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Chronic Stress, Reward Processing, and Emotional Eating as Neurobehavioral Predictors of Incident Obesity: A Retrospective Cohort Study

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Abstract

Background: Chronic stress, altered reward processing, and emotional eating may jointly promote positive energy balance, but their longitudinal contributions to the transition from non-obese weight status to obesity are incompletely characterized.

Methods: We conducted a retrospective cohort analysis of 750 adults aged 18-70 years without obesity at baseline. BMI was calculated from recorded height and weight, and incident obesity was defined as the first follow-up BMI ≥ 30 kg/m². Participants contributed 2,609.8 person-years (median follow-up, 43 months). The primary analysis used person-month complementary log-log regression with annual baseline-hazard indicators and participant-clustered robust standard errors. Baseline stress, reward-processing, and emotional-eating scores were standardized; models adjusted for age, sex, baseline BMI, and family history of obesity. An exploratory standardized neurobehavioral index summarized the three exposure scores.

Results: Ninety-eight participants (13.1%) developed obesity, an incidence rate of 37.6 per 1,000 person-years. In unadjusted models, stress (HR 1.45 per SD, 95% CI 1.20-1.75) and emotional eating (HR 1.41, 95% CI 1.17-1.70) predicted incident obesity, whereas reward processing did not (HR 1.14, 95% CI 0.95-1.38). In the complete-case multivariable model (n=619; 81 events), associations attenuated for stress (adjusted HR 1.21, 95% CI 0.92-1.59), reward processing (0.98, 0.74-1.30), and emotional eating (1.26, 0.96-1.67), while baseline BMI remained strongly predictive (4.63 per SD, 3.10-6.92). The exploratory neurobehavioral index was independently associated with incident obesity (adjusted HR 1.42 per SD, 1.13-1.79; p=0.003); crude incidence increased from 26.1 to 52.4 per 1,000 person-years from the lowest to highest tertile.

Conclusions: Individual neurobehavioral scores were attenuated after adjustment for baseline adiposity, whereas their combined standardized profile retained prognostic information. These findings support integrated assessment of stress-related, reward-related, and emotional-eating processes while emphasizing baseline BMI as the dominant near-term predictor of incident obesity.

Keywords: obesity; chronic stress; reward processing; emotional eating; neurobehavioral risk; retrospective cohort

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1. Introduction

Obesity has become one of the most consequential chronic-disease challenges worldwide. Recent pooled and Global Burden of Disease analyses document continued growth in adult overweight and obesity and project substantial further increases through 2050 [1,2]. Contemporary clinical frameworks therefore emphasize obesity as a heterogeneous, relapsing disease shaped by

biological, behavioral, environmental, and psychosocial determinants rather than by energy balance in isolation [3]. Identifying markers that precede the transition to obesity may improve the timing and targeting of prevention [4].

Stress-related neuroendocrine activity is one plausible pathway. Long-term glucocorticoid exposure has been associated with subsequent increases in BMI and waist

circumference [5], and glucocorticoids can influence appetite-regulating hormones and reward-related eating [6]. In people with obesity, perceived stress and hair cortisone have also been linked with persistent hedonic eating tendencies [7]. Daily-life studies further suggest that stress, cortisol reactivity, and eating style interact to influence snack intake and eating behavior [8,9]. These observations support stress as a potential upstream driver, but they do not establish whether stress contributes independently to new-onset obesity once baseline adiposity is considered.

Eating behavior and reward processing may represent overlapping components of the same vulnerability. A systematic review found that several eating-behavior traits are associated with energy intake and BMI [10]. Recent meta-analytic and longitudinal work has linked negative emotional eating with higher BMI and demonstrated meaningful heterogeneity and temporal instability in emotional-eating patterns [11-14]. Reward-related constructs are similarly complex: wanting, liking, dyscontrol, hedonic hunger, and neural food-value representations show associations with weight status, but effect sizes and directions vary across paradigms [15-18]. Real-world dieting studies also indicate that appetite, affect, and momentary temptations operate dynamically rather than as isolated traits [19].

We therefore examined whether baseline chronic stress, reward-processing, and emotional-eating scores predicted incident obesity in a non-obese retrospective cohort. We hypothesized that higher recorded scores would be associated with greater obesity risk and explored whether a combined standardized neurobehavioral index captured risk not evident from any single measure.

