



Prospective associations of technostress at work, burnout symptoms, hair cortisol, and chronic low-grade inflammation

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ABSTRACT

Background: Working conditions in the age of digitalization harbor risks for chronic stress and burnout. However, real-world investigations into biological effects of technostress, that is stress in the context of digital technology use, are sparse. This study prospectively assessed associations between technostress, general work stress, burnout symptoms, hair cortisol, and chronic low-grade inflammation.

Methods: Hospital employees ($N = 238$, 182 females, $M_{age} = 28.5$ years) participated in a prospective cohort study with two follow-ups six months apart (T2, T3). Participants answered standardized questionnaires on general job strain (job demand-control ratio), technostressors (work interruptions, multitasking, information overload), burnout symptoms (exhaustion, mental distance), and relevant confounders. Moreover, they provided capillary blood samples for C-reactive protein (CRP) and hair strands for hair cortisol concentration (HCC) analysis. Structural equation modelling was performed.

Results: The factorial structure of survey measures was confirmed. Burnout symptoms ($M_{T2} = 2.17$, $M_{T3} = 2.33$) and HCC ($M_{T2} = 4.79$, $M_{T3} = 9.56$; pg/mg) increased over time, CRP did not ($M_{T2} = 1.15$, $M_{T3} = 1.21$; mg/L). Adjusted path models showed that technostress was negatively associated with HCC ($\beta = -0.16$, $p = .003$), but not with burnout and CRP. General work stress in contrast, was not significantly associated with burnout, HCC or CRP. Furthermore, there were reciprocal effects of CRP on HCC ($\beta = 0.28$, $p = .001$) and of HCC on CRP ($\beta = -0.10$, $p \leq .001$). Associations were robust in additional analyses including further confounders.

Conclusion: This is the first study on prospective effects of technostress on employees' endocrine and inflammatory systems. Results suggest differential effects of technostress on the hypothalamic-pituitary-adrenocortical axis activity. Given its key role for long-term health, the findings have important implications for occupational health and safety in digitalized work environments.

1. Introduction

Stress is a major risk factor for the development of non-communicable diseases, like cardiovascular diseases, cancer or diabetes, which are the leading cause of death worldwide (WHO, 2022). The workplace can be stressful and substantially influence employees' health. There is ample evidence of the link of work stress with physical and mental morbidity as well as mortality (e.g., Kivimäki et al., 2012;

Madsen et al., 2017; Taouk et al., 2020). In the light of the profound transformation of the world of work in the age of digitalization, new forms of work-related stress emerge, that is technostress (Brod, 1982) or digital stress (Hefner and Vorderer, 2016; Reinecke et al., 2017; Weinstein and Selman, 2016). The more commonly used term technostress can be defined as “stress experienced by end users of Information and Communication Technologies (ICTs)” (Ragu-Nathan et al., 2008). In modern work environments relevant and common technostressors are

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work interruptions, multitasking, and information overload due to ICTs (Eppler and Mengis, 2004; Galluch et al., 2015; Hefner and Vorderer, 2016; Reinecke et al., 2017). Technostress has been associated with different negative consequences regarding well-being, (mental) health, and work-related outcomes in employees (Dragano and Lunau, 2020; La Torre et al., 2019). However, technostress has mostly been assessed with self-report, while objectively measurable physiological effects of technostress are under-researched. The few existing studies on physiological stress responses focus on acute stress assessed in laboratory experiments rather than chronic stress related to ICT use in real-world settings (Becker et al., 2022a; Dragano and Lunau, 2020).

One important biological mechanism that explains how chronic stress, like work stress, “gets under the skin” is the hypothalamic pituitary-adrenocortical (HPA) axis with its main effector hormone cortisol. Chronic stress has been associated both with an increase in cortisol secretion (i.e. hypercortisolism), but also with a deficiency of cortisol (i.e. hypocortisolism) depending on a range of factors, such as stressor and person characteristics (Heim et al., 2000; Miller et al., 2007). Analysis of hair cortisol concentration (HCC) is increasingly used to measure long-term integrated cortisol levels retrospectively and as an indicator for chronic stress confers substantial advantages over the use of traditional fluid-based biomarkers such as salivary cortisol, as it is less influenced by biological rhythms or acute influences (Stalder et al., 2017).

Besides the HPA-axis, stress also has complex effects on the immune system with up-regulation of some parts, primarily inflammatory pathways, and down-regulation of others, primarily cellular immunity (Chrousos, 2009; Segerstrom and Miller, 2004). While inflammation is an adaptive reaction in the short-term, sustained low-grade inflammation is involved in the development of severe chronic diseases encompassing cardiovascular, metabolic, and neurodegenerative diseases, cancer as well as depression (Couzin-Frankel, 2010; Morey et al., 2015; Slavich and Irwin, 2014). Chronic systemic low-grade inflammation can be triggered by psychological stress alone without any apparent medical source (e.g., infection or injury) and can be measured with a range of biomarkers, such as the acute-phase-protein C-reactive protein (CRP) or cytokines (Black, 2002; Rohleder, 2019). Inflammation and abnormalities in cortisol secretion have been found to co-occur in clinical samples (i.e., depression), presumably due to glucocorticoid resistance, that is, a dysfunction of the glucocorticoid receptor leading to an impaired negative feedback loop of the HPA-axis (Pariante, 2017).

Available research on work-related stress and HCC is limited with inconsistent results, and there is a lack of prospective studies (Schaafsma et al., 2021). Furthermore, work stress has been associated with low-grade inflammation, but high-level evidence is weak due to a paucity of prospective research (Kaltenecker et al., 2021; Wright et al., 2020). Regarding technostress in particular, chronic effects on the two key biological mechanisms – the HPA-axis and chronic low-grade inflammation – have largely been overlooked. To our knowledge, two recent cross-sectional studies from our work group assessed for the first time, inflammatory responses to different technostressors without finding stress-induced increases (Becker et al., 2023; Kaltenecker et al., 2023).

