Illicit Drug Use, Cigarette Smoking, and Eating Disorder Symptoms: Associations in an Adolescent Twin Sample

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ABSTRACT. Objective: Twin studies have shown that genetic factors in part explain the established relation between alcohol use (i.e., problematic use or abuse/dependence) and eating disorder symptoms in adolescent and adult females. However, studies have yet to elucidate if there are similar shared genetic factors between other aspects of substance involvement, such as illicit drug use and repeated cigarette smoking. **Method:** For those sex-specific phenotypic correlations above our threshold of .20, we used a behavioral genetic design to examine potential shared genetic overlap between self-reported lifetime illicit drug use and repeated cigarette smoking and the eating disorder symptoms of drive for thinness (DT), bulimia (BU), and body dissatisfaction (BD), as assessed with the Eating Disorder Inventory-II, in 16- to 17-year-old female and male twin pairs. **Results:** Only phenotypic correlations with

illicit drug use met our threshold for twin modeling. Small to moderate genetic correlations were observed between illicit drug use and BU in both girls and boys and between illicit drug use and DT in girls. **Conclusions:** Similar etiological factors are at play in the overlap between illicit drug use and certain eating disorder symptoms in girls and boys during adolescence, such that genetic factors are important for covariance. Specifically, illicit drug use was associated with bulimia nervosa symptoms in girls and boys, which parallels previous substance use research finding a genetic overlap between alcohol use and bulimia nervosa symptoms. Future research should prospectively examine developmental trajectories to further understand the etiological overlap between substance involvement and eating disorder symptoms. (*J. Stud. Alcohol Drugs*, 79, 720–724, 2018)

EVIDENCE SUPPORTS AN ASSOCIATION between substance involvement and eating disorder (ED) symptoms during adolescence (e.g., Mann et al., 2014). In a large, U.S. representative sample of adolescent girls and boys reporting ED symptoms, 27% and 11% reported heavy alcohol use at least once a week monthly, 32% and 10% reported weekly cigarette smoking, and 31% and 10% reported past month illicit drug use, respectively (Neumark-Sztainer & Hannan, 2000). An etiological overlap between alcohol involvement and bulimia nervosa (BN) symptoms during adolescence and adulthood exists (Baker et al., 2010; Munn-Chernoff & Baker, 2016), and similar etiological associations have been suggested between BN and other substance use disorders in adult women (e.g., illicit drug abuse/dependence; Baker et al., 2010). However, little is known about the etiological overlap between substance in-

volvement, especially illicit drug use and cigarette smoking, and ED symptoms during adolescence. Therefore, this study examines the etiological overlap between illicit drug use and repeated cigarette smoking and three core ED symptoms during adolescence.

Twin studies indicate that a common genetic predisposition partly explains the etiological overlap between substance involvement and BN symptoms (Munn-Chernoff & Baker, 2016). During adulthood, shared genetic overlap has been observed between BN symptoms and substance involvement including alcohol use disorder, illicit drug use disorder, regular cigarette smoking, and problematic alcohol use (Baker et al., 2007, 2010; Munn-Chernoff et al., 2013, 2015; Slane et al., 2012; Trace et al., 2013). Shared genetic factors have also been observed between alcohol involvement and BN symptoms during adolescence, such that alcohol involvement accounted for up to 57% of the total heritability for BN symptoms (Baker et al., 2017). However, studies to date have almost exclusively examined shared genetic factors between alcohol involvement and BN symptoms. Only two studies have explored substances besides alcohol (Baker et al., 2007, 2010), and these only included adults. Thus, it remains unclear whether there is a similar genetic overlap between other substances and ED symptoms, particularly during adolescence.

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We aim to fill gaps in the literature by examining the etiological overlap between illicit drug use and cigarette smoking and ED symptoms—for those associations in which a phenotypic association is observed—in adolescent girls and boys to ascertain if there are shared genetic factors. As studies to date have mostly focused on problematic substance involvement, we evaluate any illicit drug use (versus an illicit drug use disorder) and repeated cigarette smoking, both of which may reflect problematic substance involvement for adolescents (Johnston et al., 2013).

