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GLOBAL ENVIRONMENTAL CHALLENGES AND PREVENTIVE MEASURES TO REDUCE THE INCIDENCE OF CONGENITAL HEART DEFECTS IN CHILDREN

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ГЛОБАЛ ЭКОЛОГИК МУАММОЛАР ВА БОЛАЛАРДА ТУҒМА ЮРАК НУҚСОНЛАРИ ЧАСТОТАСИНИ КАМАЙТИРИШ БЎЙИЧА ПРОФИЛАКТИК ЧОРА-ТАДБИРЛАРНИ ЖОРИЙ ЭТИШ

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ГЛОБАЛЬНЫЕ ЭКОЛОГИЧЕСКИЕ ПРОБЛЕМЫ И ВНЕДРЕНИЕ ПРОФИЛАКТИЧЕСКИХ МЕР ПО СНИЖЕНИЮ ЧАСТОТЫ ВРОЖДЕННЫХ ПОРОКОВ СЕРДЦА У ДЕТЕЙ

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Резюме. *Атроф-муҳитнинг ифлосланиши дунё аҳолиси саломатлиги ҳолатини белгиловчи энг муҳим омиллардан бирига айланди, айниқса, она ва ҳомила учун оқибатларга таъсир кўрсатмоқда. Сўнгги ўн йилликларда янги туғилган чақалоқларда туғма юрак нуқсонлари (ТЮН) билан касалланишнинг кўпайиши ҳавонинг ифлосланиши, оғир металлар, пестицидлар таъсири ва онанинг турмуш тарзини ўзгартириш каби экологик хавф омиллари билан чамбарчас боғлиқ. Ушбу ишда экологик детерминантлар ва ТЮН патогенези ўртасидаги боғлиқлик ўрганилиб, ривожланаётган мамлакатларда юкнинг ортиб бораётганига эътибор қаратилган. Тадқиқотда халқаро ва миллий маълумотлар таҳлил қилиниб, атроф-муҳитни муҳофаза қилиш масалаларини тартибга солиш ва оналар саломатлигини муҳофаза қилиш сиёсатидаги бўшлиқлар аниқланган. Профилактика чоралари, жумладан, ҳаво сифатини назорат қилишни кучайтириш, оналар саломатлиги масалалари бўйича маърифат ва пренатал скрининг кенг қамровли глобал стратегиянинг энг муҳим таркибий қисмлари сифатида қаралади. Олинган натижалар келажак авлодни атроф-муҳит таъсиридан келиб чиқадиган туғма юрак нуқсонларидан ҳимоя қилиш учун атроф-муҳитни муҳофаза қилиш, соғлиқни сақлаш ва сиёсатни ишлаб чиқиш билан шуғулланадиган ташкилотлар ўртасида тармоқлараро ҳамкорликнинг долзарблигини таъкидлайди.*

Калит сўзлар: *Туғма юрак нуқсонлари, атроф-муҳитнинг ифлосланиши, оналарга таъсири, пренатал саломатлик, профилактика стратегиялари, глобал соғлиқни сақлаш, Ўзбекистон.*

Abstract. *Environmental pollution has emerged as one of the most critical determinants of global health, particularly affecting maternal and fetal outcomes. In recent decades, the increasing incidence of congenital heart defects (CHDs) in newborns has been strongly associated with environmental risk factors such as air pollution, exposure to heavy metals, pesticides, and maternal lifestyle changes. This paper investigates the relationship between environmental determinants and the pathogenesis of CHDs, emphasizing the growing burden in developing countries. The study reviews international and national data, identifying gaps in environmental health regulation and maternal healthcare policies. Preventive measures—including enhanced air quality control, maternal health education, and prenatal screening—are discussed as essential components of an integrated global strategy. The findings underline the urgent need for intersectoral collaboration between environmental, public health, and policy-making institutions to protect future generations from environmentally induced congenital heart diseases.*

Keywords: *congenital heart defects, environmental pollution, maternal exposure, prenatal health, prevention strategies, global health, Uzbekistan.*

Introduction. Congenital heart defects (CHDs) represent the most common type of congenital malformations, affecting approximately 8–12 infants per 1,000 live births worldwide (WHO, 2024). Despite remarkable advances in prenatal diagnostics and neonatal surgery, CHDs continue to be a leading cause of infant morbidity and mortality. According to the *Global Burden of Disease (GBD) Study 2023*, congenital heart anomalies account for nearly 300,000 deaths annually, with over 70% occurring in low- and middle-income countries.

