

# GEORGIAN MEDICAL NEWS

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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 ВИНТИ 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 ВИНТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებშიდან.

### WEBSITE

[www.geomednews.com](http://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](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. Корректур авторам не высылаются, 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 computer-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.nlm.nih.gov/bsd/uniform_requirements.html)  
[http://www.icmje.org/urm\\_full.pdf](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 საქურაღებოლ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დაიცვათ 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. რენტგენოგრაფიების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შედეგების 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 ფხიხლებში 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.

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## GENOTOXIC AND MOLECULAR STRESS EFFECTS OF DENTAL RESIN MONOMERS ON ORAL EPITHELIAL CELLS

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### Abstract.

**Background:** The dental materials based on resin substances release small amounts of their unreacted monomer components which result in continuous exposure of oral epithelial cells to these substances. The scientific community lacks sufficient knowledge about molecular reactions which occur before cells show toxic damage.

**Aim:** The research aims to determine how Bis-GMA and TEGDMA and UDMA and HEMA concentrations below cytotoxic levels affect oral epithelial cells through oxidative stress and DNA damage and changes in genes that control cell proliferation and apoptosis.

**Methods:** The MTT assay helped researchers determine the specific concentration ranges which produced sub-cytotoxic effects. The DCFH-DA assay measured the amount of ROS which cells produced inside their cells. The alkaline comet assay was used to evaluate DNA damage while qRT-PCR analyzed the relative mRNA levels of BAX, BCL-2, p53, c-Myc and Cyclin D1. **Results:** The tested monomers at low concentrations did not affect cell metabolic activity but they all caused an increase in intracellular ROS levels. The two monomers Bis-GMA and TEGDMA generated the most significant increase in ROS levels among all tested compounds. The results showed oxidative imbalance because comet parameters showed increased DNA strand breakage and qRT-PCR results showed apoptosis-related gene expression with BAX and p53 showing elevated expression while BCL-2 levels decreased and c-Myc and Cyclin D1 showed no significant changes.

**Conclusion:** The research shows that oral epithelial cells experience oxidative and genotoxic stress when they encounter standard resin monomers at levels which do not result in major cell death. The research results confirm that additional studies need to investigate how biological responses develop from first molecular changes which occur when patients follow standard medical protocols.

**Key words.** Dental resin monomers, oral epithelial cells, oxidative stress, DNA damage, gene expression, biocompatibility.

### Introduction.

The oral environment needs dental resin-based materials to perform both functional restoration and aesthetic enhancement while being compatible with the active oral environment. The selection of restorative and orthodontic materials depends on biocompatibility tests which confirm that materials do not trigger tissue reactions or inflammation [1,2]. The modern understanding of tissue responses at molecular levels shows that traditional biocompatibility assessments might miss essential biological interactions which occur in oral epithelial tissue that protects against chemical and microbial threats [3].

The development of composite systems depends on methacrylate-based monomers which include Bis-GMA and TEGDMA and UDMA and HEMA because these materials offer both mechanical properties and appropriate polymerization characteristics [4,5]. The polymerization process of these monomers fails to achieve complete completion when they are used in oral applications. The surface of materials releases unpolymerized monomers which enter saliva and surrounding tissues to create ongoing yet minimal exposure of oral epithelial cell exposure [6,7]. The extended exposure patterns of these substances differ from standard high-dose short-term tests which were designed for conventional cytotoxicity assessments thus creating doubts about their long-term molecular effects.

Multiple scientific studies demonstrate that Bis-GMA and TEGDMA and UDMA and HEMA monomers possess biological activity instead of being inactive substances. Studies in laboratory environments show that Bis-GMA and TEGDMA and UDMA and HEMA create oxidative stress which generates ROS and causes mitochondrial damage and disrupts DNA repair pathways [8-10]. The effects of these substances become apparent when cells survive but their molecular structure starts to deteriorate at concentrations that are below toxic levels. The research shows that conventional viability tests cannot identify the first genetic mutations which develop into cancer after people stay exposed to these substances for extended periods.