Method

Participants

The Ethics Committee of Karolinska Institutet, Stockholm, Sweden and the University of North Carolina Institutional Review Board approved this project. This study sampled from the Swedish Twin study of Child and Adolescent Development (TCHAD): twin pairs born in Sweden from May 1985 until December 1986 (Lichtenstein et al., 2007). For detailed information regarding TCHAD, see Lichtenstein et al. (1997, 2007). Our sample included 16- to 17-year-old same-sex female ($n = 229$) and male ($n = 219$) monozygotic (MZ) twins and same-sex female ($n = 175$) and male ($n = 157$) dizygotic (DZ) twin pairs.

Measures

Participants self-reported lifetime illicit drug use and repeated cigarette smoking. A dichotomous variable was created indicating if the participant had ever used any type of illicit drug. Three categories assessed the frequency of repeated cigarette smoking: (0) non-repeated smoker (i.e., tried smoking or quit smoking [$n = 84$ identified “quitters”]), (1) smoke cigarettes sometimes (i.e., at parties or on the weekend); or (2) smoke cigarettes repeatedly/regularly (i.e., almost daily or daily). Because repeated cigarette smoking is conditional on having initiated cigarette use, participants who denied lifetime cigarette use were coded as missing to avoid estimation bias (Agrawal et al., 2016).

The drive for thinness (DT; excessive concern with dieting, preoccupation with weight, and pursuit of thinness), bulimia (BU; tendency toward binge eating episodes sometimes followed by self-induced vomiting), and body dissatisfaction (BD; believe specific body parts are too large) subscales of the Eating Disorder Inventory-II (EDI; Garner, 1991) examined current ED symptoms. Subscale scores range from 0 to 3, where higher scores indicate greater psychopathology for each ED symptom. For each subscale, missing item values were mean imputed if the participant responded to at least 75% of the items (Baker et al., 2009). Since the substance involvement data were categorical, the EDI subscales of DT and BD were categorized (0 = score of zero; 1 = score above

zero and below one standard deviation above the mean; 2 = score at least one standard deviation above the mean), and the BU subscale was dichotomized (0 = score of zero; 1 = score greater than zero) (Baker et al., 2017).

Statistical analyses

Sex-specific phenotypic correlations were examined between substances and EDI subscales, and twin modeling was completed for variables showing a sex-specific correlation of .20 or above, which corresponds to a moderate effect size. Next, to examine the etiological overlap between substance involvement and ED symptoms, a Cholesky decomposition was completed in Mx (Neale, 1991) using a raw data approach. The association between substance involvement and ED symptoms was decomposed into genetic (A; heritability), common environmental (C; environmental factors shared by twin pairs reared together), and individual-specific environmental (E; environmental factors unique to a twin member and measurement error) components. The Cholesky decomposition also estimates the genetic (r_a), common environmental (r_c), and individual-specific environmental (r_e) correlations for each bivariate association, estimating the proportion of overlapping genetic and environmental factors between the two traits.

The full ACE model fit was compared to two submodels with the A or C parameter estimate set to zero, respectively. Model comparisons were made using the difference in twice the negative log-likelihood of the models, which is distributed as a chi-square, and Akaike's information criterion (AIC; Akaike, 1987). A submodel can be rejected according to a significant ($p < .05$) change in chi-square, whereas lower AIC values indicate a better model fit. The model with the lowest AIC value is retained as the best-fit model.

Results

Most participants endorsed never having used illicit drugs (girls: 95.4%; boys: 95.4%) and not being a repeated smoker (girls: 60.0%; boys: 44.2%). For DT, girls had a mean (SD) score of 2.1 (4.0), whereas boys had a mean score of 0.34 (1.4). The BU subscale had the lowest mean score for girls ($M = 0.54$, $SD = 1.5$) and boys ($M = 0.49$, $SD = 1.3$), and BD had the highest mean score for girls ($M = 5.7$, $SD = 6.2$) and boys ($M = 2.2$, $SD = 3.6$). After categorization, most participants scored zero for DT (girls: 57.5%; boys: 84.0%) and BU (girls: 76.0%; boys: 77.0%). For BD, most girls (61.2%; boys: 32.4%) scored above zero and less than one standard deviation above the mean, whereas most boys (53%; girls: 24%) scored zero.