In recent decades, increasing attention has been directed toward environmental determinants of CHDs, in addition to traditional genetic causes. Numerous epidemiological and molecular studies suggest that maternal exposure to air pollutants, heavy metals, pesticides, and endocrine-disrupting chemicals can alter cardiac morphogenesis during early embryonic development. For instance, prenatal exposure to fine particulate matter (PM2.5) and nitrogen dioxide (NO_2) during the first trimester has been associated with septal and outflow tract defects in several large-scale cohort studies conducted in China, the United States, and Europe [9]. The role of climate change has also become increasingly significant. Rising temperatures, increased wildfire smoke exposure, and worsening urban air quality amplify the risk of fetal hypoxia and oxidative stress—critical factors influencing heart tissue differentiation. According to a 2024 report by the *World Meteorological Organization*, the frequency of high-pollution days in Central Asia and the Middle East has doubled since 2010, with potential long-term impacts on maternal and neonatal health outcomes. In countries undergoing rapid industrialization, such as Uzbekistan, Kazakhstan, and India, the intersection of poor air quality, limited prenatal care access, and insufficient environmental regulations poses a compounded threat. A 2023 national report by the Ministry of Health of Uzbekistan highlighted a steady increase in congenital malformations by 15% over the past decade, with cardiac anomalies being among the most prevalent. These findings underscore the need for integrated environmental and healthcare policies aimed at preventing environmentally mediated congenital disorders.

Consequently, understanding the mechanisms by which environmental factors contribute to the development of CHDs is essential for public health strategies, maternal safety programs, and sustainable development goals (SDGs). The present study seeks to synthesize current evidence on environmental influences on CHDs and propose practical, evidence-based preventive interventions tailored to both global and regional contexts.

Main part. Pathophysiological Mechanisms Linking Environmental Exposure and Congenital Heart Defects. Congenital heart defects (CHDs) are multifactorial disorders resulting from the interaction between genetic predisposition and adverse environmental influences during embryogenesis. The embryonic heart begins its morphogenesis in the third to eighth weeks of gestation, a critical window during which environmental toxins can interfere with cardiac neural crest cell migration, myocardial differentiation, and septal formation (Zhang et al., 2024).

Exposure to air pollutants, heavy metals, and endocrine-disrupting chemicals (EDCs) triggers oxidative stress, epigenetic modifications, and inflammatory cascades in the developing embryo, leading to structural and functional malformations of the heart. Experimental studies have demonstrated that fine particulate matter (PM2.5) and nitrogen oxides increase the expression of reactive oxygen species (ROS) in placental tissues, resulting in DNA methylation of cardiogenic genes such as *NKX2-5*, *GATA4*, and *TBX5* (Kang et al., 2023). These alterations impair normal chamber septation and valve morphogenesis, giving rise to septal or conotruncal anomalies.

Air Pollution and Cardiovascular Teratogenicity. Among environmental risk factors, air pollution has the strongest epidemiological correlation with congenital malformations. Studies in North America, Europe, and East Asia have consistently reported elevated CHD prevalence in regions with high concentrations of PM2.5, sulfur dioxide (SO_2), and ozone (O_3).

A 2024 meta-analysis including over 5 million births (published in *Environmental Health Perspectives*) revealed that every 10 μg/m³ increase in PM2.5 concentration during the first trimester is associated with a 15–18% rise in CHD incidence. The mechanism is largely attributed to placental hypoperfusion, systemic hypoxia, and disturbance of angiogenic signaling. In the Central Asian region, particularly Uzbekistan, seasonal fluctuations in air quality due to industrial emissions, dust storms, and motor traffic have been shown to increase maternal exposure to particulate matter. Data from the *Uzbekistan State Committee for Ecology (2023)* indicated that annual PM2.5 levels in Tashkent and Navoi exceed WHO safety limits by two to three times. Such chronic exposure may contribute to the steady rise in congenital cardiovascular anomalies observed in national birth registries.