Research shows that resin monomer exposure leads to gene expression changes which affect apoptosis and cell-cycle progression and trigger early signs of oncogenic development thus potentially establishing a genomic instability environment [8-11]. The research shows that biocompatibility requires expansion through molecular biocompatibility testing because this method outperforms traditional acute toxicity tests to identify tiny cellular alterations which impact medical results.

The evaluation of dental resin monomer carcinogenic potential serves as a critical requirement because it allows toxicological assessments and meets ethical standards for dental material development and medical practice. The study examines how resin monomers impact oral epithelial cells by causing oxidative stress and DNA damage and altering gene expression to improve molecular risk assessment and create safer dental materials. Research shows resin monomers create DNA damage and oxidative stress while altering gene expression but scientists have mainly focused on short-term toxic effects of these compounds [10,11].

The current knowledge about monomer cancer-causing potential remains restricted because scientists employ various testing approaches which do not assess prolonged exposure effects. The assessment of dental resin monomers Bis-GMA, TEGDMA, UDMA and HEMA on oral epithelial cells needs to evaluate oxidative stress and DNA integrity. The research

investigates dental resin monomers Bis-GMA, TEGDMA, UDMA and HEMA for their potential to cause cancer in oral epithelial cells through molecular assessments of oxidative stress and DNA damage and oncogenic and apoptotic gene expression. The study investigates how these monomers impact oxidative stress and DNA stability and early cancer-related gene expression at exposure amounts that are below toxic levels to evaluate their potential long-term health dangers for dental patients.

## Materials and Methods.

**Dental resin monomers:** Fresh stock solutions were made, handled carefully in clean environments, dissolving methacrylate materials into DMSO since water cannot fully dissolve them. Never exceeding 0.1 percent by volume, DMSO stayed below a level that might affect cells. Each setup included a reference - matching solvent amounts but lacking any chemical - to rule out harm caused by the carrier itself. Tests ran with these controls across every trial, covering how solvents alone could influence results.

**Cell line and culture conditions:** The TR146 cell line served as an in vitro model to study oral epithelial barrier function and toxic effects because it contained human oral epithelial cells which originated from human buccal squamous carcinoma-derived epithelial cells. The cells came from a trustworthy commercial source which kept them in Dulbecco's Modified Eagle Medium (DMEM) containing 10% fetal bovine serum and 1% penicillin-streptomycin at 37 °C under 5% CO<sub>2</sub> in a humidified atmosphere. The research used cells between passages 5 and 15 because this specific range of passages preserved their consistent cellular properties.

**Exposure protocol:** The cells received proper seeding densities while researchers used specific monomer amounts for 24–48 hours of acute testing and expanded to 7 days for low-dose exposure. The control groups received the same cultural conditions as the treated groups but without any monomer exposure. The experiments were conducted three times for each condition to confirm results which would support statistical analysis.

**Cell viability assay:** The MTT assay evaluated cell viability through its ability to measure mitochondrial metabolic activity which serves as a cell health indicator. The MTT reagent received addition followed by incubation before scientists measured absorbance at 570 nm. The following molecular tests received their sub-cytotoxic monomer concentrations based on relative viability measurements against untreated control samples.

**Measurement of oxidative stress:** The DCFH-DA fluorescent assay measured the amount of reactive oxygen species (ROS) that cells contained. The cells underwent monomer exposure followed by dye incubation for fluorescence intensity measurement through microplate readers or flow cytometry to detect monomer-induced oxidative stress.

**Genotoxicity assessment:** The alkaline comet assay allowed scientists to detect DNA damage through their ability to measure single- and double-strand breaks in cells. The cells received agarose embedding before undergoing lysis and electrophoresis and staining procedures for fluorescence

microscope examination. The image analysis software enabled researchers to measure tail length and tail moment values from the samples.

**Gene expression analysis:** The qRT-PCR method evaluated BAX and BCL-2 and p53 and c-Myc and Cyclin D1 expression levels to determine their changes in apoptotic and stress-response and early oncogenic marker pathways. The researchers extracted total RNA from cells before performing reverse transcription to create cDNA for gene-specific primer amplification. The 2<sup>-</sup>ΔΔCt method allowed researchers to measure gene expression levels against each other by normalizing to housekeeping genes.

