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Indicators of repeated oral exposure to lead combined with Cadmium in non-lactating ewes

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ead (Pb) and Cadmium (Cd) pollution co-exists and humans and animals may be co-exposed to both toxics. These heavy Lead (Pb) and Cadmium (Cd) pollution co-exists and numans and annual may be a set of the simulate a repeated low oral exposure and to highlight the toxic effects after lead and lead-Cadmium repeated oral exposure for nine weeks in ewes. An experiment was conducted using "Ouled Djellal" ewes during two periods: Before exposure, where ewes are considered as controls and during exposure 10 ewes were randomly divided in two groups of five; the lead group received lead nitrate at 2.5 mg Pb/kg/day and the lead-Cadmium group received lead nitrate at 2.5 mg Pb/kg/day + Cadmium chloride at 2 mg Cd/kg/day orally for 63 days. Both groups were tested for their blood lead levels and hematological and biochemical parameters before and after receiving the treatment. Before exposure, blood lead levels were below the detection limit of 4 µg/l. Blood levels of lead during 9 weeks of exposure varied from  $135\pm57 \mu g/l$  to  $356\pm147 \mu g/l$  for the lead group and from  $192\pm75 \mu g/l$  to  $445\pm294$ µg/l for the co-exposed group. Mean blood lead levels of lead-Cadmium group were more elevated than the ones of the lead group. The transaminases (ALT, AST) and total proteins are high for the Pb-Cd group during the two last weeks of exposure. The ratio albumin/globulin is low. The rates of hematocrit and hemoglobin decreased for the Pb-Cd group to reach a value of 24% and 7.9±0.6 mg/100ml, respectively. The co-administration of Pb and Cd resulted in a significant reduction in zinc and copper plasma contents and the estimation of toxicokinetic parameters (AUC, Vs, Tmax, Cmax) revealed a greater systemic exposure. Concentrations of lead and Cadmium were determined in organs. Histopathologic lesions occurred in liver and kidney. ANOVA was used for statistical analysis.

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