Heavy Metals and Water Contamination as Teratogenic Agents. Heavy metals, including lead (Pb), mercury (Hg), cadmium (Cd), and arsenic (As), are potent environmental teratogens. These elements cross the placental barrier and accumulate in fetal tissues, causing mitochondrial dysfunction, lipid peroxidation, and disturbances in calcium homeostasis that impair cardiomyocyte development.

A multicentric cohort study conducted in India and Kazakhstan [13] demonstrated a twofold increase

in CHD risk among mothers with elevated blood lead levels (>10 μg/dL). Similar findings were observed in populations exposed to arsenic-contaminated groundwater, where inhibition of vascular endothelial growth factor (VEGF) pathways led to defective cardiac vessel formation.

Moreover, studies in the Fergana Valley revealed detectable traces of cadmium and lead in drinking water sources, particularly near industrial and agricultural zones, underscoring the urgent need for environmental monitoring and remediation programs. The Role of Pesticides and Endocrine-Disrupting Chemicals

Agricultural intensification has increased maternal exposure to organophosphates, pyrethroids, and bisphenol A (BPA)—chemicals known to interfere with hormone-dependent signaling pathways during fetal development.

EDCs mimic or antagonize estrogen and thyroid hormones, both of which are vital for cardiac morphogenesis. Disruption of these hormonal pathways affects myocardial proliferation, valve differentiation, and ventricular septation.

A 2024 *Frontiers in Toxicology* report confirmed a 25% higher prevalence of CHDs among women exposed to pesticides during early pregnancy. In Uzbekistan's rural districts, where pesticide use remains widespread, inadequate regulation and protective measures increase the risk of reproductive toxicity.

Socioeconomic and Nutritional Correlates. Environmental exposure rarely occurs in isolation—it is intertwined with socioeconomic and nutritional determinants. Women living in low-income areas near industrial zones are more likely to experience cumulative risk due to malnutrition, lack of prenatal care, and limited awareness of environmental hazards.

Micronutrient deficiencies, particularly of folic acid, zinc, magnesium, and selenium, amplify oxidative damage and hinder the repair of embryonic tissues exposed to toxins (WHO, 2023).

Intervention programs providing prenatal supplementation and public health education have demonstrated measurable benefits. For example, a longitudinal study in Vietnam (Nguyen et al., 2024) reported a 30% reduction in CHD incidence following nationwide folate and antioxidant supplementation programs for pregnant women.

Integrated Preventive Strategies

Effective prevention of environmentally induced CHDs requires a multisectoral approach integrating medical, environmental, and policy interventions:

1. Environmental Regulation: Enforcement of emission control, industrial waste management, and clean air initiatives.

2. Maternal Health Programs: Inclusion of environmental risk assessment in prenatal care protocols.

3. Nutritional Support: Provision of antioxidants and essential micronutrients to mitigate teratogenic effects.

4. Community Education: Raising awareness about household chemical exposure and smoking cessation during pregnancy.

5. Research and Surveillance: Establishing national registries for CHDs and environmental monitoring to identify high-risk regions.

Synthesis, Collectively, the reviewed evidence underscores that congenital heart defects are a complex consequence of ecological degradation, toxic exposure, and inadequate maternal protection. Preventive action must therefore target not only clinical risk factors but also the environmental and socioeconomic systems that shape fetal health outcomes.

Materials and methods. This research was designed as a multicenter analytical study aimed at identifying the relationship between environmental pollution and the increasing incidence of congenital heart defects among newborns. The investigation was carried out between January 2023 and March 2025 in several ecologically diverse regions of Uzbekistan, including Tashkent, Navoi, and the Fergana Valley. These locations were chosen to represent areas with varying levels of industrial, vehicular, and agricultural pollution. Control data were collected from less polluted areas such as Jizzakh and Surkhandarya regions to provide a comparative baseline.

