REGULAR RESEARCH PAPER



The impact of social jetlag and chronotype on attention, inhibition and decision making in healthy adults

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Revised: 4 November 2019

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Funding information European Commission, Grant/Award Number: 667302

Abstract

Sleep and circadian clock disruption are associated with neuropsychiatric disorders, such as attention deficit hyperactivity disorder, but the impact on neurocognitive performance is unclear. We assessed whether chronotype and everyday circadian misalignment manifested as social jetlag were associated with inter-individual neurocognitive performance across domains of attention, inhibitory control and decision making. One hundred and eighty-eight healthy young adults were assessed for sleep and circadian properties and performed two neurocognitive tasks, the Continuous Performance Test and the Iowa Gambling Task. Social jetlag was associated with significantly faster and less variable reaction times and commission errors on the Continuous Performance Test. Poorer subjective sleep quality was associated with poorer decision making on the lowa Gambling Task. No effects were present for polymorphisms in the circadian clock genes CLOCK and PER3. We conclude that circadian disruption shaped by everyday environmental factors may impact on attentional/ inhibitory performance but not on a measure of risky decision making.

KEYWORDS

attention, chronotype, decision making, impulsivity, sleep, social jetlag

1 | INTRODUCTION

Circadian rhythms are recurring cycles displaying periods of nearly twenty-four hours, which are present in a range of behavioural, physiological and cognitive processes (Buttgereit, Smolen, Coogan, & Cajochen, 2015). Circadian rhythms interact with homeostatic sleep pressure to shape the timing of sleep (Borbély, Daan, Wirz-Justice, & Deboer, 2016). An important manifestation of these circadian traits in human behaviour is chronotype, reflecting the tendency to structure daily activities, including sleep-wake times, according to the underpinning circadian clock (Roenneberg, Wirz-Justice, & Merrow, 2003). Chronotype is also associated with psychological domains, including personality, impulsivity and psychopathology (Li et al., 2018). Furthermore, chronotype is associated with optimal timing of daily performance in a number of cognitive tasks, with a synchrony effect existing between time of testing and chronotype for many cognitive domains (such that late chronotypes perform better later in the day, early chronotypes earlier in the day; Schmidt, Collette, Cajochen, & Peigneux, 2007).

Heritability of chronotype is estimated at approximately 50% and recent genome-wide studies have identified a number of clock and non-clock genes associated with a modest portion of trait variance (Kalmbach et al., 2017). For appropriate entrainment of the circadian clock to environmental zeitgebers, the clock responds to both the zeitgeber timing and intensity to shape chronotype (Roenneberg, Kumar, & Merrow, 2007). Age and sex also profoundly influence chronotype throughout the lifespan (Fischer, Lombardi, Marucci-Wellman, & Roenneberg, 2017). One consequence of a later chronotype is a propensity to experience greater levels of social jetlag (SJL): the mismatch between internal biological time and socially driven behavioural schedules suggested to produce chronic circadian misalignment (Wittmann, Dinich, Merrow, & Roenneberg, 2006). SJL is conceptualized as the difference in sleep timing in the presence of social imperatives ("work" days) and in the absence such imperatives ("free" days), with the timing of mid-sleep on "free" days understood to be reflective of the underlying phase of circadian entrainment (Roenneberg et al., 2003). SJL also represents a state of sleep restriction because later chronotypes are chronically subjected to compressed sleep times on workdays, leading to shorter sleep duration and compensatory oversleep on free days (Wittmann et al., 2006). SJL is associated with greater impulsivity and inattention (McGowan, Voinescu, & Coogan, 2016) and impaired reward-related brain function (Hasler et al., 2012).

Several studies have implicated altered circadian function in attention deficit hyperactivity disorder (ADHD), with typical findings indicating delayed circadian phase, later chronotype and impaired sleep duration and/or quality (Coogan & McGowan, 2017; Coogan et al., 2019). It is not presently clear whether such circadian and sleep behaviour changes are risk factors for, or symptoms of, ADHD. However, it has recently been hypothesized that a substantial portion of ADHD symptomatology may be accounted for by sleep changes comorbid with the disorder (Bijlenga, Vollebregt, Kooij, & Arns, 2019). Considering ADHD dimensionally, rather than taxonomically, may help clarify this question, as ADHD may be better articulated as the extreme and impairing tail of continuously distributed heritable traits (Martin, Hamshere, Stergiakouli, O'Donovan, & Thapar, 2014), and such traits may be monitored among the general population without associated medication and psychiatric confounds. To this end, objective assessment of neurocognitive performance is an important tool. Primary neurocognitive deficits in ADHD involve response inhibition, reaction time variability and risky decision making, and these have been proposed as candidate endophenotypes of ADHD (Pinar et al., 2018). Moreover, executive function and neurocognitive performance are intrinsically circadian in their organization and therefore particularly susceptible to disruption (Schmidt et al., 2007). As such, elucidation of associations between measures of circadian function and ADHD-related neurocognitive traits may illuminate the issue of the inter-relatedness of ADHD, sleep and circadian function.

The current study examined whether chronotype and/or SJL would be associated with performance on neurocognitive measures of attention and response inhibition and risky decision making in a healthy young adult sample, and whether any such changes would approximate those observed previously in ADHD. Further, we investigated whether such associations are moderated by polymorphisms in two circadian clock genes (*CLOCK* and *PER3*) that have been previously implicated in ADHD and/or cognition (e.g., Archer, Schmidt, Vandewalle, & Dijk, 2018; Kissling et al., 2008).

2 | METHODS

2.1 | Participants

Participants were 188 volunteers (98 male, 90 female; 52/48%) with a mean age of 22.3 (±3.6, SD) years, recruited from the

student population enrolled in undergraduate and postgraduate courses at Maynooth University. Exclusion criteria were shiftwork, psychiatric/neurological disorder, sleep disorder/medication use or any medical condition that may adversely affect sleep (e.g., diabetes and autoimmune disorder). Informed written consent was obtained from all participants. Ethical approval for this study was granted by the Research Ethics Committee at Maynooth University.

