



Polystyrene-Induced Renal Oxidative Damage and Inflammation Mediated by PPAR γ , IL-17, and MAPK8IP3 in Mice

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Abstract

Polystyrene-microplastics (PS-MPs) are pervasive environmental contaminants found in common household items, and their ingestion with dietary intake and water is linked to adverse health effects. Following ingestion, PS-MPs can translocate into the circulatory system and accumulate in tissues including kidney. Twelve 6-week-old male C57BL/6 mice were divided into a control group (n = 6) and a PS-MPs group (n = 6) administered 1 mg/kg/day of PS-MPs via oral gavage. Kidney tissue was subjected to histological analysis and thorough transcriptome profiling to identify dysregulated pathways. Histological examination revealed renal atrophy, hemorrhage, thinning of Bowman's capsules, and increased Bowman's capsules space in the PS-MPs group. TEM confirmed severe ultrastructural damage, including nuclear deformation, cytoplasmic vacuolization, and mitochondrial cristae disruption. Transcriptome analysis showed significant enrichment in the PPAR, MAPK, and inflammatory response signaling pathways. Key dysregulated genes included PPARG, ACADL, and SCP2 (PPAR pathway); MAPK8IP3, MAPK11, and FGFR1 (MAPK pathway); and IL-17, RELA, and TLR1 (inflammatory response). This study aimed to investigate the molecular mechanisms underlying PS-MPs-induced kidney injury in a mouse model, with a focus on transcriptomic changes in inflammatory and oxidative stress pathways. In male C57BL/6 mice, PS-MPs exposure induces significant kidney damage at histological, ultrastructural, and molecular levels, dysregulating genes critical to oxidative stress, inflammation, and identifies PPAR γ , IL-17 and MAPK8IP3 as pivotal mediators in PS-MPs-induced nephrotoxicity.

Key Words: Polystyrene-microplastics; PPAR γ ; IL-17; MAPK8IP3; nephrotoxicity

