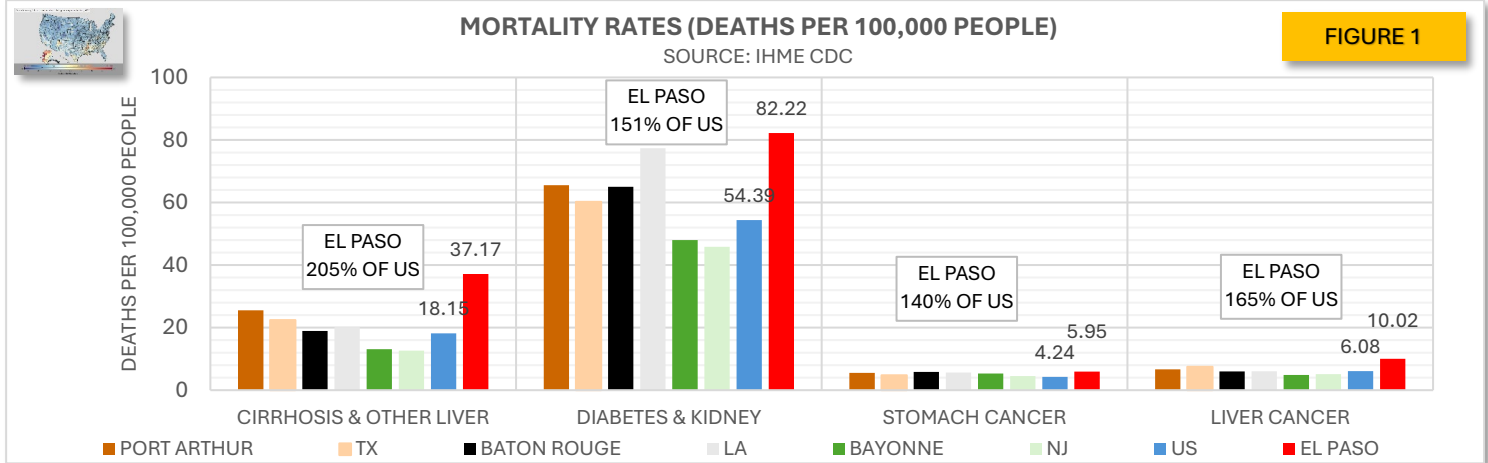


ENVIRONMENTAL & PUBLIC HEALTH IMPERATIVE



Although reducing/eliminating used motor oil contamination is a national priority since it is the #1 cause of freshwater contamination in the U.S. (EPA), reducing the contamination in the El Paso water shed is of paramount importance. That is due to the unusual ground and surface water chemistry – the high concentration of bromide (Br⁻) in the El Paso aquifers and the Rio Grande River (per the USGS) that interacts with used motor oil and other hydrocarbon contaminants – producing excessive concentrations of some of the most powerful toxins/carcinogens known – creating lethal stress (including but not limited to cancer) on the kidneys and liver. In essence, the greater the amount of hydrocarbons in the water supply – from dumped used motor oil, for example – the higher the level of toxins/carcinogens in the tap water.

Bromide in the El Paso drinking water supply is in a concentration over 50 times the U.S. average (USGS), and 9 of the 18 lethal chemicals present in excessive concentrations in El Paso's tap water are products of bromide interactions with hydrocarbons such as those in used motor oil.

The impact is not theoretical – it is very real with frighteningly higher mortality rates in El Paso (IHME & CDC) for kidney and liver diseases and cancer (the kidneys and liver of course are primary organs to remove toxins in the body).

El Paso Water Utilities

EWG's drinking water quality report shows results of tests conducted by the water utility and provided to the Environmental Working Group by the Texas Commission on Environmental Quality, as well as information from the U.S. EPA Enforcement and Compliance History database (ECHO).