One key mental health outcome in occupational health research is burnout. Burnout is suggested to develop as a consequence of chronic exposure to work stress and is expected to be associated with depletion of the HPA-axis, that is hypocortisolism (Miller et al., 2007; Rohleder, 2018). However, this notion has not been consistently supported empirically with recent studies reporting *increased* HCC in burned-out individuals (Penz et al., 2018; Wendsche et al., 2020). Besides alterations in the HPA-axis, increased systemic inflammation has been shown in burnout – yet the current evidence is inconclusive (Hänsel et al., 2010; Rohleder, 2019). Initial findings suggest associations of technostress with burnout, which are, however, mainly based on cross-sectional designs (Dragano and Lunau, 2020). In a prior study, specific forms of technostress (e.g., technology and information overload) were related to employees’ burnout symptoms, even after controlling for general work

overload (Kaltenecker et al., 2023). In sum, prospective research on technostress and burnout, as well as on the biological underpinnings of technostress and work stress in general, is limited. This highlights the need for advanced methods to gain a deeper understanding of potential health risks in modern working environments. Longitudinal designs, in which the same variables are assessed repeatedly over time in the same participants (i.e., full panel designs), provide an avenue to test the temporal order and direction of effects and best determine (reciprocal or reverse) causality (Ployhart and Vandenberg, 2010; Taris and Kompier, 2014).

Technostress may be especially relevant in healthcare settings, where health information technology is increasingly implemented, such as electronic health records or clinical decision support systems. Healthcare professionals are suggested to be an at-risk population for stress-related biological perturbations and development of burnout (Dawe et al., 2016; Maslach, 2003). Firstly defined by the psychologist Craig Brod (1982), the concept of technostress and its measurement was primarily developed in the discipline of information systems (e.g., Ayyagari et al., 2011; Ragu-Nathan et al., 2008; Tarafdar et al., 2007), but in recent years, it has also been applied to the healthcare context: For instance, Califf and Sarker (2020) found that negatively perceived technostress was associated with psychological distress in nurses, which in turn was related to low job satisfaction and high attrition, both impacting turnover intentions – a highly relevant issue in nursing. This was supported by a further study among health professionals in psychiatric hospitals, which also showed that technostress was associated with negative health consequences including burnout symptoms (Golz et al., 2021). Furthermore, in a recent cross-sectional study, university medical staff members and students reported moderate-to-high levels of technostress, which was positively associated with burnout and serum cortisol (Kasemy et al., 2022). Taken together, the emerging evidence suggests that technostress is an important phenomenon for different medical personnel, but in-depth research utilizing prospective designs is necessary.

To shed light into the possible associations of work stress, including technostress, burnout, HCC, and chronic low-grade inflammation, we conducted – to our knowledge for the first time – a prospective study with a full panel design among employees of a university hospital. As a conceptual framework, we drew upon the well-established job demand-control (JDC) model, which postulates that job strain results from a combination of high job demands and low job control (Karasek, 1979). The objective was to investigate general work stress (based on the JDC model) and technostress (work interruptions, multitasking, information overload) as predictors and burnout symptoms, HCC, and inflammation (CRP) as outcomes. In particular, we examined prospective associations of the predictors with the outcomes (research question 1) and prospective associations between the outcomes in order to identify their temporal order (research question 2).

2. Materials and methods

2.1. Design

A prospective cohort study at a large university hospital in South Germany with a full cross-lagged panel design including three measurement time points with a time lag of 6 months was conducted. Data collection took place from 06/2021 until 11/2022 with baseline

measurement (T1) from 06–11/2021,² first follow-up (T2) from 11/2021–05/2022, and second follow-up (T3) from 06–11/2022. The study was approved by the faculty's ethics committee (20–0914) and was carried out in accordance with the ethical standards of the Declaration of Helsinki. The study protocol was registered (<https://osf.io/94p6n/>). All participants gave their written informed consent.

2.2. Participants and procedure

New hospital employees were recruited for study participation after their obligatory pre-employment medical examination. As an incentive, participants were compensated monetarily (€ 50) for study participation (i.e., for completing at least two measurement time points) and were provided with a personal report on their results (i.e., biomarker levels and scores in psychological constructs) after study completion. Prior to data collection, we performed an a-priori power analysis for bivariate linear regression based on an alpha of 0.05, a power of 0.80, and a small to medium effect size ($\beta = 0.18$), revealing a required sample size of $N = 187$ (Faul et al., 2007). A total of $N = 301$ participants were included in the study at baseline (T1), of whom $n = 241$ participated at follow-up I, 6 months later (T2), and $n = 200$ at follow-up II, 12 months later (T3). For follow-up measurements, participants were contacted by the study team following a standardized and iterative procedure, and an individual appointment for each participant at the clinic was arranged. The sample consisted of healthcare personnel with various professions, such as physicians and nurses, but also research staff and other.

Before inclusion in the study and each follow-up, we checked participants' eligibility with a screening on the following exclusion criteria: temporary contract of < six months (only T1), current acute disease symptoms (like acute cold or influenza-like infection, fever, cystitis, influenza, acute injuries, etc.), pregnancy, permanent intake of anti-inflammatory medication (e.g., cortisone, hydrocortisone), intake of anti-coagulant drugs in the last 12hr, and insufficient German language skills. A posteriori, we excluded participants, who dropped out after T1 ($n = 55$), who had extreme HCC levels (i.e., > 3 standard deviations [SD] from the mean across waves, $n = 2$), CRP levels > 10 mg/L (Pearson et al., 2003) at any measurement time point ($n = 5$), as well as non-binary sex ($n = 1$).

2.3. Measures

A combination of standardized questionnaires for self-report and biomarker measurements was used. All variables were measured at each time point, except for sociodemographic information, profession, and body-mass index [BMI] (only at T1). The reliability of self-report measures was assessed by computing the Spearman-Brown statistic (ρ) for two-item scales and Cronbach's alpha (α) as well as McDonald's omega (ω) for scales with more than two items (Eisinga et al., 2013; Hayes and Coutts, 2020).

2.3.1. Self-report measures

2.3.1.1. General work stress. Based on the JDC model (Karasek, 1979), general work stress was measured with two scales derived from a well-established screening for psychological stressors at work (Glaser et al., 2020). *Job demands* was assessed with two items. A sample item is "I often have to hurry and still cannot complete my work". Reliability was

acceptable, with $\rho = 0.80$ (T2) and $\rho = 0.78$ (T3). *Job control* was measured with three items (e.g., "I can determine for myself how to do my work"; $\alpha/\omega = 0.82$ [T2], $\alpha = 0.82/\omega = 0.83$ [T3]). Response options ranged from 1 = *not at all* to 5 = *to a very great extent*. A score was calculated by summing the item scores for each scale. Because of the difference in the number of items per measure, job demands (multiplied by 10) and control (multiplied by 20/3) were weighted to obtain values between 0 and 100 (Piantella et al., 2021). We then calculated the *job demand/control (JDC) ratio*, a continuous measure for job strain, where higher scores indicate higher job strain (e.g., Theorell et al., 1990). Moreover, means for job demands and control, respectively, were computed.

