



Published in final edited form as:

J Biol Rhythms. ; : 7487304261427822. doi:10.1177/07487304261427822.

Overnight Motor Memory Consolidation in Adolescents: Effects of change in dim light melatonin onset after sleep restriction

Lindsay Stager, PhD^{1,2,3}, Caroline Gredvig-Ardito, BA^{1,3}, Stephanie Crowley, PhD⁴, Ronald Seifer, PhD⁵, Mary Carskadon, PhD^{1,3}, Jared Saletin, PhD^{1,3}

¹Department of Psychiatry and Human Behavior, Alpert Medical School of Brown University, Providence, RI, USA

²The Miriam Hospital, Providence, RI, USA

³E.P. Bradley Hospital Sleep Research Laboratory and COBRE Center for Sleep and Circadian Rhythms in Child and Adolescent Mental Health, Providence, RI, USA

⁴Department of Psychiatry and Behavioral Sciences, Rush University, Chicago, IL, USA

⁵Frank Porter Graham Child Development Institute, The University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Abstract

Introduction: Adolescents experience chronic sleep restriction and developmental changes in circadian biology. Sleep aids adolescent learning and memory; the moderating effect of circadian rhythms is largely unknown. Here we examine adolescent sleep restriction, circadian biology, and memory consolidation.

Methods: Adolescents were recruited for a larger experimental study. The present study includes a subsample of individuals from the larger study who completed the motor sequence task (MST; added toward the end of data collection). Participants ($M_{AGE}=12.7$, $SD=1.8$; 62.5% male) completed a self-selected 9-hour in bed sleep stabilization schedule for 19 nights followed by 7 nights of sleep restriction (6 hours in bed; bedtime delayed and risetime advanced equally). In-lab dim-light-melatonin-onset (DLMO) was assessed on the final nights of stabilization and restriction. The MST indexed overnight memory consolidation across the final night of sleep restriction. MST outcomes included the average number of correct sequences per trial, # of errors, and precision. We examined overnight improvement (morning-evening) in MST performance and associations between improvement, phase preference, $DLMO_{Stabilization}$, $DLMO_{Restriction}$, and $DLMO_{Shift}$ ($DLMO_{Stabilization} - DLMO_{Restriction}$), controlling for age where statistically justified.

Results: The average number of correct sequences per MST trial improved ($t(15)=-3.44$, $p<.01$, $d=0.86$) for the morning (12.94 ± 6.89) test session compared to evening (10.81 ± 5.69). There were no changes in errors or precision ($ds<.14$, $ps>.34$). Greater delays in DLMO phase (mean \pm sd: 10.34 ± 41.69 minutes) were associated with greater overnight improvement in the average number of correct sequences per trial (Adj. $R^2=0.54$, $F(2,13)=9.79$, $p<.01$) and errors (Adj. $R^2=0.21$,

$F(1,15)=4.94, p<.05$). Overnight improvement was not related to phase preference (Adj. $R^2_s<0.17$; $p>.05$).

Conclusion: These data highlight context dependent benefits of sleep for adolescent memory consolidation and indicate a potential link between circadian biology and the cognitive benefits of adolescent sleep. Understanding the influence of circadian rhythms in sleep-dependent memory may inform discussions of adolescent sleep loss and learning.

Keywords

Dim Light Melatonin Onset; Motor Memory; Adolescent; Sleep Restriction; Phase Preference

Introduction

Adolescence is characterized by changes in sleep and circadian biology¹ and significant demands for learning and memory. Over 75% of U.S. adolescents report insufficient sleep,² often due to oppositional biological and psychosocial influences. During adolescence, circadian phase is delayed, as evidenced by later dim light melatonin onset (DLMO), and sleep pressure accumulation across the waking day is slowed. These phenomena drive later bedtimes.¹ Psychosocial factors (e.g., increased technology access) exacerbate the effect.¹ Early school start times prohibit equally delayed wake times, short-changing weekday sleep.¹ Adolescent short sleep has numerous negative health impacts including impaired learning and memory,³ and decreased academic success.⁴

