Genetic and Environmental Influences on Adolescent Emotional Inertia in Daily Life

Blinded for peer review

**Abstract**

Emotional inertia represents the extent to which individuals’ emotions tend to carry over from one time point to the next. High emotional inertia indicates low emotion regulation ability and has been associated with psychological maladjustment and mood disorders. However, the extent of genetic influence on emotional inertia, particularly in adolescents, is largely unknown. The current study examined genetic and environmental influences on individual differences in emotional inertia. This study followed a sample of 447 17-year-old same-sex UK twins (41% males) with an innovative intensive longitudinal daily diary design that captured their intra-individual emotion fluctuations over one month. Adolescents reported their positive and negative emotions once a day consecutively for up to 40 days. Time series analyses were used to construct emotional inertia and classical twin analyses were used to disentangle its genetic and environmental influences. The results showed that inertia for positive emotion was only modestly heritable and inertia for negative emotion showed no heritability at all. Both measures showed predominantly non-shared environmental influences. These findings highlight the importance of unique environmental influences in shaping individual differences in how well adolescents regulate their emotions and how easily they move from one emotional state to another in daily life. The importance of identifying specific environmental influences on emotional inertia is discussed, and suggestions of what those influences might be are offered.

*Keywords*: emotional inertia; heritability; non-shared environmental influences; intensive longitudinal data; twin study

**Introduction**

Emotion regulation has a potent impact on multiple domains over the lifespan for adaptive development, especially during adolescence (Cole, Martin, & Dennis, 2004; Hollenstein & Lanteigne, 2018). Extant twin research on emotion regulation exclusively focused on early childhood and provided inconclusive evidence of genetic contributions to emotion regulation (Soussignan et al., 2009; Wang & Saudino, 2013). Intensive longitudinal data enable the researchers to measure emotional inertia–a novel construct closely related with, yet distinct from emotion regulation–that captures the temporal dynamics of emotion processes (Kuppens, Allen, & Sheeber, 2010). Notably, emotional inertia is linked with long-term psychological adjustment and well-being (Houben, van den Noortgate, & Kuppens, 2015). Recent studies have used genetically informative intensive longitudinal data to elucidate genetic contributions to emotion dynamics, and revealed substantial genetic influences on time-independent emotion dynamics such as emotional lability (Zheng, Plomin, & von Stumm, 2016). Nonetheless, currently there is a lack of knowledge of genetic and environmental contributions to temporal dynamics of emotions contained in intra-individual variability. Using a large sample of adolescent twins who provided data on their positive and negative emotions daily over an entire month, the current study examined genetic and environmental contributions to adolescent daily emotional inertia.

**Emotion Regulation and Emotional Inertia**

Emotion regulation refers to the dynamic intrinsic and extrinsic processes encompassing behaviors and strategies that aim to monitor, enhance, and inhibit the experience and expression of emotions in a way that is conducive to achieving one’s goals in particular situations (Cole et al., 2004; Gross & Thompson, 2007). Emotion regulation plays an essential role with pervasive influences in multiple domains for adaptive and normative development over the life span, such as social competence (e.g., Spinrad et al., 2006), academic performance (e.g., Eisenberg, Sadovsky, & Spinrad, 2005), and mental health (e.g., Silk, Steinberg, & Morris, 2003). Emotion regulation is particularly relevant during adolescence (Hollenstein & Lanteigne, 2018), given that adolescents tend to show more emotional reactivity and daily fluctuations (e.g., Larson, Moneta, Richards, & Wilson, 2002); demonstrate different emotion regulation skills to younger children (e.g., Cracco, Goossen, & Braet, 2017); and are more prone to psychopathology (e.g., Lerner & Galambos, 1998).

Emotion and its regulation involve dynamic, intra-individual processes happening continuously in real time that cannot be easily or fully captured by either cross-sectional or longitudinal studies with time intervals of months or years between data collection waves. Over the past decade, there has been noteworthy growth in the number of studies that have used emerging technology to gather intensive longitudinal data on psychological and behavioral phenotypes within a short time interval (Stone, Shiffman, Atienza, & Nebeling, 2007; Trull & Ebner-Priemer, 2014). The collection of intensive longitudinal data, such as daily diary data, offers a unique opportunity to investigate dynamic processes of psychological phenotypes at a nuanced, real-time level in normal daily life contexts, and to capture intra-individual short-term fluctuations that are independent of intra-individual mean changes (e.g., daily emotion fluctuations; Hamaker & Wichers, 2017; Zheng, Molenaar, Arden, Asbury, & Almeida, 2016). Notably, intensive longitudinal data provide a novel way to measure a construct closely related with emotion regulation that is not possible with conventional laboratory observations or questionnaire surveys, namely, emotional inertia.

Emotional inertia describes the resistance to change in an individual’s emotional states (Kuppens et al., 2010; Suls, Green, & Hills, 1998). High emotional inertia suggests that an individual’s emotional state tends to persist from one time to the next, being highly resistant to internal (e.g., emotion regulation efforts) and external (e.g., environmental experiences and changes) influences, whereas low emotional inertia suggests that an individual’s emotional state is particularly malleable and susceptible to change (Kuppens et al., 2010; Suls et al., 1998). An individual who is high in emotional inertia can get stuck in feeling sad or excited due to poor regulation, and struggle to move on even when circumstances and stimuli have changed. An individual who is low in emotional inertia, however, can be cheered up by a friend or a new situation and get out of a negative emotional state. Notably, emotional inertia does not provide information on the mechanisms underlying emotional processes and their changes. High emotional inertia may be impacted by interpersonal processes, and could also reflect maladaptive emotion regulation processes. Therefore, emotional inertia is related with, yet distinct from emotion regulation. High emotional inertia could indicate a less effective regulation of emotional states where adaptive emotional regulation processes are dampened, whereas more effective emotion regulation could change perturbed emotional states more quickly back to an equilibrium point, therefore rendering low emotional inertia.

The construct validity and psychometric reliability of emotional inertia has been well documented (e.g., Wang, Hamaker, & Bergeman, 2012). Particularly, emotional inertia can uniquely and robustly predict long-term outcomes above and beyond other individual characteristics. For instance, high emotional inertia, for both positive and negative emotions, is associated with psychological maladjustment and well-being (Houben et al., 2015), such as depressive disorder in adolescents (Kuppens et al., 2012) and adults (Thompson et al., 2012), above and beyond rumination (Brose, Schmiedek, Koval, & Kuppens, 2015; Koval, Kuppens, Allen, & Sheeber, 2012). For individuals with high positive emotional inertia, it suggests that once they experience positive emotions higher than their typical level (e.g., getting aroused or excited), it takes longer time for them to change and return back to their equilibrium point. While it may be adaptive to feel positive emotions, not being able to regulate one’s emotions efficiently (e.g., if a child cannot calm down after getting excited or happy, but runs around screaming for an extended time period) would be maladaptive. Therefore, individuals with high emotional inertia, even when experiencing positive emotions, tend to show more pervasive emotional states with slower responsiveness and fewer sudden changes, particularly among those characterized by low psychological adjustment (Kuppens et al., 2010).

