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Oral lead acetate elicits multi-axis testicular toxicity with incomplete post-exposure recovery in male wistar rats

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Despite established knowledge of lead's adverse effects on male reproduction, the extent and persistence of testicular injury following oral exposure to lead acetate (PbAc), a common environmental and occupational contaminant, remain incompletely characterized. A systematic investigation is required to understand the simultaneous impact on endocrine function, oxidative balance, inflammatory status, genomic integrity, and apoptotic pathways, and critically, to determine whether such damage is reversible upon exposure cessation. This study investigated testicular toxicity induced by oral lead acetate (PbAc) focusing on injury, inflammation, oxidative stress, DNA damage, apoptosis, and endocrine disruption in male Wistar rats.Thirty rats were randomized (n=10/group): control (distilled water), PbAc (60 mg/kg, 28 days), and recovery (PbAc 60 mg/kg for 28 days followed by 28 days distilled water). Endpoints included follicle stimulating hormone (FSH), luteinizing hormone (LH), and testosterone; superoxide dismutase (SOD) activity and malondialdehyde (MDA) for oxidative status; 8-hydroxy-2′-deoxyguanosine (8-OHdG) for genotoxicity; tumor necrosis factor-alpha (TNF-α) for inflammation; B-cell lymphoma 2 (Bcl-2) and caspase-3 for apoptosis; plus, testicular and epididymal histology. One-way analysis of variance (ANOVA) with Tukey's post hoc test determined significance ($P<0.05$). PbAc caused endocrine disruption (reduced FSH, LH, testosterone), oxidative imbalance (decreased SOD, increased MDA), increased 8-OHdG and TNF-α, pro-apoptotic signaling (decreased Bcl-2, increased caspase-3), and histological injury. Key finding: after 28 days without further exposure, several toxic effects persisted (elevated MDA and 8-OHdG; depressed gonadotropins; altered Bcl-2), indicating incomplete spontaneous recovery. Oral PbAc elicits multi-axis testicular toxicity that does not fully resolve after exposure cessation, underscoring the need for preventive and therapeutic strategies.

Keywords: Lead acetate, testicular toxicity, oxidative stress, apoptosis, endocrine disruption, post-exposure recovery