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Diurnal preference and sleep quality: Same genes?

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Diurnal preference and sleep quality: same genes?

A study of young adult twins

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### Abstract

The aims of this study were to examine: 1) genetic and environmental influences on diurnal preference and sleep quality; 2) the association between these phenotypes; 3) the genetic and environmental influences on this association; and 4) the magnitude of overlap between these influences. Using a twin design, data on diurnal preference (measured by the 'Morningness-Eveningness Questionnaire') and sleep quality (measured by the 'Pittsburgh Sleep Quality Index') were collected from 420 monozygotic twins, 773 dizygotic twins, and 329 siblings (mode age = 20 yrs, range = 18-27 yrs) from a population-based twin registry across the UK. Univariate analyses indicated that dominance genetic influence accounted for 52% and non-shared environment 48% of variance in diurnal preference. For sleep quality, additive genetic influence explained 43% and non-shared environment 57% of the variance. The bivariate analysis indicated a significant association between greater eveningness preference and poorer sleep quality ( $r = .27$ ). There was substantial overlap in the additive genetic influences on both phenotypes ( $r_A = .57$ ), and overlap in the dominance genetic influences common to both phenotypes was almost absolute ( $r_D = .99$ ). Overlap in non-shared environment was much smaller ( $r_E = .02$ ). Additive genetic influence accounted for 2% of the association; dominance genetic influence accounted for 94%; and non-shared environmental influences accounted for the remaining 4%. The substantial overlap in genetic influence between these phenotypes indicates that similar genes are important for diurnal preference and sleep quality. Therefore, those genes already known to influence one phenotype may be possible candidates to explore with regards to the other phenotype. (Email correspondence: ps701nh@gold.ac.uk)

Keywords: Circadian, diurnal preference, eveningness, genetics, morningness, sleep

## INTRODUCTION

The regulation of sleep-wake behavior is considered to be the product of two processes: the endogenous control of circadian rhythmicity and homeostatic regulation (Borbely, 1982; Borbely & Achermann, 1999; Daan, et al., 1984; Dijk & Lockley, 2002). The endogenous period of the circadian pacemaker is tightly constrained between individuals; however, circadian preferences and the entrainment of sleep-wakefulness to circadian rhythms show greater inter-individual variability (Kerkhof, 1985). Knowledge regarding individual differences in circadian timing may be important for organizing our daily lives, in terms of timing work and social commitments and maintaining sleep hygiene and quality, by ensuring that we sleep at times in accordance with our biological ‘clock’. It is known that differences in the phase position of this biological ‘clock’, and thus the timing of an individual’s preferred sleep-wake cycle, is to some extent under genetic control (Archer, et al., 2003). Furthermore, research has indicated that diurnal preference – the self-report analogue of circadian rhythm phase – is also heritable (Drennan, et al., 1992; Hur, et al., 1998; Koskenvuo, et al., 2007; Vink, et al., 2001). The morningness-eveningness disposition represents extremes in diurnal preference. Morning-types are so-called ‘larks’, who find it easy to arise in the morning, function best at this time, and fall asleep easily during early evening. Evening-types, on the other hand, so-called ‘owls’, find it hard to get up early, are at their peak during late evening, and go to bed late, often in the early hours of the morning. Advanced and Delayed Sleep Phase Disorders (ASPD and DSPD, respectively) represent extremes of morning and evening-type orientations (American Academy of Sleep Medicine, 2005), and are characterized by

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difficulty maintaining socially-normal sleep-wake hours even in the face of adverse social and occupational consequences.

These two chronotypes have received much attention over the last decade, with researchers investigating not only the heritability of the morningness-eveningness disposition but also the molecular genetic basis for its occurrence (Archer, et al., 2003; Archer, et al., 2008; Carpen, et al., 2005; Carpen, et al., 2006; Dijk & Lockley, 2002; Katzenberg, et al., 1998; Lee, et al., 2007) and the personality dimensions correlated with these chronotypes (Larsen, 1985; Randler, 2008b; Tonetti, et al., 2009). For instance, evening-types, compared to other chronotypes, have more irregular lifestyle habits (Monk, et al., 2004), are more prone to substance abuse (Giannotti, et al., 2002), have more psychological and emotional problems (Giannotti, et al., 2002), have lower self-control and elevated levels of procrastination (Digdon & Howell, 2008), and hold dysfunctional beliefs about sleep (Ong, et al., 2007). Some of these difficulties have previously been associated with poor sleep quality (for example, dysfunctional beliefs about sleep: Edinger, et al., 2001; and anxiety and depression: Gregory, et al., submitted; Gregory & O'Connor, 2002; Gregory, Rijdsdijk, Dahl, et al., 2006). Thus, it is possible that since both diurnal preference and sleep quality have similar correlates, there may be more explicit links between them.

