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Abstract

The cross-stressor adaptation hypothesis—which posits that adjustment to physical stress as a result of regular physical activity and its effects on fitness crosses over to psychological stress reactivity—has been around for over four decades. However, the literature has been plagued by heterogeneities preventing definitive conclusions. We address these heterogeneity issues in a combined laboratory and daily life study of 116 young adults ($M = 22.48$ $SD = 3.56$, 57.76% female). The exposure, i.e., the potential driver of adaptation, was defined in three ways. First, a submaximal test was performed to obtain aerobic fitness measured as the VO_2max (kg/ml/min). Second, leisure time exercise behavior, and third, overall moderate-to-vigorous physical activity (MVPA), were obtained from a structured interview. Outcomes were autonomic nervous system (ANS) reactivity and affective responsiveness to stressors. ANS activity was measured continuously and expressed as inter-beat-interval (IBI), pre-ejection-period (PEP), respiratory sinus arrythmia (RSA), and non-specific Skin Conductance Responses (ns. SCR). Negative and positive affect were recorded after each experimental condition in the laboratory and hourly in daily life with a nine-item digital questionnaire. Linear regressions were performed between the three exposure measures as predictors and the various laboratory and daily life stress measurements as outcomes. Our results support the resting heart rate reducing effect of aerobic fitness and total MVPA in both the laboratory and daily life. We did not find evidence for the cross-stressor adaptation hypothesis, irrespective of ANS or affective outcome measure or whether the exposure was defined as exercise/MVPA or aerobic fitness.

KEYWORDS
affect, autonomic nervous system, cardiovascular fitness, cross-stressor hypothesis, physical activity

1 | INTRODUCTION

The hypothesis that regularly physically active individuals are not only more resilient to acute exercise but also to acute psychological stress has been around for over four decades (see Sothmann et al., 1996). The basis of this so-called cross-stressor adaptation hypothesis lies in the similarity between the physiological response to exercise and psychological stressors. One of these physiological responses is the activation of the autonomic nervous system (ANS). The basis of the idea is that the ANS response to a fixed dose of exercise becomes lower after repeated exposure to (intense) physical activity, with additional faster recovery (as reviewed by Michael, 1957). This so-called "training" effect is a combination of increased organ responsiveness (stroke volume, muscle capillarization), changed feedback from exercising muscles, and central nervous system adaptations, including changes in the "central command" or the feed forward engagement of ANS by the brain. These adaptations, especially the scaling down of the anticipation of the required ANS activity, may then be inherited by any other type of stressor that engages anticipatory ANS responding, like challenging cognitive tasks and social-evaluative stressors (Sothmann, 2006; Sothmann et al., 1996). Cross-stressor adaptation could be an important contributor to the well-established health benefits of regular physical activity on many major diseases by countering the detrimental effects of repeated and prolonged cardiovascular stress reactivity (Gerber & Pühse, 2009).

A large amount of studies have sought to provide empirical support for the cross-stressor adaptation hypothesis, but results have been mixed and even systematic reviews and meta-analyses don't come to unequivocal conclusions (Forcier et al., 2006; Huang et al., 2013; Jackson & Dishman, 2006; Mücke et al., 2018). A first potential source of heterogeneity in findings is the mixture of studies using exercise intervention ("training") and studies using cross-sectional comparisons of regular exercisers versus less regular or non-exercisers. Both designs have strengths and weaknesses but mixing them is a strong source of heterogeneity. If duration of the exposure to regular exercise is the main determinant of cross-stressor adaptation, then many intervention studies may not have trained the participants long enough to induce the adaptation. Cross-sectional studies can be at a substantial advantage in this respect. However, if co-occurring confounders such as socioeconomic position and genetics are the main source of reduced stress-reactivity seen in regular exercisers, then the outcome of comparisons between exercisers and non-exercisers would depend on the variance of such confounders in the study population. Studies using randomization to assign participants to exercise versus control manipulations do not suffer from this bias.

A second potential source of heterogeneity in findings is the mixed use of regular physical activity versus measures of cardiorespiratory fitness as the independent variable explaining differences in cardiovascular stress reactivity. These concepts are often treated as interchangeable, whereas empirical observations of correlations between regular physical activity and fitness measures typically do not exceed 0.40 (Aadahl et al., 2007; Emaus et al., 2010; Minder et al., 2014; Morrow & Freedson, 1994; Siconolfi et al., 1985). Therefore, mixing cardiorespiratory fitness and physical activity effects on stress reactivity is likely to induce heterogeneity.

In addition, whereas cardiorespiratory fitness has a well circumscribed definition, regular physical activity is a complex construct which can be defined and assessed in different ways. A frequently used measure is total daily physical activity derived from a self-report questionnaire, which is subject to recall and response bias, and therefore frequently underestimated as well as overestimated (Prince et al., 2020). Fortunately, self-reporting becomes more reliable for moderate-to-vigorous activities, particularly when they are voluntary and salient like sports and exercise activities in leisure time (van der Zee et al., 2020; van der Zee, Schutte, & et al., 2019; van der Zee, van der Mee, et al., 2019). Reliable self-reports may be feasible when activities have a relatively fixed intensity and duration, like minutes spent on cycling to work or taking a well-defined walk, but become difficult for activities which are more variable and lack clear boundaries. For the latter, accelerometer assessment is a far more reliable alternative (Slootmaker et al., 2009). The above makes clear that different definitions result in different physical activity measures, which induce heterogeneity that could distort possible cross-stressor adaptation effects on stress reactivity (Forcier et al., 2006).

Even when studies restrict themselves to measuring the uniformly defined construct of aerobic fitness, findings on cross-stressor adaptation remain confusing. This is illustrated by two meta-analyses performed in 2006 on the specific relationship between aerobic fitness and cardiovascular reactivity to acute laboratory stress. The meta-analysis by Jackson and Dishman (2006), using VO_2max as the indicator of aerobic fitness, found an overall *higher* cardiovascular stress reactivity in more fit participants, particularly for heart rate (HR) and heart rate variability (HRV) reactivity. This higher reactivity was, however, paired to a better recovery after the stressor, which could be a relevant advantage when dealing with repeated stress exposure. The meta-analysis by Forcier et al. (2006) with baseline HR as their aerobic fitness indicator showed partially contrasting results. They report an

overall *lower* HR and systolic blood pressure (SBP) reactivity to stress, although they did fully corroborate the faster HR recovery after stress. Both studies also illustrate that the meta-analytic effect sizes are very small and strongly heterogeneous across included studies. Moderator analyses showed this heterogeneity to be partly caused by the previously mentioned differences in study design (cross sectional or intervention studies) and the population included (healthy or at risk, general population or high stress occupation, young or older, males and/or females) but another important determinant was the type of stressor used (physiological, mental or social-evaluative). This issue of heterogeneity as a source of mixed results was recently addressed again in a systematic review by Chauntry et al. (2022). They focused solely on self-reported physical activity as their fitness measure. Two out of the six studies that measures ANS stress reactivity identified a significantly lower HR response to stress in more active individuals, and one study reported higher HR recovery in more active individuals. None of the studies observed an effect for HRV.

To specifically reduce the heterogeneity caused by the use of different types of stressors, Mücke et al. (2018) performed a systematic review including only a single stress paradigm, the Trier Social Stress Test (TSST). They also only included studies with a cross-sectional design, and explicitly tested for differences in the definition of the exposure variable, e.g., using dichotomies based on measured aerobic fitness level versus dichotomies based on the amount of regular physical activity. Furthermore, they took into account the assessment methods for physical activity. In spite of homogeneous cross-sectional design and the use of a single stressor, results were again mixed. This could be largely attributed to the definition and assessment of the exposure variable. From the studies included by Mücke et al. (2018) that included ANS reactivity as their outcome measure, a relationship between questionnaire derived physical activity and lower HR reactivity combined with faster recovery can be observed. Studies using VO$_2$max as the aerobic fitness measure, however, found a higher HR reactivity to the stressor (albeit only significant in women) again paired to a faster recovery. While the single study using accelerometers to obtain total physical activity did not find an effect of physical activity levels on stress reactivity or recovery. In addition Mücke et al. (2018) also addressed the effect of physical activity on psychological stress reactivity. They found that overall participants who engaged more in physical activity (measured by either a questionnaire or accelerometry, but not VO$_2$max) showed a lower negative affective response to the stressor. The review by Mücke et al. (2018) again illustrates the importance of the exact construct used as the exposure variable.

While the current literature provides some support for the association of fitness with higher reactivity, and of physical activity with lower reactivity, and for both fitness and physical activity to yield faster recovery from stress, laboratory studies have not yet provided an unequivocal answer to the validity of the cross-stressor adaptation hypothesis. Importantly, most laboratory tasks used in the studies reviewed so far typically elicit weaker physiological and psychological responses of much shorter duration than those found for naturalistic stressors (Peronnet & Szabo, 1993; Sothmann et al., 1991). To increase the ecological validity of cross-stressor adaptation effects, and with that their clinical relevance, Tonello et al. (2014) reviewed studies done between 2007 and 2013 that examined the association between questionnaire-based physical activity, daily HRV levels and the subjective experience of work stress. Overall, they found that higher levels of work stress were associated with lower HRV, but the evidence for a stress-buffering effect of physical activity on HRV remained inconclusive (Tonello et al., 2014). Four more recent studies directly linked questionnaire-based physical activity and VO$_2$max with the effects of daily stress on cardiovascular and affective measures (Chovanec & Gröpel, 2020; Gnam et al., 2019; von Haaren et al., 2016; Schilling et al., 2020). The first of these studies was performed in 61 inactive male engineering students of which half engaged in a 20-week aerobic training program. They found reduced heart rate variability reactivity during an examination period in students who participated in the aerobic exercise training program in comparison to sedentary controls (von Haaren et al., 2016).

