Stress reactivity in salivary cortisol and electrocardiogram in adolescents: Investigating sleep disturbances and insomnia

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Summary
This study examined the role of sleep disturbances and insomnia in the context of stress reactivity in adolescence. One-hundred and thirty-five 11–18 year olds (M\textsubscript{age} = 14.2 years, SD = 1.9, 52% female) completed the Trier Social Stress Test for Children. Salivary cortisol and subjective stress ratings were collected at six time points, and heart rate as well as heart rate variability were measured pre-, during and post-stress induction. Additionally, sleep disturbances and insomnia diagnosis were assessed by a self-report questionnaire and a sleep interview. Robust mixed models investigated if adolescents with compared with adolescents without (a) sleep disturbances and (b) insomnia differ regarding cortisol, heart rate, heart rate variability and psychological stress reactivity considering gender effects. The results indicated that boys with high sleep disturbances showed higher cortisol activity compared with boys with low sleep disturbances, B = 0.88, p < 0.05. Moreover, in boys with insomnia, heart rate and alpha 1 significantly differ less than in boys without insomnia. These findings support the notion of sex differences regarding the association between poor sleep and increased activity of the hypothalamic–pituitary–adrenal axis, and a less adaptable autonomic nervous system in boys in response to an experimental social stress task.

KEYWORDS
heart rate analysis, hypothalamic–pituitary–adrenal dysfunction, laboratory stress test, sleep problems, stress vulnerability, youth

1 | INTRODUCTION

Increased stress associated with the beginning of puberty as a time of cognitive, social and physical change (Byrne et al., 2007) goes along with increasing sleep difficulties (Tavernier et al., 2016; Zhang et al., 2016). Striking evidence confirms the impact of poor sleep on cognitive, emotional and health problems (de Zambotti, 2018). A discussed mechanism behind the link between stress and sleep is suggested in the hyperarousal model of insomnia (Riemann et al., 2010), according to which the major component contributing to future and current sleep disturbances is increased arousal by interfering with falling and staying asleep. Physiological arousal activates two major elements of the stress regulation system: the neuroendocrine stress response of the hypothalamic–pituitary–adrenal (HPA) axis (Asarnow, 2020); and the body-regulating response of the autonomic nervous system (ANS) via sympathetic and parasympathetic branches (Kupper et al., 2021). Various studies have addressed the assumption that the experience of chronic stress is reflected in a stress system dysfunction under challenges (Zänkert et al., 2019). Dysregulations measured by cortisol and heart rate variability (HRV) have already been seen in individuals with...
stress and sleep were directly addressed as study topics. In addition, participants had one or more siblings (86%). The adolescents were recruited via grade; 1%, 13th grade; 13%, 9th grade; 22%, 10th grade; 10%, 11th grade; 7%, 12th grade (1%, 5th grade; 12%, 6th grade; 8%, 7th grade; 17%, 8th grade; 19%, 10th grade; 11%, 12th grade). Most of the students were German (88%) and had one or more siblings (86%). The adolescents were recruited via social media, newspapers, flyers and direct contact at school, whereby stress and sleep were directly addressed as study topics. In addition, participation was not recommended in cases of (diagnosed) mental health disorder or heart disease.

The participants answered questionnaires on stress, sleep and puberty status (see below for detailed information), which were filled out online at home. At a personal university appointment, they additionally responded to a sleep interview. At a second appointment, participants completed the TSST for Children (TSST-C; Buske-Kirschbaum et al., 1997) in age-appropriate versions (see below). Six saliva samples and three electrocardiogram measurements were taken before, during and after the TSST-C (Figure 1). This procedure was conducted in the afternoon (between 15:30 hours and 19:00 hours) to minimize the effects of intra-individual diurnal cortisol variation, following the recommendations of Seddon et al. (2020).

The study was part of a larger study funded by the German Research Foundation (DFG; funding codes LO 337/30-1 and SCHL 1909/8-1) and approved by the local Ethics Committee of the university. Participation was rewarded with a monetary incentive in the form of a 30€ voucher. Participation was voluntary but, as all participants were underage at the beginning of their participation, a caregiver gave written informed consent. In the overall study, 203 adolescents took part, with only 175 participants completing the TSST-C. Of this sample, 40 adolescents were excluded due to clinically relevant mental health symptoms to avoid interferences with other mental disorders, in particular symptoms of behavioural and emotional disorders with onset in childhood and adolescence as well as affective and anxiety disorders, assessed with the Diagnostic System for Mental Disorders according to ICD-10 and DSM-5 for children and adolescents (DISYPS-3; Döpfner & Görtz-Dorten, 2017) screening questionnaire. Based on the manual, a total score in the 90th percentile of the normative sample scores was considered clinically relevant.

2.2 | Measurements

2.2.1 | Trier Social Stress Test for Children

The TSST-C consists of a preparation phase (5 min), a free speech task in which the participant has to retell a story (5 min), and calculate a demanding serial arithmetic subtraction task (5 min; 11 year olds: subtraction of 7 starting from 785; all from 12 years onwards: subtraction of 23 starting from 1023). To increase the perceived stress, videotapes and voices were recorded during the speech and arithmetic part, while the participants stood in front of two white-coated confederates.

