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Does Digital Engagement Delay the Onset of Cognitive Impairment?A Target Trial Emulation of Time-to-Event Shifts in CHARLS (2011–2018)

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Does Digital Engagement Delay the Onset of Cognitive Impairment?A Target Trial Emulation of Time-to-Event Shifts in CHARLS (2011–2018)

Abstract:
As population aging accelerates alongside rapid digital transformation, whether digital engagement can delay the onset of cognitive impairment has become a question of growing public health and policy relevance. Using longitudinal data from the China Health and Retirement Longitudinal Study (CHARLS, 2011–2018), this study emulates a target trial to estimate the causal effect of sustained digital engagement on time-to-cognitive-impairment. We implement weighted discrete-time marginal structural models with stabilized inverse probability of treatment and censoring weights to address time-varying confounding, selective attrition, and reverse causation. Regular (≥weekly) digital engagement is associated with a significantly lower hazard of incident cognitive impairment and a clinically meaningful delay in onset. Counterfactual survival curves indicate a 7.4-month gain in restricted mean survival time over seven years under sustained engagement compared with non-engagement. Heterogeneity analyses reveal a compensatory pattern: the time-shift is largest among individuals with low education, rural residence, high social isolation, and lower baseline cognition. Pathway tests suggest that digital engagement operates through dual psychosocial mechanisms—psychological relief (reduced depressive symptoms) and enhanced social connectedness—with the psychological pathway exerting a relatively stronger influence. These findings move beyond static associations by demonstrating that digital engagement reshapes the temporal trajectory of cognitive aging. In aging societies, digital inclusion may therefore function not only as a technology policy but also as a scalable strategy for promoting cognitive health equity.
Keywords: digital engagement; cognitive impairment; time-to-event; target trial emulation; psychosocial mechanisms

Introduction

China is entering a phase of rapid population aging at the same time that everyday life is being reshaped by digital technologies. Online communication, mobile payments, tele-services, and platform-based information have become embedded in routine social and economic participation. Yet older adults are not equally positioned to benefit from this transformation. Digital engagement among China's midlife and older population remains socially patterned—stratified by education, rural–urban residence, income and wealth, and functional capacity—creating a "late-life digital divide" that is both structural and behavioral [1]. In parallel, cognitive impairment has emerged as a major public health and social policy concern. Even mild cognitive impairment can reduce independence, increase care needs, and elevate long-term disability risk, with profound implications for families and health systems as the size of the older population grows [2]. Against this backdrop, it is plausible that digital engagement and the onset of cognitive impairment are linked: digital tools may extend opportunities for cognitive stimulation and social participation, but they may also reflect underlying advantage or early-stage decline [3].

A central challenge is that the existing literature offers competing interpretations. A large body of research—drawing on cognitive reserve theory, "use-it-or-lose-it" accounts, and stimulation-based perspectives—suggests that mentally engaging activities can support cognitive aging by strengthening adaptive capacity and preserving function [4]. Digital engagement, in this view, can be more than a convenience; it may serve as a modern bundle of cognitively and socially stimulating behaviors. Using the internet can involve learning new skills, navigating complex interfaces, searching and evaluating information, managing tasks, and maintaining communication networks [5]. These components plausibly exercise memory, attention, executive function, and language while also improving access to resources and services that may support health management [6]. If so, digital engagement could shift cognitive aging trajectories in a meaningful way, potentially delaying the onset of impairment.

At the same time, skepticism is well founded. Digital engagement in later life is strongly correlated with socioeconomic resources and baseline cognitive capacity [7]. As a result, observed associations may reflect selection rather than influence: individuals with higher education and better health are more likely to go online, while those experiencing functional limitations may disengage. Reverse causation is particularly salient in cognitive aging: preclinical cognitive decline may reduce digital participation long before impairment is formally recognized. Moreover, digital engagement is heterogeneous in quality and purpose [8, 9]. Some online activity may be passive and displacing—crowding out physical activity or face-to-face interaction—while other forms may be interactive and socially connecting [10]. These competing narratives help explain why empirical findings across settings are mixed and why "digital engagement helps cognition" remains an unsettled claim.

