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LIFETIME HISTORY OF CIGARETTE SMOKING ASSOCIATED WITH AGGRESSION AND IMPULSIVITY IN BOTH HEALTHY AND PERSONALITY DISORDERED VOLUNTEERS

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Abstract

Cigarette smoking has been associated with several personality and behavioral traits including impulsivity and aggression, primarily in adolescents and/or pregnant women. In this study, the authors tested the hypothesis that history of cigarette smoking is also associated with dimensional measures of aggression and impulsivity in adult subjects. Subjects were 179 personality disordered (PD), and healthy volunteer control (HV), subjects in whom history of cigarette smoking and measures of both aggression and impulsivity were collected. Scores on measures of both aggression and impulsivity were elevated as a function of personality disorder status and history of cigarette smoking status; no interaction between these two factors were noted on these measures. Including socio-economic status and global psychosocial functioning in the model eliminated the difference associated with diagnostic grouping and impulsivity but not the difference in aggression associated with lifetime history of smoking. These data suggest that lifetime history of cigarette smoking is associated with elevations in life history of aggression regardless of the presence of psychiatric disorder in nontreatment seeking individuals.

Cigarette smoking has been associated with several personality and behavioral traits including impulsivity, sensation seeking, and aggression (e.g., Acton, 2003). Most studies to date have been conducted in adolescents and in the offspring of pregnant women. In adolescents, several recent studies suggest a strong association between smoking and measures of impulsivity such as the Barratt Impulsivity Scale (Papadodima et al., 2009) and delay-discounting (Melanko, Leraas, Collins, Fields, & Reynolds, 2009). Impulsivity has also been shown to be associated with cue-induced craving for cigarettes in adults (Doran, Cook, McChargue, & Spring, 2009). Sensation seeking, a subtype of impulsivity, has also been shown to be associated with cigarette smoking in adolescents (Urban, 2010). Similarly, some measures of aggression also appear to be associated with cigarette smoking (Ernst et al., 2006; Fite, Colder, Lochman, & Wells, 2008; Shaeffer et al., 2007) as also may be suicidality in adolescents (Raila et al., 2009) and bipolar patients (Ostacher et al., 2009). A history of childhood trauma, a variable related to aggression (Coccaro, Noblett, McClosky, et al., 2009) has also been shown to be associated with cigarette smoking in adolescents (Papadodima et al., 2009).

In addition, a growing body of evidence has linked prenatal maternal smoking with an increased rate of disruptive behaviors in offspring (Brook, Brook, & Whiteman, 2000;

Wakschlag et al., 1997; Wasserman, Liu, Pine, & Graziano, 2001). The influence of numerous confounding factors such as parental, social, and demographic is under debate, however, and the hypotheses that the association between maternal prenatal smoking and antisocial behavior is modulated through the influence of prenatal smoking on externalizing behaviors in offspring has not been confirmed by some recent studies (Button, Thapar, & McGuffin, 2005). More importantly, a recent genetically informed study suggests that unidentified environmental factors, rather than teratogenic effects, underlie the relationship between prenatal smoking and externalizing behavior in offspring of maternal smokers (D'Onofrio et al., 2008).

One possibility for the relationship between cigarette smoking and externalizing behaviors could be a relationship with central 5-HT system function. An inverse relationship between impulsive aggression and 5-HT system function has been demonstrated in a variety of clinical, and preclinical, studies for more than three decades (Coccaro, Lee, & Kavoussi, 2010). Life history of smoking has been shown to be associated with inverse relationships with several measures of 5-HT function in human subjects (e.g., Anthenelli & Maxwell, 2000; Malone et al., 2003; Patkar, Gopalakrishnan, Berrettini, Weinstein, & Vergare, 2003) and chronic administration of nicotine has been shown to reduce 5-HT in several brain areas in rodents exposed to nicotine levels similar to that of human smokers (Xu, Seidler, Ali, Slikker, & Slotkin, 2001).

Accordingly, we sought to determine if a life history of smoking (cigarettes) would be associated with psychometrically reliable and valid measures of externalizing behaviors in adults, a group that has received little study to date, in this regard. In this study we examined levels of aggression and impulsivity in physically healthy research volunteers recruited without selection bias for aggressive or impulsive behavior. We hypothesized that history of smoking would be associated with aggression and impulsivity in our adult subjects.

