

Apparent Protection, Uncertain Harm: An Estimand Decomposition of Internet Use and Late-Life Depressive Symptoms in a National Cohort

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Abstract

Background

Digital inclusion is widely promoted as a mental-health prevention strategy for middle-aged and older adults, yet the evidence is inconsistent and prone to selection bias, reverse causation and other methodological problems. We tested whether any protective estimate survives methods that address several of these biases.

Methods

We reanalysed the China Health and Retirement Longitudinal Study (CHARLS, 2011–2018) among community-dwelling adults aged ≥ 45 years who reported no internet use at baseline and scored below 10 on the 10-item Center for Epidemiologic Studies Depression Scale (CES-D-10). We estimated the association under several designs – target-trial emulation with clone-censor-weighting and inverse probability of censoring weighting, within-person fixed-effects models with lead and placebo-lead tests of temporal direction, and linear mixed models – and probed the estimates with negative-control outcomes and E-values for unmeasured confounding. The primary outcome was annual CES-D-10 change; the secondary, incident elevated depressive symptoms (CES-D-10 ≥ 10).

Results

Cross-sectionally, internet users appeared less depressed (crude difference – 3.07 points), but the association attenuated with covariate adjustment and was null under within-person fixed effects (+ 0.096; 95% confidence interval [CI] – 0.148 to 0.341; $p = 0.440$). A linear mixed model found symptoms rising slightly faster among users (+ 0.094 points/year; 95% CI 0.014–0.174; $p = 0.021$). In target-trial emulation, initiators had a lower hazard of elevated symptoms (per-protocol hazard ratio 0.699; 95% CI 0.559–0.875). Within-person lead analyses pointed in the opposite direction: internet use preceded later symptom increases (+ 0.50; $p = 0.01$), the placebo-lead was null ($p = 0.68$), and the estimate was largest where reverse causation was least plausible (baseline CES-D-10 < 5 ; +0.83; $p < 0.001$). The negative-control outcome, arthritis, showed the same protective direction (adjusted odds ratio 0.51–0.75) – a generic selection artefact rather than a depression-specific causal pathway.

Conclusions

The protective association between internet use and lower depressive symptoms is a selection artefact, mirrored by a negative-control outcome. A modest within-person residual, directionally consistent across designs, is compatible with harm but fragile to unmeasured time-varying confounding (robustness values < 0.005). Internet use should not be recommended to prevent depressive symptoms, and digital-

inclusion programmes should not be promoted on selection-confounded evidence — not because harm is proven, but because protection is not.

Key Message

1. In a nationally representative cohort of middle-aged and older Chinese adults, the widely reported protective association between internet use and lower depressive symptoms did not survive within-person (fixed-effects) estimation, and it was closely mirrored by a negative-control outcome (arthritis) that internet use could not plausibly protect against. The apparent benefit is better explained by healthy-user selection than by a real protective effect.
2. Once between-person selection was removed, a modest within-person residual remained, directionally compatible with harm (+0.094 points per year on the CES-D-10 scale): the lead effect was significant, the placebo-lead was null and the estimate was strongest where reverse causation was least plausible. This residual was statistically fragile, however (Cinelli–Hazlett robustness values <0.005), and cannot support a causal interpretation.
3. Internet use should not be recommended to prevent depressive symptoms, and digital-inclusion programmes should not be promoted on selection-confounded evidence.
4. For self-selected lifestyle exposures, within-person designs combined with negative-control outcomes should be the default methodological audit of an apparently protective association, not a sensitivity check run as an afterthought.

Introduction

Policy documents increasingly frame digital technologies as mental-health interventions for middle-aged and older adults.^{1,2} The World Health Organization (WHO) Decade of Healthy Ageing (2021–2030) identifies digital inclusion as a strategic priority,¹ and national programmes—China's "Smart Aging" initiative, the European Union (EU) Digital Decade, and digital-skills programmes in the United States (US)—operate on the premise that internet use protects against depressive symptoms in middle and later life.^{3,4} This carries tangible opportunity costs: resources allocated to digital-literacy training are unavailable for direct psychological services or social-support interventions with established evidence bases. Given the high prevalence of depressive symptoms in middle-aged and older adults and their association with disability and excess mortality, whether a widely promoted intervention truly prevents them is of direct public-health importance.

The empirical foundation, however, is remarkably inconsistent. Observational studies of internet use and depressive symptoms in middle-aged and older adults have produced estimates spanning the full spectrum—from apparent protection through null findings to signals of harm.^{5–11} This degree of inconsistency is not the signature of a robust causal effect; it is the signature of a literature corrupted by bias.¹²

Three methodological problems likely account for this. First, selection bias: internet users are younger, more educated, wealthier, and healthier;¹³ in the China Health and Retirement Longitudinal Study (CHARLS), the standardised mean difference for education exceeds 1.0 at every wave. The confounders driving this selection—digital literacy, motivation, social support, personality—are largely unmeasured and time-invariant, making them difficult to address through standard covariate adjustment.^{14,15} Second, reverse causation. Depressive symptoms may reduce internet use (withdrawal) or drive compulsive use (escapism); the net association depends on which pathway dominates.^{16–18} Third, exposure misclassification: items are operationalised inconsistently across studies, and in longitudinal surveys missing responses are frequently treated as non-use, generating measurement artifacts. Few prior studies have addressed all three problems simultaneously. Most rely on cross-sectional or naive longitudinal regressions that cannot, in principle, separate causation from selection.¹² We therefore treated this as a methodological audit: rather than asking whether internet use prevents depressive symptom burden, we asked whether any protective estimate survives methods that address selection bias, reverse causation and other methodological problems—and if not, what the rigorous estimates actually show.

We did not set out to test whether the internet prevents depression. The more fundamental problem is that the available designs do not estimate the same thing. Cross-sectional regression compares users with non-users; clone-censor-weighted trial emulation compares initiators with sustained non-users among baseline non-users; fixed effects compare each participant with themselves. The first two are between-person contrasts and inherit the selection problem described above; the third is a within-person contrast that differences out all time-invariant traits, measured or not. Each design rests on its own exchangeability assumption, and for a self-selected exposure these assumptions are not equally credible. When the same exposure then yields estimates of opposite sign depending on the design, the divergence itself carries information — it shows where between-person selection is doing the work, though it cannot prove causation. Figure 1 makes this concrete, placing the estimates side by side: from the crude cross-sectional difference of -3.07 CES-D-10 points, through covariate-adjusted and trial-emulated estimates, to within-person estimates of $+0.10$ to $+0.83$. The protective association collapses as identification moves from between-person to within-person. We therefore treat the within-person estimand as the primary analytical tool rather than a sensitivity check, with negative-control outcomes built into the design from the start. Using the CHARLS, 2011–2018, we asked two questions: whether any protective estimate of internet use on depressive symptoms survives methods that address several of these biases simultaneously; and, if not, what the selection-adjusted estimates actually show.

Methods

Study Population and Data

We drew on the China Health and Retirement Longitudinal Study (CHARLS), a nationally representative survey of community-dwelling Chinese adults aged ≥ 45 years and their spouses. The baseline sample in

2011 covered 28 provinces and enrolled 17,708 participants; later waves added refreshment samples to replace those lost to attrition. Follow-up interviews ran every 2–3 years. We restricted the analysis to waves 2011, 2013, 2015, and 2018. (2020-wave exclusion rationale is reiterated in Supplementary Methods, Study Population note.) Data were collected through face-to-face interviews with standardised questionnaires. The study design and sampling procedures have been described in detail elsewhere.¹³ The Ethics Committee of Peking University approved the protocol, and all participants gave written informed consent. We verified all variables against the CHARLS codebook.

