



FORENSIC TOXICOLOGY:

FROM CRIME SCENE TO VIRTUAL LAB

MODULE 1

CHAPTER 4: ALCOHOL

Alcohol Consumption: Linked to Thousands of Cancer Cases in Canada in 2020

- The modelling study from the World Health Organization's International Agency for Research on Cancer (IARC)
- Even mild to moderate drinking poses risk of developing the disease in the future
- "...alcohol is a Class I carcinogen defined by IARC." (CAMH)

HEALTH | News

Alcohol consumption linked to thousands of cancer cases in Canada in 2020: study

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The Canadian Press Staff
Contact

Published Wednesday, July 14, 2021 6:28PM EDT



A cocktail is made in Ottawa in this file photo dated April 11, 2015. THE CANADIAN PRESS/Justin Tang

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01. INTRODUCTION

What is Alcohol?

01

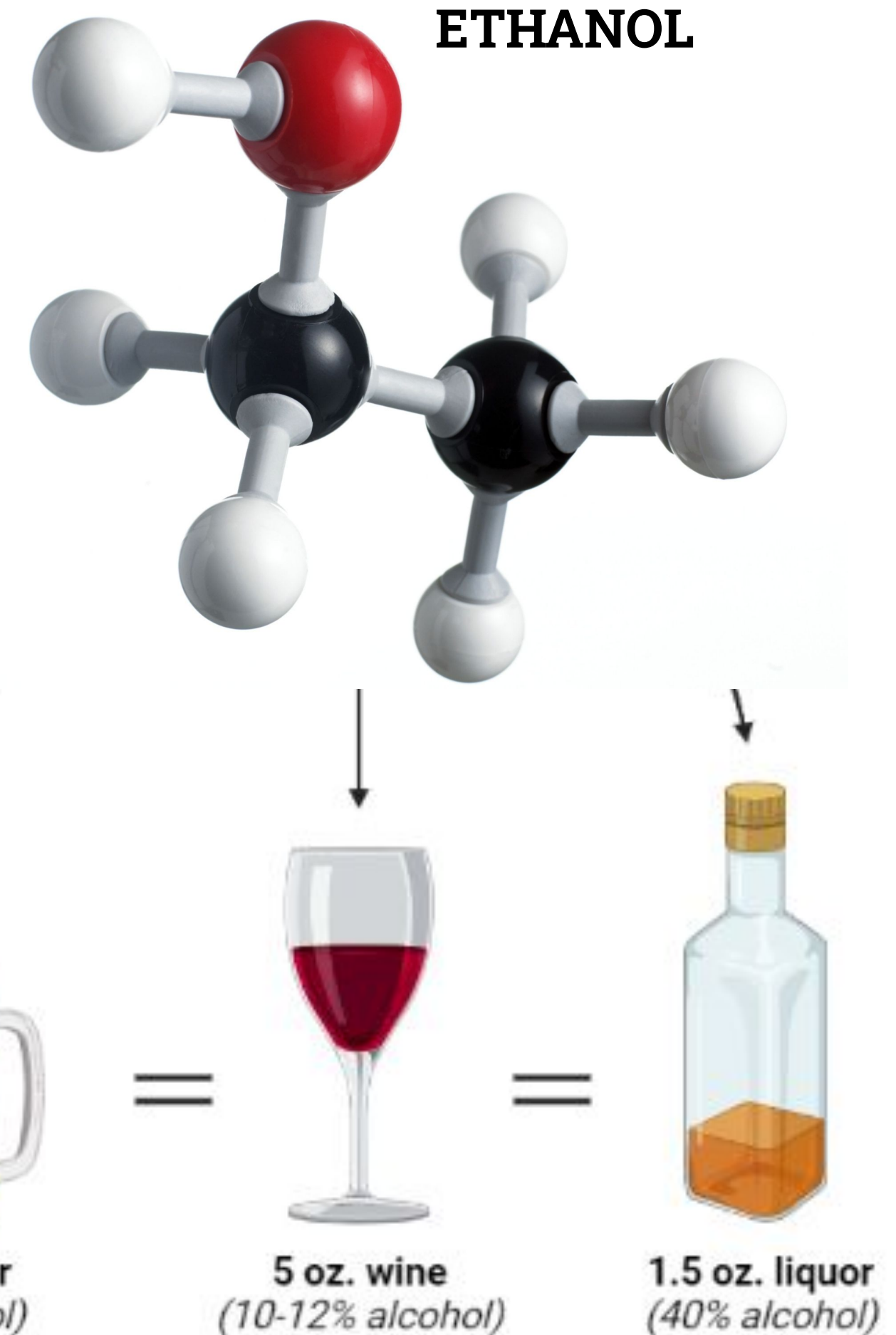
WHAT IS ALCOHOL?

Alcohol

- Depressant that slows down parts of the brain that are responsible for behaviour, breathing, heart rate, and thinking.
- Legal in Canada, has many restrictions and should be consumed moderately.
- Originates from the **fermentation** or **distillation** process of different fruits, vegetables, or grains, including beer and wine.

Fermented: beer and wine, maximum alcohol content of 15%

Distilled: liquor, spirits (rum, whisky, vodka), much higher alcohol content




01

Regulation of Alcohol

Alcoholic beverages are regulated under the provisions of the Safe Food for Canadians Act (SFCA), the Safe Food for Canadians Regulations (SFCR), and the Food and Drug Regulations (FDR).


For selling between provinces, alcohol is subject to labeling restrictions under the Food and Drugs Act (FDA) and the FDR. Listed in Division 2 of Part B of the FDR are the following alcoholic beverages:

- | | |
|-----------------------|-----------|
| → Whisky | → Vodka |
| → Rum | → Tequila |
| → Gin | → Mezcal |
| → Brandy | → Wine |
| → Liqueurs | → Cider |
| → Spirituous Cordials | → Beer |



Justice Laws Website

[Home](#) → [Laws Website Home](#) → [Consolidated Regulations](#) → [C.R.C., c. 870 - Table of Contents](#)
→ [C.R.C., c. 870](#)

Food and Drug Regulations (C.R.C., c. 870)
Full Document: [HTML](#) (Accessibility Buttons available) | [XML](#) [4203 KB] | [PDF](#) [6101 KB]
 Regulations are current to 2021-06-28 and [last amended](#) on 2021-06-23. [Previous Versions](#)

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PART B

Foods (continued)

DIVISION 2

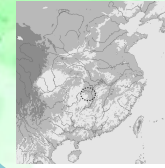
Alcoholic Beverages

B.02.001 The foods referred to in this Division are included in the term *alcoholic beverage*.
SOR/93-145, s. 3(F).

The background of the slide features a lush green forest scene with various shades of green foliage. A large, white rectangular box is centered on the slide, containing the title text. The box is divided into two horizontal sections by a thin black line.

02. HISTORICAL & CURRENT USES

HISTORICAL ALCOHOL USES



7000 B.C.

Some of the earliest drinks were thought to have originated in China, although some fermented drinks were used by the Egyptians.



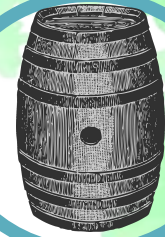
6000 B.C.

The cultivation of grapes began during the early Mesopotamia era, or present-day Iraq. By 4000 B.C., winemaking was thriving.



3000 B.C.

From 3000 - 2000 B.C., India commonly made *Sura*, a drink made from the distillation of rice.



1500 B.C.

The Greek god of winemaking and harvest, *Dionysus*, appeared in Greek literature as the first concept of drunkenness & ecstasy.



800 B.C.

In India, barley and rice beer were first created.



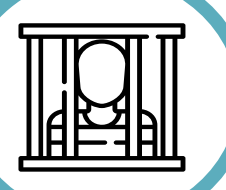
300 B.C.

Hebrews adopted wine for all ages & classes. Jewish culture also used alcohol for ritual ceremonies.



65 A.D.

Use of alcohol in moderation & for medical purposes was not yet prohibited, but drunkenness was frowned upon in most countries.



600 A.D.

Muhammad banned his followers from drinking, while Buddhists & Hindu Brahmins abstained as well.



1100 A.D.

Distillation was developed by a medical school in Italy that created a stronger & purer alcohol.



Middle Ages

Europe started creating a variety of alcohols from beer, wine, & mead, mostly made from honey.



1600s A.D.

Beer manufacturing grew popular in Germany, as beer & wine sales was mass distributed to Scotland and England. England was also popular for gin production.



1789 A.D.

1st temperance society was formed in Connecticut to reduce the amount of alcohol consumption.



19th Century

The cocktail was invented by bartenders in New York City, with 1100 legal distilleries in America producing 88 million gallons of liquor per year.



1864 - Early 1900s

1st asylum treating alcoholism opened. 18th Amendment & the Volstead Act outlawed the production of alcohol in 1920. Illicit alcohol trade boomed in 1933. Prohibition was later repealed.



Mid-20th Century

Groups like Alcoholics Anonymous (AA) were founded in 1935 and the American Medical Association (AMA) declared alcohol addictions were valid patients.



