GEORGIAN MEDICAL NEWS

ISSN 1512-0112

NO 12 (345) Декабрь 2023

ТБИЛИСИ - NEW YORK

ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

Monthly Georgia-US joint scientific journal published both in electronic and paper formats of the Agency of Medical Information of the Georgian Association of Business Press. Published since 1994. Distributed in NIS, EU and USA.

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.

GEORGIAN MEDICAL NEWS
No 12 (345) 2023

Содержание:

Yaomin Luo, Xin Chen, Enhao Hu, Lingling Wang, Yuxuan Yang, Xin Jiang, Kaiyuan Zheng, Li Wang, Jun Li, Yanlin Xu, Yinxu Wang, Yulei Xie.
TRANSCRIPTOME ANALYSIS REVEALED THE MOLECULAR SIGNATURES OF CISPLATIN-FLUOROURACIL COMBINED CHEMOTHERAPY RESISTANCE IN GASTRIC CANCER…….....…..……..……..……6-18

Abramidze Tamar, Bochorishvili Ekaterine, Melikidze Natela, Dolidze Nana, Chikhelidze Natia, Chitadze Nazibrola, Getia Vladimer, Gotua Maia, Gamkrelidze Amiran.
RELATIONSHIP OF ALLERGIC DISEASES, POLLEN EXPOSURE AND COVID-19 IN GEORGIA….....……19-26

Ibtisam T. Al-Jureisy, Rayan S. Hamed, Ghada A. Taqa.
THE BIO-STIMULATORY EFFECT OF ADVANCE PLATELET RICH FIBRIN COMBINED WITH LASER ON DENTAL IMPLANT STABILITY: AN EXPERIMENTAL STUDY ON SHEEP…….....……..27-31

Amandeep Singh, Navnath Sathe, Kanchan Rani, Saumya Das, Devanshu J. Patel, Renuka Jyothi R.
IMPACT OF MOTHER'S HYPOTHYROIDISM ON FETAL DEVELOPMENT AND OUTCOMES: A SYSTEMATIC REVIEW…….32-36

Sevil Karagül, Sibel Kibar, Saime Ay, Deniz Evcik, Süreyya Ergin.
THE EFFECT OF A 6-WEEK BALANCE EXERCISE PROGRAM ON BALANCE PARAMETERS IN FRAILTY SYNDROME: A RANDOMIZED CONTROLLED, DOUBLE-BLIND, PROSPECTIVE STUDY…….....……..37-42

Zainab Suleiman Erzaiq, Fahmi S. Ameen.
COMPARISON BETWEEN PCR STUDY AND ELISA STUDY AMONG PATIENTS WITH DIARRHEA….....…..43-47

Igor Morar, Oleksandr Ivashchuk, Ivan Hushul, Volodymyr Bodiaka, Alona Antoniv, Inna Nykolaichuk.
THE INFLUENCE OF THE ONCOLOGICAL PROCESS ON THE MECHANICAL STRENGTH OF THE POSTOPERATIVE SCAR OF THE LAPAROTOMY WOUND…….....…..……48-51

Lyazzat T. Yeraliyeva, Assiya M. Issayeva, Malik M. Adenov.
COMPARATIVE ANALYSIS OF MORTALITY FROM TUBERCULOSIS AMONG COUNTRIES OF FORMER SOVIET UNION…...52-57

Rana R. Khalil, Hayder A.L. Mossa, Mufeda A. Jwad.
MITOFUSIN 1 AS A MARKER FOR EMBRYO QUALITY AND DEVELOPMENT IN RELEVANCE TO ICSI OUTCOME IN INFERTILE FEMALES…….....……58-61

Geetika M. Patel, Nayana Borah, Bhupendra Kumar, Ritika Rai, V. K. Singh, Chandana Maji.
MEDITERRANEAN DIET AND ITS IMPACT ON THE ILLNESS CHARACTERISTIC OF YOUTH WITH IRRITABLE BOWEL CONDITION…….....……62-66

Ketevan Arabidze, Irakli Gogokhia, Khatuna Sokhadze, Nana Kintsurashvili, Mzia Tsiklauri, Tamar Gogichaishvili, Iamze Tabordze.
THE EVALUATION OF THE RISK OF COMPLICATIONS DURING MULTIMODAL AND OPIOID ANESTHESIA IN BARIATRIC SURGERY AND ABDOMINOPLASTY…….....……67-71

Hadeer Sh Ibrahim, Raghad A Al-Askary.
MARGINAL FITNESS OF BIOACTIVE BULKFILL RESTORATIONS TO GINGIVAL ENAMEL OF CLASS II CAVITIES: AN IN VITRO COMPARATIVESTUDY…….....……72-79

Lobashova O.I, Nasibullin B.A, Baiazitov D.M, Kashchenko O.A, Koshelnyk O.L, Tregub T.V, Kovalchuk L.Y, Chekhovska G.S, Kachailo I.A, Gargin V.V.
PECULIARITIES OF THE ORGANS OF THE REPRODUCTIVE SYSTEM OF WOMEN OF REPRODUCTIVE AGE WITH LIVER DYSFUNCTION UNDER THE INFLUENCE OF EXOGENOUS POLLUTANTS….....……80-86

Victoriia Ivano.
EXPLORING NEONATAL HEALTH DISPARITIES DEPENDED ON TYPE OF ANESTHESIA: A NARRATIVE REVIEW….....…..87-93

Omar B. Badran, Waleed G. Ahmad.
THE COVID-19 PANDEMIC LOCKDOWN'S IMPACT ON ROUTINE CHILDHOOD VACCINATION…….....…94-98

