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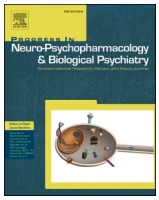

# Lower baseline plasma selenium is associated with later suicide attempts in eating disorders: A longitudinal cohort

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ABSTRACT

*Background:* Individuals with eating disorders are at high risk of suicide attempts (SA), yet nutritional correlates remain underexplored. Selenium, an essential micronutrient with antioxidant and neuroprotective functions, is often deficient in eating disorders. We examined the association between baseline plasma selenium and subsequent SA.

*Methods:* We analyzed 658 patients with eating disorders recruited at the University Hospital Center of Montpellier (2012–2014 and 2017–2020). Baseline plasma selenium was quantified by the hospital's biochemistry laboratory. SA occurring after baseline were identified through review of electronic Emergency Department records up to December 1, 2024. Semi-parametric Cox proportional hazards models estimated hazard ratios (HR) for time to first SA.

*Results:* Median plasma selenium was 82.9 μg/L. Over a median 82-month follow-up, 40 participants (6.1%) attempted suicide. Higher selenium levels were associated with lower hazard of SA (HR per 1 SD = 0.59, 95% CI 0.42–0.83, $p = 0.002$). Results were similar across models adjusted for age, sex, and BMI and, separately, past SA, global eating disorder severity, current depression, psychotropic medication use, and additional C-reactive protein, albumin, zinc, and iron indices. Sensitivity analyses using ≤83 and ≤ 70 μg/L thresholds, restricting to women, and excluding extreme values yielded similar results. Exploratory analyses suggested modest attenuation of associations between purging, tobacco use, alcohol/substance use disorder and SA after adding selenium.

*Conclusions:* Lower baseline selenium was associated with higher subsequent SA hazard. If replicated, selenium status could inform future risk models.

## 1. Introduction

Suicidal behaviors (SB) remain a leading cause of premature mortality worldwide (WHO, 2020). Among psychiatric populations, individuals with eating disorders face particularly elevated risk. Lifetime suicide attempt (SA) prevalence approaches one-third in eating disorders, with the highest rates observed in binge–purge and purging subtypes (Mandelli et al., 2019; Xu et al., 2023). Meta-analytic evidence indicates that eating disorders confer nearly a twofold increased risk of both SA and suicide death, independent of comorbid mood, anxiety, and substance use disorders (Yao et al., 2016). With the prevalence of eating disorders and self-harm rising internationally in recent years, especially among adolescent girls (Trafford et al., 2023), improving risk stratification and identifying clinically informative correlates of SB risk in this population has become a public health and clinical priority.

The links between eating disorders and SB are multifactorial. Shared vulnerabilities such as trauma exposure, affective instability, impulsivity, and psychiatric comorbidity are well documented (Smith et al., 2018). In addition, illness-related physiological states, including nutritional deficiencies, metabolic stress, and systemic inflammation, may contribute to SB risk and may index periods of heightened clinical instability. Micronutrient deficiencies are common in eating disorders

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and can persist even after partial weight restoration. In anorexia nervosa (AN), nearly half of patients have at least one trace element deficiency, with selenium deficiency among the most frequent and most pronounced in binge–purge subtypes (Hanachi et al., 2019).

Selenium is an essential trace element required for the synthesis of selenoproteins, including glutathione peroxidases and thioredoxin reductases, which participate in antioxidant defence and immune function (Burk and Hill, 2015; Rayman, 2012). Selenium status has been linked to depressive symptoms in observational studies (Chen et al., 2023; Da et al., 2025; Ferriani et al., 2022; Pasco et al., 2012), and meta-analyses report inverse associations between selenium intake and depression and possible benefits of supplementation (Ding and Zhang, 2022; Wang et al., 2023). In population data, higher dietary selenium intake was prospectively associated with lower suicidal ideation (Liu and Chen, 2025). We previously observed a cross-sectional association between selenium deficiency and SA history in AN (Strumila et al., 2022). Mendelian randomization analyses have also suggested that genetically higher selenium levels may be associated with lower AN risk (Guo et al., 2023). However, circulating selenium is state-dependent and influenced by inflammation, acute illness acuity, and nutritional instability. Therefore, in eating disorders, lower plasma selenium may plausibly reflect a broader vulnerability state (e.g., malnutrition and inflammatory burden) rather than a specific etiologic contributor to SB risk (Braunstein et al., 2020; Rock and Moos, 2009).

Importantly, selenium biology does not operate in isolation. Zinc, reported to be lower in AN, particularly binge–purge presentations, supports the synthesis and function of several selenoproteins and is also linked to inflammatory states (Leach et al., 2025). Iron status, frequently altered in eating disorders, may likewise influence oxidative stress pathways and antioxidant demand (Papillard-Marechal et al., 2012). These interrelated nutritional and inflammatory processes underscore the need to interpret selenium findings cautiously and in context.

To date, no longitudinal study has examined whether baseline plasma selenium is associated with subsequent SA occurrence in eating disorders using clinically ascertained outcomes over extended follow-up. We therefore investigated the association between baseline plasma selenium levels and later SA in a large, well-characterized eating disorders cohort, with SA identified through systematic review of Emergency Department records. We also explored whether selenium levels were associated with the strength of associations between purging, tobacco use, and alcohol/substance use disorder and later SA - behaviors linked to both lower selenium status and elevated SA risk (Arnaud et al., 2006; Baldessarini et al., 2019; González-Reimers et al., 2009; Lengvenyte et al., 2021, 2019; Luty-Frackiewicz et al., 2002). We hypothesized that lower baseline plasma selenium would be associated with higher subsequent SA risk and would index broader nutritional and inflammatory vulnerability in eating disorders.