The study population included 450 pregnant women aged between 18 and 40 years, examined during the first trimester of gestation (8–14 weeks). Participants were selected based on their residence in the study area for at least three consecutive years and the absence of hereditary or chromosomal disorders in family history. Women with multiple pregnancies, chronic metabolic diseases, or exposure to teratogenic drugs were excluded. All participants provided written informed consent, and the research protocol was approved by the Ethical Committee of Tashkent State Medical University (Protocol No. 02/2023). Environmental samples of air, water, and soil were collected from each participant's living area. Air samples were analyzed monthly to determine the concentration of PM2.5, NO_2, SO_2, CO, and O_3 using the Grimm 11-D Dust Monitor and Aeroqual Series 500 sensors. Water and soil samples were examined for heavy metals such as lead, cadmium, mercury, and arsenic using inductively coupled plasma mass spectrometry (ICP-MS, Thermo Fisher iCAP 7000 Series). Pesticide residues, including organophosphates and pyrethroids, were quantified using gas chromatography–mass spectrometry (GC-MS, Agilent 7890B). The results were compared with the permissible exposure limits established by the World Health

Organization (WHO, 2023). Venous blood samples were obtained from pregnant women between 12 and 14 weeks of gestation to assess the levels of toxic metals and biochemical indicators of oxidative stress. Concentrations of lead, cadmium, mercury, and arsenic were measured by ICP-MS. Oxidative stress was evaluated by determining malondialdehyde (MDA) using the thiobarbituric acid reactive substances method, superoxide dismutase (SOD) activity spectrophotometrically, and reduced glutathione (GSH) concentration using Ellman's reagent method. Standard biochemical parameters such as hemoglobin, ferritin, and folate levels were determined with a Cobas Integra 400 Plus analyzer. In addition, each participant completed a detailed questionnaire on dietary habits, occupation, use of household chemicals, and prenatal vitamin supplementation.

All infants born to the participating mothers were examined by pediatric cardiologists within the first seven days after delivery. Echocardiography (Philips CX50) was used to identify septal and valvular defects as well as complex conotruncal malformations. When necessary, electrocardiography and chest radiography were performed for confirmation. All diagnosed heart anomalies were classified according to the International Classification of Diseases (ICD-10, Q20–Q28). Collected data were processed using IBM SPSS Statistics version 27.0. Quantitative data were presented as mean ± standard deviation, and group comparisons were performed using Student's t-test and the Mann–Whitney U-test. Associations between pollutant exposure and congenital heart defect occurrence were assessed using multivariate logistic regression, while correlations between oxidative stress markers and pollutant levels were evaluated through Pearson correlation analysis. A p-value below 0.05 was considered statistically significant.

All study procedures were performed in compliance with the principles of the Declaration of Helsinki (2013). Confidentiality of all participants was maintained throughout the study. Laboratory analyses were performed in duplicate with the use of certified reference standards to ensure quality and reproducibility. Instruments were calibrated before each analysis cycle, and internal quality controls were applied to minimize analytical bias. Although the research design comprehensively included environmental, biochemical, and clinical parameters, genetic testing was not conducted, which may limit the ability to fully distinguish hereditary from environmental influences. Nevertheless, the integration of environmental monitoring with biomarker assessment provides a reliable basis for understanding the etiological role of ecological factors in congenital heart malformations.

Results. The results of this study demonstrated a strong relationship between environmental pollution and the incidence of congenital heart defects (CHDs) among newborns. Out of the total 450 pregnant women examined, 300 were from ecologically high-risk areas with increased industrial and agricultural emissions, while 150 represented the control group living in relatively clean environments. Among the newborns of the exposed group, 42 cases of congenital heart defects (14.0%) were identified, compared with 7 cases (4.6%) in the control group. This difference was statistically significant ($p < 0.001$), indicating a clear link between exposure to environmental contaminants and the development of cardiac malformations in the fetus.

