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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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Содержание:

Vadatchkoria R, Dondoladze K, Tsintsadze N, Kaushik A, Singh H.
HEALTH-RELATED CHALLENGES, PSYCHO-EMOTIONAL WELLBEING, AND ADAPTATION EXPERIENCES OF INTERNATIONAL STUDENTS IN A FOREIGN ACADEMIC ENVIRONMENT……6-13

Tan Kaiyou, Zhang Zhaoying.
EPIDEMIOLOGY AND POST-EXPOSURE MANAGEMENT OF RABIES EXPOSURES AT A COMMUNITY HEALTH CENTER: A 2024–2025 RETROSPECTIVE ANALYSIS……14-19

Josue Andres Orozco Pilco, Ximena del Rocío Robalino Flores, Silvia Paola Monar Basantes, Luisa Carolina González Ramírez.
RETICULOCYTE COUNT AND HEMATOLOGICAL INDICES IN MICROCYTIC HYPOCHROMIC ANEMIA: PREVALENCE AND PATTERNS AMONG INFANTS UNDER 24 MONTHS IN ANDEAN ECUADOR……20-25

Mirgul Kapakova, Jamilya Mansurova, Anar Kozhabayeva, Beshimbayeva Zhuldyz, Zhanar Toktarova, Meiramgul Tokbulatova, Galiya Assanova, Bolat Ashirov, Dariga Nurgalieva.
INDEPENDENT PREDICTORS OF SHORT-TERM MORTALITY IN PATIENTS WITH CHRONIC HEART FAILURE AND ANEMIA……26-32

Aynur Isayeva, Yusif Aliyev, Nergiz İmanova.
BASIC PRINCIPLES OF THE CONCEPT OF ACCELERATED POSTOPERATIVE RECOVERY AFTER SUBTOTAL COLECTOMY……33-36

David Gogoberidze, Irine Bakhutashvili.
MAJOR ADVERSE CARDIOVASCULAR EVENTS AFTER ACUTE CORONARY SYNDROME: DEFINITION, TIMING, RATES, MAIN RISK FACTORS, AND WAYS TO IMPROVE……37-43

Nataliya Byelyayeva, Pavlo Velychko, Olha Savitska, Zhanna Bogdan, Nataliia Ivantsev.
THE EFFECTIVENESS OF PSYCHOLOGICAL INTERVENTIONS IN MANAGING ANXIETY AND STRESS FOLLOWING TRAUMATICEVENTS……44-53

Hayfa A. Al-Olayan.
THIRTY-DAY INCIDENCE OF SURGICAL SITE INFECTION AND PERIOPERATIVE PROCESS COMPLIANCE IN A UNIVERSITY HOSPITAL DAY-SURGERY UNIT: A RETROSPECTIVE COHORT STUDY OF 200 PATIENTS……54-66

Grigoryan A.A, Makinyan L.G, Podlesnaya A.A, Imankulov M.A, Abu Zaalan V.M.
FACTORS ASSOCIATED WITH FUNCTIONAL OUTCOME AND SELECTION OF SURGICAL STRATEGY IN FOOT DROP SYNDROME ASSOCIATED WITH COMMON PERONEAL NEUROPATHY……67-72

Norah Hadi Mohammed, Tala Mustafa Ezzat, Fatima Alzahra Mukhtar, Ahmed. L. Osman, Marwan Ismail, Areeg Khalfallah, Ghina Yazbek, Sara Mohammed Ali, Abdelgadir Elamin Eltom.
CORRELATION OF HBA1C WITH INFLAMMATORY MARKERS AND NEUTROPHIL-TO-HIGH-DENSITY LIPOPROTEIN RATIO IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: A RETROSPECTIVE CROSS-SECTIONAL STUDY……73-75

Zainab Haitham Fathi, Ammar A.Y. Almulathanon.
ASSOCIATION BETWEEN INFERTILITY AND SERUM LEVELS OF ANTI-MÜLLERIAN HORMONE, FOLLICLE-STIMULATING HORMONE, LUTEINIZING HORMONE, AND PROLACTIN ACCORDING TO VITAMIN D SUPPLEMENTATION STATUS……76-82

Zhadra Kalbagayeva, Zaituna Khismetova, Nazym Iskakova, Aliya Atabayeva, Bakytkul Toktabayeva, Aigul Tugelbayeva, Sultan Seidumanov, Kamila Akhmetova, Bibinur Sydykova.
HEALTH-RELATED QUALITY OF LIFE AND ASSOCIATED FACTORS AMONG PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A CROSS-SECTIONAL STUDY……83-89

Tamta Chikobava, Ann Margvelashvili, Manana Kalandadze, Nino Tebidze, Vladimer Margvelashvili.
ASSESSMENT AND NONPHARMACOLOGICAL GUIDANCE OF CHILDREN'S BEHAVIOR AND DENTAL ANXIETY IN PEDIATRIC DENTISTRY: A STRUCTURED NARRATIVE REVIEW……90-97

Eglantina Bulica, Admir Sinamati, Alketa Tandili, Gentian Vyshka.
ETHICAL AND DEONTOLOGICAL DIMENSIONS OF RETINOPATHY OF PREMATURITY CARE: TOWARD A EUROPEAN PERSPECTIVE……98-103

Tingliang Tao, Yun Huang, Mengdan Xu, Shaoguo Wang, Lihong Bao.
MACHINE LEARNING-BASED IDENTIFICATION OF KEY GENES AND DEVELOPMENT OF A PREDICTIVE NOMOGRAM FOR PERIODONTITISDIAGNOSIS……104-111

Ahmed Abdelhadi Ahmed Nasr, Selma E. Hago Dawd, Ammar Abdelmola, Husham O. Elzein, Menna Ibrahim ALbader, Ezeldine K Abdalhabib, Elryah I Ali, Alneil Hamza, Mohammed Ibrahim Saeed, Salah Eldin Omar Hussein, Sara Mohammed Ali, Ayman H. Alfeel, Amar Mohamed Ismail, Omer Fadul Idris.
ASSOCIATION OF SER447TER, HINDIII AND PVUII LIPOPROTEIN LIPASE GENE POLYMORPHISMS WITH LIPID PROFILE IN CORONARY HEART DISEASE……112-120

Indira Adilbekova, Nishangul Bozhbanbayeva, Saule Issenova, Gulmira Altynbayeva, Dilfuza Sultanmuratova.
MATERNAL AND CORD-BLOOD HOMOCYSTEINE AND NEONATAL S100B IN PRETERM BIRTH: ASSOCIATIONS WITH IN-HOSPITAL NEONATAL OUTCOMES IN A PROSPECTIVE COHORT……121-130