In response, academic thinking has evolved in two important ways. First, the field has moved from asking whether technology use is correlated with cognitive outcomes toward asking whether it has a plausible causal interpretation under stronger research designs [11]. Methods-oriented developments—such as target trial emulation, time-varying causal inference, and time-to-event estimands—aim to clarify what causal question is being asked and how it might be approximated with observational data [12]. Second, theoretical advances in digital inequality emphasize not only access but also effective use and differential returns. This implies that even if digital engagement matters, it may not matter equally for everyone [13]. Competing frameworks propose either cumulative advantage (the already advantaged benefit most) or compensatory benefit (digital tools partly offset offline constraints, yielding larger marginal gains among disadvantaged groups) [14]. These perspectives motivate explicit tests of heterogeneity rather than reliance on average effects.

Another theoretical update concerns mechanism. If digital engagement is linked to cognitive impairment onset, through what pathway might this occur? Two psychosocial channels are especially plausible and frequently debated. A "psychological relief" pathway emphasizes mood, stress buffering, and perceived control: digital tools may reduce distress by facilitating access to information, enabling coping resources, and supporting enjoyable or meaningful activities [15, 16]. A "social connection" pathway emphasizes isolation and network maintenance: digital communication may sustain ties when mobility declines or distance increases, thereby preserving social participation and support. These pathways are not mutually exclusive and may operate jointly or sequentially [17]. Importantly, their relative importance may depend on time horizon: psychological changes may emerge quickly, while social network reconfiguration may accumulate more gradually. This possibility suggests that the mechanism itself may be time-dependent rather than fixed.

This study contributes to these debates by focusing on the timing of impairment in a nationally relevant setting. Rather than emphasizing only average cognitive differences, we examine whether digital engagement is associated with a discernible shift in the time-to-onset trajectory of cognitive impairment—an estimand with direct policy relevance. The analysis uses longitudinal data from the China Health and Retirement Longitudinal Study (CHARLS, 2011–2018) and adopts a target trial emulation logic to define eligibility, exposure strategies, follow-up, and censoring. This framing allows the study to address key threats in the literature, including time-varying confounding (health and function affecting both digital engagement and cognitive risk) and informative attrition.

Accordingly, we address three research questions. RQ1 asks: Does digital engagement relate to when cognitive impairment first emerges over follow-up? This question shifts attention from static associations to the time dimension, asking whether engagement corresponds to a delay in onset. RQ2 asks: For whom is digital engagement most consequential—are any timing shifts concentrated in particular subpopulations? This directly tests whether returns to digital engagement are socially patterned, consistent with cumulative advantage or compensatory benefit accounts. RQ3 asks: Is any observed association more consistent with a "psychological relief" pathway, a "social connection" pathway, or both? This speaks to mechanism and helps interpret whether digital engagement is best understood as primarily affective stress buffering, social infrastructure, or a combination.

The remainder of the paper is organized as follows. Section 1 reviews the literature on digital engagement and cognitive aging, highlighting

empirical inconsistencies and conceptual debates. Section 2 develops the theoretical framework and articulates hypotheses around timing, heterogeneity, and psychosocial pathways. Section 3 describes the CHARLS data, outcome and exposure construction, and the target trial emulation strategy, including the time-varying modeling approach and handling of censoring. Section 4 presents the results in four steps: (i) overall time-trajectory changes in cumulative incidence and time-shift metrics; (ii) heterogeneity analyses that identify groups for whom digital engagement is most consequential; (iii) pathway-oriented tests distinguishing psychological and social channels and their evolution across horizons; and (iv) robustness checks using alternative impairment thresholds, sample restrictions, weighting choices, additional physical-function controls, and continuous cognitive decline outcomes. Section 5 concludes with implications for digital inclusion policies and cognitive health strategies in aging societies.

1 Literature Review

1.1 Digital Engagement and Cognitive Aging

Over the past decade, the potential relationship between digital engagement and cognitive aging has garnered significant attention, particularly in the context of aging populations and the growing prevalence of cognitive impairment. Several studies have suggested that digital engagement, through activities such as internet use, social media participation, and online learning, may offer cognitive benefits by fostering cognitive reserve or stimulating mental activities [18, 19]. For instance, research indicates that older adults who engage in digital activities, including online games or communication, show a slower rate of cognitive decline compared to their less engaged counterparts [20, 21]. In these studies, cognitive reserve theory is often invoked, proposing that mental stimulation through complex tasks such as those performed online can help mitigate age-related cognitive decline by enhancing neural connections and cognitive functioning.