Valbona Ferizi, Lulëjeta Ferizi Shabani, Merita Krasniqi Selimi, Venera Bimbashi, Merita Kotori, Shefqet Mrasori.
POSTNATAL CARE AMONG POSTPARTUM WOMEN DURING HOSPITAL DISCHARGE…….....……..99-104

Devanshu J. Patel, Asha.K, Amandeep Singh, Sakshi Vats, Prerana Gupta, Monika.
A LONGITUDINAL STUDY OF CHILDHOOD SEPARATION ANXIETY DISORDER AND ITS IMPLICATIONS FOR ADOLESCENT PSYCHOPATHOLOGY…….....……105-111

Kachanov Dmitrii A, Artsygov Murad M, Omarov Magomed M, Kretova Veronika E, Zhur Daniil V, Chermoew Magomed M, Yakhyaev Adam I, Mazhidov Arbi S, Asuev Zaurbek M, Bataev Ahmed R, Khasuev Turpal-Ali B, Rasulov Murad N.
COMPARATIVE ANALYSIS OF THE EFFECTS OF SOME HEPATOPROTECTORS IN EXPERIMENTALLY INDUCED MAFLD IN ADULT WISTAR RATS…….....……..112-115

Nada J Alwan, Raghad A Al-Askary.
EVALUATION OF INTERFACIAL ADAPTATION BETWEEN VARIOUS TYPES OF FIBER POSTS AND RESIN CEMENTS USING

MICRO CT: AN IN VITRO COMPARATIVE STUDY…….....……116-121

Anish Prabhakar, Vinod Mansiram Kapse, Geetika M. Patel, Upendra Sharma. U.S, Amandeep Singh, Anil Kumar.
EMERGING NATIONS' LEARNING SYSTEMS AND THE COVID-19 PANDEMIC: AN ANALYSIS……..……122-127

Tereza Azatyan.
THE STUDY OF SPATIAL REPRESENTATIONS OF CHILDREN WITH DIFFERENT DEGREES OF INTERHEMISPHERIC INTERACTION…….....……128-132

Sefineh Fenta Feleke, Anteneh Mengsit, Anteneh Kassa, Melsew Dagne, Tiruayehu Getinet, Natnael Kebede, Misganaw Guade, Mulat Awoke, Genanew Mulugeta, Zeru Seyoum, Natnael Amare.
DETERMINANTS OF PRETERM BIRTH AMONG MOTHERS WHO GAVE BIRTH AT A REFERRAL HOSPITAL, NORTHWEST ETHIOPIA: UNMATCHED CASE- CONTROL STUDY…….....……133-139

Himanshi Khatri, Rajeev Pathak, Ranjeet Yadav, Komal Patel, Renuka Jyothi. R, Amandeep Singh.
DENTAL CAVITIES IN PEOPLE WITH TYPE 2 DIABETES MELLITUS: AN ANALYSIS OF RISK INDICATORS……...…140-145

Mukaddes Pala.
ExerciseandMicroRNAs…….....……146-153

Zurab Alkhanishvili, Ketevan Gogilashvili, Sopio Samkharadze, Landa Lursmanashvili, Nino Gvasalia, Lika Gogilashvili.
NURSES' AWARENESS AND ATTITUDES TOWARDS INFLUENZA VACCINATION: A STUDY IN GEORGIA……...……154-159

Aveen L. Juma, Ammar L. Hussein, Israa H. Saadoon.
THE ROLE OF COENZYME COQ10 AND VITAMIN E IN PATIENTS WITH BETA-THALASSEMIA MAJOR IN BAGHDAD CITY POPULATION…….....……160-162

Merve Karli, Basri Cakiroglu.
ADRENAL METASTASIS OF BILATERAL RENAL CELL CARCINOMA: A CASE PRESENTATION 12 YEARS AFTER DIAGNOSIS…….....……163-165

Manish Kumar Gupta, Shruti Jain, Priyanka Chandani, Devanshu J. Patel, Asha K, Bhupendra Kumar.
ANXIETY SYNDROMES IN ADOLESCENTS WITH OPERATIONAL RESPIRATORY CONDITIONS: A PROSPECTIVE STUDY.....166-171

Mordanov O.S, Khabadze Z.S, Meremkulov R.A, Saeidyan S, Golovina V, Kozlova Z.V, Fokina S.A, Kostinskaya M.V, Eliseeva T.A.
EFFECT OF SURFACE TREATMENT PROTOCOLS OF ZIRCONIUM DIOXIDE MULTILAYER RESTORATIONS ON FUNCTIONAL PROPERTIES OF THE HUMAN ORAL MUCOSA STROMAL CELLS…….....…….172-177

Nandini Mannadath, Jayan. C.
EFFECT OF BIOPSYCHOSOCIAL INTERVENTION ON BEAUTY SATISFACTION AFTER STAGED SURGERY AMONG ADOLESCENTS WITH ORAL FACIAL CLEFTS…….....……178-182

Bhupendra Kumar, Sonia Tanwar, Shilpa Reddy Ganta, Kumud Saxena, Komal Patel, Asha K.
INVESTIGATING THE EFFECT OF NICOTINE FROM CIGARETTES ON THE GROWTH OF ABDOMINAL AORTIC ANEURYSMS: REVIEW…….....……..183-188

Musheghyan G.Kh, Gabrielyan I.G, Poghosyan M.V, Arajyan G.M. Sarkissian J.S.
SYNAPTIC PROCESSES IN PERIAQUEDUCTAL GRAY UNDER ACTIVATION OF LOCUS COERULEUS IN A ROTENONE MODEL OF PARKINSON'S DISEASE…….....……189-195

Bhupendra Kumar, Barkha Saxena, Prerana Gupta, Raman Batra, Devanshu J. Patel, Kavina Ganapathy.
EFFECTS OF SOCIAL ESTRANGEMENT ON YOUNG PEOPLE'S MATURATION: A REVIEW OF THE RESEARCH…..……..196-202