Hayder G Oufi, Rana Hussein Kutaif, Ruaa E. Alabd, Hawraa Hussein Abd, Ranya Riyadh Hameed, Esraa Adnan Al-Muhanna, Dina Amer Abd, Riham Taha Yousif.
CLINICAL AND CULTURAL PERSPECTIVES ON POLYCYSTIC OVARY SYNDROME (PCOS) IN WOMEN BASED ON SYMPTOM CLUSTERING AND DIAGNOSTIC TRENDS…….131-141

Ferat Sallahu, Anamarija Jankulovska, Altin Sallahu.
LYMPHOVASCULAR AND PERINEURAL INVASION IN RELATION TO THE PRESENCE AND BURDEN OF NODAL METASTASIS IN RESECTED GASTRIC CANCER…….142-146

Toapanta Yugcha Iván, Toapanta Paredes Ximena, León Corrales Luis Sebastián, Maisincho Proaño Erika Fernanda, Alvarado Buenaño Marlon Andrés, Reinoso Molina Marcos Rubiño.
SOCIODEMOGRAPHIC FACTORS ASSOCIATED WITH SUICIDE RISK AMONG URBAN AND RURAL ADOLESCENTS IN AMBATO, ECUADOR…….147-151

Elmira Amanova, Gaukhar Abassova, Gulnaz Kaishibayeva, Vitaliy Kovalchuk, Yessengeldi Ismailov, Marzhan Abdrazakova, Sandugash Alipbekova, Saule Tuktibayeva, Kanatzhan Kemelbekov, Adlet Yernazarov.
FREQUENCY OF NEUROPATHIC PAIN FEATURES AMONG NEUROLOGICAL OUTPATIENTS AND THEIR IMPACT ON QUALITY OF LIFE: A CROSS-SECTIONAL SCREENING STUDY OF 1,705 PATIENTS…….152-162

Shaikha Fatmah Al Sharqia, Magdy Ali Abdou Gouda, Praveen Kumar, Shima Ibrahim Ali.
PERCEIVED CAUSES AND CONSEQUENCES OF DIAGNOSTIC ERRORS IN THE SECOND-TRIMESTER FETAL ANOMALY SCAN…….163-171

Moldir Tleubayeva, Meyirbek Aimagambetov, Kirill Krylov, Yernar Mamyrov, Marat Syzdykbayev, Samatbek Abdrakhmanov, Gulshat Alimkhanova, Assel Tulebayeva, Roman Khripunov.
NUTRITIONAL SUPPORT IN SURGICAL PATIENTS. PROTEIN-ENERGY METABOLISM DISORDERS…….172-180

Eranov Sh. N, Urinbaev Payzilla, Khodjanov Iskandar Yunusovich, Eranov Nurali Fayziyevich, Khalimova Fariza Tursunbaevna.
COMPREHENSIVE INSTRUMENTAL AND FUNCTIONAL ASSESSMENT OF POST-TRAUMATIC ELBOW CONTRACTURE IN CHILDREN AT THE INITIAL STAGE OF REHABILITATION…….181-187

Diana Dzidziguri, Irakli Chkhikvishvili, Maka Mantskava, Eka Shekiladze, Ekaterine Bakuradze, Tornike Liverashvili, Nina Kipiani, Sophio Turabelidze-Robaqidze, Tamar Sanikidze.
PHYTOCHEMICAL CHARACTERISATION, RADICAL-SCAVENGING ACTIVITY AND EXPERIMENTAL MICROCIRCULATORY RESPONSES TO GEORGIAN CUCURBITA MOSCHATA FLESH-WITH-PEEL EXTRACT…….188-195

Tchernev G, Tchernev KG Jr, Kordeva S.
DERMATOLOGIC SURGERY PEARLS: KERATINOCYTE CANCER AFFECTING HIGH RISK FACIAL REGIONS - LOGARITHMIC SPIRAL FLAP FOR NASAL ALAR RECONSTRUCTION AND V-TO-T FLAP FOR LOWER LIP RECONSTRUCTION…….196-203

Yerzhan Khochshanov, Dulat Baltabayev, Svetlana Khan, Yelena Aisenova, Zhibek Dautbayeva, Zhanar Akhmetova, Viktoriya Nazarova, Assel Meiramova.
OCULAR SURFACE AND TEAR FILM CHANGES IN GRAVES' DISEASE ACCORDING TO THYROID EYE DISEASE STATUS…….204-209

Laktionova M, Barmanasheva Z, Onglas A, Kulzhanov M, Baimuratova M, Sarsenbaeva M.
PREVALENCE AND RISK FACTORS OF PELVIC ORGAN PROLAPSE AMONG WOMEN IN ALMATY: A HOSPITAL-BASED STU DY…….210-217

Ahmed Abdelhadi Ahmed Nasr, Tagwa Yousif Elsayed Yousif, Ravshanova Maftuna Zoxidjonovna, Husham O. Elzein, HabibAllaa Eldaw Mohamed, Ezeldine K Abdalhabib, Elryah I Ali, Alneil Hamza, Mohammed Ibrahim Saeed, Salah Eldin Omar Hussein, Ayman H. Alfeel, Amar Mohamed Ismail, Omer Fadul Idris.
LIPOPROTEIN LIPASE GENE POLYMORPHISMS AND THEIR ASSOCIATION WITH APOLIPOPROTEIN A-I AND B-100 CONCENTRATIONS IN PATIENTS WITH CORONARY HEART DISEASE: A CROSS-SECTIONAL GENETIC ASSOCIATION STUDY …….218-225

Khamdamova Muhayyoxon Tukhtasinovna, Khalilova Mekhriniso Tuymurodovna, Askarova Zebo Zafarjonovna, Yusupova Mekhribon, Kambarova Dildora Noxidovna, Khalimova Fariza Tursunbaevna.
MORPHOLOGICAL FEATURES OF THE CERVICAL EPITHELIUM AND STROMA IN CHRONIC RECURRENT CERVICITIS WITH AND WITHOUT SQUAMOUS INTRAEPITHELIAL LESIONS: A COMPARATIVE CROSS-SECTIONAL STUDY…….226-233

GEORGIAN MEDICAL NEWS
No 9 (378) 2026

ASSOCIATION OF SER447TER, HINDIII AND PVUII LIPOPROTEIN LIPASE GENE POLYMORPHISMS WITH LIPID PROFILE IN CORONARY HEART DISEASE

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Abstract.

Background. Coronary heart disease (CHD) remains a leading cause of morbidity and mortality worldwide, and dyslipidaemia is one of its most important modifiable drivers. Lipoprotein lipase (LPL) is the rate-limiting enzyme for the intravascular hydrolysis of triglyceride-rich lipoproteins, and common polymorphisms in the *LPL* gene on chromosome 8p22, *Ser447Ter* (rs328), *Hin*dIII (rs320) and *Pvu*II (rs285), have been variably linked to circulating lipid levels and CHD susceptibility, with results that differ across populations.

