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Cytogenetic damage in populations with methylmercury exposure from fish consumption of Colombian Caribbean

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Mercury (Hg) is a recognized environmental contaminant since the Minamata disaster in the late 1950s. Hg contamination is directly related to mining and gold mining is responsible for most Hg pollution in developing countries. Chronic exposure to relatively low doses of Hg, especially by fish consumption, seems to activate several mechanisms that, potentially, lead to carcinogenic and/or teratogenic processes. Despite its well-known neurotoxicity and teratogenicity, the genotoxic effects of mercury on humans are not completely defined yet. In the present study, to assess whether dietary exposure to methylmercury (MeHg) leads to an increase in cytogenetic damage, human peripheral lymphocytes cells were analyzed using the cytokinesis-block micronucleus cytome assay (CBMN-cyt) parameters in populations with contaminated fish intake. Additionally, to elucidate the mechanism of micronucleus formation, an anti-kinetochore antibody (CREST staining) was used to distinguish CREST+ MN from those CREST-. Correlation between cytogenetic damage and consumption of some fish species known to be especially contaminated by MeHg was also assessed. The study population comprised 112 healthy subjects, 39 residents in the municipality of Cotorra considered as non-exposed and 73 residents of different areas around gold mining zones with evidenced Hg polluted water bodies. Data showed a significant increase (p-value≤0.05) in micronuclei (5.54±3.98), nucleoplasmic bridges (1.11±3.96) necrotic (46.66±26.30) and apoptotic (27.42±20.56) frequencies in binucleated cells of individuals with dietary exposure to MeHg compared to non-exposed individuals. Spearman correlation analysis showed a significant association between DNA damage frequency and fish intake (g/week) in exposed populations. No statistically significant increase in CREST+ micronuclei was detected in exposed individuals (18.42%) compared to the unexposed population (20.91%), supporting the notion that in vivo MeHg exposure has clastogenic effect causing chromatic breaks.

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