Target-Trial Emulation Protocol

We emulated a target trial using clone-censor-weighting, with 2011 and 2013 as sequential baselines. Participants qualified if they were aged ≥ 45 years, reported no internet use at baseline (CHARLS item da056s10), had a valid CES-D-10 score, and showed no elevated depressive symptoms (score < 10). We contrasted two treatment strategies: initiate internet use within a two-year grace period and sustain it thereafter, or never initiate. We cloned every eligible baseline non-user into both arms, so each person contributed two copies that shared baseline characteristics but were followed under different hypothetical regimes.

The primary outcome was annual change in CES-D-10 score; the secondary was incident elevated depressive symptoms (score ≥ 10). Follow-up extended through 2018, with time measured from baseline. We estimated a per-protocol (PP) effect—censoring clones when they deviated from their assigned strategy—and a modified intention-to-treat (ITT) effect. In the modified ITT, each baseline non-user with a measurable grace-end wave was assigned to one arm according to their observed behaviour at that point (initiator or sustained non-user) and censored only on loss to follow-up. Because both arms of the same person share identical observed outcomes, a true all-clones-retained ITT is unidentified; the modified ITT is therefore the standard approach in clone-censor emulation.

For the slope outcome we fitted weighted linear models; for incident symptoms, weighted Cox proportional hazards models. Inverse probability of censoring weights (IPCW) addressed informative censoring from both attrition and strategy deviation, and we calculated cluster-robust standard errors to account for the cloning structure.

Exposure

We defined the primary exposure as personal internet use (CHARLS item da056s10, "Used the Internet"), restricting the definition to respondents who completed the activity checklist. Household broadband (CHARLS item i024) served as a sensitivity exposure.

Outcome

We measured depressive symptoms with the 10-item Center for Epidemiologic Studies Depression Scale (CES-D-10; items dc009–dc018), reverse-coding dc013 and dc016. We required at least seven of ten items to be valid; when this threshold was met, we prorated the total score to a 0–30 scale. The primary outcome was annualised change in CES-D-10 score. The secondary outcome was incident elevated depressive symptoms (score ≥ 10) among participants who were not depressed at baseline.

Throughout the paper, "depressive symptoms" refers to elevated symptom levels that meet a validated screening threshold, not clinician-diagnosed major depressive disorder. We used the CES-D-10 because it is the most widely used depression-screening instrument in ageing research and has been validated in Chinese older adults. This threshold matters only for the secondary outcome; the primary outcome is continuous score change and does not depend on it.

Statistical Analysis

We began with the clone-censor-weighting analysis. Weighted linear models estimated the difference in CES-D-10 slope between treatment arms; weighted Cox models estimated the hazard ratio for incident elevated symptoms.

In parallel, we characterised the between-person association directly. In the 2013 and 2015 waves we fitted a sequence of cross-sectional linear models (M0–M4), adding demographics, socioeconomic status, household device access, and prior CES-D-10 score in turn (Table 2; Figure S4). For the 2015 wave we additionally estimated the covariate-adjusted association under propensity-score weighting—stabilised inverse-probability weighting (untrimmed and trimmed), overlap weighting, and doubly robust estimation—with mandatory weight diagnostics (effective sample size, weight range, and post-weighting covariate balance; Tables S1–S3; Figure S5). As negative-control outcomes we modelled arthritis (primary) and cancer, together with a broader panel of chronic conditions, using logistic regression adjusted for age, sex, education, and urban residence (Tables S4 and S11; Fig. 5).

We then fitted a linear mixed model by restricted maximum likelihood (REML), with CES-D-10 score as the outcome, internet use and time and their interaction as the focal predictors, and age, sex, education, and urban residence as covariates; we included a random intercept for each participant. We regarded this model as a more efficient complement to fixed-effects estimation, not a substitute. Separately, we used within-person fixed-effects regression with person-mean demeaning to estimate the contemporaneous association, augmented by lead analyses (past internet use predicting current CES-D-10) and placebo-lead analyses (future internet use predicting current CES-D-10) to test temporal directionality.

To assess robustness, we calculated E-values for unmeasured confounding.¹⁹ Details are provided in Supplementary Methods 2. We accounted for competing risks of death with cause-specific Cox models (death as censoring) as the primary approach and Fine-Gray subdistribution models as a sensitivity analysis.^{20,21} Additional sensitivity analyses included household broadband as an alternative exposure; a

CES-D-10 threshold of 12 instead of 10; a baseline CES-D-10 score below 5 to test reverse causation; stratification by age, sex, and urban residence; and complete-case analysis.

We also performed an exploratory mediation analysis of sleep duration (CHARLS item da049, which does not overlap with the CES-D-10 items) using Imai's causal mediation framework.²²

We designated the within-person CES-D-10 slope—estimated by the linear mixed-model internet-by-time interaction and the within-person fixed-effects coefficient—as the primary estimand. This choice rests on identification grounds: the within-person design differences out time-invariant confounding.²³ The target-trial-emulation hazard ratio for incident symptoms is a secondary, between-person estimand. We do not assume the within-person slope is unbiased; it remains vulnerable to unmeasured time-varying confounding, whose sensitivity we report through robustness values.

Following Rothman, we did not apply formal multiplicity adjustment.²⁴ The primary estimand and its lead and placebo-lead directionality tests were pre-specified; all other analyses—including subgroups, negative controls, competing risks, and mediation—are exploratory and were interpreted for direction and consistency. No single analysis is treated as confirmatory. Causal interpretation is reserved for the convergence of evidence across designs, and any within-person signal is regarded as provisional pending randomised or quasi-experimental evidence.

For sensitivity to unmeasured confounding, we report E-values for the hazard-ratio-based between-person estimates, following VanderWeele and Ding.¹⁹ For the continuous within-person slope estimates, which are regression coefficients rather than risk ratios, we additionally report Cinelli–Hazlett robustness values calculated with the sensemakr package (Supplementary Methods 5).²⁵ All analyses were conducted in R version 4.5.1 using the data.table, survival, sandwich, lme4, lmerTest, cmprsk, and sensemakr packages. All tests were two-sided, with $p < 0.05$ as the threshold for statistical significance.

This study is reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guideline; the completed checklist is provided in Additional file 1.

Results

Study Population

The CHARLS 2011–2018 data comprised 76,629 person-waves after excluding participants outside the target age range and removing duplicate records. Among individuals who reported no internet use at baseline (2011 or 2013) and had a CES-D score below 10, 9,346 trial-eligibilities were identified from the 2011 baseline and 8,630 from the 2013 baseline, yielding 12,081 unique persons (some individuals met eligibility criteria at both baselines) (Table 1; Fig. 2). Cloning each trial-eligibility into two hypothetical arms (A and B) produced 35,952 clones (17,976 trial-eligibilities $\times$ 2 arms). The per-protocol analytic sample consisted of 36,271 person-wave records, contributed by 17,012 adherent clones with a mean of

2.13 waves per clone; the modified intention-to-treat analytic sample comprised 34,253 person-wave records, derived from 15,173 persons with measurable outcomes at the grace-period end, with one arm assigned per person and a mean of 2.26 waves per person. Person-wave counts exceeded clone counts because each adherent clone contributed multiple wave-records; the per-protocol and modified intention-to-treat counts relied on different denominators (two-arm cloned vs. one-arm grace-end) and are therefore not directly comparable.