2.3.1.2. Technostress. For the assessment of work stressors specifically related to the use of digital technologies, three scales were used. *Work interruptions* were measured with three items (adapted from Büssing and Glaser, 2002; Glaser et al., 2020). A sample item is "I often have to interrupt my work due to electronic messages (e.g., e-mail, device message)". *Multitasking* was captured with two items (adapted from Semmer et al., 1999), such as "Due to digital technologies I have to work on several tasks at the same time". *Information overload* was also assessed with two items (Piecha and Hacker, 2020), such as "I feel that the information I receive via on-duty digital media is too much". Items were answered on a five-point scale (1 = *not at all* to 5 = *to a very great extent*). Individual scale means and an overall mean based on the three scales were calculated. The scale reliability for the overall mean was $\alpha = 0.84/\omega = 0.83$ (T2) and $\alpha/\omega = 0.86$ (T3).

2.3.1.3. Burnout symptoms. Burnout symptoms were measured with the Burnout Assessment Tool (BAT; Schaufeli et al., 2019; German translation: Glaser and Seubert, 2020). We used the two subscales *exhaustion* and *mental distance* with two items each. A sample item for *exhaustion* is "After a day at work, I find it hard to recover my energy" and for *mental distance* "I struggle to find any enthusiasm for my work". Possible responses ranged from 1 = *never* to 5 = *always* on a five-point scale. Individual subscale and a total mean for burnout symptoms were computed. Reliability for the total mean was $\alpha/\omega = 0.77$ (T2) and $\alpha/\omega = 0.81$ (T3).

2.3.1.4. Control variables. The following variables were assessed as potential confounders as suggested by previous research (de Hert, 2020; Magnusson Hanson et al., 2019; Meredith et al., 2022; Segerstrom and Miller, 2004; Stalder et al., 2017):

Sociodemographic characteristics: sex (f/m/d), age (in years);

Health-related characteristics: BMI (kg/m²), physical activity ("Overall, how much do you care about getting enough physical activity?"; 1 = *not at all* to 5 = *very much*), smoking (1 = *never smoked* to 5 = *yes, every day*), hormone medication (for contraception and for other reasons);

Employment-related characteristics: profession (nurse, physician, medical [-technical] personnel, research staff, administration, other), shift work (yes/no), full-time job (yes/no);

Hair-related information: hair dyeing (including coloring, bleaching, henna, highlighting; all: yes/no), hair treatment (perm, straightening; both: yes/no), weekly hair washing frequency;

Procedural information: To account for potential seasonal variations of biomarker levels, the date of sampling was used to calculate variables reflecting the respective season. For HCC analyses, consistent with a previous study (Abell et al., 2016), a variable with eight categories was created including the four seasons (meteorological, northern hemisphere) and four overlapping seasons reflecting HCC levels in the four weeks prior to sampling (1 = spring/summer [June], 2 = summer [July, August], 3 = summer/autumn [September], 4 = autumn [October, November], 5 = autumn/winter [December], 6 = winter [January, February], 7 = winter/spring [March], 8 = spring [April, May]). For CRP analyses, a four-category variable was generated representing the

² Based on the data from T1 one previous study has been published (Kaltenecker et al., 2023). This was a cross-sectional analysis among a subsample of employees on associations between an extended set of technostressors (and general psychosocial work factors) with burnout symptoms and C-reactive protein. In contrast, this prospective study uses data from the whole cohort and two follow-up measurements and includes an additionally relevant physiological outcome (i.e., hair cortisol).

four seasons (1 = summer [June, July, August], 2 = autumn [September, October, November], 3 = winter [December, January, February], 4 = spring [March, April, May]).

2.3.2. Biomarkers

2.3.2.1. Hair cortisol concentration (HCC). Hair sample collection was optional for participants and was conducted only after additional informed consent was obtained. In each wave, >80 % of participants provided a hair sample with $n = 251$ at T1, $n = 201$ at T2, and $n = 161$ at T3. Hair strands were taken from the posterior vertex region of the head, tied off with a thin rubber band, and cut as close as possible to the scalp with specific scissors by a trained member of the study team. Subsequently, samples were enveloped in aluminum foil and stored in a box at room temperature. HCC was analyzed in the 1 cm segment of the hair strand most proximal to the scalp. Assuming an average hair growth of 1 cm/month (Wennig, 2000), this represents hair grown over a one-month period prior to sampling.

Samples were analyzed after each study wave in the laboratory of Prof Kirschbaum at the Technical University Dresden using a column-switching liquid chromatography atmospheric-pressure-chemical-ionization tandem mass spectrometry assay (LC-APCI-MS/MS). The protocol of this efficient, highly sensitive and reliable method for the quantification of steroid hormones in human hair is described elsewhere (Gao et al., 2013; Stalder et al., 2012). For cortisol, the intra- and inter-assay coefficients of variation (CVs) were found to range between 3.7 % and 8.8 % (Gao et al., 2013). All samples were analyzed ($n = 613$, mean hair mass = 6.6 mg).

2.3.2.2. C-reactive protein (CRP). All participants provided capillary blood samples for analysis of high-sensitivity C-reactive protein (hs-CRP). We used the well-established minimally invasive dried blood spot method in which drops of whole blood from a finger prick are collected on filter papers (McDade et al., 2007). A trained member of the study team pricked the participant's fingertip with a disposable lancet under sterile conditions, and after wiping away the first drop with gauze, applied at least two blood spots of sufficient size on a filter paper. The paper was then dried at room temperature for at least 8hr before being stored with a desiccant in a sealable multi-barrier pouch at -26°C . Hs-CRP was analyzed using a "Human C-Reactive Protein/CRP Quantikine ELISA Kit" (IBL International) in the laboratory of the Chair of Health Psychology, Friedrich-Alexander University Erlangen-Nürnberg (Becker et al., 2022b for more details). The intra-assay CVs were 4.18 % (T1), 4.28 % (T2), and 4.06 % (T3). According to established cut-offs, hs-CRP values below 1.0 mg/L indicate a low, from 1.0 to 3.0 mg/L an average and above 3.0 mg/L a high risk for the development of cardiovascular diseases (e.g., Pearson et al., 2003).

