Response to: "Decreased Angiogenesis in Diabetes: New Insights into the Mechanisms Involved in the Negative Association Between Diabetes and Abdominal Aortic Aneurysm"

As noted,[1] multiple potential mechanisms suppress abdominal aortic aneurysms (AAAs) in patients with diabetes.[2–4] While aetiologically distinct, both types one and two diabetes produce the chronic hyperglycaemia causal for nearly all related pathologies. Streptozotocin produced rapid and reproducible induction of hyperglycaemia without introducing confounding influences due to the deficient leptin receptor activity or obesity present in spontaneous and diet induced type 2 murine modelling systems, respectively.[5,6]

There is limited access to vital human AAA tissue for pathogenetic analysis in the endovascular era. Available AAA tissue harvested during surgical repair represents end stage or otherwise complicated disease (e.g., infected or otherwise failed endograft explants), generating uncertainty regarding its relevance to candidate medical suppression therapies. Ironically, the reduced prevalence of AAA in patients with diabetes further limits clinical specimen availability, and thus the limitations of this study as described.

Investigations focused on deciphering diabetic AAA suppression hold great promise in identifying and advancing effective medical therapies and adjuvant treatments for this challenging disease.

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