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# Individual differences in electrodermal responsivity to predictable aversive stimuli and substance dependence

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## Abstract

To determine if the inability to take advantage of the predictability of an aversive stimulus to diminish its psychological impact reflects a deficit in inhibitory control related to the development of substance dependence, we recorded skin conductance responses (SCRs), heart rate (HR), and anticipatory electrodermal nonspecific fluctuations (NSFs) from 175 16–18-year-old boys when a white noise blast was either unpredictable or temporally predictable. Compared with boys who had moderately reduced or augmented SCRs to predictable blasts (moderate and poor modulators, respectively), boys whose SCRs were greatly reduced (good modulators) had fewer symptoms of alcohol and nicotine dependence and more anticipatory NSFs. HR appeared to index an active coping response for good and moderate modulators. The autonomic response pattern evident for good modulators may index an inhibitory control mechanism protecting them from developing substance dependence.

**Descriptors:** Inhibitory control, Substance dependence, Skin conductance responsivity, Nonspecific fluctuations, Heart rate

Substance dependence develops within a context of environmental, physiological, and psychological systems. Many studies suggest that alcohol and drug dependence are influenced by genetic factors (Bohman, Cloninger, Sigvardsson, & von Knorring, 1987; Cadoret, Troughton, O’Gorman, & Heywood, 1986; Cloninger, Bohman, & Sigvardsson, 1981; Tsuang et al., 1996; Yates, Cadoret, Troughton, & Stewart, 1996). However, substance dependence cannot develop without a facilitative environmental context which, at a minimum, includes access to the substance. Although many environmental factors are likely to contribute to an individual’s continued substance use (e.g., modeling the behavior of family and friends), a growing literature suggests that physiological systems may influence the development of substance dependence. In this report, we examined the possibility that substance dependence is associated with deficits in an inhibitory control system.

The association between psychopathy, antisocial personality disorder, and substance dependence, which are often considered to be different forms of disinhibited psychopathology, may stem from a common deficit in inhibitory control (e.g., Gorenstein & Newman, 1980). In 1988, Fowles tied the concept of disinhibited psychopathology to Gray’s two-factor motivational theory as part of a

broader motivational theory of psychopathology. Briefly, Fowles (1980, 1988) and Gray (1975) posited that approach or reward-seeking behavior is governed by the behavioral activation system (BAS), whereas inhibition of approach behavior in the face of cues signaling punishment is governed by the behavioral inhibition system (BIS). Fowles (1988) proposed that weakness in the BIS may account for physiological response deficits associated with disinhibited psychopathology.

Consistent with the weak BIS hypothesis, Finn, Kessler, and Hussong (1994) found poor electrodermal conditioning to stimuli predicting electric shock and small electrodermal responses among nonalcoholic adult males with a high-density family history of alcoholism (high-risk) as compared with nonalcoholic adult males with a low-density family history (low-risk). Additionally, the literature showing response dampening effects of alcohol (e.g., Finn, Earleywine, & Pihl, 1992; Finn, Zeitouni, & Pihl, 1990; Lyvers & Maltzman, 1991) suggests that a dysfunctional inhibitory control system may be involved in the development of substance use disorders.

One type of inhibitory control, labeled “negative preception” by Lykken (1959), is associated with individual differences in a presumed capacity to lessen the impact of predictable, aversive stimuli. Evidence of negative preception has been reported for both human and animal subjects using as aversive stimuli electric shock and white noise blasts (see Lykken & Tellegen, 1974, for a discussion of the preception literature). Although the neurobehavioral mechanism behind preception is unknown, it appears to involve an assessment of stimulus properties (aversive vs. nocive; predictable

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vs. unpredictable) followed by a manipulation of an inhibitory control system. As such, paradigms designed to induce preception may be useful for examining psychopathology (e.g., substance dependence) thought to be related to inhibitory control deficits.

