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Lipids and Obesity in CKD

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KDIGO has published a Clinical Practice Guideline on management of dyslipidemia in CKD in 2013. The gist of the guideline and the rationale will be recapitulated. More recently the link between obesity and CKD is increasingly recognized to involve chronic systemic low-grade inflammation which can eventually contribute to the development of insulin resistance. In this regard, the role of innate immunity and in particular toll-like receptor 4 (TLR4) is believed to be an important mediator that triggers the innate immune response by activating inflammatory signaling cascades. Human, animal and cell culture studies identified saturated fatty acids (SFAs), the dominant non-esterified fatty acid (NEFA) in the circulation of obese subjects, as non-microbial agonists that trigger the inflammatory response via activating TLR4 signaling. Recent advances in the impact of crosstalk between TLR4 and SFAs on inflammation and insulin resistance in multiple cell types, tissues and organs in the context of metabolic dysregulation will be discussed. In addition, our understanding of lipid metabolism suggests a critical role of endoplasmic reticulum stress in obesity-induced kidney injury. From a therapeutic perspective, the potential value of stem cell treatment in lipotoxic kidney injury will be highlighted using recent data from animal (using high-fat diet fed mice) and cell culture (using palmitic acid as a culprit molecule) models.