Cognition and Psychopathology

Lecture 7: Substance Use and Abuse

Various substances can cause alterations in mood, cognitive processes, and other mental states.

Although these states may be intentionally sought after, substance ingestion can, under some conditions, be cause for psychiatric concern.
DSM-IV notes numerous substance-specific disorders. These disorders are broken into two large categories:

- Substance Use Disorders
  - Substance Dependence and Substance Abuse

- Substance-Induced Disorders
  - e.g., Intoxication, Withdrawal, Substance-Induced Delirium, Substance-Induced Mood Disorder, etc.).

Eleven classes of substances are addressed by DSM-IV:

- alcohol; amphetamine and related drugs; caffeine; cannabis; cocaine; hallucinogens; inhalants; nicotine; opioids; PCP and related drugs; and sedatives, hypnotics, and anxiolytics.

- Other substances are included in a catch-all category (including unintentional toxin exposure).
Substance Dependence Disorder involves a cluster of cognitive, behavioural, and physiological symptoms demonstrating that the individual continues to use a substance despite significant substance-related problems. Diagnosis involves three or more of:

- tolerance
- withdrawal
- greater consumption than was intended
- persistent desire to reduce consumption that is unsuccessful

- great deal of time spent in substance acquisition, use, or recovery
- activity reduction due to substance use
- continued use despite knowledge of persistent psychological or physiological problems caused or exacerbated by the substance

(Appplies to all classes of drug except caffeine.)
Substance Abuse is a maladaptive pattern of substance use involving recurrent and significant adverse consequences related to repeated use of a substance (such as failures at work or home, use in physically hazardous situations, legal problems, or interpersonal problems). Unlike Dependence, Abuse does not involve tolerance, withdrawal, or a pattern of compulsive use.

(The category does not apply to caffeine or nicotine.)

Understanding theEffects of Substance Use

Most research investigating substance use has focussed on alcohol (it is a widely used drug; its abuse is a large social problem; it is a legal drug, and therefore has few restrictions on performing research with it).

Although models of factors in alcohol use may be specific, many of the principles transfer to other drugs.

(There are physiological and psychological accounts for a variety of different substance-related phenomena. We will concentrate on cognitive accounts of the effects that substances have.)
Physiological accounts of drug effects

Most early accounts of drug effects, including dependence, relied on physiological models.

- Substances produce physiological changes that induce desirable states, and after prolonged use failure to ingest that substance can produce the undesirable state of withdrawal.

- The psychological effects a drug produced were thought to directly result from their physiological impact.

- E.g., for alcohol, it was assumed that its behavioural effects (reduction of anxiety, disinhibition) were due to pharmacological effects that it had on perceptual, cognitive, or motivational states.
Research under this framework usually involved having subjects consume either alcohol or a placebo, and then observing them with respect to some behaviour (e.g., aggression).

However, this view assumes that subjects who think they have consumed alcohol but haven't (placebo group) are appropriate controls, i.e., that that belief does not affect their behaviour.
Cognitive Factors in Substance Use

More recent approaches have emphasized the role that beliefs about a drug's effects have on the behaviour that occurs when that drug is taken.

A central concept in this cognitive approach to substance use is expectancy.

Expectancy

Expectancy is a concept taken from general cognitive psychology.

- the relationship between objects, events, stimuli, etc. that an individual believes exists (regardless of the accuracy of that belief).

In the context of substance use research, expectancy has come to mean the effects that an individual anticipates a substance will have.
Earlier versions of this view involved *conditioned response* to substance use

- cues associated with, e.g., drinking, were thought to act as a conditioned stimulus, and elicit a conditioned state of intoxication.

More interesting, however, has been the work that has explored how expectancies themselves produce cognitive, affective, and behavioural changes.

Under this view, it is the actual belief about the substance's effects that has an influence on behaviour (perhaps, for example, through the attributions one makes about one's behaviour while under the influence).

- E.g., alcohol may in part be disinhibiting because individuals feel excused for their behaviour due to their beliefs in alcohol's disinhibiting properties ("it wasn't my fault, I was drunk at the time").
A methodology that has become fundamental to the examination of expectancies in substance use is the balanced placebo paradigm, an improvement over the original placebo research.

In this paradigm, two factors are fully crossed:

- whether a subject receives a substance or placebo,
- whether they are told they will receive a substance or placebo.

In this way, one can look at conditions that only involve expectancies (receive placebo, told alcohol), and only involve pharmacological effects (receive alcohol, told placebo).

This latter condition tests for physiological effects without expectancies, and also serves as an interesting control condition when looking at expectancies.
Effects of expectancies on alcohol behaviours

The research on expectancies in alcohol use using balanced placebo designs was examined by Hull and Bond (1986).