2. Methods

2.1 Study design and cohort

This retrospective cohort analysis included 750 adults aged 18-70 years without obesity at baseline (BMI <30 kg/m²) and with 6-60 months of observed follow-up. Longitudinal records comprised 2,969 follow-up weight observations. Participants were followed until incident obesity, the end of recorded follow-up, or the last available observation. The analysis used de-identified participant-level data.

2.2 Outcome reconstruction

BMI was calculated at baseline and at each follow-up assessment as weight (kg) divided by height squared (m²). Incident obesity was defined as the first recorded follow-up BMI ≥30 kg/m². Participants who did not develop obesity were censored at the end of recorded follow-up or at their last available observation. Person-time was calculated as observed follow-up months divided by 12.

2.3 Exposures and covariates

Baseline neurobehavioral exposures were the study-recorded stress, reward-processing, and emotional-eating scores. To permit direct comparison across measures, continuous exposure scores were standardized to z scores and effect estimates are reported per 1-SD increase. Available covariates included age, sex, socioeconomic group, smoking, family history of obesity, shift work, weight-gain medication, sleep duration, physical activity, depressive symptoms, and baseline BMI. The primary adjustment set included age, sex, baseline BMI, and family history of obesity to limit model overparameterization relative to the number of events.

2.4 Missing data

Missingness was 4.7% for stress, 8.0% for reward processing, and 5.9% for emotional eating; 619 participants had complete data on all three core exposures and primary covariates. These 619 participants contributed 81 events. No imputation was performed because the analytic file did not contain sufficient information to justify a missingness model. Event proportions were similar among included and excluded participants (13.1% vs 13.0%).

2.5 Statistical analysis

Continuous variables are summarized as mean (SD) and categorical variables as n (%). Crude comparisons used Welch t tests or chi-square/Fisher exact tests as appropriate. The crude incidence rate was calculated per 1,000 person-years. Time-to-event associations were modeled in person-month format using a complementary log-log link, four indicators for follow-up years 2-5 to allow a flexible baseline hazard, and participant-clustered robust standard errors. Continuous predictors were standardized within each analytic sample. Conventional Cox regression was examined initially, but Schoenfeld-residual diagnostics indicated non-proportionality for baseline BMI and selected covariates; the discrete-time model was therefore designated primary. There was no evidence that the effects of stress, reward processing, or emotional eating varied by follow-up year (joint interaction p=0.358, 0.688, and 0.544, respectively).

2.6 Exploratory index and sensitivity analyses

An exploratory neurobehavioral index was calculated among participants with complete core exposure data as the arithmetic mean of z-standardized stress, reward-processing, and emotional-eating scores; the resulting index was then standardized for modeling. The index was treated as a summary of the joint numerical profile across the three domains rather than as a clinical scale. Tertiles were used for descriptive cumulative-incidence curves and a log-rank test. Sensitivity analyses used a robust Poisson person-time model and a discrete-time model additionally adjusted for depressive symptoms. Two-sided p<0.05 was considered statistically significant.

3. Results

3.1 Cohort characteristics and incidence

Mean age was 42.6 (11.4) years, 52.7% were female, and mean baseline BMI was 25.53 (2.26) kg/m². Overall, 447 participants (59.6%) were overweight at baseline. During 2,609.8 person-years of observation, 98 participants developed obesity (13.1%), corresponding to 37.6 events per 1,000 person-years. Event risk was 20.4% among participants overweight at baseline versus 2.3% among those with baseline BMI <25 kg/m². Participants who developed obesity had higher mean stress (21.7 vs 19.5) and emotional-eating scores (2.78 vs 2.54) and lower physical activity (158.8 vs 182.8 min/week) than non-cases (Table 1).

3.2 Individual neurobehavioral predictors

In unadjusted discrete-time models, each SD increase in stress was associated with a 45% higher hazard of incident obesity (HR 1.45, 95% CI 1.20-1.75; $p<0.001$), and each SD increase in emotional eating with a 41% higher hazard (HR 1.41, 95% CI 1.17-1.70; $p<0.001$). Reward-processing score was not statistically associated with the outcome (HR 1.14, 95% CI 0.95-1.38; $p=0.162$). In the mutually adjusted model, these associations attenuated: stress adjusted HR 1.21 (95% CI 0.92-1.59; $p=0.177$), reward processing 0.98 (0.74-1.30; $p=0.892$), and emotional eating 1.26 (0.96-1.67; $p=0.098$). Baseline BMI was the strongest predictor (adjusted HR 4.63 per SD, 95% CI 3.10-6.92; $p<0.001$); older age was inversely associated with event hazard (adjusted HR 0.75 per SD, 95% CI 0.58-0.96; $p=0.023$) (Table 2; Figure 3).