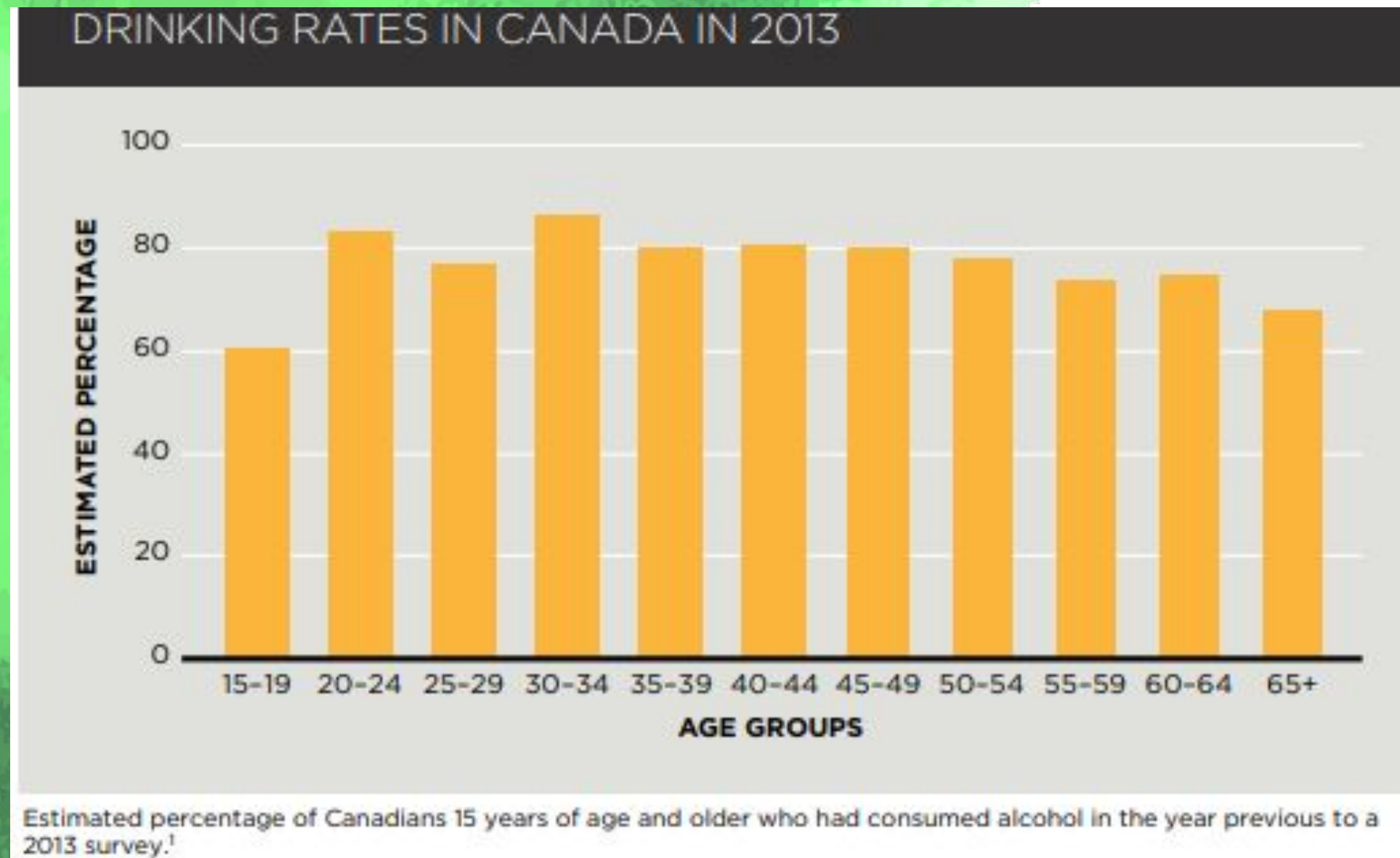
Modern Day

Today, an estimated 15 million Americans suffer from alcoholism and 40% of all car accident deaths in the US involved alcohol.

02

CURRENT USES OF ALCOHOL

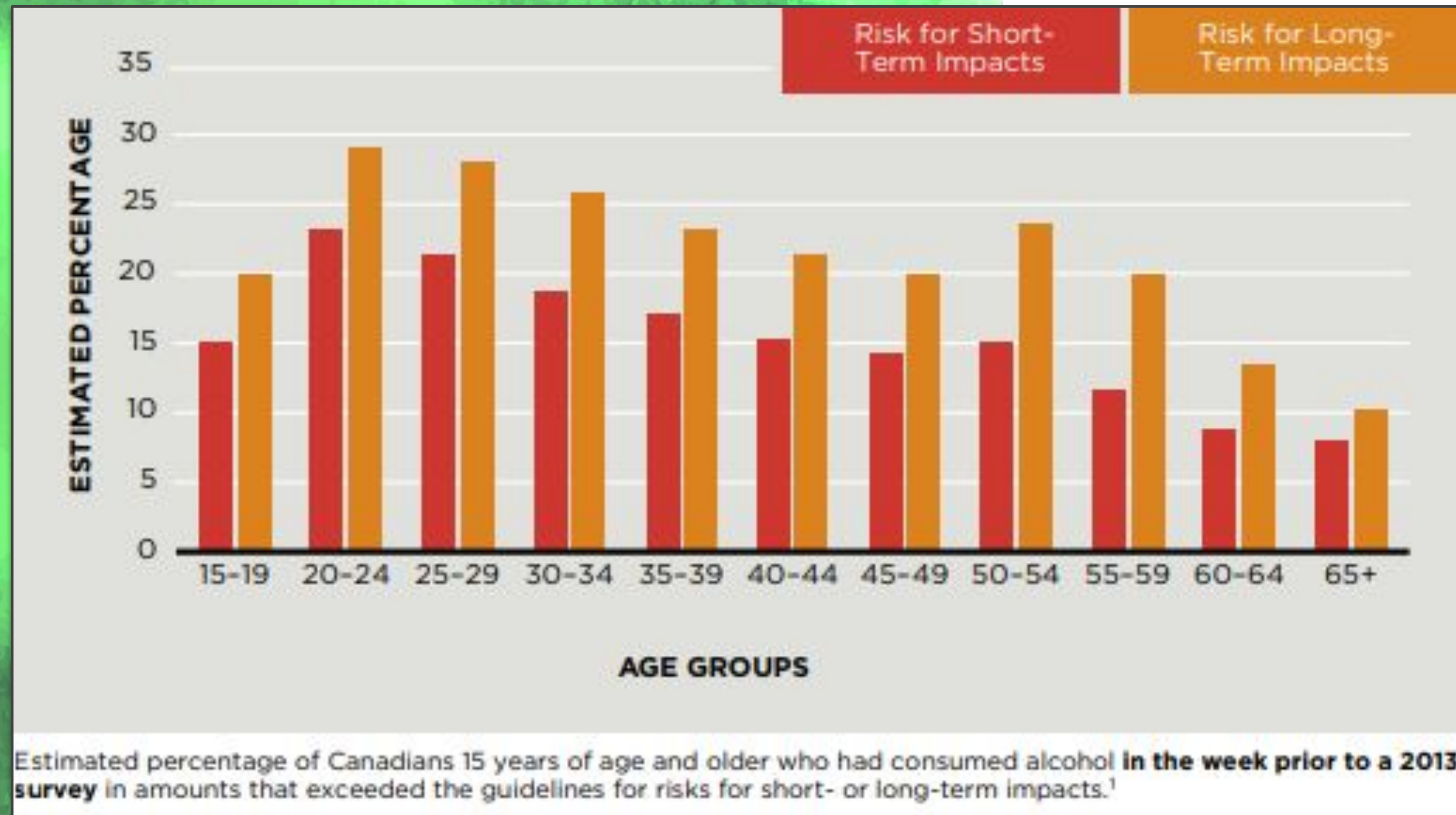
In **2013**, an estimated **22 million Canadians** reported that they drank alcohol in the previous year.



- Alcohol is the most popular drug used among Canadians, with direct dose-dependent health effects.
- Among the different age groups from a study conducted in 2013, each age group was relatively similar in estimated drinking rate percentages, with 30-34 year olds drinking slightly more often when compared to the population.



RISKY DRINKING RATES IN CANADA



- In addition to casual drinking, risky drinking is most common for 20-24 year olds
- In 2013, 56% of women admitted to binge drinking at age 15 and older at least once compared to 44% 9 years earlier.

The impacts of alcohol are dose-dependent:

- *Drinking more than 15 standard drinks per week for men or 10 standard drinks per week for women = increased risk for long-term side effects*
- *Drinking >3-4 standard drinks during any event = increases risk for short-term injury and damage to health.*



03.ALCOHOL PRODUCTION

03

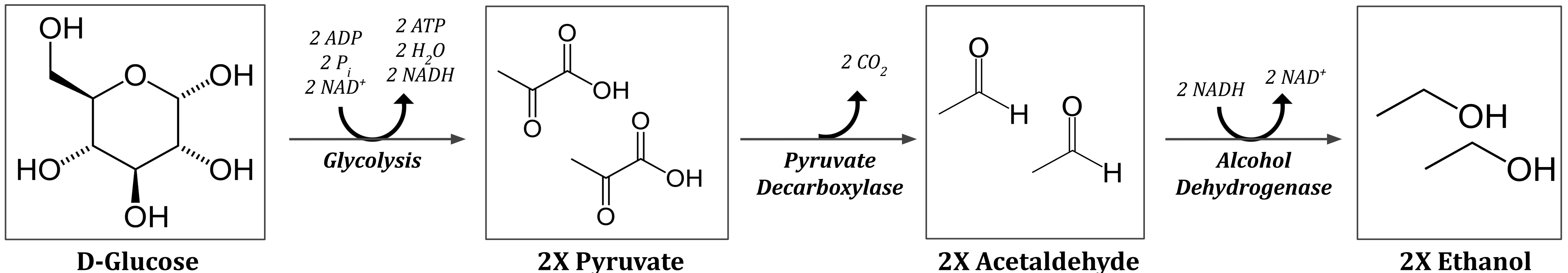
ALCOHOL FERMENTATION

Yeast Fermentation and the Making of Beer and Wine

- glucose or another sugar is converted to alcohol by microbes
- glucose is converted into pyruvic acid, which is further converted into ethanol and carbon dioxide in the absence of oxygen.
- as the yeast continues to grow and metabolize sugar, the accumulation of alcohol becomes toxic and eventually kills the yeast cells.
- after reaching an alcohol concentration of 10-15%, all yeast cells have died and alcohol remains, explaining why the majority of alcohol is within this percentage range.

For beverages with higher concentrations, the fermented products must be distilled to induce a higher ethanol concentration.

D-Glucose → *Glycolysis* → **Pyruvic Acid** → *Pyruvate Decarboxylase* → **Acetaldehyde** → *Alcohol Dehydrogenase* → **Ethanol (alcohol)**



Opposed to fermentation, the process of distillation:

- heats the fermented liquid to a boiling point where it converts into vapour that is recondensed back into a liquid as it cools
- this process concentrates the alcohol levels, while evaporating off the undesirable compounds
- typically, spirits are distilled multiple times to create a smoother and lighter body.



The fermented, lightly-alcoholic liquid is transferred to either a pot still, column still or hybrid, where it is heated to a boiling point.

The alcohol vapors that come off the boiling mash are captured and cooled, recondensing into a high-proof liquid form.

Distillers separate the "head" and "tail," or first and last parts of distilled alcohol, from the more desirable "heart" destined for consumption.

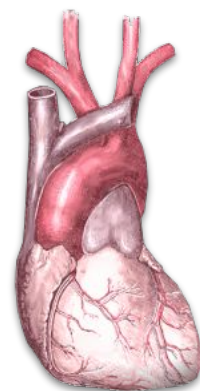
The resulting alcohol can be distilled multiple times to create a lighter, smoother or more neutral-tasting spirit.



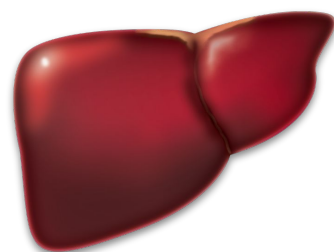
04. ALCOHOL EFFECTS ON THE BODY

**BRAIN**

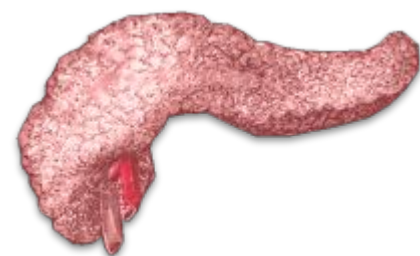
- Alcohol interferes with the brain's communication pathways
- Affects the shape and function of the brain, changing mood and behaviour and making it more difficult to think clearly with coordination

**HEART**

- Can cause cardiomyopathy (stretching & drooping of heart muscle)
- Can induce arrhythmias (irregular heartbeat), stroke, high blood pressure

**LIVER**

- Can lead to liver inflammations including steatosis (fatty liver), alcoholic hepatitis, fibrosis, and cirrhosis

**PANCREAS**

- Alcohol uses the pancreas to produce toxic substances that lead to pancreatitis (inflammation & swelling of the blood vessels in the pancreas that prevent proper digestion)

Although alcohol can induce feelings of happiness, pleasure, and sociability over short periods of time, excessive and chronic drinking can lead to alcohol dependence and addiction, commonly known as “Alcohol Use Disorder.” Chronic alcohol use is also associated with cognitive and mental health issues, including learning and memory problems.

