# Reproductive Health Impacts of Industrial Chemical Pollution: A Focus on Hexavalent Chromium

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**Abstract**

Hexavalent Chromium is a highly toxic and environmentally persistent form of Chromium which has been extensively studied due to its adverse health effects as a result of extensive human exposure. The most biologically relevant forms of chromium are Cr(VI) and trivalent chromium [Cr(III)]. While Cr(III) is a trace nutrient essential to the promotion of insulin action by carbohydrate and lipid metabolism, Cr(VI) is a strong oxidizing agent that is highly soluble and bioavailable accounting for its threat as a human health hazard [9, 14]. The toxicological importance of Cr(VI) allows it to readily cross biological membranes, undergo intracellular reductions, and generate reactive oxygen species that damage DNA, proteins, and cellular membranes. These effects establish the classification of Cr(VI) as a human carcinogen by national and international health agencies [4, 6, 13]. The oral exposure to Cr(VI) causes systemic distribution and genotoxic effects compared to the more direct inhalational exposure; this is relevant to the easier susceptibility of exposure and primary transmission of toxins to the general population [14].

**Introduction**

Environmental contamination by Cr(VI) can occur through both anthropogenic and natural processes. Industrial sources are the primary contributors to environmental pollution through stainless steel manufacturing, wood preservation, textile processing, pigment production, textile processing, and electroplating [11].

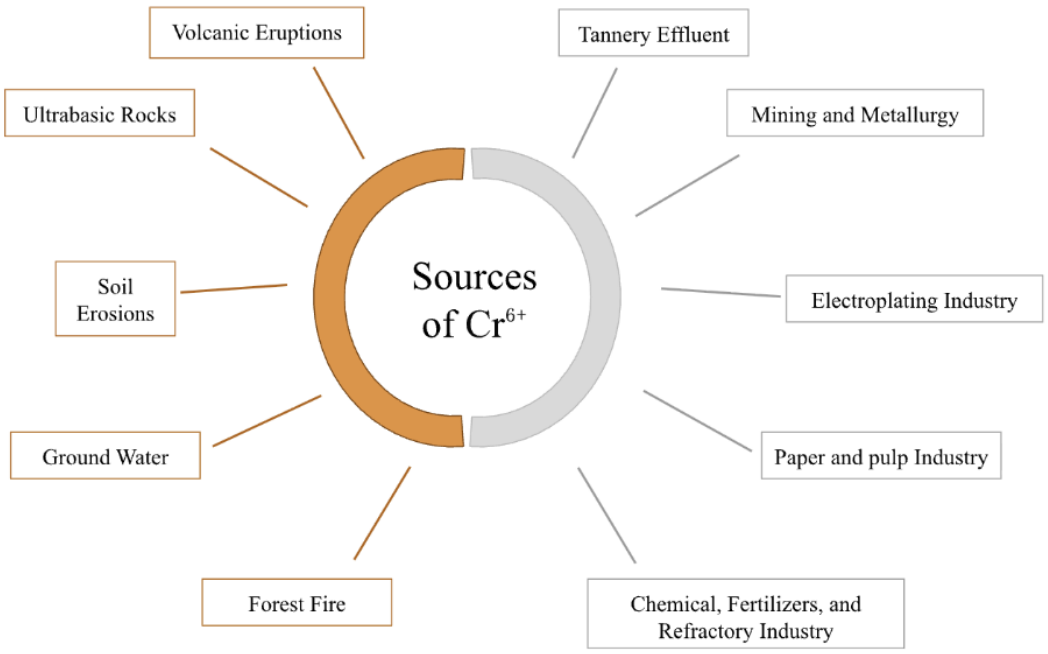


*Fig. 1. Environmental (left) and anthropogenic (right) sources of Cr 6+ in nature [17].*

**Global Efforts**

The environmental release of Cr(VI) occurs by leaching from infrastructure, improper disposal of environmental wastes, leakage from hazardous waste sites, and corrosion of manufacturing structures. In a few geological regions, the natural occurrence of Cr(VI) forms by oxidative weathering of the chromium-bearing minerals in groundwaters and eroded rock sediments [12]. Cr(III) has a high positive charge, attributing it's readily binding to soil and organic matter.