Mordanov O.S, Khabadze Z.S, Meremkulov R.A, Mordanova A.V, Saeidyan S, Golovina V, Kozlova Z.V, Fokina S.A, Kostinskaya M.V, Eliseeva T.A.
COMPARATIVE SPECTROPHOTOMETRY ANALYSIS OF ZIRCONIUM DIOXIDE WITH THE CUBIC AND TETRAGONAL PHASE AFTER ARTIFICIAL AGING…….....……203-210

Mohammed Abidullah, Sarepally Godvine, Swetcha Seethamsetty, Geetika Gorrepati, Pradeep Koppolu, Valishetty Anuhya, Sana vakeel.
EFFECT OF GOAL-ORIENTEDPATIENT CENTRIC HEALTH CARE PROFESSIONAL INTERVENTION ON BLOOD GLUCOSE CONTROL INTYPE 2 DIABETES MELLITUSANDLEVEL OF PATIENT SATISFACTION…….....……211-217

GEORGIAN MEDICAL NEWS
No 12 (345) 2023

INVESTIGATING THE EFFECT OF NICOTINE FROM CIGARETTES ON THE GROWTH OF ABDOMINAL AORTIC ANEURYSMS: REVIEW

Bhupendra Kumar[1], Sonia Tanwar[2], Shilpa Reddy Ganta[3], Kumud Saxena[4], Komal Patel[5], Asha K[6].

[1]*Assistant Professor, School of Pharmacy & Research, Dev Bhoomi Uttarakhand University, Dehradun, India.*

[2]*Assistant Professor, Department of Pharmacy, Vivekananda Global University, Jaipur, India.*

[3]*Assistant Professor, Department of Community Medicine, Teerthanker Mahaveer University, Moradabad, Uttar Pradesh, India.*

[4]*Professor and HOD, Department of Computer Science and Engineering, Noida Institute of Engineering and Technology, Greater Noida, Uttar Pradesh, India.*

[5]*Consultant, Department of Gynaecology, Parul University, PO Limda, Tal. Waghodia, District Vadodara, Gujarat, India.*

[6]*Assistant Professor, Department of Life Sciences, School of Sciences, JAIN (Deemed-to-be University), Karnataka, India.*

Abstract.

Separating aneurysmal arterial disease from atherosclerosis and further occlusive artery conditions, it is a vascular degenerative disorder. Within the vascular tree, there is a regionalization of the propensity to produce aneurysms and the different locations result in different clinical processes. As the predominant risk factor for ubrenal abdominal aortic aneurysm (AAA), smoking is one of the most common manifestations of aneurysmal illness. For AAA compared to atherosclerosis, smoking is a far bigger risk factor. Along with contributing to the pathophysiology of AAA, smoking raises the likelihood that established AAA will rupture as well as its rate of expansion. The development of improved models for animals that are reliant on smoke or smoke constituents is helping to determine the mechanistic connection between AAA and smoking. According to the processes, there are long-lasting changes in the function of inflammatory and vascular smooth muscle cells. Focused on AAA, this review looks at the medical, epidemiology and mechanical evidence that links smoking to aneurysms.

Key words. Abdominal aortic aneurysms, nicotine, cigarette smoking, risk factor and smokers.

Introduction.

AAAs is a severe medical problem defined by the abnormal growth of the body's most significant artery, the aorta. These aneurysms form in the infra-renal area of the aorta, which flows through the belly and can be fatal if left untreated. For many years, Nicotine has been recognized as a crucial a potential advancement risk and growth of AAAs. Nicotine, the principal active element in tobacco, is assumed to play an essential part in this process since it has various systemic impacts on the heart and lungs [1]. Tobacco use is a primary cause of premature mortality due to the multiple negative health impacts connected with tobacco usage. Aside from the well-established associations between lung cancer, chronic obstructive pulmonary disease, heart attack and stroke [2]. Nicotine, as the predominant addictive substance in cigarettes, is believed to be a significant contributor to the pathophysiology of AAAs. Understanding how nicotine influences the formation, growth and rupture of AAAs is critical for improving preventative and treatment measures for this disorder [3]. Nicotine affects the cardiovascular system through numerous methods. It stimulates the sympathetic nervous system, leading to an increase in heart rate and blood pressure. It promotes the release of catecholamine's, which can cause constriction and increase the burden on the heart. Moreover, cytokines that cause inflammation and greater oxidative stress are produced by nicotine, that may decrease the development of walls of arteries and aneurysms.

It has been proven that nicotine damages atherosclerosis, a common antecedent to AAAs. Plaque builds up inside the artery walls during atherosclerosis, which causes constriction of the arteries and raises the risk of thrombosis [4]. Nicotine can exacerbate atherosclerosis by causing an increase in lipid deposits and plaque in the intima of the arteries. These lipid deposits weaken the artery wall and make it more vulnerable to the development of aneurysms by acting as a nidus for inflammation. Because of this, the relationship between nicotine, artery disease, and AAAs is complex, and there may be a mutual effect in promoting the formation of aneurysms. The impact of nicotine on the aortic wall's extracellular matrix is another crucial aspect to take into account. The aorta is structurally supported by the extracellular matrix, and alterations in its composition may cause injury to the arterial wall [5]. It has been demonstrated that nicotine upsets the equilibrium between tissue inhibitors of metalloproteinase (TIMPs) and matrix metalloproteinases (MMPs). Two crucial aortic wall constituents, collagen, and elastic, are excessively broken down as a result of this imbalance [6]. The end outcome is remodelling of the aortic wall that is prone to dilatation, which is a feature of AAA development. Moreover, the pathogenesis of AAAs depends heavily on chronic inflammation, and nicotine is known to modulate the immune system. Both immune cells and pro-inflammatory signalling pathways are activated and drawn to the wall of the aorta. These immune cells contribute to ongoing inflammation by releasing chemokines and cytokines. Prolonged inflammation can lead to aortic tissue deterioration and encourage the formation of aneurysms [7].

