TACSM Abstract

Creating a Type I Diabetic Rat with Streptozotocin

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ABSTRACT

The American Heart Association (AHA) recognizes diabetes as a risk factor for developing cardiovascular disease. Type 1 diabetes (T1D) is characterized by absolute insulin deficiency due to the destruction of the insulin-producing pancreatic beta cells. A common model for studying T1D is the streptozotocin (STZ) induced rat. STZ targets and destroys beta cells of the pancreas and therefore causes the rats to develop T1D. **PURPOSE:** The purpose of this project was to describe the procedure for inducing T1D in a in vivo rat model, to study the cardiovascular responses to exercise. **METHODS:** Male and female Sprague Dawley rats were fasted overnight (12 hours) and brought in to the lab the following day for injection. Rats were anesthetized with 5% isoflurane in 100% oxygen for about four minutes. Once rats were unresponsive to mechanical stimulation, they were weighed and placed on a nosecone and ventilated through a nose cone with 3% isoflurane in 100% oxygen. The tail was pricked, and venous blood sampled for measurement of baseline blood glucose (Nova Biomedical) level. While the rat was on the nose cone, STZ (Sigma Aldrich) dosage (50mg/kg) was calculated, based on recorded body weight, and dissolved in 200 µl of citrate buffer. The drug solution was vortexed for 30 sec before being injected intraperitoneally followed by lactated Ringers (5 ml), which was injected subcutaneously to prevent dehydration. The rat was then taken off isoflurane and returned to its cage in a supine position with its head slightly elevated to prevent choking. **RESULTS:** Rats typically developed T1D within 24 to 48 hrs following STZ injection. The rats were considered diabetic when non-fasted blood glucose level was >300 mg/dl. STZ induced T1D rats had an exaggerated blood pressure response to both static contraction and passive stretch compared to healthy controls. **CONCLUSION:** We have shown that inducing rats with T1D by injecting them with STZ resulted in a T1D rat model whose blood pressure response to exercise was exaggerated compared to healthy rats. This model allows for investigations addressing neurovascular control mechanisms during exercise in T1D.