

Nighttime Light Exposure and Frailty: A Large Sample Cross-Sectional Study

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Research Article

Keywords: Outdoor light pollution, Nighttime light, Frailty, Cross-sectional study, older adults

Posted Date: June 18th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9850380/v1>

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Additional Declarations: No competing interests reported.

Abstract

Background

Nighttime light (NTL) serves as a proxy for outdoor light pollution and has been linked to various chronic diseases. Physical frailty, a prevalent geriatric syndrome characterized by decreased physiological reserve and increased vulnerability to stressors, poses a significant public health challenge among adults. However, evidence linking NTL exposure to physical frailty in aging populations is currently lacking.

Methods

This cross-sectional study analyzed data from 366,813 participants in the UKB (UK Biobank). Mixed-effects models examined the association between NTL and frailty phenotype, while multivariable logistic regression assessed relationships with individual frailty components (low physical activity, weight loss, self-reported exhaustion, low grip strength, and slow gait speed). Restricted cubic splines (RCS) were used to evaluate non-linear associations.

Results

After full covariate adjustment, higher NTL levels were associated with increased risks of pre-frailty (OR = 1.02, 95% CI:1.01–1.03) and frailty (OR = 1.06, 95% CI:1.03–1.08). Higher NTL levels were associated with an increased risk of low physical activity (OR = 1.08, 95% CI 1.06–1.09), low grip strength (OR = 1.08, 95% CI 1.07–1.09), and slow gait speed (OR = 1.03, 95% CI 1.02–1.05). RCS revealed an L-shaped non-linear relationship between NTL and frailty phenotype.

Conclusions

Higher NTL exposure was associated with elevated frailty risk, particularly affecting physical function components. There are policy implications for optimizing outdoor lighting regulations to mitigate frailty risk in ageing populations.

Introduction

As the global population ages, frailty—a geriatric syndrome characterized by reduced physiological reserve across multiple systems and increased vulnerability to stressors—has become a significant public health challenge ^[1–3]. Among the array of frailty instruments, the frailty phenotype stands as a frequently used operational definition, its five-item criterion set: unintentional weight loss, self-reported exhaustion, low grip strength, slow walking speed, and low physical activity explicitly captures vulnerability to

physiological decompensation and thus distinguishes the construct from related entities such as multimorbidity^[4–7].

Extensive studies have demonstrated that frailty significantly elevates the risk of multiple adverse health outcomes, including disability, falls, morbidity, and mortality, as well as being associated with depression, cardiovascular disease, and dementia^[8–11]. Given that frailty is a reversible clinical condition^[12, 13], early identification of vulnerable individuals is paramount. Previous research has focused primarily on individual-level determinants such as vitamin D deficiency^[7], depression^[14], dementia^[9, 15], and multimorbidity^[16], whereas the role of the broader built environment remains under-explored.

Rapid urbanization has markedly increased NTL, prolonging the natural light–dark cycle^[17]. NTL serves as an indicator of outdoor light pollution: Epidemiological studies have linked light pollution to obesity, insulin resistance, hypertension, and depression^[18–20]. However, whether NTL is associated with frailty has not been tested in large-scale adult populations.

To address this gap, we used UKB data to examine two key questions: Does higher NTL exposure independently predict pre-frail/frail status? Is NTL differentially associated with each of the five frailty components?

Study population

This cross-sectional study used data from participants enrolled in the UKB. The UKB data are publicly available at <https://www.ukbiobank.ac.uk>. This large-scale biomedical repository contains detailed phenotypic, genetic, and linked health-record information on more than 500,000 residents aged 37–73 years, recruited from England, Scotland, and Wales. Data on sociodemographic, lifestyle, and health were gathered through touchscreen surveys, interviews, and physical and biological assessments. Health outcomes were monitored by connecting to external databases, such as hospital admissions and national death records. The study received ethical approval from the North West Multi (16/NW/0382), and participants provided informed consent, in accordance with the Helsinki Declaration. Data were accessed under UKB application ID 98124.