Phenotypic correlations were significantly greater for girls ($r_s = .13-.30$) compared with boys ($r_s = .04-.20$) for a majority of the illicit drug use-EDI associations and smoking-DT (Table 1). Follow-up twin models were completed for

TABLE 1. Phenotypic correlations [95% confidence interval] between illicit drug use, repeated smoking, and eating disorder symptoms

EDI subscale	Girls		Boys	
	Illicit drug	Smoking	Illicit drug	Smoking
Drive for thinness	.30* [.13, .43]	.18* [.07, .30]	.07 [-.20, .30]	.04 [-.13, .21]
Body dissatisfaction	.17* [.02, .33]	.13 [.01, .24]	.04 [-.15, .23]	.10 [-.03, .24]
Bulimia	.21 [.04, .40]	.14 [.01, .30]	.20 [-.01, .41]	.10 [-.06, .25]

Note: EDI = Eating Disorder Inventory.

*Fisher *r*-to-*z* transformation significant indicating a significant difference in correlation estimates between girls and boys; all *ps* < .01.

illicit drug use-DT and BU for girls and illicit drug use-BU for boys as they met the correlation threshold set for twin modeling.

Twin analyses

Model-fitting results, parameter estimates, and observed correlations from twin models are provided in Table 2. Based on chi-square change and AIC, the AE model was the best-fitting model for all associations.

For illicit drug use-DT in girls, the AE model estimated $r_a = .40$, 95% confidence interval (CI) [.23, .61] and $r_e = .05$, 95% CI [-.53, .61]. Illicit drug use accounted for 15% of the total heritability of DT in girls, indicating that more than 80% of the genetic variance on DT was unique to DT. For illicit drug use-BU in girls, $r_a = .20$, 95% CI [-.13, .34], whereas $r_e = .14$, 95% CI [-.43, .70]. The AE model for illicit drug use-BU in boys estimated $r_a = .16$, 95% CI [-.28, .57] and $r_e = .38$, 95% CI [-.36, .67]. For illicit drug use-BU in girls and boys, the r_a confidence intervals included zero, suggesting nonsignificant genetic correlations. However, we were unable to drop A from the model, indicating that genetic covariance is likely important and we were underpowered to detect a significant effect. Thus, for girls, illicit drug use accounted for 4% of the total heritability of BU, whereas for boys, illicit drug use accounted for 2% of the total heritability of BU.

Discussion

Our results generally confirm a moderate phenotypic association between substance involvement and EDs (Holderness et al., 1994; Mann et al., 2014; Munn-Chernoff & Baker, 2016), with significantly greater phenotypic correlations observed between illicit drug use and most ED symptoms in girls compared with boys. However, observed phenotypic associations with repeated cigarette smoking and BD were small, which contrasts with some previous research (e.g., Solmi et al., 2016; Wiseman et al., 1998). The lower prevalence of cigarette smoking in Swedish populations (Rodu et al., 2013) compared with the United States (King et al., 2012) could explain the limited associations between repeated cigarette smoking and ED symptoms. These results

may also suggest that developmentally specific associations exist between certain substances and ED symptoms that emerge only in adulthood, that associations only exist when a threshold-level ED is present, or that BD may not be a crucial factor regarding ED overlap with substance use.

We observed small to moderate genetic associations between illicit drug use and DT and BU in girls, confirming the genetic overlap observed between an illicit drug use disorder and BN in adult women (Baker et al., 2007). A small genetic correlation ($r_a = .16$) was also observed between illicit drug use and BU in boys, which is similar to the genetic correlation observed between alcohol dependence and binge eating in adult men ($r_a = .18$) (Munn-Chernoff et al., 2013). However, the genetic correlations observed between illicit drug use and BU were smaller than the genetic correlations observed between alcohol involvement and BN symptoms in adult ($r_a = .39$) and adolescent ($r_a = .53$) females (Baker et al., 2010, 2017). Further, there was no common environmental overlap between illicit drug use and BU or DT, whereas common environmental covariance was observed between alcohol intoxication and BU in adolescents (Baker et al., 2017). Although speculative, these differences may suggest that specific ED symptoms are differentially associated with certain substances (i.e., illicit drugs vs. alcohol) and that different etiological factors contribute to the overlap between different substances and ED symptoms. Notably, however, the genetic correlations between illicit drug use and BU were nonsignificant; thus, estimates are limited because of low statistical power and may differ in a larger sample.

