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Familial and Special Twin Influences on Cigarette Use Initiation

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Background: Shared experiences within families play an important role in the initiation of cigarette use among adolescents. Behavioral genetic studies using various samples have implicated that the shared environment that twins experience is an important source of influence on whether adolescents initiate cigarette use. Whether the special twin environment, in addition to the shared environment, contributes significantly to making twin siblings more similar in cigarette initiation, and whether the influence of the special twin environment persists into adulthood, is less clear. *Methods:* Data for this study came from the National Longitudinal Survey of Adolescent Health. Twin, full-, and half-sibling pairs between the ages of 12 and 33 were separated into three age groups, with about 3,000 individuals in each age group. The proportion of variance in cigarette use initiation explained by genetic, shared, special twin, and unique environmental factors were examined. *Results:* The results of separate age-moderated univariate variance decomposition models indicate that the special twin environment does not significantly contribute to the variance in cigarette use initiation in adolescence or young adulthood. *Conclusion:* Factors shared by individuals in a family, but that are not specific to being a twin, are important in determining whether adolescents will initiate the use of cigarettes.

■ **Keywords:** adolescence, cigarette initiation, special twin environment, shared environment

Adolescence is a vulnerable developmental time for the onset of regular cigarette use (Substance Abuse and Mental Health Services Administration, 2007) and for the development of nicotine dependence (U.S. Department of Health and Human Services, 1994). Despite substantial decreases in the prevalence of 30-day cigarette use during adolescence (Johnston et al., 2013; 2015), the majority of adults report initiating cigarette use during adolescence. Epidemiological studies have suggested that cigarette use by siblings (Bricker et al., 2006; Gilman et al., 2009; O'Loughlin et al., 2009) is an important influence on adolescents' initiation, and use of, cigarettes. Having a sibling who is a smoker increases the chances that adolescents will initiate the use of cigarettes (Avenevoli & Merikangas, 2003; Rohde et al., 2003). Further, siblings of individuals who are habitual smokers have higher rates of cigarette use than siblings of controls (Bierut et al., 1998), and having a twin or a sibling who uses cigarettes is a predictor of initiating cigarette use (Vink et al., 2003).

In genetically informed studies that employ twins, the shared environment (c^2) refers to aspects of the environment that are responsible for making siblings within the same family similar, while the non-shared environment refers to factors responsible for making siblings in the same family different from one another. Aspects of the familial environment common to both siblings include the experience of parent–child conflict (Burt et al., 2003) or experiencing parental divorce (Burt et al., 2008; D'Onofrio et al., 2005). Shared environmental influences that are considered to make twins more similar to each other account for over a third of the stability in psychopathology in childhood and adolescence (Bartels et al., 2004).

Classical twin studies include data from pairs of monozygotic (MZ) and dizygotic (DZ) twins and can be extended by including pairs of individuals that differ in their genetic relatedness, but who experience the same familial environment. For instance, full-siblings are as genetically related to one another as pairs of DZ twins and share 50% of their genes, while half-siblings share

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25% of their segregating genes. When reared in the same home environment, MZ and DZ twins, as well as full-siblings, share 100% of their rearing environment. Due to disruptions in living situations, half-siblings do not experience the same rearing environment as one another. Adding full-sibling and half-sibling pairs in a genetically informed analysis provides an additional contrast of genetic relatedness and allows for the effect of the special twin environment (t^2) to be estimated in addition to the effect of the shared environment (c^2). We define the special twin environment as an influence specific and unique to being a twin that, in addition to the shared environment, has an effect on a phenotype, and makes twins more similar than full-siblings.

Twin studies have established that shared environmental factors (c^2) within a family account for about 34–55% of the variation in adolescent initiation of cigarette use (Seglem et al., 2015; Unger et al., 2011) and that there is a developmental shift in the factors that influence cigarette use initiation into adulthood (Maes et al., 2016). The strength of the shared environment is strongest in early adolescence. In middle adolescence and continuing on to adulthood, the influence of the shared environment shifts such that additive genetic influences increase in strength (Bares et al., 2015; Maes et al., 1999). Previous studies have found shared environmental influences specific to twins that play a role in the initiation of cigarette use among 12- to 19-year-olds (Rhee et al., 2003) and explain 30% of the variance in regular tobacco use (Young et al., 2006). Further, studies capitalizing on the relatedness of twins and sibling pairs indicate that among pairs that are highly socially connected, that is, have friends in common, the effect of the shared environment on cigarette use is greater (Slomkowski et al., 2005).

Whether the influence of the special twin environment in the initiation of cigarette use extends beyond adolescence and into adulthood has not been explored. Given the importance of the shared environment as a risk factor for the initiation of cigarette use in adolescence, the aims of the present study were to test whether the special twin environment, in addition to the shared environment, has a unique influence on cigarette use initiation, and to examine the developmental specificity of the influence of the special twin environment across adolescence and into adulthood. Due to the larger role that the shared environment plays in the initiation of cigarette use during adolescence relative to genetic factors, we hypothesized that the special twin environment would contribute significantly to the variance in cigarette use initiation in adolescence.

