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The use of biomarkers to assess aquatic pollution by widespread agents (detergents – SDS; pesticides – chlorfenvinphos): Feasibility, responsiveness and biological consequences in fish

B.S. Nunes[1,*], S. Rodrigues[2], S.C. Antunes[3], B.B. Castro[3], F. Gonçalves[4]

[1] Faculdade De Ciências Da Saúde, Universidade Fernando Pessoa, Porto, Portugal, [2] Departamento De Biologia, CESAM, Aveiro, Portugal, [3] Department of Biology and Cesam, University of Aveiro, Aveiro, Portugal, [4] Department of Biology and Cesam, Universidade de Aveiro, Aveiro, Portugal

Detergents and pesticides are nowadays-widespread chemicals that exert non-selective toxicity in a large number of wild organisms. However, the effects of these two classes of environmental pollutants is related to death (in extreme concentrations, not likely to occur in the majority of scenarios) or slight physiological effects, that do not impair major key aspects, such as inhibition of cholinesterases (in the case of organophosphate pesticides, such as chlorvenfinvos) and consequently survival, and that are more likely to remain unnoticed. To assess the diffuse effects of extremely diluted, albeit common, compounds from these two classes, we selected several enzymatic endpoints, such as the activities of catalase, glutathione-S-transferase, and lactate dehydrogenase in tissues of the freshwater fish *Lepomis gibbosus*. These biomarkers can give insights into significant and generic process, such as metabolism, energetic balance, and oxidative stress, and work as integrative responses reflecting the exposure to diffuse pollution, that exert toxic effects at several levels (detoxification, redox mechanisms, anaerobiosis vs aerobiosis). Our results point to a generic absence of response of the selected biomarkers, despite a significant, eventually hormetic, effect in the activity of liver catalase following exposure to the detergent. However, our results raise the questions: can generic biomarkers be applied to real scenarios of contamination, in which levels or concentrations are not likely to exert severe impairments of target mechanisms? Which is the role of compensatory physiological mechanisms that prevent us from quantifying generic responses for in vivo exposures?

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Type of acetylation as susceptibility biomarker of imidacloprid, dimethoat and sodium nitrate exposure

O. Korotun*, L. Vlasyk

Department of Medical and Ecological Problems, L.I. Medved's Institute of Ecohygiene and Toxicology, Chernivtsi, Ukraine

The individual susceptibility to chemical agents could be caused by polymorphisms in biotransformation enzymes such as transferases. The purpose of this study was to evaluate in vivo the association of N-acetyltransferase activity with susceptibility to pesticides (imidacloprid and dimethoat) and sodium nitrate exposure. White male rats were divided into two groups: animals with high (the "rapid" acetylation type) and low (the "slow" acetylation type) activity of N-acetyltransferase using the loading test with amidopyrine. The rats were given toxicants in threshold doses over a period of 28 days. The level of the animals' health was assessed using more than 20 integral and biochemical indices. We discovered that individuals with "rapid" acetylation type were more susceptible to exposure to these toxicants. Specifically they had greater changes of the integral indices (behavioral responses, body weight) and more profound changes in specific markers such as methemoglobin level in the case of sodium nitrate intoxication and the inhibition of acetylholynesterase in blood for dimethoat exposure. We also observed the signs of oxidative stress development such as the inhibition of antioxidant protective enzymes and an increase in the products level of lipid and proteins peroxidation in blood and hepar in rats with "rapid" type of acetylation. In contrast, in rats with the "slow" acetylation type, the activity of antioxidative enzymes increased, while the level of peroxidation products did not change. We concluded this was an adaptive response. Therefore, the "rapid" type of acetylation is a susceptibility biomarker of imidacloprid, dimethoat and sodium nitrate subacute exposure.