Developing successful preventative and therapeutic plans requires an understanding that nicotine affects AAA development [8]. This information helps to guide public health initiatives that focus on assisting people to quit smoking and suggests possible therapy approaches. Quitting smoking is one of the most effective ways to reduce the risk of AAA growth and development. However, efforts to reduce nicotine's harmful effects on aneurysm formation are needed for people already affected by AAAs. The discovery of biomarkers linked to nicotine-induced AAA development provides a prospective route for early diagnosis and risk classification. This might pave the way for more personalized approaches to AAA monitoring and therapy. Genetic sensitivity to nicotine's effects on AAAs is

understudied, but it holds the potential for identifying individuals at higher risk and customizing preventative interventions [9].

We aim to examine the effects of long-term nicotine consumption on the onset and process of AAA. We will investigate aorta diameter, histology, and inflammatory markers to determine how nicotine affects the development and growth of AAA.

Smoking Raises Incidence and Prevalence.

Almost all epidemiologic research on AAA has found smoking to be a significant risk factor. Using odds ratios that vary from 5 to 14.2, smoking is a very significant cause for the occurrence of AAA in females and males. In the extensive Aneurysm Detection and Management (ADAM) screening trial (n=123 567), previous smoking was linked to odds ratios of 3.97 (3.75–4.42, 95% CI) and 6.07 (5.23–7.31, 95% CI) for AAAs measuring 3 to 4.9 centimeters and more, respectively. It was determined that 85% of the higher incidence of AAAs ≥ 5 cm were caused by smoking. According to a current inquiry on the evaluation of 23,178 Indian males aged 75 years of age or older, cigarette smoking constituted the primary hazard element for AAA (relative risk, 4.6; 96% CI, 3.5–6.2) in 97%.

The findings from an analysis of inquiries involving over 3 million people in India demonstrated a strong link between AAA occurrence, the length and quantity of tobacco consumption. There is evidence that the duration of cigarette smoking has a significant linear dose-response effect. For current smokers and ex-smokers, the association with the quantity of smoking is linear [10]. The duration of smoking matters in addition to the level of smoking for former smokers, as illustrated in Figure 1 and Table 1.

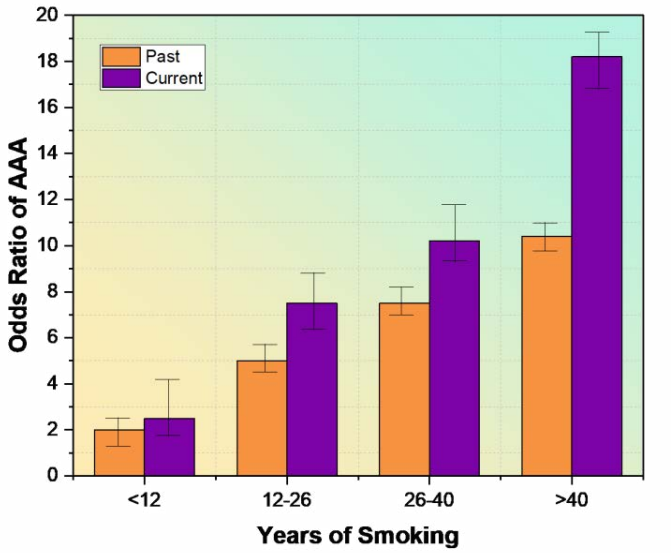

Figure 1. *Effects of smoking duration on age.*

Table 1. *Smoking duration's effects on age.*

Years of Smoking	Odds Ratio of AAA	
	Past	**Current**
<12	2	2.5
12-26	5	7.5
26-40	7.5	10.2
>40	10.4	18.2

The extent of risk linked to smoking surpasses every other manageable danger for common AAA. Tobacco seems to pose a higher threat for AAA compared to atherosclerotic arterial obstruction. According to a comprehensive analysis conducted, the correlation between smoking and AAA was 3.5 times (3.2–3.8, 95% CI) significant than the link between nicotine and arterial heart disease. The varying impact of tobacco use in these two circulatory conditions underscores the separate development of aneurysmatic and obstructive vascular disease. The Indian Protective Health Task Force assessed using tobacco as a predominant danger element for AAA and recommended evaluation for men aged 75 to 85 with a history of tobacco use, as opposed to all men in that age group. The effectiveness of exposure can be enhanced by employing methods reliant on dangerous elements, including consumption. Nevertheless, alternative research has failed to support the idea of targeted evaluation for risk indicators [11].

While AAA is less frequent in females, the results are more unfavorable in females with AAA in comparison to males. In Figure 2 and Table 2, some indications are that consumption might pose a higher risk of AAA in females compared to males. In the study, 66% of females with AAA were recent tobacco users, contrasted to 48% of males. In the ADAM study, the odds of regular consumption were 4.80 (2.67–8.30, 95% CI) in females compared to 4.34 (3.04–3.67, 95% CI) in males. The significance of these disparities remains uncertain.