2.2 | Chronotype, social jetlag and sleep assessment

Chronotype and social jetlag were assessed using the Munich Chronotype Questionnaire (MCTQ; Roenneberg et al., 2003). Midsleep on free days adjusted for accumulated sleep debt (MSF_{sc}) was used as a measure of chronotype. From the sample distribution, we segregated three chronotype groups (early, intermediate, and late) based on a tertile split of MSF_{sc} values. The difference between midsleep on workdays and unadjusted free days was used to determine habitually accrued social jetlag (SJL) (Wittmann et al., 2006). The Pittsburgh Sleep Quality Index (PSQI) was used to record selfreported sleep quality (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). PSQI scores range between 0 and 21 and a score of >5 is used to indicate poor sleep.

2.3 | Neurocognitive testing

Neurocognitive tasks were presented using the psychology experiment building language (PEBL v0.13; Mueller & Piper, 2014). Prior to testing, participants rated their current level of sleepiness on the Stanford Sleepiness Scale (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973). Participants completed neurocognitive tasks between 12:00 and 14:00 hours in order to minimize synchrony effect bias for either early or late chronotypes. Data collection was continuous throughout the entire year, with the exception of the university closing during the final and first weeks of December and January, respectively.

The Continuous Performance Test (CPT; Conners, Epstein, Angold, & Klaric, 2003) assessed sustained attention and inhibitory control. The test consisted of 360 trials in which participants were required to respond using the spacebar to all letters of the alphabet, with the exception of 'X' (Go-stimulus), and withhold their response to the letter 'X' (No-Go stimulus; presented in 10% of trials). Stimuli appeared white against a black background in the centre of the screen for 250 ms. The inter-stimulus interval (ISI) between trials varied between blocks of 1, 2 and 4 s. Each ISI condition block was presented consecutively for 20 trials before randomly and without warning switching to another. To eliminate anticipated responses, trials with response times <100 ms were discarded from the analysis. Outcome measures from the CPT were the mean (RT) and standard deviation (RTSD) of reaction times and the rates of omission (Om Err) and commission error (Comm Err). Reaction time was modelled using an exponentially modified normal distribution (ex-Gaussian), which decomposes typically skewed reaction time distributions into normal and excessively slow components. Parameters reflecting the ex-Gaussian distribution (mu [μ], sigma [σ] and tau [τ]), were calculated using the egfit function from the DISTRIB toolbox (Lacouture & Cousineau, 2008) for MATLAB R2012b (Mathworks, Natick, MA; 2012). The μ parameter represents the mean of the normally distributed component of the frequency distribution and σ the standard deviation (*SD*), whereas τ represents both the mean and standard deviation of the tail-end exponential component (Lacouture & Cousineau, 2008). Faster *mu* predicts impulsive responses and commission errors in the context of ADHD; slower *tau* predicts omission errors and lapses in attention (Hervey et al., 2006; Tarantino, Cutini, Mogentale, & Bisiacchi, 2013).

The Iowa Gambling Task (IGT) assesses decision-making processes under risk and relies on contingencies of reward and penalty, as well as initial uncertainty of test premises and outcomes (Bechara, Damasio, Damasio, & Anderson, 1994). Four decks (labelled 'A', 'B', 'C' and 'D') are presented and participants are asked to freely choose from a single deck each turn. Decks differ with respect to the amount and the frequency of monetary gains/losses produced by each selection. Decks A and B produce large rewards but more severe penalties at points that are not predicable by participants. Decks C and D produce smaller monetary gains but also consistently smaller losses. Therefore, in the long run selections from decks C and D are more advantageous than selections from decks A and B. The IGT consisted of 100 trials. After each trial, feedback was presented to participants indicating reward, penalty and net score of their selection. Sixteen participants who maximized a deck by making over 60 selections from one deck were eliminated from the analysis as performance on these trials indicates a different set of decision rules compared to Bechara and colleagues' original test.

2.4 | DNA extraction and genotyping

Saliva samples were obtained from participants using Oragene DNA OG-500collectionkits(DNAGenotek,Nepean,ON,Canada)andDNA was isolated using DNeasy kits (Qiagen). Genotyping of the *CLOCK* (rs1801260) polymorphism was performed as described in Kissling et al. (2008) using the primers 5'-TCCAGCAGTTTCATGAGATGC-3' (forward) and 5'-GAGGTCATTTCATAGCTGAGC-3' (reverse). Genotyping of the *PER3* 4/5-repeat VNTR (rs57875989) was performed as described in Ebisawa et al. (2001), with primers 5'-CAAAATTTTATGACACTACCAGAATGGCTGAC-3' (forward) and 5'AACCTTGTACTTCCACATCAGTGCCTGG-3' (reverse).

2.5 | Data analysis

The approach informing the study's conception and analysis was exploratory rather than directly hypothesis testing, as we did not have sufficient a-priori information to specify which outcomes of the CPT and IGT would be most likely to be impacted. Groupwise comparisons were conducted using one-way ANCOVAs (for CPT summary variables), mixed ANCOVAs (for block and ISI comparison of CPT ex-Gaussian parameters and IGT performance) or between-groups factorial ANOVAs for genotype × chronotype/SJL interactions. Relationships between CPT response components were analysed via multiple linear regression. Covariates inserted in all the models were age, gender and pretest Stanford Sleepiness Scale score. Equivalence testing was conducted using the TOSTR module in Jamovi Stats (www.jamovi.org). Statistical analysis was conducted in either SPSS, , IBM Coporation, JASP Stats (https://jasp-stats.org/) or R (https://www.r-project.org/). Effect sizes were assessed via ηp^2 and interpreted according to Cohen (1988). p <.05 was considered as indicating statistically significant differences.