However, the evidence is not without inconsistency. While some studies have found positive associations between digital engagement and cognitive function [22], others have reported null or even negative relationships [23]. This lack of consensus may stem from differences in operationalization of "digital engagement," varying types of cognitive outcomes measured (such as cognitive scores versus the onset of cognitive impairment), and the socio-economic and health factors influencing participation in digital activities [24]. Furthermore, while most studies have focused on average effects, the potential for digital engagement to delay the onset of cognitive impairment specifically remains largely unexplored [25]. This gap in understanding emphasizes the need to shift from a simple association to a deeper investigation of time-to-event dynamics, which captures the actual delay in the onset of cognitive decline rather than merely the speed of cognitive decline or average scores.

1.2 Competing Explanations

A critical issue in the digital engagement and cognitive aging literature lies in the competing theoretical explanations for observed relationships. Proponents of the cognitive reserve hypothesis argue that digital engagement provides cognitive stimulation, which, over time, builds neural resilience and helps delay the onset of cognitive decline [26]. On the other hand, a growing body of literature has raised concerns about selection bias and reverse causality, suggesting that people with higher cognitive function may be more likely to engage with digital technologies [27], or that cognitive impairment may discourage individuals from using digital platforms [28].

Additionally, the literature highlights another confounding factor: the role of social engagement. Digital engagement often overlaps with social activities that provide emotional and social support, both of which have been shown to be protective against cognitive decline [29]. However, social engagement and digital engagement are highly intertwined, making it difficult to disentangle their independent effects on cognitive outcomes. A growing number of studies suggest that the influence of digital engagement may not be uniform across different individuals. Older adults with better cognitive health or more education may benefit more from digital engagement compared to those who are already cognitively impaired or socially isolated [30]. This heterogeneity suggests that the impacts of digital engagement are not one-size-fits-all, and further research is needed to understand under which circumstances digital engagement may be most beneficial.

To address these concerns, more sophisticated methodologies are necessary. While traditional cross-sectional studies often suffer from selection bias and reverse causality, newer experimental designs and statistical techniques such as target trial emulation offer potential solutions. This approach allows researchers to better simulate causal relationships by creating hypothetical randomized controlled trials from observational data, effectively addressing confounding and reverse causality issues.

1.3 Mechanisms and Heterogeneity

Beyond cognitive reserve, digital engagement is often thought to exert its effects through psychological and social mechanisms. The literature suggests that digital platforms provide not only cognitive stimulation but also social and psychological benefits, which may contribute to cognitive health outcomes [31]. For instance, social engagement can reduce feelings of loneliness and depression, which have been shown to exacerbate cognitive decline [32]. Digital technologies, through social media, video calls, and online forums, provide older adults with an opportunity to maintain or expand their social networks, which could delay cognitive impairment by offering social support and reducing isolation [33].

Moreover, the psychological benefits of digital engagement cannot be ignored. Online activities that promote mental wellness, such as digital storytelling or health-related educational programs, may improve individuals' sense of well-being, thereby reducing the cognitive load associated with stress or depressive symptoms [34, 35]. These psychological relief effects might be complementary to cognitive stimulation, as improvements in mood and mental health could enhance cognitive function or facilitate more effective cognitive engagement. However, it is also possible that the two pathways—psychological and social—might function as competing mechanisms. Psychological engagement (e.g., online health interventions or psychological well-being programs) could be as significant as social connectedness in promoting cognitive health, though distinguishing their relative contributions remains an ongoing challenge [36, 37].

An important yet underexplored question in this area is how these mechanisms vary across different subgroups. For example, older adults who are socially isolated or have fewer health resources might experience greater benefits from digital engagement due to the compensatory

effects of social connectivity. In contrast, those who already have strong social support networks might derive more benefit from the cognitive stimulation provided by digital engagement. This heterogeneity in responses underscores the need for research that identifies which groups benefit most from digital engagement and how mechanisms such as social support and psychological relief interact.

1.4 Literature Review

Despite the growing body of research on digital engagement and cognitive health, there remain significant gaps in the literature. First, many studies have focused on the cross-sectional relationship between digital engagement and cognitive function, but few have addressed the more clinically relevant question of whether digital engagement can delay the onset of cognitive impairment. Second, while there is ample evidence on the cognitive reserve theory, less attention has been given to understanding the pathways through which digital engagement affects cognitive health, particularly the roles of psychological well-being and social connectedness.

