INTERACTION BETWEEN PESTICIDE-METABOLIZING GENES AND PESTICIDE EXPOSURE ON BLOOD ANTIOXIDANT ENZYMES

Antonio F. Hernández, Fernando Gil, Antonio Pla Department of Legal Medicine and Toxicology, University of Granada School of Medicine (Spain),

Miguel Rodriguez-Barranco Andalusian School of Public Health, Granada (Spain)

Marina Lacasaña Andalusian School of Public Health, Granada (Spain); Spanish Consortium for Research on Epidemiology and Public Health (CIBERESP),

Tesifón Parrón Council of Health at Almeria province (Spain).

Background and aims. Pesticide-induced oxidative stress may be a possible mechanism of toxicity involved in certain health effects. This study evaluates the potential involvement of pesticide exposure on oxidative damage by assessing levels of erythrocyte antioxidant enzymes and serum uric acid in a population of workers occupationally exposed to pesticides.

Methods. A longitudinal study was conducted on a population of intensive agriculture workers from Andalusia (South Spain), during two periods of high and low-intensity levels of pesticide application. A structured questionnaire containing questions on sociodemographic and occupational characteristics was completed by workers. Blood samples were taken for the measurement of biomarkers of exposure (serum and erythrocyte cholinesterases), susceptibility (Paraxonase-1 – PON1-) and effect (erythrocyte antioxidant enzymes, including superoxide dismutase, catalase, glutathione peroxidase, glutathione reductase, glucose-6-phosphate dehydrogenase) and serum uric acid. Interaction effects between cholinesterases and PON-1 polymorphisms on biomarkers of oxidative stress were performed by generalized estimating equations (GEE) models.

Results. Lower levels of serum cholinesterase (indicating short-term exposure to pesticides) were associated with decreased glutathione reductase, glucose-6-phosphate dehydrogenase and uric acid and with raised catalase and glutathione peroxidase levels. By contrast, cumulated pesticide exposure, as measured by a reduced erythrocyte cholinesterase, was linked to a decreased catalase activity. PON1-192 genotype was associated with catalase and to a lesser extent with uric acid. A gene-environment interaction was found between PON1-192 polymorphism and short-term exposure to pesticides on antioxidant enzymes, because reduced serum cholinesterase was associated with glutathione peroxidase, glutathione reductase and uric acid, especially in carriers of the PON1 102QR genotype.

Conclusions. Pesticide exposure appears to induce oxidative stress particularly after short-term exposure. These results suggest an association between pesticide exposure and enzymatic antioxidant defenses in individuals with certain PON1 polymorphisms, raising the possibility of a genetic risk factor for developing certain oxidative stress-induced degenerative diseases upon exposure to pesticides.