Materials and Methods

Participants

For this study, we used data from twin and siblings pairs available in the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2013; Harris et al., 2013). The Add Health participants are a nationally representative and longitudinal sample of adolescents followed over the course of 20 years through four assessments. Add Health participants provided written informed consent for participation in all aspects of Add Health in accordance with the University of North Carolina School of Public Health Institutional Review Board guidelines that are based on the Code of Federal Regulations on the Protection of Human Subjects 45CFR46.

The initial survey took place when participants were 12–17 years of age, and the most recent assessment occurred when participants were 26–33 years old (Harris, 2013). Over 3,000 pairs of the participants, who took part in the Add Health in-school survey lived in the same household (Harris et al., 2009) and were twins, full- or half-siblings, adopted siblings, or unrelated individuals (i.e., foster siblings; Harris et al., 2006; 2013). For this study, data came from pairs of MZ and DZ twins, as well as full- and half-siblings who lived in the same household at the time of the survey.

Measures

Cigarette use initiation. Participants were asked to report on whether they had ever smoked a cigarette, even just a puff. A binary variable was created for each participant that indicated whether they had ever smoked a cigarette (coded as 1) or not (coded as 0).

Zygosity. The zygosity of each individual in a twin pair was determined through self-reported answers to questions about the degree of similarity between each twin and additionally by matching on 12 molecular genetic markers. Additional details on how the biological markers were obtained have been described elsewhere (Harris et al., 2006).

Sibling type. The genetic relatedness of individuals who were part of the same family was determined through self-reported answers to an in-school survey that asked participants to identify the individuals in their family with whom they lived (Harris et al., 2006; 2013). We excluded pairs of individuals who were unrelated (e.g., foster siblings) to other individuals in the same household.

Sex. Participants' self-reported sex was used as well.

Twin and Sibling Pairs

For the present study, twin pairs were divided into five groups based on sex and zygosity; MZ males (MZM), MZ females (MZF), DZ males (DZM), DZ females (DZF), and opposite-sex DZ twins (DZO). Then, additional subgroups for the sibling pairs were created based on whether the sibling pairs were full- or half-siblings, and based on their gender. The additional six subgroups included full-sibling males (FSM), full-sibling females (FSF), full-siblings of the

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opposite sex (FSO), half-sibling males (HSM), half-sibling females (HSF), and half-siblings of the opposite sex (HSO). Due to the design of the Add Health study, pairs of twins and siblings in the sample lived in the same household at the time of the survey (Harris et al., 2006). However, it was not possible to distinguish how long half-siblings had cohabited or whether pairs of half-siblings were related through their mothers or through their fathers because more detailed questions regarding how pairs of siblings were related to each other were not part of the survey.

Age Groups

Three age groups were created that span three developmental periods: adolescence (ages 12–17), young adulthood (ages 18–25), and adulthood (ages 26–33). To be included in an age group, both individuals belonging to a twin or sibling pair had to be within the age range specified. If an individual participant fell within the age ranges but had a sibling who was not within that age range, neither one was included in the analyses.

Analysis Plan

The classical twin study examines the similarity in the variance of a phenotype between MZ and DZ twins. MZ twins share 100% of their segregating genes, while DZ twins share, on average, 50%. Three sources of phenotypic variance can be estimated by examining the similarities between pairs of twins: additive genetic (a^2), shared environment (c^2), and unique environment (e^2). Additive genetic sources include the effect of multiple genetic loci that act in an additive manner to influence the variance of a phenotype. The shared environment is thought to include aspects of the environment common to both twins within a family that are considered to make twins more similar to each other. Unique environmental influences arise from factors not shared within families and are often due to individual-specific experiences.

In this study, we extended the classical twin study by including pairs of individuals from within the same family, who differed in their genetic relatedness. Full-siblings share on average 50% of their genes and half-siblings share 25% of their genes identical by descent. When reared together, twins and full-siblings share 100% of the rearing environment. For part of their life, individuals related to one another as half-siblings will have experienced different rearing environments. Our models accounted for the fact that half-siblings do not share 100% of their rearing environment by constraining the shared environment covariance to be no larger than 50% (Figure 1, Panel A). Including, full- and half-siblings along with MZ and DZ twins creates contrasts and allows for estimates of the presence of a special twin environment (t^2) in addition to additive genetic (a^2), shared environmental (c^2), and unique environmental (e^2) influences on the phenotype of interest.