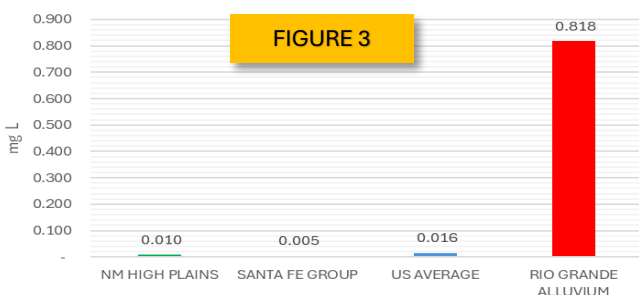
FIGURE 2

REQUIRES BROMIDE TO FORM



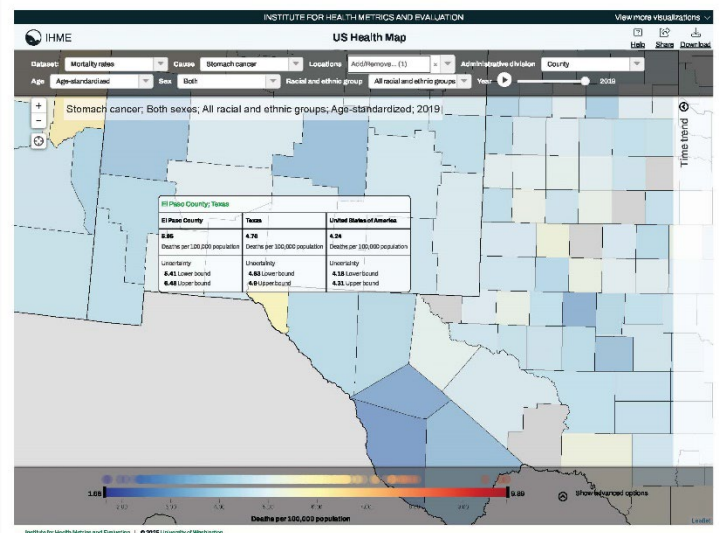
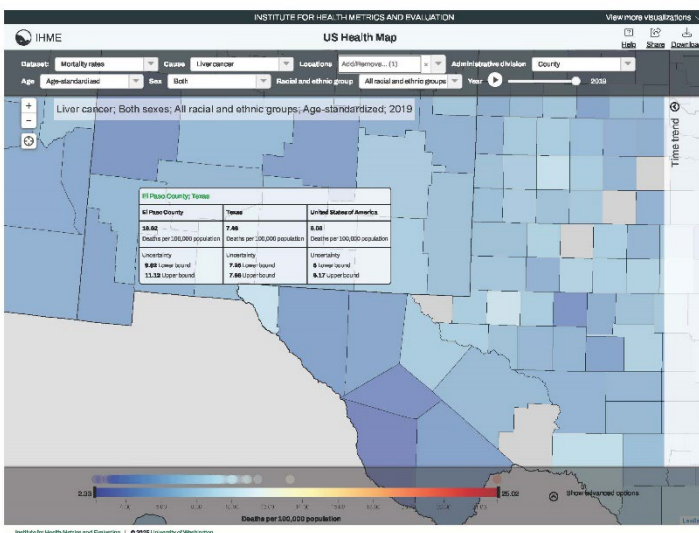
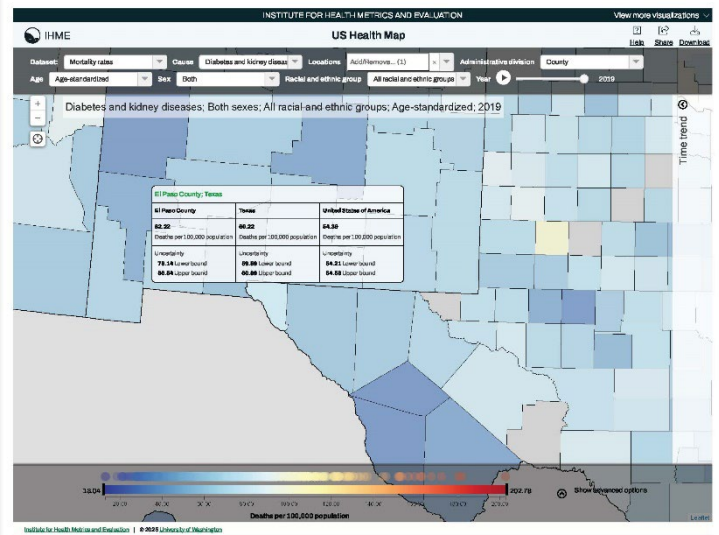
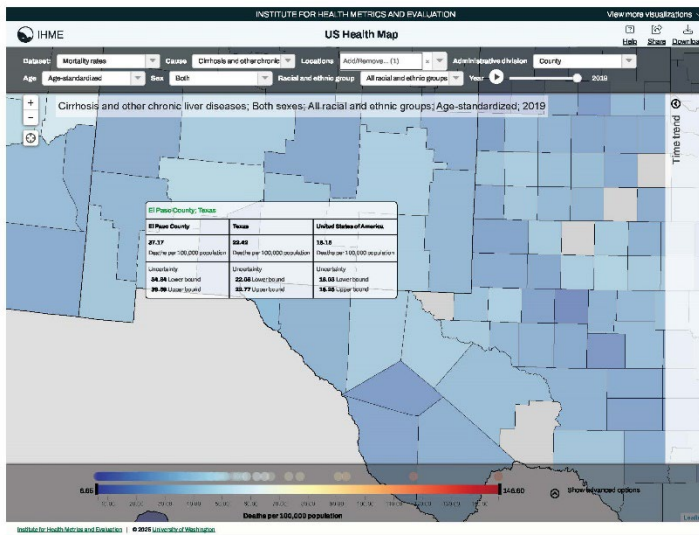
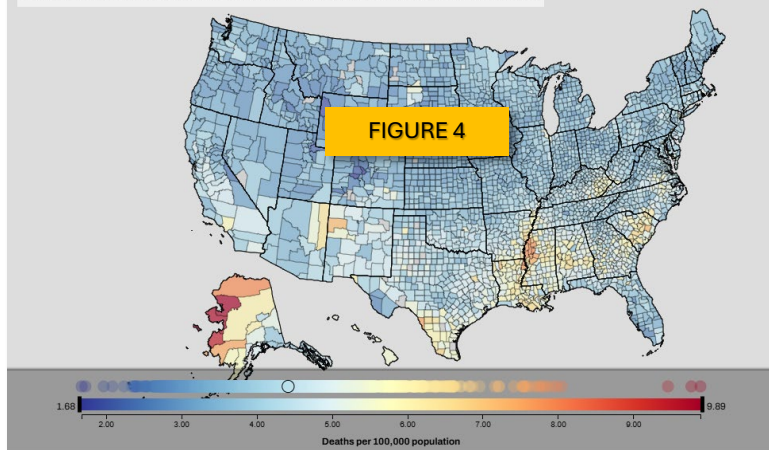
BR CONCENTRATIONS IN EL PASO DRINKING WATER SOURCES

