

# Adolescents self-reported sleep quality and emotional regulation: a discordant twin study

Emanuela Medda<sup>1</sup>, Guido Alessandri<sup>2</sup>, Davide Delfino<sup>1</sup>, Corrado Fagnani<sup>1</sup>, Maurizio Ferri<sup>1</sup>, Cristiano Violani<sup>2</sup> and Maria Antonietta Stazi<sup>1</sup>

<sup>1</sup>Registro Nazionale Gemelli, Centro di Riferimento per le Scienze Comportamentali e la Salute Mentale, Istituto Superiore di Sanità, Rome, Italy

<sup>2</sup>Dipartimento di Psicologia, Sapienza Università di Roma, Rome, Italy

## Abstract

**Aim.** This study explores the association between sleep quality and emotional regulation, and investigates the genetic and environmental bases of this association.

**Methods.** Three-hundred-eighty-two adolescent twins, from the Italian Twin Registry, and their parents filled the Youth Self-Report and Child Behavior Checklist questionnaires, from which the construct of Effortful Control (EC) was derived as a measure of emotional regulation. Twins were identified as "good" or "non-good" sleepers based on answers to the Sleep Disorders Questionnaire. EC levels were compared between same-sex sleep discordant twins.

**Results.** A significant association was detected between EC scores and sleep quality. When controlling for shared (fetal or early life) environmental factors and genetic background in the discordant twin analysis, this association weakened in dizygotic twins and disappeared in monozygotic twins.

**Conclusion.** Results support the association between sleep quality and EC in adolescence; furthermore, they suggest that sleep quality and emotional regulation may depend on common genetic or environmental factors.

## Key words

- twin
- sleep quality
- effortful control
- self-regulation

## INTRODUCTION

During the transition from childhood to adolescence, we observe dramatic changes in sleep patterns, which play an important role in growth and maturation, and in mental and physical health [1]. Adolescents, compared with children, generally start to feel sleepy later as a result of changes in the phase of circadian wake-sleep rhythms and of an enhanced capacity to sustain prolonged wakefulness [2]. During adolescence, the sleep need doesn't seem to decline while slow wave sleep (the homeostatic restorative component of sleep) decreases by approximately 40% relative to REM sleep [2]; so, to get the same amount of restorative sleep, adolescents' sleep duration should be longer compared with younger children's sleep. On the contrary, due to biological and psychosocial factors, it becomes shorter and of lower quality.

Investigating the determinants of changes in sleep duration and quality during adolescence is important, because poor sleep in adolescence may have detrimental effects on brain development due to the role of sleep in synaptic homeostasis, brain plasticity, and brain mat-

uration. Indeed, several studies found associations between poor sleep and daytime sleepiness, maladaptive conducts, internalizing and externalizing symptoms, and poor school performance [3].

There is evidence from twin studies that genetic factors play a role in adolescents' sleep quantity and quality. For example, Te Velde *et al.* showed, in a Dutch adolescent twin sample, that sleep duration was influenced at the age 12 by genetic, shared environmental, and non-shared environmental factors with similar relative contributions, while at the age 20 it was influenced by non-shared environmental and genetic factors [4]. Another twin study in adolescence found a significant genetic influence on sleep problems [5].

Sleep changes in adolescence are affected by normative biological changes, by environmental factors, as well as by experience and temperament. Around the time of puberty a delay in circadian phase has been observed in humans and other mammals. The delay has been measured on behavioral daily rhythm (sleep and activity) as well as on physiological rhythms (metabolism and endocrinology). Gonadal hormones influence

both the sleep-wake homeostasis and the circadian rhythm regulation of sleep [6]. In teenagers this is exacerbated by psycho-social and behavioral changes, such as the social media exposure and the use/abuse of caffeine and caffeine-laden products [7].