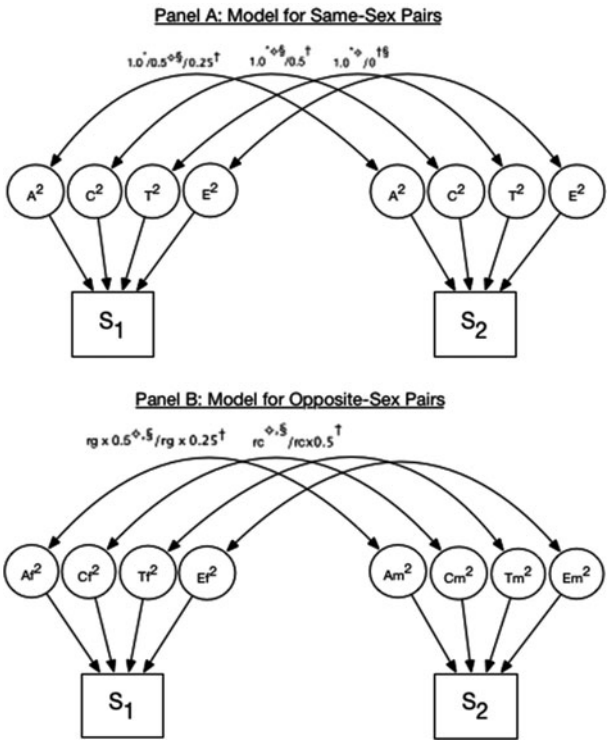

FIGURE 1

Conceptual ACTE model. Note: S_1 = Sibling 1, S_2 = Sibling 2. A^2 = Additive genetic effects, C^2 = Shared environmental effects, T^2 = Special twin environmental effects, E^2 represents the effects of unique environment. The following model constraints are applied depending on sibling type: *MZ pairs; ✧DZ pairs; § Full-sibling pairs; † Half-sibling pairs. A unique constraint (r_g, r_c) is applied to opposite-sex pairs (depicted in Panel B).

Genetic Model-Fitting

Tetrachoric correlations for the binary cigarette use initiation variable were computed separately for twin and sibling pairs with complete data on the study variables (less than 2% of pairs of twins and siblings had missing data). Twin modeling assumptions that thresholds for cigarette use initiation could be equated across twin order, sibling order, zygosity, and sibling type, for males and females, were tested next.

To test the specific role of the special twin environment, age- and sex-specific univariate variance decomposition models adjusted for the age of each sibling pair were run for cigarette use initiation (Figure 1, Panel A). Constraints were added to each covariance depending on the type of sibling relationship. The genetic covariance was constrained to be 1.0 for MZ twins, 0.5 for DZ twins and full-siblings, and 0.25 for half-siblings. We constrained the shared environment covariance to 1.0 for all twin pairs and full-siblings. However, for the half-siblings, the shared environment covariance was constrained to be 0.5. Lastly, the twin environment covariance was constrained to be 1.0 in twin pairs but it was set at zero for full- and half-siblings.

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TABLE 1

Participant Demographics (Mean [SD]/%) by Genetic Relatedness and Age Group Genetic relatedness

	All	Monozygotic	Dizygotic	Full-siblings	Half-siblings	
	12- to 17-year-olds					Sig.
n	3,078	452	802	1,360	464	–
Sex (% female)	50.3%	51.0%	49.1%	49.9%	52.8%	*ns*
Age	15.6 (1.4)	15.8 (1.3)	15.7 (1.4)	15.7 (1.4)	15.4 (1.6)	<0.001
Age difference*	-0.06 (1.8)	0.01 (0.2)	0.00 (0.1)	-0.09 (2.2)	-0.14 (2.6)	*ns*
Cigarette use initiation	44.6%	40.0%	41.4%	45.4%	51.9%	0.001
Unique families	97.7%	99.1%	100.0%	97.2%	94.0%	<0.0001
	18- to 25-year-olds					
n	3,264	490	822	1,490	462	–
Sex (% female)	50.9%	53.5%	47.5%	51.7%	51.7%	*ns*
Age	22.4 (1.7)	22.6 (1.6)	22.5 (1.6)	22.5 (1.7)	22.0 (1.9)	<0.0001
Age difference*	-0.03 (2.0)	0.01 (0.2)	0.00 (0.1)	-0.08 (2.4)	0.05 (3.0)	*ns*
Cigarette use initiation	81.5%	81.7%	81.5%	81.1%	82.9%	*ns*
Unique families	97.9%	99.2%	100.0%	97.6%	93.5%	<0.0001
	26- to 33-year-olds					
n	2,916	440	736	1,342	398	–
Sex (% female)	51.9%	53.8%	49.1%	51.6%	56.0%	*ns*
Age	29.1 (1.6)	29.2 (1.5)	28.9 (1.6)	29.1 (1.6)	28.9 (1.8)	<0.01
Age difference*	-0.01 (1.9)	0.00 (0.1)	0.00 (0.1)	-0.05 (2.3)	0.06 (2.9)	*Ns*
Cigarette use initiation	82.6%	78.8%	82.8%	82.8%	85.2%	*ns*
Unique families	98.4%	99.6%	100.0%	97.6%	97.0%	<0.0001

Note: *Difference in age between siblings in the same family.