2.4. Statistical analyses

The investigation of our research questions was based on the follow-up data, that is, T2 and T3, only. At baseline (T1), the majority of participants (66.1 %) had not started their job and almost half (45.8 %) were off duty (≥ 3 weeks) in the previous four weeks (for more information see Kaltenecker et al., 2023). Therefore, a valid assessment of participants' work situation as well as stress-related biomarkers at T1 was limited. In light of the panel attrition between T2 and T3 (~20 %), missing value analysis was performed with IBM SPSS Statistics (Version 29). 9.68 % of the values were missing and Little's MCAR test showed that missing data were not missing completely at random ($\chi^2 = 1176.16$, $df = 1056$, $p = .006$). Therefore, we imputed data using two consecutive methods: For control variables, missing T3 values were replaced by the within person mean of the respective T1 and T2 values. For key study variables, multiple imputation was conducted by creating five imputation datasets and pooling them to replace missing values. After

imputation of missing data, the final sample size was $n = 238$ for each wave.

First, descriptive analyses as well as Pearson correlations and ANCOVAs for associations between study variables were conducted in SPSS. Next, we performed structural equation modelling in Mplus (Version 8.9, Muthén and Muthén, 2017) consisting of two steps: First, a confirmatory factor analysis (CFA) was conducted to corroborate the factorial structure of the questionnaire measures (general work stress, technostress, burnout symptoms) at T2 and T3. To test for multicollinearity, we performed linear regressions and checked tolerance statistics (Field, 2009). Second, in order to test our research questions, we performed path analysis models based on the full panel design using maximum likelihood estimation with robust standard errors (MLR) to account for any skewness in the data (Yuan and Bentler, 2000).

Full panel designs, in which both predictor and outcome variables are assessed at all waves, allow for the testing of both *normal* or *stressor-to-strain*, that is, prospective effects of job characteristics on health, and *reversed* or *strain-to-stressor* effects, that is, prospective effects of health on the evaluation of job characteristics (Taris and Kompier, 2014). For research question 1, we performed a path analysis model (model I) on cross-lagged effects between the predictors (general work stress, technostress) and outcomes (burnout, HCC, CRP) including normal effects (i.e., predictors at T2 on outcomes at T3) as well as reversed effects (i.e., outcomes at T2 on predictors at T3). For research question 2 (model II), we ran the same model with additional cross-lagged associations among all outcome variables (i.e., outcomes at T2 on outcomes at T3). Both models also included cross-sectional (i.e., synchronous associations at T2/T3) and autoregressive (i.e., stability paths T2–T3) effects of all study variables as well as a predefined set of confounders. The self-report variables (general work stress, technostress, burnout symptoms) were adjusted for sex (T1), age (T1), profession (T1), shift work (T3), and full-time job (T3). The biomarkers (HCC, CRP) were additionally controlled for BMI (T1), physical activity (T3), smoking (T3), and contraceptive use (T3). Model fit was evaluated using comparative fit index (CFI), root mean squared error of approximation (RMSEA), and standardized root mean square residual (SRMR). The following cut-offs indicated adequate fit: $CFI > 0.90$, $RMSEA \leq 0.06$, $SRMR \leq 0.08$ (Hu and Bentler, 1999).

3. Results

3.1. Descriptives

In the final sample ($n = 238$), the majority of participants was female ($n = 182$, 76.5 %). Participants were mainly nurses ($n = 67$, 28.2 %), followed by physicians ($n = 53$, 22.3 %), research personnel ($n = 35$, 14.7 %), medical-technical personnel ($n = 34$, 14.3 %), administrative staff ($n = 14$, 5.9 %), and other ($n = 32$, 13.4 %), such as midwives, therapists etc.). The mean age was ($M \pm SD$) 28.5 ± 8.4 and the mean BMI was 23.47 ± 4.52 .

Main variable means at T2 and T3 are shown in Table 1. Regarding general work stress, job demands significantly increased over time. For technostress, work interruptions and information overload were significantly higher at follow-up. As for the outcome variables, burnout symptoms and HCC increased significantly, whereas CRP did not change significantly. Pearson correlations between work stressors, burnout symptoms, HCC, and CRP at T2 and T3 are depicted in Table 1A (Appendix).

3.2. Factorial structure of questionnaire measures

The CFA for both T2 (Fig. 1A, Appendix) and T3 (Fig. 2A, Appendix) showed that the scales work interruptions (T2: $\lambda = 0.77$, T3: $\lambda = 0.82$), multitasking (T2: $\lambda = 0.76$, T3: $\lambda = 0.79$), and information overload (T2: $\lambda = 0.59$, T3: $\lambda = 0.70$) loaded significantly and positively on a single latent factor "technostress". Furthermore, job demands (T2: $\lambda = 0.76$,

Table 1
Means, standard deviations (SDs), and paired t-tests of main variables at T2 and T3.

	T2 Mean (SD)	T3 Mean (SD)	Cohen's <i>d</i>	<i>p</i>
Job demands ¹	2.91 (0.99)	3.03 (0.97)	0.16	0.013
Job control ¹	3.03 (0.95)	3.00 (0.88)	0.04	0.580
General work stress (demand-control ratio) ²	1.10 (0.62)	1.14 (0.63)	0.08	0.237
Technostress: subscale work interruptions ¹	2.71 (0.93)	2.84 (0.81)	0.22	<0.001
Technostress: subscale multitasking ¹	3.22 (1.17)	3.21 (1.12)	0.01	0.896
Technostress: subscale information overload ¹	2.30 (0.89)	2.43 (1.03)	0.16	0.016
Technostress: composite score	2.74 (0.81)	2.83 (0.82)	0.17	0.008
Burnout symptoms: exhaustion ³	2.54 (0.84)	2.69 (0.90)	0.24	<0.001
Burnout symptoms: mental distance ³	1.80 (0.77)	1.97 (0.81)	0.24	<0.001
Burnout symptoms: total	2.17 (0.69)	2.33 (0.76)	0.29	<0.001
Hair cortisol concentration (HCC, pg/mg)	4.79 (4.58)	9.56 (7.98) ⁺	0.67	<0.001
C-reactive Protein (CRP, mg/L)	1.15 (1.51)	1.21 (1.37)	0.05	0.483

Note. *N* = 238; ⁺ *n* = 237.

