

The Alcohol Connection in Drug-Facilitated Crimes

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Introduction

- Most common drug linked to rape
- Victims commonly consume voluntarily
- Can be administered in a social environment without suspicion
- Can cause blackouts and unconsciousness
- Less likely to be reported
- Prosecution more challenging than with others

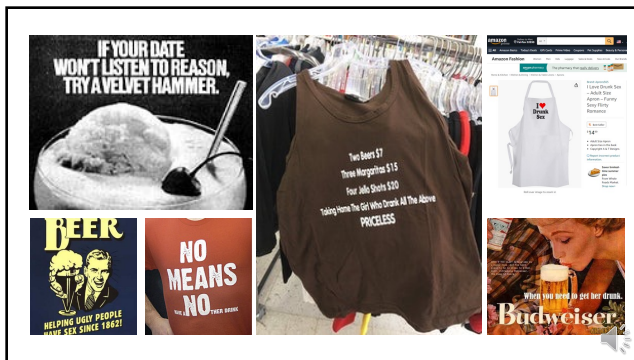
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Learning Objectives

- Society's view of using alcohol to get sex
- Standard alcoholic drinks versus the reality of what we consume
- Alcohol pharmacokinetics and the importance of rate of absorption
- Widmark-based estimations
- Dubowski's Stages of Alcohol Intoxication
- Alcohol-induced blackouts



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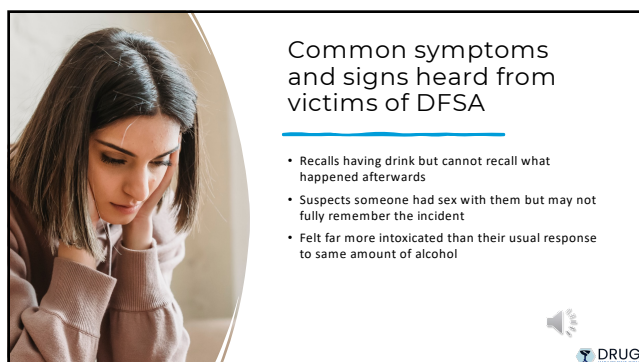
Prosecuting Alcohol-Facilitated Sexual Assault

- Alcohol use is common in many societies
 - Challenge of jurors thinking it was just consensual drunk sex
- Jurors may blame victim for putting herself into situation
- Research has shown that individuals tend to view women who drink or get drunk as more sexually available, and more likely to engage in sexual acts than women who abstain from alcohol
- Complicated by the effects of alcohol
 - Victims may not be able to clearly perceive and/or remember the details of the assault
- Three-step process:
 - Making the charging decision
 - Assessing credibility and corroboration
 - Trying the case

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CHALLENGES SURROUNDING DFC INVESTIGATIONS

Drugs

- Dosages
- Number of Candidates
- Pharmacokinetics
- Pharmacodynamics



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ETHANOL IS A CNS DEPRESSANT, BUT IT IS NOT ADMINISTERED OR USED IN "LOW DOSES"



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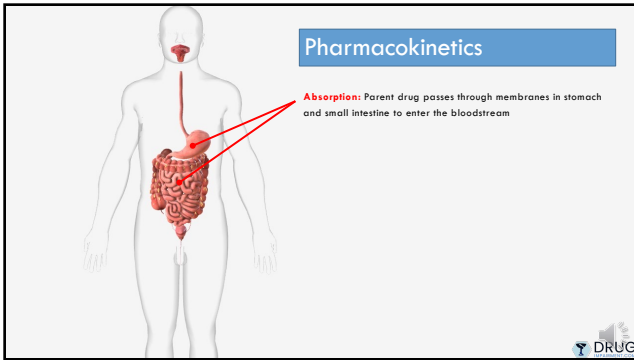
STANDARD ALCOHOLIC DRINKS

12 oz	5 oz	1.5 oz
		
5%	12%	80 proof (40%)