We must acknowledge study limitations. The modest sample size and low frequency behaviors (e.g., in particular, the sample size of illicit drug users included in twin analyses was as follows: MZ-male = 20; DZ-male = 10; MZ-female = 23; DZ-female = 17) likely affected our statistical power and estimation of significant genetic correlations. However, we could not drop the genetic covariance from models, suggesting the importance of genetic contribution. Limited power may have also influenced our estimation of common environmental effects, which have been observed in adolescents for alcohol use (Baker et al., 2017). It is possible with larger sample sizes that common environmental covariance could be observed. The sample is a community-based, non-clinical sample of late adolescents in which diagnoses were

TABLE 2. Model fit statistics, parameter estimates, and observed correlations from twin models in girls and boys

Model	Model fit statistics			Standardized parameter estimates [95% CI]								
				Substance			Eating disorder symptom			Correlation		
	-2lnL (df)	$\Delta\chi^2$	AIC	a ²	c ²	e ²	a ²	c ²	e ²	r _a	r _c	r _e
Illicit drug use-DT (Girls)												
ACE	1,671.1 (1590)	—	-1,509.0	65 [61, 86]	25 [0, 60]	10 [3, 27]	53 [17, 80]	15 [0, 45]	32 [23, 44]	.18 [-.84, .81]	1.0 [-1.0, 1.0]	.10 [-.51, .63]
AE	1,672.4 (1593)	1.3	-1,514.0	91 [74, 94]	—	9 [4, 30]	70 [58, 80]	—	31 [22, 42]	.40 [.23, .61]	—	-.53, .61]
CE	1,685.0 (1593)	13.6*	-1,501.3	—	80 [59, 90]	20 [9, 41]	—	55 [45, 65]	45 [35, 55]	—	.47 [.22, .72]	.01 [-.42, .43]
Illicit drug use-BU (Girls)												
ACE	1,140.0 (1590)	—	-2,040.3	72 [72, 88]	19 [3, 73]	10 [2, 28]	30 [0, 50]	17 [0, 50]	54 [35, 74]	.73 [.38, .92]	-1.0 [-1.0, .70]	.10 [-.54, .41]
AE	1,140.8 (1593)	1.1	-2,045.2	91 [74, 98]	—	9 [2, 26]	49 [37, 66]	—	51 [34, 71]	.02 [-.13, .34]	—	.14 [-.43, .70]
CE	1,147.0 (1593)	7.3	-2,040.0	—	79 [57, 91]	21 [10, 43]	—	40 [23, 54]	60 [46, 80]	—	.18 [-.20, .54]	.20 [-.31, .64]
Illicit drug use-BU (Boys)												
ACE	979.0 (1459)	—	-1,940.0	42 [0, 67]	40 [0, 86]	19 [5, 46]	30 [0, 61]	15 [0, 47]	56 [37, 77]	1.0 [-1.0, 1.0]	-1.0 [-1.0, 1.0]	.35 [-.36, .34]
AE	981.0 (1462)	3.0	-1,943.5	82 [55, 88]	—	18 [5, 45]	46 [23, 64]	—	54 [36, 77]	.16 [-.28, .57]	—	.38 [-.36, .67]
CE	982.0 (1462)	3.1	-1,942.3	—	75 [62, 90]	25 [10, 52]	—	37 [18, 53]	63 [47, 82]	—	.06 [-.42, .50]	.48 [-.10, .78]

Notes: Best-fit model shown in **bold**. CI = confidence interval; -2lnL = difference in twice the negative log-likelihood of the models; $\Delta\chi^2$ = chi-square difference between full model and submodel; AIC = Akaike Information Criterion; a² = heritability; c² = common environmental parameter estimate; e² = individual-specific environment parameter estimate; r_a = genetic correlation; r_c = common environmental correlation; r_e = individual-specific environmental correlation; DT = drive for thinness; BU = bulimia.

*significant chi-square change at $p < .05$.

not assessed; thus, results may not generalize to clinical populations. Importantly, however, our study had no missing cells in the substance involvement-ED symptom bivariate associations, illicit drug use and repeated cigarette smoking rates were similar to the prevalence found in Swedish 16-year-olds near the TCHAD data collection timeframe (European School Survey Project on Alcohol and Other Drugs, 2003), and mean scores for the EDI subscales were similar to other community populations (e.g., Nevonen et al., 2006; Slane et al., 2014; Stice et al., 2004). Finally, we were not able to assess the etiological overlap between ED symptoms and substance use longitudinally. Given that common environmental effects for ED symptoms have been observed during pre-puberty (Klump et al., 2012), common environmental covariance may be important during childhood and early adolescence—which we were not able to examine here.

