Editorial

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Omega-3 fatty acids: promising therapeutic agents for combating kidney injuries

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See "Omega-3 fatty acids upregulate Nrf2 expression and attenuate apoptosis, inflammation, and fibrosis in a rat model of cyclosporine-induced nephropathy" by Ji Young Lee, Young Ki Son, Mi Hwa Lee, Su Mi Lee, Seong Eun Kim, Won Suk An

Cyclosporine (CsA)-induced nephropathy is a debilitating condition characterized by renal dysfunction accompanied by inflammation, apoptosis, fibrosis, and hypoxic injury [1]. Despite extensive research, the influence of omega-3 fatty acids (O-3FA) on nuclear factor erythroid 2-related factor 2 (Nrf2) expression, a key regulator of cellular defense mechanisms, remains unclear [2,3]. However, a recent groundbreaking study sheds light on the potential benefits of O-3FA for attenuating these harmful processes in a rat model of CsA-induced nephropathy [4].

The study divided male Sprague-Dawley rats into three groups: a control group, a group treated with CsA, and a group treated with both CsA and O-3FA. The researchers observed significant kidney function impairment in the CsA-treated rats compared to the control group. Additionally, markers associated with apoptosis, such as caspase-3, caspase-7, and the Bax to Bcl2 ratio, were activated in the CsA-treated group. Remarkably, O-3FA supplementation attenuated these apoptotic activation patterns, indicating its potential anti-apoptotic effects.

Furthermore, the CsA-treated group exhibited increased expression of the inflammatory marker ED-1 and inhibition of the IκB protein. However, O-3FA supplementation effectively mitigated the inflammatory response, as evidenced by the reduced expression of ED-1 and IκB. Furthermore, CsA treatment led to the activation of Smad2/3, Smad4, and transforming growth factor-β1, all associated with renal fibrosis. Nevertheless, O-3FA prevented these activations, highlighting its potential anti-fibrotic properties.

Interestingly, the researchers discovered that Nrf2 expression was decreased in CsA-treated rats, but supplementation with O-3FA significantly increased its expression. This finding suggests that Nrf2 may act as a potential mediator induced by O-3FA supplementation, playing a crucial role in attenuating pro-inflammatory pathways, fibrotic processes, and apoptosis.

The study provides compelling evidence that O-3FA supplementation holds immense promise as a therapeutic intervention in CsA-induced nephropathy. By upregulating Nrf2 expression, O-3FA exhibits notable anti-inflammatory, anti-apoptotic, and anti-fibrotic effects, ultimately protecting the kidneys from damage. However, further investiga-

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tions are necessary to elucidate the intricate crosstalk between Nrf2 expression and the signaling pathways involved in O-3FA treatment.

These findings offer new insights into the potential mechanisms underlying the protective effects of O-3FA against kidney injuries. If translated into clinical practice, O-3FA supplementation could emerge as a valuable adjunct therapy for patients with CsA-induced nephropathy, helping to alleviate their symptoms and enhance renal function. Nonetheless, additional studies are warranted to fully understand the therapeutic potential and optimize the dosing and administration strategies of O-3FA in human subjects.

It would be premature to conclude that the efficacy of O-3FA has been proven based solely on the content of this manuscript. One limitation of this study is that it was conducted on male Sprague-Dawley rats, which may not fully represent the response to O-3FA supplementation in humans. Animal models do not always perfectly reflect human physiology, and there may be species-specific differences in the effects of O-3FA on Nrf2 expression and its associated pathways. Furthermore, the authors acknowledged the need for further studies to elucidate the crosstalk between Nrf2 expression and signals related to O-3FA treatment. This study does not provide a comprehensive understanding of the underlying mechanisms by which O-3FA influences Nrf2 and its downstream effects. Future research is necessary to fully explore and confirm these relationships.

In conclusion, despite some limitations, this study marks a significant advancement in our understanding of the protective effects of O-3FA against CsA-induced nephropathy [5]. With further exploration and clinical validation, O-3FA could become an integral component of the treatment arsenal, offering hope to patients suffering from this challenging kidney condition.

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Conflicts of interest
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