The influence of circadian rhythms on sleep-dependent learning and memory is under researched but may significantly influence outcomes, particularly among adolescents given circadian misalignment is greatest at this developmental stage. While research routinely identifies sleep-dependent gains in procedural memory (i.e., on the motor sequence task [MST]) following adult sleep,^{6–8} this finding is not always replicated in adolescents.^{9,10} It has been suggested that ceiling effects in adolescent motor abilities drive this discrepancy; adolescents with worse performance during the learning phase demonstrate greater overnight improvements compared to adolescents with better initial task performance.¹⁰ If this is the case, both initial learning and overnight motor memory consolidation may be influenced by adolescents having better cognitive performance during the times of day most optimal for their chronotype.¹¹

The present study examines overnight memory consolidation, and circadian biology in adolescents undergoing subtle sleep restriction comparable to a school-week. We hypothesized that: 1) sleep restriction would not alter the timing of adolescent DLMO when the sleep midpoint was held constant; 2) eveningness would be positively associated with evening post-training performance as adolescents with evening chronotypes would be learning at the time of day most optimal for their chronotype; 3) adolescents with morning chronotypes would have greater overnight motor sequence memory consolidation given increased room for improvement before ceiling effects in adolescent MST performance.

Methods

Participants

This study was approved by Bradley Hospital's Institutional Review Board. Participants were recruited as part of a larger study conducted in Providence, RI. Eligibility was assessed through telephone interviews and subsequent in-lab participant and caregiver questionnaires. Inclusion criteria included ages 10–15 years, English language proficiency, and good health. Exclusion criteria included personal or first-degree family history of disordered sleep, significant mental illness (e.g., suicidality, bipolar disorder), neurologic illness, metabolic disorders, chronic medical conditions, use of any prescribed psychoactive agents or other drugs that affect the sleep/wake cycle, daytime sleepiness/alertness, or the circadian timing system, inability to participate in testing, and travel beyond 2 time zones within 6 months of in-lab assessments.

Procedures

During the school year, participants completed 19 nights of sleep stabilization (Figure 1; 9-hours in bed; bedtime [mean±sd]: 20:55±21 minutes; waketime: 6:47±29 minutes) followed by 7 nights of restricted sleep (6 hours in bed; bedtime delayed and risetime advanced equally; bedtime: 22:19±30 minutes; waketime: 5:11±34 minutes). Participants achieved at least 2 weeks of stabilization and 1 week of restriction while using consecutive weekends for data collection with no gap between conditions. Wake times were self-selected with consideration of school start times. In-lab polysomnography monitored the final night of stabilization and the first and last nights of restriction. At-home actigraphy (Mini Motionlogger, Ambulatory Monitoring, Inc., Ardsley, NY) and sleep diaries monitored all other study nights. In the laboratory, illuminance was maintained at 15 photopic lux during waking activities and 0 photopic lux during scheduled sleep episodes. Light exposure during at-home portions of the study varied among participants and was not measured. In-lab DLMO assessment occurred after the final nights of sleep stabilization and restriction. Participants completed the MST before and after the final night of restricted sleep.

Measures

Demographics. Caregiver questionnaires assessed child age, sex, race, and ethnicity.

Morningness/Eveningness. Adolescents completed the Morningness/Eveningness Scale¹² assessing circadian phase preference.

DLMO. To determine DLMO_{Stabilization} and DLMO_{Restriction}, serial saliva samples (~2ml collected every 30 minutes in 15 photopic lux) were collected using the salivette system (Sarstedt, Newton, NC, USA) across a 6-hour window, beginning ~4 hours prior to a participant's stabilization bedtime regardless of condition. Participants chewed on a plain cotton cylinder that was then centrifuged for two minutes. Saliva was frozen at -20° C within 4 hours. AlpcO (Windham, NH) assay kits were used for radioimmunoassay. DLMO was defined as the time when salivary melatonin concentration surpassed 4 pg/ml as is standard for pediatric populations.^{13–15} DLMO_{Shift} was calculated as DLMO_{Stabilization}

-DLMO_{Restriction}. Consistent with circadian conventions, a positive DLMO_{Shift} indicates a phase advance and negative DLMO_{Shift} indicates a phase delay.