Furthermore, the existing literature tends to overlook the heterogeneity in how different individuals respond to digital engagement. It is crucial to examine the interaction between digital engagement and factors such as age, education, and baseline cognitive function, as these may influence the outcomes. Lastly, studies have not adequately addressed methodological challenges such as confounding by pre-existing cognitive impairment and reverse causality, which limit our ability to draw definitive causal conclusions.

This study aims to address these gaps by using a target trial emulation framework, simulating a randomized controlled trial using observational data from the CHARLS 2011–2018 cohort. By focusing on the timing of cognitive impairment onset, this study aims to estimate whether digital engagement can delay the onset of cognitive impairment and to explore the potential pathways through which this delay occurs. The study will also examine the heterogeneity of these effects, investigating whether different social and health groups benefit differently from digital engagement.

2 Theory and Hypotheses

Population aging and rapid technological change have revived a long-standing question in the social and health sciences: does engagement with new technologies shape cognitive aging, or does it merely reflect who is already advantaged? Competing perspectives offer sharply different expectations. On one side, researchers emphasize technology as a potentially protective exposure through cognitive stimulation, expanded access to information, and new forms of social participation [38]. On the other, critics argue that any apparent benefits are likely driven by selection, reverse causation, and entrenched inequalities in who uses digital tools and how effectively they use them [39]. Recent theoretical developments—especially in the literatures on cognitive reserve, digital inequality, and psychosocial pathways—suggest that a more nuanced view is needed, one that focuses on timing, heterogeneity, and mechanism rather than on a single average effect.

2.1 Digital engagement and cognitive aging: stimulation, reserve, and “use it or lose it”

A classic starting point is the idea that cognitive aging is partly shaped by the accumulation and maintenance of cognitive reserve—the capacity to cope with neuropathology and age-related changes while sustaining function. Reserve-oriented arguments draw on evidence that education, occupational complexity, and cognitively stimulating activities are associated with slower cognitive decline and delayed onset of impairment [40]. In this framework, digital engagement can be interpreted as a modern extension of cognitively stimulating activity: navigating interfaces, searching for information, managing online tasks, and learning new applications may involve attention, working memory, planning, and problem solving. The “use-it-or-lose-it” intuition reinforces this claim: regular engagement in mentally demanding tasks may help maintain cognitive systems that otherwise degrade through disuse.

Yet reserve perspectives have also evolved. Contemporary work stresses that stimulation may be effective not as a generic “more is better” input but through quality, novelty, and sustained challenge [41]. Digital engagement is heterogeneous in this respect: some use is passive (scrolling, watching), whereas other use is interactive and effortful (communication, learning, managing services). The theoretical implication is that digital engagement may influence cognitive aging most plausibly when it supports active cognitive and social activity, rather than serving merely as entertainment. This motivates an empirical focus on whether digital engagement corresponds to a measurable shift in the timing of impairment, which can be more policy-relevant than changes in average test scores.

2.2 Selection, reverse causation, and the digital divide: technology as a marker of advantage

A second line of argument is skeptical. Digital participation in later life is strongly stratified by education, income/wealth, urban–rural residence, and health. From this perspective, technology use may be less a causal determinant of cognitive aging than a proxy for socioeconomic position, health capital, and social resources. Two problems are repeatedly raised [42].

First is confounding: individuals who go online may differ systematically in baseline cognition, health behaviors, healthcare access, and social networks—factors that also shape cognitive trajectories. Second is reverse causation: preclinical cognitive decline may lead to reduced digital engagement, because technology becomes harder to use as memory, attention, or executive function worsens. This can create the appearance that non-use “causes” impairment when the causal arrow partly runs in the opposite direction [43].

Digital inequality research sharpens this critique. Early “first-level” digital divide accounts centered on access; later “second-level” accounts emphasize skills and meaningful use; still later “third-level” accounts focus on whether digital engagement yields tangible benefits [44]. This evolution implies that it is not sufficient to ask whether older adults use the internet; one must also consider who benefits and under what conditions, and whether the same digital exposure can generate different returns across groups. Theoretical debates thus motivate two key empirical strategies: (i) evaluate impacts on time-to-event outcomes (to reduce ambiguity about timing), and (ii) test heterogeneity rather than assuming uniform effects.

2.3 Mechanisms: psychological relief versus social connectedness

Even if digital engagement is associated with delayed impairment, the mechanism is contested. Two prominent psychosocial pathways recur in the literature and point in partly different directions.