Frailty

In this study, frailty was operationalized with The Fried frailty phenotype. This instrument scores five binary criteria^[5]: unintentional weight loss, self-reported exhaustion, low grip strength, low physical activity, and slow gait speed, yielding a summary score of 0–5. Participants were categorized into three groups based on their fulfilment of the criteria: robust (score = 0), pre-frail (score = 1–2), or frail (score = 3–5).

Assessment of NTL

The NTL data for 2010 were acquired from the National Earth System Science Data Center (<http://www.geodata.cn>). The dataset comprises oversaturation-corrected and time-series-corrected DMSP-OLS NTL data, annual synthetic data obtained by fusing NPP-VIIRS monthly synthetic data, laboratory-calibrated DMSP-OLS NTL products, and demographic information, all processed into a new 500 m resolution NTL dataset expressed in $\text{nW cm}^{-2} \text{ sr}^{-1}$. A total of 150,000 randomly selected pixels and 40,000 cities worldwide served as validation areas; comparison with the corresponding NPP-VIIRS annual synthetic data yielded accuracies of 0.87 at the pixel scale and 0.95 at the city scale.^[21] Participants' addresses at 100 m resolution were then aligned with the NTL exposure raster

It should be noted that NTL differs from light at night (LAN), which typically refers to individual-level exposure to artificial light sources. While LAN measurements provide detailed information at the individual level, NTL offers several unique advantages: (1) it provides objective, standardized measurements at the population level; (2) it captures long-term, spatially resolved exposure patterns; and (3) it is particularly suitable for large-scale epidemiological studies, where individual-level light monitoring is impractical.

Covariates

We considered age (years), sex (male, female), race, body mass index (BMI), average household income per year, smoking status, alcohol consumption status, $\text{PM}_{2.5}$, and NO_x . Specifically, race was classified as White and non-White. Household income was categorised as <£18000, £18000 to £100000, or >£100000.

Statistical analysis

Descriptive characteristics are reported using means and standard deviations (SDs) for quantitative variables that follow a normal distribution. For variables that were not normally distributed, medians and interquartile ranges (IQRs) were used. Categorical variables are presented as frequencies and percentages.

To investigate the association between NTL and individual frailty phenotype, a mixed-effects model was employed. Multivariable logistic regression was applied to examine the relationship between NTL and each component of the frailty phenotype. Three models were fitted with robust participants as the reference. Model 1: adjusted for demographics (age, sex, race, income, residence). Model 2: further adjusted for lifestyle factors (smoking, alcohol consumption, and BMI). Model 3: fully adjusted model adding $\text{PM}_{2.5}$, NO_x , diabetes, and hypertension. To examine the non-linear relationships between NTL and frailty phenotype, we employed RCS analysis. We further performed subgroup analyses to examine whether the association between NTL and frailty phenotypes differed by sex (male vs female), residence (urban vs rural), and age (< 60 vs $\geq$ 60 years).

Several sensitivity analyses were conducted: First, we categorized NTL into quartiles (Q1-Q4) and performed regression analyses. Second, we used segmented regression to determine the breakpoint at which the association changed significantly. All statistical analyses were conducted using R software (Version 4.1.2). $P < 0.05$ was considered statistically significant.

Results

1. Baseline characteristics of the study population

1. Baseline characteristics of the study population

After excluding participants with missing NTL and frailty phenotype data, 366,813 individuals remained for analysis. Among them, 10,291 (2.81%) were classified as frail and 133,560 (36.41%) as pre-frail. Table 1 summarises the baseline characteristics: frail participants were older (58.15 ± 7.64), more likely to be male (61.3%), and exposed to higher levels of $PM_{2.5}$ ($10.3 \pm 1.12 \mu g m^{-3}$) and NO_2 ($28.74 \pm 7.55 \mu g m^{-3}$). They also had higher prevalence rates of obesity, hypertension, and diabetes (all $P < 0.001$).

