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Chiara Costi
University of Verona
Pierluigi Sacco
University of Chieti-Pescara
Valentina Rotondi
valentina.rotondi@supsi.ch

University of Applied Sciences and Arts of Southern Switzerland

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Subjective and Biological Indicators Capture Complementary Dimensions of Adaptation to Caregiving: Evidence from the Transition to Parenthood

Chiara Costi (University of Verona, Verona, Italy)

Pierluigi Sacco (University of Chieti-Pescara, Pescara, Italy)

Valentina Rotondi (Corresponding autor: SUPSI & University of Oxford

Lugano, Switzerland. valentina.rotondi@supsi.ch)

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Abstract

Humans continuously adapt to sustained social demands, yet this process is typically assessed using subjective reports of wellbeing, implicitly assuming that self-perceptions summarize overall adaptation. Whether subjective evaluations adequately capture broader biological responses remains largely unknown. We propose and test the hypothesis that adaptation is domain-specific, such that subjective, physiological, and epigenetic indicators provide complementary rather than interchangeable information following the same life transition. We examine the transition to first-time parenthood as a natural setting in which to study adaptation to sustained caregiving using longitudinal data from the UK Household Longitudinal Study (N = 4,249), combining regression models with inverse probability weighted regression adjustment based on pre-parenthood characteristics. We compare patterns across life satisfaction, perceived health, physiological regulation, and the pace of epigenetic ageing. The transition to first-time parenthood is associated with higher life

satisfaction but no change in health satisfaction, together with lower neuroendocrine and metabolic dysregulation and a slower pace of epigenetic ageing. Similar multidimensional profiles are not observed for financial hardship, psychological distress, or sleep deprivation, which instead show either broadly convergent deterioration across domains or associations that are predominantly subjective. Together, these findings suggest that human adaptation may be best understood as a multidimensional process and that subjective and biological indicators provide complementary rather than interchangeable perspectives on this process.

Introduction

Human beings constantly adapt to sustained demands. Caring for a newborn, managing a chronic illness, or providing long-term care for an ageing parent all require profound behavioural, psychological, and physiological adjustment (McEwen and Stellar, 1993; McEwen, 2007; Sterling, 2012). Yet human adaptation is typically assessed using a single family of outcomes—most commonly self-reported wellbeing and health—implicitly assuming that subjective experience provides a reliable summary of how individuals are adapting overall. Whether this assumption is justified remains largely unknown. Throughout the paper, we treat adaptation as a latent process rather than a directly observable outcome. Subjective wellbeing, perceived health, physiological regulation, and biological ageing are therefore conceptualised as distinct indicators that may each capture different aspects of adaptation rather than direct measures of adaptation itself.

Existing research has largely studied subjective wellbeing and physiological adaptation separately. The wellbeing literature documents the psychological costs and benefits of sustained roles such as parenthood (Nelson et al., 2014; Myrskylä and Margolis, 2014; Mikucka and Rizzi, 2020), whereas neuroscience, ageing research, and stress physiology examine biological adaptation using biomarkers, allostatic load, and epigenetic ageing (McEwen, 2007; Sironi et al., 2020; Ryan et al., 2024; Hukkanen et al., 2026; Kim et al., 2025). Because these traditions have developed largely in parallel, we know remarkably little about whether subjective evaluations and physiological functioning evolve together under sustained demands. Consequently, the widespread assumption that subjective wellbeing provides an adequate summary of human adaptation has rarely been tested directly. There is no strong theoretical reason why conscious evaluations should necessarily track physiological regulation. Indeed, if subjective experience, conscious health appraisal, and physiological regulation serve different adaptive functions, they need not evolve in parallel. Rather than expecting all indicators to move together, adaptation may be expressed differently across domains. Several theoretical frameworks predict that such divergence may arise because subjective evaluations and physiological regulation operate over distinct functional domains and timescales (McEwen and Stellar, 1993; McEwen, 2007; Sterling, 2012; Pezzulo et al., 2015; Friston, 2010; Parr et al., 2022).

Sustained caregiving provides an ideal setting in which to examine this possibility. Unlike

acute stressors, sustained caregiving involves continuous behavioural, cognitive, and physiological adjustment over extended periods (Christian et al., 2023), making it possible to evaluate whether different indicators of adaptation evolve in parallel or follow distinct trajectories. If these indicators capture distinct adaptive processes rather than a single underlying dimension, they need not evolve in parallel.

As a common, well-defined, and biologically salient life transition, entry into parenthood offers an opportunity to study adaptation under sustained caregiving demands within a population setting. It entails persistent demands on time, sleep, leisure, and economic resources, and is associated with a well-documented tension between increased meaning and reduced day-to-day wellbeing—the so-called "parenthood paradox" (Kohler and Mencarini, 2016). A large body of research shows that the transition to parenthood imposes immediate constraints on time, leisure, and financial resources, leading to declines in domain-specific wellbeing even when global evaluations are more mixed (Nomaguchi and Milkie, 2003; Bianchi et al., 2012; Nelson et al., 2014; Mencarini and Sironi, 2012; Kohler and Mencarini, 2016; Rotondi et al., 2024). At the same time, parenthood is a biologically intensive process involving coordinated changes across endocrine, immune, metabolic, neural, and behavioural systems (Yu et al., 2025; Takeshita, 2024; Young et al., 2025). Pregnancy and the postpartum period are accompanied by substantial brain remodelling (Hoekzema et al., 2017; Kim et al., 2010; Barba-Müller et al., 2019; Luders et al., 2022), some of which appears to persist well beyond the perinatal period (Martínez-García et al., 2021; Rotondi et al., 2024; Straathof et al., 2026). Emerging evidence further suggests that reproductive history may be associated with more favourable brain-ageing profiles later in life, although findings remain mixed across markers and populations (Takeshita, 2024; Ryan et al., 2024; Hukkanen et al., 2026; Young et al., 2025; Sironi et al., 2020; Schneider et al., 2025). Together, these behavioural demands and enduring physiological adaptations make the transition to parenthood a natural setting for studying multidimensional human adaptation (Hoekzema et al., 2017, 2020; Pawluski et al., 2021; Luders et al., 2022; Martínez-García et al., 2023; Yu et al., 2025).

Existing theories of physiological regulation provide a useful conceptual framework for understanding why different dimensions of adaptation may not evolve in parallel. In particular,

allostatic models emphasise that physiological regulation reflects the cumulative integration of repeated demands across multiple biological systems rather than momentary experiences of stress (McEwen and Stellar, 1993; McEwen, 2007; Sterling, 2012). A complementary theoretical perspective is provided by hierarchical models of predictive regulation developed within the active inference framework. Rather than treating subjective experience and physiological regulation as different expressions of a common latent state, active inference conceptualises the organism as a hierarchical generative system in which prediction-error minimisation operates simultaneously across nested levels updating over different temporal horizons (Friston, 2010; Pezzulo et al., 2015; Parr et al., 2022). Within this framework, conscious evaluations are especially sensitive to short-term prediction errors, whereas physiological regulation reflects slower recalibration of internal states.

We evaluate whether the transition to first-time parenthood exhibits the empirical pattern predicted by the domain-specific adaptation hypothesis. Specifically, we ask whether this transition is associated with coordinated or domain-specific patterns across subjective wellbeing, perceived health, physiological regulation, and biological ageing. Our hypothesis is that sustained caregiving need not produce uniform changes across all indicators of adaptation but may instead generate distinct patterns across subjective, physiological, and biological domains. If different indicators capture complementary dimensions of adaptation, no single measure should be expected to provide a complete account of how humans adapt to sustained caregiving demands.

Two predictions follow and structure the empirical analysis. First, if these indicators capture distinct domains of adaptation, the transition to parenthood should not produce uniform changes across all measures of adaptation. Second, if this divergence reflects a distinctive feature of adaptation to sustained caregiving rather than a generic consequence of adversity, it should not be reproduced by other adverse exposures such as financial hardship, psychological distress, or sleep deprivation, which instead are expected to exhibit more convergent associations across domains.

Using longitudinal data from the UK Household Longitudinal Study (UKHLS; N = 4,249), we exploit the transition to first-time parenthood as a natural setting in which to compare subjec-

tive wellbeing, physiological regulation, and biological ageing. Our transition-anchored design follows individuals as they enter parenthood and combines conventional regression models with inverse probability weighted regression adjustment to improve comparability between first-time parents and childless individuals. We then evaluate the specificity of the observed associations through a series of falsification analyses using financial hardship, psychological distress, sleep deprivation, and relational context as comparison conditions.