The dynamic emotion processes can demonstrate inertia at different timescales (e.g., seconds, hours, days). Emotional inertia has been examined through second-to-second behavioral coding of videotaped dyadic interactions (e.g., Kuppens et al., 2010; 2012), hours apart momentary assessments to measure emotions multiple times a day (e.g., Koval et al., 2012; Suls et al., 1998; Thompson et al., 2012), and once a day through daily diary designs (e.g., Brose et al., 2015; Wang et al., 2012). The actual timescale used to assess emotions largely depends on the specific research focus (e.g., day-to-day patterns vs. real-time changes). Notably, emotional inertia from daily diary designs is situated in people’s general daily experiences and therefore captures their general emotions or feelings over the course of a day. In this sense, daily emotional inertia assesses more of a general daily mood as opposed to specific momentary emotions, and hence allows for the assessment of day-to-day change.

**Genetic and Environmental Contributions to Individual Differences in Emotion Regulation**

There are substantial individual differences in emotional inertia, and more broadly in emotion regulation, which can be explained by multiple endogenous and exogenous sources (Calkins, 1994; Calkins & Hill, 2007; John & Gross, 2007). On the one hand, emotion regulation has been associated with biological and physiological processes such as endocrine responses, heart rates (e.g., vagal tone), and brain electrical activity (Calkins, 1994; Calkins & Hill, 2007). Notably, recent studies have identified neural processes (e.g., cerebral blood flow) underlying emotional inertia (Waugh et al., 2017). Given that biological and physiological processes are genetically influenced, and that there are also genetic influences on temperament and related constructs (e.g., effortful control, negative emotionality) linked with emotion regulation (e.g., Gagne & Saudino, 2010; Ganiban, Saudino, Ulbricht, Neiderhiser, & Reiss, 2008), it is plausible that emotional inertia is under genetic influences as well. For instance, individuals genetically predisposed to a difficult temperament may develop emotion dysregulation through common genetic risk or genetically mediated environmental risk, which leads to high emotional inertia.

On the other hand, children and adolescents’ ability to regulate their emotions develops within their families and broader social contexts in the course of multitudinous social interactions. Parents are major contributors to their children’s experiences of socialization, particularly during early development, and consequently play a crucial role in explaining individual differences in emotion regulation (Bariola, Gullone, & Hughes, 2011; Eisenberg, Cumberland, & Spinrad, 1998; Morris, Silk, Steinberg, Myers, & Robinson, 2007). Furthermore, emotional inertia could be affected by external social factors such as interpersonal interactions and environmental changes. Therefore, it is reasonable to expect that emotion regulation and emotional inertia are influenced by both genetic and environmental effects.

The twin study design represents a natural quasi-experiment with the capacity to disentangle genetic effects from otherwise confounded environmental processes, and to distinguish shared environmental effects (those that make family members similar to each other) from non-shared environmental effects (those that create differences between family members brought up together) (Plomin, DeFries, Knopik, & Neiderhiser, 2016; Knopik, Neiderhiser, DeFries, & Plomin, 2016). However, there is relatively little twin research on the genetic influences of emotion regulation (Canli, Ferri, & Duman, 2009; Hariri & Forbes, 2007; Hawn, Overstreet, Stewart, & Amstadter, 2015), especially during childhood and adolescence (but see Goldsmith, Pollak, & Davidson, 2008 for a review of twin studies specifically on effortful/inhibitory control in early childhood).

One early study examined toddlers’ responses to distress simulations (e.g., experimenter pretended to catch her finger in the suitcase; mother pretended to hurt her knee as she got up from the floor) in the lab and at home, when twins were 14 and 20 months old (Zahn-Waxler, Robinson, & Emde, 1992). Toddlers’ personal distress (e.g., whimpering, crying) was the only response among several possibilities (e.g., prosocial acts, empathic concern) that showed no heritability (Zahn-Waxler et al., 1992). A later study among 5-month-old twins examined infants’ responses to presented televised sequences of neutral and happy emotional expressions of their mother and a female stranger in the lab (Soussignan et al., 2009). This study found modest to moderate heritability (19–31%) in infants’ latency and frequency of gaze aversion (an index of emotion regulation) for strangers, but no heritability for the duration of gaze aversion or self-comforting behaviors (Soussignan et al., 2009). A third study among 3-year-old twins assessed emotion regulation with the Behavior Rating Scale over broad domains (i.e., emotional expressivity, attentional skills, goal-related adaptive and motivational behaviors, and social interaction based on the situational demands) and found substantial heritability (43%; Wang & Saudino, 2013). Therefore, evidence of genetic influences on emotion regulation remains inconclusive in early childhood and unknown in adolescence.

**Genetically Informative Intensive Longitudinal Data and Emotion Dynamics**

Twin studies have only recently begun to examine genetic influences on emotions and emotion dynamics using intensive longitudinal data (e.g., Menne-Lothmann et al., 2012; Zheng, Molenaar et al., 2016; Zheng, Plomin et al., 2016). For instance, emotional lability/variability describes the average deviation from the equilibrium point in one’s emotions, and is under substantial genetic and non-shared environmental influences (Menne-Lothmann et al., 2012; Zheng, Plomin et al., 2016). Nonetheless, emotional lability only captures the amplitude of fluctuations but lacks the temporal dependency information in intensive longitudinal data (Wang et al., 2012). No study has used intensive longitudinal data to examine genetic influences on emotional inertia, capturing temporal dynamics of emotion contained in intra-individual variability. Given the importance of emotion regulation and emotional inertia in adaptive and normative development, especially during adolescence, a better understanding of the genetic and environmental architecture of emotional inertia can enhance the knowledge of their developmental origins and mechanisms, and facilitate the identification of adolescents genetically at risk for emotion dysregulation as well as amenable environmental factors to inform interventions to offset genetic risk and to achieve positive developmental outcomes.