Sunrise/Sunset. Timing of sunrise and sunset during the participant's sleep schedule was assessed retrospectively.¹⁶

Overnight Memory Consolidation. The MST¹⁷ indexed overnight memory consolidation across the final sleep restriction night. Participants repeatedly typed a five number sequence with their nondominant hand for 30 seconds (1 trial). A 30 second rest interval followed before the next trial (12 total). Outcomes included the average number of correct sequences per trial, errors (average number of mistakes per trial), and precision (average [total expected characters/total characters] per trial).¹⁰ Scores were calculated using the last 2 trials of evening training and the first two trials of the morning test to control for learning effects. Evening task administration occurred ~2 hours prior to lights out; morning task administration occurred within 3 hours of the sleep period (M=168 minutes, SD=34). The phase angle from DLMO_{Restricted} to evening MST was calculated to probe its effect on the observed outcomes.

Data Analyses

Analyses were performed using SPSS version 29. Paired samples t-tests assessed differences in MST outcomes (average number of correct sequences per trial, errors, and precision) during evening post-training and morning test sessions. Linear regressions assessed the associations among 1) phase preference, 2) DLMO_{Stabilization} 3) DLMO_{Restricted}, and 4) DLMO_{Shift}, and MST 1) evening post-training performance, 2) morning test performance, and 3) overnight performance change. Bivariate correlations assessed associations between DLMO_{Shift} and MST evening post-training performance, MST morning test performance, phase preference, demographics, and sunrise/sunset during sleep restriction, and associations between the phase angle of DLMO_{Restricted} to evening MST and MST motor memory consolidation. Statistical assumptions of tests were assessed; statistical significance was defined as $p < .05$. Regressions controlled for demographic variables only where statistically justified (i.e., when demographics were significantly associated with the outcome variable of interest) to preserve power.

Results

Circadian characterization: phase preference and dim-light-melatonin-onset [DLMO]

Forty-one participants enrolled in the larger study from 2004–2005. Only N=16 completed the MST (added later in the study; Table 1). There were no significant differences in demographics or sleep between samples.

Overnight change in MST performance

The average number of correct sequences per MST trial improved ($t(15)=-3.44$, $p < .01$, $d=0.86$ [large effect]; change= 2.1 ± 2.5) for the morning test (12.9 ± 6.9) compared to evening post-training (10.8 ± 5.7 ; Figure 2). No overnight changes in errors ($t(15)=-1.00$, $p=0.34$, $d=0.25$) or precision ($t(15)=-0.58$, $p=0.57$, $d=0.14$) were detected.

Association between circadian variables and overnight MST improvement

Later DLMO_{Shift} related to greater overnight MST improvement for the average number of correct sequences per trial ($\beta=-0.41$ [medium effect], $F(2,13)=9.79$, $p<0.01$) and errors ($\beta=0.51$ [large effect], $F(1,15)=4.94$, $p<0.05$; Figure 3). MST outcomes were not associated with phase preference (Table 2; average number of correct sequences per trial: $\beta=-0.12$, $p=0.60$; errors: $\beta=0.47$, $p=0.07$; precision: $\beta=-0.25$, $p=0.35$), DLMO_{Stabilization} (average number of correct sequences per trial: $\beta=-0.18$, $p=0.42$; errors: $\beta=0.03$, $p=0.91$; precision: $\beta=-0.08$, $p=0.78$), or DLMO_{Restricted} (average number of correct sequences per trial: $\beta=0.20$, $p=0.38$; errors: $\beta=-0.39$, $p=0.13$; precision: $\beta=0.13$, $p=0.64$).

Sensitivity analyses sought to contextualize the association between DLMO_{Shift} and MST improvement. DLMO_{Shift} was not related to MST evening post-training performance (average number of correct sequences per trial: $\beta=-0.03$, $p=0.93$; errors: $\beta=-0.05$, $p=0.85$; precision: $\beta=-0.05$, $p=0.86$), morning test performance (average number of correct sequences per trial: $\beta=-0.16$, $p=0.55$; errors: $\beta=0.25$, $p=0.36$; precision: $\beta=-0.29$, $p=0.28$), phase preference ($\beta=-0.09$, $p=0.75$), DLMO_{Restricted} ($\beta=-0.39$, $p=0.13$), demographic characteristics (age: $\beta=0.02$, $p=0.93$; sex: $t(5.45)=0.96$, $p=0.38$, $d=0.63$), or sleep restriction sunrise/sunset (sunrise: $\beta=0.33$, $p=0.22$; sunset: $\beta=-0.39$, $p=0.13$). The phase angle of DLMO_{Restricted} to evening MST was not related to MST improvement (average number of correct sequences per trial: $\beta=0.08$, $p=0.95$; errors: $\beta=-0.04$, $p=0.26$; precision: $\beta=0.01$, $p=0.55$).