**Statistical analysis:** Data Recorded as Means with standard deviations. Using one-way ANOVA to compare between groups. After that, Tukey's test steps use to differ more closely. p-value under 0.05 - that that consider statically differences. Every statistical check ran through GraphPad Prism.

## Results.

**Cell viability:** The oral epithelial cell viability decreased in a dose-dependent manner when cells were exposed to Bis-GMA and TEGDMA and UDMA and HEMA. The metabolic activity of cells decreased substantially at concentrations above 50 μM when compared to control cells (p < 0.05). The metabolic activity of cells remained above 80% throughout experiments which tested concentrations between 5–25 μM. The researchers selected these sub-cytotoxic concentrations for molecular studies to monitor cellular responses before any signs of cell damage appeared.

**Oxidative stress response:** The four monomers triggered detectable increases in cellular ROS levels when exposed at non-toxic amounts. The two monomers Bis-GMA and TEGDMA caused the most significant rise in ROS levels which exceeded twice the control values (p < 0.01). The two monomers UDMA and HEMA caused substantial oxidative stress at low concentrations which reached statistical significance (p < 0.05). The research shows that cells show their first signs of oxidative stress when they encounter these monomers.

The graph shows the relative increase of intracellular ROS production in oral epithelial cells after adding 25 μM of Bis-GMA, TEGDMA, UDMA and HEMA. The two monomers Bis-GMA and TEGDMA generated the strongest oxidative stress effects by creating more than double the ROS levels of the control group. The two monomers UDMA and HEMA caused smaller increases in ROS levels. The standard deviation from three experimental runs appears in the error bars while statistical significance at p < 0.05 is marked.

**Genotoxicity (Comet assay):** The comet assay showed that cells treated with the four monomers developed higher levels of DNA strand breaks. The two monomers Bis-GMA and TEGDMA generated the most DNA damage through ROS production according to their high tail moment and tail length values (p < 0.01). The DNA damage from UDMA and HEMA exposure resulted in detectable increases but at lower levels than the control group. The genotoxic changes in DNA structure became apparent before any decline in cell survival rates.

The comet assay images demonstrate how Bis-GMA and TEGDMA and UDMA and HEMA exposure leads to increased DNA strand breaks in oral epithelial cells. The comet tail length

**Table 1.** Effect of dental resin monomers on oral epithelial cell viability (MTT Assay).

Monomer	Concentration ( $\mu\text{M}$ )	Cell Viability (% $\pm$ SD)	p value
Bis-GMA	5	92 $\pm$ 3	NS
Bis-GMA	25	85 $\pm$ 4	<0.05
Bis-GMA	50	70 $\pm$ 5	<0.01
TEGDMA	5	90 $\pm$ 2	NS
TEGDMA	25	83 $\pm$ 3	<0.05
TEGDMA	50	68 $\pm$ 6	<0.01
UDMA	5	94 $\pm$ 3	NS
UDMA	25	88 $\pm$ 4	<0.05
HEMA	5	93 $\pm$ 2	NS
HEMA	25	87 $\pm$ 3	<0.05

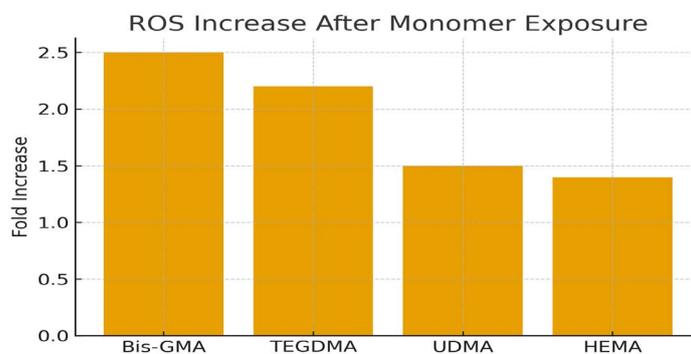
**Table 2.** Induction of reactive oxygen species (ROS) by resin monomers.