Table 1
Characteristics of internet users and non-users, by CHARLS wave (2011–2018)

Variable	Internet users	Non-users	SMD
Wave 2011			
N (participants)	425	15,138	—
Age, years, mean (SD)	53.3 (7.6)	59.5 (9.7)	−0.71
Female, %	44.6	52.9	−0.17
Education level, mean (SD)	6.6 (1.6)	3.3 (1.9)	1.87
Urban residence, %	92.9	38.9	1.39
Depressive symptoms (CES-D-10), mean (SD)	4.4 (4.2)	8.6 (6.4)	−0.78
CES-D-10 ≥ 10, %	12.7	38.3	−0.61
Owns mobile phone, %	94.6	77.7	0.51
Owns computer, %	91.7	16.8	2.28
Home broadband, %	89.5	14.5	2.27
Wave 2013			
N (participants)	509	12,931	—
Age, years, mean (SD)	54.7 (7.3)	60.9 (9.2)	−0.74
Female, %	44.4	52.8	−0.17
Education level, mean (SD)	6.1 (1.5)	3.3 (1.9)	1.70
Urban residence, %	84.7	35.7	1.16
Depressive symptoms (CES-D-10), mean (SD)	5.3 (4.4)	8.1 (5.9)	−0.55
CES-D-10 ≥ 10, %	13.4	33.3	−0.48
Owns mobile phone, %	99.4	87.0	0.51
Owns computer, %	92.9	24.0	1.96
Home broadband, %	88.5	19.0	1.94
Wave 2015			
SMD = standardised mean difference (users minus non-users), calculated with the wave-specific pooled standard deviation of that wave's analytic sample; SMD > 0.1 indicates imbalance. Mobile phone ownership, computer ownership and home broadband are household-level items; values were assigned to all members of the responding household. CES-D-10 = 10-item Center for Epidemiologic Studies Depression Scale (range 0–30).			

Variable	Internet users	Non-users	SMD
Wave 2011			
N (participants)	574	12,862	—
Age, years, mean (SD)	56.5 (7.0)	62.4 (9.0)	-0.73
Female, %	41.8	53.0	-0.23
Education level, mean (SD)	6.0 (1.5)	3.2 (1.9)	1.60
Urban residence, %	76.0	34.3	0.92
Depressive symptoms (CES-D-10), mean (SD)	5.3 (4.7)	8.5 (6.6)	-0.56
CES-D-10 $\geq$ 10, %	15.7	36.5	-0.49
Owens mobile phone, %	99.8	88.9	0.49
Owens computer, %	85.3	20.9	1.69
Home broadband, %	84.6	22.3	1.60
Wave 2018			
N (participants)	1,170	10,883	—
Age, years, mean (SD)	59.2 (6.9)	64.7 (8.4)	-0.71
Female, %	45.9	54.1	-0.16
Education level, mean (SD)	5.3 (1.7)	3.3 (1.8)	1.16
Urban residence, %	62.0	33.6	0.59
Depressive symptoms (CES-D-10), mean (SD)	6.5 (5.6)	9.3 (6.7)	-0.45
CES-D-10 $\geq$ 10, %	23.8	41.9	-0.39
Owens mobile phone, %	98.7	90.4	0.37
Owens computer, %	44.0	11.2	0.79
Home broadband, %	82.4	36.7	1.05
SMD = standardised mean difference (users minus non-users), calculated with the wave-specific pooled standard deviation of that wave's analytic sample; SMD > 0.1 indicates imbalance. Mobile phone ownership, computer ownership and home broadband are household-level items; values were assigned to all members of the responding household. CES-D-10 = 10-item Center for Epidemiologic Studies Depression Scale (range 0–30).			

Baseline Covariate Imbalance and Selection Bias

Internet users were consistently younger, more educated, more likely to reside in urban areas, and less depressed at every assessment wave (Table 1; Figure S1, Figure S2). The standardised mean difference (SMD) for education exceeded 1.0 at all waves, indicating extreme healthy-user selection bias. Symptom prevalence was markedly lower among internet users than non-users in the 2018 wave (23.8% versus 41.9%) (Table 1; Figure S3).

Collapse of the Apparent Protective Association and Emergence of a Within-Person Residual Effect

The crude cross-sectional association of -3.07 CES-D points between internet use and depressive symptoms attenuated markedly with sequential covariate adjustment and was completely null under fixed-effects estimation ($+0.096$; 95% CI, -0.148 to 0.341 ; $p = 0.440$) (Table 2; Figure S4). The propensity-score-based analyses were sensitive to the choice of weighting (Tables S1 and S3; Figure S5). With overlap weighting—our preferred approach, given the limited overlap in propensity scores—a small protective association remained (-0.318 ; 95% CI, -0.505 to -0.130 ; $p < 0.001$); truncated inverse-probability weighting produced estimates ranging from -3.29 to -0.59 ; and the doubly robust estimate was significantly positive ($+0.659$; 95% CI, 0.038 to 1.281 ; $p = 0.038$). Depending on the specification, the adjusted between-person association was protective, null or positive; we therefore do not interpret it as evidence of protection. A linear mixed model fitted to the full cohort (54,492 person-waves from 17,012 persons) yielded a modest within-person slope difference of $+0.094$ points per year (95% CI, 0.014 – 0.174 ; $p = 0.021$), suggesting that users' depressive symptoms increased slightly faster over time; however, this estimate was small in magnitude and does not support a robust causal interpretation (see E-values and Robustness). The internet main effect of -1.09 ($p < 0.001$) reflected the between-person selection advantage rather than a causal effect (Table 2; Table S13). Restricting the within-person analysis to the initiator subset—baseline non-users who subsequently initiated internet use, the target population for the target trial emulation with actual exposure variation ($n = 1,180$ ever-initiators)—substantially strengthened the contemporaneous fixed-effect estimate ($+0.255$; 95% CI, -0.005 to 0.516 ; $p = 0.055$) and the lead fixed-effect estimate ($+0.490$; 95% CI, 0.062 – 0.918 ; $p = 0.025$). The smaller full-cohort contemporaneous estimate likely reflects pooling of heterogeneous within-person switcher types—initiators, discontinuers, and intermittent users—together with attenuation from measurement error in current-use reporting. Baseline non-users who never initiated ($n = 15,304$) contribute no within-person variation in internet use and therefore no identifying information for the within-person internet coefficient, so they affect precision rather than the point estimate (Table S10).

Table 2
Internet use and depressive symptoms under sequential adjustment and fixed effects

Model1	β (CES-D-10 points)	95% CI	<i>p</i> -value
2013			
M0 Crude	-2.852	(-3.398, -2.306)	< 0.001
M1 + Demographics	-2.550	(-3.094, -2.007)	< 0.001
M2 + SES	-1.142	(-1.704, -0.580)	< 0.001
M3 + Device access	-0.336	(-0.914, 0.242)	0.254
M4 + Prior CES-D-10	-0.348	(-0.860, 0.163)	0.182
2015			
M0 Crude	-3.070	(-3.665, -2.475)	< 0.001
M1 + Demographics	-2.534	(-3.125, -1.944)	< 0.001
M2 + SES	-0.965	(-1.571, -0.359)	0.002
M3 + Device access	-0.098	(-0.721, 0.525)	0.758
M4 + Prior CES-D-10	0.069	(-0.467, 0.605)	0.801
Multi-wave			
M5 Within-person FE	0.096	(-0.148, 0.341)	0.440
¹ M0 = crude; M1 = + demographics; M2 = + socioeconomic status; M3 = + device access (household-level items: mobile phone, computer, broadband); M4 = + prior CES-D-10 score; M5 = within-person fixed effects. β = CES-D-10 points (internet users vs non-users). M0–M4 are cross-sectional models fitted separately in the 2013 and 2015 waves; M5 is estimated on the full four-wave cohort (2011–2018) and therefore on a different sample from M0–M4.			