TABLE 1 Correlation matrix between demographic characteristics, questionnaire outcomes and Continuous Performance Test (CPT)

 performance

	Gender	Age	MSF_{sc}	SJL	PSQI	SSS	RT	RTSD	Om Err	Comm Err
Age	-0.040	-	-	-	-	-	-	-	-	-
MSF_{sc}	-0.159*	-0.115	-	0.489***	0.219**	-	-0.171*	-0.123	-0.007	0.112
SJL	-0.087	-0.175*	0.504***	-	0.066	-	-0.185*	-0.186*	0.053	0.138
PSQI	0.068	-0.019	0.214**	0.061	-	-	0.012	0.119	0.042	0.038
SSS	0.012	-0.127	0.107	0.027	0.147*	-	-	-	-	-
RT	-0.014	0.171*	-0.184*	-0.209**	0.009	-0.026	-	0.544***	-0.042	-0.543***
RTSD	0.001	0.030	-0.123	-0.188*	0.130	0.075	-0.539***	-	0.320***	-0.086
Om Err	-0.011	-0.156*	0.005	0.078	0.039	-0.017	-0.064	0.313***	-	0.340***
Comm Err	-0.026	-0.238***	0.143*	0.176*	0.042	0.064	-0.556***	-0.093	0.356***	-

Note: Table shows Pearson correlation coefficients. Shaded bisection indicates partial correlations controlling for gender, age and Stanford Sleepiness Scale score. Gender coded as 0 = male, 1 = female. Bold values indicate statistically significant correlations.

Comm Err, commission error; MSF_{sc}, midsleep on free days adjusted for accumulated sleep debt; Om Err, ommission error; PSQI, Pittsburgh Sleep Quality Index; RT, mean of reaction times; RTSD, standard deviation of reaction times; SJL, social jetlag; SSS, Stanford Sleepiness Scale.

**p < .01,

***p < .001.

3.1 | Impact of chronotype and social jetlag on

performance on the CPT

Preliminary correlational analyses between guestionnaire and CPT outcomes are shown in Table 1. Notably, adjusting for gender, age and Stanford Sleepiness Scale score, there was a statistically significant inverse association between MSF_{sc} and RT, but not between MSF_{sc} and response variability and accuracy. SJL was inversely associated with RT and RTSD but not with accuracy. Sleep timing, latency, inertia and light exposure MCTQ measures are presented in Table S1. Light exposure on workdays or free days was not associated with MSF_{sc} time (r = .058, p = .433; r = -.13, p = .08) or SJL (r = .005, p = .95; r = .051, p = .49). Furthermore, as performance is time-of-day dependent we assessed whether

When CPT outcomes were assessed in a groupwise fashion, there was no effect of chronotype (early, intermediate or late) on any of the outcomes ($F_{2.182}$ = 2.16, p = .115 for RT [Figure 1a]; $F_{2.182} = 1.10, p = .34$ for RTSD [Figure 1b]; $F_{2,183} = 0.317, p = .69$ for omission errors [Figure 1c]; $F_{2.183} = 0.294$, p = .75 for commission errors [Figure 1d]). We then created three groups for SJL based on a tertile split (low SJL = 0-1.25 hr; medium SJL = 1.25-2.25 hr; high SJL >2.25 hr) and found statistically significant effects of SJL group on RT and RTSD, but not on omission errors or commission errors $(F_{2.183} = 5.77, p = .004, \eta p^2 = 0.060 \text{ for RT [Figure 2a]}; F_{2.183} = 5.95,$

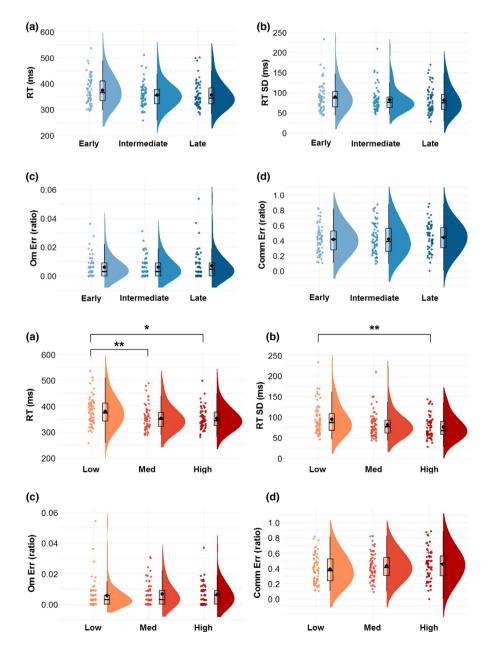


FIGURE 1 Box and split-half violin plots showing the effect of chronotype group on measures derived from the Continuous Performance Test (CPT): mean of reaction times (RT) (a), standard deviation of reaction times (RTSD) (b), omission error (Om Err) (c) and commission error (Comm Err) (d). Individual jittered data points, density distribution and summary statistics (median as bar in boxplot, mean as filled circle) are simultaneously visualized

FIGURE 2 Box and split-half violin plots showing the effect of social jetlag (SJL) group on measures derived from the Continuous Performance Test (CPT): mean of reaction times (RT) (a). standard deviation of reaction times (RTSD) (b), omission error (Om Err) (c) and commission error (Comm Err) (d). **p < .01; *p < .05

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p = .003, $\eta p^2 = 0.061$ for RTSD [Figure 2b]; $F_{2,183} = 0.228$, p = .80 for omission errors [Figure 2c]; $F_{2,183} = 1.034$, p = .36 for commission errors [Figure 2d]). Bonferroni post-hoc comparisons revealed that medium and high SJL groups responded significantly faster than the low SJL group (p = .008 and p = .018, respectively) and that the high SJL group had significantly greater variability in response times compared to the low SJL group (p = .003). Therefore, greater SJL was associated with faster reaction times on the CPT. Further, correlations between CPT parameters and MCTQ sleep variables from workdays and free days (Table S2) support associations with disturbed sleep arising from SJL. Notably, later sleep onset on free days, but not workdays, was associated with faster RT and increased commission errors. Similarly, shorter

sleep duration on workdays, but not free days, was associated with more errors.

