101 Adrenergic Receptor Expression Is Increased in Carotid Smooth Muscle from Severely Burned Rats

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Introduction: Severe burn disrupts cardiovascular function which can lead to critical shock. To counteract cardiovascular collapse, there is a systemic increase of catecholamines released in response to severe burn. Previous studies showed that β1- adrenergic receptor (AR)_ protein expression was significantly increased in the cardiac right ventricles (RV) following burn injury, which is correlated with compromised cardiac dysfunction. Vascular smooth muscle contraction served to modulate blood pressure and improve circulatory perfusion. We hypothesize that ARs expression in major arteries are modified to initiate vascular functional changes following severe burn. In the current study, we report temporal ARs expression in murine carotid artery smooth muscle following severe burn.

Methods: Thirty-four adult Sprague-Dawley male rats received a 40% total body surface area (TBSA) scald burn followed by fluid resuscitation using the Parkland formula. Control animals received a sham burn procedure. Animals were serially euthanized between 6 hours and 14 days after burn and endothelium-intact common carotid arteries were harvested for histological analysis.

Results: Immunohistochemical staining data demonstrated expression of adrenergic receptors (AR) (α1, α2, β1, and β2) were differentially changed in response to injury over time. α1a-AR expression significantly increased within the carotid artery tunica media 7-days after burn ($p< 0.05$). As a negative feedback of inhibitory of norepinephrine signaling, AR-α2a expression did not significantly change. AR-β1 expression also had no change over time after burn. Interestingly, functioning to relax vascular smooth muscle, a significant elevation of β2-AR expression within the carotid artery tunica media was observed only at 1-day after burn ($p< 0.05$).

Conclusions: In summary, immunohistochemistry showed that carotid arterial adrenergic receptor expressions of α1a-AR and β2-AR are significantly altered in response to severe burn, which may contribute to vascular contractility in burn rats.

102 Vaping of Vitamin E Acetate Causes Acute Lung Injury in a Dose-dependent Manner in Sheep

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Introduction: The Centers for Disease Control and Prevention and the Food and Drug Administration have reported an increasing number of clinical cases of pulmonary injury following the use of e-cigarette/vaping products. Although the causative factors for the national outbreak of electronic-cigarette, or vaping product use-associated lung injury (EVALI) has not been established, CDC reported that vitamin E acetate (VEA) is strongly linked to the EVALI outbreak. In this study, we tested the hypothesis that VEA vaping causes acute lung injury in a dose-dependent manner in a sheep model.

Methods: Sheep were surgically prepared under anesthesia and analgesia with multiple vascular catheters (pulmonary arterial, left atrial, and femoral arterial). To assess pulmonary edema, the mediastinal lymph node vessel draining the lung was cannulated. After a 5-day surgical recovery, a tracheostomy tube and urinary catheter were placed. Then, the sheep were placed on a mechanical ventilator and VEA vaping was immediately started in the following groups: (1) vaped with glycerol (n=1); (2) vaped with 0.4mg of VEA (n=2); (3) vaped with 0.6mg of VEA (n=1); (4) vaped with 0.8mg of VEA (n=7); and (5) not injured, not treated (Sham, n=6). Sheep were resuscitated with lactated Ringer's solution (4mL/kg/hr). Sheep in a conscious state was monitored with 4 hrs intervals for cardiopulmonary variables for 48 hrs.

Results: Pulmonary gas exchange, represented by the PaO2/FiO2 ratio, was unchanged in sham, glycerol, and 0.4 mg VEA groups. In the 0.6 VEA group, the PaO2/FiO2 ratio was decreased from 42 to 48 hrs, while in the 0.8 mg VEA group, it was strongly decreased starting at 24 hrs and remained low throughout the remaining period. Lung lymph flow, an index of pulmonary microvascular permeability, was increased more than 2-fold in 0.6 and 0.8 mg VEA groups. Lung compliance also tended to decrease in all VEA groups.

Conclusions: VEA vaping causes acute lung injury in a dose-dependent manner in sheep. Further studies should investigate underlying mechanistic aspects that cause increased microvascular permeability.