EACH CONTAIN ABOUT 14 GRAMS (0.6 OUNCES) OF PURE ETHANOL



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FACTORS THAT AFFECT ETHANOL'S ABSORPTION INTO THE BLOOD

Enhances Absorption: Faster drinking	Slows Absorption: Slower drinking
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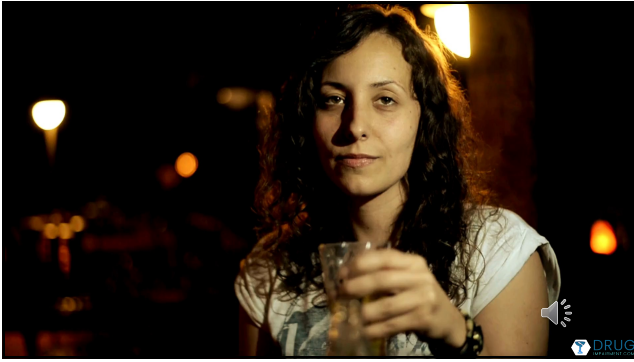
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FACTORS THAT AFFECT ETHANOL'S ABSORPTION INTO THE BLOOD

Enhances Absorption: Faster drinking Carbonated beverages	Slows Absorption: Slower drinking "Fatty" beverages
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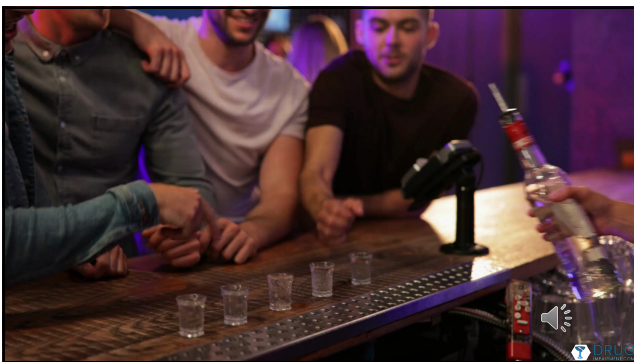
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FACTORS THAT AFFECT ETHANOL'S ABSORPTION INTO THE BLOOD

Enhances Absorption:	Slows Absorption:
Faster drinking	Slower drinking
Carbonated beverages	"Fatty" beverages
Moderate ethanol concentration	Low or high ethanol (neat) concentration




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No to low sugar content	High sugar content
Being dehydrated	Being well hydrated

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FACTORS THAT AFFECT ETHANOL'S ABSORPTION INTO THE BLOOD

Enhances Absorption:

- Faster drinking
- Carbonated beverages
- Moderate ethanol concentration
- No to low sugar content
- Being dehydrated
- Recent exposure to higher altitudes

Slows Absorption:

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Medications that speed up gastric emptying to help control acid reflux or to prevent nausea/vomiting (i.e. prokinetics) such as *Prucisid (cisapride)* or *Reglan (metoprolamide)*



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Medications that slow down gastric emptying (e.g., opioids, calcium channel blockers, diphenhydramine, tricyclic antidepressants, aluminum hydroxide antacids, phenothiazines)



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Empty stomach*

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Eating before or while drinking*



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Eating before or while drinking*
 * Amount eaten is probably more important than composition of meals. Fat is particularly important.



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DON'T FORGET THE OTHER KEY FACTORS THAT IMPACT ETHANOL ABSORPTION

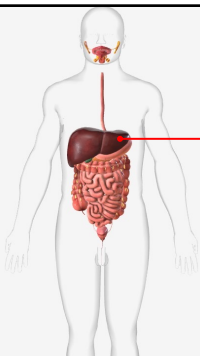
Remember to ask:

- What did they drink?
- How many did they drink?
- What size were the drinks?



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Pharmacokinetics

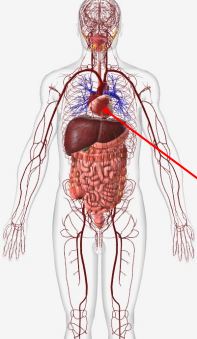


Absorption: Parent drug passes through membranes in stomach and small intestine to enter the bloodstream

Metabolism: Parent drug is modified into a different chemical substance (metabolite) that generally makes it more hydrophilic. Most metabolism occurs via enzymes located in the liver or in the blood.