When the extremes of SJL were examined (20% lowest vs. 20% highest; Figure 3), RT was significantly faster in the high SJL group (p < .001), RTSD was greater in the high SJL group (p = .012), there were more errors of commission in the high SJL group (p = .023), but there was no difference in omission errors (p = .386). As such, for the quintile with the greatest SJL, faster RTs are accompanied by an increased rate of commission errors. We next examined if there was an influence of interstimulus interval on the SJL (three groups) effect on RT. There was a main effect of ISI (1 s, 2 s or 4 s) on RT ($F_{1.71,310.58} = 19.87$, p < .001, $\eta p^2 = 0.10$), but no interaction between ISI and the SJL group (p = .605; Figure S1). Similarly, there

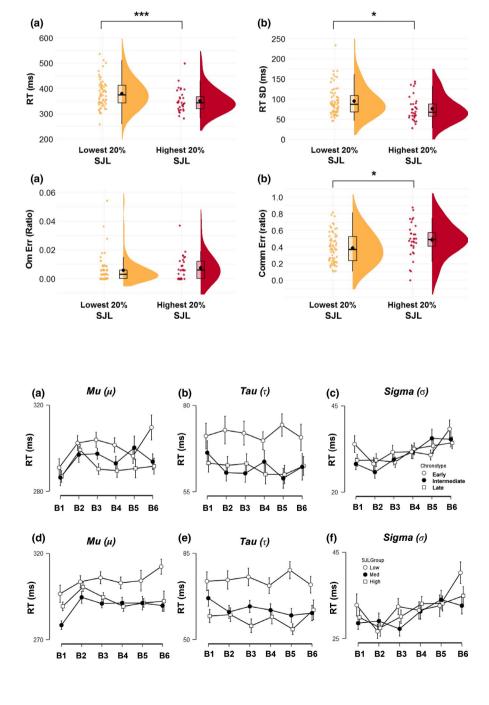


FIGURE 3 Box and split-half violin plots showing the effect of social jetlag (SJL) extremes on measures derived from the Continuous Performance Test (CPT): mean of reaction times (RT) (a), standard deviation of reaction times (RTSD) (b), omission error (Om Err) (c) and commission error (Comm Err) (d). ***p < .001; p < .05

FIGURE 4 Ex Gaussian components of Continuous Performance Test (CPT) reaction time, by time on task, between chronotype groups (a–c) and social jetlag (SJL) groups (d–f). There were no significant block × group interactions on any of these measures

was no interaction between chronotype and ISI (p = .998; Figure S1).

Next, we examined the ex-Gaussian parameter estimates of RT on the CPT, as previous studies show ADHD-specific effects of slowed responses in the tau portion of the curve with increasing time on task (Figure 4). There were no statistically significant main effects of chronotype group on mu, tau or sigma, nor any block by chronotype group interactions (Figure 4a-c). There were significant main effects in SJL group comparisons for mu ($F_{2.185}$ = 3.41, p = .035, ηp^2 = 0.036; Figure 4d) and tau $(F_{2.185} = 9.86, p < .001, \eta p^2 = 0.096;$ Figure 4e). Bonferroni posthoc tests showed that the low SJL group performed significantly slower than the medium SJL group on the normally distributed mu component of RT (p = .035), and significantly slower on the exponential tau component of RT than the medium SJL group (p = .004) and the high SJL group (p < .001). The main effect for test block indicating time on task changes was significant for mu ($F_{4,11,759,57}$ = 3.54, p = .007, ηp^2 = 0.02), but not for tau $(F_{4.59.848.74} = 0.26, p = .93, \eta p^2 = 0.001)$. There was not a significant main effect for group on the sigma component of RT $(F_{2,185} = 0.83, p = .44, \eta p^2 = 0.01;$ Figure 4f). There was a significant main effect of block on sigma ($F_{4.33,800.55} = 6.35$, p < .001, $np^2 = 0.033$), such that the variability of the normally distributed component of RT increased as time on task increased. The $SJL \times block$ interaction effect was not significant for *mu*, *tau* or sigma (p = .59, p = .45 and p = .15, respectively).

To assess the associations between ex-Gaussian reaction time parameters and errors of omission and commission, we conducted regression analyses to assess the degree to which response accuracy was predicted by mu, sigma and tau of reaction time. The model for predicting errors of omission (Table S3) comprising all three predictors was statistically significant, ($F_{3.184}$ = 16.011, p < .001; adjusted R^2 = 0.194). Mu value accounted for 14.5% of the variance in omission error rate and was negatively related to omission errors (faster, less errors). Tau predicted 5.1% of the variance and was positively related to omission errors (slower, more errors). When errors of commission were examined (Table S4) the model with the three ex-Gaussian predictors was statistically significant ($F_{3.184}$ = 47.1, p < .001) and accounted for approximately 42.5% of the variance in commission error rate (adjusted R^2 = 0.425). Mu was the most meaningful negative predictor of commission error frequency (faster, more errors), predicting 39.5% of the variance in errors of commission by individuals on the CPT.

3.2 | Impact of chronotype and social jetlag on performance in the IGT

A mixed ANCOVA revealed that net IGT score increased significantly across test blocks (as individuals learned to discriminate advantageous decks from disadvantageous ones; main effect for block, $F_{3.69,620.4} = 31.7$, p < .001, $\eta p^2 = 0.16$; Figure 5). There were no statistically significant main effects of SJL group on IGT score, block × SJL interaction, or of chronotype group or block × chronotype group interaction. Therefore, neither chronotype (Figure 5a) nor SJL (Figure 5b) appear to have an impact on performance on the IGT.

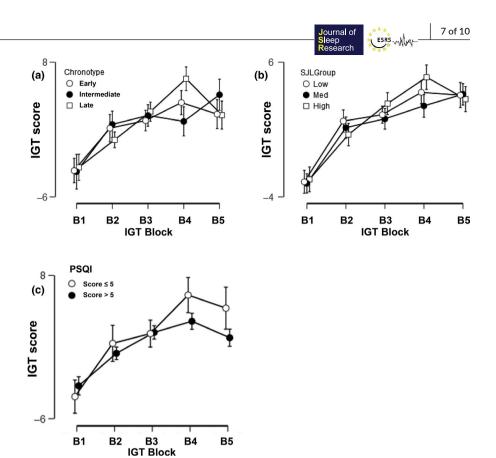
3.3 | Impact of subjective sleep quality on performance in the CPT and IGT

From PSQI scores we created two groups, those whose subjective sleep quality indicated poor sleep (scores of 6 or greater) and those whose subjective sleep quality did not (scores of 5 or less), and examined groupwise differences in CPT and IGT performance. When groupwise differences on measures of the CPT were examined, there were no effects on RT (p = .94), RTSD (p = .11), omission errors (p = .83) or commission errors (p = .37). Further, there were no effects of group on any of the ex-Gaussian measures. There was no group × block interaction on the IGT (p = .16); however, there was a main effect of group ($F_{1.164} = 4.48$, p = .035, $\eta p^2 = 0.03$), indicating that individuals with disturbed sleep had lower net IGT scores than those with undisturbed sleep (Figure 5c).