**The Current Study**

Previous literature has highlighted the importance of emotion regulation in adaptive adolescent development (e.g., Cracco et al., 2017; Hollenstein & Lanteigne, 2018), as well as its potential genetic and environmental contributions (e.g., Wang & Saudino, 2013). Research using intensive longitudinal data has also demonstrated the importance of emotional inertia, as well as its relevance with and distinction from emotion regulation (e.g., Kuppens et al., 2010). Particularly, recent research using genetically informative intensive longitudinal data has revealed substantial genetic contributions to some emotion dynamics such as emotional lability (e.g., Zheng, Plomin et al., 2016). Drawing on the literature of twin research on emotion regulation and emotion dynamics research using intensive longitudinal data, the current study aimed to further extend prior studies (e.g., Menne-Lothmann et al., 2012; Zheng, Molenaar et al., 2016; Zheng, Plomin et al., 2016) to integrate these two research fields to investigate genetic influences on emotion dynamics through intensive longitudinal data. Specifically, the current study aimed to elucidate genetic and environmental influences on day-to-day emotional inertia using a large sample of adolescent twins who provided intensive longitudinal data using a daily diary design on their positive (e.g., active, inspired) and negative (e.g., upset, nervous) emotions over an entire month. It was hypothesized that adolescent daily emotional inertia would be under both moderate to substantial genetic and environmental influences.

**Methods**

**Participants and Procedures**

Participants from the current study were drawn from the Twins Early Development Study (TEDS), a longitudinal study of twins born in England and Wales in 1994, 1995 and 1996. Twin births between January 1994 and December 1996 were identified through UK birth records. Over 13,000 families were reached at the point of first contact when the twins were about 18 months old (91.7% White, 35.5% A-levels or higher education, 43.1% mother employed, 91.7% father employed, 50.1% female), and this demographic information was comparable to the UK population at that time (93% White, 32% A-levels or higher education, 49% mother employed, 89% father employed). The sample has been longitudinally followed up multiple times in childhood and adolescence (over 74% participation rates at age of 16 years) and more recently in young adulthood (Haworth, Davis, & Plomin, 2013). Zygosity was assessed using a parental questionnaire shown to be more than 95% accurate compared with direct genetic testing (Price et al., 2000). DNA testing was conducted where results were unclear. The institutional review board at King’s College London approved the procedure. Informed consent was obtained from both parents and twins before data collection. A group of 17-year-old same-sex twins from TEDS was invited to participate in a daily mood study (610 invited families, 51.5% consent rate). The demographics of the participating families (93.0% White, standardized socioeconomic status [SES] = 0.30, *SD* = 0.96) is comparable to the total TEDS sample (91.7% White, SES = 0.00, *SD* = 1.00), but with a slightly lower participation rate for males (43.3% vs. 49.9%).

Interested twins were instructed to log-in to a customized web application every day between 3 p.m. (i.e., after school) and 2 a.m. the following morning to fill out a brief survey about their general mood that day, for 40 consecutive days. Each participant tended to fill out the questionnaire at roughly the same time each day (intra-individual standard deviation: *M* = 1.7 hr, *SD* = 0.6 hr, range = 0.1–3.2 hr) although they were not specifically asked to do so. A total of 275 pairs of twins and three unpaired twins (n = 553, 41.4% males, 93.9% White, standardized SES = 0.31, *SD* = 0.96) submitted daily reports within the designated 1-week time window to start participating in this study, including 121 monozygotic (MZ) twin pairs, 154 dizygotic (DZ) twin pairs, and three unpaired DZ twins. On average, each twin provided 33.7 days of reports (range 1–40, *SD* = 8.8), and 87.2% twins provided 30 days or more worth of valid reports. Each participant was paid £40 as compensation after completion of the diary study.

Participants with major medical problems or severe perinatal problems were excluded from analyses. Participants whose first language was not English were also excluded from analyses. The reliability of intra-individual variability is lower with fewer assessments and approximately 30 days are needed to achieve 80% power to accurately capture intra-individual variability based on simulation studies (Estabrook, Grimm, & Bowles, 2012; Wang & Grimm, 2012). Hence, to more accurately estimate emotional inertia, only data from twins with 30 or more days were used in the analyses, leaving a total of 447 twins (96 MZ and 122 DZ twin pairs, and 11 unpaired twins), with a total of more than 15,000 assessments. There was no difference between those included vs. excluded due to available days of reports with regard to average levels of emotions or demographic variables. Based on simulations following the guidelines in Verhulst (2017), the analyzed sample size has approximately 80% power to detect moderate genetic and non-shared environmental influences (~45–50%).

**Measures**

Daily emotions were measured with a short form of the Positive and Negative Affect Schedule (PANAS; Rush & Scott, 2014; Watson, Clark, & Tellegen, 1988), which has been widely used to assess participants’ emotions with good sensitivity to intra-individual variability in emotions on a daily basis, including a British non-clinical sample representative of the general population (Crawford & Henry, 2004). The 10-item short form PANAS has been validated with satisfactory psychometric properties in multiple countries including the UK (Thompson, 2007). The rating procedure followed the end-of-day daily-diary design. Participants were instructed to consider how they had felt over the course of the whole day, rather than just at the time of their response, and to rate the extent to which they had felt each of 10 emotional states, using a 5-point scale from 1 “very slightly or not at all” to 5 “extremely”. Five items were used to measure positive emotions: active, determined, attentive, inspired, and alert. Five items were used to measure negative emotions: afraid, nervous, upset, hostile, and ashamed. To reduce learning effects, item order was randomly presented each day during the short survey. The conventional internal consistency (Cronbach’s α) ranged from .75 to .84 across days for positive emotions and from .76 to .85 for negative emotions.

The average scores of all items were created for daily positive and negative emotions separately. Time series plots using raw data of two randomly chosen twin pairs (one MZ twin pair and one DZ twin pair) are shown for positive emotion (Figure 1) and negative emotion (Figure 2), respectively. Substantial intra-individual variability over days is demonstrated, especially in positive emotion. Systematic linear trend and weekly cyclic mean trend were removed from each individual time series. Consistent with the literature (Brose et al., 2015; Koval et al., 2012; Kuppens et al., 2010; 2012; Thompson et al., 2012), emotional inertia was constructed as the first-order autocorrelation $ρ\left(1\right)$ (i.e., autocorrelation at lag 1, or one day apart) of each person’s daily emotion ratings over the month, which captures the temporal dependency, for positive and negative emotions separately. It indicates the degree to which current emotional state correlates with the previous emotional state. Let $y\_{i,t}$ represents the observed value of person *i* at occasion *t*, while $T\_{i}$ denotes the number of measurement occasions for person *i*, emotional inertia can be estimated using Equation 1.

$\hat{ρ}\_{i}\left(1\right)$ = $\frac{\hat{γ}\_{i}\left(1\right)}{\hat{γ}\_{i}\left(0\right)}$ (1)

where $\hat{γ}\_{i}\left(0\right)$ represents intra-individual variance as calculated in Equation 2, while $\hat{γ}\_{i}\left(1\right)$ represents the auto-covariance at lag 1 defined as the covariance between pairs of observations $y\_{i,t}$ and $y\_{i,t+1}$ for person *i*. The $y\_{i.}$ represents intra-individual mean as calculated in Equation 3. The lag 1 auto-covariance $\hat{γ}\_{i}\left(1\right)$ is computed as in Equation 4 (Wang et al., 2012). Both positive and negative emotional inertia were normally distributed, therefore no transformation was made in following twin analyses.