## 2. Methods

### 2.1. Study cohort

This analysis used the combined sample from two harmonized prospective cohorts of patients with eating disorders recruited at the Eating Disorders Unit, Lapeyronie Academic Hospital (CHU Montpellier), Montpellier, France. The first cohort enrolled participants between February 2012 and October 2014, and the second between May 2017 and January 2020. Recruitment did not occur between 2014 and 2017 due to a planned interruption in study activity (funding), rather than selective inclusion. The two cohorts used comparable procedures for clinical evaluation and baseline blood collection, enabling pooled analyses.

Ethics approvals were granted by the Comité de Protection des Personnes Sud Méditerranée IV (approval 11–04-SC; first cohort) and CPP Sud-Est 6 (approval AU13–13; second cohort). All participants, or their legal representatives for minors, signed the informed consent, and procedures complied with the Declaration of Helsinki.

Inclusion criteria were: age 15–45 years, fluency in French, diagnosis of anorexia nervosa (AN), bulimia nervosa (BN), or other eating disorder according to DSM-5 criteria (American Psychiatric Association, 2013), and ability to provide informed consent. Participants recruited prior to DSM-5 publication were reassessed to ensure diagnostic harmonization. The upper age limit was prespecified to align with the age range targeted by the cohorts and to reduce heterogeneity related to later-life medical comorbidity and nutritional/inflammatory profiles. Exclusion criteria were inability to complete the evaluation, pregnancy or breastfeeding, and deprivation of liberty by judicial or administrative decision. Diagnoses were established by senior psychiatrists and nutritionists specialized in eating disorders. Psychiatric comorbidities were assessed using DSM-5 criteria and corroborated using medical records and collateral information when available.

### 2.2. Psychiatric and clinical assessment

Participants completed validated self-report measures at baseline, including the Eating Disorder Examination Questionnaire (EDE-Q) (Luce and Crowther, 1999), which assesses restraint, eating concern, shape concern, and weight concern over the preceding 28 days. A senior psychiatrist conducted standardized clinical interviews to assess psychiatric comorbidities, including major depressive disorder and current major depressive episode (MDE), bipolar disorder, anxiety disorders, post-traumatic stress disorder, obsessive–compulsive disorder, and alcohol or substance use disorder, using DSM-5 criteria. Lifetime history of SA was assessed by direct questioning and corroborated using medical records when available. SA history was defined as self-injurious behavior with intent to die, consistent with the Columbia Classification approach for distinguishing SA from non-suicidal self-injury (Posner et al., 2011).

Purging behaviors in the past 28 days were derived from EDE-Q items 13–15 (self-induced vomiting, laxative misuse, and diuretic misuse). Any endorsement (score $\geq 1$) on at least one of these items was coded as current purging. Tobacco use was recorded at baseline during clinical interview. Additional clinical characteristics, including illness duration, body mass index (BMI), and psychotropic medication exposure (antidepressants and antipsychotics), were collected systematically from participant interview and medical records.

### 2.3. Outcome capture and catchment assumptions

Incident SA during follow-up were ascertained through systematic retrospective review of Emergency Department records at Montpellier University Hospital, completed in December 2024. Montpellier University Hospital is the regional referral for acute psychiatric presentations in a catchment area of approximately 500,000 inhabitants, where individuals presenting with suicidal ideation or following SA are typically referred for medical and psychiatric evaluation. SA events occurring outside this hospital system (e.g., after relocation or presentation to out-of-area facilities) would not be captured, representing a potential source of under-ascertainment.

To reduce outcome misclassification, we queried all hospital encounters (psychiatry and non-psychiatry) under each participant's unique identifier and reviewed free-text clinical notes when visit reason coding was ambiguous. SA were defined as self-injurious behaviors with intent to die, consistent with established research definitions (Erlangsen et al., 2011). This approach to identifying SA-related outcomes from hospital records has been validated in prior studies (Lengvenyte et al., 2023; Pellatt et al., 2025).

Follow-up time accrued from the date of baseline blood draw to the earliest of: first SA recorded in ED records or end of observation (1 December 2024). Observation time was expressed in person-months. The median follow-up was 82 months (IQR 49–122 months). Under-ascertainment of SA would be expected to bias associations toward the

null if not systematically related to baseline selenium.

### 2.4. Laboratory measurements

Baseline blood samples were collected during admission as part of the study baseline assessment. Plasma selenium concentration was measured in EDTA-anticoagulated samples using inductively coupled plasma mass spectrometry (ICP-MS) in the hospital's certified biochemistry laboratory, in accordance with established standards for trace-element quantification. Laboratory staff were blinded to participants' clinical characteristics. Selenium concentrations are reported in μg/L. For analyses, selenium was examined as a continuous variable (z-standardized). For descriptive and sensitivity purposes, selenium was also dichotomized at the sample median (≤83 μg/L vs. >83 μg/L) and categorized using a deficiency threshold (≤70 μg/L), corresponding to concentrations below which plasma glutathione peroxidase activity is considered impaired (Perri et al., 2024). Two participants with selenium values <20 μg/L were excluded as biologically implausible.

Plasma zinc was measured as part of the same trace-element assessment. In addition, C-reactive protein (CRP), albumin, and iron indices (including ferritin and transferrin saturation) were obtained from baseline blood testing collected systematically within the study protocol. Because all elements were measured once at baseline, values may reflect short-term physiological state at the time of sampling.