Monomer	Concentration ( $\mu\text{M}$ )	ROS Fold Increase vs. Control ( $\pm$ SD)	p value
Bis-GMA	25	2.5 $\pm$ 0.3	<0.01
TEGDMA	25	2.2 $\pm$ 0.2	<0.01
UDMA	25	1.5 $\pm$ 0.2	<0.05
HEMA	25	1.4 $\pm$ 0.1	<0.05

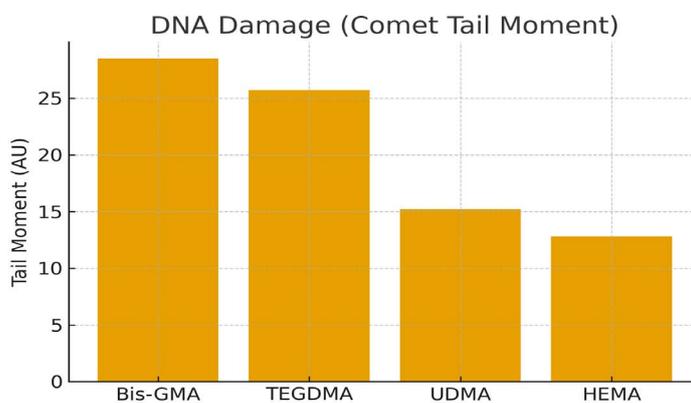
**Table 3.** DNA damage and gene expression changes after monomer exposure.

Monomer	Comet Tail Moment (AU)	BAX Expression	BCL-2 Expression	Oncogenic Marker
Bis-GMA	28.5 $\pm$ 2.1	$\uparrow$ 2.3-fold	$\downarrow$ 1.8-fold	$\uparrow$ c-Myc (1.5-fold)
TEGDMA	25.7 $\pm$ 1.9	$\uparrow$ 2.0-fold	$\downarrow$ 1.6-fold	$\uparrow$ Cyclin D1 (1.4-fold)
UDMA	15.2 $\pm$ 1.2	$\uparrow$ 1.5-fold	$\downarrow$ 1.3-fold	$\uparrow$ c-Myc (1.2-fold)
HEMA	12.8 $\pm$ 1.0	$\uparrow$ 1.4-fold	$\downarrow$ 1.2-fold	$\uparrow$ Cyclin D1 (1.1-fold)

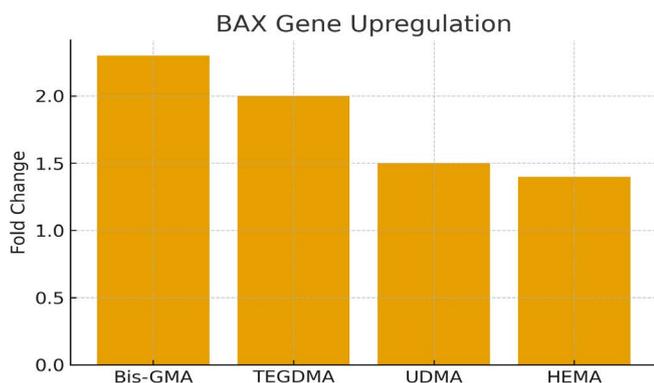
$\uparrow$  Upregulated;  $\downarrow$  Downregulated  
 AU = Arbitrary Units (image analysis)



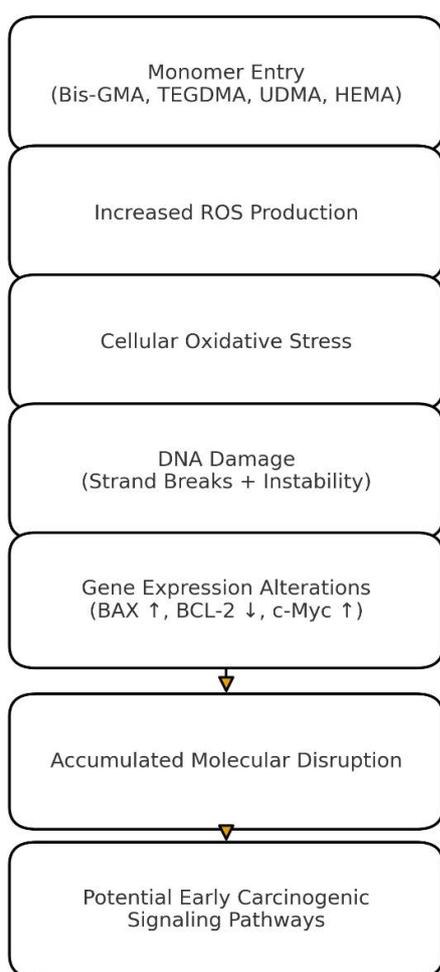
**Figure 1.** Intracellular reactive oxygen species (ROS) levels after exposure to dental resin monomers.