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Pharmacokinetics

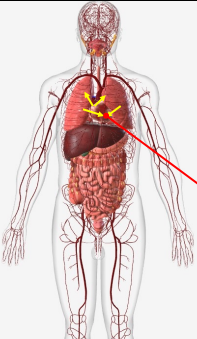
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Pharmacokinetics

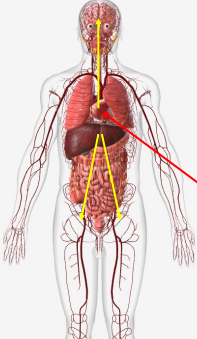
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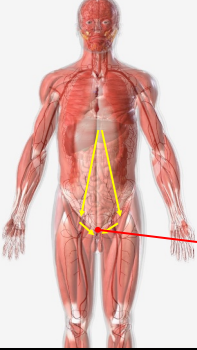
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Distribution: Drug is transported throughout the body to different organs where it can move into the tissues to have an effect

Excretion: Parent drug and metabolites are leave the body, most often through urine



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ETHANOL DISTRIBUTION

Volume of Distribution (V_d) relates concentration of a drug (alcohol) in blood (or plasma) to the total amount of drug in the body

- Total body water (TBW) is about 60% of body weight in men and 50% in women
- Blood is ~80% water

V_d for ethanol is impacted by person's age, gender, and proportion of fat to lean tissue in the body


Because ethanol distributes into the water fraction of all body fluids, the distribution ratio of ethanol between the blood and body as a whole should follow similar distribution as water:

- ~60%/80% = ~0.7 for men
- ~50%/80% = ~0.6 for women

But body composition (BMI) is very important

- V_d can be as low as 0.4-0.5 L/kg in obese females to as high as 0.7-0.8 L/kg in lean males

Current recommendations suggest using a fixed V_d range of 0.45 to 0.81 L/kg or to calculate an individualized V_d based on TBW



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WIDMARK FORMULA TO ESTIMATE A BAC

$$BAC = \frac{D}{V_d * w}$$

BAC = blood alcohol concentration (g/L)*
 D = dose of alcohol (g)
 V_d = volume of distribution (L/kg)
 w = weight (kg)

* BAC is typically expressed as g/dL, so proper conversion is important

Useful exercise to evaluate the role that ethanol may have played in contributing to the symptoms that the DFC victim described



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ETHANOL METABOLISM AND ELIMINATION


Primarily metabolized and eliminated through oxidative metabolism in the liver

Ethanol → Acetaldehyde → Acetic Acid → CO₂ + H₂O

Small amount is excreted unchanged in breath, sweat, oral fluid, and urine

Eliminated at a constant, linear rate until low concentrations are reached

- Rates range between about 0.010 – 0.025 g/100 mL per hour (until low BAC)
- Factors can impact this such as alcoholism (faster), genetic factors, liver disease (slower), and food (slightly enhances)
- Can be used to back-extrapolate a blood alcohol concentration to an earlier time




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ETHANOL METABOLISM AND ELIMINATION

Secondary non-oxidative metabolic pathways may also be important

- Small amount of ingested ethanol produces ethyl glucuronide (EtG) and ethyl sulfate (EtS)
 - Detectable for considerably longer periods of time than ethanol itself, can serve as biomarker for recent drinking
- Ethanol and phosphatidylcholine can form phosphatidylethanol (Peth)
 - Requires heavier drinking period to produce this
 - Also detectable after ethanol has been cleared




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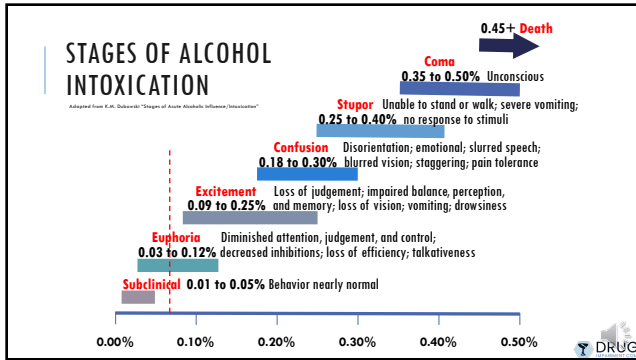
CNS DEPRESSION OF ETHANOL

