

Policy Brief:

The Need to Manage Arsenic Contamination in the Food Supply Chain and Processing to Promote Public Health

Fahimeh Abdollahimajd^{1,2,3}, Babak Arjmand⁴, Fatemeh Bandarian⁵, Masoumeh Farahani^{6*}

1. Skin Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran.
2. Research Center of Artificial Intelligence in Health, Shohada-e Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran.
3. Clinical Research Development Unit, Shohada-e Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran.
4. Hematology, Oncology and Stem Cell Transplantation Research Center, Research Institute for Oncology, Hematology and Cell Therapy, Tehran University of Medical Sciences, Tehran, Iran.
5. Metabolomics and Genomics Research Center, Endocrinology and Metabolism Molecular-Cellular Sciences Institute, Tehran University of Medical Sciences, Tehran, Iran.
6. Proteomics Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

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Masoumeh Farahani

E-mail:

mfarahani2005@gmail.com

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Abstract

Background and Objective: Long-term exposure to arsenic through food, beyond direct risks (such as skin lesions), causes immune system dysfunction, cardiovascular disease, and reduced cognitive development in children. This research provides a health policy framework based on the results of a study of cellular and molecular changes in human keratinocytes exposed to arsenic.

Material and Methods: RNA sequencing of arsenic-exposed HaCaT keratinocytes versus controls was used to identify differentially expressed genes. A protein-protein interaction network was then constructed and a critical subnet was extracted through topological analysis.

Results and Conclusion: Overall, integrating gene expression with protein interaction analysis highlighted a core protein subnetwork and revealed possible underlying mechanisms. Here, a health policy framework was built on these findings, emphasizing stricter exposure control and the development of surveillance.

Keywords: Human health risk, Food chain contamination, Arsenic toxicity

1. Introduction and statement of the problem

Arsenic is a toxic heavy metal and a pervasive environmental pollutant that can infiltrate the food chain, particularly in rice, rice-based products, vegetables, and seafood, through various sources, including drinking water and soil. Research indicates that inorganic arsenic forms pose a greater toxicity risk than their organic counterparts. Long-term exposure to arsenic can significantly endanger human health, leading to several types of cancer, skin diseases, neurological problems, and developmental disorders [1, 2]. This is not only one of the foremost challenges to individual health, but also a heavy economic burden on the health system. Traditional methods alone are inadequate to eliminate arsenic at high levels, indicating the need for scientific interventions at both macro and

household levels. This policy recommendation is derived from our research project aimed at deciphering the molecular mechanisms associated with arsenic-induced cutaneous squamous cell carcinoma (cSCC) [3].

2. Methods

Our previous study used a systematic bioinformatics and network pharmacology approach to investigate the molecular mechanisms of arsenic toxicity in human keratinocyte (HaCaT) cells and the potential of protective agents. First, we identified differentially expressed genes (DEGs) by analyzing gene expression data (RNA-seq) and enriched them using gene ontology (GO) and KEGG pathways. Then, a critical protein subnetwork was screened by constructing a protein-protein interaction (PPI) network

in Cytoscape software and applying stringent topological filters (via CytoNca and CytoHubba plugins). Finally, using the CTD database, potential chemical and natural agents to counteract arsenic toxicity were identified, and their efficacy was evaluated through drug-protein interaction analysis and detailed molecular docking simulations to predict binding affinity to target proteins [3].

3. Results

The results of our previous study indicate that in long-term arsenic-treated human keratinocytes, 705 differentially expressed genes (DEGs) were identified. Among these genes, 15 key targets were identified as critical arsenic-responsive subnetwork [3]. In the evaluation phase of protective agents, “folic acid” and “quercetin” showed the most importance with these targets.

4. Health Policy Framework

4.1. Suggested strategies for risk reduction

a) Modifying preparation and processing methods (household-based educational interventions):

- Post-harvest processing methods, standardizing cooking practices, and providing scientific guidelines for arsenic reduction [4, 5].
- Public awareness to change dietary habits and cooking practices in at-risk communities [6].

b) Integrating food biotechnology for the remediation of arsenic (structural interventions):

- Developing strategies to reduce arsenic uptake: Invest in genetic modification and targeted genome editing (CRISPR) to produce varieties of cereals that have the lowest rate of arsenic uptake by the roots [7].
- Bio-processing and environmental bioremediation: Using specific enzymes and microbial fermentation under controlled conditions to convert toxic forms of arsenic into less bioavailable compounds [8].
- Use of biosorbents for trapping arsenic: Research on biomaterials and bio-based nano materials that can be used to trap arsenic in groundwater, drinking water, and wastewater [9].

Food biotechnology can be both a complement to household-level methods and a final solution to reduce the pollution load on the water, soil-plant systems, and food products [1].

