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Unveiling the Impact of Particulate Matter on the Exacerbation of IgA Nephropathy: An Animal Experimental Study

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Objectives : Particulate matter (PM) exposure is linked to various diseases, including chronic kidney disease (CKD), yet its role in exacerbating IgA nephropathy (IgAN) remains unclear. This study investigates whether PM exposure aggravates IgAN using HIGA mice, a spontaneous IgAN animal model.
Methods : BALB/c mice were used as controls and HIGA mice as the disease model. Based on PM exposure, groups were classified as BALB/c-non exposure (NE), BALB/c-exposure (E), HIGA-NE, and HIGA-E. PM exposure was conducted in a uniquely designed sealed cage system for 12 weeks, 5 days per week, 1 hour per day. Blood, lung, and kidney pathology were analyzed, along with differential gene expression in splenic B cells. Toll-like receptor 9 (TLR-9) expression in lung tissue, aberrantly glycosylated IgA and A Proliferation-Inducing Ligand (APRIL) levels in blood, and mesangial IgA deposition with kidney pathology were assessed. Differentially expressed genes in splenic B cells were identified through RNA sequencing, followed by functional enrichment analysis.
Results : PM exposure significantly increased TLR-9 expression in lung tissues (BALB/c-E and HIGA-E groups), particularly around pigment-laden macrophages, where the pigment is presumed to originate from PM (Figure 1). The HIGA-E group exhibited a higher proportion of aberrantly glycosylated IgA along with increased APRIL levels. Kidney histopathology showed aggravated mesangial expansion, IgA deposition, and glomerular injury in the HIGA-E group (Figure 2). Transcriptomic analysis of splenic B cells revealed greater immune-related gene expression changes in HIGA than in BALB/c mice following PM exposure, indicating heightened genetic susceptibility to PM-induced immune dysregulation.
Conclusions : This study provides the first experimental evidence that PM exposure exacerbates IgAN through TLR-9-mediated immune dysregulation, aberrant IgA glycosylation, and increased mesangial IgA deposition. These findings highlight the interplay between environmental factors and genetic predisposition in IgAN progression and underscore the potential benefits of mitigating PM exposure.

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Beyond Challenges, Towards Healthier Kidney

Figure 1. Comparison of ELISA and pathology results for TLR-9 expression in lung tissues

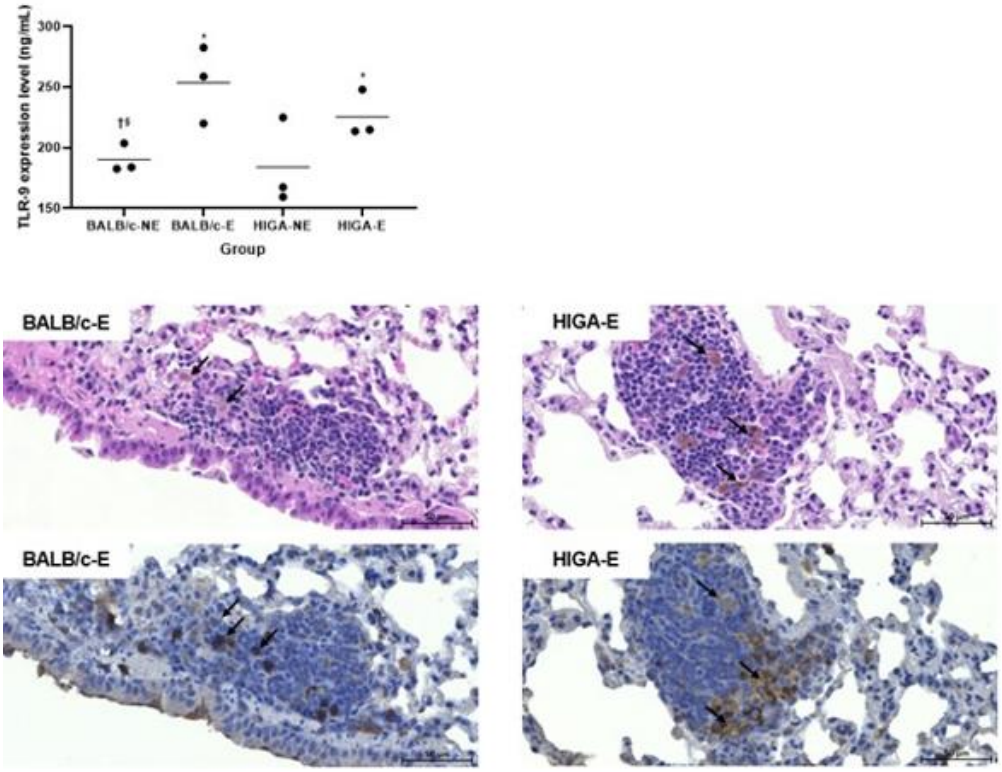

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Figure 2. Comparison of PAS stain, renal injury score, and IgA IHC results in kidney tissues