The relationship between sleep and temperament has been extensively studied in infants, toddlers and school age children. Fewer are the studies among adolescents. In a review paper, Willis [8] reports a robust association between sleep and anxiety in adolescents. In a longitudinal study of young adult twins, Alice Gregory and her team showed that while genetic effects play a modest role in insomnia and depression symptoms separately, they appear to play a more central role in concurrent and longitudinal associations between these phenotypes [9]. Higher neuroticism was reported for adolescent poor sleepers compared with good sleepers [10], and for junior high school students with later bedtimes, shorter sleep duration, more sleep problems and daytime sleepiness. According to Matthews [11], poor sleep quality in young adults is robustly associated with loneliness. A relatively recent study showed positive correlations of sleep problems with negative affectivity and sociability, and a negative correlation with effortful control [12]. Similar findings were reported by Lukowski and Milojevich [13] in a sample of university students; in their study, a poor global quality of sleeping was unrelated to extraversion, positively correlated to negative affect (sadness) and to orienting sensitivity (associative), and negatively correlated with effortful control.

Self-regulation represents a broad construct entailing attentional, cognitive, physiological, and behavioral processes that operate in concert to ensure an appropriate level of emotional, motivational, and cognitive arousal [14]. From a developmental perspective, researchers often investigate self-regulation using measures of effortful control [15]. The construct of effortful control (EC) reflects the temperamentally-based component of emotion-relevant self-regulation, and captures a set of control functions needed for voluntary and goal-directed behavior [16]. In terms of development, EC represents an early appearing component of child temperament [16]. Children high in EC reveal better social and emotional competence [17], are less likely to develop internalizing and externalizing symptoms, and show better school performances [18] compared to children with low EC. Furthermore, EC has been associated with indicators of psychological adjustment, such as optimism, self-esteem and happiness [19].

In a recent study conducted on the same sample, we showed substantial genetic influences on the EC measure [19]. Moreover, other previous twin studies found a substantial genetic component for the same measure in children [20] and in young adults [21].

Although the correlations between sleep quality and EC, found by Lukowski and Milojevich [13], could be due, at least in part, to shared genetic or environmental factors, no previous studies tested this hypothesis. Therefore, considering an adolescent sample from the Italian Twin Registry, we aimed to replicate the association between EC and sleep quality, and to explore

the role of genetic and environmental factors in this association.

## METHODS

### *Participants*

Study subjects were twins aged 14-18 years previously enrolled in the Italian Twin Registry (ITR) [22]. Of about 1600 families contacted by mail, 389 families having a twin pair among their offspring agreed to participate filling in the CBCL/YSR questionnaires. The sample included 774 twins (385 complete pairs, 4 unmatched twins) of Caucasian origin. Of these, 281 (36.3%) were monozygotic (MZ) and 493 (63.7%) were dizygotic (DZ). Twins from respondent and non-respondent families were similar for gender, zygosity and age, as well as for age of the parents, whereas parents of participating twins were significantly more educated than parents in non-participating families.

Subsequently, following a new study hypothesis, a random sub-sample of the 389 families was administered the questionnaire to assess sleep quality, and 191 families sent it back.

Parents of twins received at home an invitation letter explaining the purpose of the study, an informed consent form to be signed and a number of questionnaires regarding the traits of interest to be filled by parents and/or twins. The protocol of this observational study was under the general framework of the ITR research activities, approved by the Ethics Committee of the Istituto Superiore di Sanità (ISS, Italian National Institute of Health).

### *Zygosity assessment*

Zygosity of twin pairs was assessed by a standardized self-report questionnaire, which consists of items, filled by parents, about physical similarity and frequency of confusion of the twins by family members and strangers during infancy. This is a well-known method in twin studies that has been shown to be over 95% accurate [23].

### *Measures*

We collected behavioral information through the Child Behavior Checklist/6-18 (CBCL) and Youth Self-Report/11-18 (YSR) questionnaires [24].

The CBCL is a standardized assessment of behavior problems based on responses to 113 items that are scored 0 ("not true"), 1 ("somewhat true"), or 2 ("very true") in describing the child's behavior by parents. The YSR is a standardized assessment tool widely used to report behavioral problems and social competence among adolescents; twins self-responded to the 118 test items, scored as in the CBCL. In the analyses, we simultaneously exploited the information from parents (CBCL) and adolescents (YSR) (see below, Statistical Analyses).

