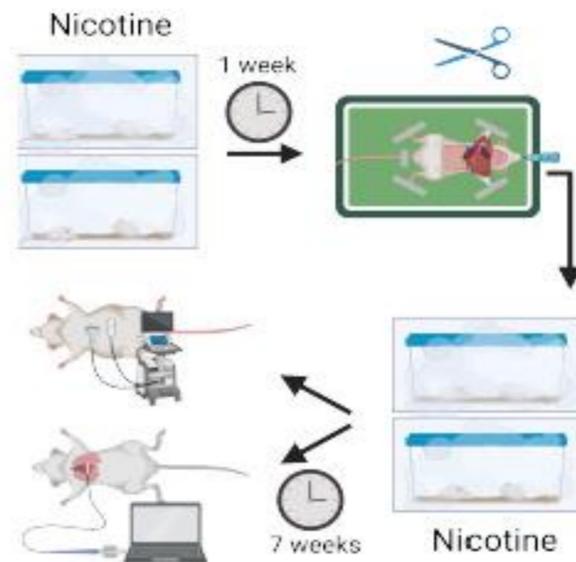


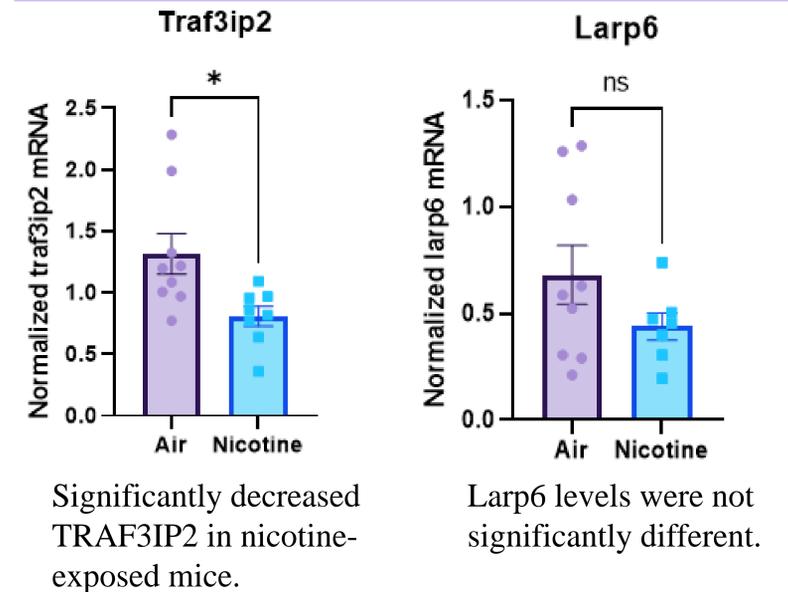
Introduction

Cardiovascular disease (CVD) is the leading cause of premature death in the world, and an independent, associated risk factor of CVD is tobacco use. Globally, the use of electronic cigarettes, containing nicotine, is increasing at an alarming rate. As a driver of cardiac remodeling, fibrosis, and inflammation, nicotine significantly contributes to the development of CVD. To assess the effects of chronic nicotine exposure on myocardial function, fibrosis, and inflammation, male C57BL/6J mice were exposed to room air or nicotine vapor for one week before undergoing a transverse aortic constriction (TAC) or a sham surgery. After, air and sham mice were exposed to nicotine vapor or room air for 7 weeks. Left ventricular (LV) catheterization data previously showed that nicotine exposure significantly impairs cardiac function and lowers ejection fraction. To evaluate potential mechanisms of fibrosis and inflammation, LV samples from the hearts of nicotine-exposed mice were assessed and compared to air-sham mice using qPCR. mRNA levels of LARP6, a fibrotic biomarker, and TRAF3IP2, an inflammatory biomarker, are presented.

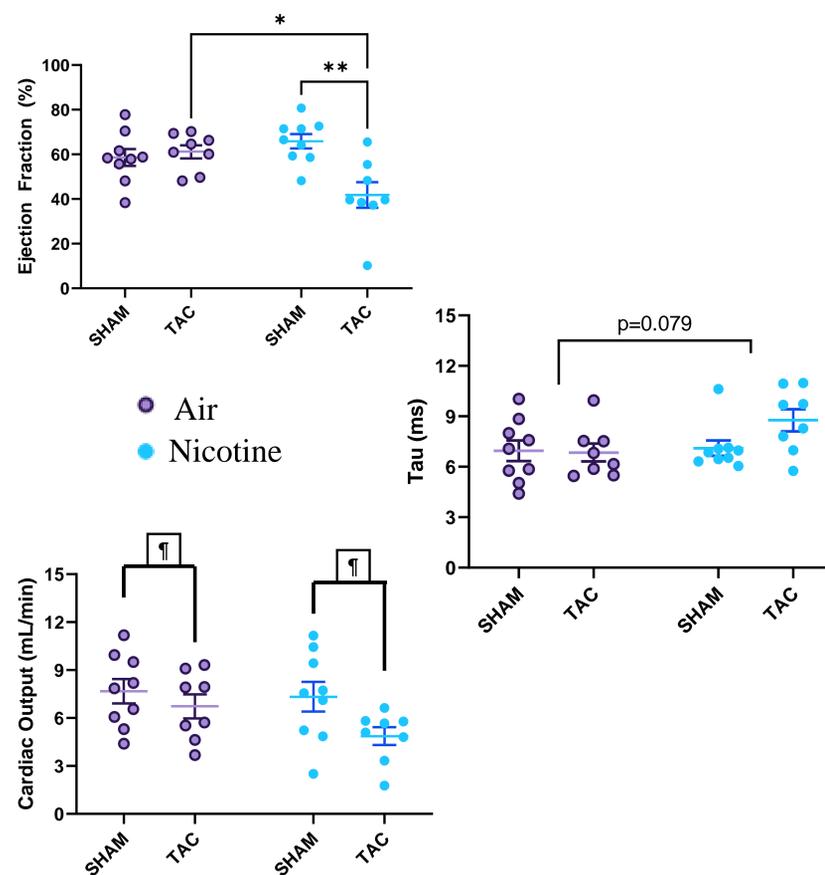
Exposure



qPCR Data



Catheterization Data



Open chest, left ventricular catheterization data (* $p < 0.05$; ** $p < 0.01$; ¶=effect of surgery).

Conclusion

We have previously seen that chronic nicotine exposure leads to a decreased ejection fraction and an accelerated cardiac decompensation following TAC, and here we present qPCR data of LARP6 and TRAF3IP2. mRNA levels of LARP6 were not significantly different in nicotine-exposed mice compared to control (air) mice. Interestingly, TRAF3IP2 levels decreased in the mice exposed to nicotine. Future investigation of other biomarkers of inflammation and fibrosis is warranted and will provide a more complete picture.

Acknowledgements

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