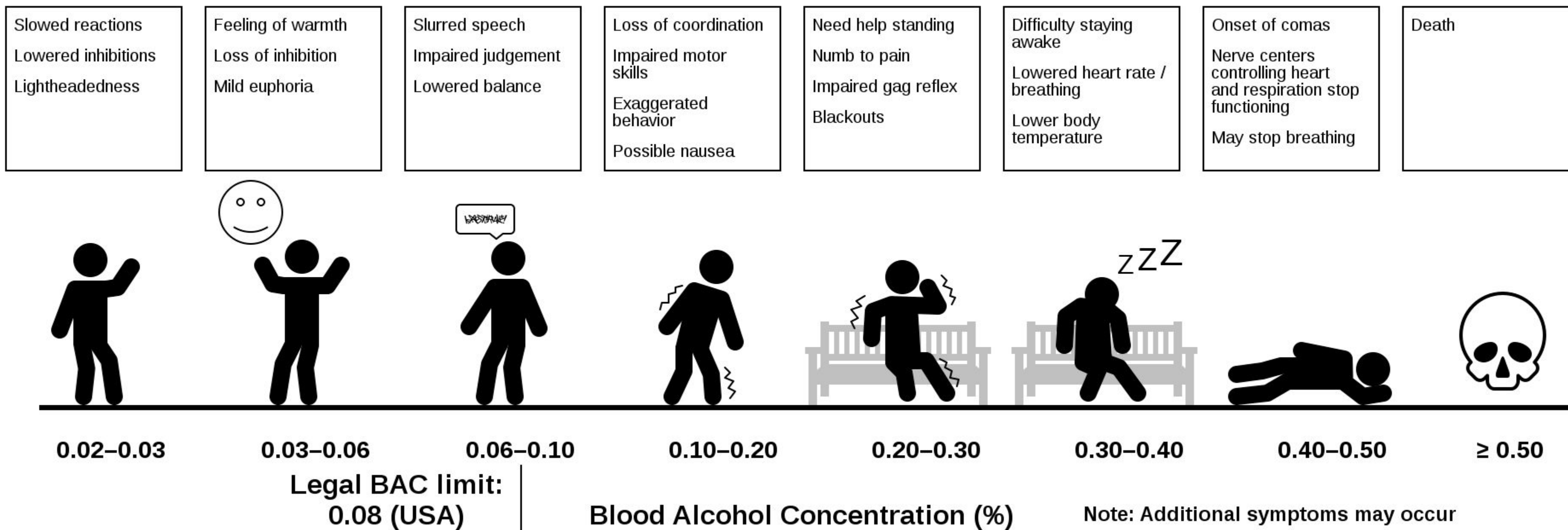
SHORT-TERM EFFECTS

- Lowered inhibitions (poor social judgement)
- Trouble concentrating
- Loss of coordination
- Loss of critical judgement
- Mood swings
- Reduced core body temperature
- Raised blood pressure
- Vomiting

LONG-TERM EFFECTS

- Diminished gray matter and white matter in the brain
- Memory loss
- Loss of attention span
- Trouble learning
- Alcoholic hepatitis
- Liver fibrosis
- High blood pressure
- Cardiomyopathy
- Stroke / irregular heartbeat

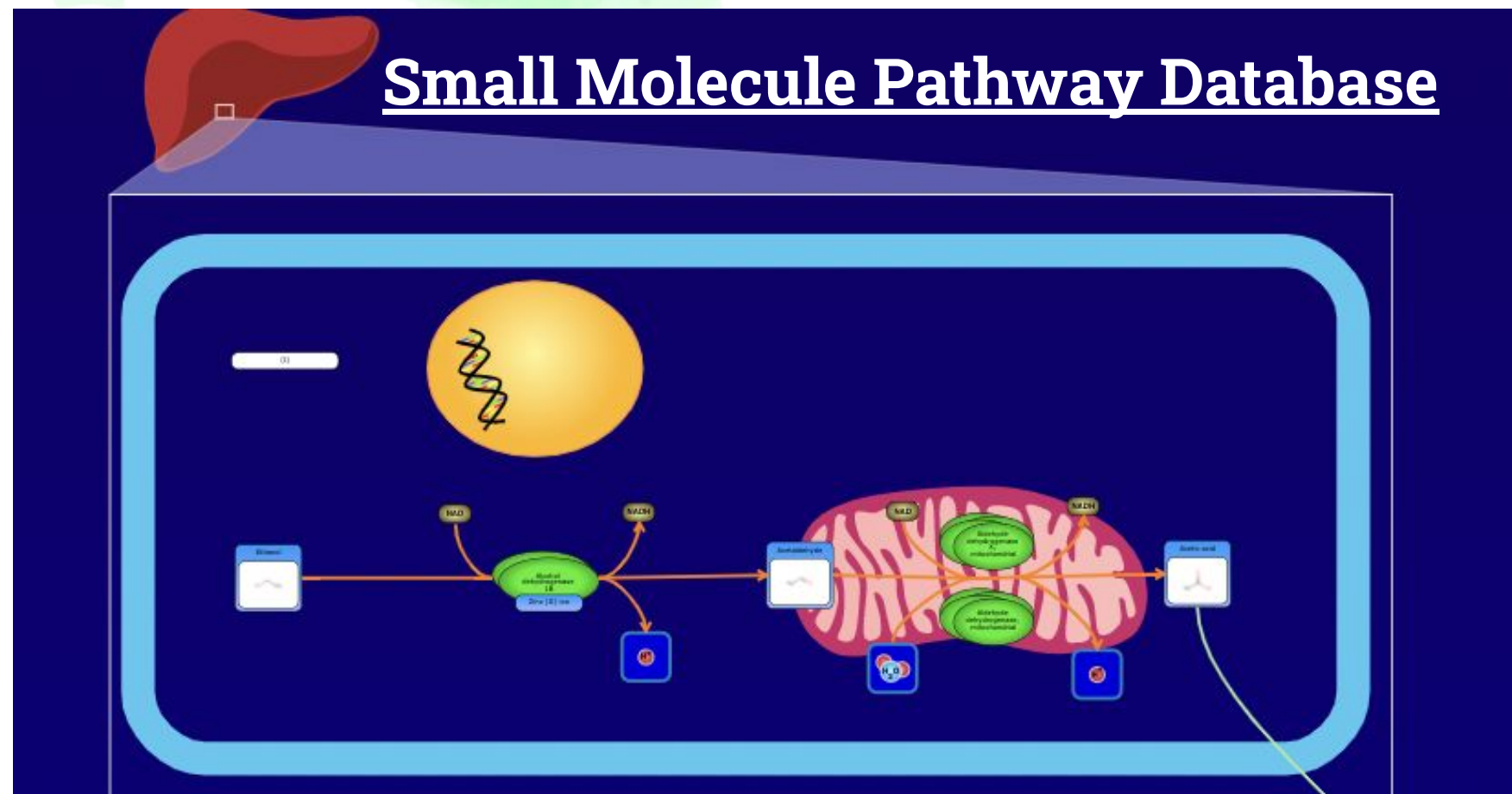
DOSE-RESPONSE FOR ALCOHOL



05.PHARMACODYNAMICS

ETHANOL AND RECEPTORS

- Ethanol also binds to GABA, glycine, NMDA receptors and modulates their effects.

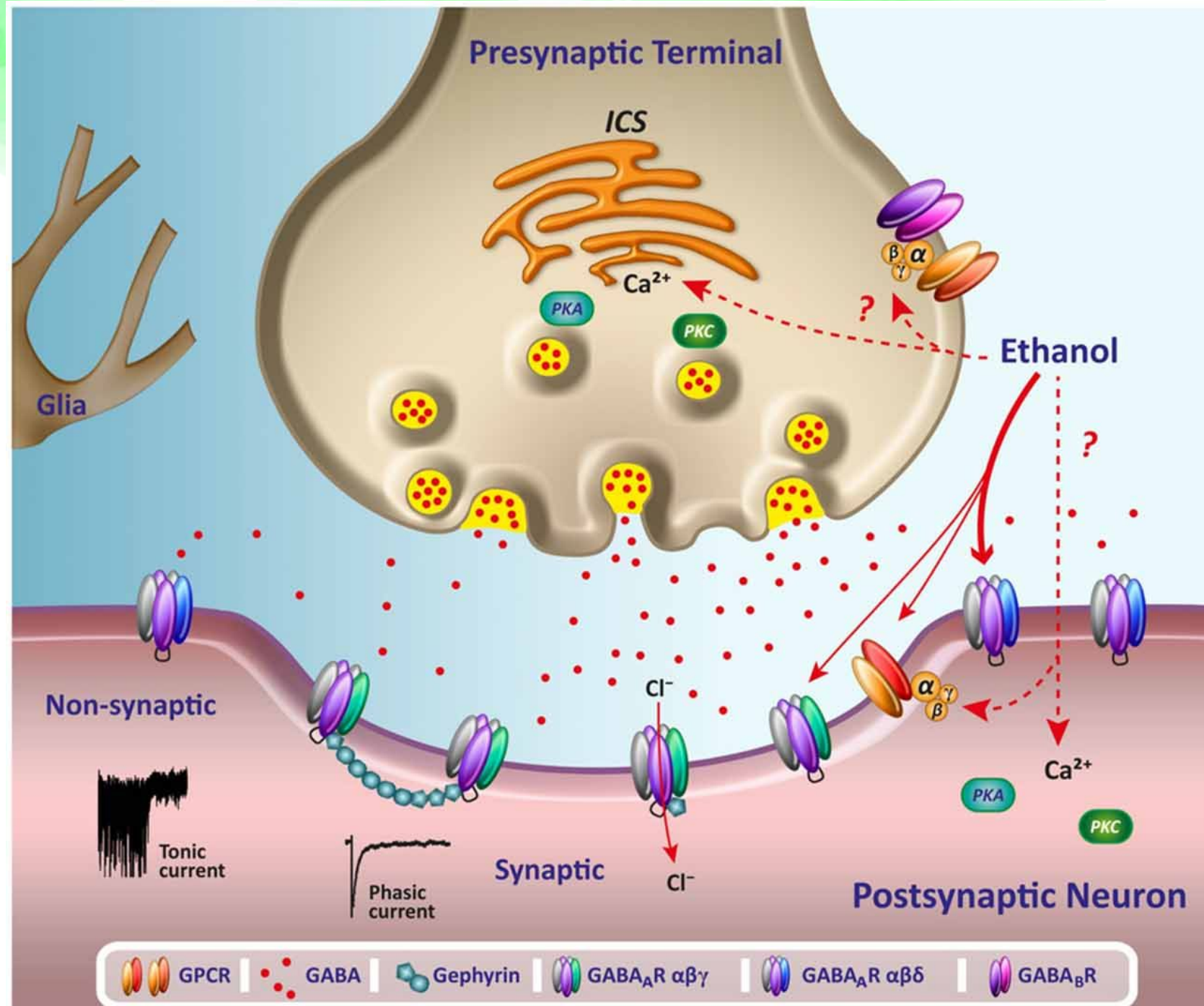


- Ethanol affects the brain's neurons by **altering their membranes as well as their ion channels**, enzymes, and receptors.
- Alcohol also binds directly to the receptors for **acetylcholine, serotonin, GABA, and the NMDA receptors for glutamate**.
- The sedative effects of ethanol are mediated through binding to GABA receptors and glycine receptors (alpha 1 and alpha 2 subunits).
- It also inhibits NMDA receptor functioning.
- In its role as an **anti-infective**, ethanol acts as an osmolyte or dehydrating agent that disrupts the osmotic balance across cell membranes

Protein binding of ethanol has not been reported.

