Silicon intake reduces hypercholesterolemia facilitating reverse cholesterol transport through intestinal activation of LXR/ABC transporters pathway in type 2 diabetic rats

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INTRODUCTION:
❖ Dyslipidemia by increased intestinal cholesterol (Chol) absorption is a risk factor in type 2 Diabetes Mellitus (T2DM).
❖ Intestinal transporters mediate Chol absorption and are an important therapeutic target to reduce hypercholesterolemia.
❖ Silicon intake (Si) has a hypolipemic effect in experimental T2DM models.
❖ Could silicon intake modulate Chol transporters levels in duodenum by lowering hypercholesterolemia in T2DM rats?

METHODS:
T2DM Model

DM-HFD
DM-HFCD-Si

Intraperitoneal injection
Streptozotocin (STZ 65mg/kg b.w.)
Nicotinamide (NAD 225 mg/kg b.w.)

Duodenum dissection

• Plasma lipid profile
• Feces lipid excretion

Immunohistochemistry analysis

RESULTS:

Cholesterol excretion

ABCG5

ABCG8

LXR

D

D-Si

Cholesterol absorption

ABCG5/8

NPC1L1

ACAT2

MTP

LXR

D- T2DM

D-Si T2DM+ Silicon

CONCLUSIONS:
The present study demonstrates that Si consumption might facilitate the cholesterol efflux into feces through upregulating LXR, ABCG5 and ABCG8 expression in duodenum and could be a potentially therapeutic nutritional ingredient for hypercholesterolemia associated to insulin resistance in T2DM treatment.

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