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Cell-Type Specific Activation of Mitogen-Activated Protein Kinases in PAN-Induced Progressive Renal Disease in Rats

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We examined the time-course activation and the cell-type specific role of MAP kinases in puromycin aminonucleoside (PAN)-induced renal disease. The maximal activation of c-Jun-NH2-terminal kinase (JNK), extracellular signal regulated kinase (ERK) and p38 MAP kinase were detected on Day 52, 38 and 38 after PAN-treatment, respectively. p-JNK was localized in mesangial and proximal tubular cells at the early renal injury. They were expressed, therefore, in the inflammatory cells of tubulointerstitial lesions. While, p-ERK was markedly increased in the glomerular regions and macrophages. p-p38 was observed in glomerular endothelial cells, tubular cells and some inflammatory cells. The results show that the activation of MAP kinases in the early renal injury by PAN-treatment involves cellular changes such as cell proliferation or apoptosis in renal native cells. The activation of MAP kinases in infiltrated inflammatory cells and fibrotic cells plays an important role in destructive events such as glomerulosclerosis and tubulointerstitial fibrosis.

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