2. Association between NTL and frailty phenotype

Compared with non-frail individuals, higher NTL levels were associated with a higher risk of pre-frailty (OR=1.06, 95% CI 1.05-1.07) and frailty (OR=1.15, 95% CI 1.13-1.18) in Model 1. These associations were slightly attenuated but remained statistically significant after further adjustment for all covariates in Model 3: pre-frail: OR=1.02, 95% CI 1.01-1.03; frail: OR=1.06, 95% CI 1.03-1.08 (Table 2).

3. Association of NTL with components of the frailty phenotype.

In Model 3 (fully adjusted), higher NTL levels were associated with an increased risk of low physical activity (OR=1.08, 95% CI 1.06-1.09), low grip strength (OR=1.08, 95% CI 1.07-1.09), and slow gait speed (OR=1.03, 95% CI 1.02-1.05) (Table 3).

4. Non-linear relationship between NTL and frailty phenotype.

Figure 1A illustrates the non-linear 'L-shaped' relationship between NTL and the risk of pre-frailty, with a cutoff point at $13.403 nW cm^{-2} sr^{-1}$, indicating a higher risk of pre-frailty above this threshold. Similarly, Figure 1B shows a non-linear 'L-shaped' pattern between NTL and the risk of frailty, with a cutoff point at $8.908 nW cm^{-2} sr^{-1}$, above which the risk of frailty is higher.

5. stratified analyses.

Stratified analyses showed consistent positive associations between NTL and both pre-frailty and frailty in most subgroups (Table 4). Notably, the association with frailty became non-significant among rural residents after full adjustment (OR=1.05, 95% CI 0.95-1.16, $P=0.347$), whereas it remained significant among urban residents (OR=1.06, 95% CI 1.04-1.09, $P<0.001$).

6. Sensitivity analyses.

Sensitivity analyses confirmed the robustness of our findings. First, categorizing NTL into quartiles showed the highest quartile associated with increased odds compared to the lowest (pre-frailty: OR=1.12, 95% CI 1.10-1.15; frailty: OR=1.25, 95% CI 1.14-1.37) (Table S1). Second, segmented regression identified significant threshold effects at 8.40 nW cm⁻² sr⁻¹ for frailty and 12.38 nW cm⁻² sr⁻¹ for prefrailty (Table S2).

Discussion

Leveraging a large population cross-sectional sample, we examined the association between NTL and frailty. The key findings are as follows: (1) Pre-frailty was common (36.41%), while frailty was present in 2.81% of participants; (2) Higher NTL levels were associated with increased risks of both pre-frailty and frailty; (3) NTL was associated with specific components of the frailty phenotype (low physical activity, low grip strength, and slow gait speed). (4) a significant urban–rural disparity in the association between NTL and frailty.

This study examined the association between NTL and frailty. Our findings align with the growing body of evidence on the adverse health effects of light pollution. Multiple studies have shown that disruption of circadian rhythms caused by light at night (LAN) can contribute to adverse health outcomes [22, 23]. A longitudinal study involving more than 1,000 elderly participants found that circadian rhythm disruptions were significantly associated with an elevated risk of frailty, suggesting that these disturbances may serve as early indicators or risk factors for frailty in older populations. [24], Moreover, Kim et al. [25] documented a high prevalence of nighttime light exposure among community-residing older adults, with significant associations observed between LAN and obesity, diabetes, and hypertension. Zhang et al. [26] analyzed the UK Biobank cohort and found that NTL significantly mediated the relationship between air pollution exposure and cardiovascular disease risk.