In comparison, Cr(VI)'s highly oxyanionic properties remains dissolved in oxygenated water as the chromate ion: ($CrO_4^{2-}$) [10]. The solubility of the Cr(VI) ion makes it to be highly

durable, allowing the ion to persist and migrate over long distances. This is significant as the routing of drinking water contamination represents the greatest risk of non-occupational human exposure. Figure 2 portrays the regional prevalence of Cr(VI) supporting its ability to travel far by its

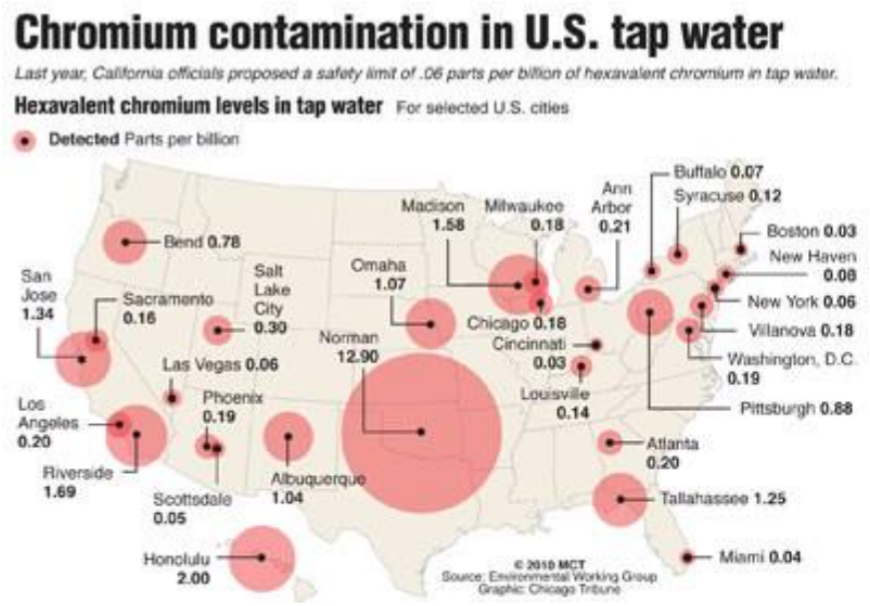


*Figure 2. Regional map of Hexavalent Chromium detection in tap water, measured in parts per billion (ppb) (Filter [20]).*

Following ingestion, the acidic environment of the stomach interacts with the Cr(VI) by a chemical reduction reaction to reduce the chemical to Cr(III). However, the incomplete mechanism results in substantial amounts of the Cr(VI) that bypass and enter systemic circulation within the body [9]. The absorption of the Cr(VI) is distributed to multiple organs, including the reproductive organs, kidney, placenta, liver, and possibly developing fetuses [1]. The anionic characteristic of Cr(VI) allows it to pass through lipid membranes by nonspecific sulfate and phosphate transport channels within cells [14]. The intracellular trapping immediately occurs, reducing the Cr(VI) to Cr(III), however, the mechanism results in large quantities of reactive oxygen species, causing oxidative stress, chromosomal aberrations, DNA strands breakages, and impairing of DNA repair [15]. This mechanism has been observed through

animal models, case-control, and mechanistic studies to determine the effects of toxic exposure by neural tube defects (NTD) [Figure 3].