Target-Trial Emulation: Between-Person Protective Association

In the clone-censor-weight emulation with inverse probability of censoring weighting (effective sample size 38,092 of 38,638 [98.6%]; maximum weight 1.22; no weight instability), the hazard of elevated depressive symptoms was lower among initiators: per-protocol hazard ratio 0.699 (95% CI, 0.559–0.875; $p = 0.002$) and intention-to-treat hazard ratio 0.703 (95% CI, 0.561–0.883; $p = 0.002$); inverse probability of censoring weighting–adjusted CES-D-10 trajectories are presented in Figure S6. The near-identical per-protocol and intention-to-treat estimates (distinguishable only at the third decimal place, confirming independent computation) indicate that protocol deviation was not strongly informative for symptom risk. The broadband internet sensitivity analysis (i024) yielded concordant results: per-protocol hazard ratio 0.796 (95% CI, 0.723–0.876; $p < 0.001$) (Table 3); estimates were robust to unweighted Cox modeling, consistent with limited informative censoring (Supplementary Methods 6). Competing-risk

models on baseline non-depressed ever- and never-initiators (n = 9,323; 869 deaths) reproduced the protective association without a mortality paradox—cause-specific Cox hazard ratio 0.739 and Fine-Gray subdistribution hazard ratio 0.742 (Fig. 3; Table S6; detail in Supplementary Results 3). All of these hazard ratios are between-person incidence measures and, as such, reflect selection bias rather than a causal effect.

Table 3

Estimand divergence: between-person protection vs within-person harm. Internet use and depressive symptoms, CHARLS 2011–2018

Estimand	Estimate (95% CI)	p-value	Direction
Between-person (incidence / levels)			
Cross-sectional β (crude, 2015)	–3.07 (– 3.67, – 2.48)	< 0.001	Protection
TTE per-protocol HR (da056s10)	0.699 (0.559, 0.875)	0.002	Protection
TTE intention-to-treat HR	0.703 (0.561, 0.883)	0.002	Protection
i024 broadband sensitivity HR (PP)	0.796 (0.723, 0.876)	< 0.001	Protection
Cause-specific Cox HR (death censored)	0.739 (0.654, 0.835)	< 0.001	Protection
Fine-Gray subdistribution sHR	0.742 (0.657, 0.838)	< 0.001	Protection
Negative-control outcome: arthritis, adjusted OR	0.73 (0.60, 0.90)	< 0.01	Protection
Within-person (slope / trajectory)			
Mixed model, internet $\times$ time (CES-D-10 points/yr)	+ 0.094 (0.014, 0.174)	0.021	Harm
Within-person FE (overall)	+ 0.096 (– 0.148, 0.341)	0.440	Harm (NS)
Lead FE (past predicts current)	+ 0.50 (0.12, 0.88)	0.011	Harm
Placebo-lead FE (future predicts current)	–0.06 (– 0.35, 0.23)	0.68	Null (directionality)
Baseline CES-D-10 < 5 subgroup FE	+ 0.83 (0.53, 1.14)	< 0.001	Harm

HR, sHR and OR compare ever-users (or initiators) with never-users; values < 1 indicate protection. β denotes the within-person effect on the CES-D-10 score (individual-demeaned); values > 0 indicate harm. All between-person estimates reflect healthy-user selection. Cox models were weighted by inverse probability of censoring where applicable. TTE = target-trial emulation (clone–censor–weight approach; effective sample size 38,092/38,638; maximum stabilised weight 1.22). The baseline CES-D-10 < 5 subgroup restricts the analysis to participants with minimal symptoms at baseline, limiting reverse causation. Cause-specific and Fine-Gray models were fitted to the same ever/never sample (n = 9,323; 3,461 depression events; 869 deaths) and yielded nearly identical estimates (0.739 and 0.742, respectively). NS = not significant.

Temporal Directionality: Lead Effects and Placebo-Lead Analysis

The within-person lead fixed-effects estimate (past internet use predicting current CES-D score) was + 0.496 (95% CI, 0.115–0.878; $p = 0.011$), indicating that past internet use was associated with subsequently higher depressive symptom scores. The placebo-lead estimate (future internet use predicting current CES-D score) was – 0.061 (95% CI, – 0.347 to 0.225; $p = 0.676$), which was null. This pattern—significant active lead and null placebo lead—is consistent with forward temporal directionality (Fig. 1), although it does not itself establish causation. Notably, the lead effect (+ 0.496) was larger than the contemporaneous within-person effect (+ 0.096). If the effect of internet use on depressive symptoms requires 1–2 years to manifest, a plausible timeframe for neurobehavioural pathways, then past use would predict current CES-D more strongly than current use. This interpretation aligns with the observed pattern, and contemporaneous measurement error in current-use reporting would further attenuate the contemporaneous estimate. The within-person autocorrelation of internet use was moderate ($r[t, t - 1] = 0.496$; Table S12); consequently, a lagged effect cannot be fully distinguished from a measurement-stability artifact. We therefore treat the lead result as suggestive of temporal directionality rather than as proof of a lagged causal effect.

Within-Person Association Strongest in Subgroup Where Reverse Causation Is Least Plausible

Among participants with baseline CES-D scores below 5 (deep non-depressed subgroup; 17,392 person-waves from 5,001 persons), the within-person fixed-effects estimate was + 0.834 (95% CI, 0.532–1.136; $p < 0.001$). This was the largest within-person estimate observed and arose in precisely the subgroup where reverse causation is least tenable. No subgroup analysis produced a statistically significant protective estimate (Fig. 4). The broadband internet sensitivity analysis (i024) yielded a within-person fixed-effects estimate of + 0.182 (95% CI, 0.047–0.317; $p = 0.008$). Although these point estimates are directionally consistent with a harmful association, they share the same limitations as the primary within-person estimates and do not support a robust causal interpretation.

Negative-Control Outcomes: Confirmation of Selection Bias

Arthritis, the primary negative-control outcome, showed a protective association with internet use across all waves (adjusted odds ratio 0.51–0.75; all $p < 0.01$). Cancer, the most stringent negative control given the absence of any plausible short-term causal pathway from internet use to cancer incidence, yielded a pooled adjusted odds ratio of 0.82 (95% CI, 0.56–1.21). Both outcomes exhibited the same protective direction as depressive symptoms in every between-person model; rather than weakening the inference, this convergence strengthens it, confirming that the between-person association reflects a generic socioeconomic-status and health-engagement selection artifact rather than a causal pathway specific to

depressive symptoms. The broader negative-control panel across nine chronic diseases was broadly null-to-protective (Fig. 5; Table S4; Table S11), the single exception being liver disease (adjusted odds ratio 1.45), which more plausibly reflects regional hepatitis B prevalence and urban diagnostic patterns than a causal internet-to-liver pathway.

E-values and Robustness Assessment

For the between-person adjusted association, the E-value was 1.11 (confidence-interval bound 1.00), meaning that an unmeasured confounder of modest strength (relative risk approximately 1.1 with both exposure and outcome) could explain away the association, and the bound of 1.00 denotes sensitivity precisely at the threshold of statistical significance (Figure S7). Because within-person slopes are regression coefficients rather than risk ratios, we report Cinelli–Hazlett robustness values (sensemakr) for continuous outcomes: the linear mixed model internet-by-time slope had a robustness value of 0.0016 and the lead fixed-effect estimate 0.0031 (the contemporaneous within-person fixed-effects estimate, already null, was approximately zero). These values are very small—an unmeasured time-varying confounder accounting for less than 0.5% of the residual variance in both internet-use transitions and CES-D changes would suffice to erase their statistical significance—so the within-person residual is directionally consistent across designs (Table S7) but not robust to unmeasured time-varying confounding (Table S9). Neither the between-person protective association nor the within-person residual is secure against unmeasured confounding, and the overall inference therefore rests on the pattern of estimand divergence (Fig. 1) rather than on any single estimate. Sensitivity analyses using alternative CES-D thresholds (≥ 12), age–sex–urban stratification and complete-case estimation consistently yielded non-significant within-person estimates spanning the null, with no statistically significant protective association (Fig. 4; Table S7).

