (5) as well as in the anterior (6, 7) and posterior (5) pituitary gland. Moreover, it has been reported that [^{125}I]ET binds with high affinity to hypothalamic tissues (8), pituitary fragments (9) and dispersed pituitary cells (10).

Since its discovery, ET-3 has been specifically associated with the nervous tissue (7, 5, 11). Thus it has been reported to be the major ET produced by the pituitary gland in humans (6) and rats (7). Recent studies have shown ET-3 to stimulate the release of luteinizing hormone, follicle-stimulating hormone and thyroid-stimulating hormone from primary monolayer cultures of anterior pituitary cells derived from female rats (12), as well as to exhibit a dose-dependent stimulatory effect on luteinizing hormone-releasing hormone secretion from luteinizing hormone-releasing hormone neurons in male rats (13).

On the other hand, Samson *et al.* (14) demonstrated that ET-3 inhibited in a specific, significant, dose-dependent and reversible fashion prolactin (PRL) release from cultured anterior pituitary cells. In addition, Kanyicska *et al.* (12) provided further evidence for the concentration-dependent decrease of PRL secretion from pituitary cell cultures elicited by ET-3. Nevertheless, the former authors claim in another study that ET-3 also exerts transient stimulatory effects on PRL release in rats (15).

So far, no data exist concerning the relationship between circulating ET-3 and PRL concentrations in various conditions *in vivo*, particularly in humans. The present study was based on the hypothesis that ET-3 may be implicated in PRL release in puerperal women. We investigated the association of blood concentrations of ET-3 and PRL on the first and fourth day after delivery in healthy women.

Materials and methods

The study was approved by the ethics committee of our teaching hospital. Twenty-five healthy post partum

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Endothelin Plasma Levels in Primary Amenorrheic Adolescents Before and After Estrogen Treatment

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OBJECTIVE: *We evaluated the effect of estrogen administration on endothelin (ET) secretion in primary amenorrheic (PA) adolescent girls.*

METHODS: *Fifteen PA adolescents (ten hypergonadotropic, group A; five hypogonadotropic, group B) were treated with estrogen and progestogen tablets. A control group of ten healthy adolescents (group C) was included in the study. The ET 1-21, FSH, and LH plasma levels were tested before treatment (PrT) and immediately after the last estrogen tablet but before the progestogen administration (PoT).*

RESULTS: *A statistically significant difference ($P < .01$) in ET 1-21 plasma values was found between PrT (9.66 ± 0.80 pmol/L) and PoT (7.56 ± 0.89 pmol/L) levels in group A cases. A similar reduction ($P < .05$) was recorded between PrT (8.06 ± 0.46 pmol/L) and PoT (5.59 ± 0.53 pmol/L) ET 1-21 plasma levels in group B cases. Endothelin 1-21 plasma PrT values were higher in both group A and B cases in comparison with controls (6.66 ± 0.44 pmol/L; $P < .01$, $P < .1$, respectively).*

CONCLUSIONS: *Estrogens administered to PA adolescents reduce ET 1-21 plasma levels in both hyper- and hypogonadotropism.* (J Soc Gynecol Invest 1996;3:350–353) Copyright © 1996 by the Society for Gynecologic Investigation.

KEY WORDS: Endothelins, primary amenorrhea, adolescence, estrogens.

Endothelins (ET-1, ET-2, ET-3) are biologically active peptides with 21–amino acid residues. They have been isolated from porcine aortic endothelial cells and produce a potent, long-lasting vasoconstriction.[1] Endothelins are secreted into many biologic fluids, including follicular fluid. It has been suggested that ET-1 found in the follicular fluid may be related to follicular development.[2] Endothelin-1 was shown to inhibit the gonadotropin-supported accumulation of cyclic adenosine monophosphate and progesterone.[3] It has been speculated that ET-1 inhibits the morphologic luteinization of porcine granulosa cells and that ET-1, possibly of intraovarian origin, acts as a luteinization inhibitor and suppresses premature luteinization.[4,5]

Moretto et al[6] indicated a direct action of ET-3 on the LH-releasing hormone (LHRH) neuronal system. However, the effects of ET-3 on LHRH neurons have been associated with a functional arachidonic acid metabolic pathway, indicating the role of prostaglandins in this kind of intracellular event. Finally, ET is as potent as hypothalamic GnRH in the stimulation of gonadotropin release. Endothelin and GnRH have a common signal-transduction mechanism, and ET itself can act as a neuropeptide to regulate anterior pituitary function.[7]

The association of 17β-estradiol and ET-1 has been examined in in vitro studies. It has been suggested that 17β-estradiol may inhibit Ca^{2+} influx in the cell.[8] On the other hand, it has been demonstrated that increased intracellular Ca^{2+} stimulates ET-1 mRNA expression,[9] leading to increased ET production. Nevertheless, no studies exist examining the effect of estrogen administration on ET secretion in humans.

We studied the hypothesis that estrogens may inhibit vasoconstriction produced by ET. The aim of the present study was to evaluate the effect of estrogen administration on ET secretion in primary amenorrheic (PA) adolescents.

MATERIALS AND METHODS

After providing informed consent, 15 PA adolescent girls aged 16–18 years were divided into two groups based on the serum gonadotropin levels of FSH and LH. Group A consisted of ten hypergonadotropic girls with the syndrome of gonadal dysgenesis, and group B included five hypogonadotropic subjects suffering from hypothalamic amenorrhea. A control group (group C) contained ten adolescent girls with normal gonadotropin serum levels and normal menstrual function, who participated voluntarily. Table 1 presents data concerning the three groups included in the study. The study was approved by the Ethics Committee of our teaching hospital.

The following treatment was applied to group A and B subjects: Premarin (Wyeth-Ayerst, St. David's, PA) tablets 0.625 mg orally three times daily for 14 days, and Provera (Upjohn, Kalamazoo, MI) tablets 5 mg twice daily for the

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Endothelin 1-21 Plasma Concentrations in Children and Adolescents with Insulin-Dependent Diabetes Mellitus

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ABSTRACT

This study is based on the hypothesis that endothelins (ETs), which are 21-amino acid peptides with vasoactive and proliferative properties, could be implicated in the development of complications of insulin dependent diabetes mellitus (IDDM) in children and adolescents. We determined plasma ET 1-21 concentrations by radioimmunoassay in 59 patients with IDDM (32 male, 27 female) and in 41 healthy siblings (20 male, 21 female) and investigated the association of ET 1-21 concentrations with age, sex, control of diabetes (expressed as % of glycosylated hemoglobin), duration of disease and presence of complications. Plasma ET 1-21 concentrations (mean ± SEM) were 14.12 ± 0.30 pg/ml in IDDM and 15.34 ± 0.47 pg/ml in healthy siblings. The difference was statistically significant (p=0.01) after controlling for age and sex by multiple logistic regression. In the group with IDDM, analysis of covariance showed duration of disease to be the only variable associated with ET 1-21 values (b=-0.2179 pg/ml/yr, p=0.04). It is concluded that in youngsters with IDDM ET plasma concentrations are lower than in healthy controls, negatively associated with duration of the disease and not directly implicated in diabetic angiopathy.

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KEY WORDS

endothelins, plasma concentrations, children, adolescents, insulin-dependent diabetes mellitus, angiopathy

INTRODUCTION

Endothelins (ETs) are potent, 21 amino acid peptides /1/ with vasoactive and proliferative effects on vasculature /1-3/. In humans, three separate isopeptides (ET-1, ET-2, ET-3) are synthesized by three different genes /4/. ET-1 is produced by endothelial cells, ET-2 is produced in the kidney and ET-3 is associated with nervous tissue /5/.

It is generally accepted that the production of ETs by endothelial cells is stimulated by a rise of intracellular calcium (Ca^{2+}) /4,6/. Intracellular Ca^{2+} has been found to increase /4/, but also to decrease /7/, causing changes in ET levels, through activation of phospholipase C and inhibition of inositol trisphosphate (IP_3) uptake respectively, in the polyol or sorbitol pathway, activated by the presence of excessive blood glucose levels /8,9/ and implicated in the pathogenesis of tissue complications in diabetes mellitus (DM) /7,10/.

Plasma ET levels have been measured in healthy adults as well as in various pathologic conditions including insulin or non-insulin dependent DM (IDDM, NIDDM) /11-20/. Nevertheless, the findings concerning DM are contradictory in the literature. Plasma ET levels have been measured by radioimmunoassay in healthy fetuses /21/, preterm /22/ and fullterm /23/ neonates as well as in healthy children up to 15 years /24/. ET levels have been found nearly constant at all ages from childhood to adulthood after the first three months of life /24/. No studies have previously been carried out to

A/A : 61

Short communication

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Endothelin 1-21 plasma levels in fetuses at 18–24 weeks of gestation

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Introduction

Endothelins (ETs) are potent vasoconstrictor 21-aminoacid peptides, which were originally isolated from supernatants of cultured porcine aortic endothelial cells [21]. In humans, endothelin-1 (ET-1) is secreted from endothelial cells and constrict a variety of blood vessels and control vascular tonus and local blood flow [1]. The latter considered very important for oxygen transfer, especially across the placenta. It has been reported that the placenta and the amniotic endothelial cells produce a large amount of ET-1 [17, 18]. Furthermore the placenta is a rich source of endothelin receptors [4] and ET-1 has been identified as an important pressor substance in the fetoplacental cotyledon [19]. Thus the overproduction of ET or increased placental receptor concentrations could result in limitation of fetoplacental blood flow and its sequelae, i.e. intrauterine growth retardation (IUGR) [10].

This study intended to investigate the physiological implications of ETs in the fetus by a) establishing normal plasma ET ranges for non IUGR fetuses of 18–24 weeks gestational age (who undergo cordocentesis for various reasons), b) by seeking a possible correlation among fetal and maternal ET levels. Future investigations concerning ET levels in pathological states might contribute to the elucidation of the pathophysiology of intrauterine growth retardation.