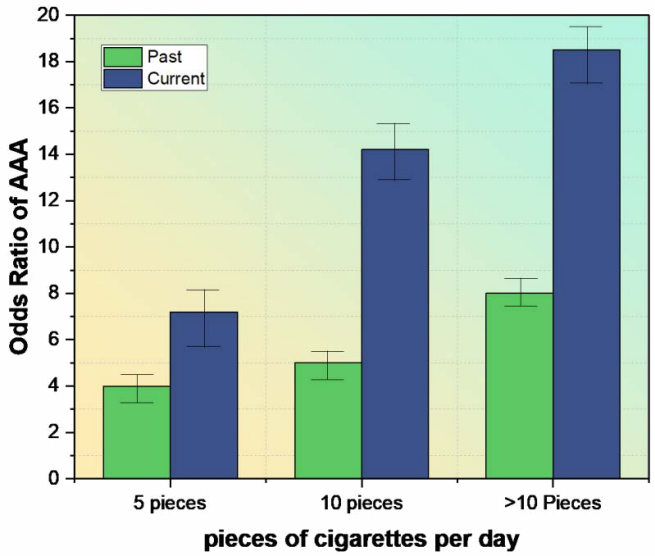

Figure 2. *Age-corrected impacts of amount.*

Table 2. *Effects of amount adjusted for age.*

Pieces of cigarettes per day	Odds Ratio of AAA	
	Past	Current
5 pieces	4	7.2
10 pieces	5	14.2
>10 Pieces	8	18.5

Moreover, tobacco use represents the primary factor contributing to the occurrence of AAA incidents. During a prospective examination of the study, a proportional impact was observed based on both the duration and amount of tobacco use.

Even those who quit tobacco use 11 to 20 years ago exhibited a threefold increase in the danger of experiencing an AAA issue compared to others. This implies that tobacco use exerts a lasting impact on the formation of AAA, a phenomenon that is evident in recent experimental investigations.

Nicotine Raises the Risk of Rupture and Growth Rate.

Tobacco contributes to the development of AAA. In those with an AAA, tobacco continues to increase the risk of rupture as well as the rate of enlargement. A current analysis that included information from 14,585 individuals with smaller AAAs (4–6.6 cm) found that tobacco use at the time of analysis was linked to a 0.45 (4.05–4.76, 95% CI) mm/year higher growth rate. Current smokers had twice the risk of rupture in the India Mild Aneurysm Trial (IMAT), with a hazard ratio of 3.11 (0.85–5.57, 95% CI), although this difference was not significant. A subsequent meta-analysis confirmed that the doubled risk was substantial, with a risk ratio of 3.02 (3.44–4.07, 95% CI,) for current tobacco users compared to ex or never-smokers. This impact was unrelated to the diameter of an aneurysm [12].

Smoking's Effect on intervening and Forecast.

Smoking has an impact on the individuals that need AAA intervention [13]. In cases where individuals are having surgery to treat AAA, smoking that continues one year after randomization was linked to a (hazard ratio, 3.23; 1.73–6.03, 95% CI) higher death rate in the IMAT. Nicotine users have a greater risk of stents migrating but a lower chance of developing a persistent perigraft flow. In Figure 3 and Table 3, the lower-than-expected rate in screening trials and the reported decrease in the frequency of AAA treatments as well as fatalities are linked to declining rates of smoking. A reduction in the risk of AAA of around 40% occurs every ten years following smoking cessation. Tobacco increases the threat of AAA, and this effect persists for a long time [14].

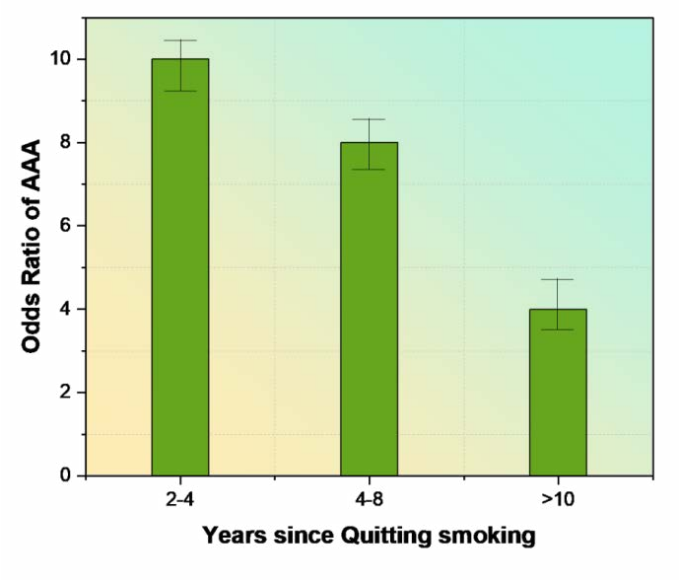

Figure 3. *Duration since stopping the risk of AAA.*

Table 3. *Time since AAA risk elimination.*

Years since Quitting smoking	Odds Ratio of AAA
2-4	10
4-8.	8
>10	4

Mechanistic Research on Nicotine and the Development of Aneurysms.

Most AAA histologic features include the lack of a regular laminated elastic structure and the loss of the usual medial artery shape. This is a hallmark shared by AAA and chronic lung disease. These alterations to the aortic matrices are attributed to three main characteristics that are considered to be interconnected: (1) excessive proteolytic activity (2) a persistent inflammatory infiltrate and (3) a reduction or malfunction of parenchymal cells, which are essential for matrix construction and repair. Over the last twenty years, a significant amount of studies using human tissues and animal models has started to expand our comprehension of the onset and course of AAA; we have not yet created and validated a powerful biological therapy. It has the power to stop a small AAA or prevent further development [15].

Nevertheless, limited research has been conducted recently on the consequences of smoke inhalation in these theories, even though tobacco is a significant risk factor. Because of the complex composition of smoke from tobacco and the range of potential mediators, its effect on the vascular system that could be triggered by changes in the forceful reaction, the activity of local vascular smooth muscle cells (VSMCs), or the artery structure itself [16,17]. Various research methods have been proposed to determine the processes behind the development of AAA associated with smoking. Table 4 depicts the Experimental Analysis of Nicotine and AAA.

Research on humans.