3.3 Combined neurobehavioral profile

The exploratory standardized neurobehavioral index was associated with incident obesity after adjustment for age, sex, baseline BMI, and family history (adjusted HR 1.42 per SD, 95% CI 1.13-1.79; $p=0.003$). Crude incidence rates rose across index tertiles from 26.1 to 34.0 to 52.4 per 1,000 person-years; cumulative-incidence curves differed overall (log-rank $p=0.031$; Figure 2). Relative to tertile 1, the adjusted HR was 1.10 (95% CI 0.59-2.04) for tertile 2 and 1.64 (0.93-2.89) for tertile 3, indicating a graded but imprecisely estimated categorical pattern. Population-standardized model curves projected 60-month cumulative incidence of 17.7%, 22.6%, and 28.3% at -1 SD, the mean, and +1 SD of the index, respectively (Figure 4).

3.4 Sensitivity analyses

The continuous index remained associated with obesity in a robust Poisson person-time analysis (IRR 1.45 per SD, 95% CI 1.19-1.76; $p<0.001$). Additional adjustment for depressive symptoms attenuated the discrete-time estimate to HR 1.29 (95% CI 0.99-1.69; $p=0.060$; $n=584$, 75 events), consistent with partial overlap between affective symptoms and the neurobehavioral construct (Table 3).

4. Discussion

Our principal finding is that stress and emotional-eating scores were associated with incident obesity in unadjusted longitudinal models, but neither remained independently significant after baseline adiposity and other core covariates were considered. By contrast, a combined standardized profile spanning stress, reward processing, and emotional eating remained associated with incident obesity. This pattern suggests that neurobehavioral susceptibility may be distributed across correlated domains rather than captured reliably by a single recorded score.

The attenuation of stress after adjustment is biologically plausible. Long-term cortisol exposure has been linked to later BMI gain [5], while glucocorticoid excess can alter appetite signaling and facilitate reward-related eating [6]. Yet perceived stress and endocrine stress markers are not interchangeable, and their relationships with eating may depend on cortisol dynamics, context, and eating style [7-9,29]. The present data cannot separate endocrine from subjective stress pathways. The adjusted estimate for stress nevertheless remained above unity, with a confidence interval compatible with both little effect and a clinically relevant increase.

Emotional eating showed a similar pattern. Recent syntheses support an association between negative emotional eating and BMI, while emphasizing heterogeneity in how emotional overeating is measured and expressed [11,12]. Treatment-seeking and longitudinal studies likewise show that emotional-eating phenotypes are multidimensional and unstable over time [13,14]. Stress-recovery experiments further demonstrate that comfort eating is context dependent and may not be uniformly maladaptive [23]. Our adjusted HR of 1.26 per SD therefore fits a literature in which emotional eating is plausibly contributory but difficult to isolate from baseline adiposity, depression, stress, and other correlated behaviors.

Reward processing was not independently predictive in this cohort. This should not be interpreted as evidence that food reward is irrelevant to obesity. Reward-related constructs include wanting, liking, dyscontrol, hedonic hunger, delay discounting, and neural salience, and their relationships with BMI differ by construct and context [15-18,30]. The weak single-score association observed here may reflect measurement specificity, restricted baseline BMI range, or overlap with stress and emotional eating. Modern work on food reward also emphasizes the importance of sensory and macronutrient properties of foods, which were not measured in this dataset [18].

The composite result is compatible with a systems view of obesity vulnerability. Eating-behavior reviews suggest that no single trait consistently explains energy intake or BMI [10], while ecological momentary evidence indicates that affect and appetite interact within real-world episodes of temptation and lapse [19]. Behavioral weight-management trials can modify some eating traits, although effects on emotional eating and hedonic constructs are variable [20,21]. Diet composition,

discretionary snacking, exercise, and adaptive responses to energy imbalance further shape appetite and weight trajectories [22,24,25]. Serotonergic pathways illustrate how homeostatic and hedonic systems are biologically integrated [26], while family transmission and habitual physical activity contribute additional background susceptibility [27,28]. Thus, the observed joint index may function less as a single causal mechanism than as a marker of converging behavioral risk.

