

## **Cognitive Factors in Adolescent Substance Abuse: Antecedents or Consequences?**

### **Introduction**

Consider two adolescents: Person One and Person Two. Person One has never used substances and exhibits relatively few behavioral (conduct) problems. On the other hand, Person Two uses substances frequently and more commonly exhibits poor conduct. If you were to guess, which of the individuals would you expect to have lower overall judgment and self-regulation? If you answered Person Two, your intuition is correct. Scientific studies have confirmed our intuition; individuals with substance use and other behavioral problems have been found to have poorer judgment and self-regulatory skills. While the association with lower judgment and self-regulation is seemingly obvious and commonsensical, the true relationship between poor judgment and substance abuse is less clear. Namely, how do we know if drug use causes poor judgment or poor judgment and low self-regulation are risk factors for substance abuse? Behavioral genetic researchers at the Institute for Behavioral Genetics and other research institutes are designing research to decipher just this.

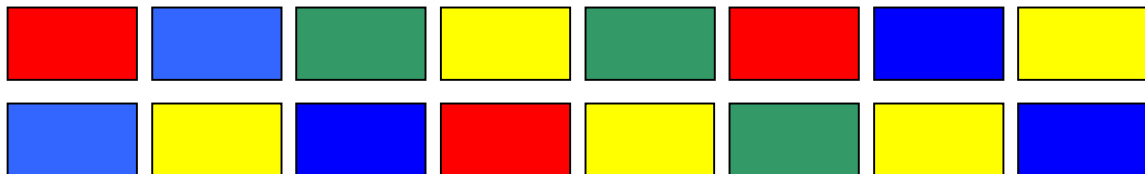
### **How do scientists measure abstract traits like judgment and self-regulation?**

To test one of the observed differences between Person One and Person Two, scientists developed the term executive cognitive functioning or ECF. ECF is broadly defined as the self-regulation of goal-directed behavior. In most contexts, it can be likened to judgment and/or self-control. Scientific studies use a variety of names for ECF including: neurobehavioral disinhibition, low self-regulation, behavioral disinhibition, inhibitory control, low constraint, and behavioral under-control. ECF can be quantitatively tested with psychological tests. For example, the Stroop test is a common measure of ECF that is thought to assess selective visual attention (a specific type of self-regulation). To experience the Stroop first hand, time how long it takes you to say the names of the ink colors out loud while ignoring the meaning of the words. Go through the list five times.

**RED BLUE GREEN YELLOW GREEN RED BLUE RED**

**YELLOW GREEN RED BLUE GREEN BLUE RED GREEN**

Now, for comparison, time yourself naming the colors of the rectangular blocks five times.



What you probably observed is that it takes longer to say the colors while inhibiting reading the words than to just say the names of the block colors. This is known as the Stroop Effect. In the case of substance abuse research, a scientist might use the Stroop test to determine if the difference in task-completion times between Person One and Person Two is significant.

### How do scientists measure substance use and behavioral problems?

To compare differences in substance involvement and conduct problems between Person One and Person Two, a clear definition of what constitutes a drug or conduct problem must be utilized. Most research is guided by the Diagnostic and Statistical Manual IV (DSM-IV). The DSM-IV defines substance dependence, substance abuse, and conduct disorder in detail. Using this as a reference, researchers use in-person interviews or questionnaires to determine if the participant meets criteria for drug abuse, drug dependence, and/or conduct disorder. The following tables display the DSM-IV criteria for substance dependence, substance abuse, and conduct disorder.

#### DSM IV Criteria

##### Substance Dependence

A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following, occurring at any time in the same twelve-month period:

- (1) tolerance, as defined by either of the following: (a) a need for markedly increased amounts of the substance to achieve intoxication or desired effect; (b) markedly diminished effect with continued use of the same amount of the substance;
- (2) withdrawal, as manifested by either: (a) the characteristic withdrawal syndrome for the substance (refer to Criteria A and B or the criteria sets for withdrawal from the specific substances); (b) the same (or closely related) substance is taken to relieve or avoid withdrawal symptoms;
- (3) the substance is often taken in larger amounts or over a longer period than was intended;
- (4) there is a persistent desire or unsuccessful efforts to cut down or control substance use;
- (5) a great deal of time is spent in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances), use the substance (e.g., chain smoking), or recover from its effects;
- (6) important social, occupational, or recreational activities are given up or reduced because of substance use;
- (7) the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (e.g., current cocaine use despite recognition of cocaine-induced depression, or continued drinking despite

#### DSM IV Criteria

##### Substance Abuse

**A.** A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by one (or more) of the following occurring within a twelve-month period:

- (1) recurrent substance use resulting in failure to fulfill major role obligations at work, school, or home (e.g., repeated absences or poor work performance related to substance use; substance-related absences, suspensions, or expulsions from school; neglect of children or household);
- (2) recurrent substance use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by substance use);
- (3) recurrent substance-related legal problems (e.g., arrests for substance-related disorderly conduct);
- (4) continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication, physical fights).