Together, these analyses provide a multidimensional assessment of human adaptation to sustained caregiving and test whether subjective, physiological, and epigenetic indicators capture complementary rather than interchangeable dimensions of adaptation.

Results

The transition to first-time parenthood is associated with divergent patterns across domains of adaptation

To evaluate whether indicators of adaptation to sustained caregiving follows coordinated or domain-specific patterns, we compare subjective wellbeing, physiological regulation, and biological ageing across the transition to first-time parenthood. Because blood-based biomarkers and epigenetic measures are collected only during the nurse assessment, all analyses are conducted within a transition-anchored cross-sectional framework. We compare individuals who become first-time parents at the nurse assessment with respondents who remain childless, excluding those who transition into parenthood during the subsequent two survey waves to minimise anticipation effects. Conventional regression estimates are complemented by inverse probability weighted regression adjustment (IPWRA), which models treatment assignment using pre-parenthood characteristics measured at $t-1$, thereby improving comparability between first-time parents and childless respondents while preserving the temporal ordering between covariates and outcomes. Unless otherwise stated, coefficients are expressed in standard deviation (SD) units. The Dunedin ageing measures are reported in their original scale.

Figure 1 (Appendix Tables A4–A9) summarises the estimated associations between the transition to first-time parenthood and outcomes spanning four domains of adaptation: life satisfac-

tion, satisfaction with health, physiological regulation, and biological ageing.

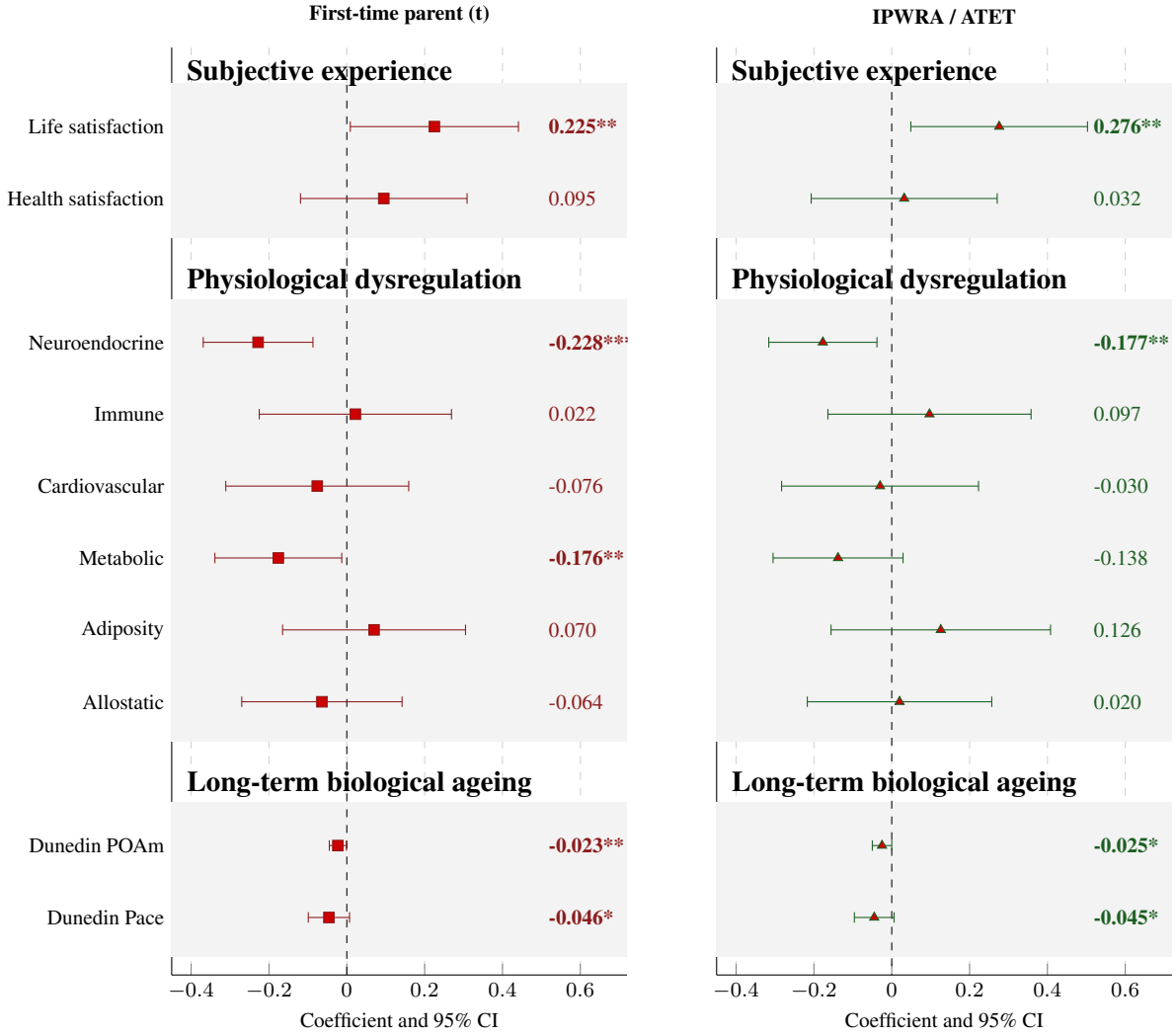
Across all outcomes, conventional regression and IPWRA yield highly similar estimates in both sign and magnitude, indicating that the overall pattern is robust to adjustment for observable differences in pre-parenthood characteristics.

A clear domain-specific pattern emerges. Among subjective outcomes, the transition to first-time parenthood is associated with higher life satisfaction in both the conventional regression ($\beta = 0.225$, $p < 0.05$) and the IPWRA analysis ($\beta = 0.276$, $p < 0.05$). In contrast, satisfaction with health remains statistically indistinguishable from that of childless individuals and close to zero under both specifications ($\beta = 0.095$ and $\beta = 0.032$, respectively).

Physiological regulation exhibits a contrasting profile. Neuroendocrine dysregulation is substantially lower among first-time parents in the conventional regression ($\beta = -0.228$, $p < 0.01$) and remains significantly lower under IPWRA ($\beta = -0.177$, $p < 0.05$). Metabolic dysregulation is likewise lower in the conventional regression ($\beta = -0.176$, $p < 0.05$), with a coefficient of similar magnitude under IPWRA ($\beta = -0.138$), although estimated less precisely. By contrast, immune, cardiovascular, adiposity, and allostatic dysregulation remain close to zero across both specifications.

A similar pattern is observed for biological ageing. Both epigenetic measures point towards slower biological ageing, although the evidence is somewhat stronger for DunedinPoAm than for DunedinPACE. Following the transition to parenthood. DunedinPoAm is lower in both the conventional regression ($\beta = -0.023$, $p < 0.05$) and the IPWRA analysis ($\beta = -0.025$, $p < 0.10$), while DunedinPACE also shows negative associations of similar magnitude ($\beta = -0.046$ and $\beta = -0.045$, respectively; both $p < 0.10$).

Taken together, the estimated associations do not indicate a uniform pattern across domains of adaptation. Instead, the transition to first-time parenthood is associated with higher life satisfaction, no detectable change in perceived health, and more favourable profiles of physiological regulation and biological ageing. Although these findings are observational, they are consistent with the possibility that subjective evaluations, perceived health, physiological regulation, and biological ageing capture complementary rather than interchangeable dimensions of human adaptation.



Note: Points denote coefficients and horizontal bars indicate 95% confidence intervals. All outcomes are standardized, except the Dunedin ageing measures. First-time parent estimates refer to the transition into parenthood. IPWRA estimates report average treatment effects on the treated. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Figure 1: Associations between the transition to parenthood and subjective, biological, and epidemiological outcomes.

Assessing selection into parenthood

A potential concern is that the observed biological profiles reflect selection into parenthood rather than differences emerging around the transition itself. Because biomarkers are available only during the nurse assessment, pre-parenthood biological trajectories cannot be observed directly. We therefore exploit the longitudinal structure of the UKHLS to conduct two complementary selection analyses (Appendix Table A10).

Panel A compares respondents who remained childless throughout the observation period (never parents) with respondents who were childless at the nurse assessment but entered parenthood subsequently (future parents). Unlike the main analyses, which exclude individuals who transition into parenthood during the subsequent two survey waves to minimise anticipation effects, this comparison is designed specifically to assess pre-transition selection and therefore includes all respondents who later become parents. If individuals selecting into parenthood already possessed more favourable subjective or biological profiles, systematic differences should be evident before the transition. Instead, future parents are broadly comparable to never parents across subjective wellbeing, physiological regulation, and biological ageing. Coefficients are generally small, statistically insignificant, and centred around zero. The only exception is metabolic dysregulation, which is modestly higher among future parents ($\beta = 0.111$, $p < 0.05$), opposite in direction to the association observed following the transition to parenthood.