Baseline BMI was by far the dominant predictor. This is clinically unsurprising: participants closer to the BMI 30 threshold required less absolute weight gain to meet the incident-obesity definition. It also explains why unadjusted psychosocial associations weakened substantially after adjustment. The result argues for careful interpretation of “prediction”: neurobehavioral measures may help refine risk within a given adiposity stratum, but they should complement rather than replace anthropometric surveillance.

This study has several strengths. The outcome was based on repeated longitudinal height and weight measurements, and event classifications were verified against visit-level data. Follow-up extended to five years, and the primary discrete-time approach avoided reliance on a proportional-hazards assumption that was not supported for key covariates. The analysis also separated individual exposure estimates from a clearly labeled exploratory composite and tested robustness using an alternative person-time model.

Several limitations should be considered. Missing core exposure data led to complete-case analysis of 619 participants, although event proportions were nearly identical in included and excluded groups. Neurobehavioral exposures were represented by baseline study-recorded scores; item-level psychometric data were not available for the present analysis, limiting assessment of internal consistency and interpretation against clinical cut points. Exposure measures were treated as baseline-fixed, so temporal changes in stress or eating behavior were not modeled. Dietary intake and objective biological stress markers were not available. Residual confounding is likely, and the exploratory index is not a validated clinical scale and requires external validation. Finally, incident obesity defined by BMI crossing 30 kg/m² is clinically interpretable but sensitive to baseline proximity to the threshold and does not distinguish fat from lean mass.

5. Conclusions

The findings support a nuanced neurobehavioral model of obesity development. Stress and emotional eating were longitudinal predictors before adjustment, but baseline BMI accounted for much of their independent association. A combined standardized stress-reward-emotional-eating profile retained prognostic information, suggesting that integrated neurobehavioral assessment may identify a subgroup with elevated risk. Prospective studies with validated instruments, repeated neurobehavioral measures, objective stress biomarkers,

dietary phenotyping, and external validation are needed before such a composite is used for clinical risk stratification.

Declarations

Ethics approval: Ethical approval was obtained for this study.

Data availability: The dataset analyzed in this study is not included in the article.

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Competing interests: None.

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Figures

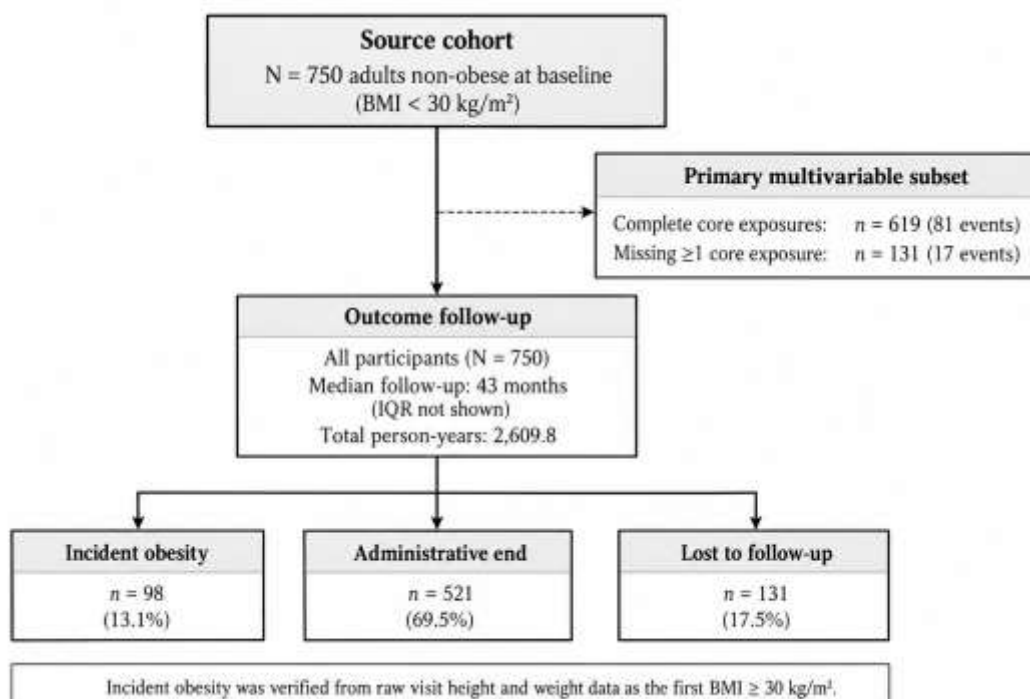


Figure 1. Cohort flow and analytic samples. Incident obesity was defined from longitudinal height and weight measurements as the first follow-up BMI ≥ 30 kg/m².