Discussion

These data demonstrate the associations between adolescent sleep-dependent motor memory consolidation and circadian timing in the context of sleep restriction. The average number of correct sequences per trial improved for the MST morning test session compared to post-training evening performance. Greater delays in DLMO phase were associated with greater overnight MST improvement in the context of sleep restriction. Phase preference was not related to memory consolidation. DLMO_{Shift} did not relate to any variables of interest within our sample.

The present findings indicate a positive effect of adolescent sleep on the average number of correct sequences per MST trial in the context of sleep restriction, suggesting an overall improvement in signal-to-noise ratio and subtle improvement in task performance. This finding differs from past findings showing no impact of sleep on motor memory consolidation in adolescents^{9,10} and may reflect the unique study context, where evening learning and subsequent testing occurred after several nights of restricted sleep. Ceiling effects may obscure sleep-dependent MST improvement among rested adolescents.¹⁸ Past research investigating sleep-dependent MST improvement among 10–13-year-olds with and without ADHD demonstrated that, while individuals with ADHD had initially reduced performance compared with controls, they achieved equivalent levels of performance following sleep. Individuals without ADHD showed no significant post-sleep changes in performance.¹⁵ If sleep-restriction created initial impairments in MST evening performance for our sample, this may have allowed for overnight improvement in the context of ceiling effects.

The present study is also the first to identify a positive association between shifts to later DLMO and adolescent overnight motor memory consolidation. Importantly, as the offset of dim light melatonin was not assessed, we cannot say whether the entire melatonin secretion episode shifted, or if it was constricted. However, prior research suggests that evening light exposure causes shifts in melatonin secretion¹⁹ and demonstrates null effects of restricted sleep on the length of adult melatonin secretion.^{20,21} While shifts in DLMO were not expected when the midpoint of sleep was maintained, this outcome may relate to differences in adolescent light exposure at home and sensitivity. As evening light can shift circadian phase,²² individuals with greater light exposure at home, and/or light sensitivity, may have experienced larger DLMO shifts to align with their restricted sleep schedule, thus mitigating some of the negative effects of circadian misalignment during the learning process in the evening.²² This may partly explain why adolescents with shifts to a later DLMO displayed improved motor memory consolidation. Given the sample size of the present study and null results of follow-up tests to contextualize this effect, this finding requires replication and further mechanistic probes.

This study had many strengths including objective assessment of sleep, memory consolidation, and DLMO, and completion of sleep conditions in a controlled environment prior to morning MST administration. It is the first to assess sleep-dependent motor memory consolidation in the context of sleep restriction, increasing the study's ecological validity, as ~75% of adolescents experience chronically short sleep.²

The study's limitations include a small sample in which only one participant had an eveningness chronotype. The results need validation in larger samples with more variation in circadian profiles. Data were collected in 2004 and 2005, and cohort differences (i.e., technology use) may emerge compared to more contemporary samples. These differences, if existing, should minimally impact the within-subjects analyses of the current work. While light exposure was controlled during the in-lab protocol, light exposure during the at-home portion of the protocol was not controlled and likely impacted DLMO stability. The context of sleep restriction increases ecological validity for adolescents;² however, future studies should compare the same protocol to adequate sleep for a greater understanding of the interactive effects of circadian factors and sleep duration on adolescent memory consolidation.

Overall, these data indicate that sleep-dependent adolescent motor memory consolidation may be most evident when initial evening performance is challenged and reveal a potential link between circadian biology and the cognitive benefits of adolescent sleep. Understanding the influence of circadian rhythms in sleep-dependent memory consolidation informs discussions of how sleep loss affects adolescent learning. Future studies should assess the roles of circadian biology and restricted sleep in other forms of memory (e.g., declarative). Leveraging adolescent circadian biology may ultimately help to protect youth from the negative cognitive effects of short sleep.