### 2.5. Statistical analysis

Analyses included participants with baseline selenium measured and available follow-up for outcome ascertainment. Sample size varied across sensitivity analyses due to missingness in additional laboratory measures. Baseline characteristics were summarized as means (SD) or medians (IQR) for continuous variables and as counts (%) for categorical variables. Comparisons between low and high selenium groups used Student's *t*-tests or Mann–Whitney *U* tests for continuous variables and $\chi^2$ or Fisher's exact tests for categorical variables.

The primary outcome was time to first SA during follow-up. Cox proportional hazards models (Efron method for ties) estimated hazard ratios (HRs) per 1 SD increase in z-standardized selenium. Continuous covariates were mean-centered. For visualization, selenium was dichotomized at the sample median (≤83 μg/L vs. >83 μg/L) and examined using Kaplan–Meier curves and log-rank tests. The distribution of plasma selenium was approximately normal (skewness = 0.65; kurtosis = 1.82), therefore no transformation was applied.

To limit overfitting given 40 events, we used a parsimonious sequence of partially adjusted models rather than a fully jointly adjusted model: (0) unadjusted; (1) adjusted for age, sex, and body mass index (BMI); and additionally adjusted separately for (2) past SA, (3) eating disorder severity (ED*E*-Q global score), (4) current major depressive episode (MDE), (5) antidepressant use, and (6) antipsychotic use. Potential residual confounding was quantified using E-values.

To account for calendar-period effects, Cox models were stratified by recruitment cohort (2012–2014 vs. 2017–2020), allowing the baseline hazard to vary across cohorts while estimating a common selenium effect. Proportional hazards assumptions were assessed using Schoenfeld residuals. Nonlinearity was evaluated by adding a quadratic selenium term and by inspection of Martingale residuals; neither suggested meaningful deviation from linearity. Dose–response patterns were additionally explored using selenium quartiles.

Exploratory attenuation analyses examined whether selenium levels were associated with the strength of associations between risk behaviors (purging, tobacco use, alcohol/substance use disorder) and SA hazard. Percent attenuation was calculated on the log-HR scale. Associations between these behaviors and selenium concentration (z-standardized) were tested using linear regression adjusted for age, sex, and BMI, with additional adjustment for EDE-Q score in secondary models. Within the attenuation analyses, q-values were calculated using the Benjamini–Hochberg procedure across the three behaviors within each adjustment set.

Sensitivity analyses included (1) selenium deficiency defined as ≤70 μg/L, (2) women-only analyses, and (3) exclusion of extreme selenium values (>150 μg/L). As a comparator laboratory measure reflecting general nutritional/inflammatory status, we repeated the primary analysis using baseline albumin. To address potential confounding by inflammatory and nutritional state, we conducted additional sensitivity analyses adjusting for CRP (log-transformed) and albumin. To contextualize selenium within trace-element status, we also examined models adjusting for plasma zinc. Iron status was explored using ferritin (log-transformed) and transferrin saturation (TSAT); ferritin models were adjusted for CRP given ferritin's acute-phase properties.

All analyses were conducted using JASP version 0.19.3. Statistical significance was defined as two-sided $p < 0.05$.

## 3. Results

### 3.1. Sample characteristics

A total of 658 participants with eating disorders were included (Table 1). Most were women (93.2%), with a mean age of 28.0 years (SD 11.0) and a mean BMI of 20.35 kg/m$^2$ (SD 6.11). The diagnostic distribution was 56.7% anorexia nervosa, 21.6% bulimia nervosa, and 21.7% other eating disorders. Baseline plasma selenium concentration had a median of 82.9 μg/L (IQR 72.0–93.7) and a mean of 83.7 μg/L (SD 16.8). Participants were categorized into low (≤83 μg/L; $n = 332$) and high (>83 μg/L; $n = 326$) selenium groups.

Compared with participants in the high selenium group, those with low selenium were younger, had shorter illness duration, and had higher EDE-Q scores. Low selenium was also associated with higher prevalence of current major depressive episode, alcohol/substance use disorder, prior SA, purging behaviors, and tobacco use, as well as more frequent antidepressant and antipsychotic use (Table 1). During a median follow-up of 82 months, 40 participants (6.1%) had at least one SA. Incidence was higher in the low selenium group than the high selenium group (9.0% vs 3.1%; $p = 0.001$), corresponding to 30 and 10 events, respectively.

### 3.2. Selenium and risk of subsequent suicide attempts

In unadjusted Cox models, higher baseline plasma selenium was associated with a lower hazard of subsequent SA (HR per 1 SD increase = 0.59, 95% CI 0.42–0.83, $p = 0.002$; Table 2, Fig. 1). The association remained significant after adjustment for age, sex, and BMI (HR = 0.58, 95% CI 0.41–0.83, p = 0.002) and persisted across partially adjusted models that additionally accounted separately for prior SA history, eating disorder severity (EDE-Q score), current major depressive episode, and psychotropic medication exposure (Table 2, Fig. 1).

To address the state-dependent nature of circulating selenium and potential confounding by inflammatory and nutritional status, we additionally adjusted for lnCRP and albumin. The inverse association between selenium and subsequent SA remained statistically significant with modest attenuation (HR = 0.61, 95% CI 0.41–0.89, $p = 0.012$). To contextualize selenium within trace-element status, adjustment for plasma zinc yielded a similar and robust association (HR = 0.55, 95% CI 0.38–0.77, $p < 0.001$). In models incorporating iron indices alongside lnCRP, selenium remained inversely associated with SA risk both with transferrin saturation (TSAT) (HR = 0.58, 95% CI 0.40–0.84, $p = 0.003$) and with additional adjustment for ferritin (HR = 0.61, 95% CI 0.43–0.89, $p = 0.009$). Results were also consistent when stratifying baseline hazards by recruitment era (HR = 0.56, 95% CI 0.40–0.79, p < 0.001).

