THE IMPACT OF ENVIRONMENTAL FACTORS ON THE DEVELOPMENT OF CHRONIC LIVER DISEASES

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Chronic liver diseases (CLD) represent a complex group of conditions that account for a significant portion of the global health burden, encompassing a clinical spectrum ranging from hepatic fibrosis to cirrhosis and hepatocellular carcinoma. Recent large-scale population-based and epidemiological analyses indicate that CLD ranks among the major causes of mortality worldwide. Globally, liver diseases are responsible for approximately 1.5–2 million deaths annually, constituting nearly 4% of all deaths. These statistics demonstrate that CLD is not only a clinical condition but also a critical public health and environmental health challenge.

Traditional etiological frameworks attribute CLD primarily to viral hepatitis (HBV, HCV), alcohol-associated liver disease, and metabolic factors—particularly metabolic dysfunction–associated steatotic liver disease (NAFLD/MASLD). However, scientific literature from the past decade increasingly highlights the direct and indirect influence of living conditions and environmental exposures—including air pollution, drinking-water quality, agrochemical contamination (pesticides and heavy metals), industrial emissions, and urbanization processes—on hepatic biology. These environmental determinants may exacerbate oxidative stress within hepatocytes, activate inflammatory signaling pathways, disrupt metabolic activity, and impair detoxification enzymes, thereby contributing to chronic liver injury and disease progression.

Emerging evidence shows a strong association between long-term exposure to fine particulate air pollution (PM2.5) and traffic-related gases (NO_2 and others) with the development of NAFLD and liver fibrosis. Numerous cohort and cross-sectional studies report statistically significant correlations between rising PM2.5 concentrations and increased liver enzyme levels (ALT, AST, GGT), alongside heightened risks of steatosis and fibrosis. Meta-analytic findings suggest that each 10 $\mu g/m^3$ increase in PM2.5 is associated with elevated CLD risk, highlighting the synergistic interplay between environmental exposure and metabolic/ gastroenterological pathways.

Heavy metals such as cadmium, lead, and mercury also exert adverse effects on liver health. These metals disrupt hepatic detoxification mechanisms and antioxidant defense systems, leading to lipid peroxidation, mitochondrial dysfunction, and chronic inflammation—processes that cumulatively drive fibrogenesis and chronic liver damage. Large-scale

observational studies and meta-analyses corroborate the link between heavy-metal exposure, elevated liver biomarkers, and worsening liver pathology.

Exposure to pesticides and organic pollutants (organochlorine, organophosphate compounds, etc.) likewise affects hepatocellular enzymatic systems. In certain agroecological regions, pesticide exposure has been linked to increased risks of NAFLD and other hepatobiliary disorders—particularly among agricultural workers and populations indirectly exposed through the food chain.

Urbanization, socioeconomic status, and changes in living-environment quality further influence the epidemiology of CLD. Lifestyle factors associated with urban living—unhealthy dietary habits, low physical activity, chronic stress, and increased alcohol consumption—create an environment conducive to NAFLD onset and progression. Data from the Global Burden of Disease and national reports demonstrate rising trends in metabolic liver diseases among urban populations, providing a clear example of the synergy between environmental and behavioral determinants.

This growing body of evidence collectively underscores the need to consider ecological exposures as integral determinants in the development and progression of chronic liver diseases.

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