Opuntia Ficus Indica fruit ameliorates glucose dysmetabolism in a murine model of metabolic syndrome: a comparative study with chromium picolinate.

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Opuntia ficus-indica fruit (OFF) contains relevant amounts of fibres, prebiotics and phytochemicals with anti-oxidative and anti-inflammatory properties as previously published in: Silva MA et al. (2021) Molecules 26, 951. In the light of the self-feeding, vicious cycle between low-grade inflammation, oxidative stress and insulin resistance (IR), we here explored whether and how OFF administration counteracts IR generated after 10 weeks in high-fat diet (HFD) fed mice as previously published in: Terzo S et al. (2022) Antioxidants 11, 80. Due to the limited seasonal availability and short shelf-life of OFF, we employed a lyophilised OFF, given per os at 40 mg/kg/day for 4 weeks, alone or with chromium picolinate (CrPi), one of the most used supplements against IR, at 2.6 µg/kg/day. Our results show that OFF ameliorated HFD-induced IR to the same extent of CrPi, as evaluated by HOMA-IR, glucose and insulin tolerance tests. Furthermore, OFF co-administrated with CrPi does not potentiate its effects on the above-mentioned parameters. From a mechanistic perspective, OFF-mediated anti-dysmetabolic effects were associated with the reduction of hepatic oxidative stress and inflammation. Indeed, OFF treatment inhibited the HFD-induced production of reactive oxygen species and malondialdehyde, evaluated by fluorimetric assays. Coherently, western blotting analysis (WB) revealed an increase of Nfr-2 nuclear translocation and SOD-2 expression levels by OFF. Accordingly, OFF administration counteracted inflammation evaluated, by WB, as NF-kB nuclear translocation and i-NOS and COX-2 expression levels. Relevantly, the OFF dose employed, if extrapolated from mice to humans, could be administrable as food supplement, while that of CrPi is the one contained in multimineral supplements. As a whole, our results show that OFF counteracts IR by modulating the expression of crucial proteins involved in the oxidative stress-dependent, inflammatory reaction underlying the HFD-induced IR.