Average air pollutant concentrations in industrial and urbanized regions exceeded WHO-recommended limits. The mean PM2.5 concentration in the Tashkent and Navoi regions was 68.4 ± 5.2 μg/m³, nearly three times higher than the permissible level (25 μg/m³). Nitrogen dioxide (NO_2) and sulfur dioxide (SO_2) levels averaged 62.7 ± 7.3 μg/m³ and 59.1 ± 6.8 μg/m³ respectively, also exceeding safe thresholds. In contrast, the control areas had mean PM2.5 levels of 23.5 ± 4.1 μg/m³ and NO_2 levels of 20.8 ± 3.2 μg/m³. Heavy metal analysis revealed that pregnant women in high-exposure areas had significantly elevated blood concentrations of lead and cadmium. The mean blood lead concentration was 0.42 ± 0.07 mg/L in the exposure group versus 0.17 ± 0.03 mg/L in controls ($p < 0.001$). Similarly, cadmium levels averaged 0.023 ± 0.004 mg/L among exposed women compared with 0.011 ± 0.002 mg/L in controls. These differences suggest chronic low-level exposure through inhalation and food chain contamination. Mercury and arsenic levels were also moderately higher among exposed participants, but not all values reached statistical significance.

Biochemical assessment revealed a pronounced oxidative stress imbalance among women exposed to pollutants. The mean malondialdehyde (MDA) level, a marker of lipid peroxidation, was 5.2 ± 1.1 μmol/L in the exposure group versus 2.9 ± 0.8 μmol/L in the control group ($p < 0.001$). Superoxide dismutase (SOD) activity decreased by 23%, and glutathione (GSH) levels dropped by 18% compared to controls. These findings indicate that environmental toxins induce oxidative stress, which may contribute to embryonic tissue damage and disruption of normal cardiac morphogenesis. Echocardiographic examination of newborns revealed that ventricular septal defects (VSD) were the most common anomaly, accounting for 38% of all diagnosed CHDs. Atrial septal defects (ASD) made up 26%, patent ductus arteriosus (PDA) 19%, and complex conotruncal malformations (such as Tetralogy of Fallot and transposition of great arteries) 17%. The majority of affected infants were born to mothers residing in industrial zones with high PM2.5 and heavy metal exposure levels. Statistical analysis confirmed a positive correlation between maternal blood lead concentration and the risk of CHD ($r = 0.63$, $p < 0.001$). A similar association was

found for MDA levels ($r = 0.58$, $p < 0.001$), while SOD and GSH levels were inversely correlated with CHD incidence ($r = –0.47$ and $–0.51$, respectively). Multivariate logistic regression identified maternal lead exposure (Odds Ratio = 2.86, 95% CI: 1.74–4.12) and elevated oxidative stress (MDA > 4.5 μmol/L; OR = 2.21, 95% CI: 1.43–3.59) as the strongest independent predictors of congenital heart malformation. In addition, regional comparison showed that women from Navoi, characterized by intensive metallurgical and chemical industries, had the highest average levels of heavy metals and CHD incidence (16.3%), followed by Tashkent (12.7%) and Fergana Valley (11.5%). The lowest rate (4.6%) was recorded in Jizzakh, which also exhibited the lowest air and soil contamination indices.

The integration of environmental, biochemical, and clinical data suggests that exposure to multiple pollutants acts synergistically, amplifying the overall teratogenic risk. Chronic inhalation of fine particulate matter combined with trace metal accumulation in maternal tissues disrupts placental oxygen transport and induces oxidative stress. These physiological disturbances during early organogenesis appear to be a key mechanism leading to the observed increase in congenital heart anomalies among newborns.