¹ Scale range: 1 = not at all – 5 = to a very great extent.

² Range: 0.2 – 5.0.

³ Scale range: 1 = never – 5 = always.

T3: $\lambda = 0.64$) loaded significantly positively and job control (T2: $\lambda = -0.18$, T3: $\lambda = -0.17$) negatively on the factor “general work stress”. The BAT subscales exhaustion (T2: $\lambda = 0.88$, T3: $\lambda = 0.89$) and mental distance (T2: $\lambda = 0.56$, T3: $\lambda = 0.63$) were significant indicators of the factor “burnout”. Model fit was excellent at both time points, *CFI* = 0.97 (T2)/0.98 (T3), *RMSEA* = 0.06 (T2&T3), *SRMR* = 0.04 (T2&T3). Therefore, composite scores (i.e., means for technostress and burnout; JDC ratio) at T2 and T3, respectively, were used. Tolerance statistics in four linear regressions with technostress, general work stress (i.e., JDC ratio), and burnout as predictors and HCC and CRP as outcomes at T2 and T3, respectively, were all > 0.2, indicating the variables satisfied the assumption of non-multicollinearity.

3.3. Prospective associations of technostress, general work stress, burnout symptoms, hair cortisol, and inflammation

We first tested the path analysis model for research question 1 (model I), that is, cross-lagged associations between predictors (technostress, general work stress) and outcomes (burnout, HCC, CRP) controlled for covariates (sex, age, profession, shift work, full-time job, BMI, physical activity, smoking, and contraceptive use). Results showed that technostress at T2 was significantly negatively associated with HCC at T3 (standardized coefficient $\beta = -0.15$, $p = .003$). In contrast, technostress at T2 was not significantly associated with burnout ($\beta = 0.08$, $p = .133$) and CRP at T3 ($\beta = 0.04$, $p = .584$). General work stress at T2 was not significantly associated with any of the outcomes at T3, that is, burnout ($\beta = 0.01$, $p = .788$), HCC ($\beta = 0.06$, $p = .328$), and CRP ($\beta = -0.02$, $p = .824$). Concerning reversed effects, there were no significant lagged associations with technostress or general work stress at T3: burnout ($\beta = 0.07$, $p = .133$; $\beta = 0.02$, $p = .721$), HCC ($\beta = 0.02$, $p = .676$; $\beta = 0.03$, $p = .370$) and/or CRP ($\beta = 0.03$, $p = .373$; $\beta = 0.01$, $p = .853$).

We then tested the adjusted path analysis model for research question 2 (model II), which additionally included cross-lagged associations between outcome variables (burnout, HCC, and CRP). The results of model II are presented in Fig. 1. Consistent with the results of model I,

there was a significant negative effect of technostress at T2 on HCC at T3 ($\beta = -0.16$, $p = .003$), but no significant associations with the other outcomes. Again, general work stress at T2 was not significantly associated with any of the outcomes at T3, and there were no significant reversed effects. Concerning associations between outcomes, there was a positive cross-lagged effect of CRP at T2 on HCC at T3 ($\beta = 0.28$, $p = .001$). At the same time, there was a small negative effect of HCC at T2 on CRP at T3 ($\beta = -0.10$, $p \leq .001$). For burnout, there were no significant associations with HCC or CRP.

3.4. Additional analyses

To check for the robustness of the results, we ran the same two models including further relevant confounders for the biomarkers. First, HCC was additionally controlled for hair-related characteristics, that is, hair dyeing, hair treatment, washing frequency. The results were similar: For research question 1, there was still a significant negative effect of technostress at T2 on HCC at T3 ($\beta = -0.17$, $p = .002$). In model II, this effect remained significant as well ($\beta = -0.17$, $p = .003$), and there was still a positive effect of CRP at T2 on HCC at T3 ($\beta = 0.24$, $p = .019$) and a negative effect of HCC at T2 on CRP at T3 ($\beta = -0.09$, $p = .002$). Next, both HCC and CRP were additionally adjusted for season and hormone medication use not for contraception ($n = 13$, e.g., use of asthma inhalers containing corticosteroids). Again, the results were similar with a negative effect of technostress at T2 on HCC at T3 (Model I: $\beta = -0.16$, $p = .003$; Model II: $\beta = -0.16$, $p = .005$), a positive effect of CRP at T2 on HCC at T3 ($\beta = 0.27$, $p = .005$), and a negative effect of HCC at T2 on CRP at T3 ($\beta = -0.08$, $p = .011$). No other cross-lagged associations between main variables were significant. Fit indices for the final full model were *CFI* = 0.82, *RMSEA* = 0.07, *SRMR* = 0.08.

In addition, to further contextualize the associations between the key variables and the covariates, we conducted partial correlations and ANCOVAs using the adjustments applied in the path analysis models (Table 2). For age, there were significant negative correlations with general work stress and HCC. Shift workers reported higher general work stress. Profession had significant effects on technostress and general work stress with physicians reporting higher strain. BMI was positively correlated with HCC as well as CRP, and physical activity only with HCC. Smoking was negatively correlated with HCC. Contraceptive use was positively correlated with both biomarkers. Regarding season, there were significant effects for HCC with higher levels in summer–autumn and for CRP with higher levels in autumn than in winter (see Table 2).

4. Discussion

4.1. Findings and contributions to the literature

To the best of our knowledge, for the first time, the biological effects of work-related technostress in terms of HPA-axis function (i.e., HCC) and chronic low-grade inflammation (i.e., CRP) were investigated in a prospective study within a naturalistic occupational setting. Results showed that technostress was consistently negatively associated with HCC (research question 1) and that CRP was positively associated with HCC, while HCC was negatively associated with CRP (research question 2) over a time lag of 6 months. Given the lack of research – especially prospective – on work stress including technostress, HCC, and low-grade inflammation, our study contributes to the current evidence base in several ways.

