TIME SEQUENCE OF OXIDATIVE STRESS IN NEURODEGENERATIVE BRAIN AFTER LONG-TERM LEAD EXPOSURE IN RATS

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Introduction A large number of studies have shown that the developmental neurotoxicity induced by lead is related to oxidative injury, meanwhile, oxidative stress is among the most common mechanisms of neurodegeneration. However, few studies have explored the role of oxidative stress in age-related cognitive impairment caused by prolonged lead exposure and oxidative stress.

Methods In the present study, rats were exposed to low-level lead from the embryonic stage to old age. Dynamic changes in neurodegeneration, endoplasmic reticulum (ER) stress, and oxidative stress in brains during postnatal weeks 3, 41 and 70 (PNW3, PNW41 and PNW70, respectively) were investigated.

Results Lead exposure resulted in neurodegeneration in PNW70 rats based on magnetic resonance imaging (MRI) scans and thionin staining analysis. Amyloid precursor protein (APP) and tau mRNA expression in PNW41 and PNW70 brains increased in a time- and dose-dependent manner. APP and Tau protein levels significantly increased with lead exposure at PNW3 and PNW70. Mechanistically, the expression of the ER stress protein glucose-regulated protein 78 (GRP78) was higher in the presence of lead than in normal controls, which was associated with high levels of 8-hydroxy-2'-deoxyguanosine (8-OHdG) in brain tissues after lead exposure in PNW3 and PNW70, their changes were like APP and tau protein that were a u- or j-shaped curve with time of lead exposure.

Conclusion Our findings suggest that the neurodegenerative injuries induced by lead exposure may be mediated by ER and oxidative stresses, and there is a critical period for prevention or intervention AD in early life and later life, however middle-aged people at the latent stage of neurodegenerative process should not be ignored.

THE ASSOCIATION OF BLOOD LEAD LEVEL AND RENAL EFFECTS MAY BE MODIFIED BY METALLOTHIONEIN 1A2 POLYMORPHISMS

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Introduction Lead toxicity plays an important role in public health. It causes multiple organs damage, and nephrotoxicity is included. Metallothionein (MT) is a cysteine-rich, low molecular weight protein with function of heavy metal detoxification. However, study about how the MT1A and MT2A single nucleotide polymorphisms (SNPs) influence the lead nephropathy is relatively scarce. Our aim is to investigate the association of blood lead levels and renal biomarkers in chronic lead exposure, and to study whether the association was influenced by MT1A2A SNPs.

Methods Blood samples were collected from 485 participants during their annual health examination after informed consent letters were obtained. The blood lead level, urinary creatinine, urinary uric acid, and urinary N-acetyl-beta-d-glucosaminidase (NAG) were measured and analysed. DNA was extracted and used for real-time PCR genotyping two MT1A SNPs.
BONE EFFECT UNDER CAUSED BY CO-EXPOSURE TO BONE METABOLISM ABNORMALITY AND RENAL Dysfunction IN CADMIUM EXPOSED FARMER FROM THAILAND

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Introduction Paddy fields in Mae Sot, Tak province of Thailand are polluted with cadmium (Cd) over safe level. Farmers exposed Cd from contaminated rice consumption and the high prevalence of kidney dysfunction was shown in the exposed population. Bone fracture is another Cd related pathology which shown in elderly with high exposure level. The identification of high risk individuals will be advantageous to provide suitable health promotion and to prevent severe pathology. This study was done to compare bone fracture risk between glomerular dysfunction, proximal tubular dysfunction and Calcium (Ca) handling abnormality.

Methods Serum osteocalcin and cross-linked N-telopeptide of type I collagen to detect bone metabolism abnormalities, whereas glomerular filtration rate, serum cystatin C, urinary β2-microglobulin (β2-MG) and fractional excretion of calcium

LOW LEVEL URINE ARSENIC CONCENTRATION AND OBSTRUCTIVE PULMONARY DISEASE AMONG U.S. WORKERS

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Introduction Arsenic (As) is a known carcinogen commonly found in drinking water. An emerging body of evidence suggests that exposure to inorganic arsenic may be associated with non-malignant respiratory disease. The aim of this study was to determine whether there is an association between As exposure at levels seen among workers in the United States and prevalence of asthma, emphysema, chronic bronchitis, and respiratory symptoms.

Methods Urinary As was collected from 5365 participants from the combined 2003–2006 National Health and Nutrition Examination Survey (NHANES) cohorts. Two methods to adjust for organic As component were incorporated into the statistical model. Linear and logistic regression models compared urinary As adjusted for organic As with diagnoses of obstructive pulmonary disease and respiratory symptoms.

Results Geometric mean concentration of urinary As were not significantly different between participants with and those without asthma, chronic bronchitis, and emphysema. Odds of having asthma was 0.71 for participants with the highest quintile of urinary As (≥17.23 µg/dl) when compared to the lowest quintile (≤5.52 µg/dl). A significant association was found between increasing urinary As concentration and decreasing age, male gender, and non-white race.

Conclusion A significant association between urinary As and obstructive pulmonary disease and symptoms was not demonstrated in the U.S. working population