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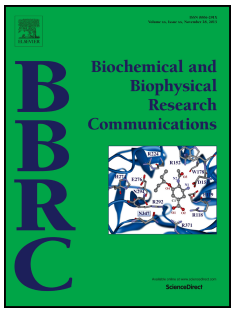

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Amelioration of streptozotocin-induced type 2 diabetes mellitus in Wistar rats by arachidonic acid

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Keywords:

Type 2 diabetes mellitus, streptozotocin, arachidonic acid, lipoxin A4, insulin, Wistar rats, anti-oxidants, lipocaln2, cytokines, glucose.

Abstract

Traditionally arachidonic acid (AA, 20:4 n-6) is considered as a pro-inflammatory molecule since it forms precursor to prostaglandins (PGs), leukotrienes (LTs) and thromboxanes (TXs) that have pro-inflammatory actions. Type 2 diabetes mellitus (type 2 DM) is considered as a low-grade systemic inflammatory condition in which circulating PGs and LTs are increased. Streptozotocin (STZ)-induced type 2 DM is used as a model of human type 2 DM in which peripheral insulin resistance, increased plasma interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) and hyperglycemia occurs. In the present study, we observed that oral supplementation of AA prevented STZ-induced type 2 DM in Wistar rats by restoring hyperglycemia, plasma levels of TNF-α and IL-6; adipose tissue NF-kB and lipocalin 2 (LPCLN2) and pancreatic tissue NF-kB and 5- and 12- lipoxygenase enzymes to normal. AA treatment enhanced insulin sensitivity and plasma lipoxin A4 (LXA4) levels, a potent anti-inflammatory molecule derived from AA. These results are supported by our previous studies wherein it was noted that plasma phospholipid content of AA and circulating LXA4 levels are low in those with type 2 DM. In a preliminary study, we also noted that high-fat-diet (HFD)-induced type 2 DM in Wistar rats can be prevented by oral supplementation of AA. These results suggest AA