Perhaps, more direct evidence for the associations between diurnal preference and sleep quality comes from noting that evening-types suffer from greater daytime sleepiness and dysfunction (Vardar, et al., 2008), experience poorer sleep than morning-types (Megdal & Schernhammer, 2007; Ong, et al., 2007; Shiihara, et al., 1998; Vardar, et al., 2008), and display other forms of sleep deficit, such as irregular sleep/wake habits (Talliard, et al., 1999). Furthermore, a relationship has been reported between the timing of an individual's biological clock and some types of

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chronic insomnia (Lack & Wright, 2007). When sleep is attempted at a time incongruent with one's biological clock, difficulties such as decreased total sleep time, impaired daytime functioning, cognitive impairment, fatigue, difficulty falling asleep, early morning awakenings, and chronic insomnia may arise - which may be seen not only in ASPD and DSPD, but also in conditions such as Shift Work Sleep Disorder (American Academy of Sleep Medicine, 2005). While there appears to be an association between diurnal preference and sleep quality, what is currently unclear is what accounts for this association. Like diurnal preference, sleep quality is influenced by genes (Gregory & Franken, in press), and one possibility is that shared genes are important in accounting for the association between these phenotypes.

Examining the extent to which genetic and environmental influences account for this association may be useful in understanding why differences between chronotypes may be associated with sleep problems. Finding genetic/environmental overlap in the genes/environments influencing diurnal preference and sleep quality would suggest that once we have found genetic/environmental influences associated with one phenotype, the same genes/environments may be worth exploring as to their role in other phenotypes with which it is associated.

Using a sample of 1,556 twin and non-twin siblings, aged between 18 and 27 yrs, the present study aimed to 1) estimate the extent to which genes and environments influence both diurnal preference and sleep quality, separately; 2) examine the phenotypic overlap between diurnal preference and sleep quality; 3) address the extent to which genetic and environmental contributions overlap for diurnal preference and sleep quality; and 4) estimate the magnitude of genetic and environmental influences on the association between diurnal preference and sleep quality.

## **METHODS**

### **Ethical Approval**

Ethical approval for different stages of this study was provided by the Research Ethics Committees of the Institute of Psychiatry, South London and Maudsley NHS Trust, and Goldsmiths, University of London. The experimental protocol conforms to international ethical standards as outlined by Portaluppi et al. (2008).

### **Participants**

The present analyses focus on wave 4 of the G1219 and G1219Twins longitudinal studies. G1219 initially comprised adolescent offspring of adults from a large-scale population-based study (GENESiS: Sham, et al., 2000). The G1219Twins are a random selection of live twin births occurring between 1985 and 1988 as identified by the UK Office of National Statistics. Health Authorities and General Practitioners then contacted families (Eley, et al., 2004). At Wave 1 of data collection (which took place between 1999 and 2002), 3,640 respondents aged between 12 and 19 yrs participated in the study. Informed consent was obtained from parents/guardians of all adolescents <16 yrs, and from the adolescents themselves when >16 yrs. At Wave 2, data were available from 2,646 individuals (73% of the original sample at Wave 1), while corresponding figures for Wave 3 were 1,777 adolescents (49% of the original sample at Wave 1).

At Wave 4 (which took place in 2007 and is the focus of this current report), we traced participants who had taken part in Wave 2/Wave 3 primarily by using websites dedicated to providing information (e.g., phone numbers and postal

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addresses) about members of the population. We successfully traced 2,550 individuals and sent them a questionnaire booklet. Three reminders were then sent (a duplicate questionnaire was sent out with the last reminder in case the former had been misplaced). Participants were also emailed and telephoned in order to determine whether they planned to take part. A total of 1,556 individuals were included in the Wave 4 dataset (61% of those targeted; 74% of those participating in Wave 3).

Zygoty was established through a questionnaire measure completed by mothers at Waves 2 and 3, assessing physical similarity between twins (Cohen, et al., 1975). When zygoty was only available on one or other wave, this rating was used. If there was disagreement between zygoty ratings at the two waves, DNA was obtained (N = 26 pairs) before final classifications were made.

At Wave 4, on which the present study is focused, 61.5% of the sample was female and the mode age was 20 yrs (range 18-27 yrs). Following the study design, the majority of participants were close in age (90% of the participants were aged 18-22 yrs), but the inclusion of siblings inevitably created some age-spread. At Wave 4, the 1,556 individuals came from 896 families: 75 MZ male (65 complete) pairs, 76 DZ male (53 complete) pairs, 155 MZ female (125 complete) pairs, 138 DZ female (111 complete) pairs, 232 DZ opposite sex (163 complete) pairs, 44 male-male sibling (28 complete) pairs, 68 female-female sibling (44 complete) pairs, 89 opposite sex sibling (56 complete) pairs. Sibling type was uncertain for a remaining 19 (15 complete) pairs.