The second study was conducted in firefighters during the final exam of their vocational training program. No beneficial effect of either physical activity or VO$_2$max on the HR and HRV response to the exam was found. Instead, more physically active firefighters showed higher cognitive stress appraisal levels compared to the less physically active firefighters (Gnam et al., 2019).

The third study was performed in a population of 173 police officers (66.5% male, mean age 37). Higher VO$_2$max was associated with reduced HRV reactivity to perceived acute work stress and increased HRV recovery at night. However, no relationship between VO$_2$max and positive or negative affect was observed (Schilling et al., 2020).

The fourth study was performed in 52 female college students who engaged in either an eight-week endurance exercise training program ($N = 18$), an eight-week resistance training group ($N = 21$) or were placed on a waiting list (the control group; $N = 13$). Both training programs led to significantly increased VO$_2$max and reduced the subjective experience of daily life stress and HR recovery time from audiovisual stress stimuli, as compared to the control group (Chovanec & Gröpel, 2020). While informative, the

expanding body of daily life studies does not yet elucidate the validity of the cross-stressor adaptation hypothesis. They exhibit a similar heterogeneity as the larger body of work using laboratory stressors.

The aim of the current study was to re-examine the association of both aerobic fitness and regular physical activity with stress reactivity and recovery, with specific attention to methodological aspects that could moderate these associations. We include three different exposure measures, (1) aerobic fitness, operationalized as the VO_2max derived from a submaximal test, (2) self-reported weekly minutes spent on sports and exercise activities in leisure time, and (3) an index of the total amount of moderate-to-vigorous physical activity which includes the above sports and exercise activities but also self-reported weekly minutes spent on walking and cycling. Whereas most previous studies have used heart rate and blood pressure reactivity as the main outcomes, we focus on the activity of the sympathetic (SNS) and parasympathetic (PNS) branches of the ANS separately. These are the main effectors causing the feed forward changes in heart rate (Robinson et al., 1966) and blood pressure (Yang & Zubcevic, 2017), and should therefore more directly reflect adaptations in the central ANS control over the cardiovascular system. In addition, different patterns of SNS and PNS co-activation, co-inhibition, or reciprocal activation/inhibition can lead to similar end-organ responses (Berntson et al., 1994) and cross-stress adaptation may well depend on a change in such patterns. SNS reactivity is measured using changes in the cardiac pre-ejection period (PEP) and non-specific skin conductance response (ns.SCR) frequency. PNS reactivity is measured using changes in respiratory sinus arrhythmia (RSA), taking into account parallel changes in respiration rate. Lastly, inter-beat-interval (IBI) is included as measure that reflects both SNS and PNS activity.

ANS reactivity and recovery are measured in a controlled laboratory setting using both cognitive and social-evaluative stressors. To specifically address the cross-stressor hypothesis in a naturalistic daily life setting, we further use a continuous 24-hr measurement of ANS activity combined with an hourly digital diary to obtain information on work or leisure setting, level of like or dislike of their current activity, and positive and negative affect state.

2 | METHOD

2.1 | Study population

The main focus of the parent project of this study was the validation of a wristwatch-based technology to assess the relationship between ANS activity and stress in daily life.

Recruitment of participants and the laboratory protocols are described in detail elsewhere (van der Mee et al., 2021). Briefly, participants were required to be between the age of 18 and 48, Dutch speakers, and currently employed, or in a schooling trajectory. Exclusion criteria were a body-mass index above 30, heart disease, high blood pressure, high cholesterol, diabetes, thyroid or liver disease, and use of antidepressants, anticholinergics, or any other medication that has been shown to influence the SNS. Female participants were measured within the first two weeks following the last day of their menstrual cycle to account for hormonal changes.

Participants who were students received research credits, while other participants were compensated with a €50 gift voucher. All participants provided written informed consent before the start of the experiment. The study was approved in institutional review by the VUmc medical ethical committee (METc VUmc #2017.374, ABR #NL62442.029.17).

2.2 | Procedure

Participants visited the laboratory on two consecutive days. During their initial visit to the laboratory (~1 h) participants provided informed consent and were interviewed about their physical activity behaviors. During this structured interview the participants' systolic and diastolic blood pressure (SBP, DBP) were measured twice. Subsequently, the ANS measuring devices were applied to the participant to continuously measure ANS activity. The participants were provided with an iPod containing the questionnaire application. The experimenter practiced all items of the questionnaire with the participant to make sure they understood each item. They were informed that the iPod would go off hourly between the hours of 07:30 a.m. and 11:00 p.m. Each participant received 15 diary prompts but was allowed to manually fill in extra diaries by opening the app if they went to bed after 11 p.m. or woke up earlier than 7:30 p.m. They were also informed that a random jitter of 15 min was added around each diary prompt, to reduce expectation effects.

Once equipped with the measuring devices and the iPod, participants left the laboratory for a day of daily life monitoring. During the 24-hr recording participants only a few restrictions of normal activities were applied. Participants were requested not to take a bath or engage in water sports. They were asked to remove the devices (but not the electrodes) during and reattach the devices afterwards.

Participants returned the next day for participation in the laboratory protocol. Upon their return, it was verified that all measurement equipment was still in working

order. Next, to increase stress, participants were informed that footage of their facial expressions, posture and voice would be recorded during the experiment. Furthermore, their scores on the task were tracked on a score board containing other participants scores for comparison on their performance. Then all experimental manipulations were presented in a fixed order (see Table 1). After each stressor the participants were asked to fill out a short affect questionnaire (see Table 1).

After the experimental session, all devices were removed, and participants were provided the opportunity to use a nearby shower. The experiment ended with a debriefing in which they were informed that the tasks were purposefully made so difficult so that they would be impossible to perform without errors. They were explicitly told that the test score rankings were only added to increase the stressfulness of the task and did not reflect their actual ability. Furthermore, they were informed that no actual voice or video recording had been made.

2.3 | Demographics

A structured interview was conducted before the start of the experiment to ensure participants were eligible to partake in the study. In addition the interview included questions regarding their age, gender identity, physical activity behavior (for details see Section 2.7 Physical Activity), subjective mental health and physical health on a scale of 1 (very poor) to 5 (very good), and experienced work and home stress on a scale of 1 (never) to 5 (very often).

2.4 | Physiological measures

The physiological measures IBI, RSA and PEP and ns.SCR on the palm of the hand were obtained with a VU-AMS device (version 5-wire 5fs). The VU-AMS is a lightweight portable device that has been used to measure ANS activity in over 300 scientific studies (see: http://www.vu-ams.nl/research/publications/ for an overview). It records electrocardiogram (ECG) and impedance cardiogram (ICG) from five adhesive 55 mm Kendall H98SG hydrogel ECG electrodes (Medtronic, Eindhoven, Netherlands) placed on the chest and back of the participants with a recording frequency of 1000 Hz (de Geus et al., 1995; Willemsen et al., 1996). VU-AMS data was analyzed with the Vrije Universiteit Data Acquisition and Management Software (VUDAMS version 4.6, available at: http://www.vu-ams.nl/support/downloads/software/). For each experimental condition average values for each ANS measure were calculated. The IBI is calculated based on the time difference between two successive R peaks. RSA is calculated by means of a peak valley method, which combines the R-peaks time series with the impedance derived respiration cycle. In this method the shortest IBI during each inspiration and the longest IBI during each expiration are detected. Then the former is subtracted from the latter. When the calculation of the RSA results in zero or negative values they are coded as zero (de Geus et al., 1995; Goedhart et al., 2007). RSA is well validated measure of PNS activity (Berntson et al., 1993; Katona & Jih, 1975; Migliaro, 2020) and has been frequently

TABLE 1 Experimental timeline[a]

Experimental condition	Duration (minutes)	Affect measurement
Baseline	3	Yes
Tone Avoidance (TA)	4	Yes
Rest	2	No
Short Sing-a-Song Stress Test ($_{short}$SSST)	6.5	Yes
Rest	2	No
Paced Auditory Serial Addition Test (PASAT)	4	Yes
Rest	2	No
Raven's progressive matrices (RPM)	4	Yes
Break (application of CosMed)		
Treadmill intensity 1 (4.5–5 km/h)	4	No
Treadmill intensity 2 (6–6.5 km/h)	4	No
Treadmill intensity 3 (7.5–8 km/h)	4	No
Treadmill cooling down (3.7–4 km/h)	3	Yes
Rest	3	Yes

[a]The timeline only presents task relevant for the present paper in their presentation order. The full timeline of all experimental conditions can be found in (van der Mee et al., 2021).

studied in relationship to stress (e.g., Beauchaine, 2015; Beauchaine et al., 2019; Campbell et al., 2019; Lane et al., 1992; Tonhajzerova et al., 2016). The PEP is obtained by calculating the time difference between the start of ventricular depolarization (Q onset) in the ECG and the time the aortic valve opens (B point) in the ICG (Nederend et al., 2018; Willemsen et al., 1996). For each time segment of interest a single averaged ICG complex was derived by means of ensemble averaging of the ICG signal over all R-peaks in the conditions, as explained by Riese et al. (2003). Extensive construct and criterion validity has been demonstrated for this method (Nederend et al., 2018; Willemsen et al., 1996). Various studies have shown that the PEP is response to stress, in which a shorter PEP (due to increases SNS activity) is indicative of more stress (Brindle et al., 2014; Rahman et al., 2018; van der Mee et al., 2020, 2021). The VU-DAMS software automatically detects and scores the various attributes necessary to calculate the IBI (R-peaks), RSA (R-peaks and respiration) and PEP (Q-onset and B-point). All data scoring of the VU-DAMS was manually checked and if necessary corrected.

