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D-ribose, an overlooked player in type 2 diabetes mellitus?

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Type 2 diabetes mellitus is a metabolic disorder that is characterized by high blood glucose due to either insulin resistance or insulin deficiency [1]. A direct correlation between D-glucose and diabetic complications has long been established, and is the focus of most research in this field. In contrast, D-Ribose has been overlooked so far as a potential risk player in the development of diabetes.

Recently published reports found abnormally high levels of D-ribose in the urine of diabetic patients [2]. Elsewhere, intravenous administration of D-ribose resulted in a decrease in blood D-glucose, concomitantly with an increase in the level of glycated serum proteins [3]. It is known that administration of high dose of D-ribose rapidly glycates proteins *in vivo* and *in vitro* [4–6], which results in the production of high levels of advanced glycation end products. D-glucose, in contrast, cannot be used to the same effect, even under identical conditions [7].

Therefore, as we hypothesized, dysfunction of the metabolism of D-ribose may play a role involving the development of complications of type 2 diabetes mellitus, and it is feasible that D-ribose could be used as a biomarker for type 2 diabetes mellitus. That is, diabetics suffer from dysfunction of energy metabolism, involving not only Dglucose, but also D-ribose. This viewpoint for D-ribose in diabetes is based on these observations: (i) Diabetics display abnormal increases in the level of uric D-ribose; (ii) Raised levels of D-ribose result in increased ribosylation activity, in the result of which more advanced glycation end products accumulate in cells; and (iii) D-ribose could be used as a new biomarker for type 2 diabetes mellitus, as it is easily measured in the urine of diabetic patients.

We suggest that this novel view of type 2 diabetes mellitus deserves further and detailed investigation, both at the level of basic and clinical research, to clarify whether and how D-ribose and its metabolism are related with diabetic complications.

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- Kumar V, Fausto N, Abbas AK. Robbins and Cotran pathologic basis of disease. 7th ed. Philadelphia: Saunders, 2005, 1194–1195
- 2 Su T, Xin L, He YG, Wei Y, Song YX, Li WW, Wang XM, He RQ. The abnormally high level of uric D-ribose for type-2 diabetics. Prog Biochem Biophys, 2013, 40: 816–825
- 3 Han CS, Lu Y, Wei Y, Liu Y, He RQ. D-Ribose induces cellular protein glycation and impairs mouse spatial cognition. PLoS ONE, 2011, 6: e24623
- 4 Wei Y, Chen L, Chen J, Ge L, He RQ. Rapid glycation with D-Ribose induces globular amyloid-like aggregations of BSA with high cytotoxicity to SH-SY5Y cells. BMC Cell Biol 2009, 10:10
- 5 Chen L, Wei Y, Wang XQ, He RQ. Ribosylation rapidly induces α-synuclein to form highly cytotoxic molten globules of advanced glycation end products. PLoS ONE, 2010, 5: e9052
- 6 Chen L, Wei Y, Wang XQ, He RQ. D-Ribosylated Tau forms globular aggregates with high cytotoxicity. Cell Mol Life Sci, 2009, 66: 2559–2571
- 7 Wei Y, Han CS, Zhou J, Liu Y, Chen L, He RQ. D-Ribose in glycation and protein aggregation. Biochim Biophys Acta-GS, 2012, 1820: 488–494

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