Panel B compares respondents who became first-time parents at the nurse assessment with the future parents from Panel A. Because both groups eventually enter parenthood, this comparison substantially reduces concerns about selection into parenthood and provides a more demanding assessment of whether the observed differences emerge around the transition itself. Consistent with the main analyses, first-time parents exhibit lower metabolic dysregulation than future parents. Although the remaining coefficients are estimated less precisely, they are uniformly aligned with the patterns observed in the main analyses, with higher life satisfaction and more favourable physiological and epigenetic profiles among first-time parents.

Taken together, these selection analyses provide little evidence that the multidimensional patterns reported above primarily reflect pre-existing differences between individuals who do and do not become parents. Instead, they are consistent with the possibility that the observed

differences emerge around the transition to first-time parenthood.

Demographic heterogeneity and variation across the parenting cycle

We next examined whether the associations identified above differed across respondents' demographic characteristics or stages of the parenting life course.

Overall, there is little evidence that the multidimensional pattern associated with the transition to first-time parenthood differs systematically by respondents' sex or age (Appendix Tables A11–A16). Women exhibit a somewhat larger increase in life satisfaction than men (interaction $\beta = 0.384$, $p < 0.10$), whereas no significant gender differences emerge for satisfaction with health. Likewise, interactions with respondents' age are generally small and inconsistent. Although a small number of age-specific interactions reach conventional levels of statistical significance, they do not form a coherent pattern across subjective, physiological, or biological outcomes. Overall, the results suggest that the multidimensional profile associated with the transition to parenthood is broadly shared across demographic groups.

We then examined whether the observed associations varied across stages of the parenting life course. Unlike the transition-anchored analyses presented above, this exercise necessarily relies on the broader sample of all parents because child age is observed only after entry into parenthood. Restricting the analysis to first-time parents would leave almost all respondents with children aged 0–2 years, providing little meaningful variation in child age.

Appendix Tables A17–A19 indicate that the strongest associations are concentrated during the early years following entry into parenthood. Parents whose oldest child is aged 0–2 years report substantially higher life satisfaction ($\beta = 0.205$, $p < 0.01$), whereas satisfaction with health remains close to zero across all child-age categories. Physiological regulation follows a similar pattern. Cardiovascular dysregulation is lower among parents of children aged 0–2 years ($\beta = -0.159$, $p < 0.01$), while immune ($\beta = -0.175$, $p < 0.05$), metabolic ($\beta = -0.157$, $p < 0.01$), and allostatic dysregulation ($\beta = -0.183$, $p < 0.01$) are most favourable among parents whose oldest child is aged 3–4 years. Across subsequent child-age categories, these associations generally attenuate towards zero.

By contrast, biological ageing exhibits little evidence of heterogeneity across the parenting

cycle. Neither DunedinPoAm nor DunedinPACE varies meaningfully with the age of the oldest child, with coefficients remaining close to zero across all categories.

Taken together, these analyses indicate that the multidimensional pattern associated with parenthood is not confined to particular demographic groups. Rather, the strongest associations are concentrated during the first years following entry into parenthood, whereas differences in biological ageing remain comparatively stable across later stages of the parenting life course.

Falsification and robustness analyses

To assess whether the domain-specific pattern associated with the transition to first-time parenthood simply reflects broader forms of adversity, reporting behaviour, or relational circumstances, we conducted a series of falsification and robustness analyses. Specifically, we compared the parenthood estimates with those associated with financial hardship (Tables A20–A22 in the Appendix), marital status (Tables A23–A25 in the Appendix), psychological distress (Tables A26–A28 in the Appendix), domain-specific satisfaction (Tables A29–A31 in the Appendix), and sleep deprivation (Tables A32–A34 in the Appendix). If the biological profile observed among first-time parents merely reflected generic stress or socioeconomic disadvantage, similar patterns should emerge under these alternative conditions.

Financial hardship provides the clearest comparison. As expected, respondents experiencing financial distress report substantially lower life satisfaction ($\beta = -0.667$, $p < 0.01$) and health satisfaction ($\beta = -0.339$, $p < 0.01$). At the same time, financial hardship is associated with poorer physiological regulation, including greater metabolic dysregulation ($\beta = 0.241$, $p < 0.01$), higher adiposity dysregulation ($\beta = 0.152$, $p < 0.05$), increased allostatic load ($\beta = 0.187$, $p < 0.01$), and accelerated biological ageing measured by both Dunedin PoAm ($\beta = 0.029$, $p < 0.01$) and DunedinPACE ($\beta = 0.032$, $p < 0.05$). Thus, financial hardship is characterised by a broadly convergent deterioration across subjective, physiological, and epigenetic outcomes. By contrast, interactions between first-time parenthood and financial distress are generally small and statistically insignificant, with the only notable exception being a more favourable Dunedin PoAm among financially distressed parents ($\beta = -0.058$, $p < 0.05$). Overall, financial hardship does not reproduce the multidimensional profile associated with the

transition to parenthood.

We next examined whether the associations depended on respondents' relational circumstances. As expected, divorced or separated respondents reported substantially lower life satisfaction ($\beta = -0.290$, $p < 0.01$), while never-married individuals also exhibited lower subjective wellbeing ($\beta = -0.186$, $p < 0.01$). However, these relational disadvantages did not systematically alter the associations between first-time parenthood and either subjective or biological outcomes. If anything, life satisfaction increased somewhat more among divorced/separated ($\beta = 0.262$, $p < 0.10$) and never-married parents ($\beta = 0.353$, $p < 0.10$). Likewise, although several individual biomarker interactions reached conventional significance—including stronger metabolic regulation among divorced parents ($\beta = -1.022$, $p < 0.01$) and lower neuroendocrine, immune, adiposity, and allostatic dysregulation among never-married parents—these effects were not accompanied by systematic differences in biological ageing, for which interaction terms remained statistically insignificant. Overall, the physiological associations linked to first-time parenthood appear broadly similar across relational contexts.

Psychological distress provides a complementary falsification test. Consistent with previous research, mental distress is strongly associated with lower life satisfaction ($\beta = -0.599$, $p < 0.01$) and poorer satisfaction with health ($\beta = -0.310$, $p < 0.01$), but shows little association with physiological biomarkers or biological ageing once covariates are taken into account. Moreover, interactions between first-time parenthood and mental distress are generally close to zero. The only exception is a somewhat stronger reduction in metabolic dysregulation among psychologically distressed parents ($\beta = -0.281$, $p < 0.05$). Thus, psychological distress does not reproduce the pattern observed following the transition to parenthood.

We further examined whether the parenthood estimates merely reflected differences in evaluations of specific life domains by controlling for satisfaction with income and leisure. Although both variables strongly predict subjective wellbeing, the estimated association between first-time parenthood and life satisfaction remains substantial ($\beta = 0.354$, $p < 0.01$). Likewise, the biological associations remain essentially unchanged, including lower neuroendocrine dysregulation ($\beta = -0.215$, $p < 0.01$), reduced metabolic dysregulation ($\beta = -0.177$, $p < 0.05$), slower Dunedin PoAm ($\beta = -0.023$, $p < 0.05$), and slower DunedinPACE ($\beta = -0.046$,

$p < 0.10$). These results indicate that the observed physiological associations cannot be explained simply by more favourable evaluations of other life domains.

Finally, we assessed whether acute physiological strain, proxied by self-reported sleep deprivation, modified the observed associations. Sleep deprivation is associated with lower life satisfaction ($\beta = -0.408$, $p < 0.01$), poorer health satisfaction ($\beta = -0.167$, $p < 0.01$), and modestly faster DunedinPACE ($\beta = 0.020$, $p < 0.10$), but shows little association with physiological dysregulation. Moreover, interaction terms between sleep deprivation and first-time parenthood are generally small and statistically insignificant across subjective, physiological, and epigenetic outcomes, with only isolated evidence for cardiovascular regulation ($\beta = -0.412$, $p < 0.05$).