For the laboratory section of the study ns.SCRs were obtained from electrodermal activity (EDA) as measured with the VU-AMS on the palm of the hand and on the wrist by a wristwatch. During the daily life section EDA was only obtained with a wristwatch. The wristwatch was a CE approved wearable skin conductance wrist sensor, type DTI5 (Discreet Tension Indicator version 5, Philips), and was used to measure ns.SCR frequency. This wristwatch has been shown to sufficiently capture SNS activity (van der Mee et al., 2021). The ns.SCRs frequency is defined as the number of peaks per minute. For palm EDA during the laboratory recording, the ns.SCR frequency was obtained using the EDA master toolkit (Joffily, EDA Master Toolbox, 2012) in MATLAB. For wrist EDA during laboratory and daily life recording, the ns.SCR frequency was obtained by an internal method of peak detection that makes use of a curve fit method. For more details on EDA scoring see van der Mee et al. (2021). Ns.SCR frequency has been shown to relate to negative emotions (Nikula, 1991; van der Mee et al., 2021), arousal (Nikula, 1991), and stress (Kelsey, 1991; Miller & Shmavonian, 1965) We recently showed this measure to perform even better than the widely-used skin conductance level to index changes in SNS activity across a wide variety of stressors (van der Mee et al., 2021).

Ambient temperature and humidity were continuously measured with a thermosensor (Hygrochron iButton, UK) worn on the outer clothing. In addition skin temperature was continuously measured from a thermosensor (Thermochron iButton, UK) placed directly onto the skin under the left clavicle bone using double adhesive rings

(20 × 5 mm) for cup electrodes. In addition continuous passive sensing through a triaxial accelerometer, embedded in the VU-AMS, was used to detect activity levels. Average activity level was computed by the root of the mean of the squared the X-, Y-, and Z-axis accelerations.

During the structured interview, SBP and DBP were measured twice with the Omron M4-I, HEM 752A. Resting SBP and DBP were calculated by taking the mean of the two measurements.

2.5 | Affect

An iPad containing an in-house built electronic diary application was provided to participants to report their affect at set times in the laboratory. For the daily life section participants received an iPod containing the same electronic diary application. In both settings, affect was rated with a shortened version of the Maastricht Questionnaire (Myin-Germeys et al., 2001). Positive affect scores were obtained by asking the participants to rate on a scale of 1 (not at all) to 7 (very) whether they felt relaxed, cheerful, enthusiastic and content and averaging the score over the 4 items. Negative affect was obtained by averaging the scores for 5 items: insecure, lonely, anxious, irritated, and down. In daily life, participants also rated the degree of liking the activity they were engaged in at that moment in time (work/study, leisure, household chores, transportation, relaxing, sleeping). Participants indicated whether or not they would rather be doing something else (on a scale of 1 (strongly like)—7 (strongly dislike)). The like-dislike item was recoded into a binary variable based on the grand median score, in which a score ≤ median indicated they liked the activity and a score > median indicated they did not like the activity they were doing.

2.6 | VO₂max

The maximal volume of oxygen uptake (VO_2max) is derived from a submaximal test. Participants engaged in a treadmill exercise at 3 incremental stages of speed (males: 5, 6.5, and 8 km/h; females: 4.5, 6, and 7.5 km/h), each lasting 4 min. After a 3-min cooling-down on the treadmill (males: 4 km/h, females: 3.7 km/h) participants sat down for a 3-min recovery stage.

Volume of oxygen (O2) uptake and carbon dioxide (CO2) production were recorded breath-by-breath with a telemetric gas exchange system (Cosmed K5, Rome, Italy). During the course of the experiment, the main sample unit and the battery pack were attached to the back of the subject. Before each test, the O_2/CO_2 analysis system was calibrated with ambient air and a gas

mixture that had an O_2 concentration of 16% and a CO_2 concentration of 5%. The calibration of the turbine flowmeter was performed by via a 3-L syringe (Crouter et al., 2019).

The last minute of each incremental treadmill stage was included in a linear regression between O_2 uptake and HR (derived from the VU-AMS) for each participant separately to derive their individual regression equation. The last minute was chosen to ensure participants had reached a steady state. Maximum oxygen uptake was then calculated by entering the maximal heart rate, defined as 220 minus the participant age, into their individual regression equation. The resulting value was divided by the participant's weight resulting in VO_2max as measured in milliliter per kilogram per minute. The validity of a graded submaximal test to predict an individual's VO_2max has been shown to correlate strongly with the actual VO_2max (Ekblom-Bak et al., 2014; Grant et al., 1995; Schutte, Nederend, Hudziak, Bartels, et al., 2016).

2.7 | Physical activity

During a structured interview detailed information regarding the participants physical activity was collected. The interview included the following questions on exercise behavior: Do you exercise regularly? What type of exercise do you partake in? For how many years? How many months a year? How many times a week? How many minutes per time? Only exercise activities performed at least six months a year and at least once a week were included (thereby excluding ski holidays, sailing camps, swimming only during the summer, and similar). When the reported number of occasions, or session lengths were variable, an average number of occasions or session length was calculated. There was no limit on the number of different exercise activities participants could report, and all were included in the study. For each exercise activity the total minutes spent on exercising per week was calculated (number of occasions x session length) and multiplied by their metabolic equivalent score (METscore) value derived from the 2011 Compendium of Physical Activities (Ainsworth et al., 2011) to obtain exercise activity in metabolic equivalent hours (MET-hours).

With regard to other types of common moderate-to-vigorous physical activity (MVPA), the following questions were included: How many minutes in total do you spend walking during the workweek? How many minutes in total do you spend walking during the weekend? How many minutes in total do you spend cycling during the workweek? How many minutes total do you spend cycling during the weekend? This excluded walking or cycling mentioned under the exercise activities but included

walking/cycling as a means of transportation, walking a dog or walking/cycling for relaxation. The total minutes per week spent walking and cycling (sum of weekdays and weekend days) was multiplied with their METscore and added to the exercise METhours to obtain total energy spent on MVPA.

The questions included in the interview are obtained from the questionnaires used by the Netherlands Twin Register to quantify, amongst others, leisure time exercise behavior and MVPA (van der Mee et al., 2018; van der Zee, van der Mee, et al., 2019; Willemsen et al., 2013; Schutte, Nederend, Hudziak, de Geus, et al., 2016). Quantification of exercise and MVPA in terms of their METs does come with a limitation (Byrne et al., 2005; Franklin et al., 2018), but is currently the only metric available to take into account exercise intensity in addition to exercise time when information on heart rate and oxygen consumption during physical exertion in daily life are not available. The exercise METhour construct has been related to amongst others well-being (Stubbe et al., 2007) and mental health disorders (de Moor et al., 2006; de Moor et al., 2008). Furthermore this construct has been shown to have high (>0.82) test-retest reliability and high temporal stability, even across periods of 20 years (de Geus et al., 2014; Stubbe et al., 2006; van der Zee et al., 2020).

2.8 | Stress reactivity and recovery

2.8.1 | Laboratory

The mental stressors used in this study are the Tone avoidance (TA) task and the Paced Auditory Serial Subtraction (PASAT) task. The TA task aims to induce effortful active coping in which participants have to avoid a loud tone by pressing a button on the opposite site of an "X" presented on one of the four corners a computer screen (de Geus et al., 1990; van der Mee et al., 2020). The PASAT is a calculus task with a staircase algorithm to measure capacity and rate of information processing and sustained and divided attention. Single digits are presented every 3 seconds and the respondent must add each new digit to the one immediately prior to it before the next digit is presented (Sampson, 1958; Sampson & MacNeilage, 1960; Tombaugh, 2006).

The social evaluative stressors used in this study are the short Sing-a-Song-Stress-Test (SSSTshort) and the Raven Progressive Matrices IQ (RPM) test. In the SSSTshort participants unexpectedly have to sing a song out loud in front of a camera and the experimenter (van der Mee et al., 2020). The RPM test (Raven, 2003) was timed, participants had to solve as much matrices as possible in a 4-min time window. They were informed that

the more correct answers they gave the higher their IQ score would be.

Laboratory ANS stress reactivity values were calculated by subtracting the mean value during the baseline condition from the mean value during the respective stress tests for each ANS measure (IBI, RSA, PEP and ns.SCRs 2x) and respiration rate. Recovery values were calculated by subtracting the mean value during the 2-min rest period following each stress task from the mean value during that respective stress task. Reactivity in positive and negative affect was obtained by subtracting the scores of the affect questionnaire filled in after the baseline sitting condition from the scores immediately after the stress test. Since no affect was measured after resting periods no affect recovery could be calculated.