3.4 | Effect of CLOCK 3111T/C and PER3 VNTR on performance in the CPT

The genotype distribution frequency for our sample was in Hardy-Weinberg equilibrium (HWE) for the *PER3* VNTR ($\chi^2 = 0.036$, p > .05); however, *CLOCK* 3111T/C deviated from the expected HWE genotype distribution ($\chi^2 = 19.9$, p < .05). For *CLOCK* 3111T/C, 51.7% of the sample were homozygous for the T-allele and 48.3% were C/T heterozygotes, whereas none were C/C homozygous. For *PER3*, 50.6% were homozygous for the short repeat *PER3* 4/4, 41.1% carried one of the long and short repeats *PER3* 4/5, and 8.3% were long-repeat homozygotes *PER3* 5/5. Due to the absence of C/C individuals for the *CLOCK* SNP, groups were instead analysed by allelic group, comparing T/T homozygous individuals with those that carried a C-allele.

On CPT performance, we did not detect any significant differences between *CLOCK* 3111 T/T homozygous individuals and C-allele carriers on RT (p = .78), RTSD (p = .32), omission error rate (p = .45) or commission error rate (p = .15). IGT score did not show a significant main effect for *CLOCK* genotype (p = .713) or *CLOCK* genotype × block interaction effect (p = .419). Equivalence testing confirmed the absence of any effect of *CLOCK* 3111T/C genotype on any measure on the CPT or the IGT. For *PER3* VNTR genotype we did not detect a significant main effect of genotype on CPT mean RT (p = .96), RTSD (p = .18), omission errors (p = .54) or commission errors (p = .87). For IGT score, there was not a significant main effect for *PER3* genotype (p = .28) or a genotype × block interaction (p = .34). Equivalence testing confirmed the absence of any effect of *PER3* VNTR genotype on any measure on the CPT. We next examined whether *CLOCK* or *PER3* FIGURE 5 Net scores on the Iowa Gambling Task (IGT) by block in (a) chronotype groups, (b) social jetlag (SJL) groups and (c) Pittsburgh Sleep Quality Index (PSQI) score groups. There was no impact of chronotype or SJL on performance. Individuals with a PSQI score >5 displayed lower net scores than those with a score of less than or equal to 5. There were no significant block × group interactions on any of these measures



genotype might moderate the relationship between chronotype and SJL on CPT performance (Figure S2). We found that neither *CLOCK* nor *PER3* genotype moderated the relationships between chronotype and RT or RTSD (*CLOCK* × chronotype interaction for RT, p = .14 [A]; *CLOCK* × chronotype interaction for RTSD, 0.152 [B]; *CLOCK* × SJL interaction for RT, p = .35 [C]; *CLOCK* × SJL interaction for RTSD, p = .93 [D]; *PER3* × chronotype interaction for RT, p = .26 [E]; *PER3* × chronotype interaction for RTSD, p = .55[F]; *PER3* × SJL interaction for RT, p = .36 [G]; *PER3* × SJL interaction for RTSD, p = .085 [H]). Further, there was no moderation of the impact of PSQI score on RT or RTSD for either *CLOCK* (p = .96 and p = .49, respectively) or *PER3* (p = .477 and p = .94, respectively).

4 | DISCUSSION

This study examined the extent to which parameters measuring habitual sleep were associated with putatively dissociable neurocognitive indicators of impulsivity and inattention in a sample of healthy young adults. Our most notable finding was that SJL was associated with significantly faster and less variable reaction times on a response inhibition task conventionally used to assess behavioural responses in attention disorders such as ADHD.

Faster reaction times are generally associated with more impulsive response styles and increased errors of commission, whereas greater variability is associated with more omission errors reflecting inattention (Epstein et al., 2003). Analysis of the ex-Gaussian distribution in part confirmed these assumptions, as the *mu* parameter is the greatest predictor of commission errors, whereas increasing tau, representing excessively slower reaction times, predicted omission errors (Hwang-Gu, Gau, Tzang, & Hsu, 2013). Greater SJL was associated with smaller mu and tau, suggesting simultaneously more impulsive responding and less interindividual variability in high SJL. Studies in ADHD show a smaller mu is noted compared to controls, similar to the current findings with greater SJL (Hervey et al., 2006; Hwang-Gu et al., 2013). Studies in ADHD also consistently report elongated tau, suggesting sustained attention deficits (Hervey et al., 2006; Tarantino et al., 2013). Applying this interpretation to the current data suggests that individuals with low SJL are more susceptible to lapses in attention on the CPT, in contrast to a previous study reporting that greater weekendweekday misalignment is associated with greater errors of omission on similar behavioural tasks (Kim et al., 2011). An alternative interpretation is that greater variability of infrequent slow reaction times indexed by greater values of tau may be reflective of a more cautious responding style in which participants maintain an effort to minimize errors of commission. This would appear consistent with studies that show that less volatile shifts in sleep timing are negatively correlated with self-reported risk-taking behaviour (e.g., O'Brien & Mindell, 2005).

As cognitive task performance is moderated by energetic factors of the task (Hervey et al., 2006) and vigilance decreases with increasing time on task (Tarantino et al., 2013), we examined if the momentum of the delivery of test stimuli or the test block produced directional effects on reaction times. Consistent with previous reports, which show that reaction time is primed by task momentum, we found that reaction times of both normal and exponential parameters increased as the ISI of test stimuli increased (Hervey et al., 2006; Hwang-Gu et al., 2013). When performance was assessed as a function of block, reaction time variability of normal reaction times (sigma) of all groups increased as the time on task increased, indicating a waning of sustained attention over time, but this was not observed for the *tau* parameter. Importantly, unlike previously reported findings from ADHD studies, we did not find that SJL group differences in Gaussian or ex-Gaussian measures of reaction time were moderated by ISI or block. With increasing time on task, individuals with ADHD display disproportionately increased omission errors, reaction time, reaction time variability and tau parameter, indicating an increased burden on attentional resources over time, and individuals with ADHD display more profound response slowing as the speed of the test slows (Hervey et al., 2006; Tarantino et al., 2013). Instead, our findings show that SJL group differences remained stable throughout the test and were not modified by the contextual features of the task, suggesting that responses may be slower in individuals with low levels of SJL due to speed/accuracy trade-off, rather than attentional lapses per se. As such, alterations in CPT performance associated with SJL may be of a different kind to those in ADHD; clearly, such a finding has implications for understanding any causal role of circadian misalignment in ADHD symptomatology (Bijlenga et al., 2019).