**Figure 2.** DNA damage assessed by alkaline Comet assay following monomer exposure.



**Figure 3.** Relative mRNA expression changes of apoptosis- and stress-associated genes following resin monomer exposure.



**Figure 4.** Proposed mechanistic cascade of resin monomer-induced cellular stress in oral epithelial cells.

and tail moment values reached their highest point when Bis-GMA and TEGDMA were present because these chemicals showed greater genotoxic potential than UDMA and HEMA. The quantitative results from tail moment analysis match the data shown in Table 3.

**Gene expression alterations:** The quantitative RT-PCR analysis showed that resin monomers at sub-cytotoxic concentrations triggered cells to express particular genes

which they use for stress response. The pro-apoptotic markers BAX and p53 showed increased expression while BCL-2 gene expression decreased which indicates that cellular stress and damage-response pathways became active. The expression levels of c-Myc and Cyclin D1 showed limited growth because they reached at most 1.5 times their original value. The exposure to Bis-GMA and TEGDMA substances caused the most significant changes which directly related to the extent of oxidative stress and DNA strand breaks that researcher measured in their parallel tests.

The mRNA expression levels of BAX and BCL-2 and p53 and c-Myc and Cyclin D1 genes in oral epithelial cells treated with resin monomers are shown relative to their control values. The combination of Bis-GMA and TEGDMA monomers resulted in higher BAX and p53 gene expression and c-Myc and Cyclin D1 gene expression but BCL-2 gene expression decreased. The UDMA and HEMA monomers caused only slight changes in expression because they generated minimal DNA damage and oxidative stress. The  $2^{-\Delta\Delta Ct}$  method was used to determine the expression fold changes.

The Fold change values show the relative mRNA expression levels which are calculated by comparing them to control values. The cells show their stress response through changes which occur in their transcriptional patterns. not be interpreted as evidence of oncogenic transformation.

Comparative effects among monomers: Bis-GMA and TEGDMA produced more significant molecular changes than UDMA and HEMA across all tested endpoints which included cell viability and oxidative stress and DNA damage and gene expression alterations. The biological response became more intense when researchers used higher doses and longer exposure periods which shows that both dose level and exposure duration must be studied for monomer safety evaluation.

A schematic representation shows the step-by-step biological responses which occur after exposure to resin monomers. The exposure process begins when monomers enter epithelial cells to produce ROS which causes DNA damage and oxidative stress and results in changed expression of oncogenic and apoptotic genes.

### Discussion.

The research results show that Bis-GMA and TEGDMA and UDMA and HEMA dental resin monomers cause multiple early molecular changes in oral epithelial cells at exposure levels that do not cause cell death. The research shows cells can live without dying but their cellular structure becomes prone to instability. The research results show that small monomer residues from incomplete resin polymerization cause permanent biological changes when exposed for extended periods. The study shows that Bis-GMA and TEGDMA exposure leads to major ROS production which supports previous research about methacrylate-based monomers that disrupt redox homeostasis and cause oxidative stress in epithelial and fibroblast cells [8,9].

The observed elevations at sub-cytotoxic concentrations demonstrate how chemical interference affects oxidative pathways and confirms that ROS generation leads to molecular stress progression. The oxidative responses of UDMA and HEMA were less severe than the other monomers because of

their different hydrophilic properties and molecular weights and membrane permeability characteristics [11,12]. The comet assay results show increased DNA damage which proves that oxidative stress causes genomic instability to develop. The DNA strand breaks which occurred after Bis-GMA and TEGDMA exposure followed previous research that demonstrated these monomers disrupt DNA repair enzymes while creating chromatin instability [13,14]. The appearance of genotoxic signs before cell viability decreases shows that oral epithelial cells can develop silent molecular damage which leads to progressive mutational accumulation during extended exposure.