Our results support a genetic overlap between illicit drug use and specific ED symptoms in adolescent girls and boys. Taken together with previous findings, our findings preliminarily suggest that the genetic overlap between substance involvement and EDs may differ according to specific substances and ED symptoms. However, this is speculative and findings must be replicated in larger sample sizes. Results also support the assessment of ED symptoms in adolescents with problematic substance use, especially assessing BU and DT symptoms in girls who use drugs and BU symptoms in boys who use drugs. Future research should use prospective,

longitudinal research designs to determine the developmental trajectories of substance use-ED etiological associations as well as examine the mechanisms underlying these associations—in particular, whether unique mechanistic/genetic factors exist for symptom-specific associations.

References

- Agrawal, A., Edenberg, H. J., & Gelernter, J. (2016). Meta-analyses of genome-wide association data hold new promise for addiction genetics. *Journal of Studies on Alcohol and Drugs*, 77, 676–680. doi:10.15288/jsad.2016.77.676
- Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, 52, 317–332. doi:10.1007/BF02294359
- Baker, J. H., Maes, H. H., Lissner, L., Aggen, S. H., Lichtenstein, P., & Kendler, K. S. (2009). Genetic risk factors for disordered eating in adolescent males and females. *Journal of Abnormal Psychology*, 118, 576–586. doi:10.1037/a0016314
- Baker, J. H., Mazzeo, S. E., & Kendler, K. S. (2007). Association between broadly defined bulimia nervosa and drug use disorders: Common genetic and environmental influences. *International Journal of Eating Disorders*, 40, 673–678. doi:10.1002/eat.20472
- Baker, J. H., Mitchell, K. S., Neale, M. C., & Kendler, K. S. (2010). Eating disorder symptomatology and substance use disorders: Prevalence and shared risk in a population based twin sample. *International Journal of Eating Disorders*, 43, 648–658. doi:10.1002/eat.20856
- Baker, J. H., Munn-Chernoff, M. A., Lichtenstein, P., Larsson, H., Maes, H., & Kendler, K. S. (2017). Shared familial risk between bulimic symptoms and alcohol involvement during adolescence. *Journal of Abnormal Psychology*, 126, 506–518. doi:10.1037/abn0000268