Our paradigm, called "Cooltest," was modeled after Lykken's (1959) procedure and was designed to measure the extent to which a subject could "stay cool" by demonstrating reduced reactivity to an aversive noise blast when the blast was made predictable in time. Good modulation was marked by large decreases in the skin conductance response (SCR) to predictable relative to unpredictable stimuli, whereas poor modulation was marked by increased SCR to predictable relative to unpredictable stimuli. Moderate modulation was marked by moderate decreases in SCR to predictable relative to unpredictable stimuli. We elected to use a community-based sample of adolescents in this investigation because, even though substance dependence may develop early in adolescence, the young age and limited resources of teens diminishes the likelihood that psychophysiological measurements reflect simply the consequences of prolonged and extensive exposure to psychoactive substances.

The principal hypothesis in this investigation was that poor SCR modulators in the Cooltest would show more evidence of substance dependence than good modulators. In addition, we had two supporting hypotheses regarding other autonomic variables. First, based on the findings of Lykken, Macindoe, and Tellegen (1972), who found that cardiac activity was associated with negative preception, we expected good SCR modulators to be more likely than poor modulators to show elevated heart rate (HR) preceding the aversive stimulus and reduced HR following the stimulus when it was predictable. Such a pattern of HR activity was interpreted by Lykken et al. (1972) as evidence of a successfully employed inhibitory control mechanism in those who show evidence of negative preception. Second, we expected poor SCR modulation, perhaps indexing a weak BIS, to be associated with electrodermal hypoactivity preceding the aversive stimulus. Thus, we expected poor SCR modulators to give fewer skin conductance nonspecific fluctuations (NSFs) while anticipating the aversive stimuli than good modulators. For each of these hypotheses, moderate modulators were expected to fall between the other two groups.

## Method

### Participants

Participants were drawn from the Minnesota Twin Family Study (MTFS), a community-based longitudinal investigation of the etiology of substance use and related disorders among twins and their families. Families in the MTFS were ascertained through a search of all Minnesota state birth records. The subjects for this investigation were 175 16- to 18-year-old (mean = 17.65 years;  $SD = 0.40$ ) boys who completed both the clinical and psychophysiological assessments. Participants who were 18 years old gave written informed consent to participate in the study. Those under 18 gave written informed assent and one of their parents gave written informed consent for the son's participation.

### Clinical Assessment

Each adolescent and his mother were independently administered structured clinical interviews by trained interviewers (each with at least a BA in psychology) as part of the MTFS clinical assessment. Three DSM-III-R (American Psychiatric Association, 1987) disorders were included as dependent variables in this investigation:

alcohol, nicotine, and cannabis dependence. Under the DSM-III-R diagnostic system, symptoms of substance abuse are subsumed in a substance dependence diagnosis, whereas certain dependence symptoms (e.g., tolerance) are not included in an abuse diagnosis. For this reason, we elected to use only the more inclusive substance dependence category.

Lifetime substance dependence symptoms in the boys were assessed with an expanded version of the Substance Abuse Module (SAM; Robins, Babor, & Cottler, 1987) developed as a supplement to the World Health Organization's Composite International Diagnostic Interview (Robins et al., 1988). Mothers gave information on their sons' lifetime symptoms of substance dependence via an MTFS-modified version of the Diagnostic Interview for Children and Adolescents-Revised, parent version (DICA-R-P; Herjanic & Reich, 1982; Reich & Welner, 1988).

Symptoms of each disorder were assigned by consensus of two or more clinical psychology graduate students using all available clinical information. A computer algorithm combined the symptom information offered by each informant and generated the best estimate symptom counts and best estimate DSM-III-R diagnoses used in our analyses. The best estimate symptom count is simply an unweighted count of all positive symptoms (regardless of whether a symptom was reported by the mother or the child). The best estimate diagnoses were assigned based on information contained in the best estimate symptom count and duration criteria. We considered participants to be substance dependent if they met either definite (three or more symptoms present for a duration of at least one month) or probable (two symptoms and the duration criterion) DSM-III-R dependence criteria. A participant was considered non-dependent if he had either zero or one symptom. The reliability of project substance dependence diagnoses generated following this procedure was high (all kappas  $> .91$ ).