**B.** The symptoms have never met the criteria for Substance Dependence for this class of substance.

SOURCE: APA, DSM-IV, 1994.

## DSM IV Criteria

### Conduct Disorder

- A.** A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, as mandated by the presence of three (or more) of the following criteria in the past 12 months, with at least one criterion present in the past 6 months: *Aggression to people and animals*—(1) often bullies, threatens, or intimidates others; (2) often initiates physical fights; (3) has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun); (4) has been physically cruel to people; (5) has been physically cruel to animals; (6) has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery); (7) has forced someone into sexual activity. *Destruction of property*—(8) has deliberately engaged in fire setting with the intention of causing serious damage; (9) has deliberately destroyed others' property (other than by fire setting). *Deceitfulness or theft*—(10) has broken into someone else's house, building, or car; (11) often lies to obtain goods or favors or to avoid obligations (i.e. "cons" others); (12) has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entry; forgery). *Serious violation of rules*—(13) often stays out at night despite parental prohibitions, beginning before age 13 years; (14) has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period); (15) is often truant from school, beginning before age 13 years.
- B.** The disturbance in behavior causes clinically significant impairment in social, academic, or occupation functioning.
- C.** If the individual is age 18 years or older, criteria are not met for Antisocial Personality Disorder.

### **What differences exist between non-substance abusing populations and substance abusing populations in terms of executive cognitive functioning?**

In terms of cognitive psychological tests, researchers have reported differences between substance abusing populations and controls, namely that individuals with a substance disorder have reduced goal persistence, abstract reasoning, cognitive flexibility, attentional control, concept formation, and verbal fluency (Giancola & Moss, 1998). Physiologically, brain-imaging studies have shown that substance-abusing populations differ neurologically from other populations in that their anterior cingulate cortex and orbitofrontal cortex are less responsive to drug-related stimuli; they exhibit overall lowered brain activation; dopamine function in cocaine-using populations is reduced; and fewer benzodiazepine receptors are found in populations with alcohol dependence (Daglish et al., 2003). Although the previous research demonstrates ECF differences between substance abusing populations and non-substance abusing populations, it does not establish a causal relationship. Namely, it does not decipher if diminished ECF is a consequence or antecedent of problematic drug use.

### **What research has been done on the causal relationship between executive cognitive functioning deficits and substance abuse problems?**

Researchers have begun to resolve this relationship by studying the ECF of adolescents who are considered high-risk for substance use disorder before the age of substance use onset. This high-risk paradigm sorts children according to high (parents or siblings who have SUD) and low (parents do not have SUD) risk pathways. Because children are studied before the age of onset, differences between control and high-risk groups cannot be attributed to drug use and are therefore found to be potential SUD risk factors (Giancola et al., 1999).

Research constructed under the high-risk paradigm has accrued evidence that poor ECF is associated with increased SUD risk. Nigg et al. (2004) tested a total of 198 families including 69 community control families, 129 families with alcohol use disorder

(AUD), 35 families with antisocial personality disorder (ASPD), and 54 families without ASPD. AUD families were selected from drunk-driving convictions and a Feighner diagnosis of alcoholism. Boys were tested three to four times from ages 3-5 to 12-15 with the Stanford-Binet (Terman & Merrill, 1973), the Delay of Gratification Task (Funder, Block, & Block, 1983), the WISC-R (Weschler, 1974), the Wisconsin Card Sorting Test (Heaton et al., 1993), the Stopping Task (Logan & Cowan, 1984), the Symbol-Digit Modalities Test (Smith, 1991), Controlled Oral Word Association Task (Benton & Hamsher, 1978), Stroop Color-Word Inference Test (Golden, 1978), and the Tower of Hanoi Procedure (Lezak, 1995). These assessments included paper-and-pencil self-reports and laboratory assessments. Although high-risk boys from families with AUD and ASPD did not differ in ECF from controls, high-risk boys from families with non-antisocial AUD greatly differed from controls demonstrating that poor ECF may contribute to a risk pathway. However, this research may not generalize to girls nor predate alcohol use due to the late age of testing (13-15). Similarly, in a sample of 275 boys ages 10-12, high-risk SUD children (defined as having fathers with SUD) demonstrated lower ECF than controls. ECF predicted tobacco and cannabis use, total number of drugs tried, and severity of drug involvement (Aytaclar et al., 1999).