General relationships between BAC and the CNS depressant effects on the brain

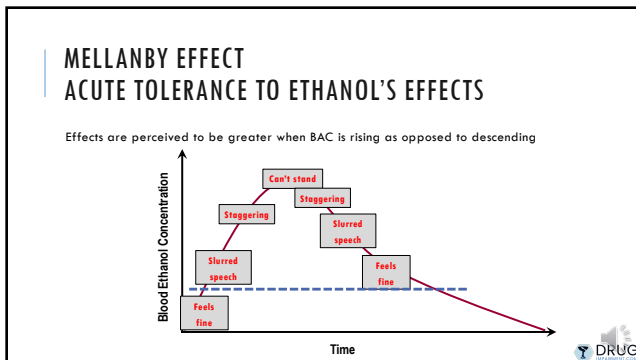
Alcoholics become habituated to ethanol so can function at high BACs, particularly when performing relatively simple tasks



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PUTTING IT ALL TOGETHER AND APPLYING THE WIDMARK EQUATION

24-year-old female (5'1" / 140 lbs) consumed 2 glasses of wine and 1 shot of vodka between 8:00 PM and 11:30 PM. At about 11:45 PM, she was reported as being unable to walk or stand on her own and seemed to be falling into a deep sleep while seated. A male friend helped her out of the bar at about midnight and took her to his place that was about 10-minute walk from the bar.

She woke the next morning to the sound of a running shower. She had no memory of how she got from the bar to his apartment. She was only wearing her panties and noticed a used condom in a nearby trashcan.

She quickly dressed and got out of the apartment before her friend got out of the shower.

Could alcohol alone cause the symptoms she experienced?

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PUTTING IT ALL TOGETHER AND APPLYING THE WIDMARK EQUATION

$$BAC = \frac{D}{V_{d*}w}$$

BAC = blood alcohol concentration (g/L)*

D = dose of alcohol (g): 3 standard drinks x 14 g/drink = 42 g

V_d = volume of distribution (L/kg): Use individualized calculated value of 0.56 L/kg (range 0.45 to 0.74)

w = weight (kg): 140 lbs x 0.454 kg/lb = 63.6 kg

$$BAC_{low} = \frac{42 g}{\frac{0.74 L}{kg} * 63.6 kg} = 0.892 \frac{g}{L} * \frac{1 L}{10 dL} = 0.089 \frac{g}{dL}$$

$$BAC_{high} = \frac{42 g}{\frac{0.45 L}{kg} * 63.6 kg} = 1.468 \frac{g}{L} * \frac{1 L}{10 dL} = 0.147 \frac{g}{dL}$$

But what about the impact of the alcohol being metabolized and eliminated? Drinks were consumed between 8:00 PM and 11:30 PM



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May not want to assume ANY alcohol was eliminated...but likely that some was.
May want to assume just 1 hour of elimination.



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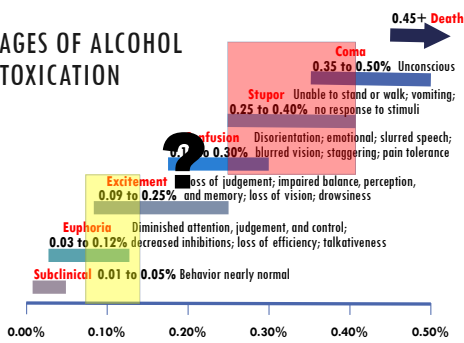
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STAGES OF ALCOHOL INTOXICATION

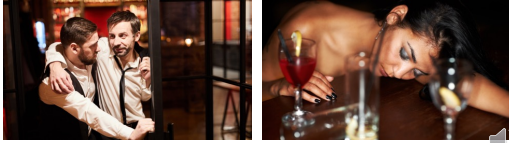


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ALCOHOL BLACKOUTS vs PASS OUTS

Blackout (Amnesia)

Pass Out (Unconscious)



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ALCOHOL-INDUCED BLACKOUTS

Memory loss (anterograde amnesia) that occurs during any part of a drinking episode without loss of consciousness

Will be awake and conscious, MAY be engaged in activity or conversation, and MAY appear to be somewhat oriented

Memory loss may be significant, but may be reversible
Information may be recalled later, sometimes spontaneously

Women seem to be more susceptible to blackouts and undergo a slower recovery from cognitive impairment than men



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AMNESIA

Retrograde

- Loss of earlier memories
- Usually from cerebral injuries or disease states
- Far more common