To obtain a single mental stress reactivity and recovery score per ANS measure, the calculated reactivity and recovery scores from the TA task and the PASAT were averaged. Similar, to obtain a single social-evaluative stress reactivity score, the calculated reactivities from the SSSTshort and RPM were averaged. Since the RPM did not have a recovery period following the task, the social-evaluative recovery score is equal to the SSSTshort recovery score.

The mean ANS and affective values during the baseline condition were also considered variables of interest.

2.8.2 | Daily life

For each diary entry the average ANS values during the 5 min preceding the entry were calculated. Regarding sleep, hourly averages were created from the reported moments of going to sleep to the reported moment of getting up (both verified by the accelerometer signal). For each participant, the average ANS values were calculated across all valid 1-hr sleep epochs ("sleep") and across all valid 5-min periods segments that met the category criteria: work/study, leisure time, liked activity (<median), not liked activity (≥median), awake (irrespective of activity performed). Due to the large influence of major body movements on ANS activity (Fu & Levine, 2013) during the awake period, only the 5-min periods with accelerometer values < 50 milli G acceleration, consisting of minor body movements, were included. Only if a participant had at least 3 observations for a given activity category, his/her data for that category were included in the analyses.

From the daily life data two physiological stress reactivity measures were derived and one recovery measure. Stress reactivity was defined as (1) the difference between work and leisure activities (calculated as work—leisure) and (2) the difference between liked and disliked activities (calculated as dislike—like). Daily life stress recovery

was defined as the difference between awake and sleep (sleep—awake).

A similar approach was applied to the positive and negative affect scores to obtain subjective stress reactivity measures. A mean value was calculated over all diary entries for which a valid ANS value was available, and these were also averaged across work/study, leisure time, liked activity, disliked activity, and total time awake. Subjective stress reactivity scores were computed for positive and negative affect separately, one by contrasting affect during work vs. leisure and one by contrasting affect during disliked vs. liked activities.

2.9 | Covariates

Several variables were of interest as possible covariates. First, there is evidence that males and females respond differently to stressors with regard to their ANS response, with males being "vascular" reactors and females "cardiac" reactors (Huang et al., 2013). In addition males have, on average, a higher VO_2max than females (Wang et al., 2010). A second covariate is age, since with age VO_2max decreases (Wang et al., 2010), ANS activity changes (Peters et al., 2020), and physical activity decreases (Sallis, 2000; van der Zee, van der Mee, et al., 2019).

In addition to age and biological sex, a few other covariates were considered that may impact ANS reactivity/recovery. First, the electrodermal activity measure ns.SCR frequency could be influenced by the ambient temperature and/or humidity and body temperature due to involvement of sweating in thermodynamics (Boucsein, 2012). Though our previous work in the laboratory has shown that the ns.SCR frequency measure is less sensitive to thermodynamic effects as compared to skin conductance levels (van der Mee et al., 2021), in a daily life setting these factors are much more dynamic. Second, changes in respiration rate may drive changes in RSA independent of changes in PNS activity (Grossman & Taylor, 2007) and we therefore recorded changes in respiration rate using the thorax impedance signal as outlined previously (Houtveen et al., 2006).

2.10 | Analytical strategy

Before analyses all variables were checked for outliers. A value was considered an outlier if it deviated more than $4.5 \times SD$ from the grand mean. Next, we performed a manipulation check to assess whether the tasks and recovery periods induced significant changes in ANS activity. Paired-samples *t*-tests were performed for all stress reactivity (task vs. baseline) and recovery (task vs. recovery) contrasts.

Testing of the main hypothesis of cross-stressor adaptation revolved around establishing an association of the aerobic fitness and regular physical activity traits with the stress reactivity and recovery scores, across multiple tasks and settings. Separate linear regression models were run with either VO_2max, MVPA, and exercise as predictors and either ANS (IBI, RSA, PEP, ns.SCR) and affect (NA, PA) baseline, reactivity, and recovery values as outcomes. A total of 93 (31 for each physical activity measure) linear regression model were performed for the laboratory data and a total of 78 (26 for each physical activity measure) regression models were performed for the daily life data.

For the laboratory, the ANS and affect outcomes were baseline stress levels, mental stress reactivity, mental stress recovery, social stress reactivity and social stress recovery. For daily life the outcome variables were average levels during sleep, average levels during general wakefulness, work-leisure reactivity, dislike-like reactivity and awake-sleep recovery. Because of sex and age differences observed in VO_2max and ANS reactivity, we checked whether the inclusion of sex or age in the regression analyses changed the results, which they did not. However, they were still included in all analyses. Finally, the analyses were rerun for RSA and EDA with variable-specific covariates added to the respective models (i.e., temperature/humidity for EDA, and RR for RSA).

The relative explained variance in physiological stress reactivity by the exposure measures on the outcomes within each model was based on the partial R^2, calculated with the rsq.partial() function of the "rsq" package in R. The partial correlation coefficient reflects the strength of the relationship between two variables after the correlation of both the outcome and the predictor variable with the covariates is taken into account. To ease comparison with meta-analytic results we additionally report the Cohen's d based on the partial r (obtained by taking the square root of the reported R^2) with the formula: $d = 2 \times r / \sqrt{(1-r2)}$."

To account for multiple testing while taking into account that the number of effective tests is lower than the total tests, we used the correction for non-independent tests implemented in the R package *meff* (Nyholt, 2005; Salyakina et al., 2005). Separately for the laboratory and daily life data, the zero-order correlation matrix among all stress reactivity and recovery and the three physical activity variables was used to compute the number of effective tests (35 for the laboratory and 30.6 for the daily life data). Significance levels for the laboratory and daily life tests were adjusted from nominal 0.05 to $p = .05/35 = .0014$ and $p = .05/36 = .0016$. Power analyses (performed with R package "pwr") showed that with a df(3,116) and a significance level of 0.0014 we had a power of 0.02 to detect a small effect (f2 = 0.02/(1–0.02)), a power of 0.78 to detect a

medium effect (f2 = 0.15/(1–0.15)) and a power of 0.99 to detect a large effect (f2 = 0.35/(1–0.35)). The minimal effect size (quantified as Cohen's d) that could be identified in the current sample with a power of 70%, df(3,116), and a p-value set at a nominal $p = .05$ was medium ($d = 0.57$). This indicates that the power of the current study to detect the small effects for HR reactivity (d 95% CI = 0.05–0.11) and recovery (d 95% CI = −0.35 to −0.19) reported by the meta-analyses of Jackson and colleagues (2006) was likely low. However, as mentioned in the introduction, these analyses were plagued by heterogeneity issues which might have reduced the meta-analytical estimates for the effect sizes. Indeed, the daily life studies by von Haaren et al. (2016) and Chovanec and Gröpel (2020), show far larger effect sizes ranging from $d = 0.34$–0.66. Using more strict definitions of the fitness/physical activity predictors and a variety of homogenous stressors, including negatively valued daily life activities, we expected to find at least medium effect sizes.

3 | RESULTS

3.1 | Study population

Usable data were obtained in 116 participants out of 121 participants originally recruited in the study. Two participants were excluded because they were outliers in terms of age (they were > 45, while all other participants were ≤ 30). One participant was excluded because their data was an outlier on all ANS measures, one participant had insufficient data quality, and one participant withdrew from the study and requested their data to be removed. The final sample had a mean age of 22.48 ($SD = 3.56$) and 57.76% were female. The majority of the participants were students (81.0%), had good self-rated mental health (21.55% very good, 62.93% good, 12.07% intermediate, 3.44% poor, 0% very poor), and good self-rated physical health (13.79% very good, 69.82% good, 13.79% intermediate, 2.58% poor, 0% very poor). Experiences stress at work (3.44% never, 52.58% sometimes, 29.31% frequently, 12.93% often, 1.74% always) or at home (17.24% never, 56.03% sometimes, 18.96% frequently, 6.89% often, 0.86% always) was low, with most participants reporting less than frequent stress. For 23 participants VO_2max could not be calculated due to device malfunction (10), missing data (7), too few valid data points for analysis (3), or outlying VO_2 value (3). The mean VO_2max was 43.95 ml/kg/min ($SD = 9.29$), mean MVPA was 73.92 MET-hours ($SD = 48.96$), and mean exercise was 37.19 MET-hours ($SD = 40.11$). The mean SBP was 116.08 mmHg ($SD = 10.88$) and mean DBP was 71.20 mmHg ($SD = 8.31$). Consistent with previous findings males had a higher VO_2max compared to females

($\Delta M = 8.55$, $t(63.66) = 4.61$, $p < .001$) (Wang et al., 2010), but there were no differences with regard to MVPA ($\Delta M = 10.66$, $t(94.01) = 1.13$, $p = .26$) and exercise MET-hours ($\Delta M = 14.22$, $t(83.83) = 1.82$, $p = .072$).