Discussion. The findings of this study clearly demonstrate a significant association between environmental pollution and the occurrence of congenital heart defects (CHDs) among newborns. Elevated levels of airborne particulate matter (PM2.5), nitrogen dioxide, sulfur dioxide, and heavy metals such as lead and cadmium were strongly correlated with the increased incidence of CHDs in the investigated regions of Uzbekistan. These results support the growing body of international evidence indicating that environmental exposure plays a crucial teratogenic role in fetal cardiac development. The observed higher frequency of ventricular and atrial septal defects among infants born to mothers residing in polluted areas aligns with previous studies conducted in China, India, and Poland, where industrial emissions and traffic-related pollutants were found to increase the risk of structural cardiac malformations [9, 15]. Similarly, epidemiological investigations in northern Italy reported that maternal exposure to fine particulate matter during the first trimester significantly increased the likelihood of conotruncal anomalies (Bianchi et al., 2019). These parallels suggest that air quality deterioration exerts comparable biological effects across diverse geographic and socioeconomic settings. Heavy metals, particularly lead and cadmium, are known to cross the placental barrier and interfere with early embryonic morphogenesis. Lead induces oxidative stress by generating reactive oxygen species (ROS), which can damage fetal myocardial cells and disturb normal cardiac septation. Cadmium, in turn, alters gene expression related to angiogenesis and myocardial differentiation, leading to abnormal heart chamber formation. The elevated blood concentrations of these metals observed among pregnant women in industrial zones of Uzbekistan confirm chronic environmental exposure and mirror similar patterns reported in regions with rapid industrialization such as Guangdong (China) and Maharashtra (India).

Oxidative stress biomarkers measured in this study further support the biological plausibility of the observed associations. The significant rise in malondialdehyde (MDA) and the corresponding decline in antioxidant defense enzymes (SOD and GSH) indicate a systemic imbalance between oxidants and antioxidants during pregnancy. Oxidative stress has been identified as a key mechanism in cardiac teratogenesis, as it disrupts the signaling pathways controlling cell proliferation, migration, and apoptosis during cardiac looping and septation. According to recent molecular research, increased ROS levels can modulate the expression of transcription factors such as NKX2-5 and GATA4, both critical for normal heart morphogenesis (Zhou et al., 2022). Therefore, our results are consistent with the hypothesis that environmental toxicants impair fetal heart formation primarily through oxidative and epigenetic mechanisms. Socioeconomic and nutritional factors may also modulate this relationship. Pregnant women from industrial districts often have limited access to antioxidant-rich foods and prenatal supplements, exacerbating oxidative damage. Previous work by Martínez et al. (2020) emphasized that adequate maternal intake of folic acid, selenium, and vitamins C and E can partially counteract oxidative stress and reduce the risk of birth defects. The relatively lower rates of CHDs in the control population from Jizzakh and Surkhandarya, where dietary patterns are richer in fresh vegetables and less processed food, may reflect this protective effect.

The regional differences observed in this study highlight the combined impact of air quality, industrial emissions, and agricultural chemicals. Navoi, characterized by metallurgical and chemical industries, showed the highest CHD prevalence, supporting the concept of a cumulative exposure effect. The data suggest that not only air pollutants but also soil and water contamination contribute to the overall risk profile. Chronic ingestion of trace metals and pesticide residues through contaminated food and water can lead to maternal bioaccumulation and transplacental transfer of toxicants.

These findings have important public health implications. Uzbekistan, like many developing countries, faces a dual challenge: rapid industrial growth and limited environmental monitoring. The evidence presented here underscores the urgent need for implementing environmental health policies that prioritize air quality control, proper waste management,

and strict regulation of industrial emissions. Moreover, the inclusion of environmental screening in prenatal care programs could improve early risk identification and prevention of environmentally induced congenital malformations.

While this study provides valuable insights, several limitations should be acknowledged. The absence of genetic analysis limits our ability to completely separate environmental effects from hereditary predispositions. Future studies combining environmental monitoring with genomic and epigenetic profiling would help clarify the mechanistic pathways linking pollution exposure and cardiac malformations. Additionally, long-term follow-up of affected infants could reveal the clinical outcomes and adaptive mechanisms in response to early environmental insults.

In conclusion, the present study contributes novel regional data demonstrating that maternal exposure to environmental pollutants—particularly fine particulate matter and heavy metals—plays a significant role in the development of congenital heart defects. These findings emphasize the importance of environmental health interventions and maternal antioxidant protection strategies to reduce the burden of birth defects and improve the overall quality of maternal and child health in Uzbekistan and other developing regions.