ETHANOL AND RECEPTORS

Acute effects of ethanol on GABAergic transmission



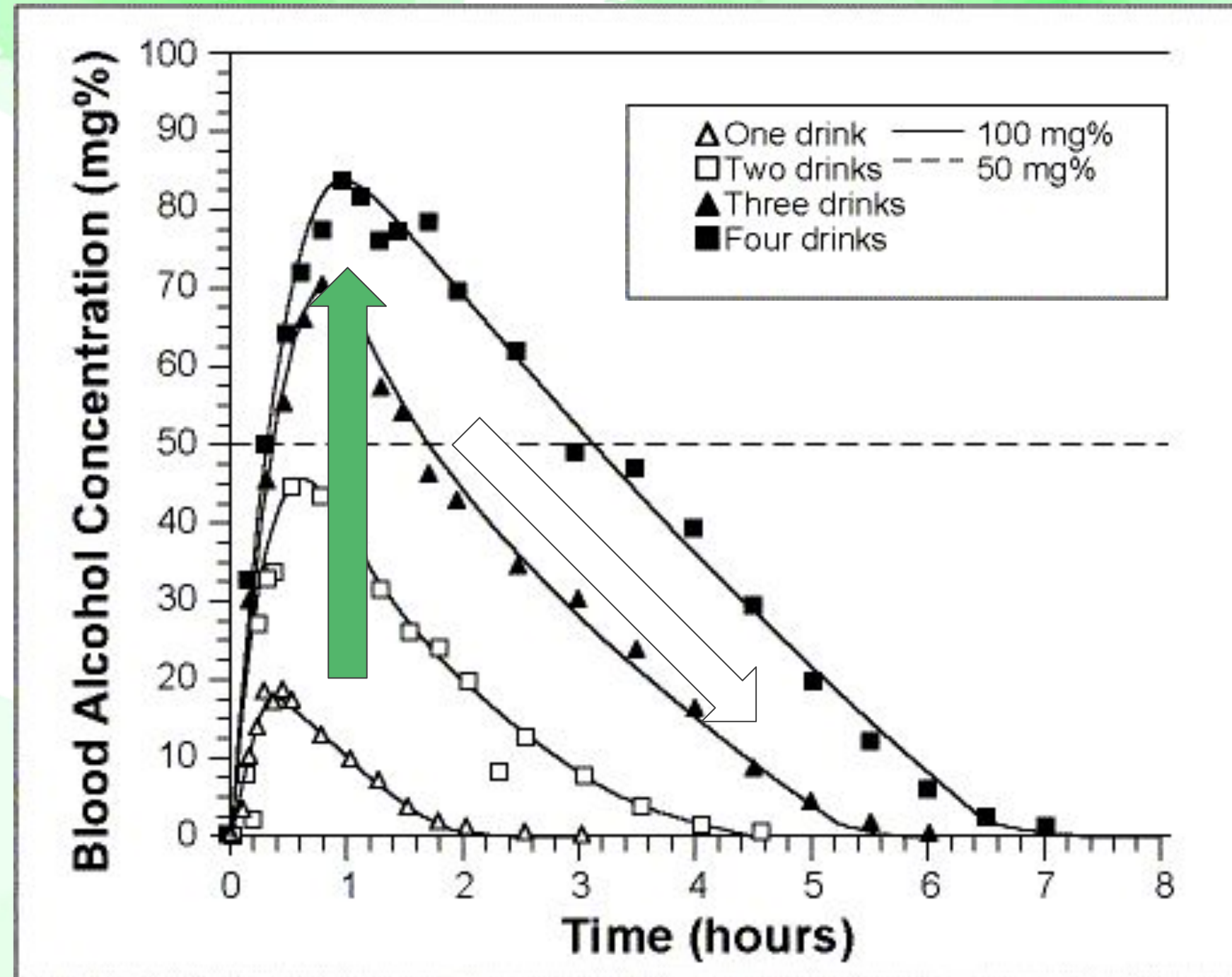
- Several potential acute pre and postsynaptic sites for the effects of ethanol on GABAergic neurotransmission.
- Reported changes on the release of presynaptic GABA might be mediated by changes on calcium release from intracellular calcium stores (ICS) following activation of G-protein coupled receptors (GPCR) or phosphorylation by protein kinase A (PKA) and C (PKC).
- Changes on the activation of GPCR, such as GABA_BR, could affect GABA release and alter the tonic Cl⁻ current associated to non-synaptic GABA_AR δ containing receptors through spillover of synaptically released GABA.
- At postsynaptic domains, acute low ethanol concentrations of alcohol appear to modulate primarily non-synaptic GABA_AR (see thicker arrow) by a mechanism that might involve direct binding to the general anesthetics site of action or by intracellular signaling pathways, G protein, PKC and PKA or calcium release.
- The scheme also shows representative traces of a phasic current, activated by synaptic receptors, and a sustained small desensitizing tonic current, mediated by non synaptic receptors.

06.PHARMACOKINETICS

06

Pharmacokinetics of EtOH

Blood alcohol concentration (BAC) after the rapid consumption of different amounts of alcohol



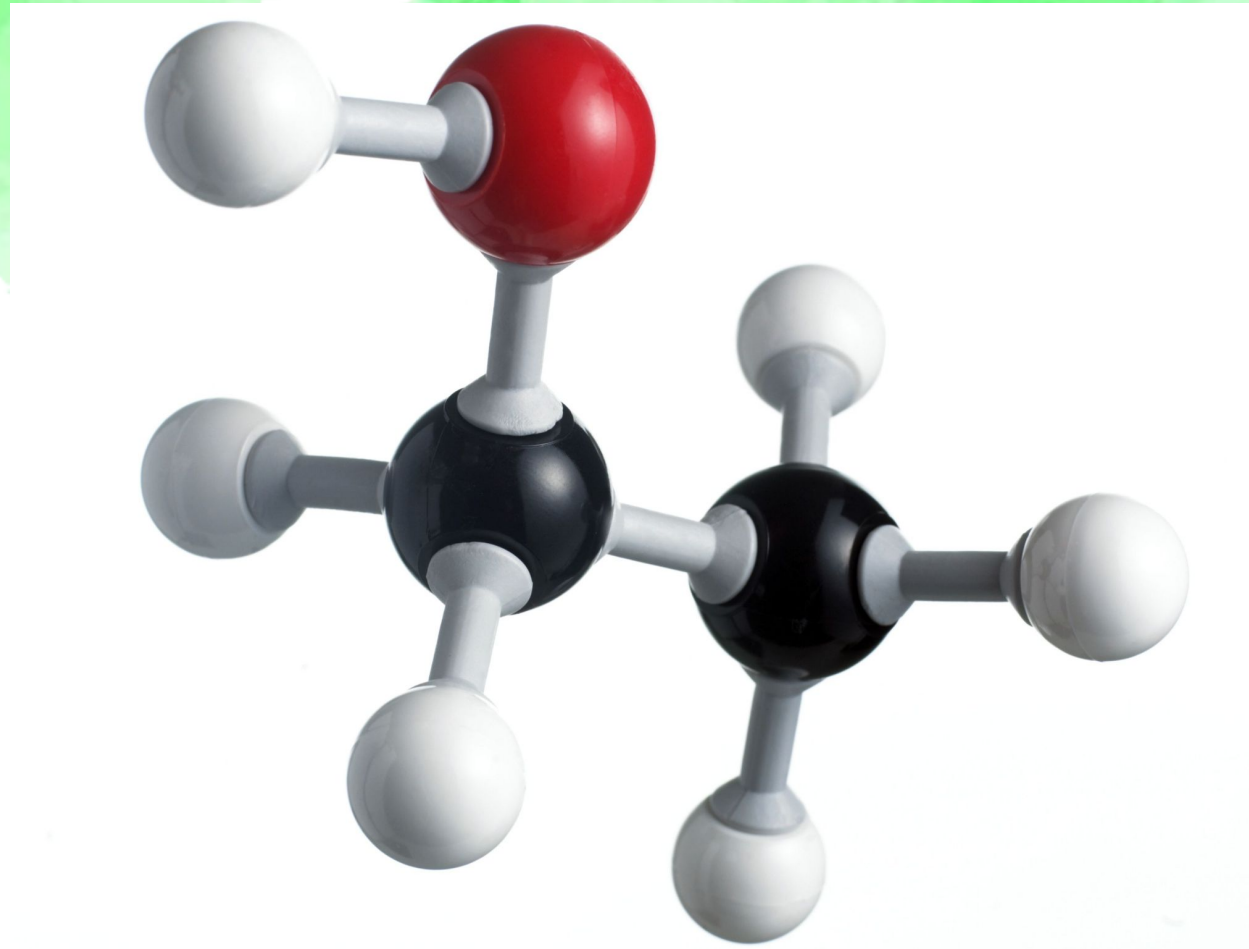
Absorption:

The maximum peak BAC value increases with increasing amount of ethanol consumed

Elimination:

1. The rates are similar regardless of the amount of ethanol consumed.
2. The longer time is needed for alcohol to clear from the blood with greater amount of ethanol consumed