FIGURE 3



ENVIRONMENTAL & PUBLIC HEALTH IMPERATIVE

Stomach cancer; Both sexes; All racial and ethnic groups; Age-standardized; 2019



ENVIRONMENTAL & PUBLIC HEALTH IMPERATIVE

BROMODICHLOROMETHANE

Bromodichloromethane can increase cancer risks and cause harm to reproduction, child development, and fetal growth and development in when present in quantities higher than 0.06 parts per billion (ppb).^[5] This data largely comes from studies reviewed or conducted by the California Office of Environmental Health Hazard Assessment.^[6] As of 2020, the chemical is considered by the [US Department of Health and Human Services](#) to be reasonably anticipated to be a human carcinogen, and by the [U.S. Environmental Protection Agency](#) (EPA) to be a probable human carcinogen.

[HEALTH EFFECTS - Toxicological Profile for Bromodichloromethane - NCBI Bookshelf](#) Hepatic Endpoints: Hepatic effects are a presumed health effect for humans based on limited evidence in humans and strong evidence in mice following acute inhalation exposure and in rats and mice following acute, intermediate, and chronic oral exposure. The liver effects include increases in serum enzymes, increases in liver weight, hepatocellular degeneration, and bile duct damage. Numerous studies confirmed that bromodichloromethane causes still births and spontaneous abortions.

BROMOFORM & DIBROMOCHLOROMETHANE

[Bromoform & Dibromochloromethane | Public Health Statement | ATSDR](#) Some studies in animals indicate that exposure to bromoform or dibromochloromethane may lead to liver and the kidney injury within a short period of time. Exposure to low levels of bromoform or dibromochloromethane do not appear to seriously affect the brain, liver, or kidneys. However, studies in animals indicate that long-term intake of either bromoform or dibromochloromethane can cause liver and kidney cancer.

DIBROMOACETIC ACID

[Dibromoacetic Acid | C2H2Br2O2 | CID 12433 - PubChem](#) [Toxicity and Carcinogenicity of the Water Disinfection Byproduct, Dibromoacetic Acid, in Rats and Mice - PMC](#) Toxic effects of DBA in the prechronic studies were detected in the liver (hepatocellular cytoplasmic vacuolization in rats and mice) and testes (delayed spermiation and atypical residual bodies in male rats and mice, and atrophy of the germinal epithelium in rats). In the 2-year studies, neoplasms were induced at multiple sites in rats and mice exposed to DBA; these included mononuclear cell leukemia and abdominal cavity mesotheliomas in rats, and neoplasms of the liver (hepatocellular adenoma or carcinoma and hepatoblastoma) and lung (alveolar adenoma or carcinoma) in mice. The increase in incidence of hepatocellular neoplasms in male mice was significant even at the lowest exposure concentration of 50 mg/L, which is equivalent to an average daily dose of approximately 5 mg/kg. These studies provide critical information for future re-evaluations of health-based drinking water standards for haloacetic acids. Several studies have shown that DCA administered in drinking water is carcinogenic to the liver of mice and rats (Herren-Freund et al., 1987; DeAngelo et al., 1991; Daniel et al., 1992; DeAngelo et al., 1996)