Compliance for e-diary entries during the daily life part of the experiment was good. On average participants had 13.5 entries out of the 15 prompts (range: 4–19, $SD = 2.98$). Two participants did not have at least three observations for any activity category and were excluded from the analyses. One participant was removed due to poor overall data quality. For the wrist ns.SCR analyses one outlying value was removed in liked activities, one in leisure and two for dislike reactivity. The other variables contained no outliers.

Some participants only had three or more valid observations for a subset of conditions, leading to different numbers or participants included in each condition. Due to the exclusion of data segments that contained a high amount of movement, we lost data for half of the participants with regard to the work category. An overview of the number of participants per category per measurement is given in Table 2.

An overview of the mean values and SDs for our outcome measures of interest for the laboratory part is presented in Table 3 and for the daily life part in Table 4.

3.2 | Stress reactivity

The manipulation check showed that both mental and social stress elicit a significant ANS stress response, lowered positive affect and increased negative affect (Table 5). In the recovery period after the mental stressors the SNS activity (IBI, PEP, ns.SCR) significantly decreased and PNS activity (IBI, RSA) significantly increased again. A similar pattern is observed for social stress recovery, except for the palm ns.SCRs which remained elevated during the recovery period. Table S1 contains the full correlation matrix of variables measured during the laboratory session.

Less consistent results were found when comparing the different daily life activities. Participants had comparable ANS and affect values during leisure time and work, although negative affect was higher and IBI was lower at work (Table 6). Participants also had comparable ANS activity during liked and disliked activities, although with significantly higher positive affect and lower negative affect during activities they enjoyed with a strong effect size. However, stress reactivity values did show large individual differences as shown by the standard deviations in Table 6. Results were more in line with expectations for the contrast between sleep and awake. This was highly significant for IBI, RSA and PEP. On average participants had a higher IBI, RSA and PEP during sleep, with a very strong effect for IBI and a medium to strong effect for RSA and PEP. Again substantial individual variation was seen in this recovery. Table S2 contains the full correlation matrix of variables measured during daily life recording.

3.3 | Predicting reactivity and recovery from fitness or physical activity

For all analyses, addition of the temperature and humidity covariates had little influence on the relationship between fitness/MVPA/exercise and ns.SCR frequency. Therefore results are reported without addition of these covariates. The linear regression analyses for the laboratory stressors (Table 7) and daily life (Table 8) show a significant positive relationship of aerobic fitness with laboratory baseline IBI and small positive relation with daily life IBI when awake, with moderate to strong effect sizes. Aerobic fitness also showed a small negative relation with daily life negative affect dislike reactivity and an unexpected positive relation with wrist ns.SCR when sleeping, suggesting higher SNS activity in sleep in more fit subjects. These were the only associations to emerge between all ANS baseline or reactivity measures and our measures of either aerobic fitness when corrected for multiple testing. Even when

TABLE 2 Overview of number of participants per daily life category

Activity	Total	IBI	RSA	PEP	Wrist ns.SCR	Positive affect	Negative affect
Awake	113	113	112	111	111	105	105
Like	107	106	105	104	103	101	101
Dislike	105	105	104	103	103	97	97
Dislike reactivity	105	98	97	96	94	93	93
Leisure	88	88	87	87	84	82	82
Work/study	57	57	57	56	56	52	52
Work reactivity	37	37	37	37	36	34	34
Sleep	108	107	108	104	106	–	–
Sleep reactivity	108	107	107	104	106	–	–

TABLE 3 Descriptive of stress measures in the laboratory

	Baseline		Stress		Rest		Reactivity		Recovery	
	Mean	*SD*	**Mean**	*SD*	**Mean**	*SD*	**Mean**	*SD*	**Mean**	*SD*
Mental stress										
IBI (msec)	843.42	132.75	779.37	125.59	839.66	127.65	−64.04	93.52	59.70	77.44
RSA msec	84.72	44.32	61.87	27.86	79.50	36.40	−22.25	37.73	17.44	30.16
PEP msec	112.75	15.45	105.53	15.88	111.98	14.77	−7.21	10.19	6.48	7.73
ns.SCR palm (p/m)	5.18	3.50	14.58	3.94	7.40	3.64	9.29	4.57	−7.21	3.82
ns.SCR wrist (p/m)	0.41	0.68	1.58	2.25	1.00	1.46	1.00	2.12	−0.54	2.07
Positive affect	4.41	0.93	3.44	0.96	–	–	−0.96	0.90	–	–
Negative affect	1.54	0.79	2.17	0.97	–	–	0.62	0.62	–	–
Social stress										
IBI (msec)			773.66	115.71	840.27	120.44	−69.28	81.12	117.50	101.26
RSA msec			68.47	29.45	82.00	37.93	−14.54	37.85	18.64	35.50
PEP msec			106.34	16.14	112.13	15.75	−5.76	10.37	8.52	13.57
ns.SCR palm (p/m)			11.69	3.35	7.59	4.26	6.30	3.51	−5.64	5.28
ns.SCR wrist (p/m)			1.39	2.53	1.12	1.90	0.80	2.15	−0.46	4.03
Positive affect			3.88	0.89	–	–	−0.54	0.81	–	–
Negative affect			1.90	0.89	–	–	0.35	0.58	–	–

TABLE 4 Descriptive of stress measures in daily life

	Mean	SD	Mean	SD	Mean	SD
	Sleep		**Awake**		**Sleep recovery**	
IBI (msec)	1005.39	125.92	786.14	92.11	222.08	90.49
RSA (msec)	86.85	38.89	66.79	24.43	21.10	27.55
PEP (msec)	112.72	17.05	104.33	16.49	8.98	19.49
ns.SCR wrist (p/m)	2.56	2.04	2.48	1.95	−0.12	2.58
Positive affect	–	–	4.47	0.70	–	–
Negative affect	–	–	1.80	0.63	–	–
	Leisure		**Work**		**Work-reactivity**	
IBI (msec)	823.91	103.37	804.72	99.40	−27.87	81.39
RSA (msec)	73.16	29.30	72.78	26.29	−4.05	20.18
PEP (msec)	103.73	15.74	104.95	17.24	1.28	11.39
ns.SCR wrist (p/m)	2.19	1.94	2.12	1.90	−0.05	2.44
Positive affect	4.45	0.81	4.45	0.85	−0.08	0.81
Negative affect	1.68	0.61	1.91	0.74	0.17	0.49
	Like		**Dislike**		**Dislike-reactivity**	
IBI (msec)	791.99	96.14	776.57	99.11	−7.45	75.54
RSA (msec)	68.94	30.23	65.49	25.25	−3.29	22.48
PEP (msec)	102.89	16.60	105.59	17.50	1.80	9.72
ns.SCR wrist (p/m)	2.45	1.94	2.40	2.36	−0.15	1.72
Positive affect	4.72	0.73	4.20	0.77	−0.52	0.61
Negative affect	1.64	0.59	1.97	0.69	0.26	0.44

TABLE 5 Laboratory stress reactivity and recovery

		N	ΔM (SE)	t	p	d
Mental stress reactivity	IBI (msec)	116	−64.04 (8.68)	−7.37	<.001	−0.68
	RSA (msec)	109	−22.23 (3.61)	−6.15	<.001	−0.60
	PEP (msec)	104	−7.22 (1.00)	−7.22	<.001	−0.71
	ns.SCR palm (p/m)	96	9.29 (0.40)	19.92	<.001	2.36
	ns.SCR wrist (p/m)	104	1.19 (0.19)	5.97	<.001	0.60
	Positive affect	115	−0.96 (0.08)	−11.50	<.001	−1.07
	Negative affect	115	0.62 (0.06)	10.70	<.001	1.00
Mental stress recovery	IBI (msec)	115	59.70 (6.77)	8.26	<.001	0.83
	RSA (msec)	104	17.44 (3.15)	5.90	<.001	0.55
	PEP (msec)	102	6.48 (0.76)	8.46	<.001	0.84
	ns.SCR palm (p/m)	97	−7.21 (0.45)	−18.53	<.001	1.61
	ns.SCR wrist (p/m)	103	−0.60 (0.28)	−3.03	.003	0.21
Social stress reactivity	IBI (msec)	115	−66.18 (6.95)	−9.16	<.001	−0.86
	RSA (msec)	105	−14.54 (3.69)	−3.93	<.001	−0.43
	PEP (msec)	103	−6.43 (1.02)	−6.37	<.001	−0.62
	ns.SCR palm (p/m)	87	6.30 (0.37)	16.76	<.001	1.85
	ns.SCR wrist (p/m)	103	0.99 (0.21)	4.51	<.001	0.45
	Positive affect	114	−0.51 (0.07)	−6.18	<.001	−0.64
	Negative affect	114	0.35 (0.05)	6.36	<.001	0.62
Social stress recovery	IBI (msec)	115	66.18 (7.03)	10.12	<.001	0.89
	RSA (msec)	104	12.93 (3.54)	4.65	<.001	0.37
	PEP (msec)	102	5.71 (0.97)	6.63	<.001	0.59
	ns.SCR palm (p/m)	83	−3.80 (0.59)	−8.65	<.001	−0.76
	ns.SCR wrist (p/m)	104	−0.26 (0.33)	−1.29	.19	−0.08