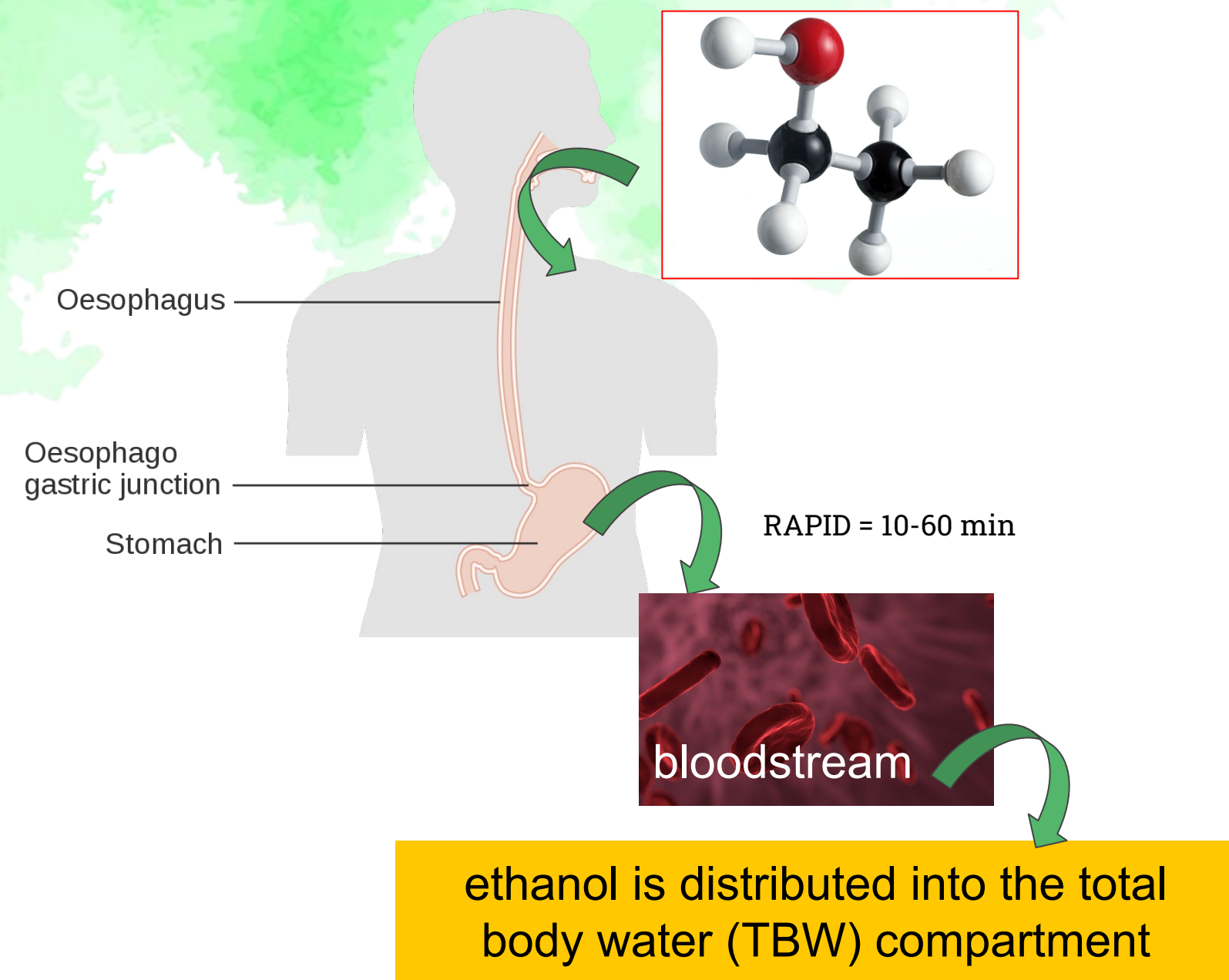
- The majority of it is absorbed from the small intestine (approximately 80%) and the stomach (approximately 20%)
- Elimination is principally by metabolism in the liver (89%) with small amounts excreted in the breath (0.7%), urine (0.3%), and sweat (0.1%)



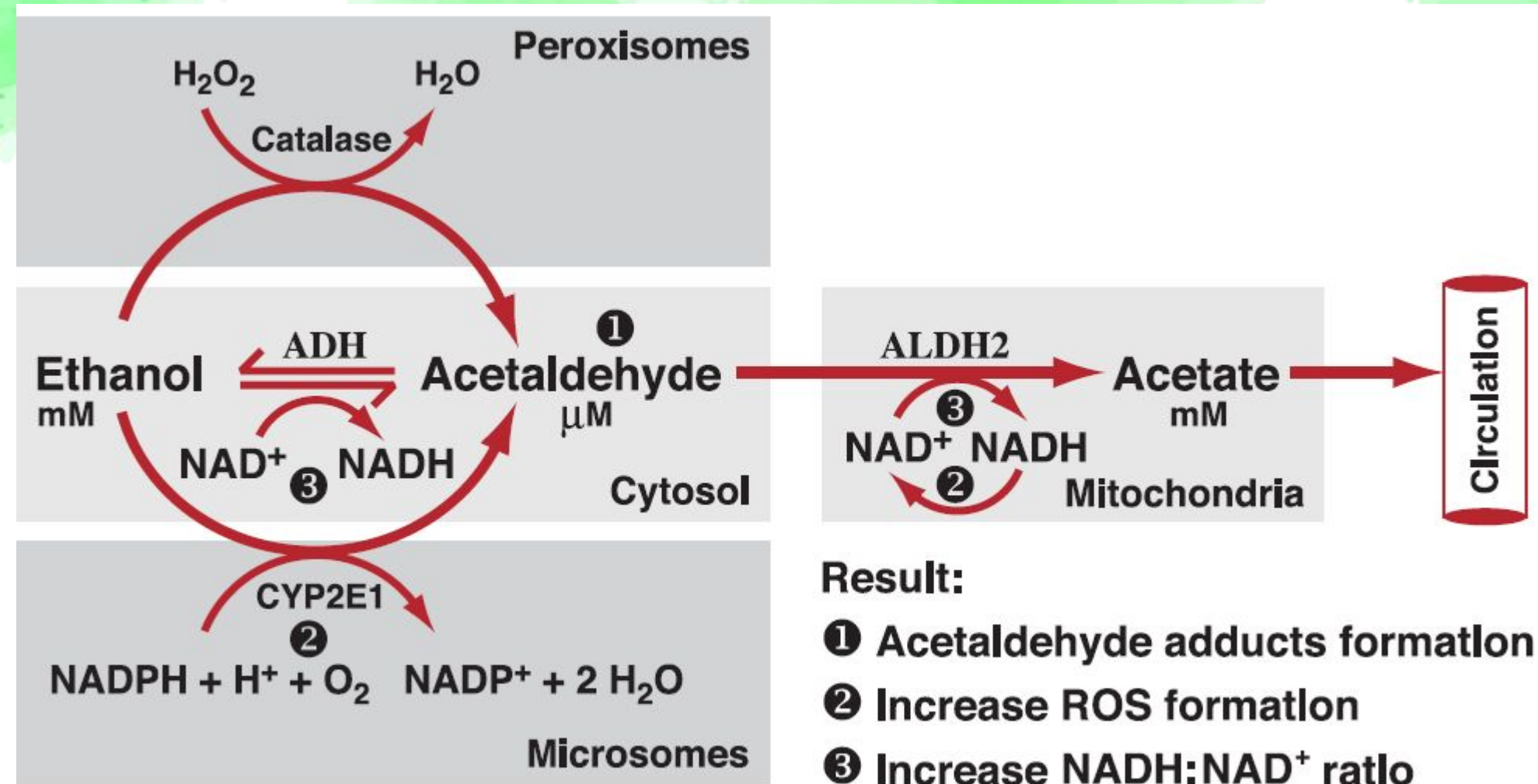
- The maximum rate of elimination of ethanol , V_{max} is 8.5 g/h/70 kg.
- This would be equivalent to a blood ethanol disappearance rate of 230 mg/L/h.
- The elimination rate is half of the elimination capacity at a peripheral blood ethanol concentration (K_m) of about 80 mg/L.
- The breath clearance at rest is 0.16 L/h.
- The renal clearance is 0.06 L/h.

Pharmacokinetics of EtOH

- The volume of distribution (V_d) of ethanol depends on a person's age, gender, and degree of adiposity (ratio of fat to lean tissue).
- The average V_d is ~0.65 L/kg.
- Elimination of ethanol from the body occurs primarily through metabolism (92–98% of dose) by hepatic alcohol dehydrogenase (ADH), an enzyme located in the liver cytosol and a microsomal enzyme, denoted CYP2E1.
- A small fraction (0.1–0.2%) of the dose of ethanol ingested undergoes nonoxidative metabolism by phase II conjugation reactions leading to formation of ethyl glucuronide and ethyl sulfate.

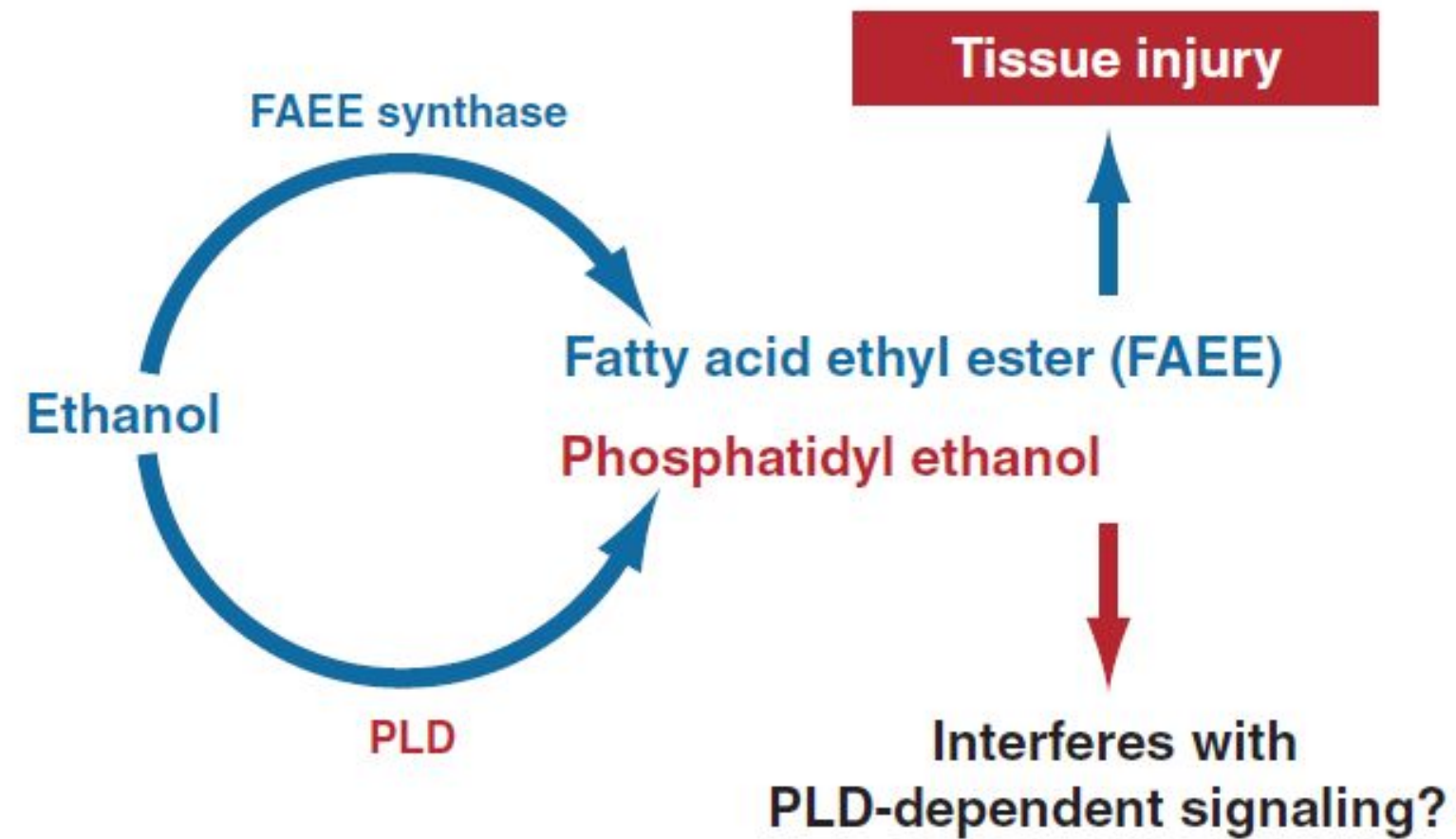


07.ALCOHOL METABOLISM



Oxidative pathways of alcohol metabolism.