Aim. To determine the distribution of the *Ser447Ter*, *Hin*dIII and *Pvu*II *LPL* polymorphisms in patients with CHD compared with healthy controls, and to examine the relationship between each polymorphism and the lipid profile after adjustment for adiposity and other cardiovascular risk factors.

Methods. A cross-sectional case–control study was conducted in 100 patients with established CHD (angiographically documented coronary stenosis ≥ 50%, prior myocardial infarction, or prior coronary revascularisation) and 50 apparently healthy, age- and sex-matched controls. Fasting venous blood was collected after a minimum 12-hour fast; serum lipid parameters were measured by standard enzymatic colorimetric methods, and LDL-C was calculated using the Friedewald equation when triglycerides were below 400 mg/dL and measured directly otherwise. Genomic DNA was extracted using the DNeasy Blood & Tissue Kit (Qiagen, Cat. No. 69504). Genotyping was performed by PCR-RFLP, with *Nla*III, *Hin*dIII and *Pvu*II used to digest the *Ser447Ter*, *Hin*dIII and *Pvu*II amplicons, respectively. Because expected cell counts were below five, genotype distributions were compared using Fisher's exact test. Between-group lipid comparisons used the Mann–Whitney U test and were repeated as analysis of covariance adjusted for BMI, age and sex. Genotype lipid relationships within the patient group were examined by multivariable linear regression adjusted for BMI, age, sex, diabetes, hypertension and smoking. Analyses were performed in IBM SPSS Statistics v26.

Results. Patients and controls did not differ in total cholesterol (median 166.5 vs 165.5 mg/dL, p = 0.485) or triglycerides (123.0 vs 129.0 mg/dL, p = 0.624). HDL-C was markedly lower in patients (39.0 vs 62.0 mg/dL, p < 0.001) and LDL-C, ApoA-I and ApoB-100 were higher (all p < 0.001); these differences persisted after adjustment for BMI, age and sex. Variant alleles were detected in 5%, 11% and 12% of patients and in 0%, 0% and 4% of controls at *Ser447Ter*, *Hin*dIII and *Pvu*II respectively. On Fisher's exact testing only *Hin*dIII was associated with CHD status (p = 0.016); *Ser447Ter* (p = 0.170) and *Pvu*II (p = 0.143) were not, and all odds ratio confidence intervals included unity. Within the patient group, *Hin*dIII variant carriage was independently associated with higher total cholesterol (adjusted β = +57.3 mg/dL, 95% CI 25.7 to 88.9, p < 0.001) and LDL-C (adjusted β = +62.1 mg/dL, 95% CI 35.1 to 89.2, p < 0.001) after adjustment for BMI, age, sex, diabetes, hypertension and smoking. No independent association was found for *Ser447Ter* or *Pvu*II.

Conclusion. Of the three loci examined, only *Hin*dIII showed a relationship with the lipid profile that was independent of adiposity and conventional risk factors, and the association ran through total and LDL cholesterol rather than through triglycerides and HDL-C. *Ser447Ter* and *Pvu*II showed no independent association. Because variant alleles were not detected in any control at two of the three loci, which is not consistent with published regional allele frequencies, the case–control genotype comparison is presented as hypothesis generating and requires confirmation with sequencing verified genotyping in a larger cohort.

Key words. Coronary heart disease, lipoprotein lipase, LPL, *Ser447Ter*, *Hin*dIII, *Pvu*II, Lipid profile, polymorphism, PCR-RFLP.

Introduction.

Coronary heart disease (CHD) remains the leading cause of mortality globally, accounting for an estimated 9.1 million deaths in 2021 [1,2]. Atherosclerosis, its underlying pathological substrate, develops over decades through the interplay of genetic predisposition, metabolic dysregulation and environmental exposure. Among modifiable determinants of atherosclerotic risk, abnormalities of lipid and lipoprotein metabolism occupy a central mechanistic role, with elevated total cholesterol (TC),

triglycerides (TG) and low-density lipoprotein cholesterol (LDL-C), together with reduced high-density lipoprotein cholesterol (HDL-C), forming the classical atherogenic lipid profile [3-5].

Beyond environmental and metabolic determinants, genetic variation in lipid-regulating enzymes contributes meaningfully to inter-individual variability in plasma lipids and, consequently, to CHD susceptibility. Lipoprotein lipase (LPL; EC 3.1.1.34) is the rate-limiting enzyme for the intravascular hydrolysis of triglycerides carried in chylomicrons and very-low-density lipoproteins (VLDL), generating free fatty acids for tissue uptake and contributing to the maturation of HDL particles [6-9]. LPL therefore operates at the intersection of the pro-atherogenic (apoB-containing VLDL/LDL) [10,11] and anti-atherogenic (HDL) branches of lipoprotein metabolism: reduced LPL activity drives hypertriglyceridaemia, accumulation of remnant lipoproteins and a more atherogenic lipid phenotype [12], whereas genetically enhanced LPL-mediated lipolysis has been associated with lower coronary disease risk in large Mendelian-randomization analyses [13].

The human *LPL* gene maps to chromosome 8p21.3, spans approximately 30 kb, and contains 10 exons interrupted by 9 introns; its product is a 448-amino-acid mature protein [6,7]. More than a hundred sequence variants have been described [14,15], but three polymorphisms detectable by restriction fragment length polymorphism (RFLP) have attracted sustained interest because of their high allele frequencies and reproducible associations with lipid traits and CHD:

The ***Ser447Ter*** (also referred to as *S447X*; rs328) polymorphism is a C→G transversion in exon 9 that introduces a premature stop codon and truncates the mature protein by two amino acids. The truncated enzyme is generally associated with enhanced lipolytic activity, lower plasma TG and higher HDL-C, and several meta-analyses have reported a protective effect against coronary artery disease, although findings are not uniform across populations [13,16].

The ***Hin*dIII** polymorphism (rs320) is a T→G substitution located in intron 8. The H+ allele has been linked in some cohorts to elevated TG and reduced HDL-C [17,18], while cumulative meta-analyses report either protective or risk-conferring effects depending on ethnicity and the genetic model tested [16,19]. Although intronic, *Hin*dIII may serve as a marker for functional variants in linkage disequilibrium across the *LPL* gene block [20].

The ***Pvu*II** polymorphism (rs285) lies in intron 6 and, although it does not alter protein structure, has been variably reported to modify CHD risk in different populations, again most plausibly through linkage disequilibrium with regulatory variants [16,20].

Despite an extensive global literature, the data for these three polymorphisms remain heterogeneous. Studies in Caucasian, East Asian, Saudi, Iranian and Turkish cohorts have variously reported significant, weak, or absent associations with both lipid parameters and CHD outcomes, reflecting differences in allele frequency, sample size, case definition and underlying environmental exposures [16-19,21,22]. There is a continuing need for population-specific data, particularly from regions where epidemiological transitions are driving rapid increases in CHD incidence [23] and where pharmacogenetic and risk-stratification frameworks remain underdeveloped.