$\hat{γ}\_{i}\left(0\right)$ = $\frac{\sum\_{t=1}^{T\_{i}}\left(y\_{i,t} - y\_{i.}\right)^{2}}{T\_{i}-1}$ (2)

$y\_{i.}$ = $\frac{\sum\_{t=1}^{T\_{i}}y\_{i,t}}{T\_{i}}$ (3)

$\hat{γ}\_{i}\left(1\right)$ = $\frac{1}{T\_{i}-2}\sum\_{t=1}^{T\_{i}-1}(y\_{i,t}-y\_{i.})(y\_{i,t+1}-y\_{i.})$ (4)

**Twin Analyses**

Standard twin model fitting was used to examine genetic and environmental influences on emotional inertia for positive and negative emotions. Twin analyses make use of the genetic difference between MZ twins, who share all of their genes, and DZ twins, who share, on average, half of their segregating alleles. In the standard Cholesky decomposition model, the phenotypic variance is decomposed into three independent components: additive genetic influences (A), shared environmental influences (C), and non-shared environmental influences as well as measurement error (E). The correlation between twins for A is 1.0 for MZ twins and 0.5 for DZ twins, reflecting their genetic resemblance. The correlation for C is 1.0 for both MZ and DZ twins. Non-shared environmental influences are not correlated between twins because by definition they make twins different from each other (Knopik et al., 2016).

Genetic and environmental influences can be roughly suggested by intra-class correlations. Higher intra-class correlations in MZ twins than in DZ twins would suggest A, which can be inferred by doubling the difference between MZ twin correlation and DZ twin correlation. The remaining familial resemblance not explained by A would suggest C, which can be estimated by subtracting estimated A from the MZ twin correlation. Non-significantly different correlations between MZ and DZ twins would indicate only shared environmental influences but no genetic influences. The extent to which the MZ twin correlation is less than 1.0 would suggest E. To the extent that the MZ twin correlation is more than double that of DZ twins, non-additive (dominance) genetic influences (D) are suggested, which models the interactions of alleles at the same locus or on different loci (epistasis). The correlation between twins for D is 1.0 for MZ twins and 0.25 for DZ twins (Knopik et al., 2016). Where D are suggested by intra-class correlations, an ADE model was also fit. A full ADCE model cannot be fit in classical twin studies because there is not enough information (i.e., degrees of freedom) to estimate all parameters simultaneously.

Because the sample size of this study does not have enough power to detect sex differences in genetic and environmental influences (males and females might be influenced differently by genes and/or environments), males and females were combined into one group for all genetic analyses. All twin analyses were conducted using the structural equation modeling package OpenMx with raw data maximum likelihood estimation to handle missing data (Boker et al., 2011). Parameter estimates, 95% confidence intervals, and model fit statistics were provided. Model goodness of fit was assessed with minus twice the log likelihood (-2LL). Difference in -2LL between a full model and a nested submodel (reduced model with fewer parameters) was assessed by χ2 tests, with the degrees of freedom equal to the difference in the number of parameters estimated between the full and the reduced model. A non-significant χ2 test suggests the reduced model as a more parsimonious model. Akaike’s Information Criterion (AIC) was also computed. A smaller value of AIC suggests a better fit.

**Results**

**Descriptive Statistics**

Descriptive statistics for emotional inertia for positive and negative emotions are presented in Table 1. The distributions show that individuals generally demonstrated modest emotional inertia for both positive emotions (*M* = 0.14) and negative emotions (*M* = 0.11), suggesting that their emotional states tended to persist to the next day to only a slight degree. However, inter-individual differences in emotional inertia were also observed (*SD*s = 0.21 and 0.22, respectively), suggesting that some individuals are more resistant to influences that may alter their emotional states and that, therefore, their emotional states are more likely to persist to the next day. Both measures were normally distributed (skewness = 0.19 and 0.34, respectively). Males and females demonstrated comparable emotional inertia for both positive (*M*s = 0.12 and 0.16, respectively) and negative emotions (*M*s = 0.09 and 0.11, respectively). There was no significant difference in emotional inertia between males and females. In addition, emotional inertia was comparable for MZ and DZ twins for both positive (*M*s = 0.15 and 0.14, respectively) and negative emotions (*M*s = 0.11 and 0.11, respectively). There was a modest positive phenotypic correlation between emotional inertia for positive and negative emotions, *r* = 0.15 (95% CI: 0.06–0.24), suggesting that those individuals whose positive emotional states tend to persist to the next day also tend to persist in their negative emotional states.

**Univariate Genetic Analyses**

Table 2 presents intra-class correlations for MZ and DZ twins. For positive emotional inertia, the correlation for MZ twins is higher than that for DZ twins, which suggest genetic influences. For negative emotional inertia, however, the correlation for MZ twins is lower, but not significantly so (overlapping 95% CIs), than that for DZ twins, indicating no genetic influence but a modest amount of shared environmental influences. Consistent with the patterns showed by intra-class correlations, the results of ACE models (see Table 3) showed that emotional inertia for positive emotion was only modestly heritable (9%, 95% CI: 0–0.25), and that heritability for emotional inertia for negative emotion was negligible (0%, 95% CI: 0–0.22). Individual differences in emotional inertia were largely explained by non-shared environmental influences: 91% for positive emotional inertia (95% CI: 0.75–1.00) and 91% for negative emotional inertia (95% CI: 0.78–1.00). In addition, shared environmental influences also explained a modest portion of individual differences in emotional inertia for negative emotion (9%, 95% CI: 0–0.22).

The most parsimonious models were chosen by comparing reduced models. The results of the most parsimonious models (see Table 3) showed that emotional inertia for positive emotion was under modest additive genetic influences (9%, 95% CI: 0–0.25). Emotional inertia for negative emotion, however, showed no genetic influences but modest shared environmental influences (9%, 95% CI: 0–0.22). Both measures showed substantial non-shared environmental influences: 91% for positive emotional inertia (95% CI: 0.75–1.00) and 91% for negative emotional inertia (95% CI: 0.78–1.00).