4.2. Policy recommendations and guidelines

a- Primary prevention and exposure control: Given its molecular carcinogenicity and other proven health effects, monitoring and reducing arsenic in drinking water and food

in high-risk areas should be prioritized as a fundamental cancer prevention measure.

b- Strengthen regulatory monitoring: Identify and map high-risk areas and require arsenic labeling on the packaging of strategic products in high-risk areas.

c- Surveillance and early detection: Strengthen screening programs for communities chronically exposed to arsenic and consider exposure history (e.g., years of drinking water with high arsenic) in risk-based screening algorithms.

d- Investment in research and development (R&D):

- Periodic updates of toxicological standards, occupational restrictions, and nutritional recommendations for at-risk populations.

- Study of the cost-effectiveness of technologies for public policy and support knowledge-based biotechnology projects to provide “arsenic-free” products to people.

e- Education and outreach: Integrate applied biotechnology training (e.g., rapid home arsenic-detection kits) into environmental health programs and human health risk assessment.

f- Agricultural management: Replacing sensitive crops in high-arsenic fields with more resistant crops through agricultural biotechnology knowledge.

Current evidence links arsenic exposure to specific molecular changes in humans. Modifying traditional food preparation methods is a necessary but insufficient step. To tackle high arsenic levels, the government must move towards “biotechnological sustainability.” Incorporating biotechnology into production and processing, along with changes in social behaviors around household practices, is the only practical way to significantly reduce the burden of arsenic-related diseases in the country. A health policy framework should focus on exposure reduction and surveillance, while explicitly promoting research to test these potential protective factors before widespread implementation in the public health arena.

5. Declarations

5.1. Acknowledgement

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5.2. Conflict of Interest

The authors report no conflict of interest.

5.3. Using chatbots

We used an AI academic search engine for scientific research (<https://consensus.app/>).

5.4. Authors' Contributions

All authors reviewed and edited the manuscript.



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خلاصه سیاستی:

نیاز به مدیریت آلودگی آرسنیک در زنجیره تأمین و فرآوری مواد غذایی برای ارتقاء سلامت عمومی

فهیمة عبداللهی مجد^{۱،۲،۳،۴}، بابک ارجمند^۴، فاطمه بندریان^۵، معصومه فراهانی^۶

۱. مرکز تحقیقات پوست، دانشگاه علوم پزشکی شهید بهشتی، تهران، ایران.

۲. مرکز تحقیقات هوش مصنوعی در سلامت، بیمارستان شهدای تجریش، دانشگاه علوم پزشکی شهید بهشتی، تهران، ایران.

۳. واحد توسعه تحقیقات بالینی، بیمارستان شهدای تجریش، دانشگاه علوم پزشکی شهید بهشتی، تهران، ایران.

۴. مرکز تحقیقات خون‌شناسی، انکولوژی و پیوند سلول‌های بنیادی، پژوهشکده انکولوژی، خون‌شناسی و سلول‌درمانی، دانشگاه علوم پزشکی تهران، تهران، ایران.

۵. مرکز تحقیقات متابولومیکس و ژنومیکس، پژوهشکده علوم سلولی-مولکولی غدد درون‌ریز و متابولیسم، دانشگاه علوم پزشکی تهران، تهران، ایران.

۶. مرکز تحقیقات پروتئومیکس، دانشگاه علوم پزشکی شهید بهشتی، تهران، ایران.

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فهیمة عبداللهی مجد

پست الکترونیک:

mfarahani2005@gmail.com

چکیده

سابقه و هدف: قرار گرفتن طولانی مدت در معرض آرسنیک از طریق غذا، فراتر از خطرات مستقیم (مانند ضایعات پوستی)، باعث اختلال در عملکرد سیستم ایمنی، بیماری‌های قلبی عروقی و کاهش رشد شناختی در کودکان می‌شود. این تحقیق بر اساس نتایج مطالعه تغییرات سلولی و مولکولی در کراتینوسیت‌های انسانی در معرض آرسنیک، یک چارچوب سیاست‌گذاری سلامت ارائه می‌دهد.

مواد و روش‌ها: توالی‌یابی RNA کراتینوسیت‌های HaCaT در معرض آرسنیک در مقایسه با گروه کنترل برای شناسایی ژن‌های با بیان متفاوت استفاده شد. سپس یک شبکه برهمکنش پروتئین-پروتئین ساخته شد و یک زیرشبکه حیاتی از طریق آنالیز توپولوژیکی استخراج گردید.

یافته‌ها و نتیجه‌گیری: به طور کلی، ادغام بیان ژن با تجزیه و تحلیل تعامل پروتئین، یک زیرشبکه پروتئینی اصلی را برجسته کرد و مکانیسم‌های اساسی احتمالی را آشکار ساخت. در اینجا، یک چارچوب سیاست‌گذاری سلامت بر اساس این یافته‌ها ساخته شد که بر کنترل سختگیرانه‌تر مواجهه و توسعه نظارت تأکید دارد.

واژگان کلیدی: خطر سلامت انسان، آلودگی زنجیره غذایی، سمیت آرسنیک