Material and methods

Forty "mother-fetus" pairs were included in this study after informed consent was obtained from the pregnant women. The study was also approved by the Ethical Committee of our Teaching Hospital. Cordocentesis and fetal blood sampling was indicated in 13 cases because of heterozygous β-thalassemia in both parents, in 4 cases because of advanced maternal age (> 35 years) and in 3 cases because of possible maternal rubella infection. Maternal age ranged from 17–38 years (mean ± SD: 27.3 ± 7.4 years). Gestational age ranged from 18–24 weeks (mean ± SD: 21.3 ± 1.4 weeks) and fetal growth was appropriate for gestational age (AGA), which was verified by ultrasonographic measurement of biparietal diameter (BPD) and femur length (FL). Furthermore no recognizable malformations were detected in the fetuses using ultrasound. Placental morpholo-

Curriculum vitae

Dr ARIADNE MALAMITSI-PUCHNER *was born in Athens. She studied Medicine at the Athens University of Athens and graduated in 1973. She did her training in Pediatrics and Neonatology at the University Clinics of Vienna, Munich and Philadelphia. Since 1978 she has been working at the Neonatal Department and the Intensive Care Unit of the "Alexandra" University and State Maternity Hospital in Athens, where she is now Assistant Professor. Most of her research activities are concerned with Perinatology.*

A/A : 62

Original Paper

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Endothelin 1-21 Plasma Concentrations on Days 1 and 4 of Life in Healthy and Ill Preterm Neonates

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Key Words
Endothelins, plasma concentrations
Neonate, preterm
Respiratory distress syndrome
Intraventricular hemorrhage
Hypoxia
Asphyxia
Vasoconstriction
Cell proliferation

Abstract
Endothelins (ETs) are highly vasoconstrictive 21-amino acid peptides possessing also cell-proliferative properties. They have been implicated in a variety of perinatal pathologic conditions, and their plasma concentrations have been found elevated in humans at birth. The purpose of this study was to determine ET 1-21 plasma concentrations in healthy and ill preterm infants and to investigate possible concentration changes with time from birth in cases of normal and abnormal adaptation to extrauterine life. The study comprised 36 preterm infants. Twenty-eight, comprising group A, were healthy (22/28) or minimally affected (6/28) and 8, comprising group B, were moderately (2/8) or severely ill (6/8) requiring continuous positive airway pressure or intermittent positive pressure ventilation as well as surfactant administration. All infants in group B had intraventricular hemorrhage grade ≥ II. Venous blood from all neonates was drawn on days 1 and 4 and ET 1-21 plasma concentrations were determined by radioimmunoassay (Amersham kit RPA 5559). ET 1-21 plasma concentrations were on day 1: 16.25 ± 8.14 and 21.81 ± 5.87 and on day 4: 12.89 ± 4.56 and 16.16 ± 5.43 pmol/l, for groups A and B, respectively. The statistical analysis showed a significant reduction in plasma ET concentrations on day 4 in both groups ($p = 0.009$ and $p = 0.025$, respectively). Nevertheless, ET 1-21 plasma concentrations were on day 4 significantly higher in ill preterm infants presenting symptoms from tissues involved in the elimination of ETs from the circulation as well as in their production.

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Original Paper

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Endothelin 1–21 Plasma Levels on the First and Fourth Postpartum Day in Normal Full-Term Neonates

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Key Words
Endothelins, plasma levels
Neonate, full-term

Abstract
Endothelins (ETs), recently discovered and highly vasoactive substances, have been implicated in the pathogenesis of various perinatal problems, such as preeclampsia, intrauterine growth retardation, intraventricular hemorrhage, pulmonary hypertension and necrotizing enterocolitis. Although fetal ET levels have been measured at birth, reference ET values for healthy newborns in the first days of life have not been established. The purpose of this study was to determine in normal healthy neonates ET 1–21 plasma values on day 1 and 4 postpartum and to investigate possible changes after adaptation of the newborn to extrauterine life. The study comprised 20 healthy full-term neonates, born after vaginal delivery (n = 10), or cesarean section (CS; n = 10) because of a previous CS. Venous blood was drawn on day 1 and 4 from all neonates and ET 1–21 levels were determined in the plasma by radioimmunoassay (Amersham kit RPA 5559). ET 1–21 values were on day 1 11.83 ± 2.39 pmol/l (n = 20) and on day 4 9.45 ± 1.88 pmol/l (n = 20). The statistical analysis showed a significant reduction of plasma ET levels on day 4 ($p = 0.004$), but no influence of the mode of delivery on plasma ET levels. In conclusion irrespective of the mode of delivery the high ET 1–21 plasma levels on day 1 postpartum are significantly reduced on day 4 of life.

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A ^{125}I-radioimmunoassay for diethylstilbestrol in serum of patients with prostatic cancer treated with stilphostrol

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Key words: Diethylstilbestrol; Prostatic cancer; Radioimmunoassay; Radioiodination; Steroids

Summary

A new radioimmunoassay for determining diethylstilbestrol in serum using *N*-(4′-OH-[3′-^{125}I]iodophenethyl)-6-(4-*O*-diethylstilbestryl)-hexanamide as a radiotracer and a double antibody as a separation reagent is described. The radiotracer is prepared by synthesizing 6-(4-*O*-diethylstilbestryl)-hexanoic acid and coupling its succinimidyl ester with mono-[^{125}I]tyramine in tetrahydrofuran (16 h, 20–22°C). The standard curve is linear (semi-log transformation) and the assay is sensitive (< 0.022 pmol/tube), reproducible (intra- and interassay coefficient of variation values, 5.3 and 8.1%, respectively), and accurate (recovery values, 95–101%), with a non-specific binding less than 3.2%. Diethylstilbestrol concentrations measured in sera of nine patients with prostatic cancer by the proposed assay ranged from 0.170 to 2.517 μmol/l, which corresponded to an only three-fold dosage variation. In all cases tested, dosing was adequate to retain markers of prostatic cancer in serum within accepted limits; nevertheless, individualization of dosing may be necessary to minimize toxicity.

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Preparation of the N-(4′-hydroxy-[3′-^{125}I]iodophenethyl)-6-(4-O-diethylstilbestryl)hexanamide for diethylstilbestrol radioimmunoassays

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The synthesis of a radioiodinated diethylstilbestrol (DES) derivative is described. This derivative was prepared by coupling the previously synthesized active ester of 6-(4-O-diethylstilbestryl)hexanoic acid with mono-[^{125}I]iodotyramine in dry tetrahydrofuran (20 to 22 C, 16 hours). The mono-[^{125}I]iodotyramine was prepared using a chloramine-T method and purified by paper electrophoresis. The final product, N-(4′-hydroxy-[3′-^{125}I]iodophenethyl)-6-(4-O-diethylstilbestryl)hexanamide, was separated by thin-layer chromatography (cyclohexane/ethanol/NH_4OH 2.5 N/acetone; 40:50:5:20, v/v/v/v); it was stable for 2 months in ethanol at 4 C and had a specific activity higher than 540 Ci/mmol. The [^{125}I]DES amide synthesized was found to retain the immunoreactivity of DES, since it competed with [^{3}H]DES or DES in an in vitro radioimmunoassay system for the binding sites of a rabbit anti-DES antibody; thus, it seems to be capable of replacing the tritiated tracer used so far in DES radioimmunoassays. (*Steroids* **57**:27–31, 1992)

Keywords: steroids; diethylstilbestrol, RIA; radioiodination, for DES RIA; radioimmunoassay, DES; N-(4′-hydroxy-[3′-^{125}I]iodophenethyl)-6-(4-O-diethylstilbestryl)hexanamide

Introduction

Diethylstilbestrol (DES), a synthetic nonsteroidal estrogen, has been widely used, mainly as an antiandrogenic agent for the treatment of patients with prostatic carcinoma[1,2] and as a growth promoter in cattle.[3]

The tritium-labeled tracers commonly used so far in all of the proposed DES radioimmunoassays[4–7] have some intrinsic problems mainly related to their instability,[4,6] low specific activity,[6,7] and detection difficulties.[8] On the other hand, the radioiodinated histamine amide of the DES derivative 4-(4-O-diethylstilbestryl)butanoic acid reported in the literature[9] does not look promising for utilization as a tracer in a DES radioimmunoassay due to its low specific activity and limited immunoreactivity.

We describe the synthesis of the N-(4′-hydroxy-[3′-^{125}I]iodophenethyl)-6-(4-O-diethylstilbestryl)hexanamide. This radioiodinated derivative, which was prepared by coupling the previously synthesized active ester of 6-(4-O-diethylstilbestryl)hexanoic acid with mono-[^{125}I]iodotyramine, was obtained in very good yield and seemed to retain the DES-related immunoreactivity, which is necessary for its utilization as a tracer in DES radioimmunoassays.

Experimental

Materials

All reagents were analytic grade. The water used was doubly distilled. Gelatin was obtained from BDH Chemicals Ltd. (Poole, England). Diethylstilbestrol, tyramine, bovine serum albumin (BSA), and N-hydroxysuccinimide (NHS) were all obtained from Sigma Chemical Co. (St. Louis, MO, USA). Dicyclohexylcarbodiimide (DCC) was obtained from Ferak-Berlin (West Berlin, Germany), and 6-bromohexanoic acid was obtained from Aldrich Chemical Co. (Milwaukee, WI, USA). The carrier-free Na ^{125}I solution (17 kCi/g; radiochemical purity,

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Direct radioimmunoassay for diethylstilbestrol in serum

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A radioimmunoassay method for the measurement of diethylstilbestrol directly on 50 µl of human serum was established using a tritium-labeled radioligand and a double antibody as a separation reagent. The assay was sensitive (<0.17 µg/L), reproducible (intra-assay and interassay coefficient of variation values, 8.14% and 8.25%, respectively), and accurate (recovery up to 95%). Factors influencing the assay are identified and discussed. (*Steroids* **55**:545–550, 1990)

Keywords: steroids; diethylstilbestrol; radioimmunoassay; prostatic cancer.

Introduction

Diethylstilbestrol (DES), a synthetic nonsteroidal estrogen, has been used in endocrine therapy for managing advanced prostatic cancer[1,2] and as a growth promoter in cattle.[3] In the past, it was used in an unsuccessful effort to prevent miscarriages as well as some of the later complications of pregnancy;[4,5] unfortunately, the offspring of women who were treated with DES for that purpose are now found to be more susceptible to vaginal cancer and other gynecologic and urologic disorders,[6] with effects possibly continuing into the third generation.

A few immunoassay methods for DES have been developed.[7–10] The concentration of DES in the serum of patients under treatment schedules using high doses of this estrogen (>200 mg daily) is high enough to be detectable directly by radioimmunoassay (RIA).[8] However, to decrease side effects,[1] recent trends recommend much lower doses (3 mg DES daily), providing low serum DES concentrations.[11] In this case, an initial extraction step is required before measuring DES by RIA, which is considered time-consuming and error-prone due to correction for recovery.[7,9]

We describe a direct, sensitive, simple, and easy to perform double-antibody RIA for measuring low DES concentrations, especially in the serum of patients with prostatic cancer under treatment with this estrogen.

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Received February 15, 1990; accepted June 8, 1990.

Experimental

Reagents

All reagents were analytic grade. The water used was doubly distilled. Gelatin was obtained from BDH Chemicals Ltd. (Poole, England). Human serum albumin (HSA) and nonradioactive DES were obtained from Sigma Chemical Co. (St. Louis, MO, USA). Freund's complete adjuvant was obtained from Difco Laboratories (Detroit, MI, USA). All the other reagents were obtained from Merck Schuchardt (Darmstadt, FRG), except as otherwise indicated.