**Discussion**

Emotion regulation impacts multiple domains over the lifespan for adaptive development. Adolescents have been found to demonstrate more emotional fluctuation, to develop new regulation strategies, and to be at heightened risk for psychopathology, making emotion regulation particularly relevant for them (Cracco et al., 2017; Larson et al., 2002; Lerner & Galambos, 1998). Emotional inertia, constructed through intensive longitudinal data, captures the temporal dynamics of emotion processes, could indicate regulation processes, and is linked with long-term psychological adjustment and well-being (Houben et al., 2015; Kuppens et al., 2010). Nonetheless, there is a lack of understanding of the relative genetic and environmental contributions to emotional inertia. This study investigated genetic and environmental influences on adolescent daily emotional inertia in a sample of adolescent twins who provided data on their positive and negative emotions daily over a month. The results found that individual differences in emotional inertia for both positive and negative emotions were predominantly explained by non-shared environmental influences, whereas the genetic influences were negligible.

**(The Lack of) Genetic Influences on Emotional Inertia**

Previous non-twin studies have demonstrated the association between multiple biological and physiological processes (e.g., endocrine responses, heart rates) and individual differences in emotion regulation (Calkins, 1994; Calkins & Hill, 2007). Notably, a prior study specifically on emotional inertia has linked it with neural processes (e.g., cerebral blood flow; Waugh et al., 2017). Given that these biological and physiological processes are genetically influenced, as well as the evidence of genetic contributions to temperament and related constructs (e.g., effortful control, negative emotionality) linked with emotion regulation (e.g., Gagne & Saudino, 2010; Ganiban et al., 2008), it was interesting and surprising that no genetic influence at all was found for negative emotional inertia and only a modest degree of genetic influence on positive emotional inertia. This finding stands in sharp contrast to the common behavioral genetic finding, and consensus, that all human behavior is heritable to some extent, typically moderate (Plomin et al., 2016). Nevertheless, the findings are consistent with two previous studies that used different measures to index emotion regulation (the focus was not on emotional inertia) in laboratory observations and in different developmental periods (i.e., infancy and toddlerhood). Specifically, there was no genetic influence on personal distress (Zahn-Waxler et al., 1992) or duration of gaze aversion and self-comforting behaviors (Soussignan et al., 2009), and modest to moderate heritability for the latency and frequency of gaze aversion (Soussignan et al., 2009).

The findings stand in contrast to a third study reporting substantial heritability for emotion regulation among 3-year-old twins (Wang & Saudino, 2013). The difference may possibly be due to the use of different constructs and measurements. Specifically, Wang and Saudino (2013) measured broader domains of emotion regulation skills with a scale, and did not measure elicited emotion regulation (i.e., reactions) or specific emotional states (e.g., upset, inspired) as in Zahn-Waxler et al. (1992) or Soussignan et al. (2009).

It is important to note that the current findings of negligible genetic influence on emotional inertia do not indicate that there is also negligible genetic influence on emotion regulation. Extant twin studies of emotion regulation, albeit scarce and primarily focused on adult populations (e.g., Coccaro, Ong, Seroczynski, & Bergeman, 2012; Hawn et al., 2015; McRae et al., 2017), and on related constructs (e.g., effortful/inhibitory control, Goldsmith et al., 2008), have found moderate genetic influences (30–50%). Despite the clear relevance, emotional inertia is distinct from emotion regulation. Emotional inertia could be due to internal factors such as emotion regulation skills and efforts, as well as external factors such as environmental changes and interpersonal experiences. The current findings suggest that adolescents’ daily emotional inertia might be more impacted by their daily environmental and interpersonal experiences than their own regulation skills.

**Non-shared Environmental Influences on Emotional Inertia**

The current findings highlight the importance of environmental influences on emotional inertia and may be interpreted as emphasizing the social mechanisms involved in the development of emotional inertia. Parent-child relationships and attachment are known to have major influences on the development of children and adolescents’ emotion regulation skills (Bariola et al., 2011; Cassidy, 1994; Morris et al., 2007). For instance, parent-child attachment security is associated with better emotion regulation skills (Brumariu, 2015; Cassidy, 1994; Zimmer-Gembeck et al., 2017). Parents’ caregiving behaviors possibly influence the development of emotion regulation skills through an emerging attachment relationship. Experience of consistent, responsive, and sensitive caregiving could provide a sense of security and continuing source of support for a child or adolescent; thereby influencing their adaptation to developmental challenges. Therefore, children and adolescents in secure parent-child relationships may develop better and more adaptive emotion regulation skills (Brumariu, 2015; Cassidy, 1994; Zimmer-Gembeck et al., 2017). A similar pattern possibly may also exist for emotional inertia, although this needs to be tested in future research.

It is interesting to note that the salient environmental influences on emotional inertia were individual-specific, that is, they were not factors that had the same effect on all children growing up within a family. This finding is largely consistent with the previous twin studies on early childhood showing either no (Soussignan et al., 2009) or modest shared environmental influences (5–9%; Wang & Saudino, 2013; Zahn-Waxler et al., 1992) on emotion regulation. Twin studies with adult samples have also found substantial non-shared environmental influences on emotion regulation (e.g., Coccaro et al., 2012; Hawn et al., 2015; McRae et al., 2017). Current literature provides multiple potential sources of socialization of emotion regulation, both within and outside of the family context (Bariola et al., 2011; Eisenberg et al., 1998; Morris et al., 2007). The findings suggest that between-family environments that affect siblings growing up in the same family in the same way play relatively a modest role in the development of negative emotional inertia. Within-family environments that are experienced uniquely and differently by family members are likely to play a major and crucial role in the development of emotional inertia. Some potential sources of such within-family environments may include differential emotion socialization by parents and differential parenting behaviors (Burt, McGue, Iacono, & Krueger, 2006; Hollenstein & Lanteigne, 2018; Morris et al., 2007). Taken together, the findings suggest that the processes involved in the development of emotional inertia are fundamentally interactive and largely depend on adolescents’ unique social interaction experiences and social contexts, although other non-social factors may still play a role.

**Unique Features of Intensive Longitudinal Data**

Interpretations of the current findings should also take into account the unique features of intensive longitudinal data. Repeatedly measuring phenotypes within a much shorter time interval than is typical in a conventional longitudinal study offers a unique opportunity to examine intra-individual variability that typically involves short-term fluctuations (Hamaker & Wichers, 2017; Zheng, Molenaar et al., 2016). Setting at such a micro timescale, emotions, as many other physiological and psychological processes, are highly dynamic and transient in that they change across different momentary emotional states or expressions under the confluences of both endogenous and exogenous influences. The highly reactive nature of human emotions determines that individual differences in emotion processes are likely to be idiosyncratic, situation-specific, and contingent upon individuals’ unique experience, especially as assessed in real time and daily life contexts (Zheng, Molenaar et al., 2016). It is therefore not surprising that non-shared environmental influences predominantly explain emotional inertia.