2.2.2 | Salivary cortisol

As presented in Figure 1, saliva samples were collected from t1 to t6 using Salivette® sample material (Sarstedt, Nümbrecht, Germany). These included the time before the first resting phase (0 min), before the preparation phase (25 min), between the talk and subtraction tasks (35 min), after the subtraction task (40 min), after the second
After the resting phase (60 min), and after the debriefing (80 min). Saliva samples were frozen and stored at −20°C until they were analysed at the laboratory of Prof. Kirschbaum (TU Dresden, Germany). After thawing, salivettes were centrifuged at 3000 rpm for 5 min, which resulted in a clear supernatant of low viscosity. Salivary concentrations were measured using a commercially available chemiluminescence immunoassay with high sensitivity (IBL International). The intra- and interassay coefficients for cortisol were below 9%.

2.2.3 | Psychological stress response to TSST-C

Each time a saliva sample was taken, the participants were asked to rate their subjective levels of stress (i.e. “How stressed do you feel now?”) in a written form on a seven-point scale from (1) none to (7) very high stress. During the assessment at t3, the investigator was instructed to keep distance and to react only briefly to the participant’s attempts of conversation.

2.2.4 | Electrocardiogram recording and HRV index measures

Using a wearable chest belt (Polar Sigma R1, Polar), heart rate and HRV parameters were measured at two 5-min intervals at rest before and after the TSST-C and during a 15-min interval throughout the TSST-C (Figure 1). In this study, heart rate and the root mean square of successive R-R interval differences (RMSSD) as well as alpha 1 were examined. The time domain parameter RMSSD represents the magnitude of fluctuation of HRV, with higher values representing higher parasympathetic activity. The detrended fluctuation analysis parameter alpha 1 is a non-linear parameter, with values higher than 1 indicating a higher quality of regulation and connectivity between the parasympathetic and sympathetic systems, while values below 1 suggest low stability of these systems with a low quality of regulation. Values near 1 are considered to indicate optimal reactivity.

2.2.5 | Insomnia

A semi-structured sleep interview was used to assess different sleep disorders. In accordance with the International Classification of Sleep Disorders (3rd edn; ICSD-3; American Academy of Sleep Medicine, 2014), insomnia was diagnosed if an adolescent reported difficulties falling asleep or staying asleep (despite having an adequate opportunity to sleep), resulting in significant distress and daytime consequences (e.g. sleepiness, difficulties with concentration and memory, mood lability, social or school difficulties). Insomnia was diagnosed if all criteria were fulfilled for at least 1 month, characterized as acute insomnia.

2.2.6 | Sleep disturbance

The Sleep Disturbance Scale for Children (SDSC; Bruni et al., 1996) investigates sleep behaviour and disturbances for the past 6 months on six subscales. A total score ranging between 26 and 130 can be calculated out of 26 items, which are answered on a Likert-scale ranging from (1) never to (5) always/daily. A value higher than the cut-off of 39 indicates clinical sleep disturbances (Bruni et al., 1996). For the total score, Bruni et al. (1996) reported a Cronbach’s alpha of 0.71 for children with sleep problems and 0.79 for a healthy control group. For this sample, Cronbach’s alpha was 0.79.

2.2.7 | Covariates

Gender, age, puberty status and body mass index (BMI) z-scores were included as covariates, as indicated by Seddon et al. (2020). Puberty status was measured by the German version of the Pubertal Development Scale (PDS; Watzlawik, 2009), differentiating five puberty scores from (1) prepubertal to (5) postpubertal. BMI scores were transformed into z-scored BMI values to allow adjustments for reference standards grouped by age and gender, as the meaning of BMI varies with age and sex (Must & Anderson, 2006). Moreover, as the data collection period covered both the time before and after the beginning of the SARS-CoV-2 pandemic, the presence of COVID-19 (no/yes) was included as an additional covariate.

2.3 | Statistical analysis

All analyses were conducted in R (v3.4.2, https://www.r-project.org/). After a pre-inspection, missing values (4% of all data values) were
TABLE 1  Unstandardized estimates for linear, quadratic and cubic changes in salivary cortisol throughout the TSST-C procedure (t1–t6)