Of note, we observed a significant urban–rural disparity in the association between NTL and frailty. While positive associations persisted among urban residents across all models, they were substantially attenuated and became statistically non-significant among rural residents after full adjustment. This finding may suggest that the health effects of light pollution may be context-dependent. Evidence indicates that urban residents are exposed to higher-intensity light pollution, whereas rural areas typically have lower ambient light pollution levels [26, 27]. Thus, the observed urban–rural disparity may be partly attributable to differences in light pollution exposure or to other unmeasured factors.

As urbanization accelerates, the brightness of nighttime electric lighting is increasing by roughly 10% per year [15]. While this illumination supports public safety and extends outdoor activity, it also disrupts natural light-dark cycles. NTL exposure may contribute to frailty development through multiple biological pathways, primarily via disruption of circadian rhythms.

Light signals travel along the retino-hypothalamic pathway to suppress pineal melatonin secretion and to disturb the central clock, consequently delaying the molecular clocks of peripheral tissues, such as

skeletal muscle^[28, 29]. The BMAL1-CLOCK circuit constitutes the core molecular mechanism of the mammalian circadian rhythm system, formed by a heterodimer of two transcription factors: BMAL1 (brain and muscle aromatase receptor-like protein 1) and CLOCK (circadian output cyclin). In skeletal muscle, the BMAL1-CLOCK circuit directly regulates the expression of myogenic factors such as MyoD, maintaining the rhythmic balance between protein synthesis and degradation in muscle fibers.^[30–32] When the circadian-skeletal-muscle axis is misaligned, proteolysis outpaces synthesis and can precipitate declines in grip strength and gait speed^[24, 33, 34]—core components of the frailty phenotype. Animal studies have demonstrated that NTL exposure interferes with circadian rhythms and metabolic processes, leading to weight gain, impaired glucose tolerance, and altered feeding behavior. These effects are primarily attributed to melatonin suppression, which plays a crucial role in regulating circadian rhythms and providing antioxidant, anti-inflammatory, and vasodilatory effects^[35, 36]—all of which may contribute to the development of frailty through multiple pathways.

The current study has several strengths: 1. First evidence on NTL-frailty association: This is the first study to establish a link between outdoor light pollution (measured by NTL) and frailty. 2. Large and representative sample: Our analysis included 366,813 participants from the UK Biobank, providing robust statistical power and enhancing the generalizability of findings. 3. Urban-rural disparity identified: We observed a significant difference in the NTL-frailty association between urban and rural areas, which adds novel insights into environmental health disparities. 4. Comprehensive frailty assessment: The standardized frailty phenotype captured multiple component-specific analyses (low physical activity, grip strength, and gait speed), which helped elucidate potential mechanistic pathways.

Several limitations should be acknowledged: 1. Cross-sectional design: The temporal relationship between NTL exposure and frailty cannot be determined, precluding causal inference. 2. Limited generalizability: As most participants were of European ancestry, caution is warranted when extrapolating findings to other populations. 3. Volunteer bias: The UK Biobank cohort is known to be healthier than the general population, potentially underestimating the true prevalence of frailty. 4. Residual confounding: Despite extensive covariate adjustment, unmeasured factors and reverse causation cannot be completely ruled out.

Conclusions

Our study provides novel evidence that exposure to outdoor light pollution, as measured by NTL, is associated with increased frailty risk in a large adult population. We observed a relationship between higher NTL levels and increased likelihood of both pre-frailty and frailty, suggesting that exposure to NTL may contribute to accelerated physiological decline. The associations were particularly evident for specific frailty components, including low physical activity, low grip strength, and slowed gait speed, highlighting potential pathways through which light pollution may impact physical function. Furthermore, the urban-rural disparity in this association underscores the need for targeted interventions in urban settings where light pollution is more prevalent. These findings call for further research to elucidate the

underlying mechanisms and inform public health strategies to mitigate the adverse effects of light pollution on populations.