In the whole G1219 sample, levels of parental education were somewhat higher (39% educated to A-level or above) than in a large nationally represented sample of parents (Meltzer, et al., 2000), where 32% were educated to  $\geq$ A-level. G1219 parents were also somewhat more likely to own their own houses (82%) than



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in the nationally representative sample (68%). To reduce the impact of any initial response bias associated with educational level, the sample was re-weighted to match the distribution of educational qualifications in a nationally representative sample of parents (Meltzer et al. 2000). For more detailed information regarding weighting and attrition, please see a previous report from this study by Gregory et al., (Gregory, et al., submitted).

## **MEASURES**

### **Morningness-Eveningness Questionnaire**

The Morningness-Eveningness Questionnaire (MEQ: Horne & Östberg, 1976) is among the most widely used measures for assessing diurnal preference and was adopted for use in this study. The MEQ is a 19-item self-report questionnaire that assesses individual preference in the timing of daytime activities, sleeping habits, hours of peak performance, and times of ‘feeling best’ and maximum alertness. Participants are required to respond mostly by indicating which response out of four best describes them. For example, for the question, “Assuming adequate environmental conditions, how easy do you find getting up in the morning?”, responses range from “Not at all easy”, “Not very easy”, “Fairly easy”, to “Very easy”. Other questions require participants to indicate during which hours they feel, for example, most tired and in need of sleep, or at what time they feel that they reach their peak. Individual items are rated on either a 4- or 5-point scale, and the responses used to give a total score on the morningness-eveningness dimension ranging from 16-86. Higher scores indicate greater ‘morningness’ and lower scores indicate greater ‘eveningness’. However, for the present analyses the total MEQ scale was reversed so that a higher score indicated greater eveningness. This procedure was employed so

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that we could decompose a positive correlation for ease of interpretation for the reader.

In order to determine whether diurnal preference was associated with actual behavior, scores on the MEQ were examined in relation to reported bed and arising times (these measures were taken from the PSQI, described below, and are typically used to calculate sleep duration). There was a significant association between diurnal preference (MEQ total score) and actual bedtimes, ( $r = .50, p < .01$ ), indicating that greater eveningness preference was associated with going to bed later, and that greater morningness preference was associated with going to bed earlier. There was also a significant association between diurnal preference and getting-up time ( $r = .42, p < .01$ ) indicating that greater eveningness was associated with later getting-up time, and greater morningness with earlier getting-up time. We note that actual bedtimes and getting-up times may be influenced by many factors other than diurnal preference, such as school and work obligations (hence, the reason why a perfect correlation was not expected).

### **Pittsburgh Sleep Quality Index**

Sleep disturbance over the past month was assessed using the Pittsburgh Sleep Quality Index (PSQI: Buysse et al. 1989), which is a widely-used questionnaire measure containing 19 items. Items include both open-ended questions (e.g., “During the past month, when have you usually gone to bed at night?”) and fixed-choice questions (e.g., “During the past month, how would you rate your sleep quality overall?” “Very good”, “Fairly good”, “Fairly bad”, or “Very bad”). A global score of overall sleep quality is derived from the sum of individual items, with scores ranging from 0 to 21. Higher scores indicate poorer sleep quality. The PSQI global score has

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demonstrated good psychometric properties, with both internal consistency and test-retest reliability in the .8 range (Backhaus, et al., 2002; Buysse, et al., 1989). The PSQI has also been shown to correspond to other self-report measures of sleep (e.g. Backhaus, et al., 2002).

## **STATISTICAL ANALYSES**

### **Data Preparation**

Skew was not considered problematic for MEQ or PSQI (MEQ skew = -.17, [SE = .09]; PSQI skew = .98, [SE = .09]), and so the variables were not transformed for this purpose. Prior to analyses, data were regressed on age and sex, as is standard in twin modelling (McGue & Bouchard, 1984). Furthermore, outliers of three or more standard deviations above and below the mean were omitted from scales, since extreme scores can significantly influence results (in total, 38 cases were excluded for this reason). All analyses focus on the transformed variables (except for descriptive statistics). Of note, analyses were also re-run on raw (untransformed) data and without excluding outliers, without notable differences in results (unreported).