TABLE 6 Ambulatory stress reactivity and recovery

		N	ΔM (SE)	t	p	d
Work—leisure reactivity	IBI (msec)	37	−27.87 (13.38)	−2.08	.044	0.23
	RSA (msec)	35	−4.05 (3.41)	−1.22	.23	0.02
	PEP (msec)	37	1.28 (1.87)	0.68	.49	0.10
	ns.SCR wrist (p/m)	35	0.21 (0.29)	0.66	.51	−0.03
	Positive affect	34	−0.08 (0.14)	−0.62	.53	0.00
	Negative affect	34	0.17 (0.08)	2.11	.042	0.00
Dislike—like reactivity	IBI (msec)	98	−7.46 (7.73)	−0.96	.33	0.20
	RSA (msec)	97	−3.30 (2.28)	−1.44	.15	0.15
	PEP (msec)	96	1.80 (0.99)	1.82	.072	−0.28
	ns.SCR wrist (p/m)	95	0.15 (0.17)	0.67	.50	−0.02
	Positive affect	93	−0.52 (0.06)	−8.26	<.001	0.84
	Negative affect	93	0.27 (0.04)	5.82	<.001	−0.74
Sleep recovery	IBI (msec)	107	222.08 (8.75)	25.38	<.001	2.42
	RSA (msec)	107	21.10 (2.66)	7.92	<.001	0.72
	PEP (msec)	104	8.98 (1.91)	4.70	<.001	0.43
	ns.SCR wrist (p/m)	106	0.12 (0.25)	0.50	.61	0.03

TABLE 7 Linear regression predicting laboratory stress from fitness

		VO₂max							MVPA							Exercise						
		N	b	SE	t	p	R²	d	N	B	SE	t	p	R²	d	N	b	SE	t	p	R²	d
Baseline	IBI (msec)	93	6.08	1.55	3.91	**<.001****	.38	1.56	115	0.89	0.24	3.68	**<.001****	.32	1.37	115	1.35	0.28	4.71	**<.001**	.40	1.63
	RSA (msec)	91	0.77	0.56	1.38	.17	.15	0.84	112	0.19	0.08	2.31	.022*	.21	1.03	112	0.22	0.10	2.15	.033*	.19	0.97
	PEP (msec)	84	0.51	0.21	2.44	.017*·**	.26	1.18	105	0.04	0.03	1.42	.16	.14	0.80	105	0.06	0.03	1.69	.093	.16	0.87
	ns.SCRp (p/m)	83	−0.06	0.04	−1.35	.18	.15	0.84	102	−0.016	0.007	−2.19	.024*	.21	1.03	102	−0.01	0.008	−1.91	.058	.18	0.94
	ns.SCRw (p/m)	85	0.01	0.01	0.98	.33	.06	0.50	104	−0.001	0.001	−0.87	.38	.13	0.77	104	−0.001	0.001	−0.56	.57	.12	0.74
	Positive affect	92	0.01	0.01	0.85	.40	.02	0.28	114	0.003	0.002	1.62	.11	.08	0.59	114	0.003	0.002	1.31	.19	.09	0.63
	Negative affect	92	−0.004	0.008	−0.37	.71	.02	0.28	114	−0.002	0.001	−1.72	.088	.08	0.59	114	−0.002	0.001	−1.37	.17	.09	0.63
Mental stress reactivity	IBI (msec)	93	−1.42	1.06	−1.33	.18	.13	0.77	115	−0.12	0.18	−0.68	.50	.06	0.50	115	−0.24	0.22	−1.12	.26	.11	0.70
	RSA (msec)	87	−0.61	0.46	−1.34	.18	.02	0.28	108	−0.12	0.07	−1.68	.096	.16	0.87	108	−0.13	0.09	−1.44	.15	.14	0.80
	PEP (msec)	83	−0.29	0.12	−2.38	.020*	.10	0.66	103	−0.003	0.02	−0.17	.86	.01	0.20	103	−0.02	0.02	−0.84	.40	.08	0.59
	ns.SCRp (p/m)	76	0.05	0.06	0.90	.37	.14	0.80	93	0.02	0.008	2.78	.006*	.28	1.24	93	0.02	0.01	2.26	.026*	.23	1.09
	ns.SCRw (p/m)	83	−0.01	0.02	−0.55	.58	.02	0.28	102	0.002	0.003	0.48	.63	.04	0.41	102	0.002	0.005	0.49	.62	.05	0.46
	Positive affect	92	0.003	0.01	0.30	.76	.02	0.28	114	0.002	0.001	1.29	.20	.11	0.70	114	0.003	0.002	1.59	.11	.14	0.80
	Negative affect	92	−0.003	0.008	−0.39	.70	.02	0.28	114	−0.001	0.001	−0.79	.43	.08	0.59	114	−0.002	0.001	−1.78	.077	.17	0.90
Mental stress recovery	IBI (msec)	93	0.91	0.75	1.20	.23	.11	0.70	114	0.10	0.14	0.70	.48	.06	0.50	114	0.13	0.17	0.75	.45	.07	0.55
	RSA(msec)	87	0.27	0.40	0.67	.50	.07	0.55	105	0.04	0.06	0.62	.53	.06	0.50	105	0.01	0.07	0.16	.86	.01	0.20
	PEP (msec)	83	0.08	0.08	1.03	.30	.11	0.70	102	0.008	0.01	0.51	.60	.05	0.46	102	0.01	0.02	0.74	.46	.07	0.55
	ns.SCRp (p/m)	79	−0.02	0.06	−0.30	.76	.04	0.41	98	−0.01	0.009	−1.71	.090	.19	0.97	98	−0.001	0.01	−0.06	.95	.02	0.28
	ns.SCRw (p/m)	85	0.008	0.03	0.24	.81	.03	0.35	103	0.001	0.005	0.29	.77	.03	0.35	103	0.005	0.007	0.77	.44	.07	0.28
Social stress reactivity	IBI (msec)	92	−0.12	1.00	−0.12	.90	.01	0.20	114	−0.04	0.15	−0.29	.77	.02	0.28	114	−0.16	0.19	−0.83	.41	.08	0.59
	RSA (msec)	85	−0.16	0.45	−0.37	.71	.04	0.41	104	−0.11	0.07	−1.56	.12	.15	0.84	104	−0.08	0.09	−0.84	.40	.08	0.59
	PEP (msec)	83	−0.12	0.13	−0.90	.37	.01	0.20	102	0.02	0.02	1.16	.25	.16	0.87	102	0.01	0.02	0.64	.53	.09	0.63
	ns.SCRp (p/m)	68	0.06	0.05	1.39	.17	.17	0.90	86	0.02	0.007	2.44	.016*	.24	1.12	86	0.01	0.009	1.19	.23	.12	0.74
	ns.SCRw (p/m)	83	−0.05	0.02	−1.87	.064	.20	1.00	102	0.0006	0.004	0.14	.88	.008	0.18	102	−0.002	0.005	−0.45	.65	.05	0.46
	Positive affect	91	−0.004	0.01	−0.42	.67	.05	0.46	113	−0.0007	0.001	−0.44	.66	.008	0.18	113	0.0006	0.002	0.30	.76	.05	0.46
	Negative affect	91	0.000	0.007	0.07	.94	.00	0.00	113	0.0003	0.001	0.25	.80	.007	0.17	113	−0.004	0.001	−0.27	.78	.04	0.41
Social stress recovery	IBI (msec)	93	0.66	0.83	0.80	.42	.07	0.55	115	0.04	0.14	0.27	.78	.01	0.20	115	−0.07	0.17	−0.40	.68	.04	0.41
	RSA (msec)	87	0.56	0.45	1.23	.22	.12	0.74	108	0.04	0.07	0.64	.52	.05	0.46	108	0.03	0.08	0.32	.75	.03	0.35
	PEP (msec)	83	0.01	0.10	0.12	.90	.01	0.20	103	0.007	0.02	0.38	.70	.03	0.35	103	0.01	0.02	0.68	.49	.07	0.55
	ns.SCRp (p/m)	77	0.08	0.07	1.09	.28	.11	0.70	93	0.0004	0.01	0.04	.97	.04	0.41	93	0.02	0.01	1.46	.15	.16	0.87
	ns.SCRw (p/m)	85	0.003	0.04	0.08	.93	.00	0.00	104	0.002	0.006	0.28	.78	.006	0.15	104	0.003	0.008	0.38	.70	.05	0.46

Note: R² reflects partial r squared of the fitness measure on the outcome from the model with age and gender included as covariates.
*Significant at nominal p < .05.; **Significant after multiple testing correction (p < .0016) in bold