### Psychophysiological Assessment

The skin conductance (SC) and HR data reported here were collected as part of a 3.5-hr battery of psychophysiological tests administered to all MTFS participants. The battery began with an eye tracking assessment and ended with the recording of central nervous system measures. The autonomic assessment described here thus composed the middle of the battery.

Participants were instructed to wash their hands with soap and water and then the skin sites were wiped with isopropyl alcohol before application of the SC sensors. A 0.79 cm<sup>2</sup> area of skin was isolated on the fingerprint regions of the index and ring fingers of both hands using electrode collars (Lykken & Venables, 1971). Ag-AgCl electrodes containing a 0.5-M NaCl electrolyte mixed in Unibase cream (Lykken & Venables, 1971) were affixed to each of the four fingers. A constant 0.5-V potential difference was generated across the two fingers on each hand by an SC signal conditioner of the type described by Lykken and Venables (1971). The output from this coupler was recorded through DC amps on a Grass Model 12A Neurodata acquisition system. These data were filtered with a 3 Hz low-pass filter and amplified 5,000 $\times$ .

The electrocardiograph (ECG) was recorded with a modified Lead II electrode configuration using Ag-AgCl electrodes filled with Hewlett Packard Redux paste placed on the right temple and left shin. A ground electrode was placed on the right shin. Skin impedance for the ECG and ground sites was kept below 10 k $\Omega$ . The ECG was recorded through a Grass AC amplifier. Half-amplitude high and low frequency settings were at 1 and 30 Hz, respectively. Both SC and ECG signals were digitized online at a rate of 128 Hz.

### The Cooltest

The Cooltest paradigm consisted of five 100-s trials. On each of the trials, participants were presented with a computer graphic of a clock face with a sweep-second hand and were instructed that they would hear a loud, unpleasant sound on each trial and they should "try to stay cool and not react to the loud noise." Participants also were told that a red hash mark on the clock face would indicate stimulus onset on some trials (making the stimulus predictable in time) but not on others.

A 2-s blast of 90-dB white noise produced by a Coulbourn white noise generator (S81-02) served as the aversive stimulus. A constant background of 55-dB white noise (used to mask extraneous lab and hallway noises) also was produced by this equipment. The stimulus was presented binaurally over head phones with immediate onset and offset. The clock was present on the screen for the entirety of each trial; the sweep-second hand began its rotation on the 10th second of each trial. A single noise blast occurred at pseudo-random times between the 25th and 75th second of each trial. Stimulus presentation times were the same for all participants and the stimulus was predictable only in Trials 1, 2, and 5. Auditory and visual stimulus presentation was under the control of an IBM-compatible PC running software written for the MTFs.

### Scoring of Psychophysiological Measures

**SCR.** SCR amplitude was scored on the right hand for each trial. Response amplitude was defined as the difference (in  $\mu$ siemens) between the SCL preceding the response and the SC at the peak of the response curve. Mean SCR amplitude was computed separately for both trial conditions.

**NSF.** NSFs were scored on the right hand for the 25-second anticipation period in each trial. An NSF was defined as a phasic change in SC of at least 0.05  $\mu$ siemens. Only NSFs with both an onset and a peak during the anticipation period were scored.

**HR.** HR was calculated by counting the number of beats in the 40 s surrounding the stimulus (the 25-s anticipation period and the 15-s response period) and then converting to HR in beats per minute (bpm). The overall mean HR in bpm for both experimental conditions was calculated separately for the anticipation and response periods in order to get assessments of pre- and post-blast HR.

All scoring of psychophysiological data was done either by an automated computer program or by someone blind to the diagnostic status of the subjects.