Taken together, these falsification analyses indicate that the multidimensional profile associated with the transition to first-time parenthood is not reproduced by other common forms of adversity or by differences in domain-specific evaluations. Financial hardship is characterised by convergent deterioration across subjective, physiological, and biological ageing outcomes, whereas psychological distress primarily affects subjective wellbeing and sleep deprivation shows comparatively limited physiological associations. By contrast, first-time parenthood is associated with a distinct multidimensional profile in which different indicators of adaptation evolve differently. Although these analyses cannot identify the mechanisms underlying this divergence, they strengthen the interpretation that the observed pattern reflects a domain-specific profile of adaptation rather than a generic consequence of stress, socioeconomic disadvantage, or reporting behaviour.

Discussion

This study examined whether subjective wellbeing, physiological regulation, and biological ageing provide convergent accounts of adaptation during the transition to first-time parenthood. Across multiple empirical specifications, they showed only partial overlap. While first-time parenthood was associated with higher life satisfaction, it was not associated with changes in satisfaction with health. At the same time, it was associated with more favourable physiological regulation and slower biological ageing. These associations were remarkably consistent across

conventional regression and IPWRA analyses and were not reproduced by alternative forms of adversity, including financial hardship, psychological distress, sleep deprivation, or differences in domain-specific satisfaction.

We interpret these findings as evidence consistent with the *domain-specific adaptation hypothesis*. Rather than providing contradictory evidence, subjective evaluations and biological measures appear to capture different dimensions of adaptation. Life satisfaction, perceived health, physiological regulation, and biological ageing therefore provide complementary rather than interchangeable information about adjustment to sustained caregiving. More broadly, our contribution is not to identify a single biological mechanism linking parenthood to health, nor to establish the causal biological consequences of parenthood. Rather, we propose a testable conceptual framework in which adaptation appears to be multidimensional. If different indicators reflect partially distinct adaptive processes operating across functional domains and timescales, no single measure can provide a complete account of how individuals adjust to prolonged caregiving demands.

Our findings bridge two literatures that have rarely been connected empirically. Within the social-science literature on the "parenthood paradox" (Kohler and Mencarini, 2016), changes in wellbeing are generally interpreted as consequences of time scarcity, fatigue, and resource constraints (Stanca, 2012). Our findings are broadly consistent with this literature for subjective outcomes. Although first-time parents report higher overall life satisfaction, they do not report better health satisfaction despite exhibiting more favourable physiological profiles. This divergence suggests that conscious evaluations of health do not necessarily mirror underlying physiological regulation and that subjective experience alone may provide an incomplete account of adaptation to parenthood.

Our results also contribute to a growing biological literature documenting persistent adaptations associated with reproduction. Pregnancy and the postpartum period are accompanied by extensive brain remodelling and long-lasting neural plasticity (Hoekzema et al., 2017; Kim et al., 2010; Barba-Müller et al., 2019; Luders et al., 2022; Martínez-García et al., 2021; Rotondi et al., 2024; Straathof et al., 2026). Likewise, pregnancy induces coordinated endocrine, immune, and metabolic changes (Yu et al., 2025), while recent evidence suggests that repro-

ductive history may be associated with more favourable trajectories of brain and biological ageing later in life (Takeshita, 2024; Young et al., 2025; Ryan et al., 2024; Hukkanen et al., 2026; Sironi et al., 2020; Schneider et al., 2025). Complementing this literature, we show that within a population-based longitudinal cohort, first-time parenthood is associated with lower neuroendocrine and metabolic dysregulation together with slower DunedinPoAm and DunedinPACE. At the same time, our findings differ from studies reporting accelerated biological ageing following reproduction, including evidence that pregnancy is associated with faster epigenetic ageing among young women (Ryan et al., 2024). Several explanations may account for this difference. First, the outcomes examined here are measured around entry into parenthood rather than during pregnancy itself, and may therefore capture a broader period of physiological recalibration rather than acute reproductive costs. Second, DunedinPACE and DunedinPoAm quantify the pace of biological ageing rather than cumulative biological age, and may capture different dimensions of biological functioning than other ageing measures. Third, evolutionary trade-off frameworks emphasise average reproductive costs across the life course, whereas short- and medium-term adaptation to caregiving demands may generate compensatory regulatory responses. Although our findings do not resolve these competing interpretations, they suggest that reproductive costs and adaptive physiological responses need not be mutually exclusive.

The heterogeneity analyses further indicate that these associations are broadly shared across demographic groups. Interactions with respondents' age and gender provide little evidence that the observed patterns are concentrated within particular subpopulations. Analyses using the broader sample of all parents further suggest that physiological differences are strongest during the first years following childbirth, whereas biological ageing varies comparatively little according to the age of the oldest child. Together, these findings support the view that parenthood represents a dynamic life-course process rather than a static exposure, while indicating that the observed multidimensional pattern is not driven by a single demographic subgroup.

The falsification analyses provide an important benchmark for interpreting these findings. Financial hardship exhibits a markedly different profile, characterised by convergent deterioration across subjective wellbeing, physiological regulation, and biological ageing. Psycho-

logical distress primarily affects subjective wellbeing while showing comparatively limited associations with physiological regulation, and sleep deprivation likewise fails to reproduce the multidimensional profile associated with first-time parenthood. Moreover, controlling for satisfaction with income and leisure leaves the estimated parenthood associations essentially unchanged. Collectively, these analyses suggest that the observed response profile is unlikely to reflect generic stress, socioeconomic disadvantage, reporting behaviour, or more favourable evaluations of other life domains. Although they cannot identify the mechanisms underlying the observed associations, they strengthen the interpretation that first-time parenthood is characterised by a distinctive pattern of adaptation across domains.

Several limitations should be acknowledged. First, despite the transition-anchored design and the use of IPWRA to improve comparability between first-time parents and childless respondents, the analyses remain observational. Several forms of selection could therefore provide alternative explanations for the observed associations. Individuals who become parents may differ systematically from those who remain childless in ways that are not fully captured by survey data, including fertility-related health, partnership stability, family orientation, and other dimensions of long-term socioeconomic advantage. Such differences could contribute to variation in subjective and biological outcomes independently of the transition to parenthood itself. Although these concerns cannot be eliminated in an observational setting, several features of our design reduce their plausibility as complete explanations. The IPWRA analyses compare first-time parents with childless respondents using pre-parenthood characteristics measured before the nurse assessment, thereby improving comparability while preserving temporal ordering. In addition, respondents who later become parents are broadly similar to those who never become parents across most subjective, physiological, and epigenetic outcomes prior to the transition, providing limited evidence for substantial baseline biological selection. Nevertheless, residual confounding cannot be ruled out, and our findings should be interpreted as evidence consistent with domain-specific adaptation rather than as causal estimates of the biological consequences of parenthood.

Second, biomarkers and DNA methylation measures are observed only once during the nurse assessment, preventing the direct observation of within-individual biological trajectories.

Third, the composite physiological indices capture broad regulatory systems rather than specific biological pathways and should not be interpreted as clinical measures.

Finally, our theoretical framework emphasises adaptation to sustained caregiving demands, whereas our empirical measure captures entry into biological parenthood rather than caregiving itself. Although parenthood is strongly correlated with caregiving responsibilities, the UKHLS does not allow us to observe caregiving intensity, division of childcare responsibilities, parental involvement, non-residential parenting arrangements, or broader family support structures. Consequently, the observed associations may reflect a combination of caregiving processes and other dimensions of parenthood, including social identity, partnership dynamics, fertility selection, and changes in life-course expectations. Future research incorporating direct measures of caregiving behaviour will be important for distinguishing these mechanisms.

These findings have implications extending beyond the study of parenthood. Much research implicitly assumes that subjective wellbeing provides a sufficient summary of human adaptation to sustained demands. Our results suggest that this assumption deserves reconsideration. Subjective evaluations, physiological regulation, and biological ageing appear to capture related but non-identical aspects of adaptation, implying that reliance on any single indicator may provide only a partial picture of how humans respond to prolonged challenges. More generally, sustained social roles may generate domain-specific adaptive profiles that cannot be adequately understood through a single dimension of wellbeing.

Future research should examine whether similar forms of domain-specific adaptation emerge in other contexts characterised by prolonged behavioural demands, including chronic caregiving, long-term illness, military service, or other sustained social roles. Longitudinal studies incorporating repeated biological measurements will be particularly valuable for establishing how subjective and physiological domains co-evolve within individuals over time. Identifying when these domains converge, when they diverge, and the mechanisms governing their coordination represents an important direction for future research on human adaptation.

More broadly, our findings suggest that human adaptation cannot be adequately characterised by any single dimension of functioning. Subjective, physiological, and epigenetic indicators appear to represent complementary adaptive systems operating across different functional

domains and timescales. Understanding how these systems interact, when they diverge, and the mechanisms governing their coordination represents an important challenge for future research on human adaptation.