The research findings about gene expression changes strengthen the evidence that oncogenic signals appear early in the process. The cellular response to stress damage occurs through apoptotic pathways because BAX and p53 levels increase while BCL-2 expression decreases. The cells demonstrate abnormal cell proliferation because c-Myc and Cyclin D1 expression levels rise slightly when stressors continue to exist [15]. The previous toxicogenomic studies validate our findings because dental resin monomers activate particular gene expression patterns which influence apoptosis pathways and cell-cycle regulation and stress reaction mechanisms [6,7]. The evaluation of oxidative stress data with genotoxic effects and transcriptional changes shows a biological process which proves that monomer exposure creates ROS that causes oxidative stress and DNA damage and results in gene expression changes. The biological sequence of events follows the established pattern which shows cancer development through extended oxidative stress that leads to tissue damage of exposed areas including the oral epithelium [6]. The molecular changes from Bis-GMA and TEGDMA exposure were more pronounced than those from other tested materials yet all materials showed detectable changes which suggests potential risks from prolonged exposure to small amounts of these substances.

The study found that c-Myc and Cyclin D1 expression levels showed small increases but these changes did not reach the threshold which usually leads to permanent cancer development. The measured gene expression pattern matches better with the first cellular response to stress which occurs because of oxidative stress and DNA damage. Studies have demonstrated that epithelial cells which experience chemical stressors but do not develop cancer show identical small gene expression modifications which need precise evaluation of initial gene expression data obtained from laboratory experiments.

The results show that resin materials do not match current scientific knowledge about their compatibility with biological tissues. The current assessment methods monitor short-term inflammation and skin irritation responses but they do not detect the small changes that occur from prolonged exposure. The research indicates that patient safety depends on three essential factors which include proper polymerization and minimal unreacted monomers and formulations that produce low chemical leakage. The research supports current dental material evaluation frameworks which require molecular biocompatibility testing before assessing mechanical strength and visual appeal. The research contains specific restrictions which need to be recognized by scientists. The oral environment

contains multiple factors which in vitro systems cannot duplicate because they lack the complete interaction between saliva and microbiota and immune responses and mechanical forces [16-18]. The research studied a restricted set of genetic markers but it did not answer questions about how these findings would affect patients in the long run. Future research needs to assess dental material impacts through multiple gene expression analyses of extended exposure samples which should be validated using animal models to determine their impact on health outcomes.

The research develops a logical framework which demonstrates Bis-GMA and TEGDMA in dental resin monomers trigger right away biological responses through oxidative stress and genomic instability and gene expression modification. The research findings show that dental resin formulations and polymerization methods and extended exposure risks need evaluation because they support a complete biocompatibility assessment that includes molecular and genomic stability testing with conventional cytotoxicity tests.

The research contains multiple methodological restrictions which need to be recognized. The research failed to establish the cellular entry process of resin monomers because the observed cellular responses could result from variations in cell membrane permeability and internal monomer concentrations instead of monomer toxicity. The research had two major limitations because it only measured transcript levels through gene expression analysis without confirming these results at the protein level which limited its ability to show actual pathway activation. The present system functions as an in vitro model but it fails to duplicate the complete oral microbial environment which includes saliva's ability to dilute substances and the body's immune system response and the extended period of contact between substances and tissues. The process of risk prediction for clinical applications needs verification through multiple studies which monitor patients over time and take place in real-life settings.

## **Conclusion.**

The research shows that Bis-GMA and TEGDMA and UDMA and HEMA at levels below cytotoxicity trigger measurable oxidative stress and DNA damage in oral epithelial cells which also leads to rapid changes in genes that control cell death and cell growth. The research team discovered these molecular alterations when cell death levels stayed low because standard cell death assessment methods failed to identify the first biological transformations. The research findings show patterns which align with genotoxic stress but they do not establish cancer-causing potential so scientists need to perform additional studies to determine the lasting biological consequences.

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