METHODS

SUBJECTS

This paper reports data from 179 physically-healthy research volunteers in whom both history of cigarette smoking, and measures of both aggression and impulsivity, were collected. All subjects were systematically evaluated as part of a larger program designed to study the biological correlates of personality traits in human subjects. Study subjects (128 male, 51 female; 78 as Healthy Volunteer Control: HV, 101 as Personality Disorder: PD) were recruited by newspaper and public service announcements seeking subjects to take part in medically related studies; this recruitment effort did not emphasize any specific interest in the study of psychopathology. Written informed consent, using an IRB-approved consent document, was obtained from all subjects after all procedures were fully explained. Medical health of all subjects was documented by medical history, physical examination, and a variety of clinical laboratory studies including a urine screen for illicit drugs.

DIAGNOSTIC ASSESSMENT

Axis I and Axis II Personality Disorder (PD) diagnoses were made according to DSM-IV criteria (American Psychiatric Association, 1994). Diagnosis of Alcoholism and Intermittent Explosive Disorder were made by modified Research Diagnostic Criteria as described in other reports (Coccaro, Kavoussi, Sheline, Lish, & Csernansky, 1996; Coccaro, Schmidt, Samuels, & Nestadt, 2004, respectively). Diagnoses were made using information from: (1) semi-structured interviews conducted by trained masters, or doctoral, level clinicians using the Schedule for Affective Disorders and Schizophrenia (Spitzer & Endicott, 1978) modified to include modules for the diagnosis of DSM Axis I disorders not covered by the original

SADS, or the Structured Clinical Interview for DSM Diagnoses (SCID-I; First, Spitzer, Williams, & Gibbon, 1995) for Axis I disorders, and the Structured Interview for the Diagnosis of DSM Personality Disorder (Pfohl, Blum, Zimmerman, & Stangl, 1989; Pfohl, Blum, Zimmerman, & University of Iowa. Dept. of, P., 1997) for Axis II disorders; (2) clinical interview by a research psychiatrist; and (3) review of all other available clinical data. Final diagnoses were assigned by team best-estimate consensus procedures (Klein, Ouimette, Kelly, Ferro, & Riso, 1994; Leckman, Sholomskas, Thompson, Belanger, & Weissman, 1982) involving at least two research psychiatrists and three clinical psychologists as previously described (Bunce, Noblett, McCloskey, & Coccaro, 2005). This methodology has previously been shown to enhance the accuracy of diagnosis over direct interview alone (Kosten & Rounsaville, 1992). Subjects with a life history of Bipolar disorder or Schizophrenia (or other psychotic disorder) were excluded from this study.

Fifty-two of the 101 PD subjects met DSM-IV criteria for a specific personality disorder (Table 1). The remaining forty-nine subjects were diagnosed as Personality Disorder—Not Otherwise Specified (PD-NOS). These subjects met DSM-IV general criteria for personality disorder, had pathological personality traits from a variety of personality disorder categories and had clear evidence of impaired psycho-social functioning (mean GAF score = 64.9 ± 7.3). In addition, most PD subjects (71 of 101: 71%) had a life history of at least one Axis I disorder (Table 1).

ASSESSMENT OF HISTORY OF CIGARETTE SMOKING, AGGRESSION, IMPULSIVITY, AND OTHER BEHAVIORAL VARIABLES

History of cigarette smoking was collected during the diagnostic interview assessment and was assessed using two questions taken from the SADS. Lifetime history of smoking (LHS) was coded positive if subjects reported smoking cigarettes on at least three separate occasions (53 of 179: 29.6%); current history of smoking was assessed by asking subjects if they were currently smoking cigarettes in the past three weeks; all but four subjects with LHS reported that they were currently smoking cigarettes (49 of 53: 92.5%) at time of study. Specific data regarding the number of cigarette packs smoked per day (or per year) were not collected systematically. Aggression and impulsivity were assessed using the Aggression scales of the Life History of Aggression (Coccaro, Beman, & Kavoussi, 1997) and the Total Score from the Barratt Impulsiveness Scale (BIS-11; Patton, Stanford, & Barratt, 1995). Secondary variables included general (i.e., Neuroticism, Psychoticism, Extraversion) personality variables from the Eysenck Personality Questionnaire (Eysenck & Eysenck, 1975).