- European School Survey Project on Alcohol and Other Drugs. (2003). *The ESPAD report 2003: Alcohol and other drug use among students in 35 European countries*. Retrieved from <http://www.espad.org/content/espad-report-2003-alcohol-and-other-drug-use-among-students-35-european-countries>
- Garner, D. (1991). *Eating disorders inventory-2: Professional manual*. Odessa, FL: Psychological Assessment Resources.
- Holderness, C. C., Brooks-Gunn, J., & Warren, M. P. (1994). Co-morbidity of eating disorders and substance abuse review of the literature. *International Journal of Eating Disorders*, 16, 1–34. doi:10.1002/1098-108X(199407)16:1<1::AID-EAT2260160102>3.0.CO;2-T
- Johnston, L. D., O'Malley, P. M., Bachman, J. G., & Schulenberg, J. E. (2013). *Monitoring the Future national results on adolescent drug use: Overview of key findings*. Retrieved from www.monitoringthefuture.org/pubs/monographs/mtf-overview2012.pdf
- King, B. A., Dube, S. R., & Tynan, M. A. (2012). Current tobacco use among adults in the United States: Findings from the National Adult Tobacco Survey. *American Journal of Public Health*, 102, e93–e100. doi:10.2105/AJPH.2012.301002
- Klump, K. L., Culbert, K. M., Slane, J. D., Burt, S. A., Sisk, C. L., & Nigg, J. T. (2012). The effects of puberty on genetic risk for disordered eating: Evidence for a sex difference. *Psychological Medicine*, 42, 627–637. doi:10.1017/S0033291711001541
- Lichtenstein, P., & Svartengren, M. (1997). Genes, environments, and sex: Factors of importance in atopic diseases in 7-9-year-old Swedish twins. *Allergy*, 52, 1079–1086. doi:10.1111/j.1398-9995.1997.tb00179.x
- Lichtenstein, P., Tuvblad, C., Larsson, H., & Carlström, E. (2007). The Swedish Twin study of CHild and Adolescent Development: The TCHAD-study. *Twin Research and Human Genetics*, 10, 67–73. doi:10.1375/twin.10.1.67
- Mann, A. P., Accurso, E. C., Stiles-Shields, C., Capra, L., Labuschagne, Z., Karnik, N. S., & Le Grange, D. (2014). Factors associated with substance use in adolescents with eating disorders. *Journal of Adolescent Health*, 55, 182–187. doi:10.1016/j.jadohealth.2014.01.015
- Munn-Chernoff, M. A., & Baker, J. H. (2016). A primer on the genetics of comorbid eating disorders and substance use disorders. *European Eating Disorders Review*, 24, 91–100. doi:10.1002/erv.2424
- Munn-Chernoff, M. A., Duncan, A. E., Grant, J. D., Wade, T. D., Agrawal, A., Bucholz, K. K., . . . Heath, A. C. (2013). A twin study of alcohol dependence, binge eating, and compensatory behaviors. *Journal of Studies on Alcohol and Drugs*, 74, 664–673. doi:10.15288/jsad.2013.74.664
- Munn-Chernoff, M. A., Grant, J. D., Agrawal, A., Sartor, C. E., Werner, K. B., Bucholz, K. K., . . . Duncan, A. E. (2015). Genetic overlap between alcohol use disorder and bulimic behaviors in European American and African American women. *Drug and Alcohol Dependence*, 153, 335–340. doi:10.1016/j.drugalcdep.2015.05.043
- Neale, M. (1991). *Statistical modelling with Mx*. Richmond, VA: Department of Psychiatry.
- Neumark-Sztainer, D., & Hannan, P. J. (2000). Weight-related behaviors among adolescent girls and boys: Results from a national survey. *Archives of Pediatrics & Adolescent Medicine*, 154, 569–577. doi:10.1001/archpedi.154.6.569
- Nevonen, L., Clinton, D., & Norring, C. (2006). Validating the EDI-2 in three Swedish female samples: Eating disorders patients, psychiatric outpatients and normal controls. *Nordic Journal of Psychiatry*, 60, 44–50. doi:10.1080/08039480500504537
- Rodu, B., Jansson, J. H., & Eliasson, M. (2013). The low prevalence of smoking in the Northern Sweden MONICA study, 2009. *Scandinavian Journal of Public Health*, 41, 808–811. doi:10.1177/1403494813504836
- Slane, J. D., Burt, S. A., & Klump, K. L. (2012). Bulimic behaviors and alcohol use: Shared genetic influences. *Behavior Genetics*, 42, 603–613. doi:10.1007/s10519-012-9525-2
- Slane, J. D., Klump, K. L., McGue, M., & Iacono, W. G. (2014). Developmental trajectories of disordered eating from early adolescence to young adulthood: A longitudinal study. *International Journal of Eating Disorders*, 47, 793–801. doi:10.1002/eat.22329
- Solmi, M., Veronese, N., Sergi, G., Luchini, C., Favaro, A., Santonastaso, P., . . . Stubbs, B. (2016). The association between smoking prevalence and eating disorders: A systematic review and meta-analysis. *Addiction*, 111, 1914–1922. doi:10.1111/add.13457
- Stice, E., Burton, E. M., & Shaw, H. (2004). Prospective relations between bulimic pathology, depression, and substance abuse: Unpacking comorbidity in adolescent girls. *Journal of Consulting and Clinical Psychology*, 72, 62–71. doi:10.1037/0022-006X.72.1.62
- Trace, S. E., Thornton, L. M., Baker, J. H., Root, T. L., Janson, L. E., Lichtenstein, P., . . . Bulik, C. M. (2013). A behavioral-genetic investigation of bulimia nervosa and its relationship with alcohol use disorder. *Psychiatry Research*, 208, 232–237. doi:10.1016/j.psychres.2013.04.030
- Wiseman, C. V., Turco, R. M., Sunday, S. R., & Halmi, K. A. (1998). Smoking and body image concerns in adolescent girls. *International Journal of Eating Disorders*, 24, 429–433. doi:10.1002/(SICI)1098-108X(199812)24:4<429::AID-EAT10>3.0.CO;2-D