CHRNA5 is a receptor subunit which mediates signal transmission æch • ^ } æ] • ^ • Èh Ö^- ^ & c • hā } h c @ ^ h \* ^ } ^ Èh • ] ^ &ā , &æll ^ h • ā } \* | ^ h } ` & | ^ [ cā å ^ h ] [ | ^ { [ phisms (SNPs), have been associated with susceptibility to nicotine dependence. It is crucial to understand CHRNA5's expression in the development of therapies for individuals struggling with nicotine consumption. To date, the mechanism through which the decreased function of CHRNA5 increases nicotine intake is not well understood. As of now, there have been several GWAS studies that have evaluated the variants in CHR-ÞŒĺÁà^Á ˘œ¦ā:ā}\*Á •œœi•œi&•Á~¦ [ {Áåā ^\^}cÁ \^\*ā [ }•Áā}Ác@^Á , [ ¦|åĖÁ P [ , ^ç^¦ĖÁ there has been minimal research done in other aspects, such as the effects of in utero nicotine exposure through maternal smoking. Maternal smoking during pregnancy exposes the developing fetus to nicotine and •c \* åi^•Å • \* \* \* ^•cÅ c@æcÅ c@i•Å i•Å æ•• [&iæc^åÅ \_ ic@Å ¦i•\Å ~æ&c[ !•Å i}Å [ •] !i} \* Å i}-&|`åå}\*Áæcc^}ci[}Áå^,&ic+ÉÁ@i\*@^¦Á&@æ}&^Á[-Á¦^+]å!æc[¦^Á]¦[à|^{ +ÉÁæ+Á \_^||Á as a higher chance of dealing with nicotine dependency. Carriers of the risk variant may be at elevated risk of struggling with smoking cessation during pregnancy. The literature review and experimental proposal aim to &[ ] clià` c^Ác[Ác@^Á, ] åi] \* • Á [ -Ác@^ÁÔPÜÞŒÍÁ \* ^ ] ^ÉÁæ} åÁ • ] ^&i, &æll^Ác[ , ælå • Å c@^Å^ ^&c+Å [ ~Å { [ c@^\!Å • { [ `\^!+Å , āc@Å c@^Å !å• \Áçæ!āæ} cÅ [ }Å c@^å!Å [ ` • ] !å} \*Åä}Å terms of dealing with alcohol and nicotine addiction in adolescence.

Although tobacco has been growing in the Americas for around 8,000 years, it was not up until around 2,000 years ago that indigenous people began chewing and smoking tobacco during cultural and religious ceremonies (Cancer Counsil NSW, n.d.). The spread of tobacco from the Americas around the world began shortly after the start of European colonization. In the 1600s, tobacco became widespread in Europe, and by the 1700s it was already established as a worldwide developed industry. Although risks associated with smoking were reported as early as the 1600s,  $x_{2}^{k} |_{k}^{k} |_$ 

•i[] kæki•^}æ]•^•kçS}i }ÈkG€FGbÈkP[\_^c,^c/iÈk•i}\*|^k]~{[diå^k][|^{[[1]@i•{k (SNP) variants in the gene are associated with an increased risk for tobacco dependence. The way that polymorphisms in this case work is that they alter the receptor composition and the binding of nicotine to the receptor. This, in fact, leads to chances in functional and behavioral outcomes (Chaity et al., 2022). Therefore, investigating into the CHRNA5 gene is of crucial interest in learning more about human nicotine dependency.

To date, there have been many human genome-wide association •c\*äi^•ÅÇŐYŒÙq•DÅ\_@i&@&c^Å@^|]^Ååi}Åi@^Åa^}œ.^â}\*Åc@^Ååi ^!^}&^DÚ çæ!iæ}c•Åi}Åc@^Å\*^}^Å&[åi}\*Å-[!Åc@^Å ÍÅ • `à`}id[-Å]i&[œ]i&Åæ&c^]&@[|i}^Å receptors (Lassi et al., 2016). These studies have contributed to determining the risks of nicotine dependency depending on the location where people live. In a research paper by Hong et al. (2020), the results indi:

H3: Adolescent mice D with in utero nicotine exposure will consume the third most alcohol at both concentrations.

H4: Adolescent mice D without in utero nicotine exposure will consume the least alcohol at both concentrations.

P [KAΌ[|^•&^>ch{i&^h}\_a:c@ki}A\*c^\[A\j&[e\*'\^A,j]|A&[}•`{^h•i\*}a,-&æ}c]^A{[!^Aæh]&[@[|Aæch]€€A \*D{ŠE

 $\mathbb{P} \in \mathsf{KkE}[\&[@[[d\land c][\bullet^{\uparrow}]\land i]] \& @ c \land i] \land \land \& c \land [] \& @ a [[\land \bullet \& \land ] c \land [i \& \land \land i] i @ [`c \land i] in utero exposure of nicotine.$ 

As of current literature review and my personal research, there has  $\frac{1}{d^{-1}} = \frac{1}{d^{-1}} = \frac{1}{d^{-1}$