We tested for two types of sex differences: qualitative and quantitative sex differences. Qualitative sex differences refer to the possibility that genetic effects influence a phenotype in one sex but not in the other sex. We tested for qualitative sex differences by constraining additive genetic, shared environment, special twin environment, and unique environment parameters to be equal across males and females and observing changes to model fit. Quantitative sex differences, however, refer to factors that have a stronger influence in one sex than the other. To test for quantitative sex differences, twin models constrain the genetic correlation, r_g, to be 1.0 (see Figure 1, Panel B) and the correlation between shared environments, r_c, to be 1.0 in opposite sex twin and sibling pairs, and examine changes to the fit of the model.

The first model run, Model 1, was a model where parameter estimates for additive genetic (a^2), shared environment (c^2), special twin environment (t^2), and unique environment (e^2) were estimated for the five twin zygosity groups (MZF, MZM, DZF, DZM, and DZO) and the six sibling groups (FSF, FSM, FSO, HSF, HSM, and HSO) with the genetic correlation (r_g) and shared environment (r_c) correlation freely estimated.

At each age group, the full model included age-moderated paths and age-moderated thresholds for cigarette use initiation. The influence of age moderation on the paths was tested (Model 2), as well as the influence of age-moderation on the threshold for cigarette initiation (Model 3). Next, differences in whether the same set of genes (Model 4) and the same set of shared environment influences (Model 5) influenced males and females to the same degree was tested. The next model tested quantitative sex differences (Model 6). Lastly, the significance of each parameter estimate was tested in Models 7 through 10 by removing it from the model and examining changes in model fit through chi-square tests.

Results

Participants for this study included 3,078 12- to 17-year-olds twins ($n = 1{,}254$) and siblings ($n = 1{,}824$); 3,264 18- to 25-year-old twins ($n = 1{,}312$) and siblings ($n = 1{,}952$); and 2,916 26- to 33-year-old twins ($n = 1{,}176$) and siblings ($n = 1{,}740$). About half of the sample at each age group was female (see Table 1). The prevalence of having ever used a cigarette was 44.6% for the 12- to 17-year-old group overall, and a significant difference was observed in cigarette use prevalence by sibling type such that half-siblings had the highest prevalence (51.9%) and MZ twins the lowest (40.0%; see Table 1).

Because multiple pairs of twins and siblings were allowed to come from the same family (e.g., a pair of MZ twins and a pair of full-siblings living in the same household), we examined the number of times that each family was duplicated. In each age group, over 97% of the pairs in the sample were unique, in that each family contributed only one pair. Only a small proportion of participants came from families that contributed multiple pairs to the sample. Less than 3% of pairs across each age group came from families that had more than one pair from the same family. Significant differences by genetic relatedness were observed in the proportion of families that were duplicated. A greater proportion of half-siblings than full-siblings or twins came from families that were included in the sample multiple times.

Twin Correlations

The tetrachoric correlations for cigarette use initiation by age group, sex, and sibling type are presented in Table 2 and

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TABLE 2
Tetrachoric Correlations (95% CI)

	Cigarette use initiation		
	Correlations	95% CI	*n*
12- to 17-year-olds (n = 1,539 pairs)			
MZF	0.68	(0.58,0.77)	116
MZM	0.57	(0.45, 0.69)	110
DZF	0.67	(0.57, 0.76)	117
DZM	0.57	(0.46, 0.68)	123
DZO	0.25	(0.13, 0.38	161
FSF	0.60	(0.51, 0.68)	194
FSM	0.44	(0.34, 0.54)	195
FSO	0.20	(0.11, 0.29)	291
HSF	0.37	(0.20, 0.55)	66
HSM	0.42	(0.23, 0.61)	52
HSO	0.27	(0.13, 0.41)	114
18- to 25-year-olds (n = 1,629 pairs)			
MZF	0.73	(0.63, 0.83)	131
MZM	0.82	(0.73, 0.90)	113
DZF	0.48	(0.34, 0.63)	109
DZM	0.27	(0.09, 0.45)	129
DZO	0.29	(0.14, 0.43)	171
FSF	0.40	(0.29, 0.51)	228
FSM	0.44	(0.33, 0.56)	204
FSO	0.41	(0.31, 0.52)	313
HSF	0.37	(0.18, 0.56)	68
HSM	0.41	(0.13, 0.70)	58
HSO	-0.02	(-0.31, 0.27)	105
26- to 33-year-olds (n = 1,438 pairs)			
MZF	0.81	(0.73, 0.90)	114
MZM	0.88	(0.81, 0.94)	96
DZF	0.46	(0.31, 0.61)	103
DZM	0.57	(0.41, 0.74)	107
DZO	0.42	(0.27, 0.57)	148
FSF	0.53	(0.42, 0.63)	206
FSM	0.39	(0.25, 0.53)	189
FSO	0.35	(0.23, 0.47)	276
HSF	0.50	(0.31, 0.68)	62
HSM	0.54	(0.19, 0.90)	37
HSO	0.07	(-0.23, 0.37)	100