To calculate the variable "effortful control" (EC), we used 8 items from the 9-item measure (one item was excluded because it showed poor psychometric properties in preliminary analyses) introduced by Luengo-Kanacri *et al.* [25]. This 8-item subscale of the CBCL and YSR captures the construct of EC in the subdimensions of attentional focusing (three items) and inhibitory con-

control (five items). This measure assesses processes related to emotional regulation, as well as the abilities to voluntarily focus and shift attention, to voluntarily inhibit or initiate behaviors such as delaying and planning. Sample items are: "He/she does not finish things he/she started", "He/she also talks when it is not his/her turn", and "His/her demands must be fulfilled immediately, easily frustrated". The items were reversed as appropriate: high scores indicated high EC. The reliability for the parent-version of the measure (CBCL) was 0.82 and for the YSR was 0.74. For the 8-item version of the EC scale, the reliability was 0.74, while it was 0.71 and 0.75 for inhibitory control and attentional focusing, respectively.

For the purposes of the analyses, a composite EC score was calculated based on CBCL and YSR (see below, Statistical Analyses).

Sleep quality was assessed by the Sleep Disorders Questionnaire, a brief self-report insomnia questionnaire including 18 yes/no questions on different sleep problems and 5 further questions concerning sleep habits [26]. The questionnaire was built according to DSM-IV and ICSD-R criteria for sleep problems; both these classifications assign a crucial role to the subjective complaint of insufficient or inadequate sleep. This assessment tool showed good convergent validity with respect to the global score of the Pittsburgh Sleep Quality Index (PSQI) in a sample of general practitioners' patients [26]. In order to define the quality of sleep during the last month, we considered only those 10 of the 23 items measuring the specific sleep symptoms of insomnia, excessive sleepiness, sleep apnea and parasomnia that were the target of our study. The first three questions concern symptoms of insomnia, the fourth question concerns non restorative sleep, while the other questions investigate excessive sleepiness (5-7), sleep apnea (8) and parasomnias (9, 10).

We defined the absence of "good sleep" on the basis of at least one positive response to the following ten items: 1) Did you take more than half an hour to fall asleep? 2) While sleeping, do you wake up often or do you remain awake for more than 30 min? 3) Have you woken up early, that is, more than an hour before you expected to? 4) Have you had non restorative sleep, that is as if you haven't slept at all? 5) Did you have problems staying awake during the day? 6) Did you have irresistible sleep attacks during the day? 7) Have you had an excessive need to sleep (10 h weren't enough)? 8) Have you realized or has someone told you that you stop breathing for a few seconds while you're sleeping? 9) Do you often (more than 3 times a month) have nightmares or dreams that cause a lot of anxiety? 10) Have there been episodes of sleep walking or of any other unusual behavior during sleep?

This definition allowed us to discriminate subjects who did not show any kind of problems ("good sleepers") from those showing slight, moderate or severe sleep problems ("non-good sleepers").

Twin pairs were considered discordant for sleep quality when one twin was a good sleeper (i.e. reported no sleep problems) while his/her co-twin was a non-good sleeper (i.e. reported at least one problem).

### Statistical analyses

Descriptive analysis was performed on twins as individuals to report means (with standard deviations) and frequencies.

Confirmatory Factor Analysis was applied to the 8 items of CBCL and YSR related to EC to estimate factor scores of the total EC scale and its subscales (attentional focusing and inhibitory control) separately for parent-reported and self-reported data. These scores were then averaged to obtain a composite EC score to be used in all subsequent analyses.

Robust regression taking account of the non-independence of data within twin pairs and including age and gender as covariates was applied on twins as individuals to estimate the association between EC and sleep quality.

Monozygotic (MZ) and dizygotic (DZ) pairs discordant for sleep quality were identified, and the estimated mean EC (and related subscales) factor scores in twins and co-twins, separately for MZ and DZ pairs, were compared using Student t test for paired samples.