Data and Methods

Study Population

The data for this study come from Understanding Society, which is a longitudinal household panel survey representative of the UK population. Data collection started under the name of British Household Panel Study (BHPS), which took place from 1991-2008, and continued to be the UK Household Longitudinal Study (UKHLS), from 2009 and ongoing. Overall, this dataset allows researchers to access data on UK households and individuals for over 30 years. Data is available at https://ukdataservice.ac.uk/.

Understanding Society contains detailed individual socio-economic and demographic characteristics, along with participants' attitudes, beliefs, health and well-being. In the main analysis, we focus on two measures of well-being indicating respondents' satisfaction with their life and their health. We also use other subjective measures in complementary analyses such as satisfaction with leisure and income, financial hardship and sleep deprivation.

Importantly for this study, a Nurse Health Assessment has been conducted and participating respondents received a visit from a qualified nurse, who collected various bio-measures. Notably, participants only completed the nurse assessment once, either in Wave 2 (for UKHLS respondents) or in Wave 3 (for BHPS respondents). Hence, bio-measures are not recorded multiple times for the same individual. The nurse measures include blood pressure, weight, height, body fat, and blood samples from consenting participants. With the analysis of blood samples, biomarkers such as hormones, proteins, haemoglobin and cholesterol are available to researchers to measure the physiological status of individuals. In this study, we consider eleven biomarkers divided into five regulatory systems: neuroendocrine, immune, cardiovascular, metabolic and adiposity. The sum of these biomarkers forms the Allostatic Load, which captures the overall individuals' physiological dysregulation. Within this framework, allostatic load serves as a cumulative indicator of physiological regulation rather than a direct measure of experienced stress. Lower allostatic load among parents does not imply the absence of strain, but may reflect the net balance between acute caregiving demands and longer-run adaptive regulation. To determine the scores for each biomarker, we use clinically relevant cut off points, as

also done elsewhere (Rodriquez et al., 2019). Specifically, we generate three categories for each biomarker based on pre-established clinically relevant values: high-risk (1 point), moderate-risk (0.5 point) and low-risk (0 points). Each regulatory system is calculated summing the categories of relevant biomarkers, with higher scores indicating greater dysregulation and, therefore, poorer physiological status. The results remain qualitatively consistent when using alternative scoring methods, confirming that our findings are not driven by the specific method used to construct the biomarkers. A summary of all biomarkers forming each regulatory systems, and their clinical cut-off points, can be found in Table A1.

Additionally, DNA methylation profiles are generated from whole blood samples provided by 3,654 eligible individuals who consented to both blood collection and genetic analysis. Using the Illumina Methylation EPIC BeadChip, over 850,000 Methylation sites across the genome have been measured. After quality control and normalisation of data, ageing algorithms are computed to create a set of epigenetic clocks which are useful to investigate the biological aging process of individuals. In this paper, we use DunedinPOAM and DunedinPACE which are third-generation clocks, measuring the pace at which individuals biologically age compared to their life years. Epigenetic clocks have emerged as powerful biomarkers for aging, mortality and disease risk, offering a more nuanced perspective than chronological age. While the latter simply refers to the time that has passed from birth to a given date, biological age reflects the actual physiological status of our cells. This provides a comprehensive, multisystemic view of an individual's health, unlike standard biomarkers that often only capture isolated biological snapshots.

Descriptive statistics of the outcome variables can be found in Table A3.

Analytical Sample

Overall, around 20,500 respondents undertook the nurse assessment either in Wave 2 or in Wave 3. Out of this sample, roughly 13,500 individuals gave blood samples and DNA methylation was quantified for about 3,500 individuals. Our analysis includes adult individuals of working age, from 16 to 65 years old. Given that the aim of this paper is to examine how parenthood affects individual outcomes, this choice aligns with the fertility cycle. Additionally, only considering

working-age individuals helps in avoiding that other factors, such as statutory retirement age, could directly affect the outcome of interest. Analytical survey weights, provided directly from the data provider, are used to ensure the representativeness of the sample.

The main explanatory variable for our study is parenthood. This information is derived from a question asking respondents how many biological children under the age of 16 are currently living in the household. Since the dataset is longitudinal, we are able to gather information on the history of parenthood. We consider all survey waves in which respondents are present, before and after the nurse assessment, so that we can assert their parenthood status from the first wave in which they appear in the survey. In this way, we can identify the exact timing in which respondents become parents within the observed period, as well as if they are never-parents or always-parents (i.e., entering the survey as a parent).

Statistical Methods

We examined cross-sectionally how being a first-time parent at the time of the nurse assessment is associated with a series of outcomes (e.g., subjective well-being, perceived health, physiological regulation, and biological ageing), compared to childless individuals - comprising both never parents and not-yet-parents. We exclude people who are childless at the nurse assessment, but then become parents within the next two survey waves. This is to avoid that their outcomes are shaped by the expectation of becoming parents. Results are robust to alternative sample selections.

We identify first-time parents as respondents who entered parenthood for the first time between the wave preceding the nurse assessment and the nurse assessment itself. This timing is important because our inverse probability weighted regression adjustment (IPWRA) approach compares first-time parents with non-parents using pre-parenthood characteristics measured at $(t-1)$, which in our setting corresponds to the survey wave immediately preceding the nurse assessment. This preserves the temporal ordering between covariates and outcomes, improves comparability between groups, and strengthens the identification strategy. At the same time, although the design anchors observations around the transition to first-time parenthood, the biological outcomes are observed only once. Consequently, we cannot directly estimate within-

individual biological changes across the transition itself, and our analyses should therefore be interpreted as comparing individuals at a common stage of the transition rather than as estimating biological trajectories.

To further investigate the relationship between parenthood and parents' outcomes, we conduct additional heterogeneity analyses across respondents' demographic characteristics (e.g., age and gender) and across stages of the parenting life course (e.g., age of oldest child).

We estimate linear regressions via ordinary least squares (OLS), with standard errors clustered at the individual level and including population weights to ensure representativeness. These regressions take the baseline form of:

$$Y_{it} = \alpha_0 + \alpha_1 Parent_{it} + \alpha_2 X_{it} + \epsilon_{it} \tag{1}$$

where Y_{it} is alternatively one outcome of the set of: subjective well-being measures, physiological outcomes, and biological age. Each outcome is measured for individual i at the nurse assessment wave (time t). $Parent_{it}$ is the parenthood status for each respondent at time t, namely, if they were first-time parents at the nurse assessment or not. A set of individual covariates measured at time t is included in all specifications to isolate the association of first-time parenthood by holding relevant factors constant as they could potentially directly influence the outcomes of interest. The covariates included are: individuals' age, age squared, gender, ethnicity, big five personality traits (e.g., agreeableness, conscientiousness, extraversion, neuroticism and openness), education, long-standing illness, equivalized household income (in log), marital status, job status and perceived financial situation. We also always control for blood-specific covariates, indicating if respondents have drunk alcohol, done vigorous exercise, eaten or smoked in the 30 minutes prior to the blood collection. It is important to control for these blood-specific variables as the measurement of biomarkers could be directly affected by those behaviours.

In the specifications where epigenetic clocks are considered as outcomes, we further include a set of variables related to cell-type composition. These methylation-based controls are: B cells, CD4+ T cells, CD8+ T cells, Granulocytes, Monocytes, and NK cells. When conducting research using blood-based DNA methylation data, it is important to control for these covariates as the estimates can be confounded by the cell-type composition.

Descriptive statistics of all covariates can be found in Table A2.

Data Availability Statement

The data that support the findings of this study are available from the UK Data Service (`https://ukdataservice.ac.uk/`) but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly available. Data are however available from the authors upon reasonable request and with permission of the UK Data Service.

Ethics approval

This study used secondary anonymised data from the UK Household Longitudinal Study (UKHLS). All methods were carried out in accordance with relevant guidelines and regulations. Ethical approval for UKHLS data collection, including biomarker and genetic data, was obtained by the UKHLS study team from the appropriate National Health Service Research Ethics Committees. Written informed consent was obtained from all participants for participation and for the collection of biomarker and genetic information. The present study involved secondary analysis of fully anonymised data and did not require additional ethical approval by the authors' institutions.