are discussed first by comparing the correlations across zygosity and siblings. The MZ and DZ correlations were similar for the 12- to 17-year-old group, suggesting shared environmental influences. The MZ correlation exceeded the DZ correlation in the two oldest age groups (18- to 25-year-olds and 26- to 33-year-olds), likely suggesting that genetic factors contribute to variation in cigarette use initiation. Comparing correlations between twins and siblings, we found that the DZ correlation was slightly higher than the sibling correlations in the youngest (12- to 17-year-old) group, possibly suggesting the influence of the special twin environment. In the 18- to 25-year-old group as well as in the oldest (26- to 33-year-old) group, the correlations between the DZ and sibling groups were similar and did not suggest the presence of a special twin environment.

Twin Model Assumptions

We next tested twin model assumptions (Table 3) that the thresholds for the binary cigarette use variable could be equated for each member of pairs of twins and siblings to test the effect of twin and sibling order, across sibling type (twin vs. siblings), as well as across same- and opposite-sex pairs, and males and females. In the 12- to 17- and 18- to 25-year-old groups, the thresholds could be equated across twin and sibling order, across sibling type, and across males and females in twin pairs. However, in the 18- to 25-year-old group, the thresholds could not be equated across same-sex and opposite-sex pairs or across males and females in sibling pairs. In the 26- to 33-year-olds, thresholds could not be equated across sibling order in sibling pairs, across same-sex and opposite-sex pairs, or across males and females in sibling pairs. Given these results, subsequent models included for all age groups separate thresholds for males and females within each zygosity and sibling group. In addition, we included each individual's age as a covariate on both the thresholds and parameter estimates in the variance decomposition models.

Genetic Models

Table 4 presents the fit statistics for each of the age-specific, variance decomposition models that were carried out for cigarette use initiation. At each age group, the full model included age-moderated, sex-specific, and sibling-specific paths, and thresholds for cigarette use initiation (Table 4, Model 1). We tested the influence of the age-moderated paths by removing them from the full model. The resulting change in model fit was not significant in any of the age groups. We then tested the effect of removing the age moderation on the threshold, which significantly reduced model fit (Model 2) in the 12- to 17-year-old and the 18- to 25-year-old groups (12- to 17-year-olds $\chi^2 = 8.34$, $p = .004$; 18- to 25-year-olds $\chi^2 = 5.09$, $p = .024$). Subsequent models included the age-moderated threshold for the 12- to 17- and 18- to 25-year-old age groups. However, in the 26- to 33-year-old group, removal of the age-moderated paths or the age-moderated thresholds did not significantly reduce the fit of the model ($\chi^2 = 2.11$, $p = ns$), suggesting that for the adult group of twins and siblings, age did not significantly influence the thresholds of cigarette use or path estimates. Therefore, the age moderation was not included in subsequent models for the 26- to 33-year-olds.

Next, we tested whether the same set of genes (Model 4) had the same influence on cigarette use initiation across males and females (qualitative sex differences) by setting the genetic correlation (r_g) to 1. In Model 5, we tested whether the same set of familial experiences influenced initiation of cigarettes in males and females by constraining the shared environmental correlation (r_c) to 1. Across each of the three age groups, the genetic correlation (r_g) was constrained to 1 without observing reductions in model fit. Only in the 12- to 17-year-old group was a significant deterioration in model fit found when both the genetic and shared environmental correlation were constrained to be 1.0 (Model 5: $\chi^2 = 4.10$, $p = .040$). Subsequent models for the 12- to -17-year old group included a constraint that r_g was estimated at 1 and r_c was allowed to be freely estimated. In the two older age groups, both r_g and r_c were constrained to be 1 in subsequent models. Constraining the parameter

