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Smoking & Disease Risk

This report explores the impacts of smoking habits on biological age and accelerated biological aging by examining associated methylation patterns at various locations of your DNA.

Developed By TruDiagnostic's Bioinformatics & Research Department

UNDERSTANDING

How smoking status & history influence disease risk.

Cigarette smoking is one of the leading causes of preventable disease, disability, and death in the United States, accounting for more than 480,000 deaths every year. Tobacco use accounts for 7 million deaths globally.
In 2020, nearly 12.5% of adults in the U.S. currently* smoked cigarettes. More than 16 million Americans live with smoking-related diseases like cancer, heart disease, diabetes, COPD, and more.

***As per the CDC, current smokers are defined as people who reported smoking at least 100 cigarettes during their lifetime and who reported smoking every day or some days.**

GENE: AHRR cg05575921

EPIGENETIC STATUS

- Altered when exposed to toxins from tobacco smoke

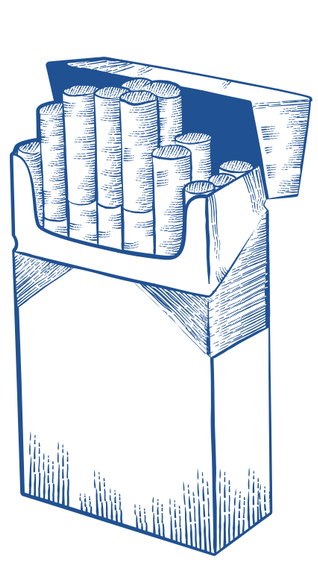
HYPOMETHYLATION OF AHRR

- Reduced AHR clearance of toxin
- Increased plaque/fat build-up in arteries

HYPOMETHYLATION OF AHRR cg05575921

- Associated with smoking + CVD

This gene has the highest level of DNA methylation change in response to smoking status.



SMOKING & DNA METHYLATION

Smoking leaves a long-term signature on DNA methylation of its exposure and is one mechanism that tobacco exposure predisposes many to adverse health outcomes. Researchers have found that CpG loci are enriched with associations of several smoking-related characteristics that contribute to damaging outcomes to health, making epigenetics a promising indicator for the impact smoking exposure has on genome-wide methylation.