### Electrodermal Response Modulation

An electrodermal response modulation (ERM) score devised by one of the authors (DTL) indexed response modulation during the Cooltest. This score reflected the percent decrease (or increase) in SCR amplitude when the aversive stimulus was made predictable in time. It was derived by the formula:

$$\text{ERM} = 100 \times (\text{SCR}_U - \text{SCR}_P) / \text{SCR}_U,$$

where  $\text{SCR}_U$  and  $\text{SCR}_P$  refer to the mean SCR on unpredictable and predictable trials, respectively. Close examination of the ERM formula reveals an inherent range correction and thus raw SCR values were entered into the formula.

### Range Correction

The response data were range-corrected by expressing SCR as a percentage of the individual's largest elicited response observed

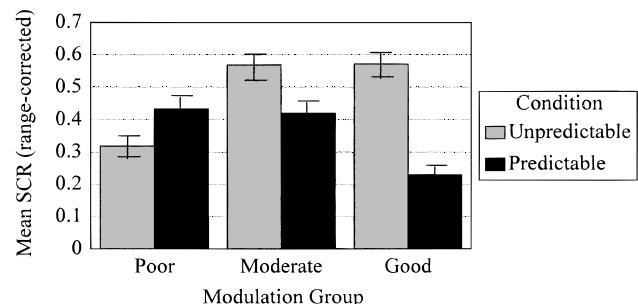
during either the Cooltest procedure or another task included for the purpose of optimizing the likelihood of eliciting an approximation of the maximum SCR (see Lykken, 1972; Lykken, Rose, Luther, & Maley, 1966; Lykken & Venables, 1971).

Prior to exposure to the Cooltest task, subjects were exposed to a single unexpected stimulus consisting of a 0.75-s blast of 90-dB white noise. Although this brief task was designed to elicit a maximum electrodermal response, the largest elicited SCR values for each subject were identified wherever they occurred during the Cooltest and single blast procedures. Range-corrected electrodermal data were used in all instances except when calculating ERM scores as noted above.

### Modulation Groups

Because the MTFs is a family study that involves many assessment procedures, it is important that each subject completes each task in the same order so that data from different family members are comparable. In addition, because the MTFs is an epidemiological study of all twins born in Minnesota who fall in the designated age bracket, participants were not excluded in advance due to qualities that might have disqualified them from a specific assessment. Consequently, decisions were made after data were collected to exclude some subjects (without regard to diagnostic status) from certain data analyses if it was believed that the assessment was compromised in some manner that could have affected the dependent measures. For this report, a total of 25 participants were excluded for various reasons, including reported hearing loss, damaged fingertips, medications, and missing data during part of the electrodermal recording session. The ERM score distribution for the remaining 150 subjects was used to select the modulation groups for this study. The 25 boys at the top of the ERM score distribution were labeled good modulators (mean = 60.25;  $SD = 10.39$ ; scores ranged from 50.4 to 84.2); the 25 boys at the bottom of the distribution were labeled poor modulators (mean = -43.17;  $SD = 31.13$ ; scores ranged from -8.2 to -121.7); and 25 boys selected at random from the 50 middle scores in the distribution were labeled moderate modulators (mean = 25.50;  $SD = 6.82$ ; scores ranged from 16.6 to 37.6).

Figure 1 contains the mean for the unpredictable (U) and the predictable (P) trials for each modulation group using the range-corrected SCR data. Both moderate and good modulators were characterized by significantly higher SCRs in the U than in the P condition,  $t(24) = -10.01$  and  $t(24) = -12.18$ , respectively. Poor modulators, however, were characterized by significantly higher SCRs in the P than in the U condition,  $t(24) = 8.30$ . Each paired-samples  $t$  test was significant at the .001 level (one-tailed).



**Figure 1.** Mean skin conductance response (SCR) in each condition for each modulation group. The vertical bar represents  $\pm 1$  standard error.

**Analyses**

The symptom count data for the substance dependence disorders were submitted to a one-way multivariate analysis of variance (MANOVA). Where the MANOVA indicated a significant test-wise effect, ANOVAs and follow-up contrasts (Fisher's Least Significant Difference tests) were run. The mean NSF counts and the mean HR data were each submitted to a two-way (modulation group by trial type) repeated measures ANOVA, with repeated measures on the trial type factor. The  $\chi^2$  statistic was used to test for differences in the number of diagnoses between modulation groups. The alpha for all tests was set at .05.