DZ twins share on average 50% of their genetic background, while MZ twins are genetically identical. Furthermore, both MZ and DZ twins share the fetal and the early-life environment. Thus, the study of twins discordant for exposure to a putative risk factor (i.e. one twin in the pair is exposed, the other twin is not exposed) allows one to investigate if there is an association between exposure and outcome while matching exposed and unexposed twins for age, genetic background (partially for DZ twins, totally for MZ twins), as well as for intra-uterine and childhood within-family environment. In particular, this design allows one to understand if and to what extent an observed association between exposure and outcome is due to genetic and/or environmental effects shared by exposure and outcome (i.e. genetic and/or environmental confounding effects). Basically, three possible scenarios can occur: 1) the exposure is causally related to the outcome and no genetic or shared environmental confounding effects are involved; in this case, exposure and outcome are expected to be significantly associated with the same strength at the individual level, as well as within DZ and MZ discordant pairs; 2) the exposure is not a causal factor and its relation with the outcome is totally due to shared genetic and environmental effects (total confounding); in this case, exposure and outcome are expected to be significantly associated at the individual level, but the association is expected to weaken in DZ pairs (partially matched for genetic background, and totally matched for pre-natal and early-life environment), and to disappear in MZ pairs (totally matched both for genetic background and for pre-natal and early-life environment); 3) the exposure has a partial causal effect on the outcome; in this case, a significant association between exposure and outcome is expected at the individual level, but the exposure effect is expected to be reduced and persistent in both MZ and DZ pairs (partial confounding effects) [27]. In our study, the exposure variable is sleep quality and the outcome variables are EC and its subscales.

All analyses were performed with the Stata software (version 13).

## RESULTS

A total of 191 twin pairs (120 MZ pairs, 71 DZ pairs), representing ages 14 through 18, provided information on sleep quality. Ninety-nine percent of twins were attending school.

Among the subjects, 120 twins (31.4%) were identified as good sleepers, of whom 33.3% were male and about 63.3% were MZ. The remaining sample ( $n = 262$ ) included those subjects who showed at least one sleep problem (1-2 problems, 39%; 3-4 problems, 38%; 5-6 problems, 16%; more than 6 problems, 7%). Demographic characteristics, zygosity and mean EC (and related subscales) factor scores of good and non-good sleepers are reported in *Table 1*.

No gender differences were observed for sleep quality (chi-square test,  $p = 0.16$ ). Moreover, age was not correlated with EC ( $r = -0.06$ ,  $p = 0.25$ ) or sleep quality ( $r = -0.02$ ,  $p = 0.65$ ).

Twins with a good sleep quality had higher temperament factor scores (i.e. EC and the subscales "attentional focusing" and "inhibitory control") compared to those with sleep problems. Accordingly, age and gender adjusted correlations between the number of sleep problems endorsed by the subjects and EC or related subscales were negative and significant [ $r = -0.26$  ( $p < 0.0001$ ) for EC;  $r = -0.18$  ( $p < 0.001$ ) for attentional focusing;  $r = -0.27$  ( $p < 0.001$ ) for inhibitory control]. Adjusting by age and gender, sleep quality (1 = "good sleeper", 0 = "non-good sleeper") was significantly associated with EC factor scores ( $\beta = 0.33$ ,  $p < 0.001$ ) and with EC subscales scores ( $\beta_{\text{Attentional}} = 0.29$ ,  $p < 0.001$ ,  $\beta_{\text{Inhibitory}} = 0.28$ ,  $p < 0.001$ ); moreover, females showed higher EC scores compared to males ( $p < 0.05$ ).

Among all same sex twin pairs, 33 were discordant for sleep quality (*Table 2*). Of these, 17 were DZ pairs and 16 were MZ pairs. Compared to co-twins with sleep problems (non-good sleepers), twins without any sleep

problem (good sleepers) showed higher mean EC levels in both MZ and DZ pairs.

Larger intra-pair differences in DZ compared to MZ discordant pairs emerged. In particular, the mean difference in EC score was 0.32 (paired t test,  $p = 0.05$ ) in DZ pairs, partially matched for genetic background, and decreased to a non-significant 0.17 in MZ pairs, fully matched for genetic background (paired t test,  $p = 0.46$ ). Therefore, these results show that, besides accounting for age and gender, when controlling also for shared (fetal or familial) environmental factors and genetic background, the association between EC and sleep quality weakened in DZ pairs and disappeared in MZ pairs. Similar results were obtained for EC subscales.

## DISCUSSION

Over several years, researchers pointed to the association of adolescents' inadequate sleep quality and irregular sleep patterns with behavioral problems in the area of emotional self-regulation, such as negative moods, increased likelihood of stimulant use, higher levels of risk taking behaviors, poor school performance, and increased risk of unintentional injuries. This association drew attention on the possible causal link between sleep problems and emotional dis-regulation, which would be of relevance in the design of interventions aimed to improve emotional self-regulation.