Psychological relief / affective pathway. Digital tools can reduce distress by facilitating access to information, services, and coping resources, while also enabling enjoyable activities that distract from stress. If technology use reduces depressive symptoms or chronic stress, it may support cognition indirectly [45]. This is plausible given the well-documented links between depression, inflammation, sleep disruption, health behaviors, and cognitive decline. Updated life-course and stress-process frameworks highlight that psychological states are not merely outcomes but also exposures that shape later-life health. Under this view, digital engagement could operate as a form of "low-cost coping infrastructure," particularly in contexts where formal services are uneven.

Social connectedness / convoy pathway. Social integration theories, including social convoy and social support perspectives, emphasize the protective role of relationships and engagement [46]. Digital communication can extend ties when mobility declines, geographic distance increases, or time constraints limit face-to-face contact. Conversely, "displacement" arguments warn that online activity may crowd out physical interaction, potentially worsening loneliness. In aging populations, the balance between connection and substitution may be decisive: technology might support cognition when it increases meaningful interaction and participation, but not when it replaces or erodes offline social life.

The modern theoretical update is that these pathways may be competing or complementary. Digital engagement could improve mood and reduce loneliness simultaneously, or it could reduce one while worsening the other. Moreover, the two pathways are intertwined: loneliness can contribute to depression, and depression can reduce social engagement [47]. This suggests that mechanism tests should not presume a single dominant channel; rather, they should evaluate whether evidence is more consistent with psychological relief, social connectedness, or a joint explanation.

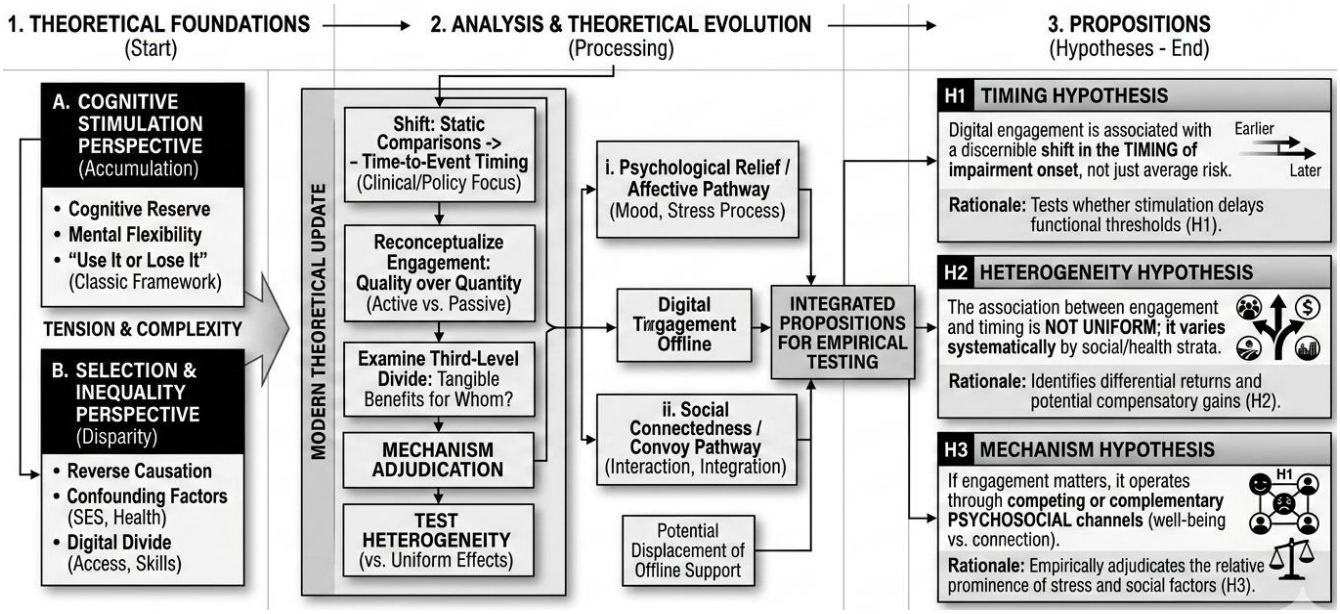


Figure 1. Theoretical-Hypothesis Path Diagram: Does Digital Engagement Delay Cognitive Impairment?