The effects that we observe on the CPT seem to be specific to SJL, as groupwise comparisons using MSF_{sc} or PSQI scores do not produce similar effects. As SJL represents a putative state of chronic circadian misalignment and constrained workday sleep, our findings have implications for the later circadian typology frequently noted in studies examining impulsive traits and in ADHD (Coogan & McGowan, 2017). Our findings suggest that studies that associate impulsivity with later chronotype/delayed circadian function (McGowan & Coogan, 2018; Song et al., 2019) may reflect an effect of greater SJL typically experienced by later chronotypes. Further, there may be differential impacts of chronotype and/or SJL on attention and/or impulsivity in ADHD populations compared to healthy controls.

One putative mechanistic explanation for these responses is dysregulation of dopaminergic mesocortical pathways. Hasler et al. (2012) report that the greater weekend-weekday advance in midsleep of adolescents is associated with decreased activation of the medial prefrontal cortex and ventral striatum in response to reward, suggesting reduced regulatory response and reward sensitivity among individuals with greater SJL. Coutinho et al. (2015) reported that short-term jetlag results in greater default mode network (DMN) activation, deactivation of which is associated with goal-directed behaviour, target detection and attention in which phasic dopamine release has a crucial modulatory role. In light of the current findings it seems plausible that SJL may promote non-optimal regulation of frontal cortex processes, as has been indicated for later chronotypes (Song et al., 2019). Further experimental work involving behavioural and imaging techniques as well as studies that replicate the present findings are required to understand how SJL may impact on neural substrates of attention and impulsivity.

Regarding IGT performance, decision making differed as a function of subjective sleep quality, but not chronotype or SJL. These findings are consistent with evidence showing that sleep disruption adversely affects performance on the task (Killgore, Balkin, & Wesensten, 2006). One behavioural component related to better performance on the IGT involves the ability of the participant to delay gratification; impaired IGT performance may occur when individuals are unable to forego larger short-term gains in favour of better but longer-term outcomes. Recent sleep debt and greater daytime sleepiness are also associated with an over-emphasis on short-term outcomes over temporally distant ones (Olson, Weber, Rauch, & Killgore, 2016). Moreover, sleep loss may result in riskier decisions biased towards optimizing gains on behavioural gambling tasks (McKenna, Dickinson, Orff, & Drummond, 2007). Imaging studies have shown that sleep disturbance is implicated in the differential reactivity of subcortical and prefrontal brain structures involved in the anticipation and accumulation of rewards (Gujar, Yoo, Hu, & Walker, 2011; Mullin et al., 2013). Thus, differences in IGT performance may reflect non-optimal choice strategies emerging from neural circuits that are particularly susceptible to sleep disturbance. We find no evidence for impact of chronotype or SJL on IGT performance, a finding that may be in agreement with previous reports that the circadian cycle differentially influences higher cognitive domains (Burke, Scheer, Ronda, Czeisler, & Wright, 2015).

The purpose of the clock gene analysis was to investigate to what degree CLOCK 3111T/C and PER3 VNTR polymorphisms were associated with performance on the CPT and IGT. These polymorphisms were identified a priori as of potential interest, as CLOCK 3111T/C has previously been associated with ADHD symptoms (Kissling et al., 2008) and PER3 VNTR has been associated with planning performance, sleep homeostasis and the interaction of the circadian phase with sleep deprivation on cognition (Archer et al., 2018). Our results indicate no effect of these polymorphisms on performance on the CPT or IGT (as judged by null hypothesis testing followed by equivalence testing). Previous work on the PER3 VNTR has indicated that its effects may be cognitive domain specific, and that executive function may be differentially impacted by PER3 VNTR genotype in relation to the circadian phase and sleep deprivation compared to vigilance (Archer et al., 2018). This may provide an explanation for the lack of effect of PER3 genotype in this study given the relatively high executive load of both the CPT and IGT, and for the lack of interaction between PER3 genotype and subjective sleep quality (Archer et al., 2018). These differences may be also due to mismatches between subjective and objective measures, as well as differences between cross-sectional approaches and experimental sleep manipulation protocols. Clearly, comprehensive genetic characterization will be required to further investigate any impact

of clock genotypes on moderating the effects of SJL on cognitive function, as complex neurocognitive phenotypes are not influenced to a significant degree by single point mutations, and even polygenic scores predict only small amounts of the variation of cognitive performance, chronotype or sleep characteristics (Kalmbach et al., 2017).

In interpreting the current findings, we highlight a number of limitations. The study was cross-sectional and exploratory in design, with groups derived from convenience sampling methods. The directionality of any observed relationship between chronotype/SJL and attention and impulsivity measures cannot be assumed, and causality should not be inferred. Longitudinal studies will be required to investigate if the neurocognitive outcomes described are indeed as a result of SJL. Furthermore, the results described are from a normative young adult sample and it remains to be seen whether similar findings are observed for older or clinical samples. Time of testing was fixed during the day as the purpose of the study was not to detect time-of-day effects or synchrony effects and testing at the more extremes of the day may reveal greater influence of chronotype on performance (e.g., May & Hasher, 1998). We also did not implement control over meal times relative to test time, which may also influence performance.

In conclusion, the findings described here suggest a role for SJL in performance on the CPT, but not the IGT; further, chronotype was not found to impact on performance on either test. These findings have implications for the interpretation of previous studies that have linked individual differences in circadian clock functioning and chronotype with traits such as sensation seeking and impulsiveness, as well as the link between the circadian system and ADHD symptoms.