Buffers

The following phosphate buffers were used: pH 7.4, 0.01 M, 9 g NaCl/L, 1 g NaN_3/L (buffer A); pH 7.4, 0.01 M, 9 g NaCl/L, 1 g NaN_3/L, and 0.5 g gelatin/L (buffer B); pH 7.4, 0.01 M, 9 g NaCl/L, 1 g NaN_3/L, and 0.5 g HSA/L (buffer C); and pH 7.4, 0.01 M, 9 g NaCl/L, 1 g NaN_3/L, and 70 g HSA/L (buffer D).

Tracer solution

[Monoethyl-^{3}H]diethylstilbestrol in toluene (70 to 120 Ci/mmol), the radiotracer solution, was purchased from the Radiochemical Center (Amersham, UK); aliquots were diluted with ethanol to a final concentration

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Evidence that vitamin E may reduce the levels of prostacyclin and thromboxane in the blood of premature neonates*

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Abstract

Vitamin E has been shown to interfere with prostaglandin production *in vitro* and in animals *in vivo*. The purpose of this study was to examine whether vitamin E administered i.m. at 20 mg/kg/day to healthy premature infants of 30–34 weeks gestational age for 3 consecutive days affects the levels of prostacyclin and thromboxane. Eleven infants were included in the experimental group and 14 in the control group. The stable metabolites of prostacyclin and thromboxane, i.e. 6-ketoPGF1α and TXB2, respectively, were measured by radioimmunoassay, on days 1 and 4 in both groups. The statistical analysis indicates that 6-ketoPGF1α and TXB2 are significantly lower on day 4 in the experimental group ($p < 0.01$), but not in the control group. It appears that vitamin E may reduce significantly the plasma levels of 6-keto PGF1α and TXB2 without causing any of the reported side effects to premature infants.

Key words
Premature neonates – vitamin E – prostaglandins

Introduction

Vitamin E, a well known antioxidant, has been widely used in neonatology for the prevention of various pathological conditions of the premature infant, such as retinopathy of prematurity (Hittner et al. 1984; Johnson et al. 1989), bronchopulmonary dysplasia (Ehrenkranz et al. 1987, Wynder et al. 1981), hyperbilirubinemia (Gross 1979), anemia (Ritchie et al. 1968) and intracranial hemorrhage (Chiswick et al. 1983).

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On the other hand, prostaglandins play a complex and not yet fully understood role in neonatal diseases. Apart from the intensively studied action on the ductus arteriosus, experiments have shown the involvement of prostaglandins in lung circulation, platelet aggregation, and cerebral, renal and mesenteric arterial function (Friedman and Fitzpatrick 1980; Heymann 1987). Substances that inhibit oxidative reactions might be expected to affect prostaglandin synthesis (Zinser and Davis 1978). Thus, vitamin E as an antioxidant may prevent the oxidation of arachidonic acid in the biosynthetic pathway leading to prostaglandins. Nevertheless, experiments *in vitro* (Vanderbrook and Lands 1973; Gwebu et al. 1980) and in animals *in vivo* (Likoff et al. 1981, Carpenter 1982) have rendered contradictory results considering the inhibition of prostaglandin synthesis from arachi-

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ΕΠΙΘΕΩΡΗΣΗ ΚΛΙΝΙΚΗΣ ΦΑΡΜΑΚΟΛΟΓΙΑΣ
ΚΑΙ ΦΑΡΜΑΚΟΚΙΝΗΤΙΚΗΣ [illegible]
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Φθορισμοανοσοαναλύσεις (FIA). Η Εναλλακτική Λύση στην Ευρεία Εφαρμογή των Ραδιοανοσοαναλύσεων (RIA) (I)

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Περίληψη: *Οι δείκτες φθορισμού και οι ανοσοαναλύσεις που βασίζονται σ' αυτούς έχουν κινήσει τελευταία το ενδιαφέρον της κλινικής ανοσολογίας, γιατί αποτελούν εναλλακτική λύση στην ευρεία εφαρμογή των ραδιοανοσοαναλύσεων (RIA) και συγχρόνως μέσα ανάπτυξης ταχέων και ευαίσθητων προσδιορισμών. Το βασικότερο πλεονέκτημά τους είναι η δυνατότητα ανάπτυξης ομογενών φθορισμοανοσοαναλύσεων. Αναλύσεις που βασίζονται στην πόλωση του φωτός φθορισμού, την απόσβεση της έντασης του φθορισμού ή στην ενζυματική απελευθέρωση δεικτών φθορισμού εφαρμόζονται στον έλεγχο των επιπέδων συγκεντρώσεων των φαρμάκων στα βιολογικά υγρά. Η ανάπτυξη κατάλληλων μεθόδων διαχωρισμού που βασίζονται στην τεχνική της στερεάς φάσης διευκολύνει τη χρήση του φθορισμού σ' ετερογενείς αναλύσεις που, γενικά, βρίσκουν εφαρμογή στον προσδιορισμό πρωτεϊνών, ιών, ακόμη και μικρομοριακών ενώσεων. Τελευταία, νέοι δείκτες φθορισμού οδήγησαν στην ανάπτυξη νέων μεθόδων. Έτσι, η χρήση φυκοβιλινοπρωτεϊνών ή παραγώγων της πορφυρίνης, που παρουσιάζουν μεγάλο μήκος κύματος εκπομπής και μεγάλη μετατόπιση Stokes ή η χρήση μετάλλων, όπως είναι μερικές λανθανίδες, υπόσχονται την ανάπτυξη περισσότερο ευαίσθητων μεθόδων.*

ΓΕΝΙΚΑ ΠΕΡΙ ΦΘΟΡΙΣΜΟΑΝΟΣΟΑΝΑΛΥΣΕΩΝ

Εισαγωγή

Οι δεσμευτικές αναλύσεις αποτελούν πλέον καθημερινή πρακτική στην κλινική χημεία εξαιτίας της εξειδίκευσης, της ευαισθησίας, της επαναληψιμότητος και της ευκολίας εφαρμογής τους. Χρησιμοποιούνται για τον προσδιορισμό ουσιών που βρίσκονται σ' εξαιρετικά χαμηλές συγκεντρώσεις στα βιολογικά υγρά, όπως είναι πρωτεΐνες (ένζυμα, υποδοχείς, αντισώματα), ορμόνες (στεροειδή, ορμόνες θυρεοειδούς, ορμόνες πεπτιδικής φύσης), φάρμακα και μικροοργανισμοί.

Οι ραδιοανοσοαναλύσεις (RIA) είναι οι ευρύτερα χρησιμοποιούμενες δεσμευτικές αναλύσεις. Αν και είναι εξαιρετικά ευαίσθητες και ακριβείς, παρουσιάζουν αρκετά μειονεκτήματα. Έτσι, η ραδιενεργή ακτινοβολία μπορεί να αποβεί επιζήμια για τον ανθρώπι-

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ΕΠΙΘΕΩΡΗΣΗ ΕΛΛΗΝΙΚΗΣ ΦΑΡΜΑΚΟΛΟΓΙΑΣ
ΚΑΙ ΦΑΡΜΑΚΟΚΙΝΗΤΙΚΗΣ Β. 115 122 (1987)
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Φθορισμοανοσοαναλύσεις (FIA). Η Εναλλακτική Λύση στην Ευρεία Εφαρμογή των Ραδιοανοσοαναλύσεων (RIA) (II)

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Ανοσοαναλύσεις πόλωσης φθορισμού (FPIA)

Ο βαθμός πόλωσης του φωτός εκπομπής, που προέρχεται από αντιγόνο που προηγούμενα διεγέρθηκε με πολωμένο φως, εξαρτάται από το χρόνο αποδιέγερσης του φθορίζοντα δείκτη (4-5 nsec για την ισοθειοκυανιούχο φλουορεσκεΐνη) και από τη δυνατότητα περιστροφής του επισημασμένου αυτού αντιγόνου. Η τυχαία περιστροφική κίνηση των μικρών μορίων μειώνει το βαθμό πόλωσης του φωτός εκπομπής. Όταν το αντιγόνο, που επισημάνθηκε με το φθορίζοντα δείκτη, συνδεθεί με το ειδικό αντίσωμα έναντι του αντιγόνου, η δυνατότητα περιστροφής του επισημασμένου αντιγόνου περιορίζεται και ο βαθμός πόλωσης του φωτός εκπομπής αυξάνεται (σχήμα 5). Οι FPIA είναι απλές, ταχείες και ακριβείς, αλλά εξαιτίας των μικρών μεταβολών του βαθμού πόλωσης του φωτός εκπομπής, οι μέθοδοι αυτές δεν εφαρμόζονται για τον προσδιορισμό αντιγόνων που το μοριακό τους βάρος υπερβαίνει τα 20.000 Da (31-33).

Ανοσοαναλύσεις αύξησης φθορισμού

Οι ανοσοαναλύσεις αυτού του τύπου βασίζονται στην αύξηση της έντασης του φθορισμού, καθώς το επισημασμένο αντιγόνο συνδέεται στο ειδικό έναντι αυτού αντίσωμα. Η απόδοση φθορισμού μερικών φθορίζόντων δεικτών (ANS, Dansyl και παράγωγα) αυξάνεται κατά τη σύνδεση με πρωτεϊνικές επιφάνειες εξ αιτίας της μειωμένης πολικότητας στην επιφάνεια των πρωτεϊνών (34). Οι φθορίζοντες δείκτες που είναι κατάλληλοι για τις μεθόδους αυτές είναι αρκετά ευαίσθητοι στη σκέδαση και στο μη ειδικό σήμα φθορισμού που προέρχεται από τον ορό. Έτσι, οι εφαρμογές των μεθόδων αυτών είναι περιορισμένες.