**Results**

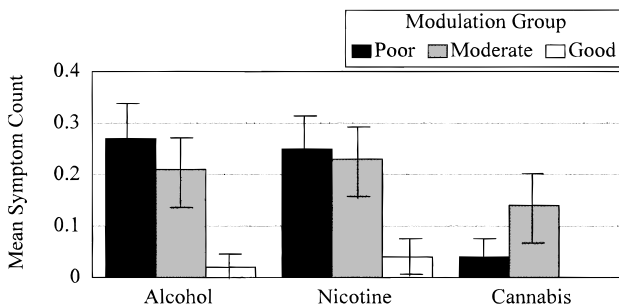
**Substance Dependence**

Due to the positive skew of the distributions, the symptom count data were transformed ( $\log_{10} [x + 1]$ ) prior to use in parametric tests. As expected, the modulation group main effect was statistically significant, not only in the overall MANOVA ( $\lambda = .766, p < .01$ ), but in each of the follow-up univariate ANOVAs of alcohol dependence,  $F(2,72) = 6.48, p < .01$ ; nicotine dependence,  $F(2,72) = 3.60, p < .05$ ; and cannabis dependence,  $F(2,72) = 3.75, p < .05$ , symptoms. Post hoc analysis of alcohol dependence symptoms revealed that good modulators had significantly fewer symptoms than moderate or poor modulators, who did not differ significantly from each other. The same pattern was found for symptoms nicotine dependence. Finally, good modulators had significantly fewer symptoms of cannabis dependence than only the moderate modulators. Figure 2 presents the log-transformed mean symptom counts for each disorder for each modulation group.

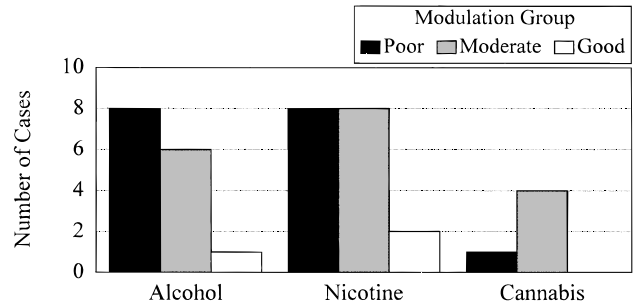
The number of DSM-III-R lifetime disorder cases for each modulation group is shown in Figure 3. Only the number of alcohol dependence disorder diagnoses differed significantly among the three modulation groups,  $\chi^2(2, N = 75) = 6.50, p < .04$ . Fisher's Exact Test revealed that good modulators had significantly fewer alcohol dependence diagnoses than the moderate and the poor modulators ( $p < .05$ , one-tailed). There was a trend toward a significant difference in the number of nicotine ( $p < .07$ ) and cannabis ( $p < .06$ ) dependence cases among the modulation groups as well.

**HR**

Mean HR was calculated for the anticipation and response periods for each of the five Cooltest trials. Figure 4 shows the changes in mean HR from before to after the noise blast after averaging across



**Figure 2.** Log-transformed ( $\log_{10} [x + 1]$ ) mean DSM-III-R disorder symptom count for each modulation group. The vertical bar represents  $\pm 1$  standard error. Alcohol, Nicotine, and Cannabis refer to the substances of dependence.



**Figure 3.** Number of DSM-III-R disorder cases in each modulation group. Alcohol, Nicotine, and Cannabis refer to the substances of dependence.