### **Genetic Analyses**

Analyses were carried out using the statistical package Mx (Neale, 1997), a widely used programme for analyzing genetically sensitive data, using the method of maximum likelihood estimation. Twin studies compare the similarity within monozygotic (MZ) twin pairs to the similarity within dizygotic (DZ) twin pairs to estimate genetic influences on traits. Since MZ twins share 100% of their genes while DZ twins share on average half of their segregating genes, this information can be used to estimate the relative contribution of four sources of variance impacting on a

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phenotype: additive genetic influences (A) (where alleles at a locus ‘add up’ to influence behavior); dominance genetic effects (D) (where genes don not simply “add up”, but the influence of one allele affects the other at a locus to influence behavior); shared environmental influences (C) (those environmental influences that act to make twins similar); and non-shared environmental influences, (E) (those environmental influences acting to make twins within a pair different. This source of variance also incorporates error). Of note, it is not possible to model both dominance genetic effects and shared environmental effects simultaneously. This is because C and D predict different MZ and DZ twin correlation ratios, and the effect of both is confounded if examined together (Neale & Cardon, 1992). Thus, these effects are examined in separate models (i.e., either by an ACE or ADE model) as appropriate. If the correlation between MZ pairs is greater than that of DZ/sibling pairs, the genetic influence may be important for that phenotype. If, however, the MZ twin pair correlation is more than twice that of the DZ twin/sibling pairs, dominance genetic influence may be playing a role.

### **Model Fitting**

The fit statistic provided by Mx for raw data modelling is -2LL (minus twice the log likelihood of the observations). Saturated models, which estimate the maximum number of parameters required to describe the variance-covariance matrix and means of observed variables and thus provide a perfect fit to the data, are first approximated to the data. The -2LL of a saturated model is then subtracted from the -2LL of the genetic model. The -2LL value, in itself, provides no information of fit; however, the difference between -2LL for the saturated and genetic models is  $\chi^2$  distributed, and so provides a relative fit of the data. A non-significant difference in fit

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between the genetic and saturated models indicates that the genetic model does not fit the data less well than a saturated model and therefore provides a good description of the data. An additional measure of fit is provided by Akaike's Information Criterion (AIC) (calculated as  $\Delta\chi^2 - 2 \times \Delta df$ ), which accounts for the number of parameters being estimated and goodness-of-fit. A good fit is indicated by lower, negative values of AIC (Neale, et al., 1989). Likelihood-based 95% confidence intervals (CIs) on the parameter estimates were obtained in order to determine their precision. Sub-models, in which certain parameters (e.g., C) are dropped in order to test their significance, can also be tested and compared against full models. However, full models are reported here for all analyses in order to provide the reader with maximum information.

### **Sex Effects**

Model fitting allows the investigation of various types of sex differences. The present study investigated: 1) quantitative sex differences – the extent to which the magnitude of genetic and environmental influences differed between males and females; 2) qualitative sex differences – the extent to which the genetic and environmental influences affecting males were the same as those affecting females; and 3) scalar sex differences – whether male and female scores differed in variance. Sex differences were also equated in all models in order to determine whether doing so would result in a significant decrement in fit compared to models incorporating sex differences. The best-fitting models (i.e., the most parsimonious and which did not result in a significantly worse fit compared to the saturated model) were selected for interpretation.

### **Univariate Analyses**

Twin correlations suggested dominance genetic effects on diurnal preference (i.e., the MZ twin correlation was more than twice that of DZ twin and sibling correlations). As such, univariate models assessing the relative contribution of A, D, and E to diurnal preference were tested in addition to ACE models. For sleep quality, only the models assessing the relative contribution of A, C, and E were examined, as dominance was not suggested by the twin correlations.

### **Bivariate Correlated Factors Model**

A bivariate correlated factors model, which allows the influences of one phenotype to correlate with the other, was tested. The influence of A, D, C, and E on the phenotypic correlation was estimated by Mx in two separate models – one examining A, C, and E, and the other examining A, D, and E. For an example of an ADE model, see Figure 1. The bivariate additive genetic correlations ( $rA$ ), dominance genetic correlations ( $rD$ ), shared environmental correlations ( $rC$ ), and non-shared environmental correlations ( $rE$ ), demonstrate the extent to which these sources overlap within the phenotypic correlation. The proportions to which A, D, and E accounted for the phenotypic correlation can be calculated, respectively, as  $\sqrt{A}$  for MEQ  $\times rA \times \sqrt{A}$  for PSQI/phenotypic correlation;  $\sqrt{D}$  for MEQ  $\times rD \times \sqrt{D}$  for PSQI/phenotypic correlation; and  $\sqrt{E}$  for MEQ  $\times rE \times \sqrt{E}$  for PSQI/phenotypic correlation.

[Insert Figure I here]

## **RESULTS**

### **Descriptive Statistics**

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Table 1 shows the means and standard deviations of scores on the MEQ and PSQI, split by sex and zygosity. There were significant sex differences in diurnal preference (fit of model incorporating sex differences compared to fully constrained model:  $\Delta\chi^2 = 29.61$ ,  $\Delta df = 2$ ,  $p < .01$ ), with males reporting slightly greater eveningness. As sex differences were important for this phenotype, sex was considered in the genetic analyses presented below. There were no significant sex differences for sleep quality (fit of model incorporating sex differences compared to fully constrained model:  $\Delta\chi^2 = 0.65$ ,  $\Delta df = 2$ ,  $p = .72$ ).