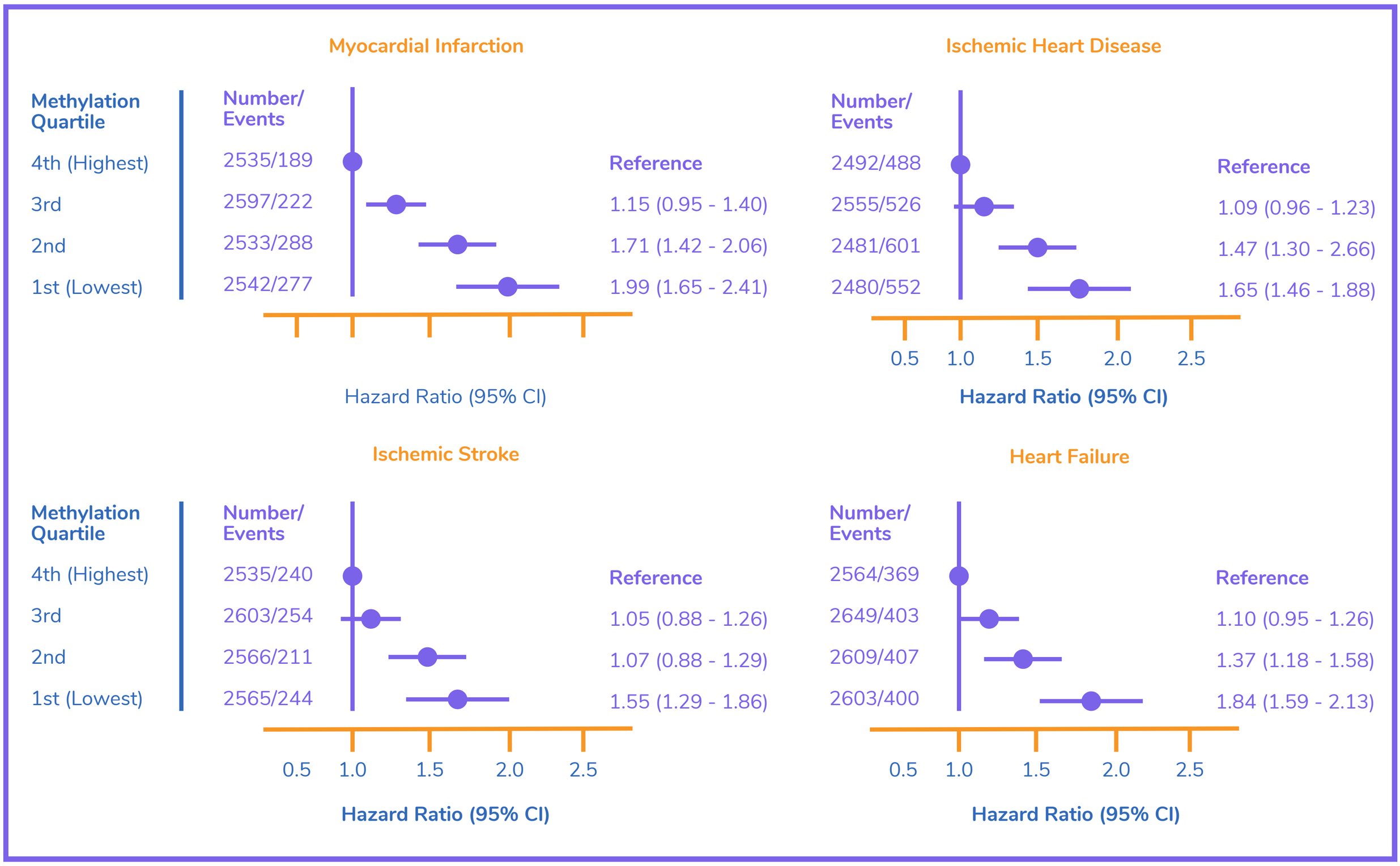
Smoking inhalation can still be considered a risk factor for almost all people. Researchers have concluded that high levels of second-hand smoke exposure are inversely associated with DNA methylation of AHRR cg05575921 in blood cells from nonsmokers.

SMOKING & DNA METHYLATION

A hazard ratio (HR) is the probability of events in a treatment group (individuals who have smoked) compared to the probability of events in a control group (individuals who have never smoked) and it determines the ratio for the probability of being diagnosed with a certain condition.

(Figure 1) Individuals from the Copenhagen City Heart Study were grouped based on their methylation levels (quartiles) determined by their smoking status. The lowest quartile (1st) is associated with the highest percentage of active smokers at baseline while the highest quartile (4th) is associated with the lowest percentage of active smokers at baseline.