Kaplan–Meier curves using a median split showed a lower cumulative incidence of SA among participants with higher baseline selenium (log-rank $\chi^2 = 10.4$, $p = 0.001$; Fig. 2.). The proportional hazards

**Table 1**
Sample description at baseline.

| Characteristic | Whole cohort ($n$ = 658) | Low selenium ≤83 μg/L (n = 332) | High selenium >83 μg/L ($n$ = 326) | $p$-value* | N |
|---|---|---|---|---|---|
| Demographics | | | | | |
| Age, years, mean ± SD | 27.99 ± 11.00 | 26.87 ± 9.54 | 29.13 ± 12.23 | 0.008 | 658 |
| Female sex, n (%) | 613 (93.2) | 310 (93.4) | 303 (92.9) | 0.828 | 658 |
| Anthropometrics / illness course | | | | | |
| BMI, kg/m$^2$, mean ± SD | 20.35 ± 6.11 | 20.68 ± 9.54 | 20.01 ± 6.05 | 0.156 | 658 |
| Duration of eating disorder, years, mean ± SD | 8.57 ± 8.99 | 7.90 ± 7.97 | 9.27 ± 9.90 | 0.037 | 560 |
| History of hospitalization for eating disorder | 239 (36.6) | 133 (40.1) | 106 (33.0) | 0.062 | 653 |
| Eating disorder characteristics | | | | | |
| Anorexia nervosa, n (%) | 378 (56.7) | 187 (56.3) | 186 (57.1) | .. | .. |
| Bulimia nervosa, n (%) | 142 (21.6) | 71 (21.4) | 71 (21.8%) | .. | .. |
| Other eating disorders, n (%) | 143 (21.7) | 74 (22.3) | 69 (21.2) | .. | .. |
| | .. | .. | .. | 0.940 | 658 |
| Eating disorder severity (EDE-Q score), mean ± SD | 3.32 ± 0.064 | 3.55 ± 1.47 | 3.08 ± 1.61 | <0.001 | 595 |
| Psychiatric comorbidity | | | | | |
| Current major depressive episode, n (%) | 185 (30.1) | 109 (34.8) | 76 (25.2) | 0.009 | 615 |
| Major depressive disorder, n (%) | 408 (65.2) | 210 (65.1) | 198 (64.5) | 0.726 | 626 |
| Anxiety disorder, n (%) | 237 (57.5) | 124 (56.6) | 113 (58.5) | 0.693 | 412 |
| Obsessive compulsive disorder, n (%) | 103 (28.2) | 58 (29.0) | 45 (27.3) | 0.715 | 365 |
| Post-traumatic stress disorder, n (%) | 63 (18.2) | 42 (21.6) | 21 (13.8) | 0.061 | 346 |
| Bipolar disorder, n (%) | 45 (7.2) | 27 (8.5) | 18 (5.9) | 0.208 | 626 |
| Substance or alcohol use disorder, n (%) | 52 (8.3) | 37 (11.6) | 15 (4.9) | 0.002 | 625 |
| Substance use disorder, n (%) | 31 (5.0) | 21 (6.6) | 10 (3.3) | 0.056 | 623 |
| Alcohol use disorder, n (%) | 30 (4.8) | 22 (6.9) | 8 (2.6) | 0.012 | 623 |
| Prior suicide attempt, n (%) | 148 (22.7) | 91 (27.6) | 57 (17.6) | 0.002 | 653 |
| Risk behaviors | | | | | |
| Purging behaviors, past-month, n (%) | 387 (64.7) | 214 (72.3) | 173 (57.3) | <0.001 | 598 |
| Tobacco use, n (%) | 240 (37.7) | 136 (41.7) | 104 (32.7) | 0.018 | 644 |
| Treatment | | | | | |
| Hypnotic use, n (%) | 37 (5.6) | 20 (6.0) | 17 (5.2) | 0.652 | 658 |
| Anxiolytics, n (%) | 133 (20.2) | 76 (22.9) | 57 (17.5) | 0.084 | 658 |
| Antidepressant use, n (%) | 235 (35.7) | 134 (40.4) | 101 (31.1) | 0.012 | 658 |
| Antipsychotic use, n (%) | 67 (10.2) | 44 (13.3) | 23 (7.1) | 0.009 | 658 |
| Outcome | | | | | |
| Suicide attempt during follow-up, n (%) | 40 (6.1) | 30 (9.0) | 10 (3.1) | 0.001 | 658 |
| Baseline blood markers | | | | | |
| Plasma selenium levels (μg /L), mean ± SD | 83.74 ± 16.78 | 70.79 ± 8.74 | 96.92 ± 12.09 | .. | 658 |
| Albumin levels (g/L), mean ± SD | 46.63 ± 3.46 | 46.06 ± 3.39 | 47.21 ± 3.45 | <0.001 | 609 |
| Prealbumin levels (g/L), median [IQR], N | 0.26 [0.23–0.29] | 0.25 [0.23–0.28] | 0.28 [0.24–0.31] | <0.001 | 359 |
| C-reactive protein levels (mg/L), median [IQR] | 0.4 [0.3–1.4] | 0.5 [0.3–1.6] | 0.4 [0.3–1.2] | 0.155 | 637 |
| Plasma zinc levels (μmol/L), mean (SD) | 13.94 ± 3.31 | 13.36 ± 3.14 | 14.53 ± 3.37 | <0.001 | 657 |
| Ferritin levels (μg/L), median [IQR] | 48.0 [26.0–83.0] | 45.0 [26.0–78.75] | 51.0 [28.75–87.25] | 0.079 | 622 |
| Transferrin saturation (%), median [IQR] | 22.3 [16.0–31.0] | 21.8 [15.0–31.0] | 23.0 [17.0–31.0] | 0.313 | 617 |