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Appendix

Table A1: Clinically relevant thresholds for biomarkers

Regulatory systems	Biomarkers	High-Risk (1); Moderate-Risk (0.5); Low-Risk (0)	Clinical cut-off points
Cardiovascular	Systolic blood pressure	High	≥150 mmHg
		Moderate	120 to 149 mmHg
		Low	<120 mmHg
	Diastolic blood pressure	High	≥90 mmHg
		Moderate	80 to 89 mmHg
		Low	<80 mmHg
Metabolic	Total/HDL cholesterol ratio	High	≥6
		Moderate	5 to <6
		Low	<5
	Glycated hemoglobin	High	≥48 mmol/mol
		Moderate	39 to 47 mmol/mol
		Low	<39 mmol/mol
	Creatinine clearance	High	<30 mL/min
		Moderate	30 to <60 mL/min
		Low	≥60 mL/min
	Triglycerides	High	≥10 mmol/L
		Moderate	2 to 9.9 mmol/L
		Low	<2 mmol/L
Immune	C-reactive protein	High	>3 mg/L
		Moderate	1 to 3 mg/L
		Low	<1 mg/L
	Clauss fibrinogen	High	≤1.4 g/L
		Moderate	1.5 to 1.9 g/L
		Low	≥2 g/L
Neuroendocrine	DHEAS (Dehydroepiandrosterone Sulfate)	High (Female & Age)	Age and gender specific. See details in Notes
		Moderate (Female & Age)	
		Low (Female & Age)	
		High (Male & Age)	
		Moderate (Male & Age)	
		Low (Male & Age)	
Adiposity	waist to height ratio	High	≥0.6
		Moderate	0.5 to <0.6
		Low	<0.5
	BMI	High	≥30 or <18
		Moderate	25 to 29.9
		Low	18 to 24.9

Note: (cholesterol ratio): total cholesterol/HDL cholesterol. Total cholesterol ≥240 mg/dL, 200 to 239 mg/dL, and <200 mg/dL; HDL cholesterol <40 mg/dL, 40 to 59 mg/dL, and ≥60 mg/dL. Allostatic Load is the sum of all biomakrers. DHEAS : High-Female >18 umol/L (age ≤ 30); >15 umol/L (age 31 to 40); >13 umol/L (age 41 to 50); >10 umol/L (age 51 to 60); >8 umol/L (age > 60) Moderate-Female >11.5 to 18 umol/L (age ≤ 30); >9.5 to 15 umol/L (age 31 to 40); >7.5 to 13 umol/L (age 41 to 50); >5.5 to 10 umol/L (age 51 to 60); >4.5 to 8 umol/L (age > 60) Low-Female ≤11.5 umol/L (age ≤ 30); ≤9.5 umol/L (age 31 to 40); ≤7.5 umol/L (age 41 to 50); ≤5.5 umol/L (age 51 to 60); ≤4.5 umol/L (age > 60) High-Male >20 umol/L (age≤ 30); >18 umol/L (age 31 to 40); >17 umol/L (age 41 to 50); >14 umol/L (age 51 to 60); >12 umol/L (age > 60) Moderate-Male >14.5 to 20 umol/L (age ≤ 30); >13 to 18 umol/L (age 31 to 40); >12 to 17 umol/L (age 41 to 50); >8.5 to 14 umol/L (age 51 to 60); >7 to 12 umol/L (age > 60) Low-Male ≤14.5 umol/L (age ≤ 30); ≤13 umol/L (age 31 to 40); ≤12 umol/L (age 41 to 50); ≤8.5 umol/L (age 51 to 60); ≤7 umol/L (age > 60)

Table A2: Descriptive statistics of covariates at time (t)

	Mean	SD	Min	Max	N
Baseline covariates					
Parenthood	0.03	0.16	0	1	4,249
At least A level	0.59	0.49	0	1	4,249
Female	0.55	0.50	0	1	4,249
White	0.97	0.18	0	1	4,249
Age	46.69	14.62	16	64	4,249
Age2	2394.15	1223.16	256	4096	4,249
Married/couple	0.61	0.49	0	1	4,249
Widowed	0.03	0.16	0	1	4,249
Divorced	0.11	0.31	0	1	4,249
Never married	0.25	0.43	0	1	4,249
Employed	0.66	0.47	0	1	4,249
Unemployed	0.05	0.22	0	1	4,249
Retired	0.14	0.35	0	1	4,249
Leave/house	0.03	0.18	0	1	4,249
Other	0.11	0.31	0	1	4,249
Eq. hh income	7.51	0.72	0.51	10.09	4,249
Agreeableness	5.61	1.01	1	7	4,249
Conscientiousness	5.51	1.08	1	7	4,249
Extraversion	4.58	1.31	1	7	4,249
Neuroticism	3.63	1.46	1	7	4,249
openness	4.62	1.28	1	7	4,249
Long-term illness	0.36	0.48	0	1	4,249
Financial situation	2.12	1.02	1	5	4,249
Blood-specific covariates					
Drunk alcohol	0.01	0.08	0	1	4,249
Exercised	0.01	0.11	0	1	4,249
Eaten	0.10	0.30	0	1	4,249
Smoked	0.07	0.26	0	1	4,249
Cell-type covariates					
B cells	0.05	0.03	0	0.23	1,128
CD4+ T cells	0.14	0.07	0	0.37	1,128
CD8+ T cells	0.16	0.06	0	0.44	1,128
Granulocytes	0.60	0.09	0.29	0.86	1,128
Monocytes	0.04	0.03	0	0.14	1,128
NK cells	0.01	0.02	0	0.18	1,128

Note: The big 5 personality traits of Agreeableness, Conscientiousness, Extraversion, Neuroticism, and openness are composite scores with higher values indicating a stronger presence of that trait. Financial situation represents the perceived financial situation of the respondents, going from 1 (Living comfortably) to 5 (Finding it very difficult).

Table A3: Descriptive statistics of outcomes at time (t)

	Mean	SD	Min	Max	N
Health & Well-being					
LifeSat	5.20	1.47	1	7	4,249
HealthSat	4.85	1.70	1	7	4,249
IncomeSat	4.60	1.72	1	7	4,242
LeisureSat	4.79	1.66	1	7	4,239
Biomarkers					
Neuroendocrine	0.04	0.15	0	1	4,249
Immune	0.46	0.39	0	2	4,249
Cardiovascular	0.52	0.53	0	2	4,249
Metabolic	0.49	0.58	0	3	4,249
Adiposity	0.95	0.72	0	2	4,249
Allostatic	2.53	1.57	0	8.50	4,249
Epigenetic Clocks					
Dunedinpoam	1.01	0.08	0.83	1.28	1,128
Dunedinpace	1.05	0.14	0.64	1.68	1,128

Note: In the statistical analysis, the health and well-being outcomes are standardized for comparison purpuses. Satisfaction variables related to overall life, health, income and leisure time are measured on a scale from 0 (completely dissatisfied) to 7 (completely satisfied). Higher values of biomarkers indicate worse physiological health. Epigenetic clocks represents the rate of aging, with values centered at 1.

Table A4: Linear regressions: Subjective outcomes (STD)

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.225**	0.095
	(0.110)	(0.109)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A5: Linear regressions: Biological outcomes (STD)

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.228***	0.022	-0.076	-0.176**	0.070	-0.064
	(0.072)	(0.126)	(0.120)	(0.083)	(0.120)	(0.105)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4073	4073	4073	4073	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A6: Linear regressions: Pace of ageing clocks

	Dunedin poam	Dunedin pace
First time parent (t)	-0.023**	-0.046*
	(0.011)	(0.027)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1108	1108

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A7: Linear regressions: Subjective outcomes (STD) - IPWRA

	Life Satisfaction	Health Satisfaction
ATET	0.276**	0.032
	(0.116)	(0.122)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	3808	3808

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t-1: at least A level, equivalized household income (log), financial situation, marital status, disability, employed. Controls at time t: white, female, age range, big 5 personality traits. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A8: Linear regressions: Biological outcomes (STD) - IPWRA

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
ATET	-0.177**	0.097	-0.030	-0.138	0.126	0.020
	(0.071)	(0.133)	(0.129)	(0.085)	(0.144)	(0.121)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	3808	3808	3808	3808	3808	3808

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t-1: at least A level, equivalized household income (log), financial situation, marital status, disability, employed. Controls at time t: white, female, age range, big 5 personality traits. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A9: Linear regressions: Pace of ageing clocks - IPWRA

	Dunedin poam	Dunedin pace
ATET	-0.025*	-0.045*
	(0.013)	(0.026)
Baseline controls	✓	✓
Methylation-based controls	✓	✓
Observations	1108	1108

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t-1: at least A level, equivalized household income (log), financial situation, marital status, disability, employed. Controls at time t: white, female, age range, big 5 personality traits. Blood-based controls not included due to nonconvergence. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A10: Selection checks: assessing timing of the transition to parenthood