- The enzymes alcohol dehydrogenase (ADH), cytochrome P450 2E1 (CYP2E1), and catalase all contribute to oxidative metabolism of alcohol. ADH, present in the fluid of the cell (i.e., cytosol), converts alcohol (i.e., ethanol) to acetaldehyde.
- This reaction involves an intermediate carrier of electrons, + nicotinamide adenine dinucleotide (NAD), which is reduced by two electrons to form NADH.
- Catalase, located in cell bodies called peroxisomes, requires hydrogen peroxide (H_2O_2) to oxidize alcohol.
- CYP2E1, present predominantly in the cell's microsomes, assumes an important role in metabolizing ethanol to acetaldehyde at elevated ethanol concentrations.
- Acetaldehyde is metabolized mainly by aldehyde dehydrogenase 2 (ALDH2) in the mitochondria to form acetate and NADH. ROS, reactive oxygen species.

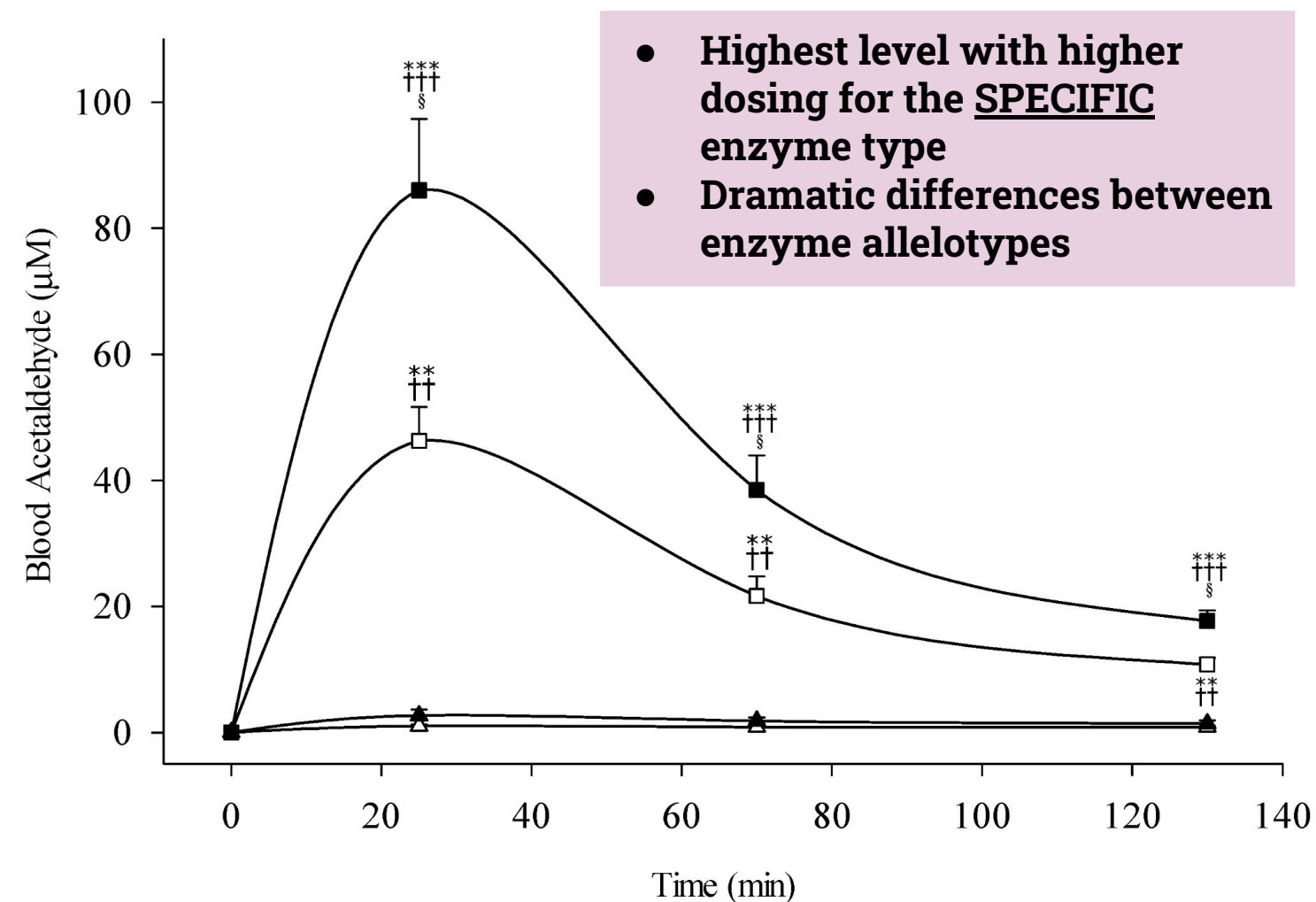


Ethanol is nonoxidatively metabolized by two pathways.

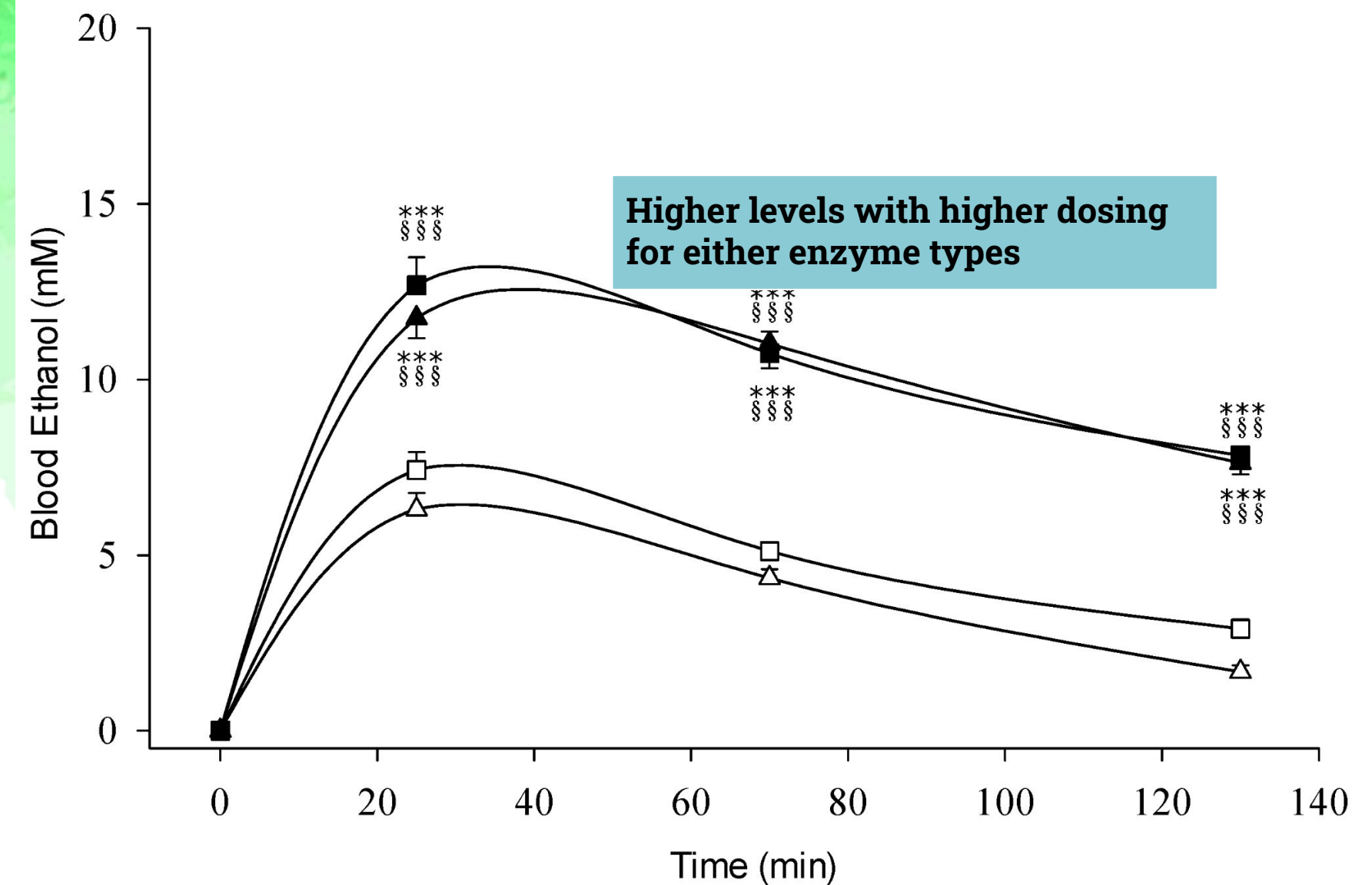
- A reaction catalyzed by the enzyme fatty acid ethyl ester (FAEE) synthase leads to the formation of molecules known as FAEEs.
- A reaction with the enzyme phospholipase D (PLD) results in the formation of a phospholipid known as phosphatidyl ethanol.

07 ENZYMES AND ETHANOL

- Alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) are the key enzymes responsible for ethanol metabolism through first-pass metabolism in the liver.
- Most ethanol elimination occurs by oxidation to acetaldehyde and acetate.
- ADH and ALDH enzymes exhibit genetic polymorphism and ethnic variation (variations in the alcohol metabolism (pharmacokinetics) and responses to alcohol (pharmacodynamics))