STATISTICAL ANALYSIS

Statistical analysis included Chi-Square, Fishers Exact Test, T-Test, and Multivariate ANOVA and ANCOVA, as appropriate, with a two-tailed alpha of .05. The primary analysis of interest was MANOVA using LHA and BIS-11 scores as dependent variables and diagnostic group (Healthy Control *vs.* Personality Disorder) and lifetime history of smoking (LHS+ *vs.* LHS-) status. Secondary analyses included: (1) MANCOVA with SES and GAF as covariates and (2) MANOVA/ MANCOVA with the general EPQ personality scores as dependent variables. Since nearly all of the LHS subjects were also currently smoking (49 of 53: 92.5%) only results of analyses using the LHS variable are reported below.

RESULTS

HV/PD GROUP AND LHS GROUP DIFFERENCES IN DEMOGRAPHIC AND PSYCHOSOCIAL FUNCTION VARIABLES (TABLE 2)

While HV and PD volunteers were similar in the frequency of lifetime smoking (32.7% vs. 25.6%, Fisher's Exact Test $p = .327$) and in race (61.5% vs. 53.5% White vs. Non-White), HV subjects differed from PD subjects with regard to age, gender, and in socioeconomic status (linear by linear χ^2) where HV subjects were more likely to come from higher (i.e., SES Class II: 14.1% vs. 7.9%) and less likely to come from lower (i.e., SES Class V: 20.5% vs. 31.7%) SES classes compared with personality disordered subjects. HV subjects also had, as expected, substantially higher GAF scores compared with those of PD subjects. Group differences as a function of LHS were similar with regard to SES and GAF score, but not for age or gender (or race) neither of which differed as a function of LHS. Compared to LHS subjects, Non-LHS subjects were more likely to come from higher (i.e., SES Class II: 13.5% vs. 3.8%), and less likely to come from lower (i.e., SES Class IV: 26.2% vs. 47.2%; linear by linear $\chi^2 = 6.45$, $df = 1$, $p = .011$), SES classes. Non-LHS subjects also had modestly higher GAF scores compared with LHS subjects (73.4 ± 12.4 vs. 68.9 ± 13.3 , $t_{92.1} = 2.10$, $p < .05$).

HV/PD GROUP AND LHS GROUP DIFFERENCES IN AGGRESSION AND IMPULSIVITY

MANOVA using LHA and BIS-11 scores as dependent variables, and HV/ PD and LHS as independent variables, revealed an effect of both HV/PD group, $F(2,172) = 11.32$, $p < .001$, and LHS on LHA and BIS-11 scores, $F(2,172) = 5.11$, $p = .007$, without interaction between HV/PD group and LHS on LHA and BIS-11 scores, $F(2,172) = 0.12$, $p = .891$. Both LHA, $F(1,174) = 6.34$, $p = .013$, and BIS-11, $F(1,174) = 5.28$, $p = .023$, scores were significantly greater as a function of LHS. Subsequent MANOVA analysis performed to assess the effects of potential demographic/functional variables (i.e., age, gender, SES, GAF) on HV/ PD group, LHS, and LHA/BIS-11 scores revealed a significant effect of SES on LHS (only) and a significant effect of GAF on both HV/PD group and on LHA and BIS-11 scores. MANCOVA analysis, using SES and GAF as covariates, revealed an effect of LHS on LHA and BIS-11 scores, $F(2,172) = 3.29$, $p < .05$, without effect of HV/PD group on LHA and BIS-11 scores, $F(2,172) = 0.08$, $p = .928$, and without interaction between HV/PD group or LHS on LHA and BIS-11 scores, $F(2,172) = 0.10$, $p = .904$. While LHA, $F(1,172) = 4.55$, $p < .05$, scores continued to be significantly greater as a function of LHS (Figure 1a), the effect of LHS on BIS-11 scores was no longer statistically significant, $F(1,172) = 2.71$, $p > .10$; Figure 1b, after accounting for differences in SES and GAF. Addition of lifetime history of major depression as a third covariate did not change these results, $F(2,171) = 3.11$, $p < .05$ for LHS overall on LHA and BIS-11 scores; $F(1,171) = 4.22$, $p < .05$ for LHA scores and $F(1,171) = 2.66$, $p = .11$ for BIS-11 scores.

HV/PD AND LHS GROUP DIFFERENCES IN GENERAL PERSONALITY VARIABLES

MANOVA, using the three general factor personality scores of the EPQ (i.e., neuroticism, extraversion, psychoticism) as dependent variables, and HV/PD and LHS groups as independent variables, was next performed to determine if the effect of LHS extended to more general personality traits. This analysis revealed a significant effect of HV/PD group on EPQ scores, $F(3,170) = 12.66$, $p < .001$, but no effect of LHS on EPQ scores, $F(3,170) = 1.18$, $p = .319$, and no interaction between HV/PD or LHS on EPQ scores, $F(3,170) = 1.04$, $p = .377$. MANCOVA, using SES and GAF as covariates, eliminated the effect of HV/PD on EPQ scores.