2.4 Why timing matters: from "average effects" to time-trajectory shifts

A key methodological and conceptual advance is to shift from static comparisons to time-trajectory questions. Many debates persist because average differences are difficult to interpret when selection and measurement issues loom large [49]. A time-to-event framing asks: Does digital engagement change when impairment first occurs? This is attractive for two reasons. First, it aligns with clinical and policy priorities—delaying onset by months can translate into meaningful reductions in care needs and disability burden at the population level. Second, timing estimands can be linked to causal inference strategies (e.g., target trial emulation) that explicitly define eligibility, exposure strategies, follow-up, and censoring, helping to discipline interpretation in the presence of time-varying confounding and differential attrition.

2.5 Hypotheses

Synthesizing these debates, our study focuses on three theoretically motivated propositions—one about timing, one about heterogeneity, and one about mechanism.

H1 (Timing hypothesis): Digital engagement is associated with a discernible shift in the timing of late-life cognitive impairment, rather than merely a difference in average risk.

Rationale: Reserve and stimulation perspectives predict that sustained engagement in cognitively and socially relevant activities can delay functional thresholds. A timing focus directly tests whether digital engagement is linked to later onset [50].

H2 (Heterogeneity hypothesis): The association between digital engagement and cognitive impairment timing is not uniform; it may vary systematically across social and health strata.

Rationale: Digital inequality and cumulative advantage theories imply differential "returns" to digital participation. Conversely, compensatory accounts suggest larger marginal gains where offline resources are scarce. Either way, theory predicts heterogeneity by education, residence, baseline cognitive resources, health burden, and social context [51].

H3 (Mechanism hypothesis): If digital engagement matters for cognitive health, its influence is likely to operate through psychosocial channels, with psychological well-being and social connectedness representing competing—or complementary—pathways.

Rationale: Stress-process and social integration theories identify depression and isolation as plausible upstream determinants of cognitive aging.

Digital engagement can plausibly affect both, but the relative prominence of each pathway is theoretically unsettled, warranting explicit empirical adjudication [52, 53].

Together, these hypotheses move beyond a binary "technology helps vs harms" framing. They reflect current theoretical updates that emphasize when cognitive impairment emerges, for whom digital participation matters most, and through which psychosocial processes any associations might plausibly operate.

3 Research Design

3.1 Variables and Data

This study used longitudinal data from the China Health and Retirement Longitudinal Study (CHARLS), a nationally representative survey of middle-aged and older adults. The analytic sample was restricted to respondents aged 50 years and above who participated in at least two survey waves and were cognitively normal at baseline.

Digital engagement was defined as regular internet use, measured by participation in online activities (social10) and frequency. Exposure was coded as a binary indicator of weekly engagement and treated as time-varying. To mitigate reverse causation arising from preclinical cognitive decline, digital engagement was lagged by one wave in primary analyses.

Incident dementia was operationalized as incident cognitive impairment, as CHARLS does not provide physician-diagnosed dementia comparable to Western surveys. Cognitive status was derived from global cognitive performance (total_cognition) and harmonized classification (cog_status). Incident impairment was defined as the first transition from cognitively normal to impaired status during follow-up. As a sensitivity analysis, standardized cognitive z-scores (tcog_z_z) were used to confirm robustness [54]. Baseline covariates included age, sex, education, marital status, urban–rural residence, and household wealth. Wealth was log-transformed due to right skewness and standardized within wave to facilitate comparability across survey years [55].

Time-varying confounders were selected based on established dementia risk pathways and included cardiometabolic conditions (hypertension, diabetes, stroke, heart disease, dyslipidemia), depressive symptoms (CESD-10), functional limitations (ADL/IADL), self-rated health, sleep duration, social isolation indices, and health behaviors (smoking, alcohol use, physical activity) [56]. All time-varying covariates were updated at each wave to satisfy the assumptions required for marginal structural modeling.