Nevertheless, twin studies investigating genetic and environmental architecture of phenotypes at a more refined microscopic level are only in their infancy. Existing twin studies using intensive longitudinal data of emotions have only examined time-independent constructs that do not make use of the temporal sequential nature of such data (e.g., emotional lability; Menne-Lothmann et al., 2012; Zheng, Plomin et al., 2016), whereas the current study represents the first effort, to our knowledge, to examine a time-dependent construct (i.e., emotional inertia). Disentangling genetic and environmental influences, as well as their interplay, using genetically informative intensive longitudinal data offers a novel opportunity to examine emotions and other phenotypes as dynamic processes at a new and exciting level of analysis.

**Limitations and Future Directions**

The current findings should be considered in the context of several limitations. First, it is important to emphasize that this study specifically examined emotional inertia, rather than emotion regulation *per se*, which would also involve its cognitive processes (e.g., attention, appraisal) and coping strategies. Recent twin studies have examined these cognitive processes (cognitive reappraisal and expressive suppression) in adult samples and found significant genetic influences (20% and 35%, respectively; McRae et al., 2017). Second, this study did not specifically look into the potential sources of social influences on emotional inertia (e.g., parent-child relationship). Unfortunately, measures of this type were not available for the sample in the current study. Future studies should consider including these measures and using multivariate genetic analysis to test genetically and environmentally mediated mechanisms linking emotional inertia with these socialization experiences. Given the current findings, it is likely that these associations are largely mediated environmentally. Future studies should include specific measures of daily events to examine how individuals regulate their emotions in the face of particular daily experiences and interactions, as positive and negative daily events (acute or chronic) possibly impact individuals’ emotions and their changes. For instance, daily hassles may perpetuate the feeling of negative emotions, hinder the return of emotional states to the equilibrium point, and lead to high emotional inertia. Third, only the short form of the PANAS was used to measure emotions. Future studies should consider using the full scale to capture a broader range of emotional states.

Fourth, there might be measurement errors for estimated emotional inertia, which could inflate non-shared environmental effects. However, the measure of positive and negative emotions, from which emotional inertia was estimated, has sound psychometric properties. Only participants with 30 or more days of observations were retained for analyses, hence providing more accurate assessment of intra-individual variability and consequently more accurate estimates of emotional inertia. Therefore, the modest genetic influences and predominant non-shared environmental influences cannot be explained solely by sample size or measurement issues. Nevertheless, the sample size of this study is admittedly smaller compared to other conventional twin studies. However, it is important to note that the current study is the largest of all extant twin studies of similar design with regard to both its sample size and time length.

Lastly, the current study adopted a daily diary design to specifically examine adolescents’ day-to-day emotion fluctuations. Previous studies on emotional inertia have adopted a momentary assessment approach to measure emotions multiple times a day, but over fewer days (e.g., Koval et al., 2012; Suls et al., 1998; Thompson et al., 2012), or even second-to-second behavioral coding of videotaped dyadic interactions (e.g., Kuppens et al., 2010; Kuppens et al., 2012). The actual timescale chosen to assess emotions largely depends on specific research questions (e.g., day-to-day patterns vs. real-time changes). Momentary assessments can provide more refined information between hours, but typically at the expense of between-day information because most momentary assessment studies only last one to two weeks. Another major benefit of a daily diary design over multiple assessments within a single day is that participants’ regular daily routine is less likely to be interrupted, especially given that the current participants were still attending school during data collection. Emotional inertia at different timescales may involve different mechanisms. Future studies should consider using different sampling designs to examine emotional inertia at different timescales. Particularly, momentary assessments could provide more information of each specific emotion (e.g., sad vs. anxious), which might reveal more nuanced differences in the associations of their inertia with long-term outcomes (e.g., Koval et a., 2012; Kuppens et al., 2010; 2012). It is likely that the current findings of predominantly non-shared environmental influences and negligible to modest genetic influence on emotional inertia will be replicated and perhaps even more dramatically at shorter timescales (e.g., hours), as future research sets to capture ever more refined and idiosyncratic dynamic processes of individuals’ unique experiences happening in real life.

**Conclusion**

Emotion regulation plays a potent and ubiquitous role in adaptive and normative development, especially during adolescence. Daily emotional inertia indicates how well adolescents regulate their emotions in their daily lives in face of various experiences and events. Higher emotional inertia is associated with more psychological maladjustment and lower well-being. There is currently a lack of knowledge of the genetic and environmental architecture of emotional inertia. This study investigated genetic and environmental contributions to adolescent daily positive and negative emotional inertia with intensive longitudinal data through daily reports over a month. Non-shared environmental influences play a major role in shaping individual differences in adolescent emotional inertia in their daily lives, whereas genetic influences are negligible. The findings highlight that adolescent daily emotional inertia is mainly affected by within-family environments experienced uniquely by adolescents, as well as other person-specific experiences outside of family. Furthermore, the findings suggest that identifying specific risk and adverse environmental factors could provide potential amenable targets for interventions that aim to promote adolescent socioemotional development. Adolescent emotion development happens continuously in real time as situated in adolescents’ daily lives and experiences. Future research should identify specific environmental factors that impact adolescent emotion processes and its regulation, and elucidate genetic contributions to other temporal emotion dynamics through intensive longitudinal data at various timescales.

**References**

Bariola, E., Gullone, E., & Hughes, E. K. (2011). Child and adolescent emotion regulation: The role of parental emotion regulation and expression. *Clinical Child and Family Psychology Review*, *14*, 198–212.

Boker, S., Neale, M., Maes, H., Wilde, M., Spiegel, M., Brick, T., . . . Mehta, P. (2011). OpenMx: An open source extended structural equation modeling framework. *Psychometrika*, *76*, 306–317.

Brose, A., Schmiedek, F., Koval, P., & Kuppens, P. (2015). Emotional inertia contributes to depressive symptoms beyond perseverative thinking. *Cognition and Emotion*, *29*, 527–538.

Brumariu, L. E. (2015). Parent–child attachment and emotion regulation. *New Directions for Child and Adolescent Development*, *2015*, 31–45.

Burt, S. A., McGue, M., Iacono, W. G., & Krueger, R. F. (2006). Differential parent-child relationships and adolescent externalizing symptoms: Cross-lagged analyses within a monozygotic twin differences design. *Developmental Psychology*, *42*, 1289–1298.

Calkins, S. D. (1994). Origins and outcomes of individual differences in emotion regulation. In N. A. Fox (Ed.), Emotion regulation: Behavioral and biological considerations. *Monograph of the Society for Research in Child Development*, *59*, 53–72.

Calkins, S. D., & Hill, A. (2007). Caregiver influences on emerging emotion regulation: Biological and environmental transactions in early development. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 229–248). New York, NY: Guilford Press.

Canli, T., Ferri, J., & Duman, E. A. (2009). Genetics of emotion regulation. *Neuroscience*, *164*, 43–54.

Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. In N. A. Fox (Ed.), Emotion regulation: Behavioral and biological considerations. *Monographs of the Society for Research in Child Development*, *59*, 228–249.

Coccaro, E. F., Ong, A. D., Seroczynski, A. D., & Bergeman, C. S. (2012). Affective intensity and lability: Heritability in adult male twins. *Journal of Affective Disorders*, *136*, 1011–1016.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development*, *75*, 317–333.

Cracco, E., Goossens, L., & Braet, C. (2017). Emotion regulation across childhood and adolescence: Evidence for a maladaptive shift in adolescence. *European Child & Adolescent Psychiatry*, *26*, 909–921.

Crawford, J. R., & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, *43*, 245–265.

Eisenberg, N., Cumberland, A., & Spinrad, T. L. (1998). Parental socialization of emotion. *Psychological Inquiry*, *9*, 241–273.

Eisenberg, N., Sadovsky, A., & Spinrad, T. L. (2005). Associations of emotion‐related regulation with language skills, emotion knowledge, and academic outcomes. *New Directions for Child and Adolescent Development*, *2005*, 109–118.

Estabrook, R., Grimm, K. J., & Bowles, R. P. (2012). A Monte Carlo simulation study of the

reliability of intraindividual variability. *Psychology and Aging*, *27*, 560–576.

Gagne, J. R., & Saudino, K. J. (2010). Wait for it! A twin study of inhibitory control in early childhood. *Behavior Genetics*, *40*, 327–337.

Ganiban, J. M., Saudino, K. J., Ulbricht, J., Neiderhiser, J. M., & Reiss, D. (2008). Stability and change in temperament during adolescence. *Journal of Personality and Social Psychology*, *95*, 222–236.

Goldsmith, H. H., Pollak, S. D., & Davidson, R. J. (2008). Developmental neuroscience perspectives on emotion regulation. *Child Development Perspectives*, *2*, 132–140.

Gross, J. J., & Thompson, R. A. (2007). Emotion regulation: Conceptual foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–27). New York, NY: Guilford Press.

Hamaker, E. L., & Wichers, M. (2017). No time like the present: Discovering the hidden dynamics in intensive longitudinal data. *Current Directions in Psychological Science*, *26*, 10–15.

Hariri, A. R., & Forbes, E. E. (2007). Genetics of emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 110–132). New York, NY: Guilford Press.

Haworth, C. M. A., Davis, O. S. P., & Plomin, R. (2013). Twins Early Development Study (TEDS): A genetically sensitive investigation of cognitive and behavioral development from childhood to young adulthood. *Twin Research and Human Genetics*, *16*, 117–125.

Hawn, S. E., Overstreet, C., Stewart, K. E., & Amstadter, A. B. (2015). Recent advances in the genetics of emotion regulation: A review. *Current Opinion in Psychology*, *3*, 108–116.

Hollenstein, T., & Lanteigne, D. M. (2018). Emotion regulation dynamics in adolescence. In P. M. Cole, & T. Hollenstein (Eds.), *Emotion regulation: A matter of time* (pp. 176–194). New York, NY: Routledge.

Houben, M., van den Noortgate, W., & Kuppens, P. (2015). The relation between short-term emotion dynamics and psychological well-being: A meta-analysis. *Psychological Bulletin*, *141*, 901–930.

John, O. P., & Gross, J. J. (2007). Individual differences in emotion regulation. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 351–372). New York, NY: Guilford Press.

Knopik, V. S., Neiderhiser, J. M., DeFries, J. C., & Plomin, R. (2016). *Behavioral genetics (7th ed.).* New York, NY: Worth.

Koval, P., Kuppens, P., Allen, N. B., & Sheeber, L. (2012). Getting stuck in depression: The roles of rumination and emotional inertia. *Cognition and Emotion*, *26*, 1412–1427.

Kuppens, P., Allen, N. B., & Sheeber, L. B. (2010). Emotional inertia and psychological maladjustment. *Psychological Science*, *21*, 984–991.

Kuppens, P., Sheeber, L. B., Yap, M. B., Whittle, S., Simmons, J. G., & Allen, N. B. (2012). Emotional inertia prospectively predicts the onset of depressive disorder in adolescence. *Emotion*, *12*, 283–289.

Larson, R., Moneta, G., Richards, M. H., & Wilson, S. (2002). Continuity, stability, and change in daily emotional experience across adolescence. *Child Development*, *73*, 1151–1165.

Lerner, R. M., & Galambos, N. L. (1998). Adolescent development: Challenges and opportunities for research, programs, and policies. *Annual Review of Psychology*, *49*, 413–446.

McRae, K., Rhee, S. H., Gatt, J. M., Godinez, D., Williams, L. M., & Gross, J. J. (2017). Genetic and environmental influences on emotion regulation: A twin study of cognitive reappraisal and expressive suppression. *Emotion*, *17*, 772–777.

Menne-Lothmann, C., Jacobs, N., Derom, C., Thiery, E., van Os, J., & Wichers, M. (2012). Genetic and environmental causes of individual differences in daily life positive affect and reward experience and its overlap with stress-sensitivity. *Behavior Genetics*, *42*, 778–786.

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development*, *16*, 361–388.

Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2016). Top 10 replicated findings from behavioral genetics. *Perspectives on Psychological Science*, *11*, 3–23.

Price, T. S., Freeman, B., Craig, I., Petrill, S. A., Ebersole, L., & Plomin, R. (2000). Infant zygosity can be assigned by parental report questionnaire data. *Twin Research*, *3*, 129–133.

Rush, J., & Hofer, S. M. (2014). Differences in within-and between-person factor structure of positive and negative affect: Analysis of two intensive measurement studies using multilevel structural equation modeling. *Psychological Assessment*, *26*, 462–473.

Silk, J. S., Steinberg, L., & Morris, A. S. (2003). Adolescents’ emotion regulation in daily life: Links to depressive symptoms and problem behavior. *Child Development*, *74*, 1869–1880.

Soussignan, R., Boivin, M., Girard, A., Pérusse, D., Liu, X., & Tremblay, R. E. (2009). Genetic and environmental etiology of emotional and social behaviors in 5-month-old infant twins: Influence of the social context. *Infant Behavior and Development*, *32*, 1–9.

Spinrad, T. L., Eisenberg, N., Cumberland, A., Fabes, R. A., Valiente, C., Shepard, S. A., ... Guthrie, I. K. (2006). Relation of emotion-related regulation to children's social competence: A longitudinal study. *Emotion*, *6*, 498–510.

Stone, A. A., Shiffman, S., Atienza, A. A., & Nebeling. L. (2007). *The science of real-time data capture: Self-reports in health research.* New York: Oxford University Press.