LHS GROUP DIFFERENCES AND SPECIFIC DIAGNOSES IN HV/PD GROUPS

LHS has previously been linked to Conduct Disorder, Antisocial PD, and Substance Use Disorders. In this sample, a significant association between LHS and lifetime history of Conduct Disorder, only, was observed (Fisher Exact Test: $p = .007$ in HV/PD groups; $p = .017$ in PD group, alone). This association remained statistically significant even after LHA scores were added as a covariate, Conduct Disorder: Wald ($df = 1$) = 4.63, $p = .031$; LHA: Wald ($df = 1$) = 4.85, $p = .028$. In contrast, there was no significant association between LHS and Antisocial PD, or Substance Use Disorders in the HC/PD group or in the PD group alone.

DISCUSSION

The results of this study indicate that a lifetime history of smoking (LHS) is associated with greater lifetime history of aggression, as ascertained by LHA Aggression scores, in both healthy volunteer control and personality disordered subjects. This finding is not accounted for by differences in other variables (e.g., GAF and SES scores) because further analyses revealed a significant positive relationship between LHS and LHA Aggression even after including these variables in the analysis. While LHS subjects also had higher scores on the BIS-11 measure, differences in these scores did not continue to be statistically significant after all other relevant variables were accounted for.

It is notable that LHS and Non-LHS subjects did not differ as a function of EPQ scores for neuroticism, extraversion, and psychoticism. This suggests that the relationship between LHS and LHA Aggression, in these subjects, is specific to this trait and not to more generalized personality trait and/or factors. Subjects did differ in their EPQ scores as a function of diagnostic status, though this was expected. Adding GAF and SES scores to the analyses, however, eliminated these differences between PD and HV subjects.

It is also notable that LHS was significantly related to the history of Conduct Disorder. This relationship continued to be present even after accounting for differences in LHA Aggression scores. An increase in the development of Conduct Disorder has been reported in the offspring of mothers who smoked during pregnancy (e.g., Wakschlag et al., 1997). As such, this result is consistent with previous data in studies of prenatal smoking. The observation that LHS is not significantly associated with an increase in the presence of Antisocial Personality Disorder (AsPD) or Substance Use Disorders (SUD) is surprising since many, though not all, individuals with Conduct Disorders meet criteria for AsPD by young adulthood. However, the number of subjects with AsPD in the sample ($n = 13$) was relatively small and may account for a lack of findings in this study. The same may be true for the absence of a significant association between LHS and SUD. For one thing, the numbers of subjects with SUD ($n = 13$ to 16) was limited to subjects with only a past history of SUD and this would have limited the numbers of overall SUD subjects as well.

Some issues limit the generalizability of these findings. These issues relate to nature of the sample, reliability of assessments, and limited information regarding the nature of the history of cigarette smoking. First, while the participants were generally recruited from the community, these study participants do not comprise a randomly selected community based sample. As such, there is some self-selection in this regard that makes this group, necessarily, different than the general population. Second, assessments regarding the history of cigarette smoking were made in the context of our diagnostic assessment procedure and were made by one rater only. Accordingly, there is no reliability data regarding our assessment of LHS. A recent study in the United States, however, reports very good reliability data for "history of smoking" compared with simultaneous measurements of urinary cotinine levels in high school smokers (Park & Kim, 2009). Third, the low threshold

for LHS was smoking on at least three separate occasions. More detailed questions regarding the full extent of history of smoking was not obtained. It is of note, however, that nearly all (92.5%) of LHS smokers were current cigarette smokers at time of assessment. These limitations notwithstanding, this study represents the first instance that an association is demonstrated between LHS and aggression using reliable and valid measures of this behavioral construct in adults.

There are several potential explanations regarding the association between LHS and aggression as observed in this study. LHS and aggressive behavior may both arise from a common vulnerability, such as Conduct Disorder, or central serotonergic (5-HT) system dysfunction. While Conduct Disorder is, by definition, associated with aggressive behavior, LHA Aggression scores remained high in LHS subjects even after those subjects with a Conduct Disorder diagnosis were removed from analysis. Accordingly, a common vulnerability for aggression via conduct disorder cannot account for increased aggression scores among most of our LHS subjects.