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TABLE 3

Testing Twin Model Assumptions by Age Group for Cigarette use Initiation

	Estimated Parameters	–2 LL	df	AIC	ΔLL	Δdf	*p*
12- to 17-year-old group (n = 1,539 pairs)							
1. Saturated model	33	4039.9	3045	-2050.10	–	–	–
2. Equal thresholds across sibling order in twins	28	4044.9	3050	-2055.14	4.96	5	.42
3. Equal thresholds across sibling order in siblings	27	4044.0	3051	-2058.04	4.06	6	.67
4. Equal thresholds across sibling order in twins and siblings	22	4048.9	3056	-2063.08	9.02	11	.62
5. Equal thresholds across zygosity (MZ vs. DZ)	20	4053.3	3058	-2062.65	4.43	2	.11
6. Equal thresholds across sibling type (Full- vs. half-siblings)	20	4052.9	3058	-2063.14	3.95	2	.14
7. Equal thresholds across relationship type (MZ/DZ vs. siblings)	16	4059.2	3062	-2064.78	10.30	6	.11
8. Equal thresholds across SS & OS	15	4063.1	3063	-2062.94	14.14	7	.05
9. Equal thresholds across f/m sex in twins	19	4053.5	3059	-2064.51	4.58	3	.21
10. Equal thresholds across f/m sex in siblings	19	4053.5	3059	-2064.53	4.55	3	.21
18- to 25-year-old group (n = 1,629 pairs)							
1. Saturated model	33	2988.6	3200	-3411.38	–	–	–
2. Equal thresholds across sibling order in twins	28	2994.0	3205	-3416.02	5.36	5	.37
3. Equal thresholds across sibling order in siblings	27	2997.4	3206	-3414.57	8.81	6	.18
4. Equal thresholds across sibling order in twins and siblings	22	3002.8	3211	-3419.21	14.17	11	.22
5. Equal thresholds across zygosity (MZ vs. DZ)	20	3005.8	3213	-3420.21	2.99	2	.22
6. Equal thresholds across sibling type (Full- vs. half-siblings)	20	3008.7	3213	-3417.33	5.88	2	.05
7. Equal thresholds across relationship type (MZ/DZ vs. siblings)	16	3012.1	3217	-3421.95	9.26	6	.16
8. Equal thresholds across SS and OS	15	3020.8	3218	-3415.20	18.01	7	.01
9. Equal thresholds across f/m sex in twins	19	3006.4	3214	-3421.59	3.62	3	.31
10. Equal thresholds across f/m sex in siblings	19	3011.6	3214	-3416.39	8.82	3	.03
26- to 33-year-old group (n = 1,438 pairs)							
1. Saturated model	33	2495.6	2843	-3190.43	–	–	–
2. Equal thresholds across sibling order in twins	28	2503.7	2848	-3192.34	8.09	5	.15
3. Equal thresholds across sibling order in siblings	27	2509.7	2849	-3188.30	14.13	6	.03
4. Equal thresholds across sibling order in twins and siblings	22	2517.8	2854	-3190.21	22.22	11	.02
5. Equal thresholds across zygosity (MZ vs. DZ)	20	2522.8	2856	-3189.24	4.97	2	.08
6. Equal thresholds across sibling type (Full- vs. half-siblings)	20	2520.9	2856	-3191.05	3.15	2	.21
7. Equal thresholds across relationship type (MZ/DZ vs. siblings)	16	2526.9	2860	-3193.08	9.13	6	.17
8. Equal thresholds across SS & OS	15	2534.9	2861	-3187.08	17.12	7	.02
9. Equal thresholds across f/m sex in twins	19	2524.3	2857	-3189.73	6.48	3	.09
10. Equal thresholds across f/m sex in siblings	19	2529.2	2857	-3184.80	11.41	3	.01

estimates (a^2, c^2, t^2, and e^2) to be equal for males and females did not deteriorate the fit of the models in any age groups (Model 6).

Lastly, the individual effect of each parameter estimate on explaining variance in cigarette use initiation was tested in Models 7 through 10. Removing the influence of the shared environment on cigarette use initiation in the 12- to 17-year-olds (Model 8) resulted in a reduction in the fit of the model ($\chi^2 = 20.98$, $p < .0001$), as did removing the influence of both the shared and special twin environments simultaneously (Model 10: $\chi^2 = 21.02$, $p < .0001$). Removing the additive genetic influence from the models resulted in worse model fit for the 18- to 25-year-olds (Model 10: $\chi^2 = 11.23$, $p < .0001$) and the 26- to 33-year-olds (Model 10: $\chi^2 = 9.75$, $p < .0001$, respectively). Removing the shared environment or the special twin environment did not deteriorate the fit of the models in the three age groups.

Variance Components

For each age group, we derived variance components from the best fitting model that included all four variance components. We present the results in Table 5 and discuss them below separately for each age group.

12- to 17-year-olds. In the case of the youngest age group, the best fitting model indicated that additive genetic factors accounted for 11.5% (95% CI [0%, 47%]) of the variance in cigarette use initiation. In this group, the shared environment accounted for the most variance in cigarette use initiation, 44.4% (95% CI [24%, 59%]), and the special twin environment accounted for 6.8% (95% CI [0%, 23%]) of the variance. Unique environmental influences accounted for 37.2% (95% CI [25%, 51%]) of the variance in 12- to 17-year-olds.

18- to 25-year-olds. The derived variance component for the 18- to 25-year-olds came from a model in which parameter estimates were allowed to be equal across sex. Additive genetic factors accounted for 68.8% (95% CI [31%, 85%]) of the variance in cigarette use initiation among 18- to 25-year-olds, and the shared environment accounted for 6.4% (95% CI [0%, 28%]) of the variance. The special twin environment did not account for any (0%; 95% CI [0%, 16%]) variance in this age group, while the unique environment accounted for 24.8% (95% CI [14%, 41%]) of the variance in cigarette use initiation.