Ανοσοαναλύσεις άμεσης απόσβεσης φθορισμού

Η απόδοση φθορισμού των περισσότερων δεικτών, σ' αντίθεση μ' εκείνους που χρησιμοποιούνται στις ανοσοαναλύσεις αύξησης φθορισμού, ελαττώνεται, όταν συνδέονται ή συζεύγνυνται με πρωτείνες, όταν

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Αρτηριακή Υπέρταση, 3 : 141 - 146, 1994

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Επίδραση της αντιυπερτασικής αγωγής με αναστολέα ασβεστίου στα επίπεδα των ενδοθηλινών του πλάσματος

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Περίληψη

Με σκοπό την αξιολόγηση της επίδρασης της αντιυπερτασικής αγωγής με ισραδιπίνη SRO 5 mg ημερησίως στα επίπεδα των ενδοθηλινών (ΕΤs) του πλάσματος, μελετήθηκαν 15 ασθενείς μετά από περίοδο άνοιχής 15 ημερών από οποιαδήποτε αντιυπερτασική αγωγή και 4 μήνες μετά από μονοθεραπεία με ισραδιπίνη SRO 5 mg άπαξ ημερησίως, συγκριτικά με 10 υγιείς μάρτυρες. Πριν τη φαρμακευτική αγωγή οι συγκεντρώσεις των τιμών των ΕΤs του πλάσματος ήταν στατιστικά μεγαλύτερες στους υπερτασικούς συγκριτικά με τους υγιείς μάρτυρες (8.0 έναντι 5.9 pmol/L, p = 0,0001). Η αγωγή με ισραδιπίνη ομαλοποίησε την αρτηριακή πίεση (162/104 σε 131/88 mmHg, p < 0,0001), μειώνοντας τη μάζα της αριστερής κοιλίας (135 σε 119 g/m², p < 0,0001) και τις περιφερικές αντιστάσεις (ΟΠΑ) (1734 σε 1492 dynes.cm⁻⁵, p = 0,0005) χωρίς αξιόλογη ταχυκαρδία ή μεταβολές της δραστικότητας renίνης πλάσματος (p = NS). Οι τιμές των ΕΤs μειώθηκαν σε 13 από τους 15 ασθενείς μετά την αγωγή (8,0 σε 6,8 pmol/L, p = 0,0008). Ο βαθμός μείωσης των ΕΤs ήταν ανεξάρτητος του βαθμού μείωσης της αρτηριακής πίεσης ή της μάζας της αριστερής κοιλίας, ενώ σχετιζόταν με την πτώση των ΟΠΑ (r = -0,50, p < 0,05). Συμπεραίνεται ότι η φαρμακευτική αγωγή με ισραδιπίνη μειώνει τις αυξημένες τιμές των ΕΤs των υπερτασικών ασθενών παράλληλα με την πτώση των ΟΠΑ.

Ο ρόλος του ενδοθηλίου στη ρύθμιση του αγγειακού τόνου, και επομένως της αρτηριακής πίεσης, είναι σημαντικός και εξαρτάται από την τελική λειτουργική έκφραση μιας πλειάδας αγγειοδραστικών ουσιών που εκκρίνει. Τα ενδοθηλιακά κύτταρα παράγουν αγγειοχαλαρωτικές ουσίες όπως είναι ο ενδοθηλιακός παράγοντας χάλασης (EDRF) και η προστακυκλίνη (PGI_2), αλλά και αγγειοσυσπαστικές ουσίες όπως είναι η θρομβοξάνη (TxA_2) και η ενδοθηλίνη (ET)[1-3].

Η ομάδα των ενδοθηλινών αποτελείται από τρία ισοπεπτίδια, την ενδοθηλίνη-1 (ET-1) και τις ενδοθηλίνες 2 (ET-2) και 3 (ET-3)[1-3]. Οι ενδοθηλίνες αποτελούνται από 21 αμινοξέα, παράγονται από το αγγειακό ενδοθήλιο[4], ενώ θεωρούνται τοπικές ορμόνες[5] και συμβάλλουν στη διατήρηση των αυξημένων περιφε-

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Γονίδια: Ταξίδι Κληρονομικότητας και προς την Καρδιά

3

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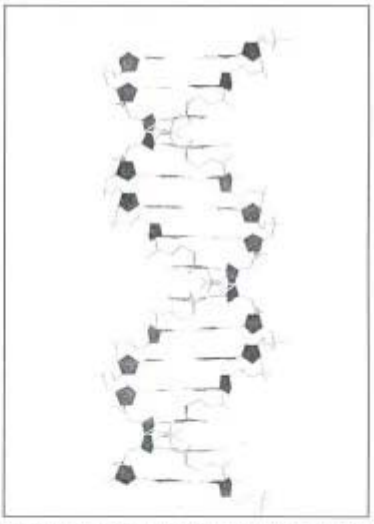
Τα γονίδια αποτελούν τη *θεμελιώδη λειτουργική μονάδα* του ανθρώπινου γενετικού υλικού γιατί αποθηκεύουν, μεταφέρουν και εκφράζουν τις γενετικές πληροφορίες που καθορίζουν την ανθρώπινη ύπαρξη. Η Καρδιολογία, στο μικρό χρονικό διάστημα της εναστρόλησης της με τη μελέτη των γονιδίων του ανθρώπινου γενετικού υλικού και στηριζόμενη στις μοντέρνες τεχνικές της *Μοριακής Βιολογίας*, είναι σε θέση να επιδείξει αξιοθαύμαστα επιτεύγματα που συνδέονται τόσο με την *έρευνα* όσο και με την *πράξη*, τη *διάγνωση* και τη *θεραπεία* σοβαρών καρδιαγγειακών ασθενειών, είτε αυτές είναι κληρονομικές είτε όχι.

Στην *έρευνα*, ο εντοπισμός των γονιδίων που φαίνεται να εμπλέκονται στην παθογένεια της υπερτροφικής μυοκαρδιοπάθειας και της κληρονομικής ιδιοπαθούς διατατικής μυοκαρδιοπάθειας *συνέβαλαν στην ορθότερη διάγνωση* και στην *έγκαιρη αντιμετώπιση* των ασθενειών αυτών. Αλλωστε, η μελέτη της σχέσης δομής-δράσης των πρωτεϊνικών καναλιών της καρδιάς που ελέγχουν τη διέλευση των ιόντων του νατρίου, με την υπερενεργοποίησή τους και την ταυτόχρονη απενεργοποίηση των γονιδίων που ελέγχουν τα αντίστοιχα πρωτεϊνικά κανάλια των λείων μυικών κυττάρων και του ηπατοκυττάρου, *συνέβαλε στη σύνθεση εξειδικευμένων φαρμάκων* που αποκλείουν μόνο τα πρωτεϊνικά κανάλια της καρδιάς με αποτέλεσμα να εμφανίζουν περιορισμένες παρενέργειες και να αντιμετωπίζουν αποτελεσματικότερα τις κοιλιακές αρρυθμίες.



Σχηματική απεικόνιση της δομής του κύριου συστατικού των γονιδίων, του γνωστού δεοξυ-νουκλεϊκού οξέος (DNA).

A/A : 5

Ελλ Καρδιολ Επιθ
Hellenic J Cardiol
40: 20-29, 1999

4

Μεταβολές της λειτουργικής και αιμοστατικής δραστηριότητας του αγγειακού ενδοθηλίου μετά από αντιϋπερτασική αγωγή με διϋδροπυριδίνες

Αικατερίνη Ε. Γιαννακοπούλου, Εμμανουήλ Β. Οικονόμου, Γρηγόριος Π. Βυσσούλης, Μαρίνα Γ. Τούτουζα, Παύλος Κ. Τούτουζας

Αντιϋπερτασικό Ιατρείο, Πανεπιστημιακή Καρδιολογική Κλινική Ιατρικής Σχολής Αθηνών, Ιπποκράτειο Π.Γ.Ν.Α.

Εισαγωγή: *Το αγγειακό ενδοθήλιο παίζει σημαντικό ρόλο στη ρύθμιση του αρτηριακού τόνου και στην αγγειακή αναδιαμόρφωση* [illegible] *(PAI-1).*

Μέθοδοι: *Με σκοπό την αξιολόγηση της λειτουργικής και αιμοστατικής δραστηριότητας του αγγειακού ενδοθηλίου μετά από χορήγηση διϋδροπυριδινών μετρήθηκαν τα επίπεδα πλάσματος* [illegible] *σε 35 ασθενείς* [illegible] *SRO 5 mg (20 ασθενείς) και λακιδιπίνη 4 mg (15 ασθενείς) συγκριτικά με 42 υγιείς μάρτυρες.*

Αποτελέσματα: [illegible]

Υποβλήθηκε στις [illegible]

A/A : 6

Ελλ. Καρδιολ. Επιθ
Hellenic J Cardiol
41: 64-74, 2000

5

Η σημασία της ενεργοποίησης του ελεύθερου ινσουλινοειδούς αυξητικού παράγοντα-1 στη διαδικασία της αναδιαμόρφωσης σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου

Κ. Ι. Καπετάνιος, Χ. Ι. Στεφανάδης, Χ. Η. Πίσσιβος, Ε. Β. Οικονόμου, Δ. Β. Παναγιωτάκος, Δ. Μ. Φαρμάκης, Κ. Ι. Βασιλειάδου, Μ. Γ. Τούτουζα, Π.Κ. Τούτουζας

Ιπποκράτειον Νοσοκομείο Αθηνών, Πανεπιστημιακή Καρδιολογική Κλινική

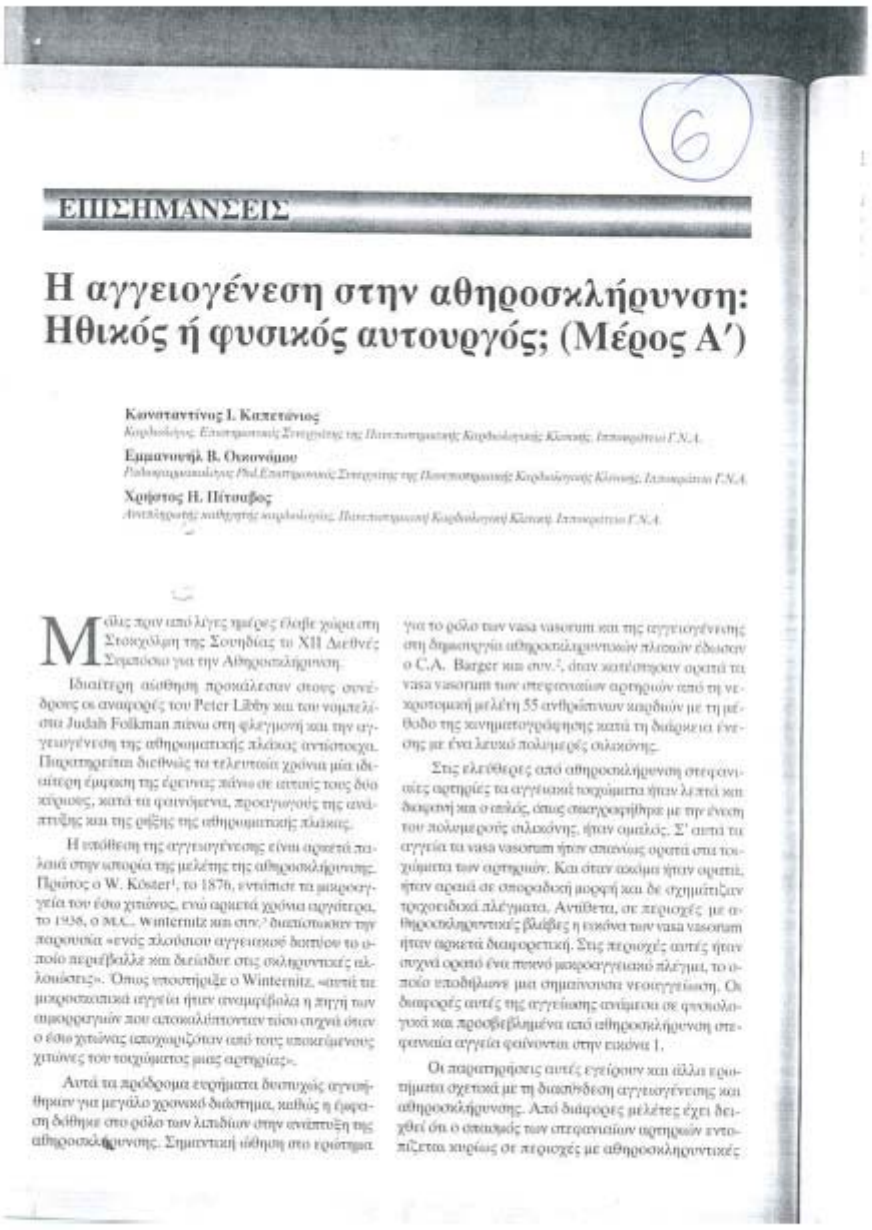
Εισαγωγή: *Τα αντικρινές άκτιμα του Ινσουλινοειδούς Αυξητικού Παράγοντα-1 (I.A.Π.-1) διεστοιβάζονται στα βαθιά μυοκύτταρα της αριστερής κοιλίας σχεδόν πάντα μετά από πειραματική στεφανιαία απόφραξη σε σκύλους, ιδίως δε σε εκείνα τα μυοκύτταρα που γειτνιάζουν προς την περιοχή της νέκρωσης. Επιπλέον η τελευταία χορήγηση ΙΑΠ-1 προήγαγε μια «φυσιολογική» πορεία καρδιακής υπερτροφίας θωρακώντας έτσι την ομαλή καρδιακή λειτουργία. Ο σκοπός της μελέτης μας ήταν να αποκαλυφθεί ο ρόλος του ελεύθερου Ινσουλινοειδή Αυξητικού Παράγοντα-1 (ε.Ι.Α.Π.-1) σε ασθενείς με οξύ έμφραγμα του μυοκαρδίου (ΟΕΜ).*