Anterograde

- Impaired information acquisition, consolidation, and storage
- Alcohol and some CNS depressants cause
- Likely due to impact on GABA-receptor complex
- Less clear when these end because people tend to fall asleep before they are over

Goodwin, D.R. et al. Am J of Psychiatry 66 (1975)


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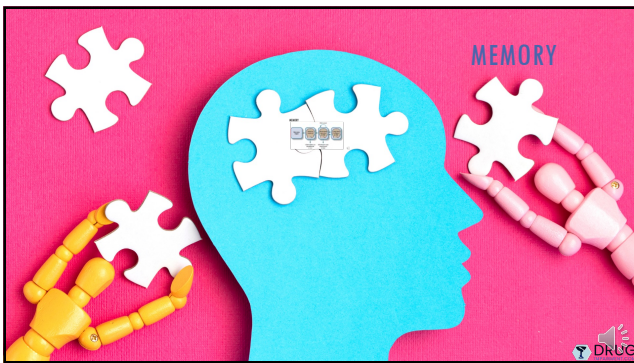
ALCOHOL-INDUCED BLACKOUTS

Two types of alcohol blackouts

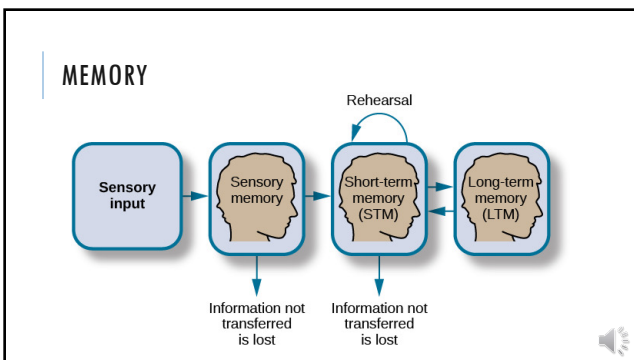
- Complete (en bloc)
 - Begins and ends at definitive points
 - Full permanent amnesia
 - Loss of time
 - Requires high BACs to disrupt memories from encoding
- Fragmentary (Gray Out)
 - Memories often recalled when prompted
 - Occur more often
 - Experienced over wider range of BACs



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ALCOHOL-INDUCED BLACKOUTS

Two mechanisms:

- Encoding deficit
 - Alcohol temporarily inhibits biochemical processes in brain needed to form new memories
- Retrieval deficit
 - Information stored as a memory while intoxicated is inaccessible when the individual is sober

Ingestion of large amounts of alcohol may have more significant effect on input, acquisition, or processing of new memories


Research suggests alcohol-induced blackouts disrupt transfer of information from short-term to long-term storage



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PROBLEMS WITH BLACKOUT STUDIES

- Most have involved only white, male alcoholics
- Many have relied upon subject's ability to recall previous episodes of blackout months or years before
 - "remember not remembering"
 - Number of drinks consumed over what period of time?
- Modern Internal Review Boards would not likely approve of normal subjects' "binge" drinking to increase BACs to levels consistent with blackouts




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RISK FACTORS FOR ALCOHOL-INDUCED BLACKOUTS

- Prior or current history of alcoholism
- Family history of alcoholism
- Age of drinking onset
- Prior history of blackouts
- Family history of blackouts
- Capacity/tolerance for high quantities of alcohol
- Ingests high quantity of alcohol such that they often exceed 0.2%
- Rapid drinking – gulping or bingeing
- Head trauma
- Loss of control
- Failure to eat properly

Fresman, ME and Cavill, DS, J Forensic Sci, Vol 25, 4, 2013



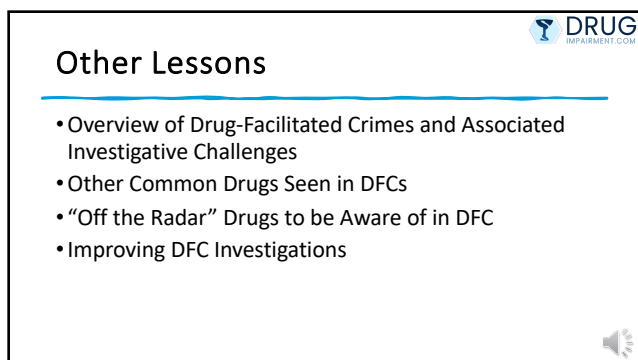
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