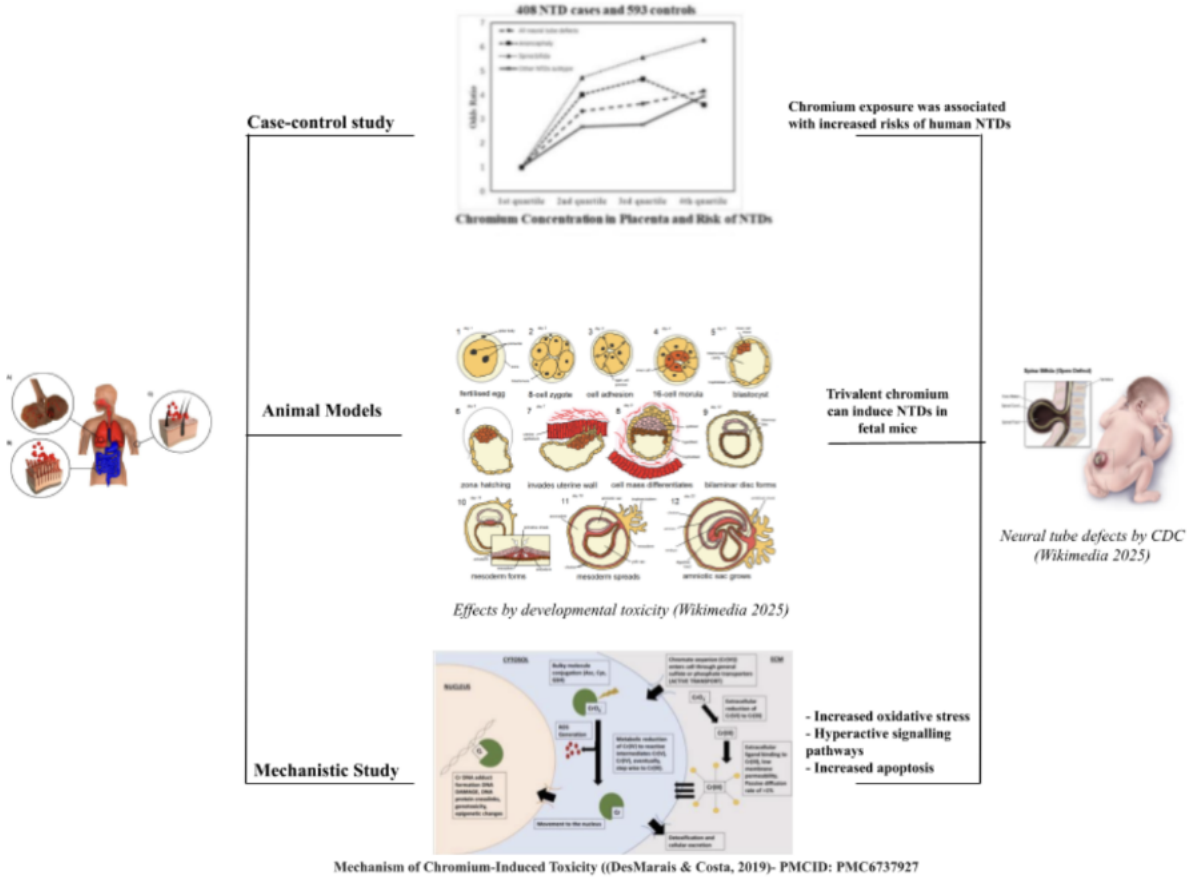


*Fig. 3. Visualization of animal models, case-control, and mechanistic studies used to study the toxic exposure of chromium within the body [21, 22, 23].*

## Fetal Challenges

The most adverse effect of Cr(VI) exposure is found in developmental toxicity in developing fetuses. Dose-dependent experimental and animal studies found the oral ingestion of Cr(VI) during gestation affects fetal development by weight, shortened crown–rump length, delayed skeletal ossification, and increased embryo reabsorption [1, 7]. This indicates that Cr(VI) has a direct impact on fetal growth and development. The ability of Cr(VI) to cross placental barriers causes the greatest expression of developmental toxicity. The chromate ions utilize the sulfate transport mechanisms along the highly active placental barriers to efficiently transfer into the

fetus from the systemic circulation of the mother. Due to the underdevelopment of antioxidant defenses, the Cr(VI) may accumulate and persist during fetal development. This threatens the genomic stability and oxidative processes of the fetus throughout development.

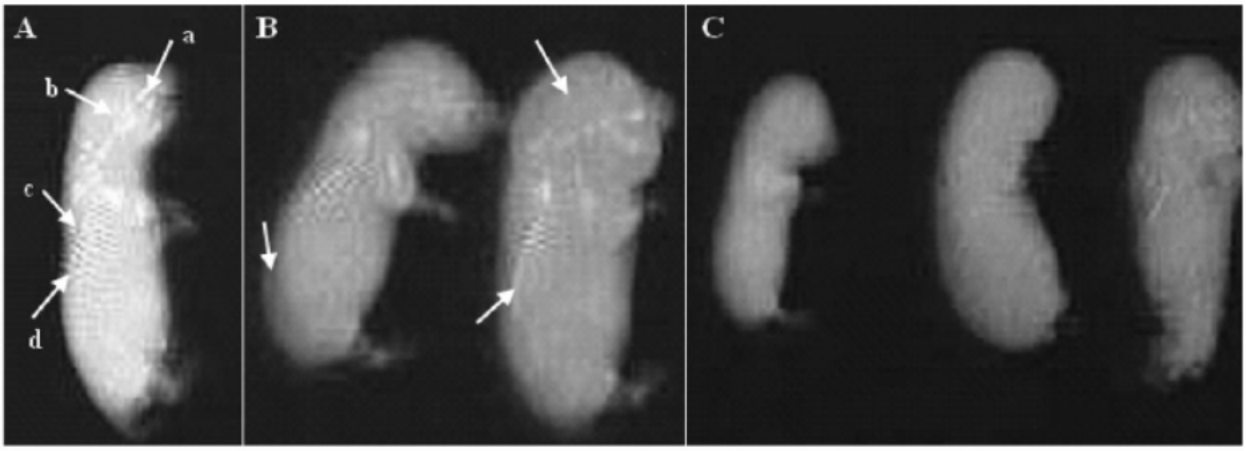


*Figure 4: A skeletal radiographic of fetuses from control ( A ) and chromium-treated rats with 1 mg/kg ( B ) and 2 mg/kg ( C ) during the organogenesis period. ( A ) Fetuses showed the presence of four sacral vertebrae (a: nasal bones, b: cranium bones, c: abdominal bones, d: caudal bones). ( B ) Fetuses showed incomplete ossification (arrowed). ( C ) Fetuses showed the absence of any vertebrae [18].*