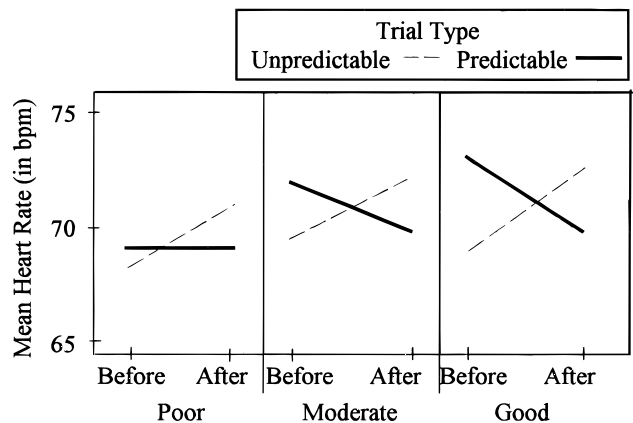
trials in each condition. The expected interaction of modulation group by trial type by period (pre- and post-stimulus) was significant,  $F(2,72) = 3.15, p < .05$ . As predicted, follow-up contrasts showed that poor modulators did not differ significantly in their pre- to post-stimulus HR in the predictable condition, whereas the moderate and good modulators both showed the negative pre- to post-stimulus relationship expected when preception is invoked. The three modulation groups did not differ significantly in their average HR before or after the stimulus in either condition.

**NSFs**

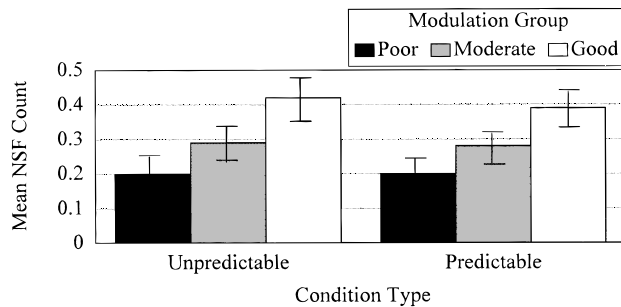
The number of NSFs produced during the anticipation period in each trial were tabulated and averaged across trials in each condition. Prior to parametric tests, the NSF data were transformed ( $\log_{10} [x + 1]$ ) due to the positive skew of the distribution. Figure 5 shows the transformed NSF means for each modulation group in each condition. The interaction between modulation group and trial type was not significant. The main effect for modulation group was significant,  $F(2,72) = 5.37, p < .01$ . Follow-up contrasts revealed that, as expected, poor modulators produced significantly fewer NSFs in both conditions than did good modulators; no other differences were significant.

**Discussion**

To our knowledge, this constitutes the first study of psychophysiological factors related to substance dependence using a community-



**Figure 4.** Mean heart rate (HR) before and after the blast in each condition for each modulation group.



**Figure 5.** Log-transformed ( $\log_{10} [x + 1]$ ) mean nonspecific fluctuation (NSF) count for each modulation group in each condition. The vertical bar represents  $\pm 1$  standard error.

ascertained sample of male high school students. Previous work suggested that substance dependence is related to deficits in an inhibitory control system (e.g., BIS), leading us to adopt a paradigm modeled after Lykken's (1959), in which the predictability of an aversive stimulus was manipulated in an effort to induce modulation of SCRs. Given the notion that substance dependence may be related to deficits in inhibitory control, we expected that poor SCR modulation to predictable aversive stimuli would be related to increased risk for substance dependence, whereas good SCR modulation would be associated with low risk for substance dependence. Specifically, we expected to find a step-wise increase in the number of DSM-III-R alcohol, nicotine, and cannabis dependence symptoms and diagnoses among good, moderate, and poor SCR modulators. Additionally, we expected (1) poor SCR modulators to have less change in HR from pre- to post-stimulus in predictable trials than moderate and good modulators, and (2) poor SCR modulators to have fewer NSFs than moderate and good modulators. The data tend to support a model with SCR modulation reflecting a protective factor in the development of substance dependence.