26- to 33-year-olds. Estimates of the variance components derived from the best fitting model in the 26- to 33-year-old age group indicated that additive genetic factors accounted for 65.6% (95% CI [25%, 91%]) of the variance, while shared environmental factors accounted for 11.6%

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TABLE 4

ACTE Univariate Models of Cigarette use Initiation by Age Group

	Estimated parameters	–2LL	df	AIC	ΔLL	Δ*df*	*p* value
12- to 17-year-old group (n = 1,539 pairs)							
1.Moderated ACTE model w/r_g and r_c	27	4038.73	3051	–2063.27	–	–	–
2. ACTE w/o moderated paths	19	4049.93	3059	–2068.07	5.89	8	.660
3. ACTE w/o moderated paths or thresholds	18	4058.27	3060	–2061.73	8.34	1	.004
4. Test qualitative sex diff. in ACTE	18	4049.93	3060	–2070.07	0.00	1	1.000
5. Constrain r_c and r_g	17	4054.04	3061	–2067.96	4.10	1	.040
6. Testing quantitative sex diff. in ACTE	**14**	**4052.72**	**3064**	**–2075.28**	**2.79**	**4**	**.590**
7. CTE model	13	4053.09	3065	–2076.91	0.38	1	.540
8. ATE model	13	4073.70	3065	–2056.30	20.98	1	.000
9. ACE model	13	4053.36	3065	–2076.64	0.64	1	.420
10. AE model	12	4073.74	3066	–2058.26	21.02	2	.000
18- to 25-year-olds (n = 1,629 pairs)							
1.Moderated ACTE model w/r_g and r_c	27	2998.29	3202	–3405.71	–	–	–
2. ACTE w/o moderated paths	19	3005.52	3210	–3414.48	7.23	8	.512
3. ACTE w/o moderated paths or thresholds	18	3010.61	3211	–3411.39	5.09	1	.024
4. Test qualitative sex diff. in ACTE	18	3005.52	3211	–3416.48	0.00	1	1.000
5. Constrain r_c and r_g	17	3005.52	3212	–3418.48	0.00	1	1.000
6. Testing quantitative sex diff. in ACTE	**13**	**3007.67**	**3216**	**-3424.33**	**2.15**	**4**	**.710**
7. CTE model	12	3018.9	3217	–3415.10	11.23	1	.000
8. ATE model	12	3008.02	3217	–3425.98	0.34	1	.560
9. ACE model	12	3007.67	3217	–3426.33	0.00	1	1.000
10. AE model	11	3008.02	3218	–3427.98	0.34	2	.840
26- to 33-year-olds (n = 1,440 pairs)							
1.Moderated ACTE model w/r_g & r_c	27	2524.20	2849	–3173.80	–	–	–
2. ACTE w/o moderated paths	19	2524.49	2857	–3189.51	0.30	8	1.000
3. ACTE w/o moderated paths or thresholds	18	2526.61	2858	–3189.39	2.11	1	.146
4. Test qualitative sex diff. in ACTE	17	2526.61	2859	–3191.39	0.00	1	1.000
5. Constrain r_c and r_g	16	2526.86	2860	–3193.14	0.26	1	.610
6. Testing quantitative sex diff. in ACTE	**12**	**2528.94**	**2864**	**-3199.06**	**2.34**	**5**	**.800**
7. CTE model	11	2538.69	2865	–3191.31	9.75	1	.000
8. ATE model	11	2529.96	2865	–3200.04	1.02	1	.310
9. ACE model	11	2529.15	2865	–3200.85	0.21	1	.650
10. AE model	10	2529.99	2866	–3202.01	1.05	2	.590

Note: Bold type indicates best-fitting model that included all four variance components.

TABLE 5

Variance Components for Cigarette use Initiation in ACTE Models by Age Group

	Additive genetic		Shared environment		Twin environment		Unique environment	
12- to 17-year-olds	11.5%	[0%, 47%]	44.4%	[24%, 59%]	6.8%	[0%, 23%]	37.2%	[25%, 51%]
18- to 25-year-olds	68.8%	[31%, 85%]	6.4%	[0%, 28%]	0.0%	[0%, 16%]	24.8%	[14%, 41%]
26- to 33-year-olds	65.6%	[25%, 91%]	11.6%	[0%, 34%]	5.0%	[0%, 25%]	17.7%	[9%, 32%]

(95% CI [0%, 34%]). The special twin environment accounted for 5% (95% CI [0%, 25%]) of the variance in cigarette use initiation. The unique environment accounted for 17.7% (95% CI [9%, 32%]) of the cigarette use initiation variance.