Discussion

In this nationally representative cohort of middle-aged and older Chinese adults, the widely reported protective association between internet use and lower depressive symptoms did not survive methods that remove selection bias. Cross-sectionally, internet users looked substantially less depressed than non-users, but the advantage vanished under within-person fixed-effects estimation – and it reappeared for a negative-control outcome, arthritis, which internet use could not plausibly protect against.²⁶ The parsimonious reading is that the apparent protection is a healthy-user selection artefact,¹⁴ not a depression-specific benefit of going online.

Why the protection disappears. Internet users are systematically younger, more educated, wealthier and healthier than non-users; in CHARLS the standardised mean difference for education exceeded 1.0 at every wave.¹³ The traits that drive adoption – digital literacy, intrinsic motivation, social engagement, cognitive reserve – are largely unmeasured and stable over time. Conventional covariate adjustment cannot touch them; within-person designs difference them out.²⁷ The disappearance of the protective signal under fixed effects, and its replication for arthritis, tells us that what earlier observational studies

read as a causal benefit^{3,28,29} is the signature of who adopts the technology, not what the technology does.

What the rigorous estimates show. Once between-person selection is removed, a modest within-person residual remains. In the linear mixed model, internet use was associated with a slightly faster annual rise in depressive symptoms (+ 0.094 CES-D-10 points per year). The lead effect was significant while the placebo-lead was null, consistent with forward temporal ordering; the estimate was largest exactly where reverse causation is least plausible (baseline CES-D-10 < 5; +0.83). The same direction held under an alternative exposure definition based on broadband access. We treat the fixed-effects estimates as primary: they difference out time-invariant selection without leaning on the orthogonality assumption that random effects require, and the mixed-model internet-by-time slope corroborates the same within-person direction with greater efficiency. One result cuts the other way — the contemporaneous fixed effect was null in the full cohort. The most plausible reading is attenuation by measurement error in current-use reporting, since the lead and subgroup estimates and the mixed-model slope all point the same way. Together these estimates point towards modest harm, but the contemporaneous null and the small robustness values ($RV < 0.005^{25}$) keep that reading provisional rather than confirmatory. We therefore do not read this pattern as evidence that internet use causes depression — only as a consistent direction that calls for randomised evaluation rather than reassurance.

Reconciling an inconsistent literature. Published estimates of internet use and depressive symptoms in later life range from apparent protection through null findings to signals of harm^{3,29–32}. This divergence appears even within a single cohort. In the English Longitudinal Study of Ageing, random-effects models linked communication-related internet use to fewer depressive symptoms, while fixed-effects estimates for depressive symptoms in the same data were null²⁹ — the protective reading depends on the estimator, not on the data. Our results suggest that this inconsistency is structure, not noise. Between-person designs are dominated by selection and lean towards protection; within-person designs strip that selection out and drift towards the null or modest harm. Divergence between estimands is itself informative — it marks the spot where selection bias is doing the work. Seen this way, the literature's contradictions are expected rather than paradoxical.

Public-health and policy implications. Digital-inclusion programmes — the WHO Decade of Healthy Ageing¹, China's "Smart Aging" initiative³³, the EU Digital Decade³⁴ and US digital-skills training efforts³⁵ — are routinely justified to the public partly on evidence of mental-health benefit that selection can explain. None of our results shows that internet use harms mental health. What they show is narrower: the protective premise behind these programmes does not survive contact with designs that address selection. The practical consequence is an opportunity cost. Resources and clinical attention spent on digital-literacy training to prevent depressive symptoms are unavailable to psychological and social-support interventions with established evidence behind them. Clinicians should not recommend internet use to prevent depressive symptoms, and policymakers should not promote digital inclusion on

selection-confounded associations. The defensible position is caution about an unproven benefit — not policy reversal on unproven harm.

Methodological contribution. For self-selected lifestyle exposures, where adoption is inseparable from the traits of the adopter, within-person designs and negative-control outcomes belong at the centre of the analysis, not in the appendix as afterthought sensitivity checks^{26,36}. The estimand-divergence logic used here transfers directly to other modifiable behaviours — physical activity, diet, social engagement — wherever the same selection structure is likely to operate.

Strengths and limitations. Strengths include the nationally representative longitudinal design; codebook-verified exposure and outcome; an exposure definition restricted to respondents, so missing responses are never recoded as non-use; clone-censor-weighted target-trial emulation with mandatory weight diagnostics; dual exposure definitions; and multiple negative-control outcomes. The limitations matter at least as much. Internet use was measured as a single binary item, with no information on frequency or purpose of use; exposure was sparse in early waves, which limits power; CES-D-10 is a self-report screening instrument, not a clinical diagnosis; and the within-person residual is statistically fragile. The full list appears in the Supplementary Material. Being observational, this analysis can neither establish nor exclude a causal effect; its target is the credibility of the literature's protective claim, and any within-person signal remains provisional until randomised or quasi-experimental evidence arrives.

Conclusion

The protective association between internet use and lower depressive symptoms in middle and later life is a selection artefact. A modest, directionally consistent within-person residual is compatible with harm but too fragile to confirm causation. On present evidence, internet use should not be recommended to prevent depressive symptoms, and digital-inclusion programmes should not be promoted on selection-confounded evidence — not because harm is proven, but because protection is not.

Abbreviations

CES-D-10

10-item Center for Epidemiologic Studies Depression Scale

CHARLS

China Health and Retirement Longitudinal Study

CI

confidence interval

EU

European Union

FE

fixed effects

HC1

heteroscedasticity-consistent (type 1) standard errors
HR
hazard ratio
IPCW
inverse probability of censoring weighting
IPTW
inverse probability of treatment weighting
ITT
intention-to-treat
LMM
linear mixed model
NCO
negative-control outcome
NS
not significant
OR
odds ratio
PP
per-protocol
REML
restricted maximum likelihood
RV
robustness value
SD
standard deviation
SES
socioeconomic status
sHR
subdistribution hazard ratio
SMD
standardised mean difference
STROBE
Strengthening the Reporting of Observational Studies in Epidemiology
TTE
target-trial emulation
US
United States
WHO
World Health Organization

Declarations

Ethics approval and consent to participate

The China Health and Retirement Longitudinal Study (CHARLS) was approved by the Biomedical Ethics Review Committee of Peking University (IRB00001052-11015), and all participants provided written informed consent. The present secondary analysis used de-identified, publicly available CHARLS data and did not require separate ethical approval.

Consent for publication

Not applicable. No individual person's data appear in any form in this manuscript.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

S.J. and X.Z. performed data curation. S.J. completed the formal analysis and visualisation, and drafted the original manuscript. X.Z. conducted the data validation. C.A. conceived the study, designed the methodology, supervised the whole study, and managed the project. X.Z. and C.A. critically revised the manuscript. All authors read and approved the final version of the manuscript.

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Data Availability

The CHARLS data are publicly available to registered researchers from the official CHARLS website (<http://charls.pku.edu.cn/>).