Acknowledgements

Thank you to the families of the greater Providence community and the Bradley Sleep Lab Team. This project was supported by funding from the NIH (R01 NR08381 and 1P20GM139743-01). Lindsay Stager received funding

from NIH's Cardiovascular Behavioral Health T32 (T32 HL076134) during conduct of this research. The content is solely the responsibility of the authors and does not necessarily represent the official views of the funding agencies.

References

1. Carskadon MA. Sleep in adolescents: The perfect storm. *Pediatr Clin North Am.* 2011;58(3):637–47. doi:10.1016/j.pcl.2011.03.003 [PubMed: 21600346]
2. Centers for Disease Control and Prevention. FastStats: Sleep in high school students. Updated May 15, 2024. Accessed September 19, 2025. <https://www.cdc.gov/sleep/data-research/facts-stats/high-school-students-sleep-facts-and-stats.html>
3. Doyon J, Korman M, Morin A, et al. Contribution of night and day sleep vs. simple passage of time to the consolidation of motor sequence and visuomotor adaptation learning. *Exp Brain Res.* 2009;195(1):15–26. doi:10.1007/s00221-009-1748-y [PubMed: 19277618]
4. Titz C, Karbach J. Working memory and executive functions: Effects of training on academic achievement. *Psychol Res.* 2014;78(6):852–868. doi:10.1007/s00426-013-0537-1 [PubMed: 24389706]
5. Roenneberg T, Allebrandt KV, Meroow M, Vetter C. Social jetlag and obesity. *Curr Biol.* 2012;22(10):939–943. doi:10.1016/j.cub.2012.03.038 [PubMed: 22578422]
6. Walker MP, Brakefield T, Morgan A, Hobson JA, Stickgold R. Practice with sleep makes perfect: Sleep-dependent motor skill learning. *Neuron.* 2002;35(1):205–211. doi:10.1016/S0896-6273(02)00746-8 [PubMed: 12123620]
7. Maquet P, Schwartz S, Passingham R, Frith C. Sleep-related consolidation of a visuomotor skill: Brain mechanisms as assessed by functional magnetic resonance imaging. *J Neurosci.* 2003;23(4):1432–40. doi:10.1523/jneurosci.23-04-01432.2003 [PubMed: 12598632]
8. Walker MP, Stickgold R, Alsop D, Gaab N, Schlaug G. Sleep-dependent motor memory plasticity in the human brain. *Neuroscience.* 2005;133(4):911–7. doi:10.1016/j.neuroscience.2005.04.007 [PubMed: 15964485]
9. Reyes S, Algarín C, Lozoff B, Peigneux P, Peirano P. Sleep and motor sequence learning consolidation in former iron deficient anemic adolescents. *Sleep Med.* 2019;64:116–122. doi:10.1016/j.sleep.2019.05.023 [PubMed: 31704427]
10. Saletin JM, Coon WG, Carskadon MA. Stage 2 Sleep EEG sigma activity and motor learning in childhood ADHD: A pilot study. *J Clin Child Adolesc Psychol.* 2017;46(2):188–197. doi:10.1080/15374416.2016.1157756 [PubMed: 27267670]
11. Videira VF, Booth JN, Saunders DH, Sproule J, Turner AP. Circadian preference and physical and cognitive performance in adolescence: A scoping review. *Chronobiol Int.* 2023;40(9):1296–1331. doi:10.1080/07420528.2023.2256901 [PubMed: 37781788]
12. Carskadon MA, Vieira C, Acebo C. Association between puberty and delayed phase preference. *Sleep.* 1993;16(3):258–62. doi:10.1093/sleep/16.3.258 [PubMed: 8506460]
13. Crowley SJ, Acebo C, Fallone G, Carskadon MA. Estimating dim light melatonin onset (DLMO) phase in adolescents using summer or school-year sleep/wake schedules. *Sleep.* 2006;29(12):1632–1641. doi:10.1093/sleep/29.12.1632 [PubMed: 17252895]
14. Carskadon MA, Acebo C, Richardson GS, Tate BA, Seifer R. An approach to studying circadian rhythms of adolescent humans. *J Biol Rhythms.* 1997;12(3):278–89. doi:10.1177/074873049701200309 [PubMed: 9181439]
15. Crowley SJ, Suh C, Molina TA, Fogg LF, Sharkey KM, Carskadon MA. Estimating the dim light melatonin onset of adolescents within a 6-h sampling window: The impact of sampling rate and threshold method. *Sleep Med.* 2016;20:59–66. doi:10.1016/j.sleep.2015.11.019 [PubMed: 27318227]
16. Sunrise Sunset. Accessed December 2023, <https://sunrise-sunset.org/us/providence-ri>
17. Walker MP, Stickgold R. Sleep, Memory, and Plasticity. *Annu Rev Psychol.* 2006;57:139–166. doi:10.1146/annurev.psych.56.091103.070307 [PubMed: 16318592]
18. Wilhelm I, Metzkw-Mészáros M, Knapp S, Born J. Sleep-dependent consolidation of procedural motor memories in children and adults: The pre-sleep level of performance matters. *Dev Sci.* 2012;15(4):506–515. doi:10.1111/j.1467-7687.2012.01146.x [PubMed: 22709400]