Overall, our results confirmed at individual level the association of quality of sleeping with emotional regulation and its components [13]. However, since the association is weaker in DZ pairs and disappears in MZ pairs, these results do not seem to support a direct causal link between sleep quality and emotional regulation as measured by effortful control, and suggest that genetic and shared environmental factors may confound this association observed also in other studies. In other words, our data seem to support the idea that quality of

**Table 1**  
Demographic characteristics and factor scores of effortful control (and related subscales) according to sleep quality

	Sleep quality						Overall sample		
	Non-good sleepers (N = 262 twins)			Good sleepers (N = 120 twins)			(N = 382 twins)		
	N	Mean $\pm$ SD or %	Range	N	Mean $\pm$ SD or %	Range	N	Mean $\pm$ SD or %	Range
Gender, male	139	53.05%		76	63.33%		215	56.28%	
Age, years	262	16.37 $\pm$ 1.22	14 ; 18	120	16.34 $\pm$ 1.24	14 ; 18	382	16.37 $\pm$ 1.22	14 ; 18
Age, percentiles									
25 <sup>th</sup>		15.3			15.2			15.2	
50 <sup>th</sup>		16.6			16.4			16.6	
75 <sup>th</sup>		17.5			17.5			17.5	
Zygosity									
MZ	80	30.53%		40	33.33%		120	31.41%	
DZ same sex	103	39.31%		39	32.50%		142	37.17%	
DZ opposite sex	79	30.15%		41	34.17%		120	31.41%	
Effortful control	262	-0.04 $\pm$ 0.82	-2.82 ; 1.22	120	0.30 $\pm$ 0.69	-3.14 ; 1.22	382	0.06 $\pm$ 0.80	-3.14 ; 1.22
Attentional focusing	261	-0.05 $\pm$ 0.82	-2.62 ; 1.01	120	0.23 $\pm$ 0.69	-2.32 ; 1.01	381	0.04 $\pm$ 0.79	-2.62 ; 1.01
Inhibitory control	262	-0.03 $\pm$ 0.79	-2.78 ; 1.08	120	0.28 $\pm$ 0.66	-3.05 ; 1.08	382	0.07 $\pm$ 0.76	-3.05 ; 1.08

SD: standard deviation; MZ: monozygotic; DZ: dizygotic.

**Table 2**

Comparison of good and non-good sleepers in terms of effortful control (and related subscales)

	Discordant same-sex DZ pairs					Discordant MZ pairs				
	N	Good sleepers	Non-good sleepers	Difference	P*	N	Good sleepers	Non-good sleepers	Difference	P*
		Mean (SD)	Mean (SD)	Mean (SD)			Mean (SD)	Mean (SD)	Mean (SD)	
Effortful control	17	0.44 (0.61)	0.11 (0.64)	0.32 (0.62)	0.05	16	0.27 (0.67)	0.10 (0.87)	0.17 (0.87)	0.46
Attentional focusing	17	0.44 (0.53)	0.10 (0.69)	0.34 (0.76)	0.09	16	0.21 (0.72)	-0.05 (0.76)	0.26 (0.69)	0.15
Inhibitory control	17	0.32 (0.63)	0.09 (0.56)	0.23 (0.63)	0.16	16	0.25 (0.55)	0.20 (0.83)	0.04 (0.88)	0.85

\* One-sample t test; SD: standard deviation; MZ: monozygotic; DZ: dizygotic.

sleeping and emotional regulation may depend upon a common set of genetic and environmental causes. This result may reinforce the evidence found by Diaz *et al.* [28] that EC moderates the relation between sleep and academic achievement in children: children with low EC need plenty of sleep in order to be successful at school. Furthermore, looking from the other direction, Berger *et al.* [29] found that sleep duration moderates the association between children's temperament and academic achievement. These phenotypes might have then a complex genetic and environmental architecture, and our findings suggest the need to investigate deeply the composite relation between sleep and children's EC: in the absence of a causal dependency between sleep quality and emotional regulation, difficulties in sleeping behavior may not represent a good direct target for regulation-enhancing interventions.

The results of our study should be interpreted in the light of the following main limitations: a) the use of self-reported data; b) our strict classification of subjects as good sleepers (and therefore our broad classification of subjects as non-good sleepers), which is new and does not allow comparisons with other studies; c) the dependency of our results on the arbitrary definition used for sleep quality; d) the low number of discordant pairs; however, the (high-degree) genetic and environmental matching of subjects may partly counterbalance the reduction in power imposed by a limited sample size.