Declarations

Author contributions

Y.D. and J.H. contributed to the study concept and design. Y.W. and D.N. contributed to the acquisition of data. H.C and K.T contributed to the analysis and interpretation of data. J.Z., Y.Y., S.Y., and L.C. contributed to the drafting of the manuscript. Y.D. and L.W. contributed to the critical revision of the manuscript for important intellectual content. All authors reviewed and approved the final manuscript.

Competing interests

All authors declare no competing interests.

Funding

This work was supported by the Key R&D projects in Ningxia (grant number 2024BEH04011) and Natural Science Foundation of Ningxia (grant number 2025AAC030727).

Acknowledgments

We thank all investigators and participants of the UK Biobank. Acknowledgement for the nighttime light data support from the National Earth System Science Data Center, National Science & Technology Infrastructure of China.

Data availability

Data availability UK Biobank is an open-access resource ([https:// www. ukbiobank.ac.uk/](https://www.ukbiobank.ac.uk/)). All bona fide researchers can apply to use its data for health-related research that is in the public interest ([http:// www. ukbiobank.ac.uk/register- apply](http://www.ukbiobank.ac.uk/register-apply)).

Ethics approval and consent to participate

The study was approved by both the National Health Service National Research Ethics Service (Ref.: 11/NW/0382) and the Institutional Review Board of Tulane University (2018–1872). All participants gave written informed consent to participate. This research conforms to the Declaration of Helsinki.

Clinical trial number

Not applicable.

Consent for publication

Not applicable.

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Tables

Table 1
Baseline characteristics of participants

Characteristic	No-frailty	Pre-frailty	Frailty	p-value
	N = 222,962(60.78%)	N = 133,560(36.41%)	N = 10,291(2.81%)	
Age	56.49 ± 8.06	57.06 ± 8.13	58.15 ± 7.64	< 0.001
Sex				< 0.001
male	112,001 (50.23%)	73,621 (55.12%)	6,310 (61.32%)	
female	110,961 (49.77%)	59,939 (44.88%)	3,981 (38.68%)	
Race				< 0.001
White	214950 (96.41%)	124673 (93.3%)	9122 (88.61%)	
Non-White	7561 (3.44%)	8575 (6.42%)	1133 (11.0%)	
BMI	26.62 ± 4.16	28.36 ± 5.15	31.37 ± 6.77	< 0.001
Hypertension, n(%)				< 0.001
No	146,370 (65.65%)	72,315 (54.14%)	3,469 (33.71%)	
Yes	76,592 (34.35%)	61,245 (45.86%)	6,822 (66.29%)	
Residence, n (%)				< 0.001
Rural	18,328 (8.22%)	8,155 (6.11%)	356 (3.46%)	
Urban	204,634 (91.78%)	125,405 (93.89%)	9,935 (96.54%)	
Smoking, n (%)				< 0.001
No	125,139 (56.13%)	69,857 (52.30%)	4,664 (45.32%)	
Yes	97,823 (43.87%)	63,703 (47.70%)	5,627 (54.68%)	
Drinking, n (%)				< 0.001
No	30,534 (13.69%)	30,484 (22.82%)	4,624 (44.93%)	
Yes	192,428 (86.31%)	103,076 (77.18%)	5,667 (55.07%)	
Diabetes, n (%)				< 0.001
No	216,618 (97.15%)	123,501 (92.47%)	8,243 (80.10%)	
Yes	6,344 (2.85%)	10,059 (7.53%)	2,048 (19.90%)	
Income, n (%)				< 0.001
<£18000	39,051 (17.51%)	36,989 (27.69%)	5,732 (55.70%)	

Characteristic	No-frailty	Pre-frailty	Frailty	p-value
	N = 222,962(60.78%)	N = 133,560(36.41%)	N = 10,291(2.81%)	
£18000 to £100000	169,389 (75.97%)	91,136 (68.24%)	4,455 (43.29%)	
>£100000	14,522 (6.51%)	5,435 (4.07%)	104 (1.01%)	
PM _{2.5}	9.92 ± 1.04	10.05 ± 1.06	10.30 ± 1.12	< 0.001
NO _x	26.07 ± 7.55	27.06 ± 7.6	28.74 ± 7.78	< 0.001
NTL	11.32 ± 10.16	12.45 ± 10.58	14.49 ± 11.76	< 0.001

Table 2
Association between NTL and frailty phenotype.