[Insert Table I here]

### Univariate Correlations

Univariate twin correlations for MZ, DZ twins and siblings (e.g., the correlation in MEQ score for twin 1 and twin 2) are presented in Table 2. For MEQ, MZ correlations were more than twice that of both DZ twins and siblings, suggesting dominance genetic effects on this phenotype. As such, dominance effects with regards to this phenotype were explored. Sibling correlations were greater than DZ twin pairs, but confidence intervals on the phenotypic correlations for both DZ twin pairs and siblings overlapped; and so were not significantly different.

For PSQI, MZ twin correlations were greater than DZ and sibling correlations, suggesting additive genetic influence on sleep quality. For both diurnal preference and sleep quality, MZ correlations were less than unity, suggesting that non-shared environmental factors may be important.

[Insert Table II here]

### Univariate Genetic Models

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None of the genetic models fit the data significantly worse than saturated models: they provide a good fit to the data. For diurnal preference, an ADE model, which allowed for scalar sex differences, provided the best fit to the data (ADE scalar sex difference model fit compared to saturated model:  $\Delta\chi^2 = 17.31$ ,  $\Delta df = 20$ ,  $p = .63$ ,  $AIC = -22.69$ ). Male variance was 9% greater than the female variance. Additive genetic influence on this phenotype was estimated at 0% (95% Confidence Intervals [CI], .00-.29); dominance genetic effects at 52% (95% CI, .20-.61); and non-shared environmental influences at 48% (95% CI, .39-.59).

For sleep quality, the best-fitting model was an ACE model that equated sex differences (ACE fit compared to saturated model:  $\Delta\chi^2 = 16.64$ ,  $\Delta df = 21$ ,  $p = .73$ ,  $AIC = -25.36$ ). Additive genetic influence on this phenotype was estimated at 43% (95% CI, .12-.52); shared environmental influence at 0% (95% CI, .00-.21); and non-shared environmental influences at 57% (95% CI, .48-.71).

### **Bivariate Correlations**

There was a significant phenotypic correlation between diurnal preference and sleep quality ( $r = .27$ , 95% CI, .21-.32), suggesting that greater eveningness preference is associated with poorer sleep quality (and conversely that greater morningness preference is associated with better sleep quality). Cross-twin cross-trait correlations (e.g., the correlation between diurnal preference in twin 1 and sleep quality in twin 2) are presented in Table 2. Greater MZ versus DZ correlations indicated that genes influenced the association between phenotypes. The MZ cross-twin cross-trait correlation for the association between phenotypes, being more than double that of the DZ and sibling correlations, implies that dominance genetic influences may be important for the association.

### **Bivariate Genetic Model**



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Since the cross-twin cross-trait correlations gave some indication that dominance genetic influences may be important for the association between diurnal preference and sleep quality, an ADE bivariate correlated factors model was tested and compared against an ACE model. An ADE model in which sex differences were equated provided the best fit to the data compared to the ACE model and models in which sex differences were free to vary (ADE bivariate model fit compared to saturated model:  $\Delta\chi^2 = 66.09$ ,  $\Delta df = 71$ ,  $p = .64$ ,  $AIC = -75.91$ ). The bivariate additive genetic correlation between phenotypes ( $rA = .57$ , 95% CI,  $-.99-.99$ ) indicated that there was substantial overlap in the genes influencing diurnal preference and sleep quality. The overlap in the dominance genetic influences, however, was very high ( $rD = .99$ , 95% CI,  $-.99-.99$ ), indicating that dominance genetic influences impacting on diurnal preference may be almost entirely shared with those impacting on sleep quality. There was negligible overlap in the non-shared environmental influences for these phenotypes ( $rE = .02$ , 95% CI,  $-.11-.16$ ). The proportion of the phenotypic correlation accounted for by additive genetic, dominance genetic, and non-shared environmental influences was also estimated in this model. Overall, additive genetic influence accounted for 2% (95% CI,  $-.52-.85$ ) of the phenotypic association between diurnal preference and sleep quality; dominance genetic influence accounted for 94% (95% CI,  $.02-1.57$ ); and non-shared environmental influence accounted for the remaining 4% (95% CI,  $-.22-.31$ ) of the covariance.