Values are mean ± SD, median [IQR] or n (%). $p$-values compare low vs. high selenium groups (Student's t-test or Mann–Whitney U for continuous variables; $\chi^2$ or Fisher's exact test for categorical variables).
Abbreviations: BMI, body mass index; EDE-Q, Eating Disorder Examination-Questionnaire; MDE, major depressive episode.
Other eating disorders include binge eating disorder and other specified eating disorders. Purging behaviors include laxative use, vomiting, or diuretic use.

assumption was not violated (global Schoenfeld $p$ = 0.27; selenium-specific p = 0.27). A quadratic selenium term did not improve fit ($p$ = 0.15), supporting an approximately linear association on the log-hazard scale.

### 3.3. Exploratory attenuation analyses

We examined whether baseline selenium levels were associated with the magnitude of associations between selected risk behaviors and subsequent SA (Table 3). Purging behaviors were associated with increased hazard of SA in models adjusted for age, sex, and BMI (HR = 2.76, 95% CI 1.17–6.52, $p$ = 0.021), with similar results after adjustment for prior SA history. Adding selenium to these models reduced the estimated association on the log-HR scale by 22.7% and 16.7%, respectively. With additional adjustment for eating disorder severity (EDE-Q), the purging association weakened (HR = 2.26, 95% CI 0.90–5.67, $p$ = 0.083), with 15% attenuation after adding selenium.

Tobacco use showed HRs ranging from 1.86 to 2.39 across covariate sets, with 7–24% attenuation after adding selenium. Alcohol/substance use disorder was associated with higher hazard in base models (HR = 2.46, 95% CI 1.09–5.57, $p$ = 0.031), with 7–15% attenuation after adding selenium. These exploratory analyses suggest that adding selenium to the models was associated with modest attenuation of the estimated hazard ratios for SA. However, they do not establish mediation.

### 3.4. Associations between risk behaviors and selenium

Risk behaviors were associated with lower baseline selenium (Table 4). Purging behaviors were associated with reduced selenium after adjustment for age, sex, and BMI (β = −0.30 SD, 95% CI −0.48 to −0.13, $p$ < 0.001), persisting after additional adjustment for EDE-Q (β =

**Table 2**
Associations between baseline selenium concentration and subsequent suicide attempt.

| Model | Adjustment | HR (95% CI) per 1 SD increase in selenium | p-value | N | Events |
|---|---|---|---|---|---|
| 0 | Unadjusted | 0.59 (0.42–0.83) | 0.002 | 658 | 40 |
| 1 | Age, sex, BMI | 0.58 (0.41–0.83) | 0.002 | 658 | 40 |
| 1a | Age, sex, BMI, lnCRP, albumin | 0.61 (0.41–0.89) | 0.012 | 595 | 34 |
| 1b | Age, sex, BMI, zinc | 0.55 (0.38–0.77) | <0.001 | 657 | 40 |
| 1c | Age, sex, BMI, lnCRP, TSAT | 0.58 (0.40–0.84) | 0.003 | 604 | 37 |
| 1d | Age, sex, BMI, lnCRP, TSAT, ferritin | 0.61 (0.43–0.89) | 0.009 | 587 | 37 |
| 1, era stratified | Age, sex, BMI, baseline hazard stratified by recruitment era | 0.56 (0.40–0.79) | <0.001 | 658 | 40 |
| 2 | Age, sex, BMI and SA history | 0.630 (0.434–0.903) | 0.012 | 653 | 40 |
| 3 | Age, sex, BMI and EDE-Q score | 0.655 (0.454–0.944) | 0.023 | 595 | 36 |
| 4 | Age, sex, BMI and MDE | 0.595 (0.416–0.850) | 0.004 | 615 | 38 |
| 5 | Age, sex, BMI and antidepressant use | 0.604 (0.425–0.859) | 0.005 | 658 | 40 |
| 6 | Age, sex, BMI and antipsychotic use | 0.596 (0.418–0.849) | 0.004 | 658 | 40 |

HRs are per 1 SD increase in baseline plasma selenium (z-standardized) from semi-parametric Cox proportional hazards models (Efron ties). Model 0 is unadjusted; Model 1 adjusts for age, sex, and BMI. Models 1a–1d additionally adjust Model 1 for laboratory measures (lnCRP+albumin; zinc; TSAT with lnCRP; TSAT with lnCRP plus ferritin [log-transformed]). "Era stratified" indicates baseline hazards stratified by recruitment cohort (2012–2014 vs 2017–2020). Models 2–6 additionally adjust Model 1 separately for clinical covariates (prior SA, EDE-Q, current MDE, antidepressant use, antipsychotic use). N and events vary due to complete-case estimation. Proportional hazards were assessed using Schoenfeld residuals.
Abbreviations: BMI, body mass index; CI, confidence interval; CRP, C-reactive protein; EDE-Q, Eating Disorder Examination Questionnaire; HR, hazard ratio; lnCRP, log-transformed CRP; MDE, major depressive episode; SA, suicide attempt; TSAT, transferrin saturation.