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Figures

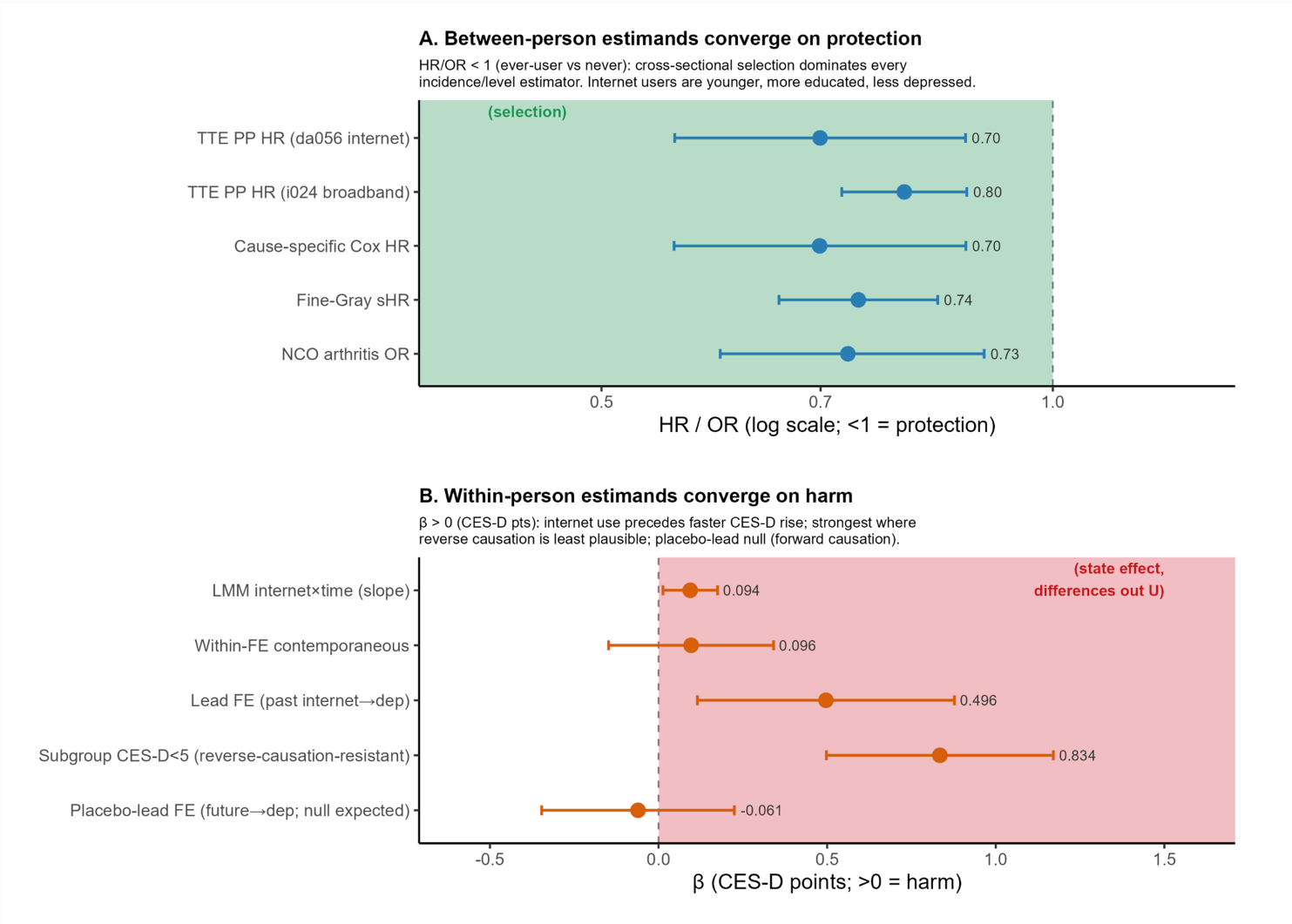


Figure 1

Estimand divergence between between-person and within-person analyses of internet use and depressive symptoms, CHARLS 2011–2018.

(A) Between-person estimands (ever-user vs never-user): per-protocol hazard ratios from clone-censor-weighted target-trial emulation (personal internet use, da056s10; household broadband, i024), cause-specific Cox hazard ratio and Fine-Gray subdistribution hazard ratio for incident elevated depressive symptoms (CES-D-10 ≥ 10), and the pooled adjusted odds ratio for the negative-control outcome arthritis.

(B) Within-person estimands (β in CES-D-10 points): linear mixed-model internet $\times$ time slope, contemporaneous within-person fixed effect, lead fixed effect (past use $\rightarrow$ current CES-D-10), the baseline CES-D-10 < 5 subgroup, and the placebo-lead fixed effect (future use $\rightarrow$ current CES-D-10). Error bars are 95% confidence intervals. CES-D-10 = 10-item Center for Epidemiologic Studies Depression Scale; FE = fixed effects; NCO = negative-control outcome; SHR = subdistribution hazard ratio; TTE = target-trial emulation.

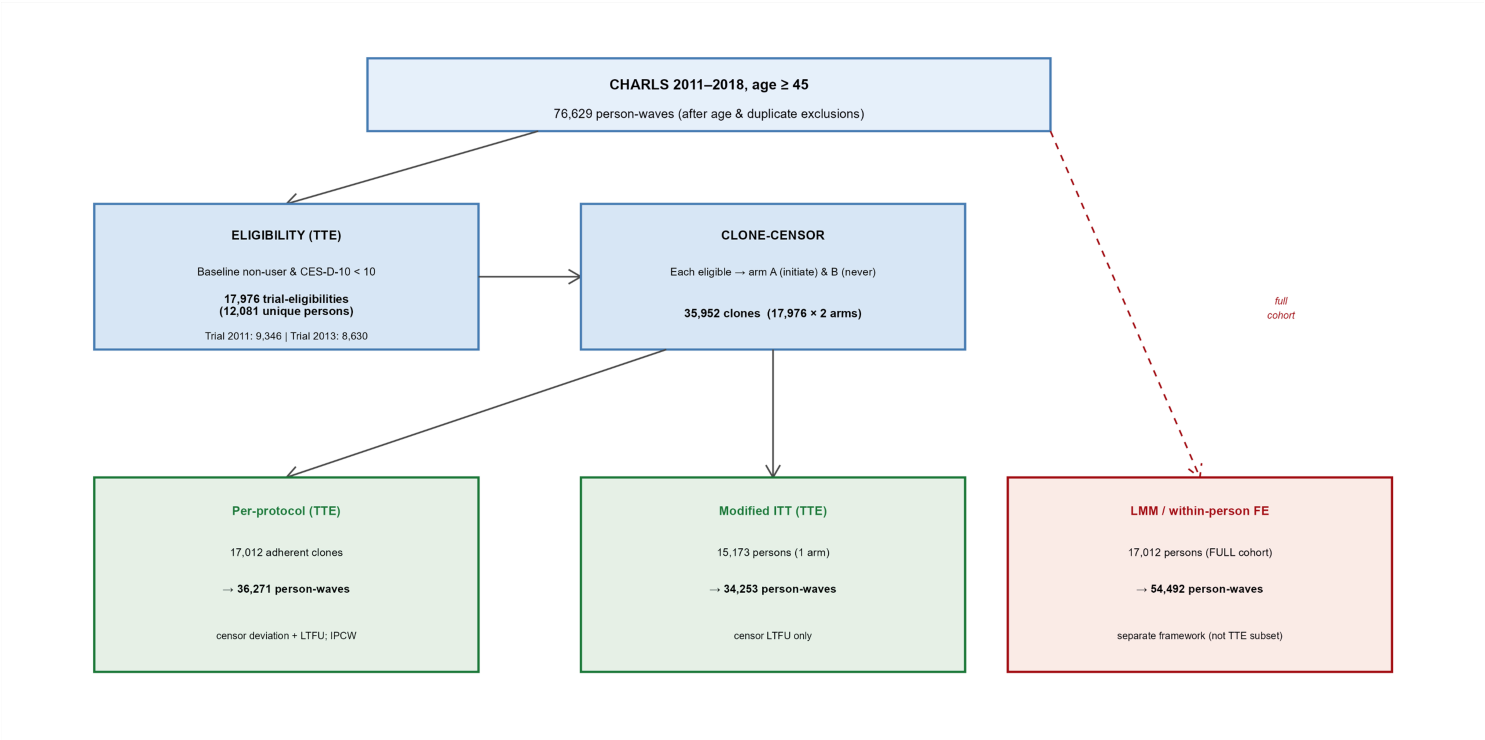


Figure 2

Sample flow for the sequential target-trial emulation and the full-cohort within-person analyses, CHARLS 2011–2018.