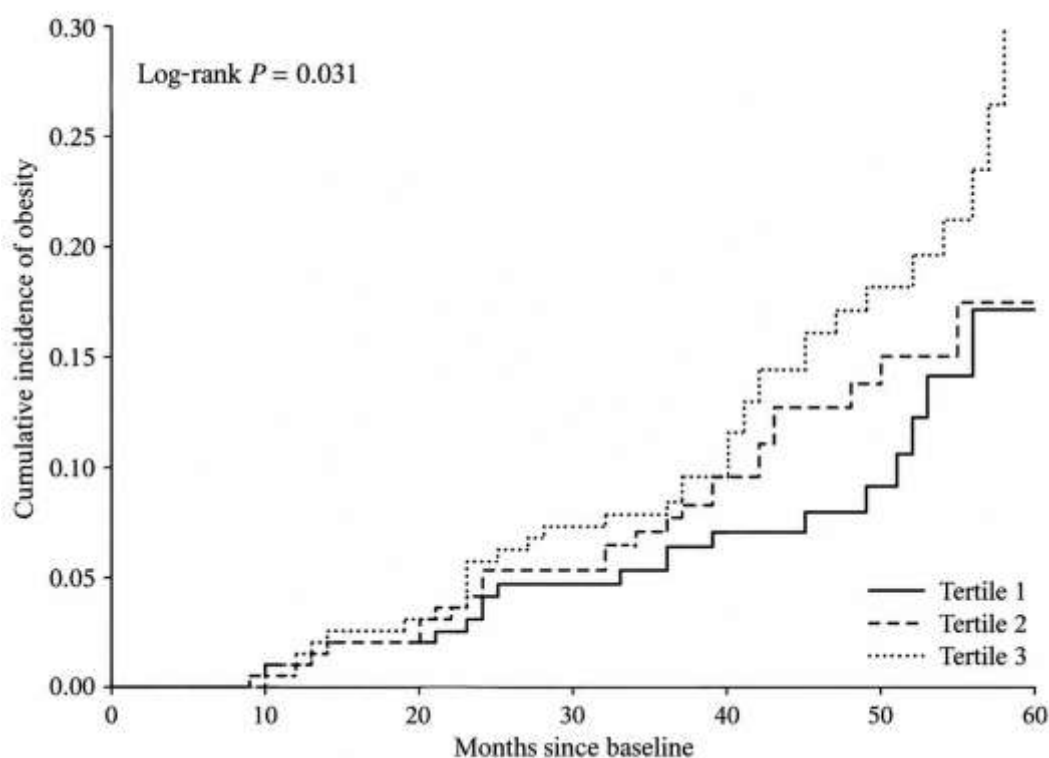


Figure 2. Kaplan-Meier-derived cumulative incidence of obesity by tertile of the exploratory neurobehavioral index (n=619). Tertile cut points were -0.346 and 0.343 on the mean-z index; log-rank $p=0.031$.