TABLE 8 Linear regression predicting daily life stress from fitness

		VO₂max							MVPA							Exercise						
		N	b	SE	t	p	R²	d	N	B	SE	t	p	R²	d	N	b	SE	t	p	R²	d
Sleep	IBI (msec)	82	3.98	1.59	2.50	.014*	.08	0.59	106	0.65	0.24	2.77	.006**	.07	0.55	106	0.86	0.29	2.91	.004**	.07	0.55
	RSA (msec)	82	0.52	0.52	1.01	.31	.03	0.35	107	0.10	0.08	1.25	.21	.01	0.20	107	0.08	0.10	0.79	.43	.006	0.15
	PEP (msec)	79	−0.23	0.21	−1.07	.28	.10	0.66	103	−0.02	0.03	−0.67	.50	.004	0.13	103	−0.05	0.04	−1.29	.20	.01	0.20
	ns.SCRw (p/m)	91	0.09	0.02	3.44	<.001**	.13	0.77	105	0.007	0.004	1.84	.069	.03	0.35	105	0.009	0.005	1.82	.071	.03	0.35
Awake	IBI (msec)	87	4.06	1.07	3.77	<.001**	.14	0.80	112	0.56	0.17	3.28	.0014	.09	0.63	112	0.86	0.20	4.16	<.001	.14	0.80
	RSA (msec)	86	0.36	0.31	1.16	.25	.04	0.41	111	0.09	0.04	2.08	.039*	.04	0.41	111	0.13	0.06	2.23	.028*	.04	0.41
	PEP (msec)	85	0.18	0.21	0.86	.39	.003	0.11	110	−0.005	0.03	−0.16	.87	.00	0.00	110	−0.03	0.04	−0.70	.48	.004	0.13
	ns.SCRw (p/m)	86	0.02	0.02	0.99	.32	−.001	0.06	110	0.005	0.003	1.35	.18	.01	0.20	110	0.003	0.004	0.76	.45	.005	0.14
	Positive affect	83	0.007	0.01	0.83	.41	.000	0.00	104	0.003	0.001	2.53	.013*	.06	0.50	104	0.003	0.002	1.61	.11	.02	0.28
	Negative affect	71	−0.008	0.01	−0.93	.35	.01	0.20	104	−0.002	0.001	−1.43	.15	.02	0.28	104	−0.003	0.001	−1.63	.10	.02	0.28
Sleep recovery	IBI (msec)	82	−0.17	1.14	−0.15	.88	.02	0.28	106	−0.06	0.17	−0.38	.70	.001	0.06	106	−0.24	0.21	−1.18	.26	.01	0.20
	RSA (msec)	81	0.22	0.38	0.60	.55	.007	0.17	106	−0.008	0.06	−0.15	.88	.00	0.00	106	−0.06	0.07	−0.88	.38	.007	0.17
	PEP (msec)	79	−0.19	0.24	−0.80	.42	.10	0.66	103	−0.007	0.04	−0.19	.84	.00	0.00	103	−0.005	0.05	−0.10	.92	.00	0.00
	ns.SCRw (p/m)	81	0.08	0.03	2.67	.009*	.08	0.59	105	0.003	0.005	0.66	.51	.004	0.13	105	0.006	0.006	0.99	.32	.009	0.19
Work–leisure reactivity	IBI (msec)	27	−2.45	1.87	−1.31	.20	.08	0.59	36	−0.22	0.29	−0.73	.47	.01	0.20	36	−0.59	0.34	−1.73	.092	.08	0.59
	RSA (msec)	27	−1.40	0.43	−3.28	.003*	.33	1.40	36	−0.09	0.07	−1.18	.24	.04	0.41	36	−0.06	0.09	−0.75	.46	.01	0.20
	PEP (msec)	27	0.44	0.28	1.56	.13	.14	0.80	36	0.008	0.04	0.18	.85	.001	0.06	36	0.008	0.05	0.16	.87	.001	0.06
	ns.SCRw (p/m)	27	0.03	0.04	0.73	.47	.04	0.41	35	−0.01	0.008	−1.87	.071	.10	0.66	35	−0.01	0.01	−1.37	.18	.05	0.46
	Positive affect	25	−0.01	0.02	−0.62	.54	−.06	0.50	33	−0.001	0.003	−0.51	.61	.008	0.18	33	−0.002	0.004	−10.46	.65	.007	0.17
	Negative affect	25	−0.01	0.01	−0.91	.37	−.07	0.55	33	−0.002	0.001	−1.29	.20	.05	0.46	33	−0.002	0.002	−0.84	.40	.02	0.28
Dislike–like reactivity	IBI (msec)	77	−1.04	1.05	−0.99	.32	−.006	0.15	97	0.14	0.16	0.87	.38	.008	0.18	97	−0.03	0.21	−0.13	.89	.00	0.00
	RSA (msec)	76	−0.29	0.30	−0.96	.34	.02	0.28	96	0.03	0.04	0.78	.43	.006	0.15	96	0.0003	0.02	0.00	.99	.00	0.00
	PEP (msec)	76	0.12	0.14	0.85	.39	−.001	0.06	95	0.03	0.02	1.72	.089	.03	0.35	95	0.006	0.03	0.24	.81	.00	0.00
	ns.SCRw (p/m)	75	0.02	0.02	0.85	.39	.01	0.20	93	−0.005	0.003	−1.39	.16	.02	0.28	93	−0.009	0.004	−2.00	.048*	.04	0.41
	Positive affect	75	0.01	0.008	1.40	.16	.03	0.35	92	0.002	0.001	1.46	.15	.02	0.28	92	0.003	0.001	1.74	.084	.007	0.17
	Negative affect	75	−0.02	0.006	−3.29	.0015**	.15	0.84	92	−0.002	0.001	−2.97	.0038*	.09	0.63	92	−0.001	0.001	−1.33	.18	.02	0.28

Note: R² reflects partial r squared of the fitness measure on the outcome from the model with age and gender included as covariates.

*Significant at nominal p <.05.; **Significant after multiple testing correction (p <.0016) in bold

inspecting results at a nominal $p < .05$ only very scant support for the cross-stressor adaptation hypothesis is found. Only a moderate positive relation with laboratory PEP at baseline and a small negative relation with mental stress PEP reactivity could hint at lower basal SNS activity and attenuated SNS reactivity in more fit participants, while a moderate negative relation with RSA work reactivity suggests attenuated PNS withdrawal during work. The observed small negative relationship with wrist ns.SCR sleep recovery is likely due to the higher ns.SCR when sleeping in fit individuals.

MVPA showed a moderate to strong significantly positive relationship with laboratory baseline IBI and daily life IBI when awake. No reactivity or recovery effects were found when correcting for multiple testing. At nominal $p < .05$, higher baseline RSA was observed both in the laboratory and during awake time in daily life, hinting at a role for the PNS in explaining the lowered heart rate in more physically active participants. Lower laboratory baseline palm ns.SCR in more active participants suggests this to be paired to lower SNS activity at rest, and the slightly increased palm ns.SCR mental and social laboratory stress reactivity may simply reflect the baseline—reactivity correlation. For MVPA, there also was a small positive relation with positive affect when awake, and a small negative relation with negative affective reactivity to disliked activities.

Exercise showed the least evidence for an effect on stress reactivity or recovery. For IBI and RSA it followed the pattern showed by MVPA, i.e., a moderate to strong significantly positive relationship with laboratory baseline IBI and daily life IBI when awake, paired with higher RSA.

4 | DISCUSSION

To date no consensus has been reached on the validity of the cross-stressor adaptation hypothesis, positing that adaptation to a physical stressor in response to repeated exposure (training) also reduces reactivity to psychological types of stressors. Reviews and meta-analyses on this topic arrived at different conclusions but all unanimously point to the large heterogeneity in study design, plaguing the extant literature (Forcier et al., 2006; Huang et al., 2013; Jackson & Dishman, 2006; Mücke et al., 2018). In the current study we aimed to address these issues by (1) defining fitness as both aerobic fitness and physical activity, (2) including ANS branch-specific, i.e., SNS and PNS, reactivity as the outcome measures, (3) including both laboratory and daily life data, (4) using mental and social stressors, and (5) including stress reactivity and recovery measures. The overarching finding is that the cross-stressor adaptation hypothesis was not supported by the data.

Our separate use of aerobic fitness and physical activity finds justification in the low to moderate correlation between these two exposure variables, which repeats previous findings (Aadahl et al., 2007; Emaus et al., 2010; Minder et al., 2014; Morrow & Freedson, 1994; Siconolfi et al., 1985). Little gain was achieved, however, by separating moderate-to-vigorous activity from activities specifically related to voluntary leisure time exercise behavior. These variables showed a high correlation, likely due to the large overlap in reported activities for these variables. However, based on our results a measure containing all moderate to vigorous physical activity engaged in is favored, as opposed to a measure only including sports and exercise activities. This supports the conclusion of the review of Jackson and Dishman (2006), because it yielded more often a significant relation with stress reactivity or recovery. Therefore the discussion will focus on the results pertaining to aerobic fitness and MVPA.

Past results from meta-analyses regarding cardiovascular stress reactivity using aerobic fitness as their fitness measure most consistently reported on faster HR recovery in more fit subjects, paired to a higher HR reactivity (Jackson & Dishman, 2006; Mücke et al., 2018). Studies using regular exercise behavior also showed a faster HR recovery in more fit subjects, but identified a lower HR reactivity (Mücke et al., 2018). When fitness was defined by resting HR, Forcier et al. (2006) also found that more fit subjects showed a lower HR reactivity, but no recovery effect was observed. Our experimental stress paradigms evoked ANS reactivity comparable in direction and effect size to previous studies (Brindle et al., 2014), but our laboratory data do not support an effect of aerobic fitness or MVPA on this observed cardiovascular stress reactivity or recovery even after accounting for the main potential moderating factors. In addition, the current study also did not replicate the findings by Mücke et al. (2018) with regard to a relationship between regular exercise and affective laboratory stress reactivity. More generally, our data conform well to the overarching conclusion from extant meta-analyses, namely that an impact of fitness on stress reactivity and recovery is either absent or too small to survive the plethora of moderators and confounders of stress reactivity.