19. Wright KP Jr., McHill AW, Birks BR, Griffin BR, Rusterholz T, Chinoy ED. Entrainment of the human circadian clock to the natural light-dark cycle. *Curr Biol*. 2013;23(16):1554–8. doi:10.1016/j.cub.2013.06.039 [PubMed: 23910656]
20. Rogers NL, Dinges DF. Interaction of chronic sleep restriction and circadian system in humans. *J Sleep Res*. 2008;17(4):406–11. doi:10.1111/j.1365-2869.2008.00681.x [PubMed: 19090952]
21. Möller-Levet CS, Archer SN, Bucca G, et al. Effects of insufficient sleep on circadian rhythmicity and expression amplitude of the human blood transcriptome. *Proc Natl Acad Sci U S A*. 2013;110(12):E1132–41. doi:10.1073/pnas.1217154110 [PubMed: 23440187]
22. Crowley SJ, Eastman CI. Human adolescent phase response curves to bright white light. *J Biol Rhythms*. 2017;32(4):334–344. doi:10.1177/0748730417713423 [PubMed: 28651468]

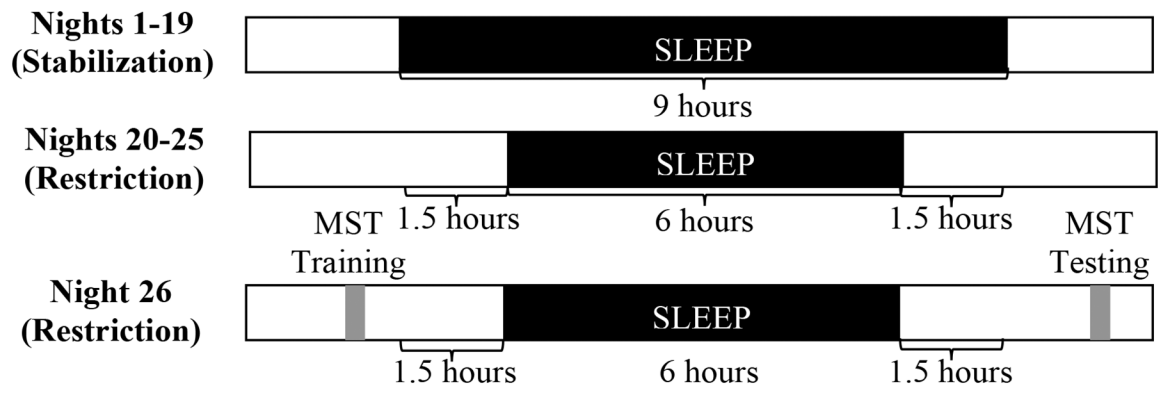


Figure 1.
 Participant Sleep and Motor Sequence Task (MST) Schedule

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

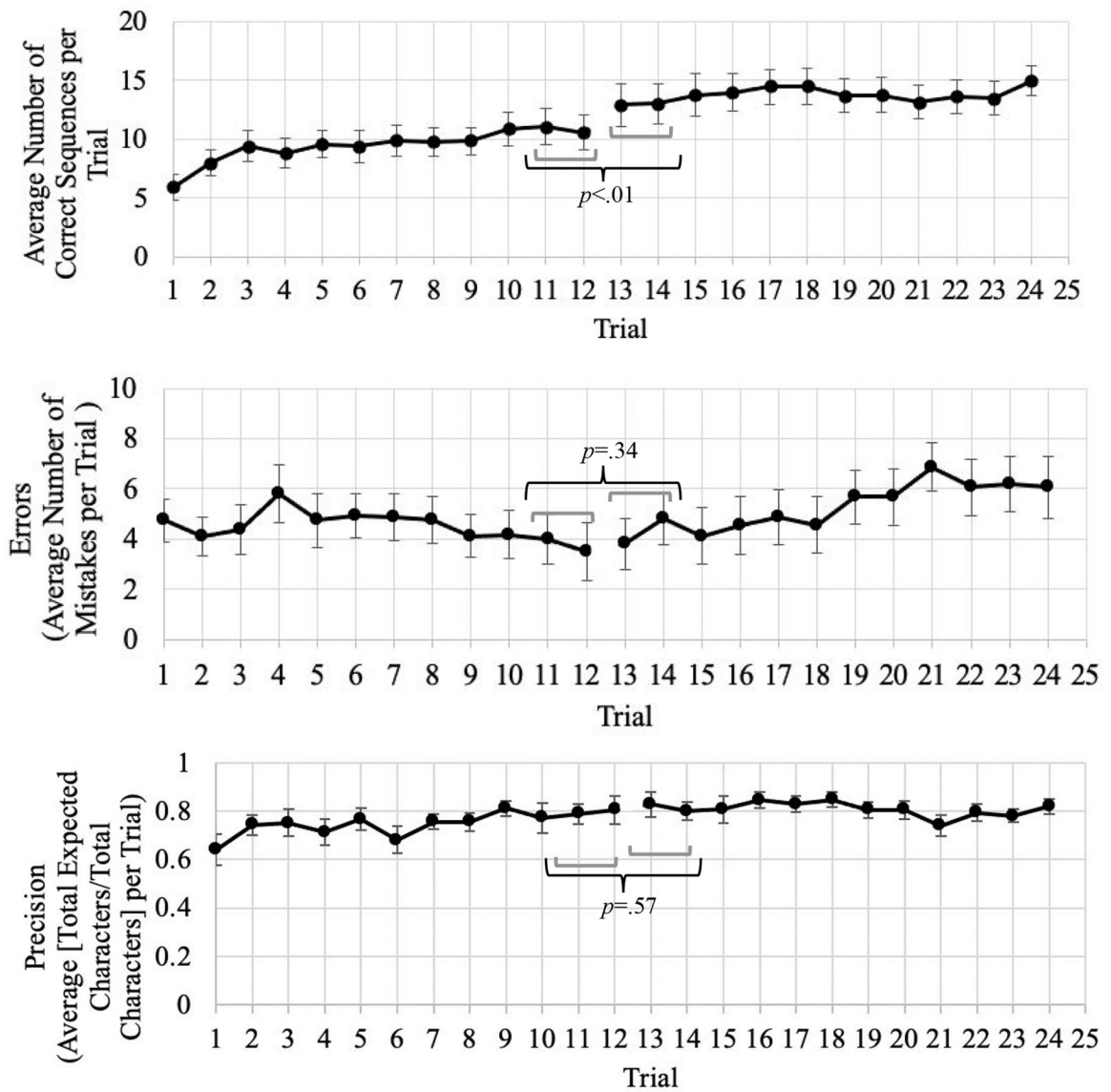


Figure 2.