Baseline internet non-users aged ≥ 45 years with CES-D-10 scores < 10 at the 2011 or 2013 baseline were eligible (17,976 trial-eligibilities; 12,081 unique persons) and cloned into two strategy arms—initiate internet use within a two-year grace period (arm A) or never initiate (arm B)—yielding 35,952 clones. The per-protocol analysis censored clones at strategy deviation or loss to follow-up and applied inverse

probability of censoring weighting; the modified intention-to-treat (ITT) analysis assigned each participant to the arm matching their observed grace-end behaviour and censored only at loss to follow-up. The linear mixed model (LMM) and within-person fixed-effects (FE) analyses (red box) form a separate framework drawn from the full analytic cohort, not the target-trial subset; the FE estimates further require ≥ 2 waves per person. The count of 17,012 appears for two distinct quantities (adherent clones; full-cohort persons) by coincidence. CES-D-10 = 10-item Center for Epidemiologic Studies Depression Scale; IPCW = inverse probability of censoring weighting.

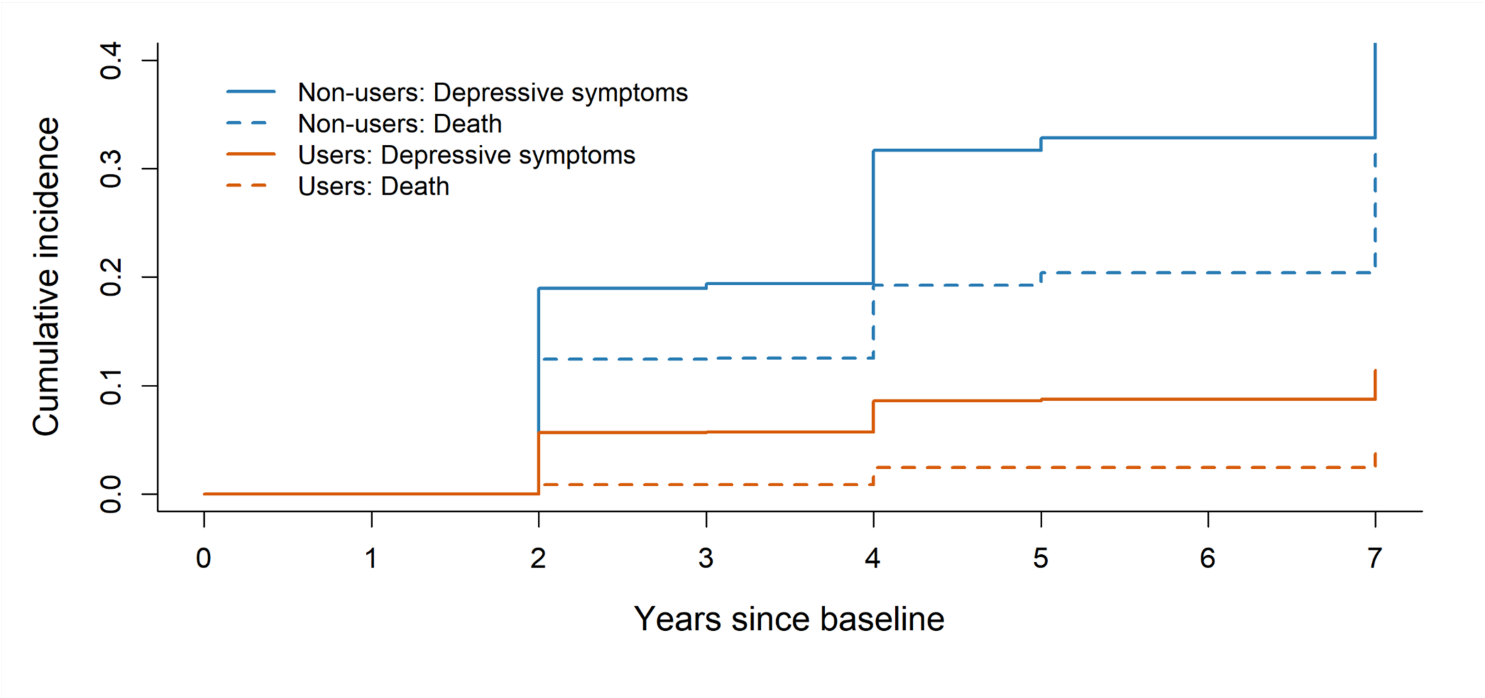


Figure 3

Cumulative incidence of elevated depressive symptoms and death by internet-use status, CHARLS 2011–2018

Step functions are unadjusted cumulative incidence functions with death as the competing event. Solid lines are incident elevated depressive symptoms ($\text{CES-D-10} \geq 10$), dashed lines are death, estimated separately for users (orange) and non-users (blue). Jumps occur at the assessment waves: $\approx 2, 4, 5$ and 7 years after baseline across the 2011 and 2013 sequences. Cumulative incidence is lower among users for both outcomes, consistent with healthy-user selection rather than a protective effect (within-person analyses: Figures 1, 4 and 5). CES-D-10 = 10-item Center for Epidemiologic Studies Depression Scale.