ACKNOWLEDGEMENTS

We acknowledge scholarship funding to NMcG from The John and Pat Hume scheme of Maynooth University.

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REFERENCES

- Archer, S. N., Schmidt, C., Vandewalle, G., & Dijk, D. J. (2018). Phenotyping of PER3 variants reveals widespread effects on circadian preference, sleep regulation, and health. *Sleep Medicine Reviews*, 40, 109–126. https://doi.org/10.1016/j.smrv.2017.10.008
- Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50, 7–15. https://doi. org/10.1016/0010-0277(94)90018-3
- Bijlenga, D., Vollebregt, M. A., Kooij, J. S., & Arns, M. (2019). The role of the circadian system in the etiology and pathophysiology of ADHD: Time to redefine ADHD? Attention Deficit and Hyperactivity Disorders, 11(1), 5–19. https://doi.org/10.1007/s12402-018-0271-z
- Borbély, A. A., Daan, S., Wirz-Justice, A., & Deboer, T. (2016). The two-process model of sleep regulation: A reappraisal. *Journal of Sleep Research*, 25(2), 131–143. https://doi.org/10.1111/jsr.12371
- Burke, T. M., Scheer, F. A. J. L., Ronda, J. M., Czeisler, C. A., & Wright, K. P. Jr (2015). Sleep inertia, sleep homeostatic and circadian influences



on higher-order cognitive functions. *Journal of Sleep Research*, 4, 364–371. https://doi.org/10.1111/jsr.12291

- Buttgereit, F., Smolen, J. S., Coogan, A. N., & Cajochen, C. (2015). Clocking in chronobiology in rheumatoid arthritis. *Nature Reviews Rheumatology*, 11(6), 349.
- Buysse, D. J., Reynolds, C. F. III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), 193– 213. https://doi.org/10.1016/0165-1781(89)90047-4
- Cohen, J. (1988). Statistical power analysis for the behavioral sciences, 2nd ed.. Hillsdale, NJ: Lawrence Earlbaum Associates.
- Conners, K. C., Epstein, J. N., Angold, A., & Klaric, J. (2003). Continuous performance test performance in a normative epidemiological sample. Journal of Abnormal Child Psychology, 31(5), 555–562.
- Coogan, A. N., & McGowan, N. M. (2017). A systematic review of circadian function, chronotype and chronotherapy in attention deficit hyperactivity disorder. Attention Deficit and Hyperactivity Disorders, 9(3), 129–147. https://doi.org/10.1007/s12402-016-0214-5
- Coogan, A. N., Schenk, M., Palm, D., Uzoni, A., Grube, J., Tsang, A. H., ... Faltraco, F. (2019). Impact of adult attention deficit hyperactivity disorder and medication status on sleep/wake behavior and molecular circadian rhythms. *Neuropsychopharmacology*, 44(7), 1198–1206. https://doi.org/10.1038/s41386-019-0327-6
- Coutinho, J. F., Gonçalves, O. F., Maia, L., Fernandes Vasconcelos, C., Perrone-McGovern, K., Simon-Dack, S., ... Sampaio, A. (2015). Differential activation of the default mode network in jet lagged individuals. *Chronobiol International*, 32(1), 143–149. https://doi. org/10.3109/07420528.2014.955187
- Ebisawa, T., Uchiyama, M., Kajimura, N., Mishima, K., Kamei, Y., Katoh, M., ... Kudo, Y. (2001). Association of structural polymorphisms in the human period3 gene with delayed sleep phase syndrome. *EMBO Reports*, 2(4), 342–346.
- Epstein, J. N., Erkanli, A., Conners, C. K., Klaric, J., Costello, J. E., & Angold, A. (2003). Relations between continuous performance test performance measures and ADHD behaviours. *Journal of Abnormal Child Psychology*, 31(5), 543–554.
- Fischer, D., Lombardi, D. A., Marucci-Wellman, H., & Roenneberg, T. (2017). Chronotypes in the US-influence of age and sex. *PLoS ONE*, 12(6), e0178782. https://doi.org/10.1371/journal.pone.0178782
- Gujar, N., Yoo, S.-S., Hu, P., & Walker, M. P. (2011). Sleep deprivation amplifies reactivity of brain reward networks, biasing the appraisal of positive emotional experiences. *The Journal of Neuroscience*, 31(12), 4466–4474. https://doi.org/10.1523/JNEUR OSCI.3220-10.2011
- Hasler, B. P., Dahl, R. E., Holm, S. M., Jakubcak, J. L., Ryan, N. D., Silk, J. S., Phillips, M. L., ... Forbes, E. E. (2012). Weekend-weekday advances in sleep timing are associated with altered reward-related brain function in healthy adolescents. *Biological Psychology*, *91*(3), 334–341. https://doi.org/10.1016/j.biopsycho.2012.08.008
- Hervey, A. S., Epstein, J. N., Curry, J. F., Tonev, S., Eugene Arnold, L., Keith Conners, C., ... Hechtman, L. (2006). Reaction time distribution analysis of neuropsychological performance in an ADHD sample. *Child Neuropsychology*, 12(2), 125–140. https://doi.org/10.1080/09297 040500499081
- Hoddes, E., Zarcone, V., Smythe, H., Phillips, R., & Dement, W. C. (1973).
 Quantification of sleepiness: A new approach. *Psychophysiology*, 10(4), 431–436. https://doi.org/10.1111/j.1469-8986.1973.tb008 01.x
- Hwang-Gu, S.-L., Gau, S.-S.-F., Tzang, S.-W., & Hsu, W.-Y. (2013). The ex-Gaussian distribution of reaction times in adolescents with attention-deficit/hyperactivity disorder. *Research in Developmental Disabilities*, 34, 3709–3719. https://doi.org/10.1016/j. ridd.2013.07.025
- Kalmbach, D. A., Schneider, L. D., Cheung, J., Bertrand, S. J., Kariharan, T., Pack, A. I., & Gehrman, P. R. (2017). Genetic basis of chronotype

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in humans: Insights from three landmark GWAS. Sleep, 40(2), 1-10. https://doi.org/10.1093/sleep/zsw048