The epidemiological analysis of these experimental findings aligns with its application into humans as well [4, 9]. A large case study justified the association between maternal chromium exposures and infant birth outcomes determined the rise in maternal chromium concentrations has a strong association to the low birth weight of newborns [13]. Within these maternal participants, chromium was detected in maternal blood, placental tissues, and umbilical cord blood, which all impacted the fetal exposure in utero. The analysis of low birth weight is a recognized indicator of developmental growth restrictions. Often, babies with low birth weights are disposed to increased risk of cardiovascular disease, metabolic disorders, immune dysfunctions, and greater malignant growth into adulthood. The exposure to Cr(VI) is supported to have adverse prenatal conditions that impact physiological development across human lifespans.

Alongside the developmental toxicity, Cr(VI) impacts the female reproductive system. Similar studies found consistent evidence in the reduction of ovarian follicle numbers, increase in follicular atresia, and impairment of ovulation in mice and rats. This impacts hormonal disturbances by damage to granulosa and theca cells that are responsible for estrogen and progesterone synthesis. Additionally, Cr(VI) disrupts endocrine regulation within the reproductive cycle and affects the normal regulation and maintenance of fertility. Possible indications of the damage to the uterine endometrium structure are a concern as it reduces the uterine receptivity and increases the likelihood of implantation failure and early pregnancy losses.

Similar experimentation found the rate of spontaneous abortions, prolonged pregnancy, and reduced litter size in affected animals [1]. These studies indicate reproductive impairment even after the removal of exposure, theorizing Cr(VI) exposure to be partially irreversible. Moreover, the Cr(VI) induces mitochondrial dysfunction, lipid peroxidation, and apoptosis in ovarian tissues. This causes accelerated depletions of the ovum, threatening female fertility and affecting material life expectancy.

Additionally, the exposure of Cr(VI) has adverse effects on the male reproductive system. Cr(VI) exposure results in chemical accumulation in the testes and damages the seminiferous tubules, possibly impairing spermatogenesis [1]. The exposure was seen with a reduction in sperm counts, decreased sperm motility, and increased abnormal sperm morphologies. This impacts the fertilization capacity and disrupts the Leydig cell function, both contributing to reduced testosterone synthesis. The role of testosterone in the maintenance of spermatogenesis and male reproductive function is critical to defend against endocrine disruptions, which would further cause testicular damage. Cellularly, the mechanism is comparative to the female reproductive system, where the lipid peroxidation in sperm membranes would cause increased embryonic losses, reduced fertilization success, and increased

risk of transferring contaminant damage to offspring. Sperm damage also has long-term implications on male fertility. The shared mechanisms of oxidative stress, endocrine disruptions, and genomic instability are closely linked by the reproductive and toxic exposure of Cr(VI). The gametic damages reduce fertility and increase the risk of transmitting genetic damage to offspring, while direct fetal exposures produce growth restriction pathways alongside long-term physiological impairment.

**Long term risks**

Cr(VI) exposure by drinking water has long-term population-level health effects [11]. Populations in industrial regions have elevated occurrences of cancer mortality by accompanied exposure to chemical pollutants. The increased mortality is found in stomach, liver, and lung cancers [6]. The impact of neighborhood health is a necessary consideration for the public health implications of industrial pollution. According to the national average by EPA survey study, populations that inhabit superfund sites by a three-mile radius are economically disadvantaged (13.7%), under the age of 5 (23%) and minority (39.6%) [16]. These statistics are represented in comparison to the nation, indicating the trend socioeconomic factors have on health (figure 5). Economically disadvantaged young families have tendencies to live in these superfund sites, which increase the threat of reproductive and developmental origins of disease. Cr(VI) follows systemic toxicity exposure patterns that are supported by the experimental study into its biological effects.