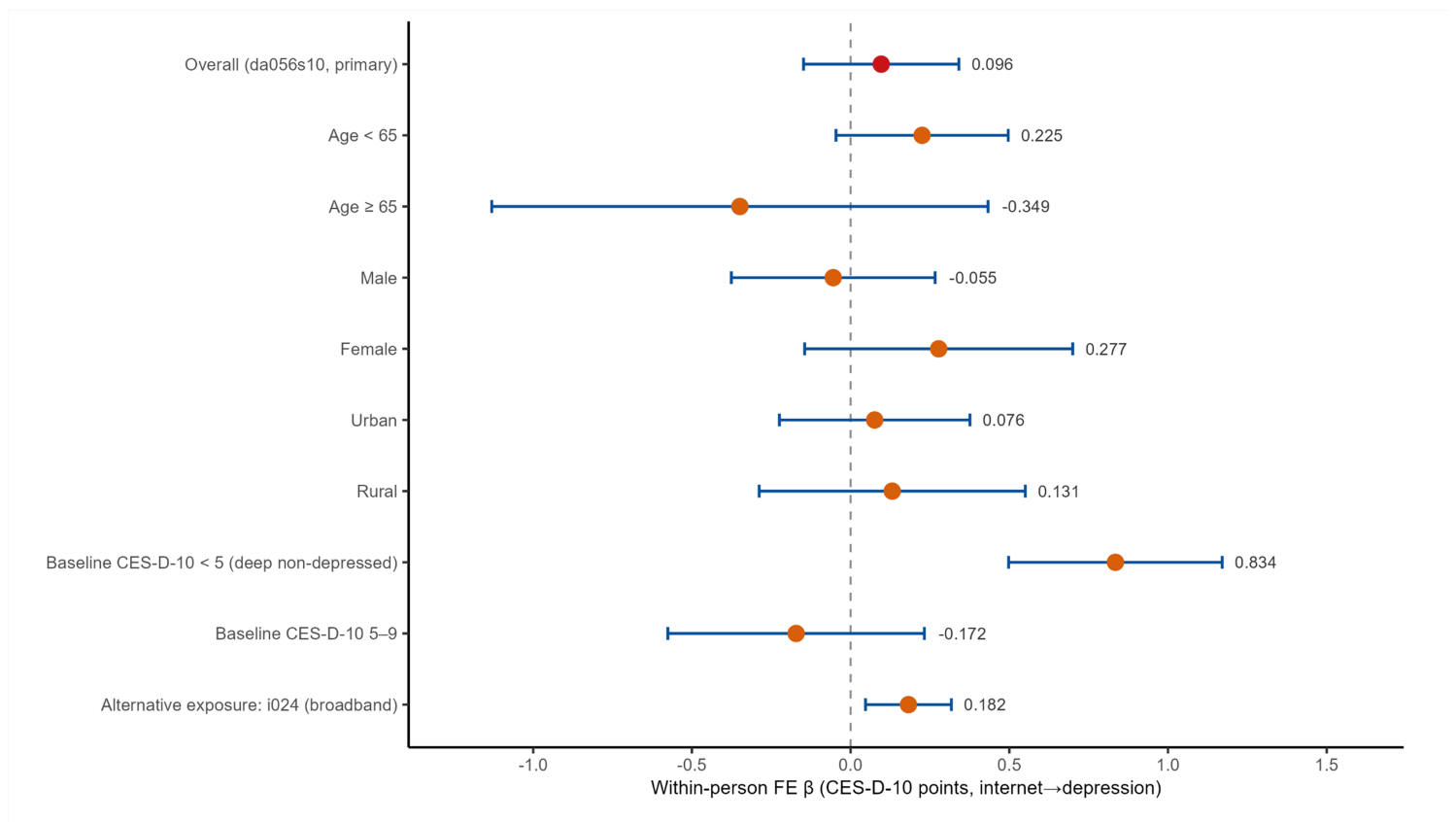


Figure 4

Within-person fixed-effects estimates across subgroups and exposure definitions, CHARLS 2011–2018.

Points are coefficients from person-mean-demeaned regressions of CES-D-10 score on internet use; bars are 95% cluster-robust confidence intervals (HC1, person level), and positive values indicate higher symptom scores among users. The red point is the overall estimate under the primary exposure (personal internet use, da056s10); orange points are subgroup estimates restricted to participants contributing ≥ 2 waves within the subgroup, with baseline-CES-D-10 strata limited to scores < 10 . The final row replaces the exposure with household broadband (i024). CES-D-10 = 10-item Center for Epidemiologic Studies Depression Scale; FE = fixed effects.

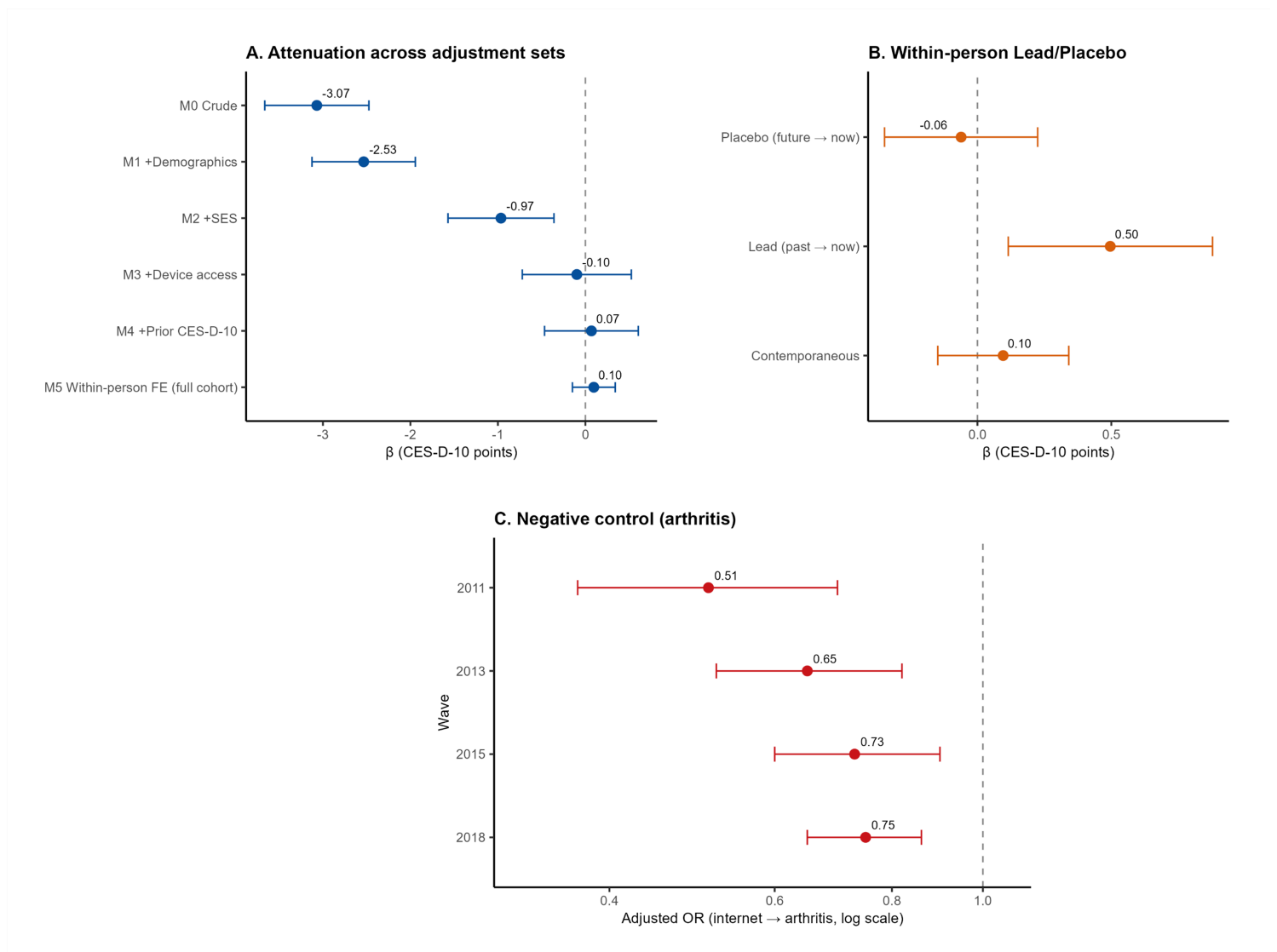


Figure 5

Progressive attenuation of the cross-sectional association, within-person directionality tests, and negative-control outcome arthritis, CHARLS 2011–2018.

(A) Successive adjustment of the cross-sectional association between internet use and CES-D-10 score (2015 wave): M0, crude; M1, + demographics; M2, + socioeconomic status; M3, + device access; M4, + prior CES-D-10 score; M5, within-person fixed effects. (B) Within-person fixed-effects directionality tests: the contemporaneous estimate; the lead estimate (past internet use predicting current CES-D-10); and the placebo-lead estimate (future use predicting current CES-D-10), the latter expected to be null under forward temporal ordering. (C) Adjusted odds ratios for the negative-control outcome arthritis at each wave (logistic regression adjusted for age, sex, education, and urban residence); arthritis has no plausible protective pathway from internet use. Error bars are 95% confidence intervals. CES-D-10 = 10-item Center for Epidemiologic Studies Depression Scale; FE = fixed effects.

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