More likely, perhaps, is a relationship with central 5-HT system function. An inverse relationship between aggression and 5-HT system function has been demonstrated in a variety of clinical, and preclinical, studies for more than three decades (Coccaro et al., 2010). Consistent with this possibility, life history of smoking has been shown to be associated with dose-related inverse relationships in cerebrospinal fluid levels of 5-hydroxyindolacetic acid levels (Malone et al., 2003), prolactin responses to d,l-fenfluramine in depressed (Malone et al., 2003) or alcoholic (Anthenelli & Maxwell, 2000) subjects, and in the numbers of 5-HT transporters on human platelets (Patkar et al., 2003). This is consistent with data from studies which report that acute administration of nicotine is associated with an increase in 5-HT release (Ramos et al., 2004; Rausch, Fefferman, Ladisich-Rogers, & Menard, 1989) and a reduction in 5-HT uptake (Rausch et al., 1989). In contrast, chronic administration of nicotine is associated with a reduction of 5-HT in several brain areas in rats and in a reduction in brain 5-HT transporter sites in the brains of rats exposed to nicotine similar to that of human smokers (Xu et al., 2001). If so, there may be a role for 5-HT agents in the treatment of smokers with histories of aggressive behavior. If 5-HT agents can reduce aggression (Coccaro et al., 2009), can they also reduce smoking behaviors as has been reported in depressed, alcoholic patients (Cornelius, Perkins, Salloum, Thase, & Moss, 1999)?

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FIGURE 1a. Association of Life History of Smoking (LHS) with LHA Aggression Scores in Healthy Volunteers and Personality Disordered Subjects Controlled for SES & GAF. * $p < .05$.

FIGURE 1b. Association of Life History of Smoking (LHS) with BIS Impulsivity Scores in Healthy Volunteers and Personality Disordered Subjects Controlled for SES & GAF. * $p > .10$.

TABLE 1

Lifetime Axis I and II Disorders in the Personality Disorder Sample

Lifetime Axis I Disorders	Axis II Disorders
Major Depression: <i>n</i> = 19	Paranoid PD: <i>n</i> = 13
Dysthymia: <i>n</i> = 4	Schizoid PD: <i>n</i> = 8
Depressive-NOS: <i>n</i> = 12	Schizotypal PD: <i>n</i> = 2
Any Anxiety Disorder: <i>n</i> = 14	Narcissistic PD: <i>n</i> = 14
Alcoholism: <i>n</i> = 16	Antisocial PD: <i>n</i> = 13
Drug Dependence: <i>n</i> = 13	Histrionic PD: <i>n</i> = 4
Intermittent Explosive Disorder: <i>n</i> = 19	Obsessive-Comp: <i>n</i> = 15
Adjustment Disorder: <i>n</i> = 6	Avoidant PD: <i>n</i> = 4
Eating Disorder: <i>n</i> = 2	Dependant PD: <i>n</i> = 1
Other Axis I: <i>n</i> = 13	PD-NOS: <i>n</i> = 42

TABLE 2

Demographic, Functional and Behavioral Data on Subjects

Variable	Healthy Control (<i>n</i> = 78)	Personality Disorder (<i>n</i> = 101)	Statistic	<i>P</i>
Age	28.2 ± 7.5	30.9 ± 7.3	$t_{177} = 2.45$	.015
Gender (Male/Female)	48/30	80/21	Fisher Exact	.015
Race (White/Non-White)	48/30	54/47	Fisher Exact	.291
Hollingshead Socio-Economic Status (I/II/III/IV/V)	0/11/26/25/16	0/8/28/33/32	$\chi^2 (df = 1) = 3.98$	<.05
Global Assessment of Function (GAF)	84.3 ± 5.0	62.6 ± 8.1	$t_{169} = 22.10$	<.001
LHA Aggression	5.4 ± 4.5	8.3 ± 5.3	$t_{176} = 3.85$	<.001
BIS-11 Impulsivity	57.3 ± 8.2	63.7 ± 10.2	$t_{177} = 4.52$	<.001
EPQ Neuroticism	5.4 ± 3.2	9.9 ± 5.2	$t_{166} = 6.60$	<.001
EPQ Psychoticism	3.0 ± 2.2	4.2 ± 2.7	$t_{175} = 3.43$	.001
EPQ Extraversion	14.5 ± 4.2	13.3 ± 4.7	$t_{174} = 1.73$	.085