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Funding Source:

University of Missouri Department of Nutrition & Exercise Physiology

Sympathetic Discharge Patterns in Human Type 2 Diabetes

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Objective: Sympathetic nervous system activity is predictive of cardiovascular morbidity and mortality. Muscle sympathetic nerve activity (MSNA) can be directly measured in humans and has been previously shown to be elevated in individuals with type 2 diabetes. Sympathetic neural recordings are often rectified and integrated to give multi-unit MSNA that represents activity of several axons recorded simultaneously. Unfortunately, multi-unit MSNA does not account for firing patterns of single action potentials (AP). AP firing patterns provide unique information about central neural processing and may have implications for end organ responses. With this, we hypothesized individuals with type 2 diabetes would display augmented AP firing patterns compared to non-diabetic controls. We further hypothesized these AP firing patterns would be associated with end organ responses (i.e., blood pressure, BP).

Methods: Heart rate (HR, ECG), arterial BP (finger photoplethysmography), and multi-unit MSNA (microneurography) were measured in 5 non-diabetic adults (59 ± 4 yrs, HbA1c $5.6 \pm 0.1\%$) and 6 adults with type 2 diabetes (51 ± 2 yrs, HbA1c $7.6 \pm 0.7\%$). Sympathetic AP firing patterns were assessed using matched wavelet-based methodology.

Results: No differences in multi-unit MSNA were observed between healthy controls and individuals with type 2 diabetes (26 ± 2 vs 23 ± 3 bursts/min; $p=0.65$). The firing probability of sympathetic APs (167 ± 47 vs 177 ± 57 AP/min; $p=0.80$) and the components of an integrated burst (6 ± 2 vs 7 ± 1 , $p=0.58$ AP/burst; 4 ± 1 vs 4 ± 1 clusters/burst, $p=0.45$) did not differ between control and type 2 diabetes. An association between firing patterns of sympathetic AP and diastolic BP were observed, such that those individuals with greater sympathetic neural discharge (clusters/burst) exhibited higher diastolic BP ($R=0.628$, $p=0.05$).

Conclusions: These data support a role for augmented sympathetic neural firing in the development of higher resting diastolic BP; however, this relationship does not differ between controls and individuals with type 2 diabetes.