Discussion

In an effort to understand the role of the special twin environment that is shared between twin siblings on initiating cigarette use, the present study tested the contribution of additive genetic, shared, and special twin environmental factors by examining similarities in cigarette use initiation among MZ, DZ, full-, and half-sibling pairs. In our sample, half-siblings in each of the three age groups reported higher rates of cigarette use initiation than other pairs of individuals. The higher rate of cigarette use among pairs of half-siblings is consistent with previous work, indicating that half-siblings in blended families have higher rates of externalizing problems, including substance use (Kendler et al., 2012) and criminal behavior (Kendler et al., 2015). Studies of the emotional wellbeing and adjustment of children and adolescents who experience changes in family structure reveals that externalizing problems might be due to receiving lower levels of parental emotional availability, experiencing multiple family transitions in family structure, or living in home environments characterized by parental discord (Marcynyszyn et al., 2008; Soloski & Berryhill, 2016). Such family stressors extend to initiating substances use in adolescence as well (Gutman et al., 2011).

Our results regarding how additive genetic and shared environment factors contribute to the initiation of cigarette

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use is consistent with previous work with this population. Although 11.5% of the variance in cigarette use initiation is due to additive genetic factors, the confidence intervals included zero, suggesting a non-significant effect. In the youngest group, about half of the variance in cigarette initiation was accounted for by the shared environment. The finding that shared environmental factors contribute more to the variance in cigarette use initiation than additive genetic factors in adolescence has been previously reported in studies using various adolescent twin samples (Bares et al., 2015; Do et al., 2015; Maes et al., 2016; Unger et al., 2011). In addition, the shared environment has been found to decrease while the influence of additive genetic factors increases as individuals move from adolescence into adulthood (Bares et al., 2015; Öncel et al., 2014). In fact, while the shared environment explains slightly less than half of the variance in cigarette use initiation in adolescence, this decreases in the 18- to 25-year-olds and 26- to 33-year-olds. We suspect that the decrease observed in the contribution of the shared environment comes about because as individuals progress past the initiation of stage of cigarette use, genes involved in metabolizing nicotine begin to shape and influence future cigarette use behaviors and therefore play a more critical role. We found that more than 60% of the variance in cigarette use initiation was explained by additive genetic factors in the two oldest age groups, in line with previous reports (Sullivan & Kendler, 1999).

Because we conceptualized that the special twin environment as part of the shared environment that twins experience within a family, we had expected that the effect of being a twin would decrease as individuals aged into young adulthood and beyond. Our findings did not support our predictions that the effect of the special twin environment would be stronger during adolescence. In fact, the special twin environment did not contribute significantly to the variance in cigarette initiation in any age group. Without a direct measure of the length of cohabitation between half-siblings, we might be underestimating the effect of the special twin environment for initiating cigarette use. Theories and studies of friendship selection (Kandel, 1978) suggest that individuals are actively involved in creating their own social environment and, as a result, adolescent twins may be choosing to include their co-twin in their social group (Rose, 2002) and, as a unit, may also choose friends who have similar views as themselves (Rushton & Bons, 2005). As a result of including similar others in their social environment, the drug use of one twin may influence the initiation and use of drugs by the co-twin. These social processes may be most relevant during adolescence, a time when individuals are most at risk for the influence of peers and friends to initiate drugs. Adolescent twins may be bringing shared friends who hold similar views on substance use to the social milieu they have created with their co-twin, and together they may all develop more similar behaviors to one another, including drug use. However, with our current sample we could not confidently detect these influences. Future studies of adolescent twins and siblings may need to take into account how much contact twins and siblings have had with one another to make confident conclusions regarding the role that the special twin environment plays.

Our findings, should be considered with the following limitations in mind. While the survey asked participants to list the members of their family with whom they currently lived allowing us to include pairs of siblings who were currently living together, we do not have information about the length of time that pairs of individuals lived together. Despite not having a direct measure of the time that half-siblings had lived together, we accounted for the different rearing environment that half-siblings likely experience by constraining the shared environment for half-siblings to be 50%. A second limitation of our study has to do with the small sample size in each of the sibling groups; in particular, the half-siblings part of the Add Health study that could have contributed to the wide confidence intervals. Lastly, although previous work suggests that there exist racial and/or ethnic differences in the degree to which genetic and shared environmental factors contribute to cigarette use (Bares et al., 2016), the small number of African–American twins and siblings available in the sample prevented us from testing both the influence of t^2 and racial/ethnic differences simultaneously.

Notwithstanding these limitations, we have observed that shared environmental influences that are not specific to being a twin explain a large proportion of the cigarette initiation variance in adolescence. Additive genetic effects appear after age 18 and remain strong into adulthood. Continuing to unpack aspects of the shared environment that influence cigarette use initiation is useful when we consider just how important familial risk factors and familial experiences are in initiation of cigarettes and other substances in adolescence.

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Conflict of Interest

None.

Ethical Standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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