Our principal hypothesis regarding SCR modulation and substance dependence was supported. As expected, differences in SCR modulation ability were significantly related to differences in symptom counts of alcohol, nicotine, and cannabis dependence and the diagnosis of alcohol dependence. Boys who took advantage of the predictability of the aversive stimulus and showed the largest inhibition in SCR were least likely to be substance dependent. Conversely, boys who did not take advantage of the predictability of the aversive stimulus and subsequently failed to inhibit their SCRs were likely to be dependent on alcohol and nicotine. Consistent with a protective factor model, good modulators had significantly fewer symptoms of substance dependence than both moderate and poor modulators, who were similar in their levels of substance dependence. Evidence for psychophysiological protective factors also have been found for other forms of disinhibited psychopathology (Brennan et al., 1997).

Why would individual differences in electrodermal responsivity to aversive stimuli be related to the development of substance dependence? One possibility is that some substance abusers use substances to reduce life stress in part because of a deficit in an inhibitory control system that prevents them from blunting the effects of at least one type of stressful stimuli (viz., the predictable type). Support for this line of thinking comes from studies that show the response dampening effects of alcohol (e.g., Finn et al., 1990; 1992; Lyvers & Maltzman, 1991).

Our two supporting hypotheses regarding HR and anticipatory NSF count were largely supported by the data. First, the expected HR interaction for trial type by period (pre- vs. post-stimulus) was significant; this replicates the results of Lykken et al. (1972). Our results are consistent with the notion that cardiac acceleration indicates an active coping response (Obrist, 1976). As Figure 4 shows, the modulation groups did not differ in their HR pattern during the U trials as the stimulus came without warning and the subjects could not prepare for it. In the P condition, however, the good and moderate modulators began preparing for the stimulus before the stimulus occurred (as evidenced by their cardiac acceleration), which served to lessen its impact (as evidenced by their lowered SCR in the P trials). The poor modulators, on the other hand, failed to prepare for the predictable stimulus (as evidenced by only slight cardiac acceleration), which may have served to increase the impact of the stimulus (as evidenced by their increased SCR in the P trials).

Second, the anticipatory NSF data revealed a relationship between anticipatory electrodermal under-responsivity (evidenced among the poor modulators) and substance dependence. Poor modulators evidenced significantly fewer NSFs than good modulators in anticipation of the blast in both conditions. Although the moderate modulators did not differ significantly from either of the two extreme groups, the data were orderly and conformed to the pattern we expected. In sum, boys who (on average) evidenced little anticipatory electrodermal activity evidenced symptoms of substance dependence, whereas boys with few substance dependence symptoms evidenced the greatest level of anticipatory electrodermal activity (on average).

A few limitations of this investigation deserve mention. First, we elected to test our hypothesis using a male sample because most of the previous research in this area has been conducted with males. Therefore, we cannot say with any certainty whether these findings are similar for females. Although there is no reason to suspect that females would not evidence individual differences in electrodermal responsivity to aversive stimuli, we cannot be certain that modulation would be related to substance use disorders specifically. Replication with a female sample would begin to address this question.

Second, our noise blast (90 dB) was certainly aversive; however, it is possible that with an even louder blast (e.g., 110 dB) or electric shock, that the results in the poor modulation group may be qualitatively different. Although we suspect that the poor modulators have an inhibitory control deficit that hinders their ability to reduce the impact of predictable aversive stimuli, possibly these boys simply did not fear the 90-dB noise blast enough to bother modulating their responses to the blast.

Finally, although we interpret our results as having implications for assessing risk for substance dependence, we recognize the limitation of our sample that contains individuals who are already substance dependent. To address this issue, a study would have to measure psychophysiological characteristics of people not yet exposed to alcohol and drugs (e.g., 10- or 11-year-olds) and then assess them for substance dependence at a later age (i.e., once they have entered the risk period for developing the disorder) to evaluate whether individual differences on a psychophysiological index predicts substance dependence.

Substance dependence is a complex phenomenon that cannot be fully understood through examination of single domains (e.g., environment). Our investigation lends further support to the growing literature that suggests that examination of physiological systems will likely provide important pieces in the puzzle of substance dependence.

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