The present investigation was therefore designed to (i) determine the distribution of the *Ser447Ter*, *Hin*dIII and *Pvu*II *LPL* polymorphisms in patients with CHD and matched controls; (ii) characterize the lipid profile across genotype categories; and (iii) quantify the association of each polymorphism with the lipid profile and with CHD susceptibility using models adjusted for adiposity and conventional cardiovascular risk factors. The same participants have been examined in a companion analysis of apolipoprotein A-I and apolipoprotein B-100 concentrations; the two analyses address different outcomes in the same cohort and neither duplicates the other.

Materials and Methods.

Study design and setting:

This was a cross-sectional case–control study conducted at Sudan Heart Centre, Khartoum, Sudan. The protocol was reviewed and approved by the Central Institutional Review Board of Al Neelain University, Sudan (approval date 1 March 2015. All subjects enrolled in this study were Sudanese residents with similar dietary patterns. Key demographic data were recorded including age, gender, weight and height, and the study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from every participant prior to enrolment. The study is reported in accordance with the STREGA extension of the STROBE statement for genetic association studies.

Participants:

A total of 150 individuals were enrolled: 100 patients with established CHD and 50 apparently healthy controls.

Cases: Adult patients of either sex with established CHD, defined as: a history of acute myocardial infarction; documented coronary artery stenosis ≥ 50% on coronary angiography; or prior coronary revascularisation (percutaneous coronary intervention or coronary artery bypass grafting). Patients were consecutively recruited from the cardiology service. Eligible patients were aged 25–74 years.

Controls: Apparently healthy adults with no clinical, electrocardiographic or laboratory evidence of cardiovascular disease, age- and sex-matched to the case group, recruited from outpatient clinics and the surrounding community. Controls were recruited separately from the general Sudanese population and were not drawn from the Sudan Heart Centre patient pool. They were adult volunteers with no personal history of coronary artery disease, confirmed at enrolment using a Medical History and Symptom Questionnaire. Controls were selected to reflect the age and sex distribution of the CHD group at group level, giving an approximate 2:1 case-to-control design; individual one-to-one matching was not performed. Controls were not screened on lipid values, and no lipid-based exclusion was applied.

Exclusion criteria (applied to both groups): Secondary dyslipidaemia attributable to thyroid, renal or hepatic disease; acute infectious or inflammatory illness; pregnancy; malignancy; women receiving hormonal therapy; patients receiving cancer therapy; and inability to provide informed consent.

A structured interviewer-administered questionnaire was used to record demographic data, anthropometric measurements

(height, weight, body mass index BMI), smoking status, history of hypertension and diabetes mellitus, family history of premature CHD, and current medications. Body mass index was classified using World Health Organization categories as normal (< 25 kg/m²), overweight (25.0–29.9 kg/m²) and obese (≥ 30 kg/m²).

Sample collection and lipid profiling:

After a minimum 12-hour overnight fast, 10 mL of venous blood was collected from each participant. The sample was divided between an EDTA tube (for DNA extraction) and a plain tube (for serum biochemistry). Serum was separated by centrifugation at 3,000 rpm for 10 minutes within one hour of collection, aliquoted, and stored at −80 °C until analysis. Total cholesterol (TC), triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) were measured by standard enzymatic colorimetric methods on an automated clinical chemistry analyser , using commercial reagents according to the manufacturer's instructions. Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald equation [25] when TG values were < 400 mg/dL; in the two patients whose triglycerides exceeded this threshold, LDL-C was measured directly. Serum apolipoprotein A-I and apolipoprotein B-100 concentrations were determined by immunoturbidimetry using standardised commercial kits. Internal quality-control material was run with each batch, and the laboratory participated in an external quality-assurance programme throughout the study period.

DNA extraction:

Genomic DNA was extracted from peripheral blood leukocytes using the DNeasy Blood & Tissue Kit (Qiagen, Hilden, Germany; Cat. No. 69504) in accordance with the manufacturer's protocol. DNA purity and concentration were assessed spectrophotometrically (A260/A280 ratio in the range 1.7–1.9), and DNA integrity was verified on a 1% agarose gel. Extracted DNA was stored at −20 °C until genotyping.

Genotyping by PCR-RFLP:

Genotyping of the three *LPL* polymorphisms was performed by polymerase chain reaction–restriction fragment length polymorphism (PCR-RFLP) using primer sequences and reaction conditions adapted from previously validated protocols [17,18,20].

***Ser447Ter* (rs328).** A fragment encompassing exon 9 was amplified using forward primer 5′-TAC ACT AGC AAT GTC TAG GTG A-3′ and reverse primer 5′-TCA GCT TTA GCC CAG AAT GC-3′. The PCR product was digested overnight at 37 °C with the restriction endonuclease ***Nla*III** ([**insert supplier**]), generating distinguishable fragment patterns for the homozygous wild-type (G/G), heterozygous (G/C) and homozygous variant (C/C) genotypes.

***Hin*dIII (rs320).** A fragment encompassing the intron 8 region was amplified using forward primer (H1) 5′-TGA AGC TCA AAT GGA AGA GT-3′ and reverse primer (H2) 5′-TAC AAG CAA ATG ACT AAA-3′. The amplicon was digested overnight at 37 °C with ***Hin*dIII** (Promega Corporation, Madison, WI, USA), yielding the H+ (restriction-site present) and H− (restriction-site absent) alleles, and producing distinguishable patterns for the H+H+, H+H− and H−H− genotypes.

***Pvu*II (rs285).** A fragment encompassing the intron 6 region was amplified using forward primer (P1) 5′-ATG GCA CCC ATG TGT AAG GTG-3′ and reverse primer (P2) 5′-GTG AAC TTC TGA TAA CAA TCT C-3′. The amplicon was digested overnight at 37 °C with ***Pvu*II** (Promega Corporation, Madison, WI, USA), yielding the P+ (restriction-site present) and P− (restriction-site absent) alleles, with corresponding P−P−, P−P+ and P+P+ patterns.

Each 25 µL PCR reaction contained 3 µL of genomic DNA (150 ng), 12.5 µL of 5× FIREPol® Master Mix (Solis BioDyne, Tartu, Estonia), 2 µL of each primer and distilled water to final volume. Thermal cycling consisted of an initial denaturation at 94 °C for 2 minutes; 40 cycles of 94 °C for 15 s, annealing at 50 °C for 30 s and extension at 72 °C for 1 minute; and a final extension at 72 °C for 2 minutes. Restriction digestion reactions (20 µL final volume) contained 10 µL of PCR product, 1 µL of the appropriate restriction enzyme and 2 µL of the corresponding 10× buffer, made up to volume with distilled water; reactions were briefly centrifuged (2 minutes at 3,913 × g) and incubated at 37 °C overnight. Digestion products were resolved on 2.5% agarose gels at 80 V for 60 minutes, stained, and visualised under ultraviolet trans-illumination. These conditions are those used for the companion analysis of the same samples [39] and supersede the PCR and electrophoresis conditions given in the original submission, which were transcribed in error. Negative (no-template) and previously genotyped positive controls were included in every run, and approximately 10% of samples were re-genotyped to confirm reproducibility; concordance was 100%. Restriction-fragment sizes for each genotype call were not recorded at the time of genotyping and cannot be reported [author to supply if gel records survive].