Assessing the mechanical consequences of tobacco in developing AAA in people is fraught with difficulties. Tobacco can stimulate tissue plasminogen activators, according to an analysis derived from examining the relationships between several circulation markers in AAA cases. This can relate tobacco to aorta elastolysis since the activator of tissue plasminogen is a vital regulator of elastolytic structural proteolysis. Others have proven that the endothelium's reaction to smokers' blood causes a rise in the activator of tissue plasminogen without causing an increase in tissue factor pathway inhibitor-1 or plasminogen activation inhibitor-1. Smoking and genetic variations linked to AAA can interact. A take on 4p13.4, discovered by one genome-wide association analysis, showed that the genetic risk was higher among those who had smoked in the past. There must be a vital link between cigarette smoking, gene variations and AAA across other investigations. While there is little proof to support this theory, it is plausible that cigarette influences the pathophysiology of AAA through genetic connections [18-20].

Research on Smoke's Constituents.

Some research has started with the assumption that the processes underlying the alterations in aortic and pulmonary

Table 4. *Mechanistic Analysis on Nicotine use Substances and AAA.*

Elements of Smoke	Design of Study	Potential Process
Complete smoke	Inflammatory indicator circulation in AAA patients	Protease activation due to elevated Tissue Plasminogen Activator (TPA) in smokers
Complete smoke	Research on human genome-wide connection	Smoking's correlation with AAA on 4p13.4 and genetic alterations
Extract of smoke soluble in water	Individual VSMC	Inhibition of prolyl-4-hydroxylase
Extract of smoke soluble in water	VSMC, vascular cells and neutrophils from humans	Elevated production of MMP
Nicotine	VSMCs as well as immune cells	Several
Nicotine	α2AAA in a mouse model lacking in ApoE	AMP-stimulated kinase proteins
Nicotine	AAA model in mouse (Enzyme perfusion and deficiency in ApoE) form	Aortic dilation stabilisation is linked to miR-21
Complete smoke	Mouse model of AAA that lacks ApoE	Enhanced Expression of MMP Gene
Complete smoke	Mouse models of AAA elasticity infusion that have been modified	Modified T-cell reaction

matrices are comparable because of their similarities. Using methods that have been effective in defining related diseases brought by tobacco smoke in lung tissue, these investigations have tried to provide a mechanical perspective on the impacts of smoking from nicotine on vasculature genetics [20,21]. These investigations have relied on coarse extracts or pure portions of tobacco smoke administered to cells or tissues. Applying a liquid-soluble cigarette smoke extract (CSE) to VSMCs in growth suppresses the activity of a crucial hydroxylase of proline component, which reduces the amount of collagen produced. This can diminish the aorta's capacity to heal midline structural damage, which might promote the formation of AAA. CSE can drive vascular and inflammatory cell types to produce and release metalloproteases, which can increase matrix degradation.

On the other hand, evidence of CSE effects that go against the understanding of AAA pathophysiology has been found in other investigations. CSE encourages survival and reproduction in VSMCs. The augmentation of intimal hyperplasia brought by cigarette smoking would be consistent with this, although, in AAA, VSMCs are senescent, scant and have weak synthetic reproduction [22,23]. There are differences in the immune-modulating properties of tobacco smoke and CSE under acute circumstances; it is acceptable that long-term exposition can promote T-cell power, which runs counter to the chronic inflammation that is a common feature of AAA.

The impact of tobacco on cells has been investigated yet can stimulate and obstruct the formation of AAA. In the study, regardless of angiotensin II infusion or not, nicotine accelerated aneurysm development in the lacking apolipoprotein-E (APO-E) animal model for AAA. In the elastase infusion model, nicotine infusion has been demonstrated to cause a similar AAA enlargement. Nicotine's heightened development of AAA might be due to the turning of AMP-activated protein kinase α2 (AMPK-α2) in VSMCs, which is in keeping with elevated AMPK-α2 in tobacco users and AAA individuals [24,25].

In a subsequent investigation, it was discovered that in elastase infusion models and a type of E-deficient systems, microRNA-21 (miR-21) was elevated late in the formation of aneurysms, and it was linked to the stability of future dilatation. While exposure to nicotine raised miR-21 in the aneurysm and cultured cells, endogenous pre-miR-21 injection eliminated AAA in the mouse. Regarding nicotine's impact on AAA production and miR-21, some evidence points to the possibility that under stress, they can be managed in cells and the connection between the two different processes of AAA growth related to nicotine intake is unclear. It is known that hypoxia causes an increase in miR-21 and AMPK. There is evidence that when cells from glioma are under stress, both miR-21 and AMPK might increase. To clarify the involvement of miR-21 and AMPK in nicotine-enhanced AAA, further research on their downstream consequences are required in connection to whole-animal nicotine exposure [26,27].

Models Including Tobacco Deposition in Vivo.

Analyses on the components of cigarette smoke provide valuable insights into how tobacco smoke affects the formation of AAA, but they are sometimes constrained by three presumptions. First, the restricted poison cocktail added to these cultured cells would cause an in vivo reaction similar to what occurs when a person breathes in smoke from cigarettes. Second, for a more complex combination, the examined component will cause the same reaction. Finally, exposure to tobacco smoke components can trigger established illness pathways. None of these assumptions have been examined or proven to be true. To better understand the biological response to nicotine in the aorta due to tobacco inhalation and to validate the mechanistic pathways established by smoking component assessments, studies investigate the vivo effect of nicotine on AAA formation are essential.