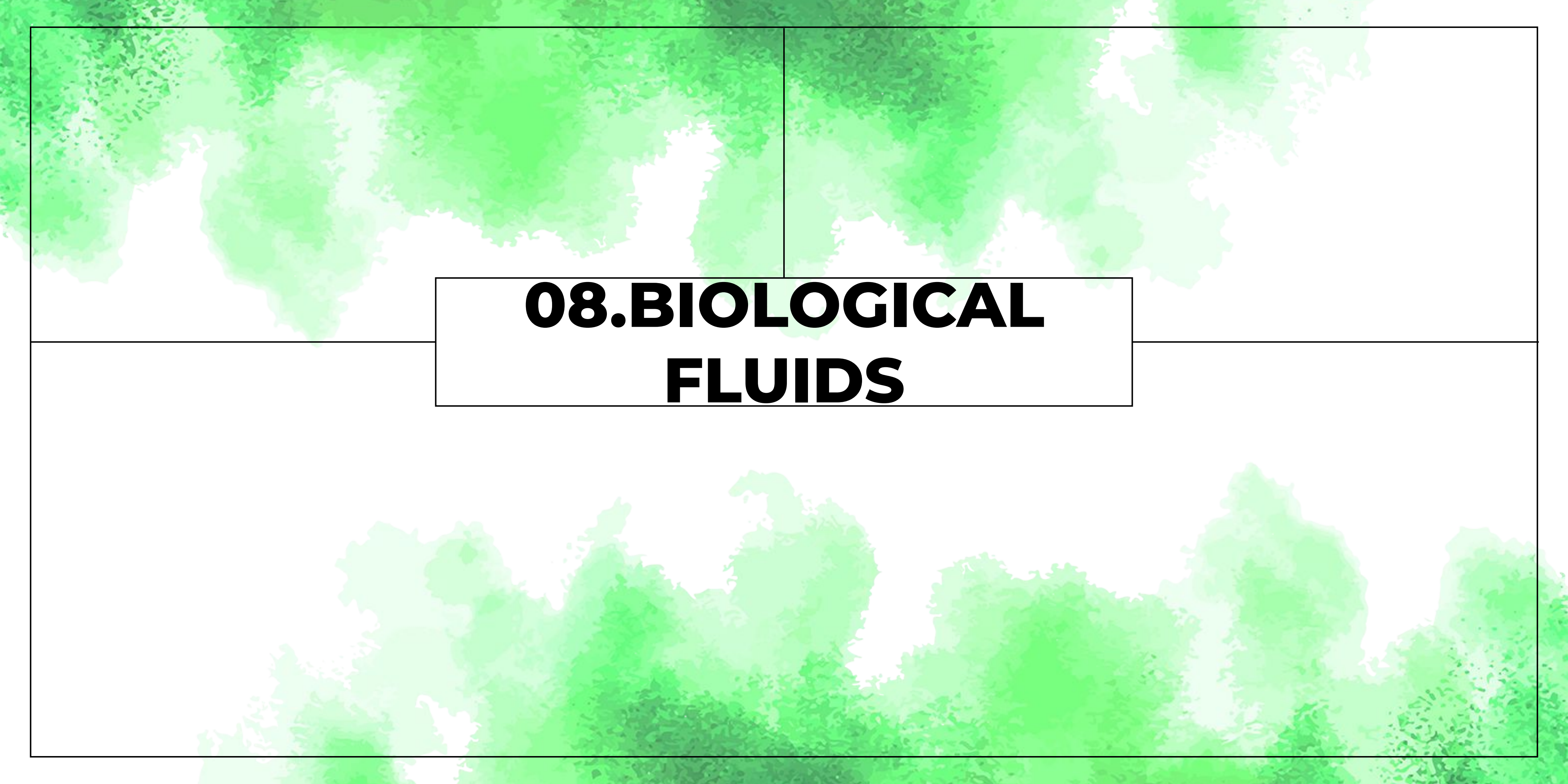


Biomolecules 2021, 11(8), 1183;
<https://doi.org/10.3390/biom11081183>



Blood ethanol concentrations of nonalcoholic subjects with different *ALDH2* allelotypes following different doses of ethanol (0.3 g/kg body weight or 0.5 g/kg)

△, *ALDH2**1/*1 with 0.3 g/kg ethanol;
 ▲, *ALDH2**1/*1 with 0.5 g/kg ethanol;
 □, *ALDH2**1/*2 with 0.3 g/kg ethanol;
 ■, *ALDH2**1/*2 with 0.5 g/kg ethanol;

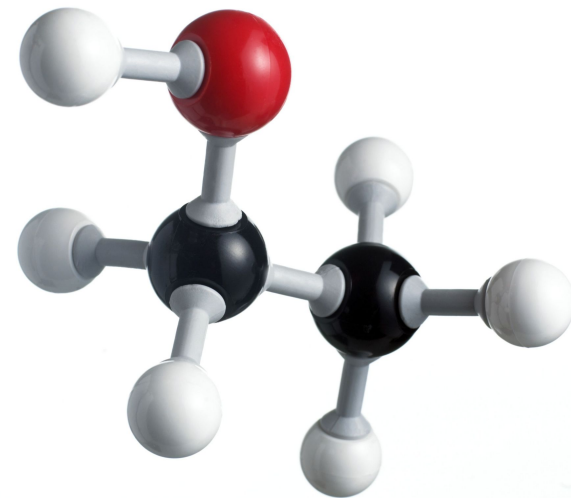
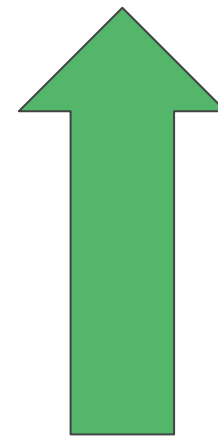
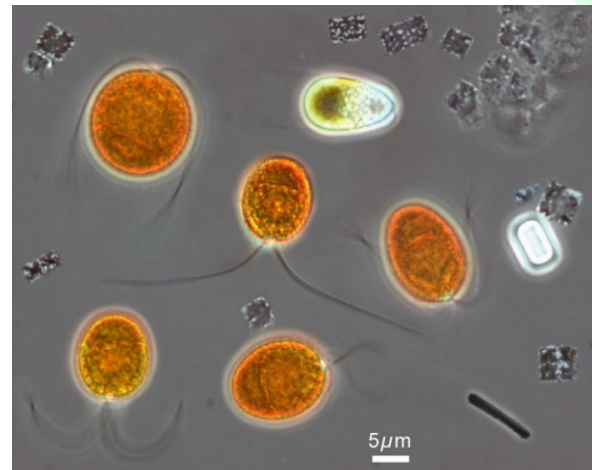
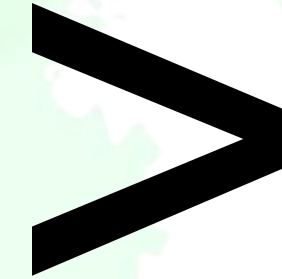


08.BIOLOGICAL FLUIDS

ETHANOL in various fluids_comparison

Vitreous fluid ethanol concentrations are higher than blood ethanol.

Urine ethanol concentrations are higher than the blood ethanol concentrations.



Postmortem specimen contamination with ethanol producing microorganisms increases with an increasing extent of trauma to the body

The maternal and fetal blood ethanol concentration–time curves were virtually superimposable, which indicated unimpeded bidirectional placental transfer of ethanol.



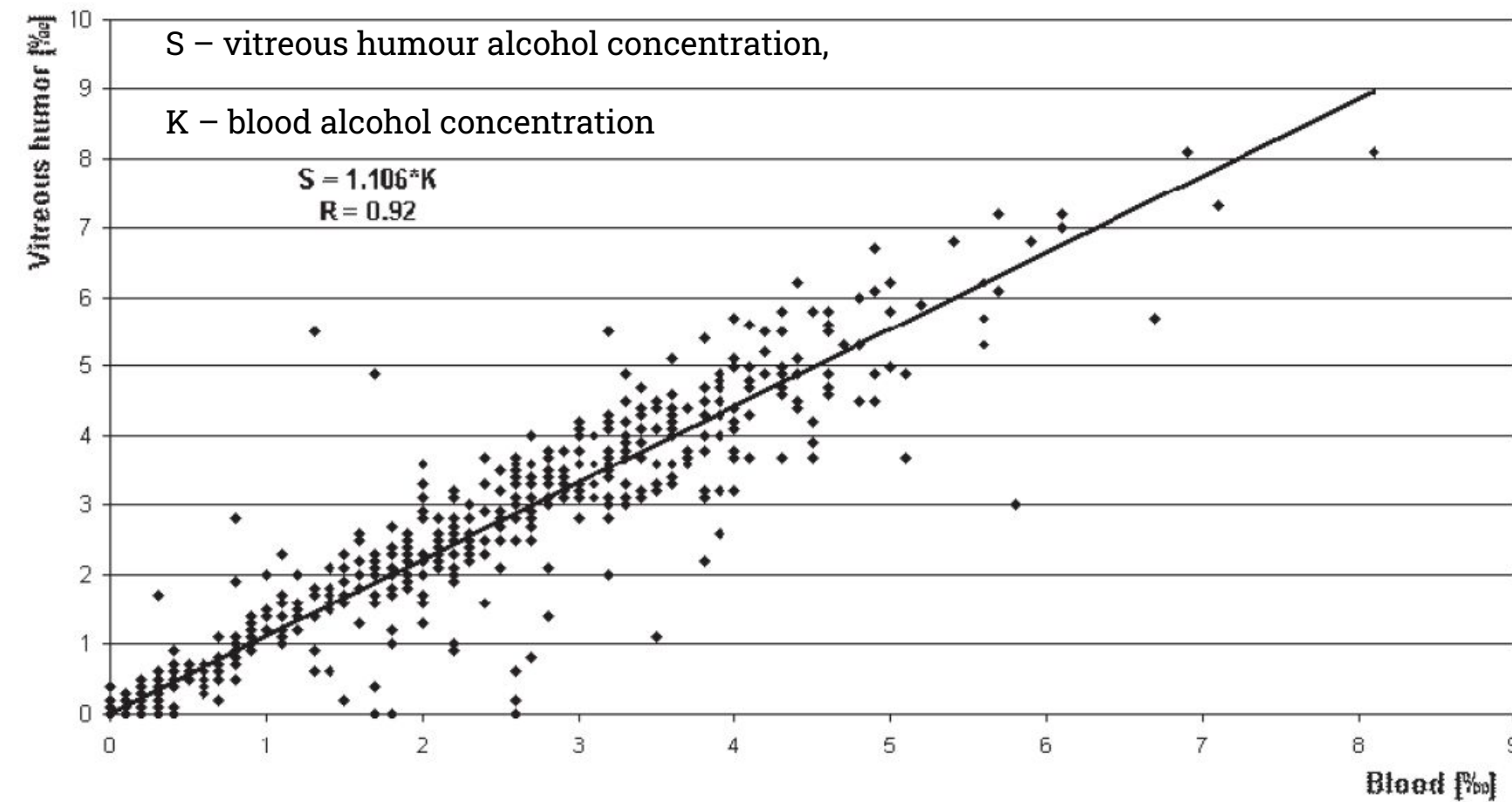


Fig. 1. Concentration of ethanol in vitreous humour (S) *versus* ethanol concentration in blood (K).

A COMPARATIVE STUDY OF THE ETHYL ALCOHOL CONCENTRATION IN VITREOUS HUMOR IN RELATION TO ETHYL ALCOHOL CONCENTRATION IN BLOOD AND URINE

What about vitreous humor versus blood?

- Linear relationship (0.92) between concentrations in two biological fluids indicates that the two fluids are comparable for testing and analysis purposes.
- Vitreous humor ethanol concentration is always **1.106 x higher** than in blood

What about vitreous humor versus urine?

- The relationship was less linear (0.90), indicating that the two fluids are less comparable then above fluids
- Vitreous humor ethanol concentration is always **0.907 x lower** than in urine

The maximum blood-alcohol concentration (BAC) is usually reached between 10 and 60min post-dosing

<https://wires.onlinelibrary.wiley.com/doi/full/10.1002/wfs2.1340>

An ambulatory care pharmacist contacted the laboratory concerning an unexpected positive urine-ethanol test result.