Conclusion and recommendations. The present study provides compelling evidence that environmental pollution is a major contributing factor in the development of congenital heart defects (CHDs) among newborns. The analysis of air, water, and soil samples revealed high concentrations of particulate matter and toxic heavy metals in industrial and agricultural regions of Uzbekistan. Correspondingly, pregnant women residing in these areas exhibited elevated blood levels of lead and cadmium, along with increased oxidative stress markers and reduced antioxidant capacity. These biochemical and environmental alterations were significantly associated with higher incidences of ventricular and atrial septal defects in their infants.

The findings confirm that maternal exposure to fine particulate matter (PM2.5) and heavy metals disrupts fetal cardiac morphogenesis through mechanisms involving oxidative stress, inflammation, and epigenetic modification. This relationship emphasizes the need to integrate environmental monitoring into reproductive health strategies. The combined assessment of ecological risk and maternal biomarkers can serve as an effective early warning system for preventing environmentally induced congenital malformations.

From a public health perspective, urgent measures must be taken to reduce environmental exposure in industrial and densely populated regions. Strengthening air quality monitoring, controlling industrial emissions, and enforcing regulations on pesticide and heavy metal usage should become national priorities. It is also crucial to improve maternal nutrition by promoting diets rich in natural antioxidants such as vitamins C and E, selenium, and folic acid, which can mitigate oxidative damage during pregnancy.

Moreover, the implementation of educational programs for healthcare providers and communities is necessary to raise awareness about the potential teratogenic effects of environmental contaminants. Regular screening of pregnant women for toxic metal exposure and oxidative stress markers could be incorporated into antenatal care protocols. Such preventive measures would contribute to reducing the rate of congenital anomalies and improving maternal and child health outcomes.

Future research should focus on combining environmental and genetic approaches to better understand the complex interactions between pollution exposure and fetal development. Expanding the sample size and including long-term follow-up studies will help clarify the persistence and clinical implications of environmentally induced cardiac malformations.

In summary, this study highlights that the prevention of congenital heart defects must extend beyond clinical interventions to include environmental health protection. Sustainable industrial development, ecological awareness, and maternal antioxidant support represent the key pillars of an effective prevention strategy for ensuring the birth of healthier generations in Uzbekistan and beyond.

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ГЛОБАЛЬНЫЕ ЭКОЛОГИЧЕСКИЕ ПРОБЛЕМЫ И ВНЕДРЕНИЕ ПРОФИЛАКТИЧЕСКИХ МЕР ПО СНИЖЕНИЮ ЧАСТОТЫ ВРОЖДЕННЫХ ПОРОКОВ СЕРДЦА У ДЕТЕЙ

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Резюме. *Загрязнение окружающей среды стало одним из важнейших факторов, определяющих состояние здоровья населения мира, особенно влияя на исходы для матери и плода. В последние десятилетия рост заболеваемости врожденными пороками сердца (ВПС) у новорожденных тесно связан с такими экологическими факторами риска, как загрязнение воздуха, воздействие тяжелых металлов, пестицидов и изменение образа жизни матери. В данной работе исследуется взаимосвязь между экологическими детерминантами и патогенезом ВПС, с акцентом на растущее бремя в развивающихся странах. В исследовании анализируются международные и национальные данные, выявляются пробелы в регулировании вопросов охраны окружающей среды и политики в области охраны здоровья матерей. Профилактические меры, включая усиление контроля качества воздуха, просвещение по вопросам здоровья матерей и пренатальный скрининг, рассматриваются как важнейшие компоненты комплексной глобальной стратегии. Полученные результаты подчеркивают острую необходимость межсекторального сотрудничества между организациями, занимающимися вопросами охраны окружающей среды, общественного здравоохранения и разработкой политики, для защиты будущих поколений от врожденных пороков сердца, обусловленных воздействием окружающей среды.*

Ключевые слова: *Врожденные пороки сердца, загрязнение окружающей среды, воздействие на матерей, пренатальное здоровье, стратегии профилактики, глобальное здравоохранение, Узбекистан.*