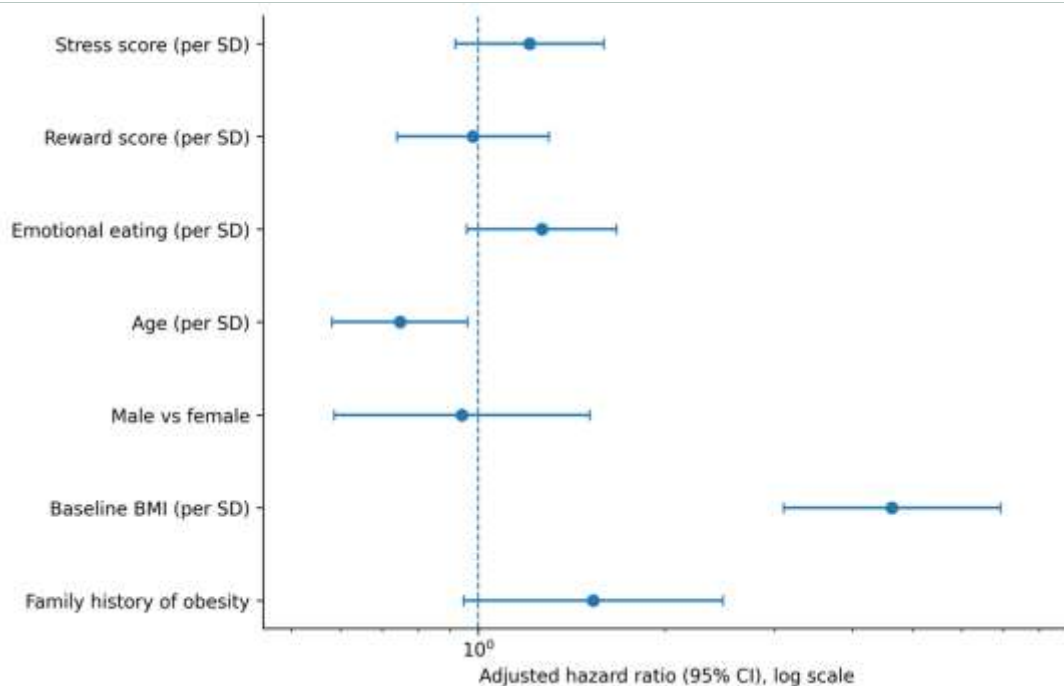


Figure 3. Adjusted hazard ratios for incident obesity in the primary discrete-time model. Error bars show 95% confidence intervals; the dashed line denotes HR=1.

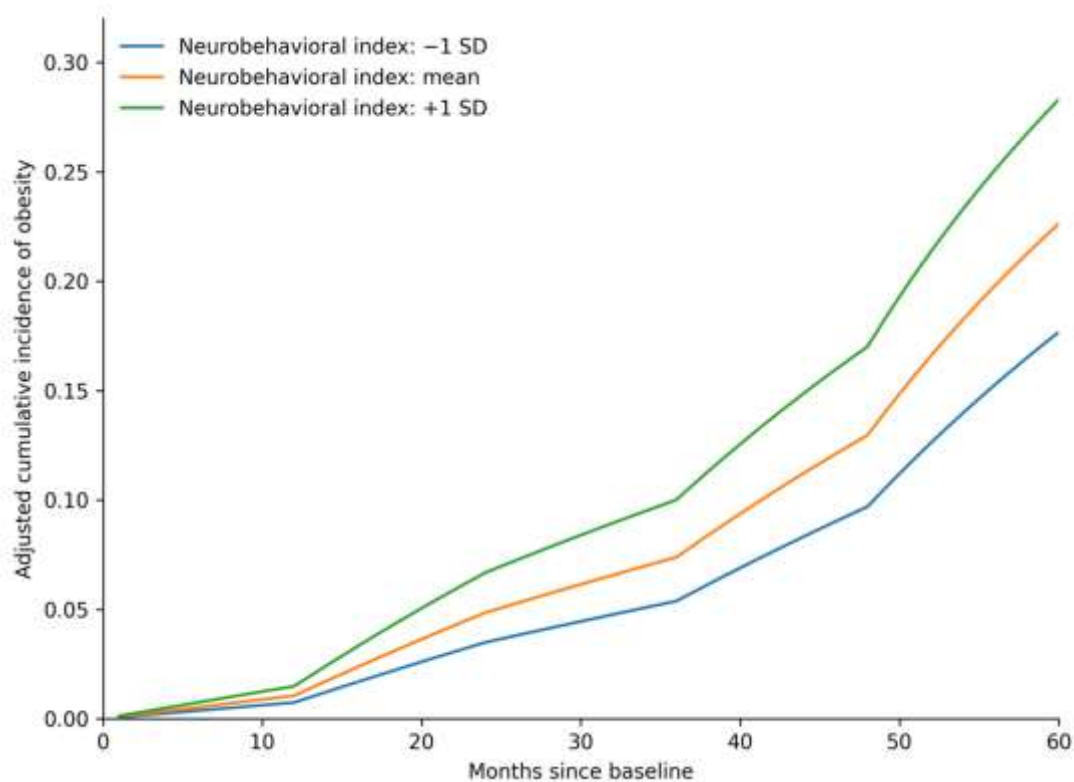


Figure 4. Covariate-standardized model-based cumulative incidence at -1 SD, the mean, and +1 SD of the exploratory neurobehavioral index. Curves average predicted risk over the empirical age, sex, baseline BMI, and family-history distribution.