Μέθοδος: *Σε 27 ασθενείς με ΟΕΜ ως 1η εκδήλωση στεφανιαίας νόσου και χωρίς προηγούμενο ιστορικό καρδιακής νόσου, μετρήθηκαν τα επίπεδα πλάσματος του ε. ΙΑΠ-1 με τη μέθοδο ELISA και συγκρίθηκαν με τα αντίστοιχα επίπεδα 20 υγιών μαρτύρων με μέσες τιμές 7.81±0.15 ng/ml. Οι ασθενείς χωρίστηκαν σε 2 ομάδες ανάλογης κλινικής και φλεβ. Ομάδα Α: Ασθενείς με περιορισμένο ΟΕΜ, ΚΕ>45% και χωρίς υπολειπόμενη δυσλειτουργία της αριστερής κοιλίας. Ομάδα Β: Ασθενείς με εκτεταμένο έμφραγμα, ΚΕ<45% και επακόλουθη καρδιακή ανεπάρκεια. Τα δείγματα πλάσματος εκβράθηκαν κατά την ώρα της εισαγωγής στο νοσοκομείο (ώρα 0) καθώς και 3, 6, 9, 12, 18, 24, 36, 48 ώρες και 3, 4, 5, 7, 15, 30 ημέρες αργότερα. Η μέση λανθάνουσα περίοδος από την «έναρξη του πόνου» μέχρι την εισαγωγή ήταν 2.0±2.0 ώρες. Όλοι οι ασθενείς θρομβολύθηκαν, υφίσταντο αντιπηξική μετεμφραγματική θεραπεία και υποβλήθηκαν σε στεφανιακή αγγειογραφία και αριστερή κοιλιογραφία.*

Αποτέλεσμα: *Η στατιστική ανάλυση έγινε με μη παραμετρικό Wilcoxon test. Στατιστικά σημαντική θεωρήθηκε η διαφορά με $p < 0,05$. Η ομάδα Α εμφάνισε ήδη από το πρώτο δείγμα στατιστικά σημαντική αύξηση των ε.ΙΑΠ-1 (21,7 ± 2,8 ng/ml), σε σχέση με τους ίδιους μάρτυρες, ο οποίος στη συνέχεια συνεχίζει να αυξάνεται σταδιακά με κορύφωση (26,3±3,8 ng/ml) 24 ώρες μετά την έναρξη του ΟΕΜ. Όλα πάντως τα δείγματα της ομάδας Α ήταν στατιστικά σημαντικά αυξημένα σε σχέση με τους μάρτυρες ($p=0,008$), ενώ η κορύφωση ήταν στατιστικά σημαντικά αυξημένη σε σχέση και με το πρώτο δείγμα ($p=0,012$). Αντίθετα η ομάδα Β εμφάνισε αρχικά επίπεδα 5,04 ± 0,6 ng/ml και μια σταδιακή μικρή αύξηση μέχρι τη μέγιστη τιμή 9,41 ± 0,6 ng/ml που δεν ήταν όμως στατι-

Υποβλήθηκε στις 10/5/99. Αποδεκτή στην διορθ. στις 15/11/99.

A/A : 7



6

ΕΠΙΣΗΜΑΝΣΕΙΣ

Η αγγειογένεση στην αθηροσκλήρυνση: Ηθικός ή φυσικός αυτουργός; (Μέρος Α΄)

Κωνσταντίνος Ι. Καπετάνιος
Καρδιολόγος. Επιστημονικός Συνεργάτης της Πανεπιστημιακής Καρδιολογικής Κλινικής. Ιπποκράτειο Γ.Ν.Α.
Εμμανουήλ Β. Οικονόμου
Ραδιοφαρμακολόγος Phd.Επιστημονικός Συνεργάτης της Πανεπιστημιακής Καρδιολογικής Κλινικής. Ιπποκράτειο Γ.Ν.Α.
Χρήστος Η. Πίτσαβος
Αντιπλάρχης καθηγητής καρδιολογίας. Πανεπιστημιακή Καρδιολογική Κλινική. Ιπποκράτειο Γ.Ν.Α.

Μόλις πριν από λίγες ημέρες έδωσε χώρα στη Στοκχόλμη της Σουηδίας το XII Διεθνές Συμπόσιο για την Αθηροσκλήρυνση.

Ιδιαίτερη αίσθηση προκάλεσαν στους συνέδρους οι αναφορές του Peter Libby και του νομπελίστα Judah Folkman πάνω στη φλεγμονή και την αγγειογένεση της αθηρωματικής πλάκας αντίστοιχα. Παρατηρείται διεθνώς τα τελευταία χρόνια μία ιδιαίτερη έμφαση της έρευνας πάνω σε αιτιώδες τους δύο πόλους, κατά τα φαινόμενα, προαγωγούς της ανάπτυξης και της ρήξης της αθηρωματικής πλάκας.

Η υπόθεση της αγγειογένεσης είναι αρκετά παλαιά στην ιστορία της μελέτης της αθηροσκλήρυνσης. Πρώτος ο W. Köster[1], το 1876, εντόπισε τα μικροαγγεία του έσω χιτώνος, ενώ αρκετά χρόνια αργότερα, το 1938, ο M.L. Winternitz και συν.[2] διαπίστωσαν την παρουσία «ενός πλούσιου αγγειακού δικτύου το οποίο περιέβαλλε και διείσδυε στις σκληρυντικές αλλοιώσεις». Όπως υποστήριξε ο Winternitz, «αυτά τα μικροσκοπικά αγγεία ήταν αναμφίβολα η πηγή των αιμορραγιών που εντοπιζόταν τόσο συχνά όταν ο έσω χιτώνας αποχωριζόταν από τους υποκείμενους χιτώνες του τοιχώματος μιας αρτηρίας».

Αυτά τα πρόδρομα ευρήματα δυστυχώς αγνοήθηκαν για μεγάλο χρονικό διάστημα, καθώς η έμφαση δόθηκε στο ρόλο των λιπιδίων στην ανάπτυξη της αθηροσκλήρυνσης. Σημαντική ώθηση στο ερώτημα για το ρόλο των vasa vasorum και της αγγειογένεσης στη δημιουργία αθηροσκληρυντικών πλακών έδωσαν ο C.A. Barger και συν.[3], όταν κατέστησαν ορατά τα vasa vasorum των στεφανιαίων αρτηριών από τη νεκροτομική μελέτη 55 ανθρώπινων καρδιών με τη μέθοδο της κονιματογράφησης κατά τη διάρκεια ενέσης με ένα λευκό πολυμερές σιλικόνης.

Στις ελεύθερες από αθηροσκλήρυνση στεφανιαίες αρτηρίες τα αγγειακά τοιχώματα ήταν λεπτά και διαφανή και ο αυλός, όπως σκιαγραφήθηκε με την ένεση του πολυμερούς σιλικόνης, ήταν ομαλός. Σ' αυτά τα αγγεία τα vasa vasorum ήταν σπανίως ορατά στα τοιχώματα των αρτηριών. Και όταν αυτά ήταν ορατά, ήταν αραιά σε σποραδική μορφή και δε σχημάτιζαν τριχοειδικά πλέγματα. Αντίθετα, σε περιοχές με αθηροσκληρυντικές βλάβες η εικόνα των vasa vasorum ήταν αρκετά διαφορετική. Στις περιοχές αυτές ήταν συχνά ορατό ένα πυκνό μικροαγγειακό πλέγμα, το οποίο υποδήλωνε μια σημαντική νεοαγγείωση. Οι διαφορές αυτές της αγγείωσης ανάμεσα σε φυσιολογικά και προσβεβλημένα από αθηροσκλήρυνση στεφανιαία αγγεία φαίνονται στην εικόνα 1.

Οι παρατηρήσεις αυτές εγείρουν και άλλα ερωτήματα σχετικά με τη διασπάθεση αγγειογένεσης και αθηροσκλήρυνσης. Από διάφορες μελέτες έχει δειχθεί ότι ο σπάνιος των στεφανιαίων αρτηριών εντοπίζεται κυρίως σε περιοχές με αθηροσκληρυντικές

A/A : 8

Καρδιά & Αγγεία
vol V(5), 436 – 442
2005

ΕΠΙΣΗΜΑΝΣΕΙΣ

(7)

Η αγγειογένεση στην αθηροσκλήρυνση: Ηθικός ή φυσικός αυτουργός; (Μέρος β′)

Κωνσταντίνος Ι. Καπετάνιος
Καρδιολόγος, Επιστημονικός Συνεργάτης Πανεπιστημιακής Καρδιολογικής Κλινικής, Ιπποκράτειο Γ.Ν.Α.
Εμμανουήλ Β. Οικονόμου
Ρεύμα νεφρολόγος Phd, Επιστημονικός Συνεργάτης Πανεπιστημιακής Καρδιολογικής Κλινικής, Ιπποκράτειο Γ.Ν.Α.
Χρήστος Η. Πέτσαβος
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Εκτός του κλινικού συνδρόμου της ασταθούς στηθάγχης, τους μελετητές απασχόλησε και η επίδραση της νεοαγγείωσης στην καρωτιδική νόσο. Είναι γνωστό ότι στενώσεις της έσω καρωτίδος αρτηρίας μπορούν να προκαλέσουν ετερόπλευρα ισχαιμικά εγκεφαλικά επεισόδια θρομβοεμβολικής αιτιολογίας. Υπάρχουν σαφείς ενδείξεις ότι τα έμβολα αυτά προέρχονται από τις αθηρωματικές πλάκες και ανευρίσκονται συχνότερα σε περιπτώσεις μαλακών εξελιγμένων πλακών σε σχέση με τις ασβεστωμένες ή ινώδεις πλάκες. Ο A. Petersen και συν.[23] ανέφεραν αρχικά ότι η αιμορραγία εντός της αθηρωματικής πλάκας οδηγεί στην εμφάνιση κλινικών νευρολογικών συμπτωμάτων από τη σπυρά που επισυμβαίνει ρήξη της πλάκας προς την ενδοαυλική επιφάνεια αυτής.