First, in contrast to previous studies that often rely on subjective evaluations, we investigated physiological effects of technostress by measuring two key biological systems through which chronic stressors “get under the skin” and lead to disease, that is, the HPA-axis and the inflammatory system. The small literature that has assessed the association between technostress and biological stress responses have predominantly focused on acute stress responses (Becker et al., 2022a;

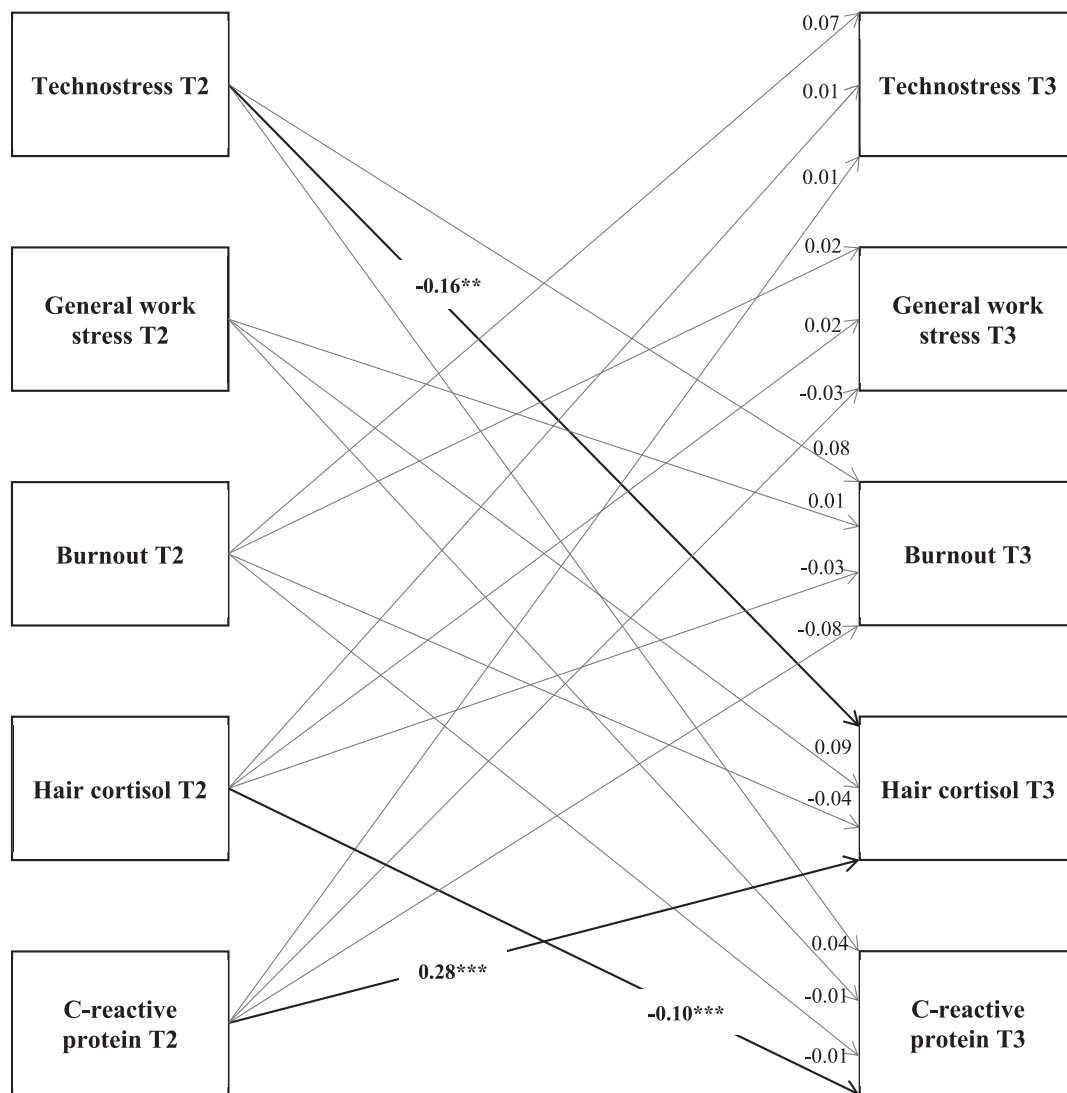


Fig. 1. Path model showing cross-lagged associations between technostress, general work stress (job demand-control ratio), burnout symptoms, hair cortisol concentration, and C-reactive protein at T2 and T3. *Note.* Standardized estimates. Adjusted for age, sex, profession, shift work, full-time job, and hair cortisol and C-reactive protein additionally for BMI, physical activity, smoking, use of contraceptives; cross-sectional and autoregressive associations are not shown; bold arrows indicate significant associations; ** $p \leq 0.01$, *** $p \leq 0.001$.

Riedl, 2012). In the present study, technostress (as measured by work interruptions, multitasking requirements and information overload due to digital technologies) was associated with reduced HCC, indicating a specific effect of technostress in terms of a longer-term alteration of HPA-axis activity beyond the influences of general work stress (i.e., JDC ratio). Consistent with our previous studies (Becker et al., 2023; Kaltenecker et al., 2023), we did not identify effects of technostress on the inflammatory system. However, these two studies were not prospective. Furthermore, general work stress was not significantly related to either HCC, or CRP, which adds to the limited and mixed evidence base on prospective associations of work stressors with HCC and low-grade inflammation (Kaltenecker et al., 2021; Schaafsma et al., 2021). Notably however, in model II, there was a weak positive, yet non-significant, association of general work stress with HCC. This aligns with a recent study in medical students showing a positive association of demands with HCC (Heming et al., 2023).

Second, given the paucity of longitudinal studies on technostress and mental health (Berg-Beckhoff et al., 2017; Dragano and Lunau, 2020), we contribute empirical insights into prospective associations with burnout as a key psychological outcome in chronic stress experience. Apart from a weak, non-significant association, we did not find

prospective associations between technostress and burnout symptoms. This is not in line with findings from cross-sectional studies, which identified positive associations between constructs (e.g., Kaltenecker et al., 2023; Kasemy et al., 2022). Moreover, to provide a more comprehensive understanding of the pathways from burnout to health problems, we analyzed associations of burnout with the two biomarkers, revealing no significant prospective associations. Research on burnout and HCC is scarce with initial findings suggesting a non-linear relationship between accumulated burnout symptomatology and elevated HCC (Penz et al., 2018; Wendsche et al., 2020). Given that the burnout symptom levels in our sample were average and below clinical cut-offs (Schaufeli et al., 2023; Schaufeli et al., 2019), the null result seems plausible.