| Effect                  | Sleep disturbances      |                | Insomnia              |
|-------------------------|-------------------------|----------------|-----------------------|
|                         | All         | Girls     | Boys      | All         | Girls     | Boys      |
| Fixed effects            |             |           |           |             |           |           |
| Intercept               | 2.19 (0.31)**| 2.53 (0.50)**| 2.03 (0.36)**| 2.66 (0.19)**| 2.89 (0.25)**| 2.58 (0.23)**|
| Female                  | -0.02 (0.18) | -           | -         | 0.06 (0.18) | -           | -         |
| Age/puberty             | -0.42 (0.09)**| -0.38 (0.13)**| -0.52 (0.14)**| -0.41 (0.09)**| -0.34 (0.13)**| -0.50 (0.14)**|
| BMI z-score             | -0.11 (0.07) | -0.13 (0.11) | -0.10 (0.10) | -0.09 (0.08) | -0.10 (0.11) | -0.08 (0.10) |
| COVID-19                | 0.55 (0.18)**| 0.36 (0.26) | 0.67 (0.29)* | 0.54 (0.18)**| 0.32 (0.26) | 0.67 (0.29)**|
| Group                   | 0.65 (0.32) | 0.39 (0.56) | 0.88 (0.40) | 0.16 (0.29) | 0.03 (0.42) | 0.38 (0.41) |
| Time—linear change      | 12.40 (6.49) | 17.36 (11.92) | 9.52 (6.76) | **18.76 (3.28)** | **22.12 (5.30)** | **15.80 (3.77)** |
| Time2—quadratic change  | -2.00 (2.89) | -5.93 (4.61) | 0.38 (3.74) | -2.47 (1.46) | -1.70 (2.14) | -3.97 (2.05) |
| Time3—cubic change      | -18.63 (2.89)** | -21.52 (4.61)** | -16.76 (3.74)** | -22.43 (1.46)** | -22.93 (2.14)** | -21.78 (2.05)** |
| Group × Time            | 4.79 (1.79) | -0.61 (12.87) | 8.62 (7.70) | -9.13 (6.26) | -16.85 (9.45) | 0.41 (7.85) |
| Group × Time2           | 0.23 (3.20) | 6.23 (4.98) | -5.80 (4.26) | 2.40 (2.79) | 3.09 (3.81) | 0.55 (4.26) |
| Group × Time3           | -4.50 (3.20) | -0.59 (4.98) | -7.59 (4.26) | 0.73 (2.79) | 2.53 (3.81) | -2.40 (4.26) |

Random effects—variance components

| Effect   | Intercept | Time | R² marginal | R² conditional | F² | ICC |
|----------|-----------|------|-------------|----------------|----|-----|
|          | 1.72 (1.31) | 790.05 (28.11) | 0.28 | 0.71 | 0.08 | 0.44 |
|          | 2.06 (1.44) | 1129.64 (36.61) | 0.23 | 0.72 | 0.06 | 0.49 |
|          | 1.51 (1.23) | 444.71 (21.09) | 0.33 | 0.71 | 0.12 | 0.38 |
|          | 1.86 (1.37) | 789.14 (28.09) | 0.25 | 0.73 | 0.07 | 0.46 |
|          | 2.23 (1.49) | 1056.08 (32.50) | 0.22 | 0.70 | 0.05 | 0.51 |
|          | 1.63 (1.28) | 469.75 (21.67) | 0.28 | 0.09 | 0.09 | 0.42 |

Note: Group = Sleep disturbances (low versus high)/Insomnia (without versus with). N all = 135, girls = 70, boys = 65. Time = linear effect of time points t1–t6; Time² = quadratic effect of time points t1–t6; Time³ = cubic effect of time points t1–t6; COVID-19 = TSST-C was conducted before/during SARS-CoV-2 pandemic (0/1). Values indicate the estimated effect and corresponding standard error (SE). Bold values indicate significant variables of interest. *p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: BMI, body mass index; ICC, intraclass correlation coefficient.

Interpreted by multiple imputation using the package Multivariate Imputation by Chained Equations (MICE). For imputation, predictive mean matching, logistic regression imputation and polynomial regression were used with regard to continuous and categorical data. Only variables were used that correlated with each other by at least 0.1. All analyses were based on aggregated data. Total and sub-scores were calculated as sum scores of the respective item values. In accordance with Bruni et al. (1996), a cut-off score of 39 was used to group adolescents with low and high sleep disturbances. To avoid multicollinearity between covariates, age and puberty scores were combined via factor analysis.

Multilevel modelling was used to evaluate group differences (sleep disturbances: low versus high; insomnia: without versus with) in cortisol reactivity, psychological response and HRV over time with crossed random effects. The mixed models included time, group, and the covariates sex, age/puberty factor, BMI z-score and COVID-19 as fixed effects, and time crossed by subject as random effects. To address the violation of a normal distribution, robust mixed models with crossed random effects are reported in the following. Because cortisol responses and psychological and electrocardiological profiles do not typically increase linearly throughout the TSST procedure, each set of mixed-effects models with time as a linear function (time1), time as a quadratic function (time2) and time as a cubic function (time3) was compared using a likelihood ratio test. To find the best model, non-robust mixed models were calculated in this process, as robust mixed models do not display indices for direct model comparison. Log-likelihood ratio tests indicated that the inclusion of quadratic time for the electrocardiogram variables and cubic time for the cortisol and psychological responses significantly improved model fit (all p < 0.01) according to which the appropriate model is reported in the following. R² and intraclass correlation coefficient (ICC) were calculated according to Nakagawa et al. (2017). For all tests, a p-value < 0.05 was considered as significant.