Σε ενίσχυση των ανωτέρω ο S. Carr και συν.[24] υποστήριξαν ότι η λέπτυνση και η διάβρωση της ινώδους κάψας από ωχρόδη κύτταρα και η φλεγμονώδης διήθηση αυτής προδιαθέτουν προς τη ρήξη της αθηρωματικής πλάκας, πράγμα το οποίο σχετίζεται άμεσα με συμπτωματική καρωτιδική νόσο. Φαίνεται λοιπόν ότι η αστάθεια της πλάκας αποτελεί κυρίαρχο παράγοντα στη δημιουργία θρομβοεμβολικών επεισοδίων.

Ο M.J. McCarthy και συν.[25] προσπάθησαν να διερευνήσουν αν υφίστανται κάποια σχέση ανάμεσα στην πυκνότητα, τη θέση και τη μορφολογία των νεοαγγειακών σχηματισμών από τη μία μεριά, και τη δημιουργία θρομβοεμβολικών επεισοδίων ή κλινικών συμπτωμάτων σε ασθενείς με γνωστή καρωτιδική νόσο από την άλλη. Οι ερευνητές μελέτησαν ενδοαισθησιομετρικά καρωτιδικές πλάκες μετά από ενδαρτηρεκτομή σε 65 ασθενείς, συμπτωματικούς ή μη, που είχαν υποβληθεί σε αυτή την επέμβαση, χρησιμοποιώντας χρώση με αιματοξυλίνη και ηωσίνη καθώς και ένα μονοκλωνικό αντίσωμα έναντι του δείκτη CD-31 των ενδοθηλιακών κυττάρων. Ως νεόπλαστα αγγεία χαρακτηρίσθηκαν μόνο εκείνα τα οποία περιείχαν μια μονήρη στιβάδα ενδοθηλιακών κυττάρων που δεν περιστοιχιζόταν από λεία μυϊκά κύτταρα.

Διαπιστώθηκε ότι υπήρχαν περισσότερα νεόπλαστα αγγεία σε στατιστικά σημαντικό βαθμό στις αθηρωματικές πλάκες συμπτωματικών ασθενών σε σχέση με εκείνες των ασυμπτωματικών ασθενών (εικόνα 10).

Στην πλειονότητά τους αυτά τα μικροαγγεία ήταν παρόντα στις πλευρικές γωνίες των αθηρωματικών πλακών, ενώ λιγότερα βρίσκονταν και γύρω από τη βάση των αθηρωματικών πλακών. Πάντως ένας σημαντικός αριθμός συμπτωματικών πλακών διέθετε νεόπλαστα αγγεία και εντός της ινώδους κάψας της αθηρωματικής πλάκας, ενώ παρόμοια τα νεοαγγεία αυτά ήταν μεγαλύτερα σε μέγεθος.

A/A : 9

8

ΕΠΙΣΗΜΑΝΣΕΙΣ

Νευροπροστασία αντιϋπερτασικής αγωγής

Ιωάννης Σ. Ελευσινιώτης
Ειδικός Παθολόγος, Επιστημονικός Συνεργάτης Ε.Λ.Ι.ΚΑΡ.
Εμμανουήλ Β. Οικονόμου
Ερευνητής Ph.D. Επιστημονικός Συνεργάτης Πανεπιστημιακής Καρδιολογικής Κλινικής Πανεπιστημίου Αθηνών, Ιπποκράτειο Γ.Ν.Α.
Χρήστος Η. Πίτσαβος
Αναπληρωτής Καθηγητής Καρδιολογίας, Ιπποκράτειο Π.Γ.Ν.Α.

Η εγκεφαλοαγγειακή νόσος αποτελεί την τρίτη κατά σειρά συχνότητας αιτία θανάτου, μετά τη στεφανιαία νόσο και τις διάφορες μορφές καρκίνου, και έναν από τους σημαντικότερους λόγους νοσοκομειακής περίθαλψης και μακροχρόνιας αναπηρίας στις βιομηχανικά αναπτυγμένες κοινωνίες της Δ. Ευρώπης και της Β. Αμερικής. Σύμφωνα με πολυπληθείς και μακροχρόνιες επιδημιολογικές μελέτες, 10-12% του συνόλου των θανάτων στις βιομηχανοποιημένες χώρες οφείλονται στην εγκεφαλοαγγειακή νόσο και τις επιπλοκές της και παρ' όλο που έχει επιτευχθεί σημαντική μείωση στην επίπτωση της νόσου την τελευταία δεκαετία, ο απόλυτος αριθμός των θυμάτων της συνεχώς αυξάνεται θεαματικά καθώς αυξάνεται ο μέσος όρος ηλικίας του πληθυσμού[1]. Αξιοσημείωτο επίσης είναι το γεγονός της σημαντικής αύξησης της νοσηρότητας και της θνητότητας εις της νόσου σε συγκεκριμένους πληθυσμούς, με κύριο εκπρόσωπο τους λαούς της Ανατολικής Ευρώπης[2].

Έναν από τους σημαντικότερους, καλά αναγνωρισμένους και προσποιήσιμους προδιαθεσικούς παράγοντες της εγκεφαλοαγγειακής νόσου, αποτελεί η υπέρταση. Διαδραματίζει κεντρικό ρόλο στην παθογένεια τόσο αθηροθρομβωτικού εγκεφαλικού εμφράκτου και της ενδοεγκεφαλικής πηγεγχυματικής αιμορραγίας, ενώ ανευρίσκεται ως παράγοντας κινδύνου σε μεγάλο ποσοστό ασθενών που εμφανίζουν εμβολικά και παροδικά ισχαιμικά αγγειακά εγκεφαλικά επεισόδια. Αποτελεί τον αιτιολογικό παράγοντα της υπερτασικής εγκεφαλοπάθειας και η παρουσία της συσχετίζεται θετικά όχι τόσο με την εμφάνιση όσο με την εξέλιξη και την πρόγνωση της υπαραχνοειδούς αιμορραγίας[3]. Εντούτοις οι κλινικά έκδηλες μορφές της εγκεφαλοαγγειακής νόσου, μέσω των οποίων καθορίζονται η νοσηρότητα και θνητότητα, καθώς και η αποτελεσματικότητα της αντιυπερτασικής αγωγής σε επίπεδο πρωτογενούς και δευτερογενούς πρόληψης, φαίνεται να αποτελούν την κορυφή του παγόβουνου, σε ό,τι αφορά την προσβολή του κεντρικού νευρικού συστήματος στους υπερτασικούς ασθενείς. Η προσβολή των μεγάλων εγκεφαλικών αγγείων αλλά κυρίως των μικρών αρτηριδίων και αρτηριολίων, των καλουμένων αγγείων αντίστασης της εγκεφαλικής μικροκυκλοφορίας, αποκτούν πολύ σημαντική σημασία στην πορεία της νόσου κλινική οντότητα και διακρίνονται ως κλινικά σύνδρομα, λόγω των πολύπλοκων μηχανισμών αυτορρύθμισης της εγκεφαλικής κυκλοφορίας και της τροποποίησης του μεταβολισμού των νευρικών κυττάρων του εγκεφάλου, με μηχανισμούς προσαρμογής σε αντίξοες μικροπεριβαλλοντικές συνθήκες.

Από τα τέλη του 1800, όταν ο Alzheimer και ο Binswanger πρωτοεισήγαγαν τον ορισμό της "αρτηριοσκληρυντικής εγκεφαλικής εκφυλιστικής νόσου", διεξάγεται συστηματική και εντατική έρευνα για το βαθμό συμμετοχής ή και την αιτιολογική κατά άλ-

ΠΡΩΤΗ ΣΕΛΙΔΑ ΕΛΛΗΝΙΚΩΝ ΔΗΜΟΣΙΕΥΣΕΩΝ ΣΕ ΠΡΑΚΤΙΚΑ ΚΑΙ ΕΙΔΙΚΕΣ ΕΚΔΟΣΕΙΣ ΠΑΝΕΛΛΗΝΙΩΝ ΣΥΝΕΔΡΙΩΝ

A/A : 1

500

K45

ΜΕΛΕΤΗ ΤΗΣ ΕΠΙΔΡΑΣΗΣ ΧΡΩΣΤΙΚΩΝ ΣΕ ΣΥΣΤΗΜΑ ΡΑΔΙΟΑΝΟΣΟΑΝΑΛΥΣΗΣ ΓΙΑ ΤΗΝ ΜΕΤΡΗΣΗ ΘΥΡΟΞΙΝΗΣ

Ε.ΟΙΚΟΝΟΜΟΥ, Σ.ΚΑΚΑΜΠΑΚΟΣ, Ε.ΛΙΒΑΝΙΟΥ, Κ.ΣΩΤΗΡΙΑΔΗΣ - ΒΛΑΧΟΣ, Γ.ΕΥΑΓΓΕΛΑΤΟΣ.
Εργαστήριο Ραδιοανοσοχημείας, ΕΚΕΦΕ Δημόκριτος, Αγία Παρασκευή Αττικής 153 10.

1. ΕΙΣΑΓΩΓΗ

Η ευρεία χρησιμοποίηση των ραδιοανοσοαναλύσεων (1), τόσο σε ερευνητικό επίπεδο όσο και σε μετρήσεις ρουτίνας, οδήγησε στην ανάπτυξη προωθημένης τεχνολογίας ως προς τον τεχνικό εξοπλισμό και στην εφαρμογή διαφόρων βελτιώσεων που διευκολύνουν τον αναλυτή.