Young et al. (2000) investigated behavioral disinhibition as a combination of conduct disorder, attention deficit hyperactivity disorder, substance experimentation, and novelty seeking symptoms in 334 adolescent twin pairs. They found behavioral disinhibition to be highly heritable (i.e. influenced by genetic factors). In addition, neurobehavioral disinhibition has been found to predict SUD and an early age of SUD onset. Tarter et al. (2004) examined 170 high-risk and control boys at age 10-12 and again at age 19 and concluded that parental SUD predicted child neurobehavioral disinhibition that in turn predicted SUD between the ages of 10-12 and at 19. In another study, Tarter et al. (2003) found that neurobehavioral disinhibition at age 16 predicted SUD with 85% accuracy at the age of 19.

Low inhibitory control has similarly been labeled a SUD risk factor in neurophysiological research. The P300 amplitude of the event-related potential (ERP) has been linked to inhibitory control: reduced P300 amplitude corresponds to low inhibitory control (Taylor et al., 1999). Research utilizing P300 amplitude as a measure of neurobehavioral disinhibition can be used to distinguish the antecedent or consequent relationship of observed ECF differences. For example, if reduced P300 amplitude is a physiological marker of low inhibitory control, it can be measured in high-risk subjects (i.e. children or siblings of individuals with SUD) who have not initiated substance experimentation. In such a research design, differences in P300 amplitude between high-risk and low-risk individuals cannot be attributed to the effect of substance use.

In one inhibitory control study, Brigham et al. (1995) examined fifty-four 10-12 year old boys, of which 28 were considered high-risk. Due to the early age of testing, it was assumed that subjects had not engaged in substance use. Participants were gathered from clinical substance treatment facilities, newspaper advertisements, and public service announcements. High-risk was defined as having a biological father with a lifetime diagnosis of the DSM-III-R Psychoactive Substance Use Disorder as

determined by the SCID (Spitzer & Williams, 1983). An auditory ERP oddball task was given to subjects in the laboratory. Findings indicated group differences in the P300 amplitude with the high-risk group having smaller overall amplitudes and thus reduced inhibitory control. Limitations of the study include a small sample size, restricted generalization due to the lack of female subjects, and incomplete subject drug use histories.

Hill et al. (1999) expanded on the previous study by examining the cause of reduced P300 amplitude in high-risk children. One-hundred fifty-six children of both sexes from high- and low-risk families between the ages of 8 and 18 and their parents were interviewed with the Schedule for Affective Disorder and Schizophrenia for School-Aged Children (K-SADS) (Chambers et al., 1985). High-risk families were defined as having at least two adult alcoholic brothers. Children were assessed longitudinally in the laboratory with an auditory and visual ERP task. The study found that reduced P300 amplitude (inhibitory control) in high-risk boys was due to developmental delay. That is, the P300 response recorded was similar to the amplitude expected from a younger, less developmentally mature age group. The study is limited by the late age of testing (18) that may not predate substance use.

Utilizing another method of neurophysiological research, Schweinsburg et al. (2004) conducted functional magnetic resonance imaging (fMRI) scans of 12-14 year old children of both sexes while they participated in a go/no-go task in the laboratory. The study consisted of 12 high-risk youth (youth with one parent or two second-degree relatives with AUD) and 14 low-risk youth (youth with no family history of AUD) with no previous substance use. Although restricted by small sample size, results indicated that high-risk youth have reduced response inhibition within the frontal lobe in the left middle frontal gyrus, the left medial/superior frontal, bilateral middle frontal, right superior frontal, and right inferior frontal gyri. Outside the frontal lobe, high-risk youth demonstrated low response inhibition in the right temporal gyrus, right precuneus/superior parietal lobule, and bilateral inferior parietal lobule.

## **Conclusion**

In summary, there is considerable literature substantiating correlations between ECF deficits and substance abuse research. However, most of this literature has demonstrated cognitive deficits in substance abusing populations, making it impossible to determine whether ECF deficits are risk factors or consequences of chronic substance use. Clearly, some of the cognitive deficits seen in substance abusing populations are likely to be consequences of chronic substance use. However, high-risk paradigms, assessing subjects at risk for SUD—but who have not yet initiated substance use, are beginning to provide evidence that ECF deficits may also be important risk factors for substance abuse vulnerability. Future research, conducted at the Institute for Behavioral Genetics and other research institutes, will provide evidence on the consequent or antecedent relationship between ECF deficits and substance abuse problems.

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