3  | RESULTS

3.1  | Descriptive statistics

Adolescents with low (M = 13.28, SD = 1.51, n = 25) compared with high (M = 14.41, SD = 1.93, n = 110) sleep disturbances were younger (t44 = −3.19, p < 0.01) and in an earlier phase of puberty (t40 = −2.88.
p < 0.01), but did not differ with regard to sex or BMI z-score. For those with (n = 37) compared with those without insomnia (n = 98), no differences in sex, BMI z-score and puberty status were found (Tables S1 and S2), but individuals with insomnia again were younger ($t_{66} = -2.23$, $p = 0.03$). Adolescents with severe sleep disturbances or insomnia primarily reported disorders of excessive somnolence, disorders of initiating and maintaining sleep, and sleep wake transition disorders (Table S3). Age was associated with sleep disturbances ($r = 0.27$, $p < 0.001$) and insomnia ($r = 0.18$, $p = 0.04$), but not with overall cortisol level or psychological stress (all $p > 0.05$). Female sex was related to sleep disturbances ($r = 0.31$, $p < 0.001$), but not to insomnia, nor again with overall cortisol level or psychological stress (all $p > 0.05$).

### 3.3 Comparisons of sleep groups

#### 3.3.1 Salivary cortisol

Robust mixed models controlling for sex, age/puberty, BMI z-score, and COVID-19 were performed to assess the response to the TSST in salivary cortisol for two group comparisons: (a) high versus low overall sleep disturbances; and (b) with versus without a diagnosis of insomnia (Figure 2; Table 1).

The first model showed significant differences between adolescents with low and high sleep disturbances in the total sample and more specifically in boys, but not in girls, revealing higher cortisol activity in boys with high sleep disturbance. There were no significant interaction effects. A second mixed model testing differences between insomnia diagnosis and no insomnia showed no main or interaction effect for insomnia.

### 3.3.2 Heart rate and HRV

Investigating the heart rate and HRV (RMSSD and alpha 1), all models (Figure 2; Tables 2–4) showed significant time effects, indicating higher activation during the TSST-C compared with before and after. For heart rate and alpha 1, the models reveal significant

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**Table 2** Unstandardized estimates for linear and quadratic changes in heart rate in the period from before until after the TSST-C

| Effect                      | Sleep disturbances | Insomnia         |
|-----------------------------|--------------------|------------------|
|                             | All    | Girls | Boys | All    | Girls | Boys |
| Fixed effects               |        |       |      |        |       |      |
| Intercept                   | 79.85  | 79.02 | 81.38| 78.74  | 79.08 | 80.18|
| Female                      | 1.80   | –     | –    | 1.87   | –     | –    |
| Age/puberty                 | 0.63   | 1.16  | –0.26| 0.62   | 1.17  | –0.13|
| BMI z-score                 | −1.21  | −0.07 | −1.74| −1.33  | −0.15 | −0.87 |
| COVID-19                    | 2.68   | 6.69  | 2.58 | 2.58   | 6.64  | 2.44 |
| Group                       | −2.15  | 1.32  | −2.44| −2.55  | −1.28 | −3.67|
| Time-linear change          | 0.59   | 24.06 | −11.48| 12.71  | 23.64 | 3.78 |
| Time²—quadratic change      | 246.15 | −264.86| −235.14| 242.22 | −245.56| −241.02|
| Group × Time                | 11.13  | −6.55 | 16.11| −10.87 | −15.70| −12.47|
| Group × Time²               | 8.22   | 18.34 | 6.12 | 11.33  | 8.77  | 52.26|
| Random effects—Variance components |        |       |      |        |       |      |
| Intercept                   | 110.67 | 82.65 | 137.72| 109.86 | 82.59 | 132.45|
| Time                        | 107.54 | 176.54| 129.22| 73.23  | 166.96| 86.25 |
| $R^2$ marginal              | 0.44   | 0.52  | 0.39 | 0.45   | 0.51  | 0.40 |
| $R^2$ conditional           | 0.87   | 0.89  | 0.89 | 0.87   | 0.83  | 0.89 |
| $F^2$                       | 0.24   | 0.36  | 0.18 | 0.25   | 0.36  | 0.20 |
| ICC                         | 0.42   | 0.32  | 0.50 | 0.42   | 0.32  | 0.49 |

Note: Group = Sleep disturbances (low versus high)/Insomnia (without versus with). $N_{all} = 135$, girls = 70, boys = 65. Time = linear effect of time points before, during and after TSST-C; Time² = quadratic effect of time points before, during and after TSST-C; COVID-19 = TSST-C was conducted before/during Sars-Cov-2 pandemic (0/1). Values indicate the estimated effect and corresponding standard error (SE). Bold values indicate significant variables of interest.

$^*$p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: BMI, body mass index; ICC, intraclass correlation coefficient.

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### 3.2 Effectiveness of the stress-induction task

The TSST successfully induced stress. Specifically, participants reported significantly more psychological stress during the TSST-C ($t_3 = 4.47$, $SD = 1.35$) compared with stress prior to stress exposure ($t_2 = 1.94$, $SD = 1.00$; $t_{59} = -17.54$, $p < 0.001$) and after recovery ($t_6 = 1.64$, $SD = 0.84$; $t_{59} = 2.70$, $p < 0.01$), respectively. The stress induction effect is shown by the main effects of time in salivary cortisol (Table 1) and in the electrocardiogram (Tables 2–4), demonstrating successful manipulation within the TSST.
TABLE 3 Unstandardized estimates for linear and quadratic changes in RMSSD in the period from before until after the TSST-C