**TABLE 2: Detailed Data on the Population within 1 and 3 miles of Superfund sites**

Below are data on the demographic characteristics of the population surrounding Superfund sites. The table indicates whether certain population demographics near sites are above (in bold) or below (in italics) the U.S. average.

| Demographics | Population Within 1 Mile Of Sites (Approximate) | | Population Within 3 Miles Of Sites (Approximate) | | US Population (Approximate) | |
|---|---|---|---|---|---|---|
| **Race** | Percent | Number | Percent | Number | Percent | Number |
| **White** | *65.2%* | 13,465,608 | *65.0%* | 47,390,464 | 72.7% | 237,188,538 |
| **Black** | *12.6%* | 2,610,976 | **14.8%** | 10,806,046 | 12.7% | 41,281,123 |
| **Asian** | **8.5%** | 1,759,682 | **7.7%** | 5,646,019 | 5.4% | 17,581,234 |
| **Native American** | *0.7%* | 146,605 | *0.6%* | 449,849 | 0.8% | 2,707,577 |
| **Hawaiian/Pacific Islander** | **0.4%** | 72,953 | **0.3%** | 192,261 | 0.2% | 583,021 |
| **Other** | **12.6%** | 2,596,713 | **11.5%** | 8,384,355 | 8.3% | 26,948,478 |
| **Ethnicity** | | | | | | |
| **Hispanic (any race)** | **25.6%** | 5,291,125 | **24.2%** | 17,605,908 | 18.7% | 60,867,275 |
| **Non-Hispanic (any race)** | *74.4%* | 15,361,412 | *75.8%* | 55,263,086 | 81.3% | 265,422,696 |
| **Minority** | | | | | | |
| **Minority** | **49.8%** | 10,285,751 | **49.4%** | 35,986,864 | 39.6% | 129,080,779 |
| **Income** | | | | | | |
| **Households below the poverty level** | **15.4%** | 1,170,285 | **15.1%** | 4,037,341 | 13.7% | 16,567,346 |
| **Households with a ratio of income to poverty level of two and over** | *64.6%* | 12,973,217 | *65.6%* | 46,572,969 | 67.7% | 215,350,011 |
| **Education** | | | | | | |
| **Less than a high school education** | **14.9%** | 2,078,629 | **14.1%** | 6,907,708 | 12.5% | 27,526,286 |
| **Linguistically isolated** | | | | | | |
| **Linguistically isolated households** | **7.8%** | 596,375 | **7.3%** | 1,950,794 | 5.1% | 6,149,144 |
| **Age** | | | | | | |
| **Under 5 years of age** | **6.4%** | 1,330,018 | **6.3%** | 4,607,310 | 6.1% | 19,994,488 |
| **Under 18 years of age** | *22.2%* | 4,588,465 | *22.4%* | 16,304,103 | 22.8% | 74,241,185 |
| **Over 64 years of age** | *13.3%* | 2,744,745 | *13.8%* | 10,066,711 | 15.3% | 49,879,204 |
| **Total** | | **20,652,537** | | **72,868,994** | | **326,289,971** |

*Fig. 5. Table of the demographic characteristics of the populations living within a 1-mile and 3-mile radius from surrounding super fund sites [16].*

These studies are indicative of Cr(VI) characteristics as a carcinogen and potent toxicant to reproduction and development. The chronic exposure has significant outcomes to reproduction in both sexes, by fetal growth restriction, impaired fertility, hormonal disruptions, genomic instability, and increased early pregnancy losses. The consistent effect of exposure supports the conclusion of Cr(VI) as a trigger for chronic disease. Populations reliant on contaminated groundwater or private sector welling have higher risks of exposure without routine monitoring or treatment due to a lack of regulation [5, 2]. However, the social impact and maximum benefit of regulatory standards are lower than the output cost, which may result

in inadequate regulatory policies and solutions. This will result in systemic failure for vulnerable populations.

## Conclusion

Hexavalent chromium's bio accessibility and uptake contribute to its toxicity by developmental and reproductive health effects. The ingestion of contaminated drinking water results in the diffusion of Cr(VI), causing widespread distribution to reproductive and fetal tissues, as well as in the stomach, liver, and pancreas. Experimental and epidemiological studies demonstrate the effect prenatal exposure has on the development and growth of restrictions into adulthood, as well as the impairment of reproductive function within adults. Due to Cr(VI) established carcinogenicity, the threat of its widespread contamination is a necessary consideration for industrial and public health interventions. Alongside, regulating the discharge and safe management of Chromium, there is increased potential in the engineering of the infrastructure used within the transportation of drinking water. The use of steel pipes, seals, and coatings may be improved to prevent corrosion and limit the leaching of Cr(VI) into tap water.

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