

The Clinical Effects of Nicotinamide Riboside on Inflammatory Parameters

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Objectives: Nicotinamide adenine dinucleotide (NAD⁺) coenzymes are the central regulators of cellular metabolism. While the NAD⁺ system is dysregulated by metabolic stress and age-related conditions, nicotinamide riboside (NR) augments NAD⁺ levels and may contribute to the reduction of inflammation. As advancing age is often accompanied by chronically elevated inflammation (i.e., ‘inflammaging’), targeting chronic inflammation may be a viable strategy to preserve aspects of health and function. The objective of this study was to investigate the clinical evidence of NR’s mitigation of inflammation in humans.

Methods: We systematically evaluated peer-reviewed, published clinical trials of oral NR supplementation that included analysis of inflammatory markers. There were no restrictions for age, sex, health status, or use of other bioactives. Key study details were assessed to compare experimental designs and results.

Results: We identified 5 clinical trials of oral NR supplementation that measured inflammation. There was substantial heterogeneity in inflammatory endpoints evaluated. NR dose and treatment duration ranged from 1–2 g/day and 5-d–6-wks, respectively. Participants varied in age (18–78 years) and health status. In one study, NR was given as part of a nutritional cocktail, while NR was the sole intervention in the remaining studies. Of the 3 studies assessing circulating inflammatory markers, two investigated IL-6 and reported significant reductions in its levels with NR treatment, while effects on MCP-1 and TNF- α were inconsistent. NR significantly lowered gene expression of IL-6 and IL-18, but not NLRP3 or IL-1B in peripheral blood mononuclear cells in one study. Another study documented significant reductions in the gene expression of IFNB and CXCL10, but no change to TNFA expression, in the monocytes of the NR-treated group. Moreover, NR treatment led to lower concentrations of IFN β , but not TNF- α , in monocytes.

Conclusions: Preliminary evidence suggests that NR may improve some inflammatory measures, with potential for more robust effects among populations with compromised NAD⁺ and higher inflammation status, e.g., elderly or diseased. More work is needed to clarify which inflammatory markers are targeted by NR, and the extent to which they are further modified by dose and participant demographics.

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