| Effect                | Sleep disturbances | Insomnia        |
|-----------------------|-------------------|-----------------|
|                       | All | Girls | Boys | All | Girls | Boys |
| Intercept             | 58.73 (8.12)**   | 77.02 (12.12)** | 55.73 (9.58)** | 63.60 (5.37)** | 78.34 (6.21)** | 60.56 (2.10)** |
| Female                | 8.04 (6.24)      | —              | —    | 8.71 (6.27)      | —              | —    |
| Age/puberty           | 10.88 (3.23)**   | 11.83 (4.63)†  | 8.09 (4.47) | 10.50 (3.24)**  | 11.83 (4.53)†  | 7.83 (4.52) |
| BMI z-score           | 4.59 (2.56)      | 1.05 (3.99)    | 5.82 (3.22) | 4.70 (2.58)     | 0.98 (4.03)    | 5.78 (3.27) |
| COVID-19              | -14.34 (6.15)‡   | -28.34 (9.19)** | -4.96 (8.47) | -13.95 (6.30)‡  | -28.35 (9.16)** | -4.02 (8.57) |
| Group                 | 6.90 (8.15)      | 1.97 (13.29)   | 7.23 (10.27) | 1.43 (7.07)     | 1.34 (9.74)    | 1.55 (10.31) |
| Time—linear change    | 29.60 (72.84)    | 59.13 (138.05) | 8.86 (86.20) | -11.66 (37.09)  | -52.12 (63.35) | 14.47 (46.58) |
| Time²—quadratic change| 360.97 (71.27)**| 491.96 (136.47)** | 319.22 (80.55)** | 381.79 (36.32)** | 426.43 (62.59)** | 349.04 (44.04)** |
| Group x Time          | -69.47 (80.69)   | -118.57 (149.11) | -31.61 (98.28) | -58.50 (70.85)  | 8.99 (113.00)  | -115.45 (96.97) |
| Group x Time²         | 27.15 (78.95)    | -66.63 (147.41) | 26.49 (91.84) | 8.12 (69.37)    | 19.80 (111.65) | -39.58 (91.67) |

Random effects—Variance components

|                     | Intercept | Time       | R² marginal | R² conditional | F² | ICC         |
|---------------------|-----------|------------|-------------|----------------|----|-------------|
|                     | 955.10 (30.90) | 5287.20 (72.71) | 0.22        | 0.67           | 0.05 | 0.46       |
| All                  | 880.90 (29.68) | 4045.60 (63.60) | 0.26        | 0.60           | 0.07 | 0.45       |
| Girls               | 946.40 (30.76) | 13220.60 (114.98) | 0.18        | 0.71           | 0.03 | 0.52       |
| Boys                | 964.20 (31.05) | 5213.80 (72.21) | 0.21        | 0.67           | 0.05 | 0.46       |
| Time                | 5213.80 (31.05) | 4276.40 (65.39) | 0.26        | 0.60           | 0.07 | 0.35       |
| Time/C0             | 892.60 (29.88) | 10794.50 (103.90) | 0.18        | 0.71           | 0.03 | 0.53       |

Note: Group = Sleep disturbances (low versus high)/Insomnia (without versus with). N = 135, girls = 70, boys = 65. Time = linear effect of time points before, during and after TSST-C; Time² = quadratic effect of time points before, during and after TSST-C; COVID-19 = TSST-C was conducted before/during Sars-Cov-2 pandemic (0/1). Values indicate the estimated effect and corresponding standard error (SE). Bold values indicate significant variables of interest.

*p < 0.05; **p < 0.01; ***p < 0.001.

Abbreviations: BMI, body mass index; ICC, intraclass correlation coefficient.

Time² × Insomnia interaction effects for boys. Post hoc t-tests revealed a significantly smaller change in heart rate from during to after TSST-C for boys with compared with boys without insomnia ($t_{36.71} = 2.18$, $p = 0.04$). Relatedly, alpha 1 differences from during to after TSST-C were significantly smaller in boys with compared with without insomnia ($t_{13.96} = 2.16$, $p < 0.05$).

3.3.3 Psychological stress response

When testing for differences in subjective stress between adolescents with low and high sleep disturbances, there was neither a significant main effect of sleep disturbances nor an interaction.

The model testing differences for insomnia diagnosis showed a main effect of insomnia only in girls, showing that girls with insomnia reported higher levels of psychological stress. Moreover, a significant interaction Time² × Insomnia in the total sample and more precisely in girls indicated that the increased stress is only observable prior to the experimental stressor at time 1 in girls ($t_{24.60} = -2.62$, $p = 0.02$) and in the total sample ($t_{45.04} = -2.20$, $p = 0.03$), but at no other time points (Table 5). However, girls and boys again did not differ in their overall stress response ($p > 0.05$).

4 DISCUSSION

The present study examined the HPA, ANS and psychological stress reactivity related to the TSST-C in adolescents with high/low sleep disturbances and with/without diagnosed insomnia. The TSST-C elicited the intended stress response in the HPA axis, ANS and psychological stress levels. Particularities associated with poor sleep were most evident in more striking overall cortisol activity, less oscillating ANS and higher stress in the initial phase of the TSST-C procedure. Even if girls and boys did not differ in their average cortisol reactivity and psychological stress response, some important alterations depending on biological sex were visible for the relation between sleep and stress reactivity.