Genotyping quality control and its limitations:

Repeat genotyping used the same PCR-RFLP assay, so it establishes reproducibility but not accuracy: a systematically incomplete restriction digestion would be reproduced rather than detected, because undigested product is scored as the homozygous common-allele genotype. No independent method such as Sanger sequencing was used to verify genotype calls, and heterozygote positive controls were not run on every digestion gel. The implications of this for the observed allele frequencies are considered in the Limitations.

Statistical analysis:

Continuous variables were tested for normality using the Shapiro–Wilk test. Because total cholesterol, triglycerides, HDL-C, LDL-C, ApoA-I, ApoB-100, BMI and age were all non-normally distributed in at least one group, continuous data are presented as median with interquartile range and compared using the Mann–Whitney U test; means with standard deviations are also given to allow comparison with earlier reports. Categorical variables were summarised as frequencies and percentages and compared using the Pearson chi-square test or Fisher's exact test where any expected cell count was below five.

Genotype and allele frequencies were calculated by direct counting. Conformity to Hardy–Weinberg equilibrium was assessed in both groups using the exact test of Wigginton et al. [40] rather than the chi-square goodness-of-fit approximation, because several genotype cells contained small counts; the test is informative only where the group is polymorphic at that locus.

Because the minimum expected cell count was below five at all three loci, the association between each *LPL* polymorphism and CHD status was tested using Fisher's exact test rather than the Pearson chi-square. Odds ratios are reported with 95% confidence intervals; where no control participant carried a variant allele, a Haldane–Anscombe continuity correction of 0.5 was applied to every cell, and the resulting estimate is identified as such. Relative risks were not estimated, because incidence cannot be derived from a case–control sample.

Between-group lipid comparisons were repeated as analysis of covariance with BMI, age and sex as covariates, and adjusted mean differences are reported with 95% confidence intervals. The relationship between genotype and the lipid profile within the patient group was examined by multivariable linear regression of total cholesterol and LDL-C on genotype (variant allele present versus absent), adjusted for BMI, age, sex, diabetes, hypertension and smoking; regression coefficients are reported with 95% confidence intervals. Comorbidity data were collected for the patient group only, so case–control models could be adjusted for BMI, age and sex alone. A logistic model of CHD status on genotype could not be fitted for *Ser447Ter* or *Hin*dIII because the absence of variant carriers among controls produces complete separation and the genotype coefficient does not converge; this is reported rather than worked around. The genotype–lipid analyses are unadjusted for multiple comparisons and are reported as exploratory. A two-tailed p value of less than 0.05 was considered statistically significant. All analyses were performed using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA).

Results.

Demographic and genetic characteristics:

One hundred patients with established CHD and 50 age- and sex-matched healthy controls were enrolled. The gender distribution was balanced between groups (54% male in patients vs 50% in controls). Most participants in both groups were older than 40 years (70% of patients and 76% of controls). Body mass index differed markedly between the groups: 68% of patients were obese (BMI ≥ 30 kg/m²) compared with 4% of controls, and 98% of patients were overweight or obese (BMI ≥ 25 kg/m²) compared with 30% of controls ($p < 0.001$). We note that the figures of 98% and 30% reported in the original submission corresponded to the overweight-or-obese category and were incorrectly labelled as obesity; the corrected classification is given in Table 1.

Variant alleles were detected in 5% of patients at *Ser447Ter* (all heterozygous G/C, with no C/C homozygotes), in 11% at *Hin*dIII (H+H− 8%; H−H− 3%) and in 12% at *Pvu*II (P−P+ 9%; P+P+ 3%). No control participant carried a variant allele at *Ser447Ter* or *Hin*dIII. At *Pvu*II, two controls (4%) were heterozygous (P−P+); the original submission reported the control *Pvu*II distribution as entirely wild-type, which was a transcription error, and the corrected counts are given in Table 1. The control *Pvu*II genotype distribution was consistent with Hardy–Weinberg equilibrium ($\chi^2 = 0.021$, df = 1, $p = 0.885$, minor allele frequency 0.020). Hardy–Weinberg equilibrium could not be tested at *Ser447Ter* or *Hin*dIII because the control group was monomorphic at both loci.

Clinical characteristics and lipid profile:

Clinical and biochemical profiles are summarised in Table 2. Age was comparable between groups (median 49.0 vs 49.5 years, $p = 0.538$), while BMI was substantially higher in patients (median 32.5 vs 24.1 kg/m², $p < 0.001$).

Among the conventional lipid parameters, neither total cholesterol (median 166.5 vs 165.5 mg/dL, $p = 0.485$) nor triglycerides (123.0 vs 129.0 mg/dL, $p = 0.624$) differed significantly between groups. HDL-C was markedly lower in patients (39.0 vs 62.0 mg/dL, $p < 0.001$) and LDL-C was higher (104.0 vs 80.0 mg/dL, $p < 0.001$). Both apolipoproteins were higher in patients: ApoA-I 140.0 vs 115.0 mg/dL ($p < 0.001$) and ApoB-100 118.0 vs 81.5 mg/dL ($p < 0.001$). Triglyceride values in the patient group were strongly right-skewed, with two patients exceeding 1,000 mg/dL, which is why median-based comparison is reported in preference to the mean.

Because obesity was far more prevalent among patients, the lipid comparisons were repeated with adjustment for BMI, age and sex (Table 3). The differences in HDL-C, LDL-C, ApoA-I and ApoB-100 all persisted after adjustment and were, if anything, larger; total cholesterol and triglycerides remained non-significant. Adiposity therefore does not account for the lipid differences observed between the two groups.

Lipid profile across LPL genotype strata and adjusted genotype–lipid analysis:

Mean lipid values stratified by genotype within the CHD patient group are shown in Table 4, together with multivariable regression coefficients adjusted for BMI, age, sex, diabetes, hypertension and smoking.

Ser447Ter. The five G/C heterozygotes had higher mean TC (212.6 vs 175.9 mg/dL) and LDL-C (140.6 vs 107.0 mg/dL) than the 95 G/G carriers. Neither difference reached significance after adjustment (TC adjusted $\beta = +40.0$ mg/dL, 95% CI −6.4 to +86.3, $p = 0.090$; LDL-C adjusted $\beta = +37.4$ mg/dL, 95% CI −3.7 to +78.5, $p = 0.074$). With only five carriers these estimates are imprecise and are reported descriptively.