	Panel A	**Panel B**
Outcome	Future parents vs Never parents	First-time parents vs Future parents
Subjective outcomes		
Life satisfaction	0.014	0.110
	(0.060)	(0.129)
Health satisfaction	0.015	-0.068
	(0.061)	(0.132)
Physiological regulation		
Neuroendocrine	0.009	-0.110
	(0.076)	(0.101)
Immune	0.043	-0.103
	(0.067)	(0.147)
Cardiovascular	-0.025	-0.047
	(0.048)	(0.135)
Metabolic	0.111**	-0.329***
	(0.054)	(0.127)
Adiposity	0.037	0.050
	(0.063)	(0.133)
Allostatic load	0.049	-0.125
	(0.054)	(0.134)
Biological ageing		
Dunedin PoAm	0.003	-0.012
	(0.007)	(0.013)
DunedinPACE	-0.007	-0.028
	(0.013)	(0.028)
Controls	Yes	Yes
Observations	4,127	538
Epigenetic clocks	1,127	164

Note: Panel A compares respondents who remained childless throughout the observed study period (never parents) with respondents who were childless at the nurse assessment but entered parenthood after the nurse assessment (future parents) up to the subsequent 11 survey waves. Panel B compares respondents who transition into first-time parenthood at the nurse assessment (first-time parents) with the same group of future parents. All coefficients are estimated from separate linear regression. Standard errors (clustered at the individual level) are reported in parentheses. All models includinge the same covariates used in the main analyses. Sample sizes for the biological epigenetic ageing models are reported separately because DNA methylation data are available only for a subsample.

Table A11: Linear regressions: Subjective outcomes (STD) by gender

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.124	-0.042
	(0.209)	(0.192)
Female	0.068*	0.005
	(0.041)	(0.041)
First time parent (t) × Female	0.384*	0.306
	(0.229)	(0.228)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A12: Linear regressions: Biological outcomes (STD) by gender

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.203*	0.144	-0.057	-0.266**	0.251	0.037
	(0.110)	(0.200)	(0.160)	(0.123)	(0.178)	(0.152)
Female	-0.059	0.206***	-0.405***	-0.168***	-0.120***	-0.193***
	(0.042)	(0.039)	(0.034)	(0.035)	(0.038)	(0.034)
First time parent (t) × Female	-0.057	-0.121	0.012	0.246*	-0.003	0.033
	(0.116)	(0.242)	(0.227)	(0.139)	(0.237)	(0.204)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4073	4073	4073	4073	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A13: Linear regressions: Pace of ageing clocks by gender

	Dunedin poam	Dunedin pace
First time parent (t)	-0.028**	-0.079**
	(0.014)	(0.040)
Female	-0.016***	-0.011
	(0.005)	(0.009)
First time parent (t) $\times$ Female	0.029	0.087*
	(0.018)	(0.048)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1108	1108

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A14: Linear regressions: Subjective outcomes (STD) by age

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.191	-0.059
	(0.230)	(0.283)
Age 26-35	-0.278***	-0.211***
	(0.076)	(0.079)
Age 36-45	-0.328***	-0.127*
	(0.074)	(0.075)
Age 46-55	-0.379***	-0.188***
	(0.070)	(0.069)
Age 56-65	-0.248***	-0.151**
	(0.071)	(0.071)
First time parent (t) × Age 26-35	0.180	0.354
	(0.267)	(0.300)
First time parent (t) × Age 36-45	-0.022	-0.124
	(0.267)	(0.351)
First time parent (t) × Age 46-55	-1.364***	0.077
	(0.408)	(0.582)
First time parent (t) × Age 56-65	0.344	0.277
	(0.251)	(0.303)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A15: Linear regressions: Biological outcomes (STD) by age

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.466***	-0.157	-0.143	-0.202	0.019	-0.178
	(0.100)	(0.223)	(0.138)	(0.167)	(0.225)	(0.189)
Age 26-35	-0.094	0.024	0.147**	0.242***	0.053	0.147**
	(0.095)	(0.088)	(0.065)	(0.071)	(0.082)	(0.071)
Age 36-45	-0.074	-0.014	0.420***	0.361***	0.441***	0.466***
	(0.090)	(0.082)	(0.072)	(0.070)	(0.082)	(0.073)
Age 46-55	-0.107	0.017	0.614***	0.530***	0.600***	0.683***
	(0.081)	(0.075)	(0.062)	(0.062)	(0.074)	(0.064)
Age 56-65	-0.072	0.077	0.706***	0.559***	0.683***	0.806***
	(0.082)	(0.078)	(0.067)	(0.066)	(0.076)	(0.067)
First time parent (t) × Age 26-35	0.228*	0.184	-0.041	0.010	0.099	0.105
	(0.125)	(0.263)	(0.162)	(0.189)	(0.267)	(0.222)
First time parent (t) × Age 36-45	0.396**	0.127	0.405	0.108	0.076	0.243
	(0.192)	(0.376)	(0.413)	(0.214)	(0.367)	(0.323)
First time parent (t) × Age 46-55	0.173	0.826**	0.289	-0.535*	0.065	0.103
	(0.137)	(0.359)	(1.028)	(0.288)	(0.680)	(0.479)
First time parent (t) × Age 56-65	0.129	0.163	0.094	-0.798***	1.248***	0.906***
	(0.163)	(0.247)	(0.176)	(0.196)	(0.255)	(0.220)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4073	4073	4073	4073	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A16: Linear regressions: Pace of ageing clocks by age

	Dunedin poam	Dunedin pace
First time parent (t)	-0.006	0.019
	(0.026)	(0.056)
Age 26-35	0.008	0.022
	(0.009)	(0.016)
Age 36-45	0.032***	0.068***
	(0.011)	(0.019)
Age 46-55	0.028***	0.089***
	(0.009)	(0.016)
Age 56-65	0.020**	0.105***
	(0.009)	(0.017)
First time parent (t) × Age 26-35	-0.024	-0.079
	(0.028)	(0.064)
First time parent (t) × Age 36-45	-0.009	-0.069
	(0.034)	(0.070)
First time parent (t) × Age 46-55	0.023	-0.021
	(0.028)	(0.061)
First time parent (t) × Age 56-65	0.000	0.000
	(.)	(.)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1108	1108

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A17: Linear regressions: Subjective outcomes (STD) by age of oldest child

	Life Satisfaction	Health Satisfaction
Oldest child aged 0-2	0.205***	0.067
	(0.059)	(0.066)
Oldest child aged 3-4	0.032	-0.024
	(0.074)	(0.068)
Oldest child aged 5-11	0.031	-0.005
	(0.041)	(0.041)
Oldest child aged 12-15	0.017	-0.019
	(0.042)	(0.040)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	6699	6699

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A18: Linear regressions: Biological outcomes (STD) by age of oldest child

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
Oldest child aged 0-2	-0.061	-0.041	-0.159***	-0.019	0.106	-0.034
	(0.086)	(0.071)	(0.055)	(0.059)	(0.072)	(0.062)
Oldest child aged 3-4	-0.011	-0.175**	-0.087	-0.157***	-0.081	-0.183***
	(0.097)	(0.084)	(0.064)	(0.057)	(0.079)	(0.064)
Oldest child aged 5-11	-0.037	-0.072	-0.067	-0.090**	0.054	-0.066
	(0.046)	(0.046)	(0.041)	(0.042)	(0.044)	(0.040)
Oldest child aged 12-15	-0.001	-0.079*	-0.024	-0.048	0.047	-0.026
	(0.049)	(0.045)	(0.043)	(0.043)	(0.042)	(0.041)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	6699	6699	6699	6699	6699	6699

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A19: Linear regressions: Pace of ageing clocks by age of oldest child

	Dunedin poam	Dunedin pace
Oldest child aged 0-2	0.004	0.007
	(0.007)	(0.017)
Oldest child aged 3-4	0.015	0.017
	(0.009)	(0.018)
Oldest child aged 5-11	-0.004	-0.003
	(0.005)	(0.010)
Oldest child aged 12-15	-0.008	-0.015
	(0.005)	(0.009)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1900	1900

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A20: Linear regressions: Subjective outcomes (STD) by financial distress

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.246**	0.104
	(0.105)	(0.113)
Financial distress	-0.667***	-0.339***
	(0.072)	(0.063)
First time parent (t) $\times$ Financial distress	-0.487	-0.234
	(0.674)	(0.418)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A21: Linear regressions: Biological outcomes (STD) by financial distress