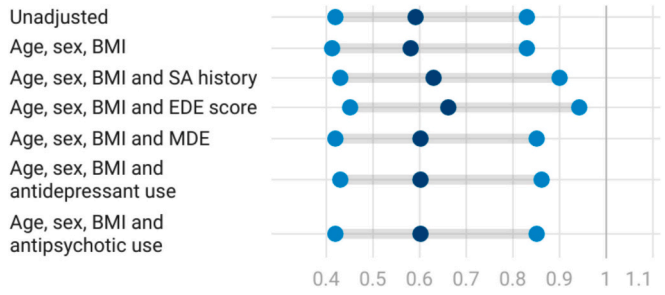


**Fig. 1.** Forest plot of hazard ratios for the association between baseline plasma selenium and subsequent suicide attempt. Hazard ratios (dots) and 95% confidence intervals (horizontal lines) per 1 SD increase in z-standardized plasma selenium concentration, estimated from semi-parametric Cox proportional hazards models (Efron ties). Model adjustments are indicated on the y-axis. All models include age, sex, and BMI as base covariates; additional clinical covariates are added separately per row.

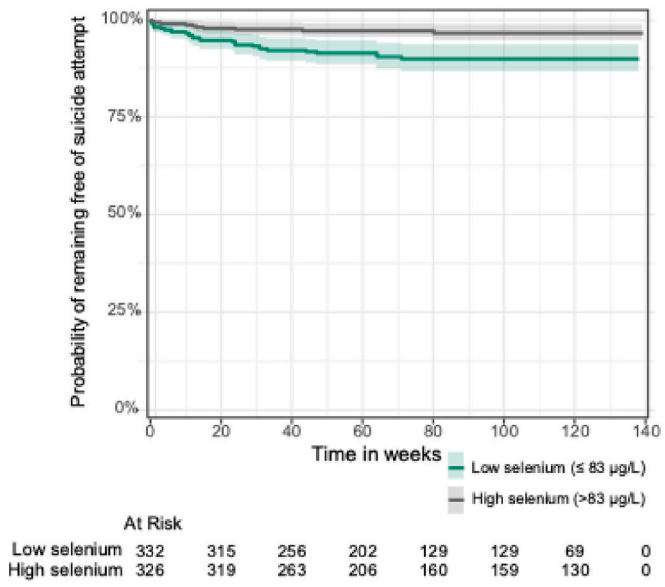


**Fig. 2.** Kaplan–Meier curves for survival free from suicide attempt in participants with low versus high baseline selenium concentrations.

−0.20 SD, $p = 0.048$). Tobacco use was similarly associated with lower selenium (β = −0.22 SD, 95% CI -0.38 to −0.06, $p = 0.008$), with results unchanged after EDE-Q adjustment. Alcohol/substance use disorder was associated with lower selenium in base models (β = −0.36 SD, 95% CI -0.65 to −0.08, $p = 0.012$), but was not statistically significant after EDE-Q adjustment (β = −0.27 SD, $p = 0.074$).

### 3.5. Sensitivity analyses

Findings were consistent across robustness checks (Supplementary Tables S1–S4; Supplementary Figs. S1–S2). When selenium was modeled using a deficiency threshold (≤70 µg/L), selenium-deficient participants had a higher hazard of SA compared with non-deficient participants (HR = 2.00, 95% CI 1.03–3.88, $p = 0.040$). Results were similar when restricting analyses to women (HR per 1 SD increase = 0.60, 95% CI 0.42–0.84, $p = 0.004$) and when excluding extreme selenium values (>150 µg/L; HR = 0.59, 95% CI 0.41–0.83, $p = 0.003$). Exploratory quartile analyses suggested lower hazards in the higher selenium quartiles compared with Q1 (Supplementary Table S3). In addition, exploratory joint selenium–zinc analyses using median splits showed that the combined low selenium/low zinc category was not associated with subsequent SA compared with all other participants (HR = 1.13, 95% CI 0.57–2.23; $p = 0.724$).

As a comparator laboratory measure reflecting general nutritional/inflammatory status, baseline albumin was not associated with subsequent SA when analyzed analogously (HR = 0.92, 95% CI 0.82–1.03, $p = 0.14$, adjusted for age, sex, BMI, and EDE-Q). In a restricted subset with prealbumin available ($n = 349$; 14 events), additional adjustment for prealbumin yielded the same direction of association between selenium and subsequent SA (HR for selenium per 1 SD increase = 0.34, 95% CI 0.17–0.68, $p = 0.002$), but precision was limited due to the small number of events. Potential unmeasured confounding was assessed using E-values (E-value = 2.84 for the point estimate and 1.70 for the lower confidence limit for the model adjusted for age, sex, and BMI; Supplementary Table S4).