***Hin*dIII.** Non-H+H+ carriers (n = 11) had substantially higher mean TC (220.0 vs 172.5 mg/dL) and LDL-C (156.8 vs 102.7 mg/dL) than H+H+ carriers (n = 89). These were the only genotype–lipid relationships that survived multivariable adjustment: TC adjusted $\beta = +57.3$ mg/dL (95% CI +25.7 to +88.9, $p < 0.001$) and LDL-C adjusted $\beta = +62.1$ mg/dL (95% CI +35.1 to +89.2, $p < 0.001$). Adjustment for BMI and the other covariates increased rather than attenuated both coefficients (unadjusted $\beta = +47.5$ and +54.1 mg/dL respectively), indicating that the association is not a consequence of the greater adiposity of the patient group. No relationship was observed with TG or HDL-C.

***Pvu*II.** Non-P−P− carriers (n = 12) had mean TC and LDL-C values close to those of P−P− carriers (183.6 vs 176.9 and 112.2 vs 108.2 mg/dL). Neither association approached significance after adjustment (TC adjusted $\beta = +12.8$ mg/dL, 95% CI −18.7 to +44.4, $p = 0.421$; LDL-C adjusted $\beta = +4.1$ mg/dL, 95% CI −24.0 to +32.2, $p = 0.773$).

Association between LPL genotype and CHD status:

Genotype distributions were compared between patients and controls using Fisher's exact test, because the minimum

expected cell count was below five at all three loci (Table 5).

*Hin*dIII was the only locus associated with CHD status (11% versus 0% carriers, p = 0.016). *Ser447Ter* (5% versus 0%, p = 0.170) and *Pvu*II (12% versus 4%, p = 0.143) were not significantly associated. Odds ratios were 12.98, 5.82 and 3.27 respectively, and every confidence interval included unity, so none of the three loci can be said to show a statistically significant elevation in odds. The *Ser447Ter* and *Hin*dIII odds ratios required a continuity correction because no control carried a variant allele, and both are correspondingly imprecise, with intervals spanning more than two orders of magnitude.

Two features of these data require explicit statement. First, the significance levels differ from those reported in the original submission, which were based on Pearson chi-square statistics that could not be reproduced from the genotype counts and on a control *Pvu*II distribution that has since been corrected. Second, the absence of variant carriers among controls at two loci means that the corresponding effect estimates rest on a structural zero and cannot be adjusted for confounders, since a logistic model containing these terms does not converge. These results are therefore presented as descriptive and hypothesis-generating rather than as risk estimates. Relative risks, which were reported in the original submission, have been removed, as incidence is not estimable from a case–control sample.

Hardy–Weinberg equilibrium:

Exact testing for deviation from Hardy–Weinberg equilibrium was performed separately in each group (Table 6). Among patients, *Ser447Ter* was in equilibrium (p = 1.000), whereas *Hin*dIII (p = 0.005) and *Pvu*II (p = 0.009) both deviated significantly. In each case the deviation took the same form: fewer heterozygotes and more variant homozygotes than expected from the observed allele frequency. At *Hin*dIII, 8 heterozygotes and 3 variant homozygotes were observed where 13.0 and 0.5 would be expected; at *Pvu*II, 9 and 3 were observed where 13.9 and 0.6 would be expected. Among controls, the *Pvu*II distribution was in equilibrium (p = 1.000), and the remaining two loci were monomorphic and could not be tested.

A heterozygote deficit accompanied by an excess of variant homozygotes at two independent loci, in the same direction and in the same samples, is the pattern expected when a restriction digest does not run to completion and a proportion of heterozygous gels are scored as one or other homozygote. It is also compatible with population stratification or with sampling variation in small cells. These possibilities cannot be

Table 1. *Demographic and genetic characteristics of CHD patients and controls.*

Characteristic	Category	CHD n	CHD %	Control n	Control %
Gender	Male	54	54.0	25	50.0
	Female	46	46.0	25	50.0
Age group	< 40 years	30	30.0	12	24.0
	≥ 40 years	70	70.0	38	76.0
BMI category	Normal (< 25 kg/m²)	2	2.0	35	70.0
	Overweight (25.0–29.9 kg/m²)	30	30.0	13	26.0
	Obese (≥ 30 kg/m²)	68	68.0	2	4.0
Ser447Ter	GG (wild-type)	95	95.0	50	100.0
	GC (heterozygote)	5	5.0	0	0.0
	CC (homozygote)	0	0.0	0	0.0
HindIII	H+H+ (wild-type)	89	89.0	50	100.0
	H+H− (heterozygote)	8	8.0	0	0.0
	H−H− (homozygote)	3	3.0	0	0.0
PvuII	P−P− (wild-type)	88	88.0	48	96.0
	P−P+ (heterozygote)	9	9.0	2	4.0
	P+P+ (homozygote)	3	3.0	0	0.0

CHD: coronary heart disease; BMI: body mass index; LPL: lipoprotein lipase. BMI categories follow World Health Organization definitions. Control PvuII counts and the BMI classification are corrected from the original submission.

Table 2. *Clinical characteristics and lipid profile of CHD patients versus controls.*

Parameter	CHD patients (n = 100)	Controls (n = 50)	p value*	Mean ± SD, CHD / control
Age (years)	49.0 [37.8–65.0]	49.5 [40.0–58.8]	0.538	51.8 ± 16.8 / 48.8 ± 13.1
BMI (kg/m²)	32.5 [29.3–37.4]	24.1 [23.2–25.1]	< 0.001	34.2 ± 6.6 / 24.5 ± 2.4
TC (mg/dL)	166.5 [147.5–198.8]	165.5 [158.0–177.0]	0.485	177.7 ± 49.2 / 167.1 ± 13.6
TG (mg/dL)	123.0 [93.8–162.8]	129.0 [117.2–141.8]	0.624	151.5 ± 153.5 / 127.8 ± 16.4
HDL-C (mg/dL)	39.0 [34.0–46.2]	62.0 [58.0–65.8]	< 0.001	42.1 ± 15.0 / 61.7 ± 7.0
LDL-C (mg/dL)	104.0 [85.0–128.0]	80.0 [71.2–90.0]	< 0.001	108.6 ± 42.9 / 79.2 ± 12.5
ApoA-I (mg/dL)	140.0 [124.8–164.5]	115.0 [98.5–143.2]	< 0.001	144.0 ± 37.5 / 120.9 ± 30.5
ApoB-100 (mg/dL)	118.0 [82.8–149.0]	81.5 [65.0–98.5]	< 0.001	121.1 ± 45.0 / 87.5 ± 30.4

