

Sample Results:



Report Date: MM/DD/YY

Wine Gen™ Test Report

Kit ID #: 0000

Customer Name: John Doe

Collection Date: MM/DD/YY

Reviewed by: Product Manager

Laboratory Director(s): Chinh Bach, Ph.D.

Results:

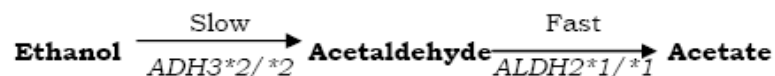
Alcohol Metabolism:

John Doe is a "SLOW" metabolizer conversion of ethanol to acetaldehyde and a "FAST" metabolizer conversion of acetaldehyde to acetate.

Alcohol Dehydrogenase 2 genotype: ADH2*1/*1 "Slow"

Alcohol Dehydrogenase 3 genotype: ADH3*2/*2 "Slow"

Aldehyde Dehydrogenase 2 genotype: ALDH2*1/*1 "Fast"



The ADH3*1*1 "slow" variant slowly converts ethanol into acetaldehyde, a carcinogen, while the ALDH2*1*1 "fast" variant rapidly converts acetaldehyde into acetate.

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Summary and Interpretations of Results:

Alcohol (ethanol) is carried from the bloodstream to the liver where it is converted into acetate by 2 liver enzymes. The enzyme Alcohol Dehydrogenase (ADH) is responsible for the initial metabolism of alcohol into acetaldehyde, a carcinogen and neurotoxin. Acetaldehyde is then converted into Acetate and Water by the Aldehyde Dehydrogenase the (ALDH) enzyme. Both enzymes are encoded by three genes. Each of these genes can have a “fast” or “slow” version, and when grouped together can determine your acetaldehyde levels and overall metabolism of alcohol.

As a MODERATE DRINKER with a “slow” ADH3*2*2 and “fast” ALDH2*1*1:

- You may benefit from drinking by **decreasing** their risk of heart attack by **31%** [1].
- You may benefit from drinking by **increasing** their high density lipoprotein levels (HDL) by **12%** which lowers cholesterol in your arteries [2].
- You may benefit from drinking by **lowering** their C-reactive protein (CRP) level which in high amounts is associated with tissue inflammation [3].
- You with the “fast” ALDH2*1*1 variant have **no** known risk of alcohol related cancers.

Laboratory specimen was analyzed using DNA isolation and PCR Quantitative Analysis.

Disclaimer








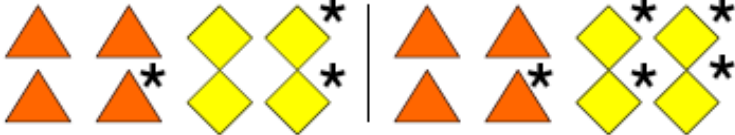

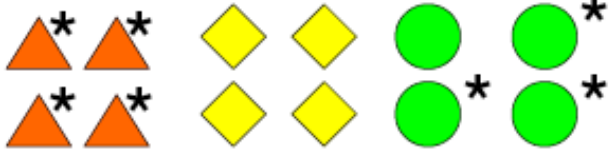

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[1]Heidrich

[2] Hines LM, Stampfer MJ, Ma J, Gaziano JM, Ridker PM, Hankinson SE, Sacks F, Rimm EB, Hunter DJ. Genetic variation in alcohol dehydrogenase and the benefit effect of moderate alcohol consumption on myocardial infraction. N Eng J Med 2001; **344**: 549-55.

[3] Furman K, Castelnovo DI, Zito F, Gaetano DE, Iacovello L. Genetic variation in alcohol dehydrogenase 3 and the decrease of hs-C-reactive protein levels by moderate alcohol consumption. J Thromb Haemost 2005; **3**: 801-2.

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Alcohol Metabolism Enzymes ALDH: alcohol dehydrogenase ADH: aldehyde dehydrogenase (ADH) (ALDH) $C_2H_5OH \rightarrow CH_3CHO \rightarrow CH_3COOH$ (Ethanol) (Acetaldehyde) (Acetic Acid)	Genes Type  ALDH2*1 <i>FAST allele</i>  ALDH2*2 <i>SLOW allele</i>  ADH2*2 <i>FAST allele</i>  ADH2*1 <i>SLOW allele</i>  ADH3*1 <i>FAST allele</i>  ADH3*2 <i>SLOW allele</i>		
Genotypes	Phenotypes		
	ALDH2 metabolism: <i>slow</i> ADH2 metabolism: <i>slow</i> Effect: Low MI risk and increase of cancer to moderate drinkers. Note: <i>ADH3</i> enzyme does not effect phenotype.		
	ALDH2 metabolism: <i>slow</i> ADH2 metabolism: <i>fast</i> Effect: 30-fold increase cancer and high MI risk to moderate drinkers. Note: <i>ADH3</i> enzyme does not effect phenotype.		
	ALDH2 metabolism: <i>fast</i> ADH2 metabolism: <i>fast</i> Effect: No known increase or decrease of MI to moderate Note: <i>ADH3</i> enzyme does not effect phenotype.		
	ALDH2 metabolism: <i>fast</i> ADH2 metabolism: <i>slow</i> ADH3 metabolism: <i>fast</i> Effect: Slight increase HDL level but increased risk MI for moderate drinkers.		
	ALDH2 metabolism: <i>fast</i> ADH2 metabolism: <i>slow</i> ADH3 metabolism: <i>slow</i> Effect: 26% increase of HDL level, decrease of MI and CRP for moderate drinkers.		

References:

1. Yokoyama A, Omori T. Genetic polymorphism of alcohol and Aldehyde Dehydrogenases and risk for esophageal and head and neck cancers. *Jpn J Clin Oncol* 2003; **33**: 111-21.
2. Brennan P, Lewis S, Hashibe M, Bell DA, Boffetta P, Bouchardy C, Caporaso N, Chen C, Coutelle C, Diehl S, Hayes RB, Olshan AF, Schwartz SM, Sturgis EM, Wei Q, Zavras AI, Benhamou S. Pooled Analysis of Alcohol Dehydrogenase Genotypes and Head and Neck Cancer: A HuGE Review. *Am J Epidemiol* 2004; **159**: 159-16.
3. Hines LM, Stampfer MJ, Ma J, Gaziano JM, Ridker PM, Hankinson SE, Sacks F, Rimm EB, Hunter DJ. Genetic variation in alcohol dehydrogenase and the benefit effect of moderate alcohol consumption on myocardial infraction. *N Eng J Med* 2001; **344**: 549-55.
4. Furman K, Castelnovo DI, Zito F, Gaetano DE, Iacovello L. Genetic variation in alcohol dehydrogenase 3 and the decrease of hs-C-reactive protein levels by moderate alcohol consumption. *J Thromb Haemost* 2005; **3**: 801-2.
5. Visapaa JP, Gotte K, Benesova M, Li J, Homann N, Conratt C, Inoue H, Tisch M, Horrmann K, Vakevainen S, Salaspuro M, Seitz HK. Increased cancer risk in heavy drinkers with the alcohol dehydrogenase 1C*1 allele, possibly due to salivary acetaldehyde. *Gut* 2004; **53**: 871-76.
6. Chen C, Lu R, Chen Y, Wang M, Chang Y, Li T, Yin S. Interaction between the functional polymorphisms of the alcohol-metabolism genes in protection against alcoholism. *Am. J. Hum. Genet.* 1999; **65**: 795-807.

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