## DISCUSSION

The first aim of the present study was to estimate the extent to which genes and environments influence diurnal preference and sleep quality. For diurnal preference, additive genetic influence accounted for 0%, dominance genetic influence

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52%, and non-shared environmental influences 48%. For sleep quality, additive genetic influence accounted for 43%, shared environmental influences 0%, and non-shared environmental influences 57%. The second aim was to examine the phenotypic association between diurnal preference and sleep quality, and it was found that eveningness was associated with poorer sleep quality ( $r = .27$ ). Thirdly, we aimed to address the extent to which genetic and environmental contributions overlap for diurnal preference and sleep quality. There was 57% overlap in the additive genetic influences common to both phenotypes, and overlap in the dominance genetic influences were 99%, indicating that almost exactly the same genes may be responsible for the co-occurrence between traits. Overlap in the non-shared environmental influences common to both phenotypes, however, was much smaller, being only 2%. The fourth aim was to estimate the extent to which genetic and environmental influences contribute to the association between diurnal preference and sleep quality. Overall, additive genetic influences accounted for 2% of the association between diurnal preference and sleep quality, dominance genetic influences accounted for 94%, and non-shared environmental influences accounted for the remaining 4%. Further discussion of the main results and limitations of this study are presented below.

### **Univariate Analyses**

Univariate estimates, first on diurnal preference, indicate that dominance genetic influences - where alleles at a given locus interact to influence behavior - account for over half of the variability in the phenotype. This is somewhat consistent with other studies of the broad-sense heritability (including both additive and dominance genetic effects) of diurnal-type, where genes were found to account for around 50% of the total variability in the phenotype (Hur, 2007; Hur, et al., 1998;

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Koskenvuo, et al., 2007; Vink, et al., 2001). Also in accordance with all other reports, the remaining source of variance in diurnal preference was accounted for by non-shared environmental factors. One could postulate that such environmental factors influencing diurnal preference may be work and social commitments, since the time that one goes to bed may be influenced by external factors. These external pressures may thus influence the development of a preference for timing activities and sleep patterns in accordance with them.

With regards to sleep quality, the present results are almost identical to those reported by Partinen et al. (1983) and similar to others (Heath, et al., 1990), with additive genetic influence accounting for 43% of variance in this phenotype. Like diurnal preference, the remaining source of variance was accounted for by non-shared environment (like the majority of studies reported here, shared-environmental influences were absent for these phenotypes). Possible non-shared environmental factors influencing sleep quality can be gleaned from previous research that suggest this may be affected by life events e.g., relationship issues (Ohayon, 1996), family conflict (Gregory, Caspi, et al., 2006), unemployment (Virtanen, et al., 2008), or ill health (Walder, et al., 2007).

### **Sex Differences**

Like previous research, females were significantly more morning-oriented than males (Vink, et al., 2001); yet, also consistent with twin research there were no significant differences in the magnitude of genetic/environmental effects on this sex difference (Koskenvuo, et al., 2007). However, the present findings did note scalar sex differences, (i.e., male's scores varied to a greater extent than did female's), indicating that diurnal preference may be a more variable trait for males than females.

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With regards to symptoms of sleep disturbances, no sex differences were found for prevalence and estimates of heritability. This is contrary to both clinical and epidemiological studies, which suggest that females experience greater sleep disturbance than males (Ohayon, 2002), and also a recent twin study in which sleep quality for females was more heritable than for males (Paunio, et al., 2009). As such, further studies investigating sex differences for sleep quality are essential in both clinical and non-clinical populations in order to determine whether males and females do differ with regards to the magnitude of genetic and environmental influences on this phenotype.

### **Bivariate Analyses**

The current analyses demonstrate that eveningness preference is associated with poor sleep quality, which is consistent with previous suggestions (Koskenvuo, et al., 2007; Megdal & Schernhammer, 2007; Shiihara, et al., 1998; Vardar, et al., 2008). This finding may be related to intrinsic properties of the circadian system. It has been suggested that the phase position of the endogenous circadian oscillator of evening-types is delayed compared to that of morning-types (Kerkhof & Van Dongen, 1996), and evidence has demonstrated that the core body temperature minimum in evening-types occurs much later in the night-time period than morning-types (Baehr, et al., 2000). As such, evening-types sleep on an earlier part of their temperature cycle, and their temperature nadir occurs closer to waking compared to morning-types, i.e., the phase angle between sleep and wake-time is smaller in evening types (Baehr, et al., 2000; Waterhouse, et al., 2001). Since alertness is lowest near the temperature minimum and evening-types awaken closer to the time of this nadir, this may account for their feeling less alert upon awakening. It is also possible that the difficulty awakening of evening-types leads them to infer that they have slept poorly. Of course,

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it is also possible that evening-types actually sleep shorter overall, given a preference for later self-selected bedtimes in combination with earlier than desired wake-times constrained by school or occupational demands (of note, there was a small but significant association between diurnal preference and sleep duration [ $r = .08, p < .05$ ], indicating that evening-types slept for a shorter duration than morning-types).