Motor Sequence Task (MST) Outcomes across Trials

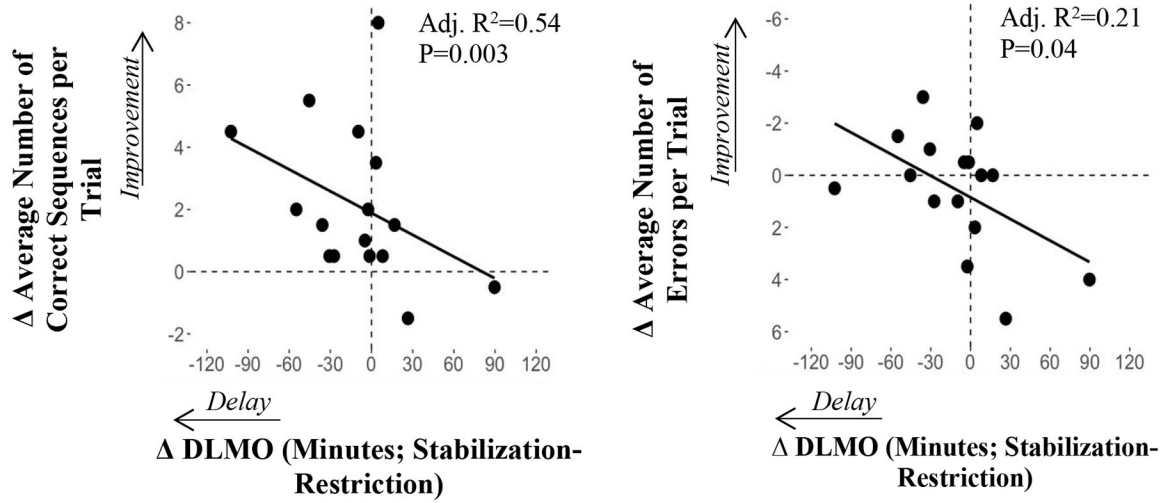


Figure 3.
Relationship between Change in DLMO and MST Performance

Table 1.

Participant Demographics

Analytic Sample (N=16)	
Age M(SD)	12.7 (1.8)
Female	6 (37.5)
Race	
<i>White</i>	14 (87.5)
<i>Non-White</i>	2 (12.5)
Morningness/Eveningness	
<i>Score Range</i>	22 – 40
<i>Morning Type</i>	11 (68.75)
<i>Neither Type</i>	4 (25)
<i>Evening Type</i>	1 (6.25)

Note: all demographic values listed as N (%) unless otherwise specified. The Motor Sequence Task was added late in the study and only completed by a subset of participants, the analytic subsample for this study.

Table 2.
Effects of Morningness/Eveningness, Change in Dim Light Melatonin Onset (DLMO), and Restricted DLMO on Memory Consolidation

	N	df	Adj. R ²	F	p	β
Morningness/Eveningness						
Average Number of Correct Sequences per Trial	16	2,13	0.36	5.23	0.02	
<i>Morningness/Eveningness</i>					0.60	-0.12
<i>Age</i>					0.01	3.22
Errors	16	1,14	0.16	3.92	0.07	
<i>Morningness/Eveningness</i>					0.07	0.47
Precision	16	1,14	-0.00	0.93	0.35	