*Data are median [interquartile range]. TC: total cholesterol; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; ApoA-I: apolipoprotein A-I; ApoB-100: apolipoprotein B-100. *Mann–Whitney U test, used because the Shapiro–Wilk test indicated non-normality for every variable in at least one group. Mean ± SD values are given in the final column for comparability with earlier reports and were recomputed from the primary dataset.*

Table 3. *Lipid and apolipoprotein differences between groups before and after adjustment for BMI, age and sex.*

Parameter	Unadjusted difference (CHD − control)	Adjusted difference (95% CI)*	Adjusted p
TC (mg/dL)	+10.65	+11.40 (−7.01 to +29.81)	0.223
TG (mg/dL)	+23.72	+32.08 (−23.99 to +88.16)	0.260
HDL-C (mg/dL)	−19.62	−23.94 (−29.60 to −18.27)	< 0.001
LDL-C (mg/dL)	+29.40	+30.71 (+14.56 to +46.86)	< 0.001
ApoA-I (mg/dL)	+23.12	+27.73 (+12.46 to +43.00)	0.001
ApoB-100 (mg/dL)	+33.61	+43.41 (+25.38 to +61.44)	< 0.001

**Analysis of covariance with BMI, age and sex as covariates. A positive value indicates a higher mean in the CHD group.*

Table 4. *Lipid profile by LPL genotype within CHD patients, and adjusted regression coefficients.*

Polymorphism	Genotype	TC	TG	HDL-C	LDL-C	Adjusted β (95% CI), p*
Ser447Ter	GG (n = 95)	175.9 ± 48.5	152.9 ± 157.2	41.8 ± 15.1	107.0 ± 42.4	TC +40.0 (−6.4 to +86.3), p = 0.090
	GC (n = 5)	212.6 ± 54.5	124.2 ± 40.0	47.2 ± 10.4	140.6 ± 42.8	LDL-C +37.4 (−3.7 to +78.5), p = 0.074
*Hin*dIII	H+H+ (n = 89)	172.5 ± 48.0	152.1 ± 162.2	42.5 ± 15.5	102.7 ± 39.8	**TC +57.3 (+25.7 to +88.9), p < 0.001**
	Non-H+H+ (n = 11)	220.0 ± 37.4	146.8 ± 40.6	38.7 ± 8.9	156.8 ± 37.1	**LDL-C +62.1 (+35.1 to +89.2), p < 0.001**
*Pvu*II	P−P− (n = 88)	176.9 ± 50.6	153.1 ± 162.6	42.4 ± 15.7	108.2 ± 43.2	TC +12.8 (−18.7 to +44.4), p = 0.421
	Non-P−P− (n = 12)	183.6 ± 38.0	139.5 ± 52.6	39.6 ± 7.0	112.2 ± 41.9	LDL-C +4.1 (−24.0 to +32.2), p = 0.773

*Lipid values are mean ± SD in mg/dL. Non-H+H+ combines H+H− and H−H−; Non-P−P− combines P−P+ and P+P+. *Multivariable linear regression within the 100 CHD patients, with variant allele carriage as the predictor and BMI, age, sex, diabetes, hypertension and smoking as covariates. β is the adjusted mean difference in mg/dL for variant carriers relative to wild-type carriers. The point-biserial correlations reported in the original submission have been replaced by these adjusted coefficients in response to peer review.*

Table 5. *Association between LPL polymorphisms and CHD status.*

Polymorphism	CHD carriers n (%)	Control carriers n (%)	Fisher's exact p	Odds ratio (95% CI)
Ser447Ter (rs328)	5 (5.0)	0 (0.0)	0.170	5.82 (0.32–107.33)†
*Hin*dIII (rs320)	11 (11.0)	0 (0.0)	0.016	12.98 (0.75–224.90)†
*Pvu*II (rs285)	12 (12.0)	2 (4.0)	0.143	3.27 (0.70–15.23)

Carriers are participants with at least one variant allele (heterozygote plus homozygote). †Odds ratio calculated after a Haldane–Anscombe continuity correction of 0.5 per cell, applied because no control participant carried a variant allele at this locus. All confidence intervals include unity. Relative risks are not reported, as incidence is not estimable from a case–control design.

Table 6. *Exact test for Hardy–Weinberg equilibrium by group.*

Polymorphism	Group	Observed (wild / het / variant hom)	Expected under HWE	Exact p	Interpretation
Ser447Ter	CHD	95 / 5 / 0	95.1 / 4.8 / 0.1	1.000	In equilibrium
	Control	50 / 0 / 0	—	—	Monomorphic, not testable
*Hin*dIII	CHD	89 / 8 / 3	86.5 / 13.0 / 0.5	0.005	Heterozygote deficit
	Control	50 / 0 / 0	—	—	Monomorphic, not testable
*Pvu*II	CHD	88 / 9 / 3	85.6 / 13.9 / 0.6	0.009	Heterozygote deficit
	Control	48 / 2 / 0	48.0 / 2.0 / 0.0	1.000	In equilibrium

Exact test of Wigginton et al. [40]. Expected counts are derived from the observed allele frequency within each group.

separated with the present data and are considered further in the Limitations.

Discussion.

In this case–control study of 100 Sudanese patients with angiographically or clinically established CHD and 50 healthy controls, one of the three *LPL* loci examined showed a relationship with the lipid profile that was independent of adiposity and conventional cardiovascular risk factors. Carriage of a non-H+H+ *Hin*dIII genotype was associated with a mean increase of 57 mg/dL in total cholesterol and 62 mg/dL in LDL-C after adjustment for BMI, age, sex, diabetes, hypertension and smoking, and *Hin*dIII was also the only locus whose genotype distribution differed between patients and controls. Neither *Ser447Ter* nor *Pvu*II showed an independent association with either the lipid profile or CHD status. The lipid phenotype of the patient group was characterised by low HDL-C and raised LDL-C, ApoA-I and ApoB-100, with no difference in total cholesterol or triglycerides, and these differences persisted after adjustment for BMI.

These findings sit against a literature that is itself markedly heterogeneous. The largest pooled analysis to date, a HuGE review of 89 studies in more than 70,000 subjects, found that carriers of the minor *Hin*dIII and *Ser447Ter* alleles tended to have a more favourable lipid profile than common-allele homozygotes [27], and a later cumulative meta-analysis similarly classed both *Ser447Ter* and *Hin*dIII as protective for CAD, with only D9N conferring risk and *Pvu*II showing no association [28]. A meta-analysis focused specifically on *Ser447Ter* estimated an approximately 22% reduction in CAD risk per copy of the minor allele [29], and the case–control studies from Denmark and the USA that established the directionality now widely used in the

field found the common *Hin*dIII(+) allele and H+H+ genotype, not the minor allele, associated with lower HDL-C, higher triglycerides and a modestly elevated risk of angiographic CAD [30,31]. Our *Pvu*II result, a null association, is consistent with this literature. Our *Hin*dIII result is not: it was carriage of the minor allele that tracked with higher total and LDL cholesterol, broadly the reverse of the pattern reported in much of the foundational literature. This is not without precedent: a case–control study from Shiraz, Iran, similarly found the *Ser447Ter* minor allele over-represented among CHD cases rather than controls [32].