HALOACETIC ACIDS (HAA5)

[Human Exposure - Report on Carcinogens Monograph on Haloacetic Acids Found as Water Disinfection By-Products - NCBI Bookshelf](#) The major characteristic of the source water that determines the formation of disinfection by-products is the type and quantity of potentially reactive natural organic matter (NOM) and the inorganic halogen precursors, **bromide** and iodide. Anthropogenic and natural sources of bromide and iodide (see Table 2-1) can increase concentrations of these halide ions in source waters (e.g., due to incomplete removal or non-removal in wastewater treatment plants) and create brominated and iodinated HAAs and other disinfection by-products such as trihalomethanes and bromate (McTigue et al. 2014). Elevated levels of bromide and iodide in source water will likely shift production of HAAs during water disinfection toward brominated and iodinated species. [Exposure Characterization of Haloacetic Acids in Humans for Exposure and Risk Assessment Applications: An Exploratory Study - PMC](#) HAAs are mutagenic, cytotoxic, genotoxic, teratogenic, and carcinogenic [10,11,12,13,14,15]. Recent research has shown that HAAs can affect pyruvate dehydrogenase activity and disrupt cellular metabolism [16]. Chlorinated HAAs (e.g., DCAA and TCAA acid) induce oxidative stress in rats by reducing glutathione (GSH) synthesis [17]. Monohaloacetic acids (including chloro-, bromo-, and iodoacetic acid) can cause genomic DNA damage in Chinese hamster ovary cells, and inhibit follicle growth and steroidogenesis in mice ovary [13,18,19,20]. This report also confirms detrimental impact on fetus development, etc.

HALOACETIC ACIDS (HAA9)

[Analysis of Cumulative Cancer Risk Associated with Disinfection Byproducts in United States Drinking Water - PMC](#) A toxicological assessment indicated that haloacetic acids, and in particular brominated haloacetic acids, are more carcinogenic and are associated with a greater number of attributable cancer cases than trihalomethanes. Based on the toxicological analysis, cumulative lifetime cancer risk due to exposure to trihalomethanes and haloacetic acids for community water systems monitored under UCMR4, estimated with standard default parameters for body weight and water intake, corresponds to 7.0×10^{-5} (3.5×10^{-5} – 1.3×10^{-4}). The same analysis conducted with age sensitivity factors to account for elevated risk in infants and children yielded a cumulative risk estimate of 2.9×10^{-4} (1.7×10^{-4} – 6.2×10^{-4}). However, epidemiological research has reported associations between adverse health effects and the presence of disinfection byproducts in tap water at concentrations that generally meet applicable national drinking water standards [3]. Multiple studies have reported an increased risk of bladder cancer in association with exposure to drinking water disinfection byproducts [4,5,6], and the risk of other cancers has been suggested [7,8]. Birth defects [9] and miscarriages [10] have also been linked to disinfection byproducts. Toxicity and carcinogenicity of disinfection byproducts are mediated through pathways involving genotoxicity, cell cycle disruption, and oxidative stress [11].

TOTAL TRIHALOMETHANES (TTHMs)

[Tap Water and Trihalomethanes: Flow of Concerns Continues - PMC](#) Now a study by government and academic researchers adds to previous evidence that dermal absorption and inhalation of THMs associated with everyday tap water use can result in significantly higher blood THM concentrations than simply drinking the water does [EHP 113:863–870]. [Associations of public water system trihalomethane exposure during pregnancy with spontaneous preterm birth and the cervicovaginal microbial-immune state - PubMed](#) [Analysis of Cumulative Cancer Risk Associated with Disinfection Byproducts in United States Drinking Water - PMC](#) Toxicological, epidemiological, and mechanistic studies of disinfection byproducts have provided strong evidence for the carcinogenicity of disinfection byproducts.

The concentration of these toxins in tap water is dependent on the bromide concentration in the water supply and the available hydrocarbons – such as from used motor oil contaminating the ground water – when using chlorine and/or chloramine as El Paso does to treat its drinking water