Frailty phenotype	No-frailty	Pre-frailty	p-value	Frailty	p-value
Model 1, OR (95% CI)	1 (Reference)	1.06 (1.05–1.07)	< 0.001	1.15 (1.13–1.18)	< 0.001
Model 2, OR (95% CI)	1 (Reference)	1.05 (1.04–1.06)	< 0.001	1.13 (1.11–1.15)	< 0.001
Model 3, OR (95% CI)	1 (Reference)	1.02 (1.01–1.03)	< 0.001	1.06 (1.03–1.08)	< 0.001
Model 1: Adjust: age, income, sex, residence, race.					
Model 2: Adjust: age, income, sex, residence, race, smoking, alcoholism, BMI.					
Model 3: Adjust: age, income, sex, residence, race, smoking, alcoholism, BMI. PM _{2.5} , NO _x , diabetes, hypertension					

Table 3
Association of NTL with components of the frailty phenotype.

Frailty phenotype	No-frailty	Pre-frailty	<i>p</i>-value	Frailty	<i>p</i>-value
Model 1, OR (95% CI)	1 (Reference)	1.06 (1.05–1.07)	< 0.001	1.15 (1.13–1.18)	< 0.001
Model 2, OR (95% CI)	1 (Reference)	1.05 (1.04–1.06)	< 0.001	1.13 (1.11–1.15)	< 0.001
Model 3, OR (95% CI)	1 (Reference)	1.02 (1.01–1.03)	< 0.001	1.06 (1.03–1.08)	< 0.001
Model 1: Adjust: age, income, sex, residence, race.					
Model 2: Adjust: age, income, sex, residence, race, smoking, alcoholism, BMI.					
Model 3: Adjust: age, income, sex, residence, race, smoking, alcoholism, BMI. PM _{2.5} , NO ₂ , diabetes, hypertension					

Table 4
stratified analyses

Frailty phenotype	No-frailty	Pre-frailty	p-value	Frailty	p-value
Male					
Model 1, OR (95% CI)	1 (Reference)	1.05 (1.04–1.06)	< 0.001	1.14 (1.11–1.17)	< 0.001
Model 2, OR (95% CI)	1 (Reference)	1.04 (1.02–1.05)	< 0.001	1.11 (1.08–1.14)	< 0.001
Model 3, OR (95% CI)	1 (Reference)	1.01 (1.00–1.03)	0.035	1.06 (1.03–1.09)	0.0002
Female					
Model 1, OR (95% CI)	1 (Reference)	1.07 (1.06–1.08)	< 0.001	1.16 (1.13–1.20)	< 0.001
Model 2, OR (95% CI)	1 (Reference)	1.06 (1.05–1.08)	< 0.001	1.15 (1.11–1.18)	< 0.001
Model 3, OR (95% CI)	1 (Reference)	1.03 (1.02–1.05)	< 0.001	1.06 (1.02–1.10)	0.001
Age45-60					
Model 1, OR (95% CI)	1 (Reference)	1.05 (1.04–1.06)	< 0.001	1.12 (1.09–1.15)	< 0.001
Model 2, OR (95% CI)	1 (Reference)	1.04 (1.03–1.06)	< 0.001	1.10 (1.07–1.13)	< 0.001
Model 3, OR (95% CI)	1 (Reference)	1.02 (1.01–1.03)	0.001	1.05 (1.02–1.08)	0.003
Age ≥ 60					
Model 1, OR (95% CI)	1 (Reference)	1.07 (1.05–1.08)	< 0.001	1.17 (1.14–1.21)	< 0.001
Model 2, OR (95% CI)	1 (Reference)	1.05 (1.04–1.07)	< 0.001	1.14 (1.11–1.17)	< 0.001
Model 3, OR (95% CI)	1 (Reference)	1.03 (1.01–1.04)	< 0.001	1.07 (1.03–1.10)	< 0.001
Urban					
Model 1, OR (95% CI)	1 (Reference)	1.06 (1.05–1.07)	< 0.001	1.15 (1.13–1.17)	< 0.001
Model 2, OR (95% CI)	1 (Reference)	1.05 (1.04–1.06)	< 0.001	1.12 (1.10–1.15)	< 0.001
Model 3, OR (95% CI)	1 (Reference)	1.02 (1.01–1.03)	< 0.001	1.06 (1.04–1.09)	< 0.001
Rural					
Model 1, OR (95% CI)	1 (Reference)	1.08 (1.05–1.11)	< 0.001	1.13 (1.04–1.22)	0.004
Model 2, OR (95% CI)	1 (Reference)	1.07 (1.04–1.10)	< 0.001	1.06 (0.97–1.17)	0.181
Model 3, OR (95% CI)	1 (Reference)	1.06 (1.03–1.09)	< 0.001	1.05 (0.95–1.16)	0.347