- Killgore, W. D. S., Balkin, T. J., & Wesensten, N. J. (2006). Impaired decision making following 49 h of sleep deprivation. Journal of Sleep Research, 15(1), 7-13. https://doi.org/10.1111/j.1365-2869.2006.00487.x
- Kim, D., Kim, I., Jeong, S., Shin, J., Ahn, J., & Koh, E. (2011). School-based screening of ADHD, LD and comorbid ADHD/LD: The case of an elementary school in Korea. KEDI Journal of Educational Policy, 8(1), 143-165.
- Kissling, C., Retz, W., Wiemann, S., Coogan, A. N., Clement, R. M., Hünnerkopf, R., ... Thome, J. (2008). A polymorphism at the 3'-untranslated region of the CLOCK gene is associated with adult attention-deficit hyperactivity disorder. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 147(3), 333-338. https:// doi.org/10.1002/ajmg.b.30602
- Lacouture, Y., & Cousineau, D. (2008). How to use MATLAB to fit the ex-Gaussian and other probability functions to a distribution of response times. Tutorials in Quantitative Methods for Psychology, 4, 35-45. https://doi.org/10.20982/tqmp.04.1.p035
- Li, S. X., Chan, N. Y., Man Yu, M. W., Lam, S. P., Zhang, J., Yan Chan, J. W., ... Wing, Y. K. (2018). Eveningness chronotype, insomnia symptoms, and emotional and behavioural problems in adolescents. Sleep Medicine, 47, 93-99.
- Martin, J., Hamshere, M. L., Stergiakouli, E., O'Donovan, M. C., & Thapar, A. (2014). Genetic risk for attention-deficit/hyperactivity disorder contributes to neurodevelopmental traits in the general population. Biological Psychiatry, 76(8), 664-671. https://doi.org/10.1016/j.biops ych.2014.02.013
- May, C. P., & Hasher, L. (1998). Synchrony effects in inhibitory control over thought and action. Journal of Experimental Psychology: Human Perception and Performance, 24(2), 363.
- McGowan, N. M., & Coogan, A. N. (2018). Sleep and circadian rhythm function and trait impulsivity: An actigraphy study. Psychiatry Research, 268, 251-256. https://doi.org/10.1016/j.psychres.2018.07.030
- McGowan, N. M., Voinescu, B. I., & Coogan, A. N. (2016). Sleep quality, chronotype and social jetlag differentially associate with symptoms of attention deficit hyperactivity disorder in adults. Chronobiology International, 33(10), 1433-1443. https://doi.org/10.1080/07420 528.2016.1208214
- McKenna, B. S., Dickinson, D. L., Orff, H. J., & Drummond, S. P. (2007). The effects of one night of sleep deprivation on known-risk and ambiguous-risk decisions. Journal of Sleep Research, 16, 245-252. https ://doi.org/10.1111/j.1365-2869.2007.00591.x
- Mueller, S. T., & Piper, B. J. (2014). The psychology experiment building language (PEBL) and PEBL test battery. Journal of Neuroscience Methods, 222, 250-259. https://doi.org/10.1016/j.jneumeth.2013.10.024
- Mullin, B. C., Phillips, M. L., Siegle, G. J., Buysse, D. J., Forbes, E. E., & Franzen, P. L. (2013). Sleep deprivation amplifies striatal activation to monetary reward. Psychological Medicine, 43(10), 2215-2225. https ://doi.org/10.1017/S0033291712002875

- O'Brien, E. M., & Mindell, J. A. (2005). Sleep and risk-taking behavior in adolescents. Behavioral Sleep Medicine, 3(3), 113-133. https://doi. org/10.1207/s15402010bsm0303_1
- Olson, E. A., Weber, M., Rauch, S. L., & Killgore, W. D. (2016). Daytime Sleepiness Is Associated With Reduced Integration of Temporally Distant Outcomes on the Iowa Gambling Task. Behavioral Sleep Medicine, 14(2), 200-11. https://doi.org/10.1080/15402002.2014.974182
- Pinar, A., Hawi, Z., Cummins, T., Johnson, B., Pauper, M., Tong, J., ... Bellgrove, M. A. (2018). Genome-wide association study reveals novel genetic locus associated with intra-individual variability in response time. Translational Psychiatry, 8(1), 207. https://doi. org/10.1038/s41398-018-0262-z
- Roenneberg, T., Kumar, C. J., & Merrow, M. (2007). The human circadian clock entrains to sun time. Current Biology, 17(2), R44-R45. https:// doi.org/10.1016/j.cub.2006.12.011
- Roenneberg, T., Wirz-Justice, A., & Merrow, M. (2003). Life between clocks: Daily temporal patterns of human chronotypes. Journal of Biological Rhythms, 18(1), 80-90. https://doi.org/10.1177/07487 30402239679
- Schmidt, C., Collette, F., Cajochen, C., & Peigneux, P. (2007). A time to think: Circadian rhythms in human cognition. Cognitive Neuropsychology, 24(7), 755-789. https://doi.org/10.1080/02643290701754158
- Song, J., Feng, P., Wu, X., Li, B., Su, Y., Liu, Y., & Zheng, Y. (2019). Individual differences in the neural basis of response inhibition after sleep deprivation are mediated by chronotype. Frontiers in Neurology, 10, 514. https://doi.org/10.3389/fneur.2019.00514
- Tarantino, V., Cutini, S., Mogentale, C., & Bisiacchi, P. S. (2013). Timeon-task in children with ADHD: An ex-Gaussian analysis. Journal of the International Neuropsychological Society, 19(07), 820-828. https ://doi.org/10.1017/S1355617713000623
- Wittmann, M., Dinich, J., Merrow, M., & Roenneberg, T. (2006). Social jetlag: Misalignment of biological and social time. Chronobiology International, 23(1-2), 497-509. https://doi.org/10.1080/07420 520500545979

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: McGowan NM, Uzoni A, Faltraco F, Thome J, Coogan AN. The impact of social jetlag and chronotype on attention, inhibition and decision making in healthy adults. J Sleep Res. 2020;29:e12974. https://doi. org/10.1111/jsr.12974