They looked at physiological (alcohol consumption) and psychological (alcohol expectancy) effects across 34 studies by performing a meta-analysis (a statistical approach to summarizing the results of multiple studies).

Analysis was conducted in part to compare the conditioned response view of expectancies (where expectancies should have similar effects to actual consumption) and the attributional account (consumption provides excuse to engage in deviant/illicit acts).
They concluded that:

- Alcohol consumption impairs memory, increases mood, and increases certain internal sensations (e.g., feelings of warmth, anesthesia). Expectancies do not appear to impact on these effects.

- By contrast, alcohol expectancies reliably increase sexual arousal, and increase further consuming of alcohol. Alcohol itself appears to have no physiological influence on these effects.

Overall, these results suggest that:

- expectancies can independently influence alcohol-related behaviours (importantly, one such behaviour is further consumption of alcohol)

- expectancies do not appear to follow conditioned responses, but primarily influence deviant/illicit behaviours (attribution account)
Further studies have shown that expectancies influence consumption patterns, with heavier drinkers showing greater expectation of social and physical pleasure, assertiveness, and tension reduction.

Expectancies can also work against physiological effects, by causing the individual to compensate.
Williams, Goldman, and Williams (1981):

- balanced placebo design

- subjects perform a number of cognitive tasks: a letter cancellation task (cross out as many Cs as possible on a large sheet of random letters in one minute), a digit span task (recall as many digits as possible in correct order from a list), and a standardized task measuring abstract intelligence (Raven's Progressive Matrices).

Williams et al. found that, when individuals were administered alcohol but expected placebo, their performance on these tasks deteriorated.

However, when correctly told that they would receive alcohol, performance was much better (and close to normal, placebo/placebo performance for the two easier tasks of letter cancellation and digit span).
Williams et al. concluded that expectations about alcohol's impairing effects helped individuals to compensate when they knew they were receiving alcohol. At the dosages used, this compensation allowed normal performance for less challenging tasks, and less-impaired performance for the more difficult task.

Effects of expectancies on caffeine behaviours

Although most expectancy work has been done with alcohol, this approach has been used with other substances as well, such as caffeine.

Although there is no doubt that caffeine is a stimulant, and can produce changes in alertness and mood through physiological means, can some of its effects be attributed to expectancies?
Kirsch and Weixel (1988):

- subjects drink a cup of coffee that was made with from 1 to 8 teaspoons of ground coffee.

- One subject group *(deceived)* was told that the coffee was caffeinated, another *(double-blind)* was told they were in a double-blind trial, and that the coffee might be decaffeinated.

- In reality, all coffee used was decaf.

Results:

- for deceived group, an increase in apparent dosage caused increases on measures of alertness, tension, and even systolic blood pressure and pulse rate (for all but the highest dose).

- double-blind group, who thought they might receive decaf, actually showed a decrease in these measures as dosage increased.
Kirsch and Weixel concluded that expectancies can have a strong impact on effect of caffeine. (They also argued that, because of the different results in the double-blind and deception conditions, the double-blind design for drug testing is not ideal. What expectancies are active under double-blind conditions is not clear. They advocated using balanced-placebo designs when testing new drugs to ensure that physiological and expectancy effects are explicitly measured.)

Fillmore and Vogel-Sprott (1992) also found expectancy effects for caffeine. In this study, subjects performed a motor task. The pursuit rotor task involved using a computer mouse to guide a crosshair onto a small target following a circular path around a computer screen. Percentage of time on target (% TOT) was the primary measure.
Prior to the task, some subjects were either told that caffeine would enhance performance [E(+)\)], or decrease performance [E(-)]. These subjects, and a further group that was given no information about caffeine effects [E(?)] , then drank a cup of strong-tasting coffee that actually contained no caffeine. These groups, and a fourth that received no coffee or information on caffeine effects [E(0)] performed the pursuit rotor task.

As anticipated, E(+) performed better on the task than the two control groups E(?) and E(0), and E(-) performed worse than the controls.

In addition, all coffee groups showed changes in alertness and tension that were in keeping with their prior expectations of caffeine's effects.
To what extent are expectancies automatically activated?

Expectancies can affect behaviour when individuals are explicitly aware of them. To what extent can such expectancies be automatically activated?

A few studies suggest expectancies can indeed become active without conscious intervention.

Chenier and Goldman (1992) had subjects complete word fragments, involving both expectancy and neutral words, after either sitting in a bare room or a room with alcohol advertising. Although unaware of the importance of the room condition, subjects who earlier saw the advertising completed more expectancy words.

Roehrich and Goldman (1992) found that doing a Stroop task involving alcohol expectancy words increased consumption of beer in a later, ostensibly unrelated, task.