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Title:
Example given: Adiponectin homolog novel osmotin protects obesity/diabetes-induced NAFLD by upregulating AdipoRs/PPARα signaling in ob/ob and db/db transgenic mouse models

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Abstract:
Background: In metabolic disorders, adiponectin and adiponectin receptors (AdipoR1/R2)
signaling has a key role in improving nonalcoholic fatty liver disease (NAFLD) in obesityassociated diabetes.
Objective: To the best of our knowledge, here, we reported for the first time the underlying
mechanistic therapeutic efficacy of the novel osmotin, a homolog of mammalian adiponectin,
against NAFLD in leptin-deficient ob/ob and db/db mice.
Methods: The ob/ob and db/db mice were treated with osmotin at a dose of 5 μg/g three times a week for two weeks. To co-relate the in vivo results we used the human liver carcinoma HepG2 cells, subjected to knockdown with small siRNAs of AdipoR1/R2 and PPARα genes and treated with osmotin and palmitic acid (P.A.). MTT assay, western blotting,
immunohistofluorescence assays, and plasma biochemical analyses were applied.
Results: Osmotin stimulated AdipoR1/R2 and its downstream
APPL1/PPARα/AMPK/SIRT1 pathways in ob/ob and db/db mice, and HepG2 cells exposed to P.A. Mechanistically, we confirmed that knockdown of AdipoR1/R2 and PPARα by their respective siRNAs abolished the osmotin activity in HepG2 cells exposed to P.A. Overall, the in vivo and in vitro results suggested that osmotin protected against NAFLD through activation of AdipoR1/R2 and its downstream APPL1/PPAR-α/AMPK/SIRT1 pathways as shown by the reduced body weight, blood glucose level and glycated hemoglobin, improved glucose tolerance, attenuated insulin

resistance and hepatic glucogenesis, regulated serum lipid parameters, and increased fatty acid oxidation and mitochondrial functions.

Conclusion: Our findings strongly suggest that novel osmotin might be a potential novel therapeutic tool against obesity/diabetes-induced NAFLD and other metabolic disorders.

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