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ABSTRACT BOOK

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Ultrastructural Alterations of Liver Sinusoidal Endothelial Cells Following Chronic Monosodium Methylarsonate Exposure in Rats

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ABSTRACT

Introduction: Monosodium methylarsonate (MSMA) is an organic arsenical herbicide still used in several developing regions despite limited toxicity data. While most arsenic research focuses on hepatocyte injury, the effects of MSMA on liver sinusoidal endothelial cells (LSEC), a specialised, fenestrated endothelium increasingly recognised for its involvement in early liver injury and toxicant-induced hepatic pathogenesis remain poorly understood. This study aimed to characterise ultrastructural changes in LSEC following chronic MSMA exposure. **Materials and method:** Twenty-eight male Sprague Dawley rats were assigned to either control or MSMA-treated groups (63.20 mg/kg/day) for 2 or 6 months (n = 7/group). Livers were perfusion-fixed and examined using scanning electron microscopy (SEM) and transmission electron microscopy (TEM). LSEC fenestrations, sinusoidal morphology, and space of Disse features were evaluated descriptively. **Results:** SEM of control animals showed preserved hepatocyte plates, intact sinusoids, and abundant LSEC fenestrae arranged in sieve plates. MSMA-exposed rats demonstrated reduced fenestral groups at 2 months and more pronounced defenestration with gap formation at 6 months. TEM findings supported these observations, showing fewer hepatocyte microvilli within the space of Disse, loss of the typical attenuated LSEC cytoplasm, chromatin condensation, and occasional caveolae formation. The space of Disse became indistinct in exposed groups, suggesting impaired hepatocyte–sinusoid exchange. **Conclusion:** Chronic MSMA exposure induces LSEC defenestration and ultrastructural changes consistent with early capillarisation, which may represent initial pathogenic events in MSMA-related liver toxicity. Further quantitative fenestration analyses and molecular studies are warranted to elucidate mechanisms of LSEC injury and explore their potential as therapeutic or preventive targets.

Keywords: Defenestration; electron microscopy; liver sinusoidal endothelial cells; monosodium methylarsonate; ultrastructure