Third, our cohort study in a real work context complements and extends previous laboratory experiments providing external validity. The study was carried out in a high-risk environment for work-related stress in general, such as high work load or emotional stressors related to patient care, and technostress in particular due to health information technology use (Dawe et al., 2016; Melnick et al., 2020). Our results showed that technostress was rated as moderate across the sample with highest ratings in physicians, indicating that the assessed

Table 2
Associations of technostress, general work stress (job demand-control ratio), burnout symptoms, hair cortisol concentration (HCC), and C-reactive protein (CRP) with control variables (partial correlations and ANCOVAs).

Partial correlations (r , p)	Control variables				
	Technostress T3	General work stress T3	Burnout T3	HCC T3	CRP T3
Sex T1 (male, female)	-0.01; 0.848	0.05; 0.421	0.11; 0.104	0.12; 0.102	0.09; 0.178
Age T1	-0.01; 0.912	-0.18; 0.007	-0.11; 0.095	-0.17; 0.020	-0.07; 0.308
Shift work T3 (no, yes)	0.06; 0.358	0.19; 0.004	0.04; 0.511	-0.09; 0.225	0.00; 0.962
Full-time T3 (no, yes)	0.12; 0.075	0.06; 0.336	0.10; 0.117	-0.06; 0.444	0.09; 0.190
BMI T1	-	-	-	0.29; <0.001	0.36; <0.001
Physical activity T3 ¹	-	-	-	0.21; 0.004	-0.03; 0.701
Smoking T3 ²	-	-	-	-0.18; 0.011	-0.07; 0.289
Contraceptive use T3 (no, yes)	-	-	-	0.15; 0.038	0.27; <0.001
Hormone medication T3 (no, yes)	-	-	-	-0.13; 0.077	-0.03; 0.665
Hair dyeing T3 (no, yes)	-	-	-	0.10; 0.159	-
Hair treatment T3 (no, yes)	-	-	-	-0.01; 0.916	-
Hair washing frequency per week T3	-	-	-	-0.05; 0.487	-
ANCOVAs (F (df); p)					
Profession	$F(5) = 6.23$; <0.001 ³	$F(5) = 3.05$; 0.011 ⁴	$F(5) = 0.30$; 0.911	$F(5) = 0.26$; 0.934	$F(5) = 0.23$; 0.951
Season, for HCC (T2 & T3)	-	-	-	$F(7) = 17.49$; <0.001 ⁵	-
Season, for CRP (T2 & T3)	-	-	-	-	$F(3) = 4.95$; 0.002 ⁶

Note. Text in bold if significant at $p < 0.05$.
¹ Scale range: 1 = not at all – 5 = very much.
² Scale range: 1 = never smoked – 5 = yes, every day.
³ Post-hoc tests with Bonferroni correction: Technostress at T3 was significantly higher in physicians than in nurses, medical-technical personnel, research staff, and other professions.
⁴ Quade non-parametric ANCOVA with Bonferroni correction: general work stress at T3 was significantly higher in physicians than in nurses.
⁵ Quade non-parametric ANCOVA with Bonferroni correction: HCC in summer, summer/autumn, and autumn was significantly higher than in autumn/winter, winter, winter/spring and spring, and HCC in spring/summer was significantly higher than in winter/spring.
⁶ Quade non-parametric ANCOVA with Bonferroni correction: CRP in autumn was significantly higher than in winter.

technostressors – especially multitasking with the highest means – played a relevant role at participants’ workplaces. The identification of at-risk persons and specific adverse working conditions is a crucial starting point for the prevention of stress-related diseases in healthcare professionals.

4.2. Post-hoc explanations for observed findings

As our main finding of an inverse association of technostress with

HCC contradicts the traditional view of HPA-axis activity increases with stress, we suggest the following possible post-hoc explanations. On the one hand, this finding could be explained by hypocortisolism as a consequence to chronic stress. In their large-scale meta-analysis Miller et al. (2007) found that timing plays a critical role with elevated HPA-axis activity at stressor onset but a reduction over time, hence providing an explanation for the formerly conflicting findings of both hyper- and hypocortisolism in response to stress. Concerning work stress in particular, this two-stage notion of HPA-axis activation was also supported by a previous study, which found that increased effort-reward-imbalance was prospectively associated with decreased HCC indicating a blunted cortisol response (Penz et al., 2019). Regarding our results, one could hypothesize that although HCC increased over time, participants who experienced higher amounts of technostress had a lower HCC response at the next time point, which suggests dampened HPA-axis activity due to long-term work stress. Between baseline (T1) and T2 during the phase of organizational socialization in the new job, participants might have perceived high stress levels due to intensive learning and adaptation requirements. In addition, we can only speculate that before commencing their new employment, some participants might have been exposed to chronic stressors, such as high job demands in former jobs, demanding medical education, unemployment, or also other chronic stressors in their private lives. However, burnout levels in our sample, although increasing over time, were rather low, which might be due to the early phase of employment in most of the participants. In contrast to burnout and HCC, CRP did not change significantly over time, and this could possibly be explained by high starting values facilitating a ceiling effect. Yet, baseline CRP levels in our sample were comparable to levels in other samples including healthy (and young) adults and analyzed with the same method (Becker et al., 2023; Becker et al., 2022b).

On the other hand, another plausible explanation could be that participants who reported high levels of technostress in fact showed less physiological stress as indicated by decreased HCC. According to the integrated specificity model, the physiological stress response is not uniform, but shaped by the nature of the stressor and the individual cognitive appraisal of it (Kemeny, 2003). Drawing upon the Transactional Model of Stress (Lazarus and Folkman, 1984) physiological responses are substantially influenced by the appraisal of the stressor, that is, whether it poses a challenge or a threat, its perceived controllability and whether it threatens social status or self-esteem (Kemeny, 2003). In our study, participants might have evaluated the technostressors as a challenge with high chances of mastery and sufficient coping capabilities to meet the work demands. Technostressors that are appraised as challenge stressors, that is, as beneficial for accomplishing work tasks, were shown to be associated with positive emotions, which in turn was related to high job satisfaction in nurses (Califf and Sarker, 2020). Furthermore, even though technostressors, such as work interruptions, may be perceived as uncontrollable, they might also be regarded as a legitimate, integral part of the job in healthcare (Semmer et al., 2019) and therefore, as predictable or even “self-chosen”. Finally, our operationalization of technostressors did not directly include a social-evaluative component, what together with uncontrollability is suggested to elicit a strong HPA-axis activation (Dickerson and Kemeny, 2004). Taken together, the specific nature of technostress and its cognitive appraisal by the employees might have led to a more favorable physiological response.