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Asymmetric dimethylarginine (ADMA) as an early cardiovascular biomarker in a children population exposed to inorganic arsenic

C. Osorio-Yañez[1,*], J.C. Ayllon-Vergara[2], E. Hernandez-Castellanos[1], L. Arreola-Mendoza[3], E.M. Melgar-Paniagua[1], A. De Vizcaya-Ruiz[1], G. Aguilar-Madrid[4], L.M. Del Razo[1]

[1] Toxicology, Cinvestav, Distrito Federal, Mexico, [2] Cardiology, Español Hospital, Distrito federal, Mexico, [3] Biosciences and Engineering, CIIEMAD-IPN, Distrito Federal, Mexico, [4] Occupational Health Research, IMSS, Distrito Federal, Mexico

Epidemiological studies have shown that chronic arsenic exposure accelerates the atherosclerosis process in the absence of traditional coronary risk factors even at pediatric stages. Asymmetric dimethylarginine (ADMA) has been used as an early atherosclerosis biomarker in populations for future cardiovascular risk and it has been associated with carotid intima-media thickness (cIMT). cIMT is a well established marker for early, preclinical atherosclerosis. The aim of this study was to examine the serum concentration of ADMA, endothelial dysfunction biomarkers (sICAM-1, sVCAM-1) and cIMT in children exposed to arsenic thought drinking water. A cross-sectional study was conducted with 195 children (3–14 years old) residents of central Mexico with historically high arsenic levels in water supplies. Concentrations of total arsenic (tAs) species in urine, inorganic arsenic, monomethyl arsenic and, dimethyl arsenic, were 3–360 μg/L. Multiple linear regression analysis revealed that ADMA concentrations, atherogenic factor, and urinary tAs were the main markers associated with cIMT increase ($R^2 = 0.1443$; $p = 0.0015$). In addition, sVCAM-1, triglycerides, and relative proportion of inorganic arsenic associated with ADMA ($R^2 = 0.2015$; $p = 0.0012$). To our knowledge, this is the first epidemiological study that has established a relationship between ADMA and arsenic exposure. Our results suggest that ADMA could be implicated in the early atherosclerosis stages by arsenic exposure in children populations. The detection of early cardiovascular biomarkers in children populations may allow the application of intervention measures to prevent clinic manifestation in adulthood.

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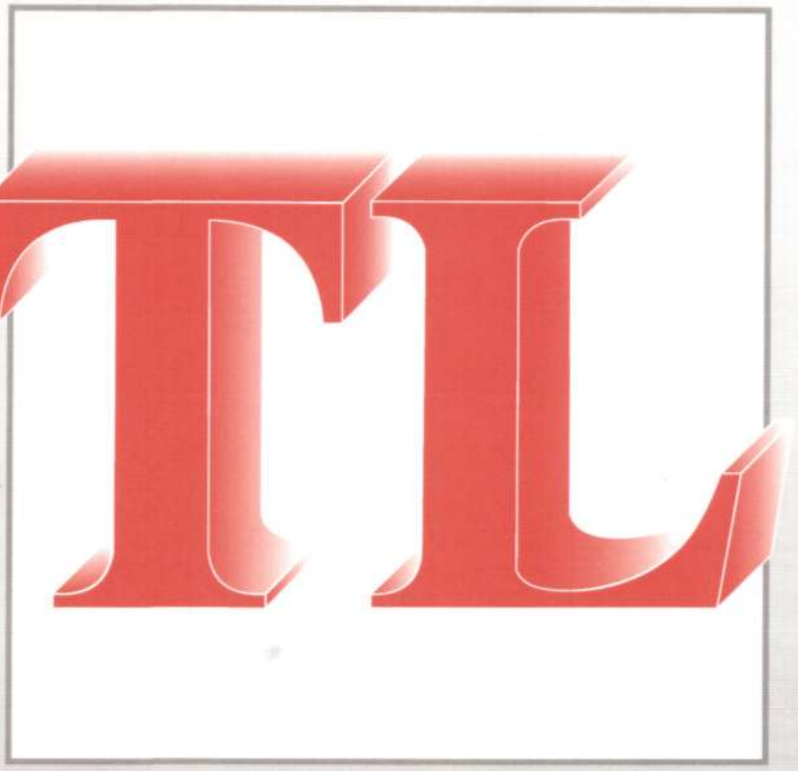

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