With this in mind, one may infer that circadian rhythm sleep disorders, such as DSPD (characterized by extreme eveningness), may also encompass poor sleep quality. Although the diagnostic criteria for DSPD describe normal sleep quality and duration when individuals are allowed to choose their sleep schedule (American Academy of Sleep Medicine, 2005), truly *ad lib* sleep schedules are difficult to attain in the real world. Thus, even though the present analysis represents individuals in the full range of circadian preference, rather than the extremes (as represented by DSPS), an evening-type diurnal preference was associated with poor sleep quality. Empirical research examining sleep quality in individuals with ASPD and DSPD would be beneficial to refine existing diagnostic criteria and our understanding of sleep quality-diurnality associations.

### **Genetic Influence on the Association between Diurnal Preference and Sleep Quality**

A novel finding presented here is that the association between phenotypes is almost entirely explained by genetic influences, and there is substantial overlap in the genes influencing both phenotypes. This suggests that the genes associated with greater evening preference are also associated with increased sleep disturbance. This is informative for future research into diurnal preference and sleep quality, since it suggests that genes already known to be associated with one phenotype should be considered as possible candidates for exploration with regards to the other. For

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example, extensive research has indicated that polymorphisms of the *CLOCK* gene, 3111 T/C allele influence eveningness and sleep timing (Katzenberg, et al., 1998), and *PER1*, *PER2*, and *PER3* extreme circadian preference (Archer, et al., 2003; Carpen, et al., 2005; Carpen, et al., 2006). Both the *CLOCK* 3111 T/C (Serretti, et al., 2003) and serotonin 5HTTLPR (Brummett, et al., 2007) polymorphisms have been related to sleep quality. In the search for genes common to both phenotypes, it has been found that homozygosity for the 5-repeat allele in the *PER3* variable number tandem repeat polymorphism is associated with both morning preference (Archer, et al., 2003; Ellis, et al., 2009; Jones, et al., 2007) and increased sleep pressure (i.e., shorter sleep latency, more theta and alpha activity in wake and REM sleep, more slow wave activity in non-REM sleep, and more slow wave sleep (Viola, et al., 2007), which is broadly associated with good sleep quality. This suggests that it may be beneficial to investigate the role of genes associated with one phenotype in relation to the other.

### **Environmental Influence on the Association between Diurnal Preference and Sleep Quality**

Environmental influences accounted for only a small proportion of the association between phenotypes, and overlap between environmental influences was also small. This again demonstrates the importance of genetic factors in explaining reasons behind the association between diurnal preference and sleep quality in the normal range.

### **Limitations**

There are four main limitations to this research. The first regards the use of self-report measures to determine both diurnal preference and sleep quality. However, the MEQ and PSQI are widely used, and good psychometric properties of both measures have been well established (Anderson, et al., 1991; PSQI: Backhaus, et al.,

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2002; Buysse, et al., 1989; Chelminski, et al., 1997; for example, MEQ: Smith, et al., 1989). Furthermore, the large sample size required to perform a genetic decomposition of a phenotype limits the ability to obtain polysomnographic measures of these constructs and would be too costly and time-consuming (Gregory, Rijdsdijk, & Eley, 2006). Simple techniques for evaluating circadian phase and sleep, such as wrist actigraphy and simplified ambulatory EEG monitors, may be useful additions to future behavioral genetic studies.

The second limitation regards the age range used in the present analysis. Previous research has suggested that diurnal preference and the influence of specific genes on this phenotype change with age (Carrier, et al., 1999; Jones, et al., 2007; Talliard, et al., 1999; Vink, et al., 2001); thus, this would be useful to be investigated in relation to sleep quality. However, since our participants were all young adults, we were unable to investigate this important issue.

Third regards the generalizability of the results. Since heritability is a population statistic, the results may only be applicable to the population under study. Indeed one study found evidence that different climates throughout the world influence differences in chronotypes (Randler, 2008a). As such, replications in different populations would be necessary before we can confidently extrapolate our findings.

The final limitation regards power. Although we found strong overlap in the additive genetic and dominance genetic influences accounting for the association between phenotypes, confidence intervals for some parameters were wide and often spanned zero. This is common in twin research and largely reflects our sample size and consequent power limitations. Although our sample size was relatively large, this

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finding highlights the need for replications in much larger twin populations before our conclusions can be confidently drawn.

### **Conclusion**

Eveningness preference is associated with poor sleep quality and this association is largely under genetic control. Those genes influencing diurnal preference are substantially shared with those influencing sleep quality. Further exploration of specific genotypes and environmental factors influencing this association will aid in the progression to understanding the complexities of sleep and the circadian system.