Although the cross-sectional study design did not allow us to resolve the directionality of the association between EC and sleep quality, we were able to show a possible dependency of emotional regulation and quali-

ty of sleeping upon common genetic and environmental factors. As regards the modifiable factors, a crucial role in the expression of both these phenotypes is played by parenting style and family functioning [30, 31], which may thus represent two of the key environmental exposures common to the twin pair.

Our results may have significant implications for theories aimed to explain the links between sleep quality and emotional regulation; furthermore, they highlight the need to identify the relevant common environmental exposures in order to plan interventions aimed to improve both sleep quality and emotional regulation.

#### Author's contribution statement

EM: planned and performed statistical analysis, contributed interpreting the results and writing the manuscript. GA: discussed the results and contributed to the final manuscript. DD: collected data, aided in interpreting the results. CF: performed statistical analysis, contributed interpreting the results and writing the manuscript. MF: aided in data collection and contributed to database preparation. CV: contributed to the phenotypes definition and commented on the paper. MAS: contributed to the design and implementation of the research and provided critical feedback. All authors read and approved the final manuscript.

#### Conflict of interest statement

Authors have no conflicts of interest to disclose.

Received on 7 November 2018.

Accepted on 1 April 2019.

## REFERENCES

1. Carskadon MA. Sleep in adolescents: the perfect storm. *Pediatr Clin North Am.* 2011;58(3):637-47. doi: 10.1016/j.pcl.2011.03.003.37
2. Moore M, Meltzer LJ. The sleepy adolescent. Causes and consequences of sleepiness in teens. *Paediatr Respir Rev.* 2008;9(2):114-21. doi: 10.1016/j.prrv.2008.01.001
3. Fallone G, Owens JA, Deane J. Sleepiness in children and adolescents. Clinical implications. *Sleep Med Rev.* 2002;6(4):287-306.
4. te Velde SJ, van der Aa N, Boomsma DI, et al. Genetic and environmental influences on individual differences in sleep duration during adolescence. *Twin Res Hum Genet.* 2013;16(6):1015-25. doi: 10.1017/thg.2013.74
5. Moore M, Slane J, Mindell JA, et al. Genetic and environmental influences on sleep problems: a study of preadolescent and adolescent twins. *Child Care Health Dev.* 2011;37(5):638-41. doi: 10.1111/j.1365-2214.2011.01230.x