## 4. Discussion

In this longitudinal cohort of 658 individuals with eating disorders, higher baseline plasma selenium was associated with a lower hazard of subsequent SA during follow-up. The association was consistent across a series of parsimonious Cox models accounting for age, sex, BMI, recruitment era, eating disorder severity, current major depressive episode, prior SA, and psychotropic medication exposure. Results were

**Table 3**
Association of risk behaviors with subsequent suicide attempt, and percent change in effect after adding selenium.

| Exposure | Adjustment | HR (95% CI) | p-value | HR after adding selenium (95% CI) | % attenuation | N | Events |
|---|---|---|---|---|---|---|---|
| Purging (yes vs no) | Age, Sex, BMI | 2.76 (1.17–6.52) | 0.021 | 2.36 (0.99–5.62) | 22.7 | 598 | 35 |
| | Age, Sex, BMI, and past SA | 2.74 (1.17–6.43) | 0.021 | 2.45 (1.04–5.75) | 16.7 | 593 | 35 |
| | Age, Sex, BMI, and EDE-Q score | 2.26 (0.90–5.67) | 0.083 | 2.00 (0.80–4.99) | 15.0 | 581 | 35 |
| Tobacco use (yes vs no) | Age, Sex, BMI | 2.39 (1.28–4.49) | 0.007 | 2.24 (1.20–4.20) | 7.5 | 644 | 40 |
| | Age, Sex, BMI, and past SA | 1.88 (0.84–4.21) | 0.109 | 1.62 (0.85–3.08) | 23.5 | 639 | 40 |
| | Age, Sex, BMI, and EDE-Q score | 1.86 (0.96–3.60) | 0.067 | 1.78 (0.92–3.45) | 7.1 | 584 | 36 |
| Alcohol/Substance use disorder (yes vs no) | Age, Sex, BMI | 2.46 (1.09–5.57) | 0.031 | 2.14 (0.94–4.85) | 15.4 | 625 | 40 |
| | Age, Sex, BMI, and past SA | 1.41 (0.61–3.25) | 0.418 | 1.35 (0.59–3.11) | 12.5 | 621 | 40 |
| | Age, Sex, BMI, and EDE-Q score | 1.82 (0.70–4.72) | 0.217 | 1.74 (0.67–4.49) | 7.4 | 569 | 36 |

Percent attenuation calculated on the log-HR scale using identical complete-case samples per row.
HR = hazard ratio; CI = confidence interval. % attenuation = (lnHR base – lnHR with selenium) / lnHR base × 100.
Cox models used Efron's method for ties; proportional hazards assumption confirmed by Schoenfeld tests. Continuous covariates were mean-centered. Selenium modeled as a z-score.

**Table 4**
Associations between risk behaviors and baseline selenium levels.

| Exposure (independent) | Adjustment | β (SE), z-selenium | 95% CI | p-value | q-value |
|---|---|---|---|---|---|
| Purging (yes vs no) | Age, Sex, BMI | −0.30 (0.09) | −0.48 to −0.13 | <0.001 | 0.003 |
| | Age, Sex, BMI, EDE-Q score | −0.20 (0.10) | −0.39 to −0.01 | 0.048 | 0.072 |
| Tobacco use (yes vs no) | Age, Sex, BMI | −0.22 (0.08) | −0.38 to −0.06 | 0.008 | 0.012 |
| | Age, Sex, BMI, EDE-Q score | −0.21 (0.09) | −0.38 to −0.05 | 0.012 | 0.036 |
| Alcohol/ Substance use disorder (yes vs no) | Age, Sex, BMI | −0.36 (0.14) | −0.65 to −0.08 | 0.012 | 0.012 |
| | Age, Sex, BMI, EDE-Q score | −0.27 (0.15) | −0.57 to 0.03 | 0.074 | 0.074 |

Dependent variable: z-standardized selenium concentration. β coefficients represent the mean difference in selenium (in SD units) between exposed and non-exposed participants. Models adjusted for age, sex, and BMI; EDE-Q score added in secondary models. P-values are two-sided. q-values control the false discovery rate across the three exposures per adjustment set (Benjamini–Hochberg).
Abbreviations: BMI, body mass index, EDE-Q, Eating Disorder Examination-Questionnaire.

also consistent in sensitivity analyses using a clinical deficiency threshold (≤70 μg/L), restricting analyses to women, excluding extreme selenium values, and in exploratory dose–response analyses indicating lower hazards at higher selenium quartiles. Exploratory attenuation analyses further suggested modest attenuation of the estimated associations between selected risk behaviors (purging, tobacco use, and alcohol/substance use disorder) and subsequent SA after inclusion of selenium, without establishing mediation.

These findings extend accumulating evidence linking selenium status to mental health outcomes and suicidal phenomena. We previously reported a cross-sectional association between selenium deficiency and prior SA in AN (Strumila et al., 2022). Population-based studies have reported inverse associations between selenium intake or status and depressive symptoms and related outcomes (Chen et al., 2023; Da et al., 2025; Ferriani et al., 2022; Pasco et al., 2012), and meta-analyses suggest inverse associations between selenium intake and depression and possible benefits of supplementation (Ding and Zhang, 2022; Wang et al., 2023). Higher dietary selenium intake has also been prospectively associated with lower suicidal ideation (Liu and Chen, 2025). The present study adds to this literature by examining selenium in relation to clinically ascertained SA over extended longitudinal follow-up in a clinically high-risk eating disorder cohort.

Plasma selenium is a state-dependent measure that can be influenced by acute nutritional intake, illness acuity, and inflammatory redistribution (Braunstein et al., 2020; Rock and Moos, 2009). In line with this interpretation, the selenium association persisted but was modestly attenuated after adjustment for lnCRP and albumin, suggesting that part of the association may reflect broader vulnerability states (e.g., malnutrition and inflammatory burden) rather than a specific etiologic effect. The association also remained robust after adjustment for plasma zinc and in models incorporating iron indices (transferrin saturation with lnCRP and additional adjustment for ferritin), supporting the view that selenium captures risk-relevant variance not fully explained by these correlated nutritional/inflammatory measures. Exploratory joint selenium–zinc analyses using median splits did not identify a distinct "low selenium + low zinc" profile associated with subsequent SA compared with other participants, suggesting that combined micronutrient patterns may be subtle and may require larger samples and repeated measures to characterise reliably.