<i>Morningness/Eveningness</i>					0.35	-0.25
Change in DLMO Phase						
Average Number of Correct Sequences per Trial	16	2,13	0.54	9.79	0.003	
<i>DLMO Phase</i>					0.04	-0.41
<i>Age</i>					0.002	0.67
Errors	16	1,14	0.21	4.94	0.04	
<i>DLMO Phase</i>					0.04	0.51
Precision	16	1,14	-0.01	0.91	0.36	
<i>DLMO Phase</i>					0.36	-0.25
Stabilization DLMO						
Average Number of Correct Sequences per Trial	16	2,13	0.38	5.57	0.02	
<i>Stabilization DLMO</i>					0.42	-0.18
<i>Age</i>					0.01	0.61
Errors	16	1,14	-0.07	0.01	0.91	
<i>Stabilization DLMO</i>					0.91	0.03
Precision	16	1,14	-0.07	0.08	0.78	
<i>Stabilization DLMO</i>					0.78	-0.08
Restricted DLMO						
Average Number of Correct Sequences per Trial	16	2,13	0.39	5.71	0.02	
<i>Restricted DLMO</i>					0.38	0.20
<i>Age</i>					0.01	0.72
Errors	16	1,14	0.10	2.57	0.13	
<i>Restricted DLMO</i>					0.13	-0.39
Precision	16	1,14	-0.05	0.24	0.64	
<i>Restricted DLMO</i>					0.64	0.13

Note: Age was only included as a covariate in models when significantly associated with the outcome variable of interest to preserve power.