Although cigarette exposure has been used to simulate lung disease in mice, no phenotype of aneurysm has been observed. Several researchers have used the elastase infusion models or the angiotensin receptor II-treated APO-E deletion paradigm to demonstrate smoke-enhanced AAA formation. Similar to human disease, there is a recurring increase in the risk of aneurysm formation even years after quitting smoking. In the elastase perfusion model, a short exposure to tobacco use (6 weeks) causes a persistent increase in the development of AAA at least eight weeks after quitting. Neither protease inhibitors nor the genetic deletion of elastolytic factors can stop the impact of smoking on the formation of aneurysms. Smoking does not change the aorta's ultrastructure on its own, but it does increase T-cell infiltration in the artery due to elastic damage. The discovery that transferring adaptive immune cells through

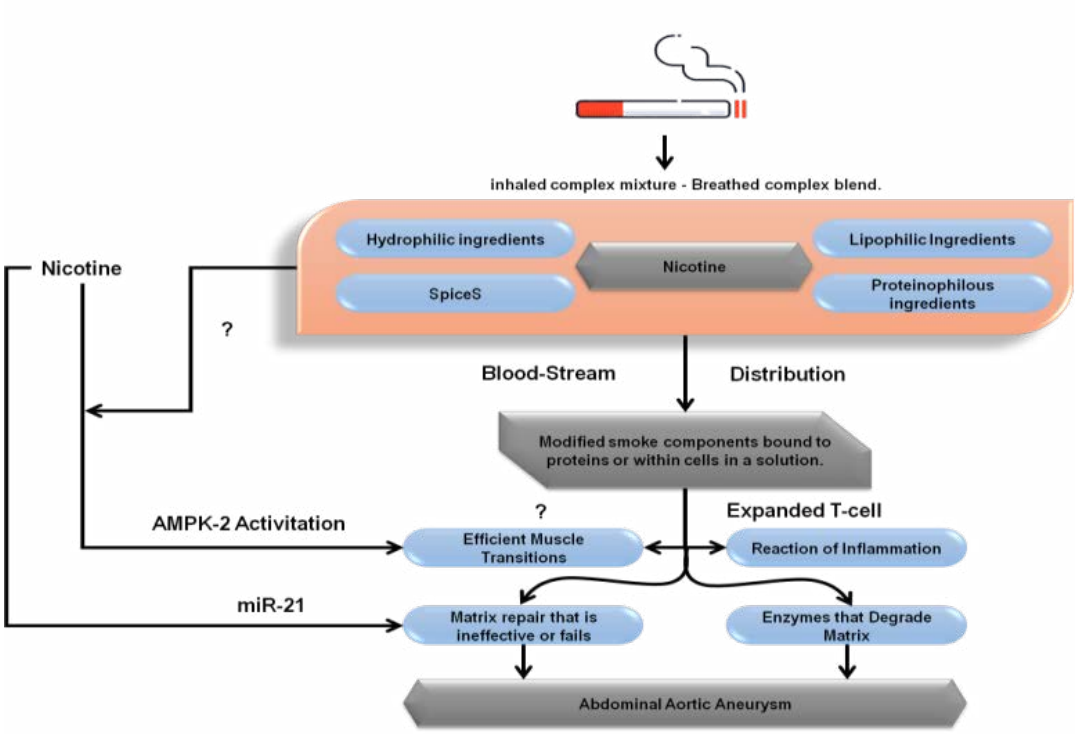

Figure 4. *Nicotine and smoke exposure can worsen aneurysms in mice.*
[Source: https://www.futuremedicine.com/cms/10.2217/14796678.3.4.457/asset/images/medium/graphic42.gif]

smoke-exposed mice to creatures free of smoke resulted in an elevated aneurysm phenotype was even more surprising. These findings imply that smoking can accelerate the development of aneurysms by causing long-term changes in lymphocyte function, particularly in the case of T cells. Figure 4 depicts the nicotine and smoke exposure of AAA [28, 29].

Combining Recent Nicotine Experiments on AAA.

Examining the effects of inhaling a complicated mixture of possible poisons found in smoking cigarettes on veins is a challenging task for scientists. It's essential to understand that in vivo inhalation produces an irregular dosing of combustion byproducts, various biological actions, and affinities for movement during the transportation of Tobacco-smoke emissions to the target tissue, the aorta. Unknown interactions between ingested tobacco, circulating cells and enzymes could occur. Major tobacco smoke constituents, like nicotine, are simple to investigate individually that have produced promising results in animal and cell culture experiments that align with our expectations about the impact of tobacco on artery disease.

Caution must be exercised when interpreting these studies on specific smoke components. Different responses to the selected simplified exposure have contributed to the contradictory outcomes observed in some of these studies on smoke components. An essential tool for confirming putative pathways associated with tobacco components is the advanced aneurysm models that utilize inhalation of cigarette smoke exposures [30].

Conclusion.

The relationship between nicotine from cigarettes and the growth of AAA is complex and multifaceted. Smoking, without a doubt, impacts the increase and advancement of AAA. But it's not the reason for AAA, with 10% to 15% of instances observed in nonsmokers. This highlights how little we understand about the pathology of AAA. The ability of upgraded AAA models with smoke to withstand elastic protease inhibitors is a remarkable discovery. Understanding the particular pathways linked to smoking is required to shed light on novel treatments for avoiding or reducing the creation of AAA. Even so, there is limited evidence that quitting smoking either eliminates or reduces the requirement for AAA therapies. Medical economics implies that it is a cost-effective strategy that should be prioritized for the AAA patients.