Eventually, we identified reciprocal associations between CRP and HCC. The finding of a positive effect of CRP on HCC supports the notion of glucocorticoid resistance, meaning that inflammation leads to an impairment of the negative feedback loop of the HPA-axis, which in turn leads to hypercortisolism (Pariante, 2017). At the same time, the finding of a negative effect of HCC on CRP confirms the established understanding of an anti-inflammatory effect of cortisol (see Sorrells and Sapolsky, 2007).

4.3. Limitations

Our findings need to be reflected in the light of several important limitations. First, regarding internal validity, it remains an open question whether the technostress scales measured stress *induced* by ICTs or rather work stress per se simply *mediated* by ICTs, that is, ICTs as a primary stressor versus medium transmitting common work stressors (Benlian, 2020). However, scale reliability was good, and factorial validity was confirmed. Moreover, we assessed burnout symptoms with a well-established, yet for the sake of practicability and efficiency, an abbreviated measure. We acknowledge that hence, the full burnout symptomatology was not captured. We used a parsimonious measure consisting of the two core dimensions of burnout, inability (i.e., exhaustion) and unwillingness (i.e., mental distance) to spend work-related effort, as suggested in previous literature (Schaufeli and Taris, 2005). Only two items per subscale were used, yet even single-item measures for burnout in healthcare providers have proven useful (Rohland et al., 2004; West et al., 2009). Although we controlled for a broad set of covariates, we cannot preclude confounding influences on stress perceptions and physiology by external factors, e.g., due to the Covid-19 pandemic or geopolitical events. Furthermore, because of threats to validity at T1, we had to constrain our design to two waves with a time lag of 6 months. It remains thus unclear, if the length of this interval was appropriate to capture the “true” effect, and the inclusion of a third (or even more) measurement time point(s) would have provided deeper insights into trajectories or potential mediating effects (Ployhart and Vandenberg, 2010; Taris and Kompier, 2014). Nonetheless, the application of the full panel design allowed us to test for normal, reversed, and reciprocal causality at the same time to unveil potential interactions of work characteristics, psychological states, and stress physiology (Taris and Kompier, 2014).

Concerning external validity, our data stemmed from young hospital employees, and that limits the generalizability of our findings to other age groups and professions. Moreover, although representative for healthcare, our sample was predominately female. Hence, our findings ought to be replicated among more experienced workers in different professional fields with a higher proportion of males.

4.4. Implications for research and practice

Given the infancy of research on health-related effects of technostress, our exploratory study provides important implications for future research. First, it advocates the viability of biomarker measurements in the quest for physiological correlates of technostress. For the rather novel approach of HCC analysis, our study provides further evidence for associations with covariates, which should be considered in future research. Compared to the meta-analysis by Stalder et al. (2017), we also identified significant associations with relevant covariates like BMI, age, and contraceptive use (the latter two however in different directions), but not with others (such as sex, hair washing frequency, and hair treatment), although comparability with our sample and method was limited. Moreover, our results suggested seasonal variation of HCC with higher concentrations in the summer and autumn than winter and spring months. This is in line with some of the few existing studies (Braig et al., 2015; Staufenbiel et al., 2015), but not with others (Abell et al., 2016). Regarding low-grade inflammation, CRP is an important indicator for the risk of cardiovascular diseases, which was in the average range in our sample. Yet, future studies should also consider further biomarkers, such as cytokines or cytokine imbalance for a more comprehensive understanding of inflammation and interactions with cortisol (Kaltenecker et al., 2020 for a list of inflammatory markers, Sorrells and Sapolsky, 2007). Building on our preliminary findings, more prospective studies with advanced, that is, full panel, designs and longer follow-ups are needed to investigate chronic psychophysiological effects of technostress and long-term health consequences.

If supported by future research, our findings have important

implications for occupational health and safety in digitalized work environments. Chronic alterations of the HPA-axis activity are involved in a broad range of medical conditions, such as diabetes or obesity, and psychiatric conditions, such as depression or psychosomatic disorders (Chrousos, 2009; Miller et al., 2007). Technostress at work might therefore pose a health risk, which warrants the development of targeted prevention and intervention measures. At the same time, technology can be a useful tool for stress management at work, as was shown for a smartphone-based mindfulness meditation training intervention which reduced pro-inflammatory gene expression in customer service workers (Dutcher et al., 2022).

4.5. Conclusions

In conclusion, for the first time, this cohort study explored associations of technostress, general work stress, burnout symptoms, HCC, and chronic low-grade inflammation in a prospective repeated measurement design. The results provide preliminary indications for HCC alterations in hospital employees due to technostress. Moreover, the study yields insights into the complex interplay of the HPA-axis and inflammation. More prospective studies on the biological mechanisms linking chronic stress with disease are essential to improve our understanding of the potential health risks for workers in digitalized work settings.

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CRedit authorship contribution statement

Helena C. Kaltenecker: Writing – review & editing, Writing – original draft, Visualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Mathew D. Marques:** Writing – review & editing, Visualization, Software, Methodology, Formal analysis, Conceptualization. **Linda Becker:** Writing – review & editing, Methodology, Conceptualization. **Nicolas Rohleder:** Writing – review & editing, Methodology, Conceptualization. **Dennis Nowak:** Writing – review & editing, Resources, Funding acquisition, Conceptualization. **Bradley J. Wright:** Writing – review & editing, Validation, Supervision, Software, Methodology, Formal analysis, Conceptualization. **Matthias Weigl:** Writing – review & editing, Validation, Supervision, Resources, Project administration, Methodology, Funding acquisition, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

After internal approval of the data security committee of LMU university hospital, the study’s minimal and anonymized underlying data set will be available from the corresponding author [HCK] upon

reasonable request.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.bbi.2024.01.222>.

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