The comparisons that bear most directly on the present cohort come from the same region. Two independent Saudi Arabian case–control studies, together comprising 346 CHD patients and 175 controls, found no significant association between any of the three *LPL* loci examined here and CAD [33,34], and an earlier Saudi study using the same *Hin*dIII and *Pvu*II assays reached the same conclusion [35]. The only previous Sudanese study of *LPL* and CHD, which examined the D9N, N291S and S447X variants, also found no significant association between *LPL* genotype and CHD, and reported that patients had significantly lower TC and LDL-C than controls [36]. The S447X carrier frequency in that cohort (7.1%) was close to the 5% observed here, suggesting that the *Ser447Ter* allele frequency in Sudan is not markedly unusual. By contrast, an angiographic cohort from Tunisia found a *Hin*dIII genotype significantly associated with coronary stenosis severity, but mediated through elevated triglycerides and reduced HDL-C rather than through TC or LDL-C [37]. Even within North African and Arab populations, therefore, the polymorphism–lipid relationship is not consistent in direction or in the lipid fraction affected.

Mechanistically, LPL hydrolyses the triglyceride core of chylomicrons and VLDL, and most functional accounts of these polymorphisms emphasise downstream effects on triglycerides and HDL-C [30,31]. Our *Hin*dIII signal instead appeared in total and LDL cholesterol, with no relationship to TG or HDL-C. One plausible explanation is that reduced LPL-mediated clearance of triglyceride-rich remnants increases delivery of cholesterol-enriched remnant particles to the LDL pool, so that in a cohort where triglycerides themselves did not differ between groups, a genotype effect would surface first in the cholesterol-carrying fractions [4,25]. Because rs320 is intronic and does not alter the protein sequence, any functional effect is most plausibly mediated through linkage disequilibrium with regulatory variants elsewhere in the *LPL* block [20], and we did not measure LPL mass or activity, so this interpretation remains inferential.

The reviewer of an earlier version of this manuscript raised the possibility that the genotype–lipid associations were driven by adiposity rather than by genotype, and this deserves a direct answer. Obesity was indeed far more common among patients (68% versus 4%). However, the *Hin*dIII association with total and LDL cholesterol was examined within the patient group, where BMI was included as a covariate alongside age, sex, diabetes, hypertension and smoking, and the adjusted coefficients were larger than the unadjusted ones. Adiposity therefore does not explain this association. The same applies to the case–control lipid differences, which persisted essentially unchanged after adjustment for BMI, age and sex. What adiposity cannot be excluded from is the case–control genotype comparison, because the structural zero at two loci prevents a confounder-adjusted model from being fitted at all.

A further observation bears on the reliability of the genotype data. Both *Hin*dIII and *Pvu*II deviated from Hardy–Weinberg equilibrium among patients, in each case through a deficit of heterozygotes and an excess of variant homozygotes. The same pattern at two independent intronic loci in the same samples is difficult to explain biologically, since these loci are not in linkage disequilibrium with each other in a way that would produce identical departures, and it is the pattern that incomplete restriction digestion would generate. Read together with the absence of variant alleles among controls at two loci, it points toward an assay problem rather than a population characteristic.

Strengths and Limitations.

The principal strengths of this study are the assessment of three *LPL* loci in the same cohort, the use of angiographic or event-based case definitions rather than symptom-based ones, the availability of apolipoprotein measurements alongside the standard lipid panel, and the use of multivariable models that adjust the genotype–lipid relationship for adiposity and conventional risk factors.

The limitations are substantial. The most important concerns the control genotype data. No control participant carried a variant allele at *Ser447Ter* or *Hin*dIII. Using control genotype frequencies reported for regional populations in the studies cited above, the probability of observing zero carriers among 50 controls at *Hin*dIII is of the order of 10^{-15} [17,33,35], and the observed carrier frequency in the patient group (11%) is also well below the 50 to 58% reported in those studies. The *Pvu*II minor allele frequency in our controls (0.020) is likewise far below published values. Taken together, these observations suggest systematic under-detection of variant alleles rather than a genuine population difference, most plausibly incomplete restriction digestion scored as the homozygous common-allele genotype. Because repeat genotyping used the same assay, it would not have detected such an error. The case–control genotype comparison in Table 5 must therefore be regarded as unreliable, and confirmation by Sanger sequencing in a future study is required before these frequencies are used elsewhere. The within-patient genotype–lipid analysis is less affected, since under-detection of heterozygotes would misclassify some carriers as wild-type and would bias the estimated coefficients toward the null rather than away from it.

Second, the study was underpowered for genetic association. To detect an allelic odds ratio of 1.5 at *Hin*dIII would require approximately 200 to 320 participants per group depending on the assumed allele frequency, and at *Ser447Ter* more than 1,000 per group; the present sample is a fraction of this. Third, the dispersion of the control lipid values is narrower than would be expected in an unselected adult population, particularly for triglycerides (coefficient of variation 12.8%, against roughly 40% in unselected adults). Controls were not screened on lipid values, so restriction of range by selection does not account for this, and we are unable to offer a satisfactory explanation; the case–control lipid comparisons should be read with that reservation. Fourth, comorbidity data were available for the

patient group only, so case–control models could be adjusted for BMI, age and sex alone. Fifth, lipid-lowering therapy was recorded but was not incorporated into the lipid analyses, which will have attenuated the case–control cholesterol differences. Sixth, the genotype–lipid analyses were not corrected for multiple comparisons and should be treated as exploratory. Finally, LPL mass and activity were not measured, and the design is cross-sectional, so no temporal or causal inference is possible.

Conclusion.

In this Sudanese cohort, carriage of a non-H+H+ *Hin*dIII genotype was independently associated with higher total and LDL cholesterol among patients with coronary heart disease, and the association was not explained by adiposity, age, sex, diabetes, hypertension or smoking. Neither *Ser447Ter* nor *Pvu*II showed an independent association with the lipid profile, and on exact testing only *Hin*dIII differed in genotype distribution between patients and controls. The case–control genotype comparison rests on a control group in which variant alleles were not detected at two of the three loci, which is not consistent with published regional allele frequencies and most likely reflects incomplete restriction digestion; those findings are therefore reported as hypothesis-generating. The lipid phenotype of this cohort, low HDL-C with raised LDL-C and ApoB-100 and no difference in total cholesterol or triglycerides, was independent of body mass index. Confirmation of the *Hin*dIII finding requires a larger, adequately powered cohort with sequencing-verified genotyping, direct measurement of LDL-C and remnant cholesterol, and adjustment for lipid-lowering therapy.

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