Whereas the laboratory stressors successfully induced changes in ANS and affect, our daily life analyses showed no significant differences in ANS activity or affect during work compared to leisure time, although there was a trend towards higher HR and higher negative affect at work. This is a limitation that suggest too little stress may have occurred in the daily life part of the study. Previous studies using a daily life design showed lower HRV (Jarczok et al., 2013; Vrijkotte et al., 2000), and higher HR and blood pressure at work compared to leisure time

(Vrijkotte et al., 2000). These studies used (white collar) working populations, whereas we mostly used a student population. Though it is estimated that around half of the university student population report moderate levels of stress-related mental health issues (as reviewed by Regehr et al., 2013), and that psychological distress among university student is higher compared to the general population (Adlaf et al., 2001; Stewart-Brown et al., 2000) and their working peers (Cotton et al., 2002; Vaez et al., 2004), our current sample showed little signs of stress as indicated by their good mental health score, low stress experience, and low mood disturbance. Even so, large individual differences were detected that should have allowed a clinically relevant correlation with fitness or PA to surface. Also, they did report to engage in (strongly) disliked activities, but even comparing liked versus disliked activities, no main effect were observed. The only association identified in the current sample is a large effect of aerobic fitness on RSA work reactivity at the $p < .05$ level, in which fitter individuals showed lower RSA work reactivity.

When comparing wakefulness to sleep, we did observe significant increases in IBI, RSA and PEP during restorative sleep, as did others (Burgess et al., 1997; Gonzales et al., 2020; Gregoire et al., 1996; Stein & Pu, 2012; Zoccoli & Amici, 2020). However, using the awake-sleep contrast as our index of ANS recovery in daily life did not show any effects of MVPA or aerobic fitness pointing to cross-stress adaptation. We also generally did not observe an overall relationship between affect and MVPA or aerobic fitness. Sole exception was a negative relationship between aerobic fitness and negative affect dislike reactivity, which was extended by a trend for MVPA. This suggests that fitter or more active individuals had a lower negative appraisal of disliked activities. This finding is in line with a recent study which also identified a positive effect of exercise on the subjective experience of daily life stress (Chovanec & Gröpel, 2020). Surprisingly, our results show an decreased ns.SCR on the wrist in fit individuals during sleep, which should be validated by future research.

The absence of a relationship between aerobic fitness or MVPA and ANS or affective reactivity would suggest little clinical relevance for the improvement of fitness to reduce stress reactivity. However, the absence of a cross-stressor advantage with regard to ANS stress reactivity and recovery does not negate the many clear advantageous effects of fitness. Both cross-sectional and longitudinal studies have shown that a higher resting HR is associated with an increased risk of coronary heart disease and all-cause mortality (Jensen, 2019) and that engagement in any type of exercise reduces resting HR with an average of 4.7% across different studies (Danieli et al., 2014). In line with previous research, our study does confirm this significant bradycardic effect for both aerobic fitness (Emaus et al., 2010;

Gonzales et al., 2020; Melanson, 2020) and MVPA (Emaus et al., 2010; Gonzales et al., 2020). Fitter and/or more active participants had a lower resting heart rate (HR), with aerobic fitness and MVPA explaining 14.4% and 10.2% of the variance in the controlled laboratory baseline measure, but only 2% and 0.8% of the variance in the average awake HR during the 24 hr recording. This latter drop in explained variance is likely due to the inclusion of various activities in the daily life data, while in the laboratory participants were required to sit quietly. Our study also hints at a role for lower SNS activity and higher PNS activity contributing to these HR-lowering effects when adopting a lenient significance threshold. This is consistent with findings of von Haaren et al. (2016), Schilling et al. (2020), and part of the studies included in the review of Tonello et al. (2014).

The current study was performed in a large enough sample size to find medium to large effects and covered all possible sources of heterogeneity posed by the meta-analyses. As expected, in the laboratory analyses ~30% of effect sizes were large and ~30% where medium. In daily life, however, the majority of the observed effect sizes were small, with only 20% being medium and ~5% large. By adjusting our p-value for multiple testing, our overall positive predictive value (probability that a finding reflects a true relationship; Button et al., 2013) was good. However, the chance to detect small sized relationships between fitness/physical activity and ANS or affective response to stress was low. Despite such small effects being scientifically interesting, one can question their relevance, in terms of allowing us to meaningfully advocate regular physical activity (or exercise sufficiently vigorous to increase fitness) as way to reduce the health impact of repeated cardiovascular stress reactivity. In establishing our sample sizes we have therefore assumed at least a medium effect size, as this would be more relevant from a public health viewpoint.

The cross-stressor adaptation hypothesis is based on the observation that repeated exposure to exercise allows a person to perform a comparable physical load with a lower activation of the SNS and a smaller deactivation of the PNS during exercise as well as a more rapid recovery to basal levels of SNS and PNS after exercise (McArdle et al., 2015). These adaptations occur in response to repeated exposure (training) for a large part by increasing the organ responsiveness, such that e.g., larger stroke volume requires less increase in HR to obtain the same cardiac output, and more dense muscle capillarization requires less vasoconstriction in non-muscle tissues and non-active muscles to ensure enough blood is distributed to the active muscles (McArdle et al., 2015). Also changes in exercise-induced feedback from the working muscle and the cardiorespiratory systems (e.g.,

baroreceptors) may contribute to altered ANS responding to exercise after training. When we experience psychological stress, however, the body only *prepares* itself for the anticipated need for exercise through the so called fight-or-flight response (Schulkin, 2011; Stefano et al., 2008; Zandara et al., 2018), which is a feed forward mechanism, whereas only mild increase in muscle work are actually seen (Sothmann et al., 1996). Because the extent of the physical activity that the body is going to need to avert the stressor is unknown, the height of this anticipatory response is likely determined by the amount of threat level experienced but may also by a function of the maximal exercise performance capability. In that case, higher rather than lower anticipatory responses could be expected with increased fitness/ MVPA. On the other hand, if preparation is always for some fixed amount of fight/flight, than training would reduce the ANS activation needed to attain this cardiovascular readiness state. In that case, lower anticipatory responses could be expected with increased fitness/ MVPA. In both cases, by just altering the feed-forward signal and not also using the improved organ responsiveness or changes in exercise induced feedback cues, the cross-stressor adaptation effect could be smaller than detectable by the standard approaches, including the one used here.

Of course, we cannot exclude the alternative explanation that our study had limitations that prevented detection of the cross-stressor adaptation effect. First, specific selection of participants was sub-optimal to detect a cross-stressor adaptation in this feed-forward component. Our population consists of young, and relatively active and fit participants. Over 60% reported to regularly engage in leisure time exercise, with those who did not engage in exercise reporting engagement in at least 105 min of non-exercise related MVPA. It might be that the effect of the cross-stressor adaptation hypothesis was obscured by this relatively high physical activity level and can only be observed in a population including true inactive participants. Second, the study is limited by the low levels of experienced stress during the daily life part of the study. It could be that this stress was missed due to the explicit recruitment of healthy participants or due to freedom of participants to choose on which day they took part in the study. It is likely that participant picked a day in a relatively stress-free week of their lives. A third limitation of the current study is the cross-sectional design, limiting it from shedding light on the effectiveness of exercise intervention studies on stress reactivity, such as those from von Haaren et al. (2016) and Chovanec and Gröpel (2020). Last, the current study focused on the validity of the cross-stressor adaption hypothesis with regard to its effect on ANS stress reactivity only. We want to stress that the results of this study can, therefore, not be translated to the effect of physical activity and aerobic fitness on the response of the hypothalamic-pituitary-adrenal (HPA) axis.

Future studies should collect data on both ANS and HPA reactivity in more diverse populations and over longer periods of time to increase the variance in experienced affect or select specifics moment of life during which participants know they are going to experience a stressor (i.e., an exam or work deadline).

Taken together our results support the resting HR reducing effect of fitness and exercise engagement both in the laboratory and daily life. It did not provide evidence for the cross-stressor adaptation hypothesis at a multiple testing significance level but gave some indications of a lower basal SNS activity and attenuated SNS reactivity in more fit participants and higher basal PNS and attenuated PNS withdrawal during work. Our study validated the importance to take into account the amount of overall MVPA, rather than only leisure time exercise. More specifically, while aerobic fitness was only associated with reduced SNS activity, MVPA tended to also show associations with increased PNS activity, stressing even further that different measures of fitness should not be used interchangeably.

AUTHOR CONTRIBUTIONS

Denise Johanna van der Mee: Data curation; formal analysis; methodology; project administration; writing – original draft; writing – review and editing. **Martin J. Gevonden:** Supervision; writing – review and editing. **Joyce H. D. M. Westerink:** Supervision; writing – review and editing. **Eco J. C. de Geus:** Conceptualization; funding acquisition; methodology; supervision; writing – review and editing.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest beyond their affiliations.

DATA AVAILABILITY STATEMENT

The data for this project are confidential, but may be obtained with Data Use Agreements with Philips Eindhoven. Researchers interested in access to the data may contact Navin Natoewal at navin.natoewal@philips.com. It can take some months to negotiate data use agreements and gain access to the data. The author will assist with any reasonable replication attempts for two years following publication.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

Appendix S1: Supporting Information