In more detail, as expected, adolescents with sleep disturbances had higher levels of salivary cortisol. When considering the two sexes separately, the effect could only be discovered for boys, but not for girls. This is in contrast with the findings of Mrug et al. (2016), who also utilized the TSST-C in 84, 11-16 year olds of low-income families. They found a prediction of sleep problems for the area under the curve with respect to ground and with respect to increase in the TSST-C only for girls but not for boys. Low income is associated with a blunted cortisol reactivity, which in combination with
TABLE 4 Unstandardized estimates for linear and quadratic changes in alpha 1 in the period from before until after the TSST-C

| Effect                  | Sleep disturbances |          |          |          | Insomnia |          |          |          |
|-------------------------|--------------------|----------|----------|----------|----------|----------|----------|----------|
|                         | All                | Girls    | Boys     |          | All      | Girls    | Boys     |          |
| Fixed effects           |                    |          |          |          |          |          |          |          |
| Intercept               | 1.07 (0.04)***      | 0.95 (0.06)*** | 1.11 (0.05)*** | 1.09 (0.03)*** | 1.02 (0.03)*** | 1.12 (0.03)*** |
| Female                  | -0.07 (0.03)†       | -        | -        | -0.04 (0.02)† | -0.04 (0.02)† | -0.04 (0.02)† |
| Age/puberty             | -0.04 (0.02)†       | 0.00 (0.02) | -0.08 (0.02)† | -0.04 (0.02)† | -0.02 (0.02)† | -0.04 (0.02)† |
| BMI z-score             | -0.04 (0.01)**      | -0.02 (0.02) | -0.04 (0.02) | -0.04 (0.01)† | -0.02 (0.02)† | -0.04 (0.02)† |
| COVID-19                | 0.12 (0.03)***      | 0.18 (0.02)*** | 0.05 (0.05) | 0.12 (0.03)*** | 0.18 (0.05)*** | 0.05 (0.05) |
| Group                   | 0.02 (0.03)         | 0.07 (0.07) | 0.02 (0.06) | -0.00 (0.04) | -0.01 (0.05) | 0.04 (0.06) |
| Time—linear change      | 0.45 (0.51)         | 0.67 (0.75) | 0.45 (0.66) | 0.77 (0.26)† | 0.55 (0.34) | 0.97 (0.36)† |
| Time²—quadratic change  | -4.12 (0.49)***     | -4.75 (0.75)*** | -3.71 (0.63)*** | -4.05 (0.25)*** | -4.53 (0.34)*** | -3.62 (0.34)*** |
| Group × Time            | 0.49 (0.56)         | 0.14 (0.81) | 0.65 (0.75) | 0.29 (0.49) | 0.63 (0.61) | -0.07 (0.74) |
| Group × Time²           | 0.30 (0.55)         | 0.38 (0.81) | 0.56 (0.72) | 0.63 (0.48) | 0.34 (0.61) | 1.49 (0.71)† |

Random effects—Variance components

|                  | Intercept | Time | R² marginal | R² conditional | F² | ICC |
|------------------|-----------|------|-------------|----------------|----|-----|
| Fixed effects    | 0.02 (0.16) | 0.02 (0.15) | 0.16 (0.72) | 0.65 | 0.70 | 0.26 |
| Time             | 0.34 (0.58) | 0.01 (0.08) | 0.14 (0.14) | 0.65 | 0.70 | 0.26 |
| R² marginal      | 0.40      | 0.48 | 0.35        | 0.40             | 0.43 | 0.29 |
| R² conditional   | 0.65      | 0.70 | 0.64        | 0.65             | 0.70 | 0.22 |
| F²               | 0.19      | 0.30 | 0.14        | 0.19             | 0.23 | 0.22 |
| ICC              | 0.26      | 0.22 | 0.29        | 0.26             | 0.26 | 0.22 |

Note: Group = Sleep disturbances (low versus high)/Insomnia (without versus with). N_int = 135, girls = 70, boys = 65. Time = linear effect of time points before, during and after TSST-C; Time² = quadratic effect of time points before, during and after TSST-C; COVID-19 = TSST-C was conducted before/during Sars-Cov-2 pandemic (0/1). Values indicate the estimated effect and corresponding standard error (SE). Bold values indicate significant variables of interest.

Abbreviations: BMI, body mass index; ICC, intraclass correlation coefficient.

Environmental-based gender-specific effects might explain the contradictory gender results. Our findings might also be due to the fact that males release more cortisol than females during the TSST-C (Liu et al., 2017). Cortisol secretion appears to be boosted by sleep-induced stress vulnerability as in this study boys and girls did not differ on average in their total cortisol levels. The results of the current study are more consistent with those of Bassett et al. (2015), who showed in college students that men’s stress responses were more dependent on self-reported sleep quality than women’s. The authors argued that effects of sex hormones on stress responsivity may outweigh possible effects of perceived sleep quality, which may also be a possible explanation for our results.

Moreover, one should note that although higher hormonal stress was evident, boys seemed to be unaware of this, as the subjectively rated stress levels were comparable for boys with high and low sleep disturbances, which may point to an inhibited perception of arousal.