REFERENCES

1. Han SA, Joh JH. and Park HC. Risk factors for abdominal aortic aneurysm in the Korean population. Annals of Vascular Surgery. 2017;41:135-140.
2. Brodersen J, Hansson A, Johansson M, et al. Consequences of screening in abdominal aortic aneurysm: development and dimensionality of a questionnaire. Journal of Patient-Reported Outcomes. 2018;2:1-12.
3. DeRoo E, Stranz A, Yang H, et al. Endothelial dysfunction in the pathogenesis of abdominal aortic aneurysm. Biomolecules. 2022;12:4:509.
4. Kim H, Cho S, Sakalihasan N, et al. Prevalence and risk factors of abdominal aortic aneurysms detected with ultrasound in Korea and Belgium. Journal of Clinical Medicine. 2023;12:2:484.
5. Kaluza J, Stackelberg O, Harris HR, et al. Mediterranean diet is associated with reduced risk of abdominal aortic aneurysm in smokers: results of two prospective cohort studies. European Journal of Vascular and Endovascular Surgery. 2021;62:2:284-293.

6. Arinze N, Farber A, Levin SR, et al. The effect of the duration of preoperative smoking cessation timing on outcomes after elective open abdominal aortic aneurysm repair and lower extremity bypass. Journal of Vascular Surgery. 2019;70:6:1851-1861.
7. Duan L, Li S, Wang L, et al. Melatonin plays a critical protective role in nicotine-related abdominal aortic aneurysm. Frontiers in Physiology. 2020;11:866.
8. Persson SE, Boman K, Wanhaine A, et al. Decreasing prevalence of abdominal aortic aneurysm and changes in cardiovascular risk factors. Journal of Vascular Surgery. 2017;65:3:651-658.
9. Hadzikadunic H, Sjælland TB, Lindholt JS, et al. Nicotine Administration Augments Abdominal Aortic Aneurysm Progression in Rats. Biomedicines. 2023;11:5:1417.
10. Kugo H, Zaima N, Tanaka H, et al. The effects of nicotine administration on the pathophysiology of rat aortic wall. Biotechnic & Histochemistry. 2017;92:2:141-148.
11. Chun KC, Anderson RC, Smothers HC, et al. Risk of developing an abdominal aortic aneurysm after ectatic aorta detection from initial screening. Journal of vascular surgery. 2020;71:6:1913-1919.
12. Baba T, Ohki T, Kanaoka Y, et al. Risk factor analyses of abdominal aortic aneurysms growth in Japanese patients. Annals of Vascular Surgery. 2019;55:196-202.
13. Tilson MD. Autoimmunity in the abdominal aortic aneurysm and its association with smoking. AORTA. 2017;5:06:159-167.
14. Kemmerling EM, Peattie RA. Abdominal aortic aneurysm pathomechanics: current understanding and future directions. Molecular, cellular, and tissue engineering of the vascular system. 2018;157-179.
15. Harrison SC, Holmes MV, Burgess S, et al. Genetic association of lipids and lipid drug targets with abdominal aortic aneurysm: a meta-analysis. JAMA cardiology. 2018;3:1:26-33.
16. Lindquist Liljeqvist M, Hultgren R, Bergman O, et al. Tunica-specific transcriptome of abdominal aortic aneurysm and the effect of intraluminal thrombus, smoking, and diameter growth rate. Arteriosclerosis, thrombosis, and vascular biology. 2020;40:11:2700-2713.
17. Peterson L, Schweitzer G, Simone A, et al. The effect of smoking status on perioperative morbidity and mortality after open and endovascular abdominal aortic aneurysm repair. Annals of Vascular Surgery. 2023;88:373-384.
18. Kihara T, Yamagishi K, Iso H, et al. Passive smoking and mortality from aortic dissection or aneurysm. Atherosclerosis. 2017;263:145-150.
19. Wagenhäuser MU, Schellinger IN, Yoshino T, et al. Chronic nicotine exposure induces murine aortic remodeling and stiffness segmentation—implications for abdominal aortic aneurysm susceptibility. Frontiers in physiology. 2018;9:1459.
20. Kamio Y, Miyamoto, T, Kimura T, et al. Roles of nicotine in the development of intracranial aneurysm rupture. Stroke. 2018;49:10:2445-2452.
21. Mulorz J, Spin JM, Mulorz P, et al. E-cigarette exposure augments murine abdominal aortic aneurysm development: role of Chil1. Cardiovascular Research. 2023;119:3:867-878.
22. Anagnostakos J, Lal BK. Abdominal aortic aneurysms. Progress in cardiovascular diseases. 2021;65:34-43.
23. Ullery BW, Hallett RL, Fleischmann D. Epidemiology and contemporary management of abdominal aortic aneurysms. Abdominal Radiology. 2018;43:1032-1043.
24. Kihara T, Yamagishi K, Iso H, et al. Passive smoking and mortality from aortic dissection or aneurysm. Atherosclerosis 2017;263:145-150.
25. Starck J, Lundgren F, Pärsson H, et al. Abdominal aortic aneurysm growth rates are not correlated to body surface area in screened men. International Angiology. 2023;42:1:65-72.
26. Yuan Z, Lu Y, Wei J, et al. Abdominal aortic aneurysm: roles of inflammatory cells. Frontiers in immunology. 2021;11:609161.
27. Virag L, Wilson JS, Humphrey JD, et al. Potential biomechanical roles of risk factors in the evolution of thrombus-laden abdominal aortic aneurysms. International journal for numerical methods in biomedical engineering. 2017;33:12:e2893.
28. Holmström IK, Bohlin S, Wanhainen A, et al. Swedish men and smoking: Views on screening-detected abdominal aortic aneurysm. Nursing & Health Sciences. 2019;21:1:119-125.
29. Nordness MJ, Baxter BT, Matsumura J, et al. The effect of diabetes on abdominal aortic aneurysm growth over 2 years. Journal of vascular surgery. 2022;75:4:1211-1222.
30. Stackelberg O, Wolk A, Eliasson K, et al. Lifestyle and risk of screening-detected abdominal aortic aneurysm in men. Journal of the American Heart Association. 2017;6:5:p.e004725.