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Editorial Note

Editorial Note for Role of Interleukin-6 in Diabetic Nephropathy

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Interleukin-6 (IL-6) was originally referred by diverse names as interferons (Weissenbach et al., 1980; Zilberstein et al., 1986), 26K factor (Content et al., 1982; Haegeman et al., 1986), B-cell stimulatory factor 2 (Hirano et al., 1985), hybridoma growth factor (van Snick et al., 1986; Brakenhoff et al., 1987), plasmacytoma growth factor (Nordan et al., 1987), hepatocytestimulatory factor (Gauldie et al., 1987), a hematopoietic factor (Ikebuchi et al., 1987), and cytotoxic T-cell differentiation factor (Takai et al., 1988)-each name reflecting a different biological activity controlled by the same protein. It is now clear that IL-6 plays a central role in diverse host defense mechanisms such as the immune response, hematopoiesis, and acute-phase reactions.While IL-6 appears to have little to do with the day-to-day “housekeeping” functions of the body, along with other cytokines it represents an important frontline component of the body’s armory against infection or tissue damage. Due to its property of functional pleiotropy, IL-6 is responsible for the pathology of many diseases like multiple myeloma, rheumatoid arthritis, Castleman’s disease, AIDS, mesangial proliferative glomerulonephritis, psoriasis, Kaposi’s sarcoma, sepsis and osteoporosis. IL-6 gene is located at chromosome 7p21 and chromosome 5 in humans and rats respectively. The IL-6 gene of humans, rat and mouse were cloned and sequenced. The sequencing conveyed the information that in the entire gene contained four introns and 5 exons. Within the protein coding region the positions of exon/intron boundaries, exon lengths, and location of cysteine residues within exons are conserved across species. Differences occur at the 5' boundary of exon 1 and the 3' boundary of exon 5, which lie outside the coding region.). Amongst species there is considerable amino acid sequence identity in the central portion of the molecule (residues 40-100) and the region close to the C-terminus (residues165-184).

Inflammatory response plays an important role in the development of pathogenesis of Diabetic nephropathy, which was first time demonstrated by Hasegawa, et.al. sekizuka ,et.al.gave a suggestion that pro-inflammatory cytokines like IL-1, IL-6 and TNF-alpha may a play a role in the pathology of DN. Various results from experiments and human studies suggest that IL-6 signaling pathways are involved in the progression of DN. For example: Diabetic patients with DN have high serum IL-6 levels when compared to diabetic patients without DN. Type 2 diabetic patients with polymorphism in IL-6 gene show increased risk for development of DN. Increased mesangialexpansion, thickening of GBM are the characteristic stru ctural changes in the DN. Where mesangial expansion correlates with the glomerulosclerosis. Recent studies suggest that IL-6 has effects on mesangial proliferative glomerulonephritis. Il-6 is a multifactorial cytokine that is produced by variety of cells including monocytes, lymphocytes, epithellial cells, fibroblasts and renal mesangial cells. IL-6 is associated with renal fibrosis as it is capable of stimulating fibroblasts to produce extra-cellular matrix and the inhibitor of metalloproteinase (capable of degenerating ECM). It was reported that level of urinary IL-6 correlates with degree of mesangial expansion. IL-6 can be used as a marker to detect the progressionofDN