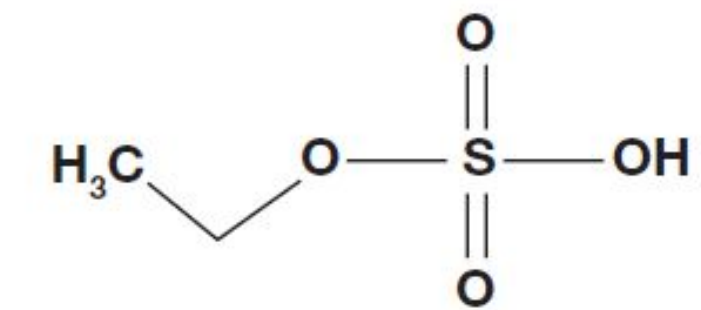
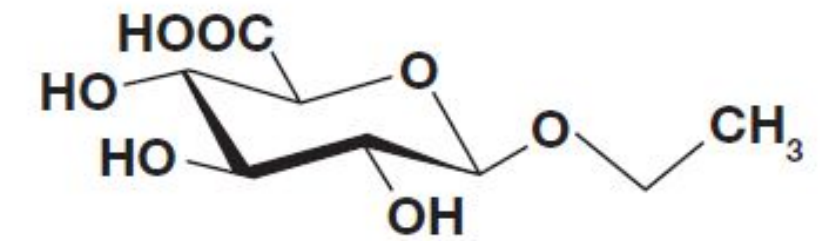
Table 1. Screening Results for the Patient, a 44-Year-Old Caucasian Man

Drug Screen Results		
Drug (Urine)	Result	Positive Cutoff Value
Amphetamines	Negative	500 ng/mL
Barbiturates	Negative	200 ng/mL
Benzodiazepines	Negative	200 ng/mL
THC	Negative	50 ng/mL
Cocaine	Negative	150 ng/mL
Ethanol	Positive	10 mg/dL
Creatinine	41.1 mg/dL	NA

Laboratory Medicine, Volume 49, Issue 3, August 2018, Pages 276–279

Positive Urine Alcohol with Negative Urine Ethyl-Glucuronide | Laboratory Medicine | Oxford Academic

- A urine ethyl-glucuronide (ETG) and ethyl sulfate (ETS) test was ordered on an aliquot of the urine (the same specimen that was already obtained and had tested positive for ethanol).
- The ETG/ETS are also metabolites of ethanol.
- The ETG/ETS test result came back 7 days later as being negative.



So how can a sample be positive for ethanol but negative for its metabolites?

- The presence of ethanol detected in the urine was not due to ingestion of ethanol but, rather, due to ethanol being formed as the product of microbial fermentation of glucose

Widmark's Blood Alcohol Calculator:**BAC for Women and Men**

Widmark's basic BAC estimator alcohol formula for calculating a person's estimated Blood Alcohol Content (BAC) at a particular time may be expressed as follows:

BAC Calculator Formula Widmark

$$\% \text{ BAC} = (A \times 5.14 / W \times r) - .015 \times H$$

A = liquid ounces of pure alcohol consumed

W = a person's weight in pounds

The r (rho) factor = a gender constant of alcohol distribution (0.73 for men and 0.66 for women), due to differences in the percentages of adipose tissue (body fat) for female subjects. The research indicated that women had higher fat content (on average) than men, due to the importance of bod fat when giving birth and nurturing babies.

H = hours elapsed since drinking commenced

Table 1. Blood Alcohol Concentration Table^[1-7]

Blood-Alcohol Concentration (percent)	# drinks for Male to achieve BAC _{a, c}	# drinks for Female to achieve BAC _{b, c}	Prominent Clinical Signs	Risk for Car Crash ^d
0.01-0.05	1.5-3.1	1.0-2.0	Behavior nearly normal by ordinary observation. Impairment detectable by special tests.	ND-4.3
0.02	1.9	1.2	Driving impairment starts around 0.02% BAC.	1.8
0.03-0.12	2.3-6.0	1.5-3.8	Mild euphoria, sociability, talkativeness. ↑ self-confidence; ↓ inhibitions, attention, judgment and control; loss of efficiency in critical performance tests.	2.4-33
≥0.04	2.7	1.7	Driving under the influence for professional driver with CDL license.	3.2
≥0.08	4.3	2.8	Driving under the influence for normal driver.	10
0.09-0.25	4.7-11.2	3.0-7.2	Emotional instability; ↓ in perception; memory and comprehension; sensory-motor in-coordination; impaired balance; slurred speech; vomiting; drowsiness.	14- >330
0.15	7.2	4.6	>50% of social drinkers are visibly intoxicated at 0.15 %.	78
0.20	9.2	5.9	84% of all drinkers (including heavy drinkers who develop tolerance) are visibly intoxicated at 0.20 %.	330
0.18-0.30	8.4-13.3	5.4-8.5	Disorientation; mental confusion; vertigo; exaggerated emotional states (fear, rage, grief); ↑ pain threshold; staggering gait; ataxia; memory loss; apathy.	185- >330
0.25-0.40	11.2-17.3	7.2-11.1	Approaching loss of motor function; Marked ↓ response to stimuli; inability to stand or walk; incontinence; impaired consciousness.	>330
0.35-0.50	15.3-21.4	9.8-13.6	Complete unconsciousness; coma; depressed reflexes; ↓ temperature; circulation and respiration impairment. Possible death from respiratory or cardiac arrest.	>330

Blood Alcohol Concentration (BAC) & Associated Clinical Signs – Expert Reference | Robson Forensic

08

ETHANOL in blood

Your blood alcohol concentration, or BAC, is the amount of alcohol in your blood.

For example, if your BAC is .05%, that means you have 50 milligrams of alcohol in 100 millilitres of blood.

In Canada, the **Criminal Code BAC limit is .08%**. This is the level at which Criminal Code impaired driving charges can be laid.

BACs (%) for Males in Relation to Time, Weight¹ and Standard Canadian Drinks²

Standard Drinks ³	2 hours						3 hours						4 hours					
	165 lbs.	180 lbs.	195 lbs.	210 lbs.	225 lbs.	240 lbs.	165 lbs.	180 lbs.	195 lbs.	210 lbs.	225 lbs.	240 lbs.	165 lbs.	180 lbs.	195 lbs.	210 lbs.	225 lbs.	240 lbs.
2	.020	.016	.012	.009	.007	.004	.005	.001	.000	.000	.000	.000	.000	.000	.000	.000	.000	.000
3	.045	.039	.034	.029	.025	.022	.030	.024	.019	.014	.010	.007	.015	.009	.004	.000	.000	.000
4	.070	.062	.055	.049	.043	.039	.055	.047	.040	.034	.028	.024	.040	.032	.025	.019	.013	.009
5	.095	.085	.076	.068	.062	.056	.080	.070	.061	.053	.047	.041	.065	.055	.046	.038	.032	.026
6	.120	.108	.097	.088	.080	.073	.105	.093	.082	.073	.065	.058	.090	.078	.067	.058	.050	.043
7	.145	.130	.118	.108	.098	.090	.130	.115	.103	.093	.083	.075	.115	.100	.088	.078	.068	.060
8	.170	.153	.139	.127	.117	.108	.155	.138	.124	.112	.102	.093	.140	.123	.109	.097	.087	.078

Provincial infraction⁴

Criminal offence⁵

ETHANOL: in field and onsite testing

Testing for ethanol in breath

- Ethanol is readily detectable in expired air.

Common field testing for alcohol



- Additional analysis is required using in lab ethanol testing of blood samples
- The usual blood:expired air ratio is 2300:1
- Alcohol analysis is typically performed in
 - A. driving under the influence (DUI) and
 - B. driving while intoxicated (DWI) investigations and
 - C. traffic accidents where people have been critically injured or killed

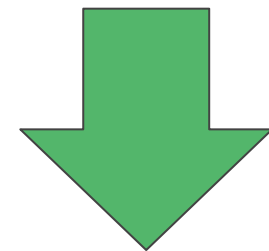


09. CanLII CASE STUDY

R v Dewey, 2016 ONSC 7536

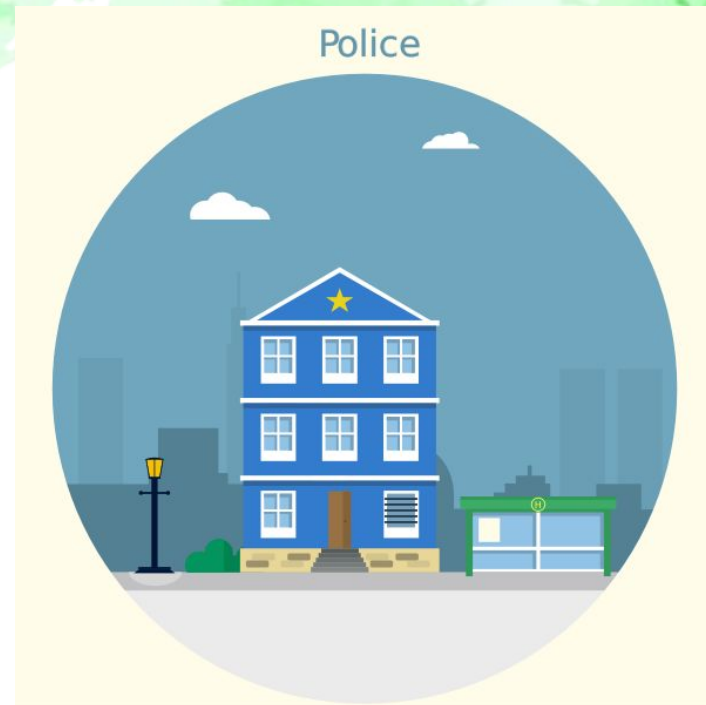
On August 10, 2011, prior to 6:00 p.m., Mr. Dewey had been operating a motor vehicle. He had been observed driving erratically, hitting the curb repeatedly, going through signs and a construction site. His vehicle eventually came to stop on the roadway. The vehicle was running. Mr. Dewey was passed out in the driver's seat. The wheels of Mr. Dewey's car had been damaged by his driving, the tires were flat and the rims were damaged.

- 1) **Police arrived**
Mr. Dewey's eyes were unfocused
There was a strong smell of alcohol on his breath.

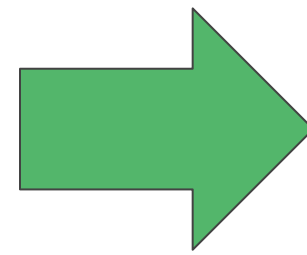


- 2) **Mr. Dewey was arrested around 6:00 p.m.**
Mr. Dewey's vehicle was about a 22 to 60 minute drive from The Keg restaurant depending on traffic. The distance travelled was approximately 18.3 kilometres.

R v Dewey, 2016 ONSC 7536



- At approximately 6:35 p.m., Mr. Dewey was taken before a qualified breath technician.
- Mr. Dewey refused to provide a breath sample



NOW AT THE PROCEEDINGS:

The Defence called Dr. Moftah

.....an expert on absorption and elimination of alcohol from the body and the effects of alcohol and drugs on the body.

R v Dewey, 2016 ONSC 7536

Dr. Moftah testified:

- 1) That a glass and a half of wine would result in less than 80 milligrams of alcohol in the blood
- 2) Mr. Dewey's blood alcohol concentration would have been approximately 32 milligrams of alcohol less whatever alcohol had been eliminated by Mr. Dewey's body while at the bar.

"body's elimination rate of alcohol being 10 to 20 milligrams of alcohol per hour"

- 3) this amount of alcohol alone would not produce Mr. Dewey's symptoms or behaviour observed at the scene where he was found stopped and asleep in his car.

Hence, Mr. Deweys

"blood alcohol level at approximately 6:00 p.m. that day would be very low."

"blood alcohol level would have been approximately zero milligrams of alcohol by the time he was in the breath room"

< 80 mg



*Elimination rate =
10 -20 mg/h*

List of References

List of References

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