Η χρησιμοποίηση των χρωστικών στα αντιδραστήρια των ραδιοανοσοαναλύσεων είναι μια σχετικά πρόσφατη βελτίωση που μειώνει σημαντικά την πιθανότητα λάθους προσθήκης αντιδραστηρίων, κάνει εμφανή μια πιθανή μόλυνση από το ραδιενεργό αντιδραστήριο, μειώνει το χρόνο της ανάλυσης και διευκολύνει τον αναλυτή.Για να χρησιμοποιηθεί όμως μια ουσία σαν χρωστική αντιδραστηρίων ραδιοανοσοανάλυσης, ιδιαίτερα ουσιών μικρού Μ.Β. όπως είναι η θυροξίνη, θα πρέπει να μην επεμβαίνει στην αντίδραση αντιγόνου -αντισώματος, να μην σχηματίζει ίζημα, να μην αντιδρά με κάποιο από τα αντιδραστήρια της μεθόδου, να μην αλλάζει το pH του μίγματος της αντίδρασης, να μην επηρεάζει την ικανότητα δέσμευσης, την μη ειδική δέσμευση (N.S.B.), την καμπύλη προτύπων,την ανάγνωση των τιμών των ορών ελέγχου στην καμπύλη προτύπων,κλπ.

Στην παρούσα εργασία μελετήθηκε η επίδραση διαφορετικών συγκεντρώσεων κάθε μιάς από τις χρωστικές : Tartrazine, Congo Red, Ponceau-S, Coomasie Blue, Erioglaucin, σε σειρά παραγόντων που υπεισέρχονται στη μέτρηση, όπως είναι η ικανότητα δέσμευσης, η μη ειδική δέσμευση, η καμπύλη προτύπων, η ανάγνωση

A/A : 2

Κα

ΣΥΓΚΡΙΤΙΚΗ ΜΕΛΕΤΗ ΤΩΝ ΜΕΘΟΔΩΝ ΧΛΩΡΑΜΙΝΗΣ-Τ, ΓΑΛΑΚΤΟΠΕΡΟΞΕΙΔΑΣΗΣ ΚΑΙ ΙΩΔΟΓΟΝΟΥ ΓΙΑ ΤΗΝ ΕΠΙΣΗΜΑΝΣΗ ΤΗΣ ΑΝΘΡΩΠΙΝΗΣ ΟΧΡΙΝΟΠΟΙΗΤΙΚΗΣ ΟΡΜΟΝΗΣ ΜΕ ^{125}I.

Σ.ΚΑΚΑΜΠΑΚΟΣ, Ε.ΟΙΚΟΝΟΜΟΥ, Ε.ΛΙΒΑΝΙΟΥ, Κ.ΣΩΤΗΡΙΑΔΗΣ-ΒΛΑΧΟΣ, Γ.ΕΥΑΓΓΕΛΑΤΟΣ.

Εργαστήριο Ραδιοανοσοχημείας, ΕΚΕΦΕ Δημόκριτος, Αγία Παρασκευή Αττικής 153 10.

ΠΕΡΙΛΗΨΗ

Η ανθρώπινη ωχρινοποιητική ορμόνη, h-LH, ραδιοεπισημάνθηκε ($Na^{125}I$), με τη μέθοδο της χλωραμίνης-Τ ελαφρώς τροποποιημένη, με τη μέθοδο της γαλακτοπεροξειδάσης και με τη μέθοδο του ιωδογόνου, ώστε να παρασκευασθεί ραδιοεπισημασμένη ορμόνη κατάλληλη να χρησιμοποιηθεί σε ραδιοανοσοανάλυση της ορμόνης. Η επισημασμένη ορμόνη ελέγχθηκε ως προς την ειδική ραδιενέργεια, την ραδιοχημική σταθερότητα, την ανοσοδραστικότητα και την μη ειδική δέσμευση. Μεταξύ των μεθόδων που χρησιμοποιήθηκαν για την επισήμανση της ορμόνης, επιλέχθηκε η μέθοδος της χλωραμίνης-Τ, γιατί εμφανίζει ικανοποιητικές τιμές αλλά και καλή επαναληψιμότητα των παραμέτρων που μελετήθηκαν για διαφορετικές επισημάνσεις, σε αντίθεση με τις δύο άλλες μεθόδους.

ΕΙΣΑΓΩΓΗ

Στην βιβλιογραφία αναφέρεται ένας αριθμός μεθόδων ραδιοϊωδίωσης των πρωτεϊνών, μεταξύ των οποίων οι πιο σημαντικές είναι : η μέθοδος της χλωραμίνης-Τ (1), της γαλακτοπεροξειδάσης (2), του ιωδογόνου (3,4), της επισήμανσης με σύζευξη (5), κ.ά. Κάθε μια από τις μεθόδους που προαναφέρθηκαν χαρακτηρίζεται από σημαντικά πλεονεκτήματα, αλλά και κάποια μειονεκτήματα (6).

Στην παρούσα εργασία περιγράφεται η προσπάθεια εξεύρεσης

A/A : 3

730

Δ_{45}

ΡΑΔΙΟΑΝΟΣΟΑΝΑΛΥΤΙΚΟΣ ΠΡΟΣΔΙΟΡΙΣΜΟΣ ΤΗΣ ΔΙΑΙΘΥΛΟΣΤΙΛΒΕΣΤΡΟΛΗΣ (DES) ΣΕ ΒΙΟΛΟΓΙΚΑ ΥΓΡΑ ΚΑΙ ΕΚΧΥΛΙΣΜΑΤΑ ΙΣΤΩΝ. ΥΠΑΡΧΟΥΣΕΣ ΜΕΘΟΔΟΙ-ΠΡΟΒΛΗΜΑΤΑ-ΠΡΟΣΑΝΑΤΟΛΙΣΜΟΙ.

Ε.Β. ΟΙΚΟΝΟΜΟΥ[*,1], Γ.Π. ΕΥΑΓΓΕΛΑΤΟΣ[1], Θ. ΣΙΑΤΡΑ-ΠΑΠΑΣΤΑΙΚΟΥΔΗ[2], Α.Σ. ΙΘΑΚΗΣΙΟΣ[3].

1. Εργ. Ραδιοανοσοχημείας, ΕΚΕΦΕ"ΔΗΜΟΚΡΙΤΟΣ",Αγία Παρασκευή 153 10
2. Εργ. Φαρμ/κής Χημείας, Πανεπ. Αθήνας, Σόλωνος 104, 106 80.
3. Εργ. Φαρμ/κής Τεχνολογίας, Πανεπ. Πάτρας, Πάτρα 261 10.

ΠΕΡΙΛΗΨΗ

Η ευρύτατη χρήση της διαιθυλοστιλβεστρόλης (DES) ως αντιανδρογόνο και αναβολικό, σε συνδυασμό με τις ποικίλες παρενέργειες που προκαλεί και τις ελλειπώς μελετημένες υπάρχουσες μεθόδους προσδιορισμού της, καθιστούν αναγκαία την ανάπτυξη νέας αξιόπιστης ραδιοανοσοαναλυτικής μεθόδου (RIA) για τον προσδιορισμό της στα βιολογικά δείγματα. Η πειραματική μελέτη για τον έλεγχο των παραμέτρων που επηρεάζουν μια τέτοια ανάλυση έδειξε οτι η συγκέντρωση της αιθανόλης και της ζελατίνης στο τελικό μίγμα της επώασης επηρεάζει σημαντικά την ικανότητα δέσμευσης του τριτιωμένου ιχνηθέτη DES (^{3}H-DES) στο αντίσωμα της DES (Ab-DES), ενώ η ιονική ισχύς δεν επεμβαίνει στην ανοσοαντίδραση αυτή. Η προτεινόμενη πρότυπη καμπύλη είναι ευαίσθητη και αναπαραγώγιμη, ενω η ελαχιστοποίηση του ανταγωνισμού των πρωτεινών του τελικού μίγματος επώασης με το Ab-DES, η αντικατάσταση του ^{3}H-DES και της χρησιμοποιούμενης μεθόδου διαχωρισμού (εναιώρημα άνθρακα) με ραδιοϊωδιωμένο ιχνηθέτη και εναιώρημα δευτέρου αντισώματος (RIP), ως μέθοδο διαχωρισμού, αναμένεται να συνεισφέρει σημαντικά στη βελτίωσή της.

ΕΙΣΑΓΩΓΗ

Η DES και το διφωσφορικό παράγωγο αυτής, η διφωσφορική διαιθυλοστιλβεστρόλη (DES-DP), χρησιμοποιείται τα τελευταία είκοσι χρόνια στη θεραπεία του καρκίνου του προστάτη (1,2) . Επιπλέον, η DES τα τελευταία δέκα χρόνια χρησιμοποιείται ως αναβολικό για την πάχυνση των βοοειδών (3) . Προβλήματα από την χρήση της ορμόνης αυτής συνδέονται με τις συνήθεις παρενέργειες των αντιανδρογόνων (θηλυκοποίηση, μείωση του Libido κ.λ.π.), με θρομβοεμβολικά και άλλα καρδιοαγγειακά προβλήματα, όπως επίσης και με την πιθανολογούμενη καρκινογόνο δράση της DES στους καταναλωτές κρεάτων βοοειδών όταν αυτή χρησιμοποιείται ως αναβολικό για την πάχυνσή τους (4,5,6). Έτσι, είναι φανερή η ανάγκη μέτρησης των επιπέδων της DES σε βιολογικά δείγματα για την παρακολούθηση της θεραπείας του καρκίνου του προστάτη. Εξάλλου, η αξιολόγηση ιατρικών εκχυλισμάτων βοοειδών ως προς την περιεκτικότητά τους σε DES κρίνεται απαραίτητη για την προστασία της δημόσιας υγείας, ιδιαίτερα στις χώρες εκείνες που η χρήση της ορμόνης αυτής ως αναβολικό έχει απαγορευθεί. Ως επικρατέστερη μέθοδος προσδιορισμού της DES in vitro, θεωρείται σήμερα η ραδιοανοσοανάλυση (RIA), αλλά η υπάρχουσα μεθοδολογία είναι ελλειπής και δεν προσφέρεται για αδρόα αξιολόγηση δειγμάτων (7,8,9).

A/A : 4

Σ_5

ΡΑΔΙΟΑΝΟΣΟΑΝΑΛΥΤΙΚΟΣ ΠΡΟΣΔΙΟΡΙΣΜΟΣ ΤΗΣ ΔΙΑΙΘΥΛΟΣΤΙΛΒΕΣΤΡΟΛΗΣ ΑΠΕΥΘΕΙΑΣ ΣΕ ΟΡΟ ΑΣΘΕΝΩΝ ΜΕ ΚΑΡΚΙΝΟ ΤΟΥ ΠΡΟΣΤΑΤΗ.

Ε.Β. ΟΙΚΟΝΟΜΟΥ*,[1], Σ.Η. ΚΑΚΑΜΠΑΚΟΣ[1], Γ.Π. ΕΥΑΓΓΕΛΑΤΟΣ[1], Δ.Σ. ΙΘΑΚΗΣΙΟΣ[1,2].