6. Hagenauer MH, Perryman JI, Lee TM, Carskadon MA. Adolescent changes in the homeostatic and circadian regulation of sleep. *Dev Neurosci*. 2009;31(4):276-84. doi: 10.1159/000216538
7. Carskadon MA, Tarokh L. Developmental changes in sleep biology and potential effects on adolescent behavior and caffeine use. *Nutr Rev*. 2014;72(Suppl. 1):60-4. doi: 10.1111/nure.12147. Review.
8. Carskadon MA, Tarokh L. Developmental changes in sleep biology and potential effects on adolescent behavior and caffeine use. *Nutr Rev*. 2014;72 (Suppl. 1):60-4. doi: 10.1111/nure.12147. Review.
9. Willis TA, Gregory AM. Anxiety disorders and sleep in children and adolescents. *Sleep Med Clin*. 2015;10(2):125-31. doi: 10.1016/j.jsmc.2015.02.002
10. Monroe LJ, Marks PA. MMPI differences between adolescent poor and good sleepers. *J Consult Clin Psychol*. 1977;45(1):151-2.
11. Matthews T, Danese A, Gregory AM, Caspi A, Moffitt TE, Arseneault L. Sleeping with one eye open: loneliness and sleep quality in young adults. *Psychol Med*. 2017;47(12):2177-86. doi: 10.1017/S0033291717000629
12. Moore M, Slane J, Mindell JA, et al. Sleep problems and temperament in adolescents. *Child Care Health Dev*. 2011;37(4):559-62. doi:10.1111/j.1365-2214.2010.01157.x
13. Lukowski AF, Milojevich HM. Sleep quality and temperament among university students: differential associations with nighttime sleep duration and sleep disruptions. *Behav Sleep Med*. 2015;13(3):217-30. doi: 10.1080/15402002.2013.855214
14. Blair C, Diamond A. Biological processes in prevention and intervention. The promotion of self-regulation as a means of preventing school failure. *Dev Psychopathol*. 2008;20(3):899-911. doi: 10.1017/S0954579408000436
15. Rueda MR, Posner MI, Rothbart MK. The development of executive attention. Contributions to the emergence of self-regulation. *Dev Neuropsychol*. 2005;28(2):573-94. doi: 10.1207/s15326942dn2802\_2
16. Rothbart MK, Rueda MR. The development of effortful control. In: *Developing individuality in the human brain. A tribute to Michael I. Posner*. Mayr U, Awh E, Keele S (Eds). Washington, DC: American Psychological Association; 2005. pp. 167-188.
17. Eisenberg N, Valiente C, Morris AS, et al. Longitudinal relations among parental emotional expressivity, children's regulation, and quality of socioemotional functioning. *Dev Psychol*. 2003;39(1):3-19.
18. Brock LL, Rimm-Kaufman SE, Nathanson L, et al. The contributions of "hot" and "cool" executive function to children's academic achievement and learning-related behaviors, and engagement in kindergarten. *Early Child Res Quart*. 2009;24(3):337-49. doi: 10.1016/j.ecresq.2009.06.001
19. Fagnani C, Medda E, Alessandri G, et al. The genetic architecture of effortful control and its interplay with psychological adjustment in adolescence. *J Res Pers*. 2017;68:5-14.
20. Lemery-Chalfant K, Doelger L, Goldsmith HH. Genetic relations between effortful and attentional control and symptoms of psychopathology in middle childhood. *Infant Child Develop*. 2008;17:365-85. doi: 10.1002/icd.581
21. Yamagata S, Takahashi Y, Kijima N, Maekawa H, Ono Y, Ando J. Genetic and environmental etiology of effortful control. *Twin Res Hum Genet*. 2005;8:300-6. doi: 10.1375/1832427054936790
22. Brescianini S, Fagnani C, Toccaceli V, et al. An update on the Italian Twin Register. Advances in cohort recruitment, project building and network development. *Twin Res Hum Genet*. 2013;16(1):190-6. doi: 10.1017/thg.2012.85
23. Goldsmith HH. A zygosity questionnaire for young twins: a research note. *Behav Genet*. 1991;21(3):257-70.
24. Achenbach TM, Rescorla LA (Eds). *Manual for the ASEBA School-Age Forms & Profiles*. Burlington, VT: University of Vermont, Research Centre for Children, Youth & Families; 2001.
25. Luengo-Kanacri BP, Pastorelli C, Eisenberg N, et al. The development of prosociality from adolescence to early adulthood: The role of effortful control. *J Pers*. 2013;81(3):302-12. doi: 10.1111/jopy.12001
26. Violani C, Devoto A, Lucidi F, et al. Validity of a Short Insomnia Questionnaire. *The SDQ*. *Brain Res Bull*. 2004;63(5):415-21.
27. McGue M, Osler M, Christensen K. Causal inference and observational research. The utility of twins. *Perspect Psychol Sci*. 2010;5(5):546-56.
28. Diaz A, Berger R, Valiente C, Eisenberg N, VanSchyndel S, Tao C, Spinrad TL, Doane LD, Thompson MS, Silva KM, Southworth J. Children's sleep and academic achievement. The moderating role of effortful control. *Int J Behav Dev*. 2017;41(2):275-84. doi: 10.1177/0165025416635284
29. Berger RH, Diaz A, Valiente C, Eisenberg N, Spinrad TL, Thompson MS, Hernández MM, VanSchyndel SK, Southworth J. Sleep duration moderates the association between children's temperament and academic achievement. *Early Educ Dev*. 2018;29(5):624-40. doi: 10.1080/10409289.2017.1404884
30. Buckhalt JA1, El-Sheikh M, Keller PS, Kelly RJ. Concurrent and longitudinal relations between children's sleep and cognitive functioning: the moderating role of parent education. *Child Dev*. 2009;80(3):875-92. doi: 10.1111/j.1467-8624.2009.01303.x
31. El-Sheikh M, Tu KM, Erath SA, Buckhalt JA. Family stress and adolescents' cognitive functioning: sleep as a protective factor. *J Fam Psychol*. 2014;28(6):887-96. doi: 10.1037/fam0000031