In contrast to the emphasis on boys with sleep disturbances, girls with insomnia had experienced initial psychological stress, which, however, was not notable in physiological measures. This fits in with females reporting more fear, irritability and less happiness compared with males with regard to the TSST (Kelly et al., 2008), and when facing an unknown situation (Byrne, 2000). On the other hand, it reveals a gender gap in the perception of hormonal stress reactivity. Another explanation for this difference in perception would be that boys have a lower bodily awareness of emotions (Rueh et al., 2019). As low emotional awareness is associated with various mental disorders (Sendzik et al., 2017), this could also be an explanation for the particular stress reactivity in sleep-impaired boys. Alternatively, it is also possible that there was a systematic response behaviour by which boys attempt to conceal their actual level of stress, as the investigators were predominately female.

Regarding the ANS, contrary to expectations, we found no remarkable overall differences with respect to sleep in heart rate or HRV, or more precisely in parasympathetic activity (i.e. RMSSD), and in the quality of parasympathetic and sympathetic regulation (i.e. alpha 1) for the total sample. This is surprising as in general insomnia is associated with a higher heart rate (de Zambotti et al., 2018), although particularly for the TSST other authors found no differences in heart rate in young adults after sleep deprivation (Schwarz et al., 2018). Some previous studies also indicated an elevated sympathovagal imbalance with regard to sleep (Chen et al., 2017; Martin-Piñón et al., 2021), while others found no effect (Schwarz et al., 2018). In the present study, the sex-stratified analysis, however, revealed that boys, but not girls, with insomnia showed irregularities in heart rate and alpha 1 in relation to the recovery phase in the way of having smaller differences between measures during and after stress induction. These may indicate poorer adaptability of the ANS after an effort in boys with insomnia. The relationship between the HPA axis
and ANS was investigated by Agorastos et al. (2019), whose results support a vital role of the parasympathetic nervous system in the interplay between the ANS and the HPA axis in healthy adults. For adolescents with sleep disturbances, our findings suggest that poor sleep may more likely affect the stress response of the HPA axis and the ANS in boys than in girls. Future studies should gain a deeper insight into the physiological dynamics of the stress response and the effects of insomnia on it in this developmental period, not only for stress reactivity under current stress but also at rest, taking into account sex differences.

Our results should be understood in the light of some limitations. First of all, adolescents with diagnosed insomnia surprisingly did not show elevated cortisol secretion, as has been shown for those with self-reported high sleep disturbances. At the same time, this could provide an explanation: 55% of those with high self-reported sleep disturbances did not have an insomnia diagnosis, stressing that even those without insomnia often experience poor sleep and daytime sleepiness, as it is frequently the case in adolescence (Hein et al., 2020). This has methodological implications for future studies in terms of which sleep measure is used and how the control group is defined. As a consequence, it seems beneficial to distinguish at least three groups: individuals with healthy sleep; with minor sleep problems; and those with severe problems, the latter of which would include those with insomnia.

Secondly, in response to the fact that sleep disturbances are often accompanied by mental health problems, adolescents with clinically relevant psychological symptoms were excluded in this study. However, there was no control for medication use, which was not recorded in this study, but could potentially influence the results (Granger et al., 2009).

Thirdly, this study explored only the mere presence of insomnia. Previous studies have found greater alterations in the HPA axis and ANS in individuals with insomnia of the short sleep duration type (Fernandez-Mendoza et al., 2014; Jarrin et al., 2018). Therefore, sleep duration might be considered as a factor for stress reactivity in adolescents in the future. In this vein, an inclusion of objective measures of sleep, such as actigraphy, on stress reactivity would provide a more complete picture. For example, in adults higher cortisol and poorer cardiac sympatho-vagal balance were found to be associated with longer wake phases after sleep onset in the night immediately following the TSST, while neither sleep-onset latency nor total sleep time were affected (Chen et al., 2017).