1. Εργ. Ραδιοανοσοχημείας, ΕΚΕΦΕ "ΔΗΜΟΚΡΙΤΟΣ", Αγ.Παρασκ. 153 10.
2. Εργ. Φαρμ/κής Τεχνολογίας, Πανεπιστήμιο Πάτρας, Πάτρα 261 10.

ΠΕΡΙΛΗΨΗ

Στην εργασία αυτή παρουσιάζεται η ανάπτυξη ραδιοανοσοαναλυτικής μεθόδου για τον προσδιορισμό της διαιθυλοστιλβεστρόλης (DES) απευθείας σε 50μL ανθρώπινου ορού με τη χρήση τριτιωμένης DES ως ιχνηθέτη και δευτέρου αντισώματος ως αντιδραστηρίου διαχωρισμού των φάσεων. Η προτεινόμενη μέθοδος είναι ευαίσθητη (ευαισθησία < 0.17μg/λίτρο), επαναληψιμη (ενδο-και διαναλυτικός συντελεστής διακύμανσης 8.14% και 8.25% αντίστοιχα) και ακριβής (ανάκτηση >95%).

ΕΙΣΑΓΩΓΗ

Η διαιθυλοστιλβεστρόλη (DES), ένα συνθετικό μη στεροειδικό οιστρογόνο, χρησιμοποιείται για την αντιμετώπιση προβλημάτων στα τελευταία στάδια της κύησης, κυρίως όμως χρησιμοποιείται στην θεραπεία του καρκίνου του προστάτη (1,2,3).

Αρκετές ραδιοανοσοαναλυτικές μέθοδοι έχουν αναπτυχθεί για τον προσδιορισμό της DES στα βιολογικά υγρά (4-7).

Η χορήγηση υψηλών θεραπευτικών δόσεων (> 200mg DES ημερησίως) επιτρέπει τον απευθείας προσδιορισμό των επιπέδων του οιστρογόνου αυτού στον ορό τών ασθενών.

Στεχιτικά πρόσφατες μελέτες έδειξαν ότι η χορήγηση μικρών δόσεων DES (< 3mg DES ημερησίως) είναι εξίσου αποτελεσματικές ενώ συγχρόνως προκαλούν σημαντικά λιγότερες παρενέργειες (2,4). Στην περίπτωση αυτή, ο ραδιοανοσοαναλυτικός προσδιορισμός της DES στον ορό των ασθενών προϋποθέτει την εκχύλιση του βιολογικού δείγματος πριν από την ανάλυσή του, ένα στάδιο που είναι χρονοβόρο, επίπονο και αποτελεί πηγή σφαλμάτων για τον τελικό προσδιορισμό του οιστρογόνου αυτού (4,6).

Στο εργαστήριό μας αναπτύξαμε μία απλή, εύχρηστη και ευαίσθητη ραδιοανοσοανάλυση για τον προσδιορισμό χαμηλών συγκεντρώσεων της DES απευθείας σε 50μL ορού ασθενών με καρκίνο του προστάτη που ακολουθούν θερεπεία με το οιστρογόνο αυτό.

ΥΛΙΚΑ ΚΑΙ ΜΕΘΟΔΟΙ

Όλα τα αντιδραστήρια ήταν αναλυτικής καθαρότητας. Η ξελατίνη ήταν της BDH, Αγγλία, η ανθρώπινη οραλβουμίνη (HSA) και η διαιθυλοστιλβεστρόλη (DES) ήταν της Sigma, Η.Π.Α., και το αντίσωμα έναντι της DES (Ab-DES) ήταν της Roussel-Uclaf, Γαλλία.

Ρυθμιστικά διαλύματα: Α) ρυθμιστικό διαλυμα φωσφορικών (PB), pH 7.4, 0.01M περιείχε 9g NaCl, 1g NaN_3 και 0.5g ξελατίνης στο λίτρο. Β) PB pH 7.4, 0.01M περιείχε 9g NaCl, 1g NaN_3 και 0.5g HSA

A/A : 5

ΠΡΑΚΤΙΚΑ 8ου ΠΑΝΕΛΛΗΝΙΟΥ ΣΥΝΕΔΡΙΟΥ ΠΕΡΙΓΕΝΝΗΤΙΚΗΣ ΙΑΤΡΙΚΗΣ
Αθήνα, 16 και 17 Απριλίου 1994

ΕΠΙΠΕΔΑ ΤΩΝ ΠΟΛΥΑΚΟΡΕΣΤΩΝ ΛΙΠΑΡΩΝ ΟΞΕΩΝ ΣΤΟ ΠΛΑΣΜΑ ΦΥΣΙΟΛΟΓΙΚΩΝ ΤΕΛΕΙΟΜΗΝΩΝ ΚΑΙ ΠΡΟΩΡΩΝ ΝΕΟΓΝΩΝ

Α. Μαλαμίτση-Puchner, Σ. Σεβαστιάδου, Θ. Ενσταθόπουλος, Ε. Οικονόμου Ζ. Χατζησταμιατίου, Δ. Νικολόπουλος

Τμήμα Νεογνολογίας, ΠΓΝ "Αλεξάνδρα"

Τα υπεροξείδια των πολυακορέστων λιπαρών οξέων (LPs) αποτελούν σταθερά προϊόντα της επίδρασης των ελευθέρων ριζών οξυγόνου (EPO) στα πολυακόρεστα λιπαρά οξέα των κυτταρικών μεμβρανών (1). Σημαντική αιτία αθρόας παραγωγής EPO και επομένως LPs θα μπορούσε να αποτελεί η απότομη αύξηση της συγκέντρωσης του οξυγόνου στα αερόβια κύτταρα με την έναρξη της αναπνοής κατά τη γέννηση. Είναι γνωστό ότι η τάση του O_2 στους πνεύμονες του νεογνού αυξάνει με τη γέννηση από <30 mmHg σε περίπου 100 mmHg. Μελέτες όμως που έγιναν σε ζώα δεν κατόρθωσαν προς το παρόν να αποδείξουν με βεβαιότητα ότι η έκθεση σε υψηλή συγκέντρωση οξυγόνου αυξάνει in vivo την υπεροξείδωση των λιπαρών οξέων (2). Επιπλέον παραμένει το κρίσιμο ερώτημα, άν αυτή η υπεροξείδωση των λιπαρών οξέων των κυτταρικών μεμβρανών εμπλέκεται στην τοξική επίδραση του οξυγόνου in vivo (3). Μολαταύτα η υπερπαραγωγή EPO έχει σχετισθεί με την παθογένεση ποικίλων προβλημάτων στα νεογνά, όπως η αμφιβληστροπάθεια της προωρότητας, η ενδοκοιλιακή αιμορραγία, η νεκρωτική εντεροκολίτιδα και η βρογχοπνευμονική δυσπλασία (4). Ενδεχομένως η εκδήλωση αυτών των παρενεργειών να οφείλεται στην ανεπάρκεια κυτταρικών αντιοξειδωτικών προστατευτικών μηχανισμών των οποίων οι συγκεντρώσεις εξαρτώνται από την ηλικία.

Σκοπός αυτής της μελέτης ήταν η διερεύνηση των μεταβολών των επιπέδων των LPs στο 1ο και 4ο 24ωρο της ζωής στο πλάσμα φυσιολογικών τελειομήνων και προώρων νεογνών, που δεν χρειάστηκαν υποστήριξη της αναπνευστικής λειτουργίας με χορήγηση οξυγόνου, καθώς και η συσχέτιση των επιπέδων των LPs με το είδος του τοκετού.

A/A : 6

ΠΡΑΚΤΙΚΑ 8ου ΠΑΝΕΛΛΗΝΙΟΥ ΣΥΝΕΔΡΙΟΥ ΠΕΡΙΓΕΝΝΗΤΙΚΗΣ ΙΑΤΡΙΚΗΣ
Αθήνα, 16 και 17 Απριλίου 1994

– 24 –

ΕΠΙΠΕΔΑ ΕΝΔΟΘΗΛΙΝΗΣ (ΕΤ) 1-21 ΣΤΟ ΠΛΑΣΜΑ ΦΥΣΙΟΛΟΓΙΚΩΝ ΕΜΒΡΥΩΝ 18-24 ΕΒΔΟΜΑΔΩΝ

Α. Μαλαμίτση- Puchner, Α. Αντσακλής, Ε. Οικονόμου, Ν. Παπαντωνίου, Σ. Μεσογίτης, Ν. Κούτρα, Σ. Παπαχαρίτωνος, Γ. Μπίτσιου, Δ. Αραβαντινός

Τμήμα Εμβρυομητρικής Ιατρικής, Α' Μαιευτική και Γυναικολογική Κλινική Πανεπιστημίου Αθηνών.

Η ενδοθηλίνη-1 (ΕΤ-1) είναι ένα εξαιρετικά δραστικό αγγειοσυσπαστικό πεπτίδιο, που εκκρίνεται από τα ενδοθηλιακά κύτταρα των αγγείων, τον πλακούντα και τα κύτταρα του αμνίου (1). Στον άνθρωπο συμβάλλει στη ρύθμιση του αγγειακού τόνου, της τοπικής αιματικής ροής (2) και επομένως και της μεταφοράς του οξυγόνου μέσω του πλακούντα στο έμβρυο. Ο τελευταίος είναι πλούσιος σε υποδοχείς ΕΤ-1 (3), η υπερπαραγωγή της οποίας εξασκεί συσπαστική δράση στην εμβρυοπλακουντιακή κοτυληδόνα, με συνέπεια τον περιορισμό της εμβρυοπλακουντιακής αιματικής ροής και πιθανόν την ενδομήτρια καθυστέρηση της ανάπτυξης του εμβρύου (ΕΚΑΕ). Εντούτοις η ΕΤ-1 εξασκεί και μια τελείως διαφορετική δράση, προάγοντας αμέσως την κυτταρική αύξηση και ενισχύοντας τη μιτογόνο δραστικότητα πολλών αυξητικών παραγόντων (4).
Σκοπός αυτής της μελέτης ήταν η διερεύνηση της φυσιολογικής λειτουργίας της ΕΤ-1 στο έμβρυο α) προσδιορίζοντας τα επίπεδα της ΕΤ 1-21 σε φυσιολογικά έμβρυα 18-24 εβδομάδων, που υφίστανται παρακέντηση του ομφαλίου λώρου για διάφορους λόγους και β) αναζητώντας πιθανή συσχέτιση μεταξύ των εμβρυϊκών και μητρικών επιπέδων της ΕΤ 1-21.

Υλικό και Μέθοδος

Η μελέτη περιέλαβε 20 ζεύγη "μητέρων-εμβρύων". Η ένδειξη για λήψη εμβρυϊκού αίματος με παρακέντηση του ομφαλίου λώρου ήταν σε 13 περιπτώσεις η ετερόζυγος β- μεσογειακή αναιμία και στους δύο γονείς, σε 4 περιπτώσεις η προχωρημένη ηλικία της μητέρας (>35 ετών) και