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“The Effects of Chronic Nicotine Inhalation through Vaping on the Anticoagulant, Protein S, a Marker of Endothelial Cell Dysfunction, in Mice”

Blood clotting, or coagulation, is an important process that prevents excessive bleeding when a blood vessel is injured. Platelets and proteins in plasma work together to stop the bleeding by forming a clot over the injury. Protein S (PS) is an essential natural anticoagulant and is present in the plasma in free and bound forms. PS deficiency is a major contributor to hereditary or acquired hypercoagulability. Hypercoagulability causes myocardial infarction, stroke, and deep vein thrombosis in millions of individuals. Protein S is associated with endothelial cell dysfunction. Decrease in Protein S and increase in thrombin generation suggest endothelial dysfunction. Chronic nicotine inhalation is also associated with the hardening of arteries, which can lead to cardiovascular disease, heart disease, and conceivably heart attack. According to 2020 statistics, 12.5% of the United States population were confirmed as nicotine users. In addition, 15% of global deaths are attributed to nicotine-induced heart disease. It is also known that cigarette smoking and nicotine use causes a prothrombotic state for its users.

It has been reported that Protein S is significantly downregulated in cigarette smokers. These alterations in Protein S may contribute to the thrombotic complications associated with smoking (1). In this study, we aim to determine whether chronic nicotine inhalation via vaping influenced thrombin generation and overall Protein S concentration in mice.

We measured thrombin generation using automated thrombin generation assay as described (2), and we measured overall Protein S concentration in each sample by immunoblotting. Our initial data indicate that thrombin generation is higher in mice receiving Nick Salts compared to mice receiving air (control) and VGPG (vaping control). Similarly, there is a decrease in Protein S (20% decrease) concentration in the mice receiving Nick Salt compared to the mice receiving air. These preliminary data indicate a downregulation of Protein S and increased thrombin generation in the mice receiving Nick Salt. Therefore, the mice receiving nicotine are at risk of becoming thrombotic.

Currently, work is underway to interrogate whether other endothelial cell markers like Thrombomodulin are affected with chronic use of nicotine in mice.

References:

- 1) B D Scott 1, C T Esmon, P C Comp. The natural anticoagulant protein S is decreased in male smokers. *Am Heart J.*, 1991,122:76-80.
- 2) Chattopadhyay, Rima., Sengupta,Tanusree., and Majumder, Rinku. Inhibition of Intrinsic Xase by Protein S. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 2012, 32:2387-2393.