FIGURE 1

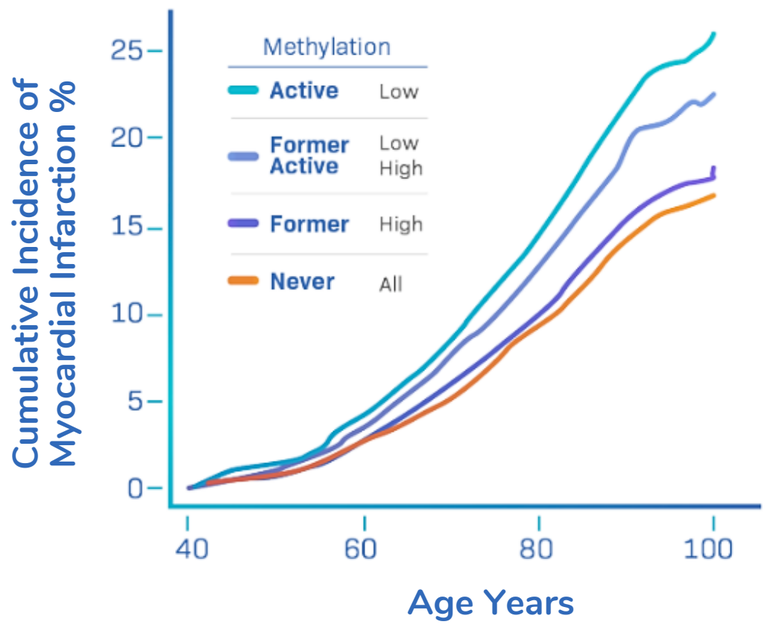


MYOCARDIAL INFARCTION

Myocardial infarction, commonly known as a heart attack, is caused by atherosclerosis, a condition where plaque builds up inside the arteries. Studies suggest measurement of AHRR cg05575921 hypomethylation is a **better marker of risk of myocardial infarction than self-reported smoking history**, even after adjusting for smoking status.

Langsted et al. conducted an 18-year follow-up of the Copenhagen City Heart Study, and approximately 1,000 incidences of myocardial infarction events occurred. The cumulative incidence of myocardial infarction **increased with age but was lowest in never-smokers and highest in active smokers with the lowest degree of methylation (Figure 2)**.

FIGURE 2



AGING

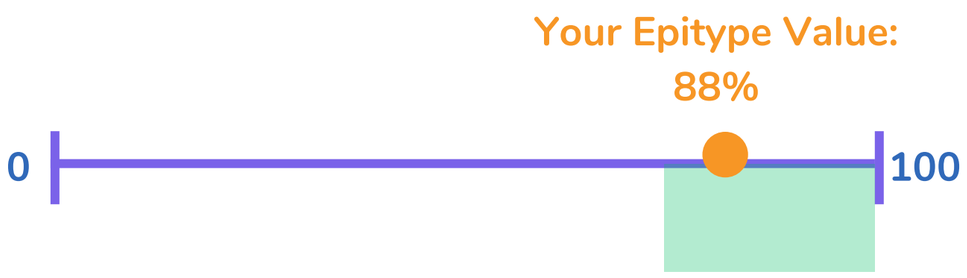
Although smoking is known to increase biological age by 5 to 7 years, promising insights have determined that smoking cessation may reduce biological age.

A pilot investigation of the impact of smoking cessation on biological age-tested smokers who averaged at baseline 13 cigarettes a day. The researchers assessed the AHRR cg0557592 methylation status and found **that 3-4% demethylation occurred just after one month of smoking cessation.** The rapid and substantial reversal of accelerated aging associated with smoking cessation supports that reversal of biological age of a former smoker.

Another study conducted by Wu et al., showed that smoking increased the epigenetic age of airway cells by **an average of 4.9 years and lung tissue by 4.3 years.** After smoking ceased, the epigenetic age acceleration in airway cells (but not in lung tissue) slowed to a level that non-smokers had. Patients who quit smoking, heavy smokers included, appeared biologically younger than they were at baseline. The connection between aging, smoking, and cessation serves as a powerful incentive for patients to quit smoking.

Your Results.

AHRR (cg05575921)
Average Beta Value %:



[64%-100%]
No Epigenetic Risk

The impact that tobacco smoke exposure has on the epigenome is based on the level of methylation at the AHRR gene locus cg05575921.

Your DNA methylation score was **88%** at the AHRR locus, meaning that your methylation score aligns with the status of **non-smokers,** putting you at **low risk** for developing smoking-related conditions.

TREATMENT

It has been noted that specific genes, such as AHRR, exhibited a reversal of DNA methylation changes in ex-smokers who quit smoking at least 10 years prior to collecting their sample. 10 years+ post-cessation, as seen in **Figure 2**, **leads to a reversal in DNA methylation at AHRR**, resulting in methylation levels similar to those observed in never-smokers.

By limiting tobacco exposure, hypomethylation at the AHRR gene will reduce, decreasing the chance of smoking-related diseases. The longer the time since cessation, the more likely the epigenome will revert back to the healthy methylation levels.

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