| TABLE 5 | Unstandardized estimates for linear, quadratic and cubic changes in psychological stress throughout the TSST-C procedure (t1–t6) |
|----------------------------------|----------------------------------|----------------------------------|
| **Effect** | **Sleep disturbances** | **Insomnia** |
| | **All** | **Girls** | **Boys** | **All** | **Girls** | **Boys** |
| Fixed effects | | | | | | |
| Intercept | 2.68 (0.19)*** | 3.09 (0.28)*** | 2.55 (0.24)*** | 2.69 (0.13)*** | 2.71 (0.14)*** | 2.75 (0.16)*** |
| Female | 0.13 (0.15) | — | — | 0.03 (0.15) | — | — |
| Age/puberty | 0.20 (0.08)† | 0.12 (0.11) | 0.28 (0.12) | 0.21 (0.08)** | 0.21 (0.11)† | 0.26 (0.12)† |
| BMI z-score | 0.02 (0.06) | 0.04 (0.09) | 0.02 (0.09) | 0.03 (0.06) | 0.07 (0.09) | 0.02 (0.09) |
| COVID-19 | −0.21 (0.15) | −0.18 (0.21) | −0.22 (0.23) | −0.21 (0.15) | −0.14 (0.21) | −0.18 (0.23) |
| Group | 0.08 (0.19) | −0.25 (0.31) | 0.22 (0.26) | 0.21 (0.17) | 0.60 (0.23)** | −0.24 (0.26) |
| Time—linear change | −4.19 (3.11) | −5.66 (5.18) | −3.67 (3.82) | −3.02 (1.56) | −3.05 (2.33) | −2.88 (2.07) |
| Time²—quadratic change | −23.44 (3.09)*** | −26.25 (5.13)*** | −21.50 (3.82)*** | −25.19 (1.56)*** | −24.86 (2.32)*** | −25.40 (2.07)*** |
| Time³—cubic change | −3.46 (3.09) | −7.99 (5.13) | −0.36 (3.81) | −4.40 (1.56)** | −6.50 (2.32)** | −2.38 (2.07) |
| Group × Time | −0.57 (3.44) | −0.37 (5.60) | 0.44 (4.35) | −6.18 (2.99)** | −9.60 (4.15)** | −1.85 (4.32) |
| Group × Time² | −2.02 (3.42) | 0.73 (5.54) | −3.64 (4.35) | 0.60 (2.98) | −2.57 (4.14) | 5.08 (4.32) |
| Group × Time³ | −1.41 (3.42) | 1.54 (5.54) | −2.77 (4.45) | −0.93 (2.98) | −1.02 (4.14) | −0.54 (4.32) |
| Random effects—Variance components | | | | | | |
| Intercept | 0.46 (0.68) | 0.42 (0.65) | 0.52 (0.72) | 0.46 (0.68) | 0.43 (0.66) | 0.54 (0.73) |
| Time | 2.34 (1.53) | 5.35 (2.31) | 0.02 (0.14) | 1.34 (1.16) | 1.98 (1.41) | 0.00 (0.01) |
| $R^2_{marginal}$ | 0.29 | 0.29 | 0.28 | 0.29 | 0.31 | 0.28 |
| $R^2_{conditional}$ | 0.46 | 0.45 | 0.50 | 0.46 | 0.47 | 0.51 |
| $F^2$ | 0.09 | 0.09 | 0.09 | 0.09 | 0.11 | 0.08 |
| ICC | 0.17 | 0.16 | 0.22 | 0.17 | 0.16 | 0.23 |

Note: Group = Sleep disturbances (low versus high)/Insomnia (without versus with); N_all = 135, girls = 70, boys = 65. Time = linear effect of time points t1–t6; Time² = quadratic effect of time points t1–t6; Time³ = cubic effect of time points t1–t6; COVID-19 = TSST-C was conducted before/during SARS-CoV-2 pandemic (0/1). Values indicate the estimated effect and corresponding standard error (SE). Bold values indicate significant variables of interest.

* $p < 0.05; ** p < 0.01; *** p < 0.001.

Abbreviations: BMI, body mass index; ICC, intraclass correlation coefficient.
FIGURE 2  Means and 95% confidence intervals of salivary cortisol (a, b), heart rate (c, d), RMSSD (e, f), alpha 1 (g, h) and psychological stress response (i, j) separately for insomnia and sleep disturbances, and grouped by gender. B_lowSD/G_lowSD = boys/girls with low sleep disturbances; B_highSD/G_highSD = boys/girls with high sleep disturbances; B_noI/G_noI = boys/girls without insomnia; B_withI/G_withI = boys/girls with insomnia.
Fourthly, some studies found blunted, not elevated cortisol levels in relation with early lifetime stress (Young et al., 2021), depression and attention-deficit/hyperactivity disorder (Bernhard et al., 2021). In this study, we focused on acquiring deeper insight into overall individual-stress reactivity during the TSST-C and therefore refrained from directly comparing cortisol responders (i.e. those with a normal or high cortisol response) with non-responders (i.e. those with a blunted cortisol response). However, future longitudinal studies might address sleep as a factor for altered cortisol levels in adolescents, and thereby distinguish between hypo- and hypercortisolism as a long-term consequence of sleep problems.

Nevertheless, a strength of the present study was the inclusion of both psychological and physiological measurements of stress, which allowed a more comprehensive insight into the implications of dysregulated stress reactivity.

Taken together, the findings of higher overall cortisol reactivity in boys with sleep disturbances provides further evidence in favour for the hyperarousal model of insomnia. A more rigid ANS in boys with insomnia after stress suggests adjustment difficulties and potential long-term health problems (Goldberger et al., 2019).

Given that dysfunctions of the HPA axis and ANS are linked to poor sleep specifically in boys, future research should consider the intensity and duration of sleep problems to further understand why and when poor sleep causes dysregulated biological functions, taking into account sex differences. The centrality of integrating arousal-reducing techniques known to impact the stress system (e.g. mindfulness-based stress reduction; Reive, 2019) into sleep interventions is supported by our findings of increased cortisol secretion in youth with disturbed sleep.

**CONFLICT OF INTEREST**

The funders had no role in study design and administration, data analysis or interpretation, manuscript writing, or the decision to submit the paper for publication. All authors declare that they have no conflicts of interest.

**AUTHOR CONTRIBUTIONS**

Maren-Jo Kater was the principal author, who analysed the data, and drafted and revised the paper. All authors designed and monitored data collection, and revised the draft paper.

**DATA AVAILABILITY STATEMENT**

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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