Several biological pathways provide plausible hypotheses for why selenium status may be associated with SA risk, although these mechanisms remain hypothesis-generating in the context of observational data. Selenium is required for selenoproteins such as glutathione peroxidases and thioredoxin reductases, which regulate oxidative balance and immune function (Burk and Hill, 2015; Rayman, 2012). Deficiency may increase vulnerability to oxidative and nitrosative stress pathways implicated in SB (Lengvenyte and Courtet, 2025; Meng et al., 2023; Vasupanrajit et al., 2022). Emerging work also links selenium to mitochondrial redox regulation, RNA methylation, and ferroptosis suppression through GPX4 (Alim et al., 2019; Ingold et al., 2018; Lan et al., 2025; Lee et al., 2024; Li et al., 2023). Selenium deficiency has been associated with altered dopaminergic signalling in animal models (Solovyev, 2015), which may be relevant in eating disorders where

reward processing and decision-making are disrupted and oxidative stress is elevated (Solmi et al., 2015). Dopaminergic neurons are particularly vulnerable to oxidative stress (Qi et al., 2010). Inflammatory and oxidative stress alterations have also been repeatedly associated with SB (Lengvenyte et al., 2024; Lengvenyte and Courtet, 2025; Vasupanrajit et al., 2022), including evidence of NOX2 upregulation in suicide decedents (Schiavone et al., 2016), and increased nitric oxide metabolites and lipid peroxidation in suicide attempters (Odebrecht Vargas et al., 2013). Selenium supplementation has been reported to downregulate inflammatory mediators (e.g., TNF-α) and enhance antioxidant defences (Jamilian et al., 2018; Zhang et al., 2002), and vascular homeostasis disturbances have been implicated in SB (Lengvenyte and Courtet, 2025). These pathways warrant direct testing in future studies.

### 4.1. Strengths and limitations

Strengths of this study include its relatively large sample size for an eating disorders cohort, its prospective design with over six years of mean observation period, and its detailed psychiatric characterization. The use of electronic Emergency Department records minimized recall bias and allowed objective ascertainment of SA (Lengvenyte et al., 2023; Pellatt et al., 2025). The selenium association was consistent across multiple model specifications and sensitivity analyses, supporting robustness.

However, several limitations warrant consideration. First, selenium was measured once at baseline. Because plasma selenium is state-dependent, repeated measures and functional selenium indices (e.g., selenoprotein P) would strengthen interpretation (Braunstein et al., 2020; Rock and Moos, 2009). Second, causal inference is not possible in this observational design. Residual confounding by unmeasured dietary patterns, socioeconomic factors, or other nutritional exposures, such as omega-3 fatty acids, which are correlated with selenium and may independently influence SB risk, may remain (Lewis et al., 2011; Wang et al., 2024). Sensitivity analyses using *E*-values suggested that the observed associations between lower selenium and increased risk of SA were moderately robust to unmeasured confounding, suggesting that residual confounding cannot be fully excluded and need further studies. Third, the number of SA ($n = 40$) limited precision for subgroup analyses and more complex modelling (Narita et al., 2025). Fourth, the cohort was predominantly female and recruited from a single specialized centre, limiting generalizability to men and to community settings. Fifth, SA were captured within a single hospital system; events occurring outside the catchment area may have been missed, which would likely bias associations toward the null. Sixth, although quartile analyses supported approximate linearity, given evidence for U-shaped associations between selenium and cardiometabolic outcomes and selenium toxicity to dopaminergic neurons at high levels (Naderi et al., 2018; Rayman, 2012; Zhou et al., 2020), both deficiency and excess may be associated with unfavourable outcomes, any supplementation should be approached cautiously and evaluated in interventional designs.

## 5. Conclusion

Lower baseline plasma selenium was associated with higher subsequent SA risk in individuals with eating disorders. The persistence of this association after contextual adjustment for inflammatory, nutritional, and trace-element measures supports interpretation of selenium as a risk correlate that may integrate aspects of nutritional instability and inflammatory state. Replication in larger and more diverse cohorts is needed, ideally incorporating repeated selenium measurements, functional selenium indices, and multi-marker nutritional/inflammatory profiling. Interventional studies would be required before drawing conclusions about whether selenium repletion can modify SB risk. Until then, selenium may be considered as part of comprehensive nutritional and medical assessment in high-risk eating disorder populations, interpreted in clinical context.

## CRediT authorship contribution statement

**Aiste Lengvenyte:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Conceptualization. **Robertas Strumila:** Writing – review & editing, Investigation, Conceptualization. **Maude Seneque:** Writing – review & editing, Project administration, Investigation. **Philippe Courtet:** Writing – review & editing, Supervision, Resources. **Sebastien Guillaume:** Writing – review & editing, Supervision, Resources, Investigation, Conceptualization.

## Ethical statement

Ethics approvals for the studies were granted by the Comité de Protection des Personnes Sud Méditerranée IV (approval number 11–04-SC, first cohort) and by the CPP Sud-Est 6 (approval number AU13–13, second cohort), allowing subsequent analyses. All participants, or their legal representatives for minors, signed the informed consent, and procedures complied with the Declaration of Helsinki.

## Declaration of competing interest

The authors report no actual or potential conflict of interest that could influence or bias this work.

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## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.pnpbp.2026.111672.

## Data availability

Data will be made available on request.

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