Tables

Table 1. Baseline characteristics by incident obesity status

Characteristic	Overall (N=750)	Incident obesity (n=98)	No incident obesity (n=652)	p value
Age, years	42.6 (11.4)	40.9 (11.4)	42.8 (11.4)	0.128
Baseline BMI, kg/m ²	25.5 (2.3)	27.6 (1.7)	25.2 (2.2)	<0.001
Stress score	19.8 (5.9)	21.7 (5.8)	19.5 (5.9)	<0.001
Reward-processing score	49.3 (15.2)	51.4 (13.0)	49.0 (15.5)	0.103
Emotional-eating score	2.6 (0.6)	2.8 (0.6)	2.5 (0.6)	<0.001
Sleep duration, h/night	7.0 (0.8)	6.8 (0.9)	7.0 (0.8)	0.066
Physical activity, min/week	179.6 (102.1)	158.8 (80.8)	182.8 (104.7)	0.012
Depressive-symptom score	6.0 (3.4)	6.9 (3.6)	5.9 (3.4)	0.014
Female sex	395 (52.7%)	52 (53.1%)	343 (52.6%)	0.933
Family history of obesity	262 (34.9%)	40 (40.8%)	222 (34.0%)	0.190
Shift work	138 (18.4%)	14 (14.3%)	124 (19.0%)	0.260
Weight-gain medication	73 (9.7%)	12 (12.2%)	61 (9.4%)	0.368
Overweight at baseline (BMI 25-29.9)	447 (59.6%)	91 (92.9%)	356 (54.6%)	<0.001
Socioeconomic group, Low / Middle / High	262 (34.9%) / 248 (33.1%) / 240 (32.0%)	40 (40.8%) / 32 (32.7%) / 26 (26.5%)	222 (34.0%) / 216 (33.1%) / 214 (32.8%)	0.337
Smoking, Never / Former / Current	479 (63.9%) / 149 (19.9%) / 122 (16.3%)	64 (65.3%) / 23 (23.5%) / 11 (11.2%)	415 (63.7%) / 126 (19.3%) / 111 (17.0%)	0.282

Values are mean (SD) or n (%). p values are descriptive unadjusted comparisons (Welch t test for continuous variables; chi-square/Fisher exact test for categorical variables). Missing values: stress 35, reward 60, emotional eating 44, sleep 37, physical activity 52, depressive symptoms 41.

Table 2. Associations with incident obesity in discrete-time complementary log-log models

Predictor	Unadjusted HR (95% CI)	p	Adjusted HR (95% CI)	p
Stress score (per SD)	1.45 (1.20-1.75)	<0.001	1.21 (0.92-1.59)	0.177
Reward-processing score (per SD)	1.14 (0.95-1.38)	0.162	0.98 (0.74-1.30)	0.892
Emotional-eating score (per SD)	1.41 (1.17-1.70)	<0.001	1.26 (0.96-1.67)	0.098
Age (per SD)	—	—	0.75 (0.58-0.96)	0.023
Male vs female	—	—	0.94 (0.59-1.51)	0.802
Baseline BMI (per SD)	—	—	4.63 (3.10-6.92)	<0.001
Family history of obesity	—	—	1.53 (0.95-2.48)	0.082

Adjusted model: n=619, 81 events; mutually includes the three core exposures, age, sex, baseline BMI, family history of obesity, and annual baseline-hazard indicators. HRs for continuous variables are per 1-SD increase.

Table 3. Exploratory combined neurobehavioral index and sensitivity analyses

Analysis	N	Events	Incidence /1,000 PY	Adjusted effect (95% CI)	p
Continuous index, per SD	619	81	—	1.42 (1.13-1.79)	0.003
Tertile 1 (lowest)	207	19	26.1	Reference	—
Tertile 2	206	24	34.0	1.10 (0.59-2.04)	0.770
Tertile 3 (highest)	206	38	52.4	1.64 (0.93-2.89)	0.087
Sensitivity: robust Poisson, per SD	619	81	—	IRR 1.45 (1.19-1.76)	<0.001
Sensitivity: + depressive symptoms, per SD	584	75	—	1.29 (0.99-1.69)	0.060

Primary composite and tertile HRs are adjusted for age, sex, baseline BMI, family history of obesity, and annual baseline-hazard indicators. IRR, incidence rate ratio; PY, person-years.