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Table 1: Descriptive statistics. Means (SD) of scores on the Morningness and Eveningness Questionnaire (MEQ) and the Pittsburgh Sleep Quality Index (PSQI)

	Males	Females	MZ	DZ	Sibs
MEQ	54.88 (8.38)*	52.42 (7.78)*	51.77 (7.67)	54.10 (8.20)	53.64 (8.20)
PSQI	5.58 (3.00)	5.72 (3.01)	5.45 (2.86)	5.74 (3.10)	5.70 (2.93)

*Note:* MZ = monozygotic twins; DZ = dizygotic twins. Means and standard deviations (SD) of raw (untransformed) data. Note that the MEQ variable has been reversed so that higher scores indicate greater eveningness. Sex differences for means were tested, \*  $p < .01$ . All analyses were run in Mx incorporating a weight to account for initial participation bias and attrition.

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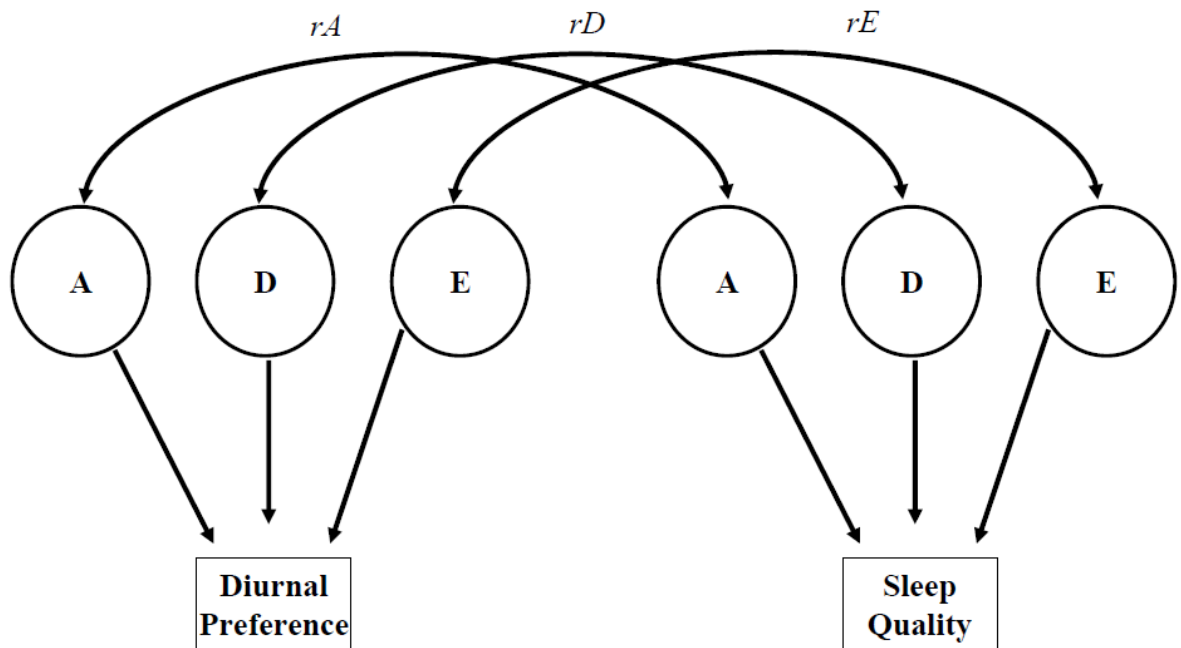
Table 2: Phenotypic correlations for Monozygotic twins (MZ), Dizygotic twins (DZ) and siblings (Sibs) (95% Confidence Intervals)

	MEQ-MEQ	PSQI-PSQI	MEQ-PSQI
Within Twins	/	/	.27 (.22 - .32)
Cross Twins			
MZ	.50 (.39 - .60)	.42 (.29 - .53)	.25 (.16 - .33)
DZ	.10 (-.01 - .20)	.25 (.14 - .35)	.04 (-.04 - .12)
Sibs	.17 (-.01 - .34)	.11 (-.08 - .30)	.08 (-.06 - .20)

*Note:* All analyses are run on transformed (i.e. age and sex regressed) data and include a weight variable to account for initial selection bias and attrition. The model was constrained where appropriate. For example, the twin correlations are constrained so that those of the randomly selected twin 1's are the same as the randomly selected twin 2's.

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Figure 1. Bivariate Correlated Factors Model.



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*Note.* A = Additive genetic influence; D = Dominance genetic influence; E = Non-shared environmental influence;  $r_A$  = Bivariate additive genetic correlation;  $r_D$  = Bivariate dominance genetic correlation;  $r_E$  = Bivariate non-shared environmental correlation.