Figures

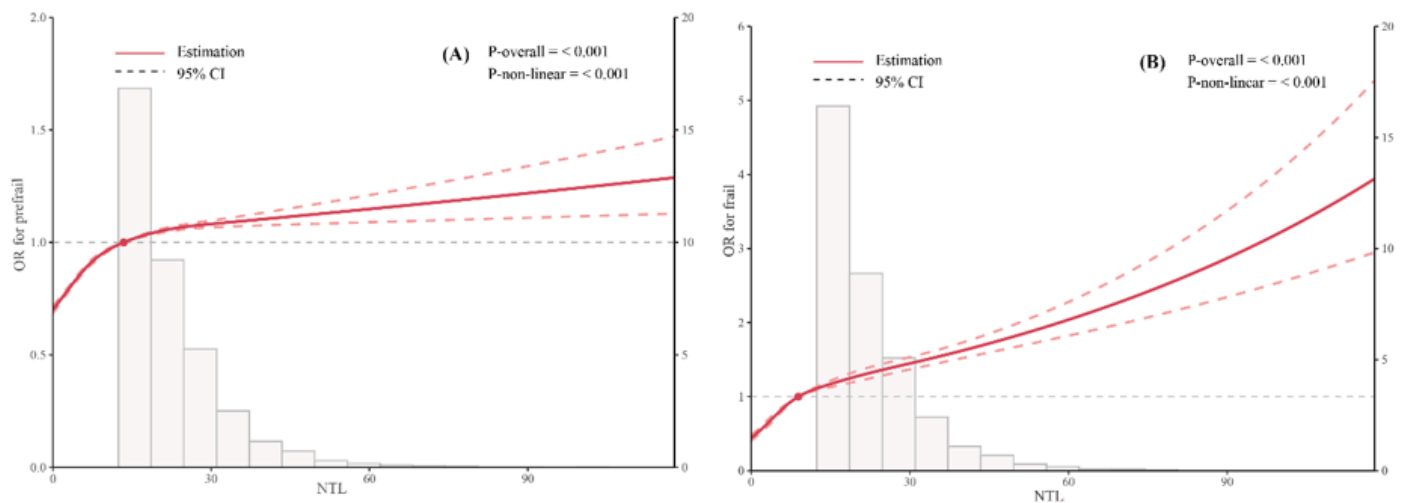


Figure 1

RCS of the association between NTL and frailty status. (A) For prefrail: The cutoff point at 13.403 nW cm⁻² sr⁻¹. (B) For frail: The cutoff point at 8.908 nW cm⁻² sr⁻¹.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Supplementarydata.docx](#)