Suls, J., Green, P., & Hillis, S. (1998). Emotional reactivity to everyday problems, affective inertia, and neuroticism. *Personality and Social Psychology Bulletin*, *24*, 127–136.

Thompson, E. R. (2007). Development and validation of an internationally reliable short-form of the Positive and Negative Affect Schedule (PANAS). *Journal of Cross-Cultural Psychology*, *38*, 227–242.

Thompson, R. J., Mata, J., Jaeggi, S. M., Buschkuehl, M., Jonides, J., & Gotlib, I. H. (2012). The everyday emotional experience of adults with major depressive disorder: Examining emotional instability, inertia, and reactivity. *Journal of Abnormal Psychology*, *121*, 819–829.

Trull, T. J., & Ebner-Priemer, U. (2014). The role of ambulatory assessment in psychological science. *Current Direction in Psychological Science*, *23*, 466–470.

Verhulst, B. (2017). A power calculator for the classical twin design. *Behavior Genetics*, *47*, 255–261.

Wang, L., & Grimm, K. (2012). Investigating reliabilities of intraindividual variability indicators. *Multivariate Behavioral Research*, *47*, 771–802.

Wang, L. P., Hamaker, E., & Bergeman, C. S. (2012). Investigating inter-individual differences in short-term intra-individual variability. *Psychological Methods*, *17*, 567–581.

Wang, M., & Saudino, K. J. (2013). Genetic and environmental influences on individual differences in emotion regulation and its relation to working memory in toddlerhood. *Emotion*, *13*, 1055–1067.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of a brief measure of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*, 1063–1070.

Waugh, C. E., Shing, E. Z., Avery, B. M., Jung, Y., Whitlow, C. T., & Maldjian, J. A. (2017). Neural predictors of emotional inertia in daily life. *Social Cognitive and Affective Neuroscience*, *12*, 1448–1459.

Zahn-Waxler, C., Robinson, J. L., & Emde, R. N. (1992). The development of empathy in twins. *Developmental Psychology*, *28*, 1038–1047.

Zheng, Y., Molenaar, P. C. M., Arden, R., Asbury, K., & Almeida, D. M. (2016). Person-specific non-shared environmental influences in intra-individual variability: A preliminary case of daily school feelings in monozygotic twins. *Behavior Genetics*, *46*, 705–717.

Zheng, Y., Plomin, R., & von Stumm, S. (2016). Heritability of intraindividual mean and variability of positive and negative affect: Genetic analysis of daily affect ratings over a month. *Psychological Science*, *27*, 1611–1619.

Zimmer-Gembeck, M. J., Webb, H. J., Pepping, C. A., Swan, K., Merlo, O., Skinner, E. A., ... Dunbar, M. (2017). Review: Is parent–child attachment a correlate of children’s emotion regulation and coping? *International Journal of Behavioral Development*, *41*, 74–93.

Table 1

*Descriptive Statistics (M and SD) for Inertia in Positive and Negative Emotion*

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
|  | Overall | Skewness | Range | Male | Female | MZ | DZ |
| Positive Emotion | 0.14 (.21) | 0.10 | -0.37–0.68 | 0.12 (.20) | 0.16 (.22) | 0.15 (.23) | 0.14 (.20) |
| Negative Emotion | 0.11 (.22) | 0.34 | -0.45–0.87 | 0.09 (.23) | 0.11 (.21) | 0.11 (.23) | 0.11 (.21) |

Table 2

*Twin Correlations for Inertia in Positive and Negative Emotion*

|  |  |  |
| --- | --- | --- |
|  | Positive Emotion | Negative Emotion |
| MZ | 0.12 (-0.06, 0.29) | 0.004 (-0.18, 0.19) |
| DZ | -0.01 (-0.21, 0.18) | 0.17 (-0.02, 0.34) |

*Note*. Intra-class twin correlations (95% confidence intervals) for monozygotic (MZ) and dizygotic (DZ) twins.

Table 3

*Univariate Model-fitting Results and Fit Statistics for Inertia in Positive and Negative Emotion*

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Model | -2LnL (df) | AIC | Comparison Model | Δχ2 (Δdf) | *p* | A (95% CI) | C/D (95% CI) | E (95% CI) |
| PEI |  |  |  |  |  |  |  |  |
| ACE | -122.87 (442) | -1006.87 | –– | –– | –– | 0.09 (0–0.25) | 0 (0–0) | 0.91 (0.75–1.00) |
| ADE | -123.12 (442) | -1007.12 | –– | –– | –– | 0 (0–0.25) | 0.11 (0–0.28) | 0.89 (0.72–1.00) |
| AE | -122.87 (443) | -1008.87 | ACE | 0 (1) | 1 | 0.09 (0–0.25) | –– | 0.91 (0.75–1.00) |
| AE | **-122.87 (443)** | **-1008.87** | **ADE** | **0.25 (1)** | **.62** | **0.09 (0–0.25)** | **––** | **0.91 (0.75–1.00)** |
| CE | -122.38 (443) | -1008.38 | ACE | 0.49 (1) | .48 | –– | 0.06 (0–0.19) | 0.94 (0.81–1.00) |
| NEI |  |  |  |  |  |  |  |  |
| ACE | -101.31 (441) | -983.31 | –– | –– | –– | 0 (0–0.22) | 0.09 (0–0.22) | 0.91 (0.78–1.00) |
| ADE | -100.34 (441) | -982.34 | –– | –– | –– | 0.07 (0–0.24) | 0 (0–0.21) | 0.93 (0.76–1.00) |
| AE | -100.34 (442) | -984.34 | ACE | 0.97 (1) | .33 | 0.07 (0–0.24) | –– | 0.93 (0.76–1.00) |
| AE | -100.34 (442) | -984.34 | ADE | 0 (1) | 1 | 0.07 (0–0.24) | –– | 0.93 (0.76–1.00) |
| CE | **-101.31 (442)** | **-985.31** | **ACE** | **0 (1)** | **1** | **––** | **0.09 (0–0.22)** | **0.91 (0.78–1.00)** |

*Note*. PEI = positive emotional inertia; NEI = negative emotional inertia; A = standardized additive genetic influences; D = standardized dominant genetic influences; C = standardized shared environmental influences; E = standardized non-shared environmental influences; 95% CI = 95% confidence interval; -2LnL = -2 log-likelihood; df = degrees of freedom, AIC = Akaike information criterion. The p-values indicate no significant deterioration in model fit between the full and the reduced models. Most parsimonious model bolded.

*Figure 1*. Trend and intra-individual variability of positive emotion of one randomly chosen monozygotic and dizygotic twin pair.

*Figure 2*. Trend and intra-individual variability of negative emotion of one randomly chosen monozygotic and dizygotic twin pair.