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.220***	-0.038	-0.051	-0.151*	0.074	-0.057
	(0.076)	(0.117)	(0.127)	(0.086)	(0.124)	(0.111)
Financial distress	0.011	0.074	0.000	0.241***	0.152**	0.187***
	(0.073)	(0.066)	(0.059)	(0.065)	(0.068)	(0.060)
First time parent (t) × Financial distress	-0.090	0.844	-0.352	-0.266	-0.032	-0.059
	(0.125)	(0.757)	(0.244)	(0.245)	(0.525)	(0.335)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4073	4073	4073	4073	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A22: Linear regressions: Pace of ageing clocks by financial distress

	Dunedin poam	Dunedin pace
First time parent (t)	-0.015	-0.041
	(0.012)	(0.030)
Financial distress	0.029***	0.032**
	(0.009)	(0.014)
First time parent (t) × Financial distress	-0.058**	-0.031
	(0.023)	(0.054)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1108	1108

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A23: Linear regressions: Subjective outcomes (STD) by marital status

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.197*	0.116
	(0.116)	(0.114)
Widowed	-0.188*	0.097
	(0.105)	(0.106)
Divorce/Separated	-0.290***	-0.090*
	(0.055)	(0.050)
Never married	-0.186***	-0.039
	(0.050)	(0.052)
First time parent (t) × Widowed	0.000	0.000
	(.)	(.)
First time parent (t) × Divorce/Separated	0.262*	-0.419
	(0.158)	(0.320)
First time parent (t) × Never married	0.353*	-0.222
	(0.200)	(0.296)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A24: Linear regressions: Biological outcomes (STD) by marital status

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.235***	0.083	-0.062	-0.155*	0.114	-0.025
	(0.068)	(0.132)	(0.130)	(0.086)	(0.127)	(0.111)
Widowed	-0.059	0.012	0.162	0.092	0.168*	0.190*
	(0.094)	(0.107)	(0.118)	(0.117)	(0.097)	(0.104)
Divorce/Separated	0.034	0.084	0.034	0.057	-0.103*	0.024
	(0.064)	(0.057)	(0.060)	(0.059)	(0.055)	(0.055)
Never married	0.112**	0.011	0.057	0.008	-0.073	0.007
	(0.054)	(0.059)	(0.049)	(0.051)	(0.055)	(0.049)
First time parent (t) × Widowed	0.000	0.000	0.000	0.000	0.000	0.000
	(.)	(.)	(.)	(.)	(.)	(.)
First time parent (t) × Divorce/Separated	2.035*	-0.901*	-0.134	-1.022***	0.269	-0.131
	(1.168)	(0.491)	(0.166)	(0.140)	(0.505)	(0.513)
First time parent (t) × Never married	-0.319***	-0.695**	-0.173	-0.080	-0.682***	-0.524**
	(0.115)	(0.277)	(0.237)	(0.208)	(0.240)	(0.266)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4073	4073	4073	4073	4073	4073

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A25: Linear regressions: Pace of ageing clocks by marital status

	Dunedin poam	Dunedin pace
First time parent (t)	-0.028**	-0.058**
	(0.012)	(0.028)
Widowed	0.005	0.035
	(0.016)	(0.026)
Divorce/Separated	0.007	0.024*
	(0.008)	(0.014)
Never married	-0.005	0.010
	(0.006)	(0.012)
First time parent (t) × Widowed	0.000	0.000
	(.)	(.)
First time parent (t) × Divorce/Separated	0.000	0.000
	(.)	(.)
First time parent (t) × Never married	0.035	0.099
	(0.033)	(0.067)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1108	1108

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A26: Linear regressions: Subjective outcomes (STD) by mental distress

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.165	0.042
	(0.107)	(0.133)
Mental distress	-0.599***	-0.310***
	(0.038)	(0.040)
First time parent (t) × Mental distress	0.112	0.115
	(0.230)	(0.199)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4052	4052

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A27: Linear regressions: Biological outcomes (STD) by mental distress

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.246***	-0.015	-0.069	-0.080	0.051	-0.047
	(0.084)	(0.135)	(0.149)	(0.095)	(0.149)	(0.135)
Mental distress	-0.028	0.045	0.010	0.056	0.009	0.044
	(0.046)	(0.043)	(0.036)	(0.037)	(0.041)	(0.037)
First time parent (t) × Mental distress	0.040	0.127	-0.013	-0.281**	0.076	-0.030
	(0.115)	(0.258)	(0.233)	(0.139)	(0.232)	(0.191)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4052	4052	4052	4052	4052	4052

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A28: Linear regressions: Pace of ageing clocks by mental distress

	Dunedin poam	Dunedin pace
First time parent (t)	-0.018	-0.037
	(0.014)	(0.033)
Mental distress	0.005	0.013
	(0.005)	(0.010)
First time parent (t) × Mental distress	-0.018	-0.027
	(0.018)	(0.046)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1106	1106

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A29: Linear regressions: Subjective outcomes (STD) with domain-specific satisfaction controls

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.354***	0.188*
	(0.089)	(0.102)
Satisfaction with income	0.394***	0.351***
	(0.023)	(0.024)
Satisfaction with leisure	0.335***	0.219***
	(0.019)	(0.020)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4058	4058

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A30: Linear regressions: Biological outcomes (STD) with domain-specific satisfaction controls

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.215***	0.025	-0.078	-0.177**	0.060	-0.068
	(0.072)	(0.126)	(0.119)	(0.084)	(0.119)	(0.105)
Satisfaction with income	0.056**	0.008	0.026	0.008	-0.027	0.011
	(0.026)	(0.025)	(0.021)	(0.021)	(0.024)	(0.021)
Satisfaction with leisure	-0.043*	-0.007	-0.040**	-0.008	-0.033	-0.042**
	(0.024)	(0.022)	(0.019)	(0.019)	(0.020)	(0.018)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4058	4058	4058	4058	4058	4058

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A31: Linear regressions: Pace of ageing clocks with domain-specific satisfaction controls

	Dunedin poam	Dunedin pace
First time parent (t)	-0.023**	-0.046*
	(0.011)	(0.027)
Satisfaction with income	-0.002	-0.003
	(0.003)	(0.006)
Satisfaction with leisure	-0.001	0.003
	(0.003)	(0.005)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1103	1103

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A32: Linear regressions: Subjective outcomes (STD) by sleep deprivation

	Life Satisfaction	Health Satisfaction
First time parent (t)	0.209*	0.077
	(0.107)	(0.120)
Sleep loss	-0.408***	-0.167***
	(0.049)	(0.048)
First time parent (t) × Sleep loss	-0.079	0.041
	(0.345)	(0.207)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Observations	4067	4067

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A33: Linear regressions: Biological outcomes (STD) by sleep deprivation

	Neuroendocrine	Immune	Cardiovascular	Metabolic	Adiposity	Allostatic
First time parent (t)	-0.224***	-0.063	-0.014	-0.200**	0.025	-0.096
	(0.080)	(0.115)	(0.136)	(0.087)	(0.128)	(0.116)
Sleep loss	-0.034	0.051	0.045	0.017	-0.017	0.034
	(0.049)	(0.052)	(0.043)	(0.047)	(0.049)	(0.043)
First time parent (t) × Sleep loss	-0.050	0.606	-0.412**	0.181	0.305	0.237
	(0.092)	(0.434)	(0.178)	(0.177)	(0.312)	(0.212)
Baseline controls	✓	✓	✓	✓	✓	✓
Blood-based controls	✓	✓	✓	✓	✓	✓
Observations	4067	4067	4067	4067	4067	4067

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table A34: Linear regressions: Pace of ageing clocks by sleep deprivation

	Dunedin poam	Dunedin pace
First time parent (t)	-0.025*	-0.046
	(0.013)	(0.031)
Sleep loss	0.001	0.020*
	(0.006)	(0.011)
First time parent (t) $\times$ Sleep loss	0.008	0.007
	(0.019)	(0.039)
Baseline controls	✓	✓
Blood-based controls	✓	✓
Methylation-based controls	✓	✓
Observations	1107	1107

Note: Standard errors in parentheses, clustered at individual level. Balanced sample of respondents aged 16-65 y/o with valid subjective and biological outcomes. Controls at time t: at least A level, white, female, marital status, job status, age, age2, equivalized household income (log), big 5 personality traits, disability and financial situation. Blood-based controls measured at time t: smoked/drunk alcohol/eaten/exercised in past 30 minutes. EML is computed on white respondents of European descendents. Methylation-based controls measured at time t: bcell cd4t cd8t gran mono nk. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

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