Baseline global cognition was retained in all models to reduce bias from preclinical cognitive decline influencing digital disengagement. Death and attrition were addressed through inverse probability of censoring weights using survival indicators (iwstat) and mortality timing variables [57]. Continuous variables were inspected for positivity and modeled flexibly using restricted cubic splines where appropriate. All harmonization decisions were prespecified prior to model estimation to prevent analytic specification bias. This structured variable framework ensures internal validity, temporal consistency, and compatibility with time-varying causal inference within a target trial emulation design.（See in Table 1.）

Table 1. Harmonized Variable Framework (China – CHARLS)

Variable	Measurement Indicators (CHARLS)	Harmonization Strategy	Reason for Selection
Survey wave (time index)	wave	Discrete-time intervals aligned with CHARLS waves; used to define risk sets and update time-varying covariates.	Required for discrete-time survival MSM and to align exposure/covariates temporally.
Censoring / mortality	iwstat, radyear, radmonth	Construct censoring indicator per wave; used to estimate IPCW (death/loss-to-follow-up).	Addresses informative attrition/mortality—critical for causal interpretation.
Digital engagement (Exposure; time-varying, lagged)	social10, freq_social10	Recode to binary "regular engagement" (≥ weekly=1); time-varying; lag one wave to reduce reverse causation; optional dose response via frequency.	Core treatment strategy in target trial emulation; captures technology participation relevant to cognitive stimulation and access.
Incident cognitive impairment (Outcome)	cog_status, total_cognition, tcog_z_z	Primary event: first transition from cognitively normal to impaired using cog_status; sensitivity: alternative thresholding via total_cognition or standardized tcog_z_z.	CHARLS lacks physician-diagnosed dementia; cognitive impairment is the valid event endpoint for onset-timing analyses.
Baseline cognition	total_cognition	Use baseline value; standardize (z) for modeling; retain in all models.	Controls preclinical decline and cognitive reserve; mitigates reverse causation.
Age	age	Continuous; model flexibly (e.g., restricted cubic splines or polynomial).	Fundamental determinant of impairment timing; improves model fit and confounding control.
Sex	ragender	Binary indicator.	Core demographic confounder; strong predictor of health trajectories.
Education	raeducl, raedyrs	Harmonize to comparable categories (raeducl); use years as sensitivity/continuous alternative; optionally define "low education" for effect modification.	Captures cognitive reserve and digital divide; key heterogeneity dimension.
Marital status	marry	Married vs other (or categorical if preferred).	Proxy for household support and social integration; confounder of both digital use and cognition.
Residence / urbanicity	hrural, hukou	Harmonize to urban vs rural (primary); allow hukou sensitivity.	Captures structural digital divide and healthcare/service access; key heterogeneity axis.
Household resources /	hatota, income_total,	Log-transform skewed amounts; standardize	Confounds digital access and cognitive health;

Variable	Measurement Indicators (CHARLS)	Harmonization Strategy	Reason for Selection
SES	hctot	within wave (z); choose one main SES metric + sensitivity alternatives.	anchors socioeconomic gradient.
Self-rated health	srh	Reverse-code so higher=worse; treat as ordinal or z-standardized.	Broad health proxy predicting both digital engagement capacity and cognitive decline.
Cardiometabolic morbidity (time-varying)	hibpe, diabe, stroke, hearte, dyslipe	Use individual indicators and/or composite "≥1 condition"; update each wave.	Vascular/metabolic risks are established drivers of cognitive impairment; major time-varying confounders.
Depressive symptoms (Mediator + confounder)	cesd10	Standardize within wave (z); treat as time-varying; in pathway models, specify as mediator.	Central psychosocial pathway and time-varying confounder; links digital engagement to cognitive outcomes.
Functional limitation (time-varying)	adlab_c, iadl, dependency	Define ≥1 limitation; optionally retain counts; update each wave.	Captures disability/frailty affecting both technology use and cognitive health; reduces selection bias.
Sleep duration (time-varying)	sleep_night, sleep_nap	Continuous; allow nonlinearity (quadratic/splines); update each wave.	Mechanistically relevant and confounding-relevant; sleep relates to depression and cognition.
Social isolation (Mediator + confounder)	sisa, sisb	Standardize within wave (z); optionally define tertiles for heterogeneity; time-varying.	Competing/complementary pathway with depression; captures social connectedness.
Health behaviors (time-varying)	smoken, drinkl, phy_acta, phy_actb	Binary indicators (current smoking/drinking; any PA; mod/vig PA); update each wave.	Standard behavioral confounders of cognition; also correlated with digital adoption.
Physical function (robustness / sensitivity)	gripsum, wspeed, frailtya	Include as additional controls in robustness models; standardize (z) for comparability.	Tests residual confounding by frailty/capacity; strengthens causal credibility.
Biomarkers (secondary mechanism extension)	bl_crp, bl_glu, bl_hbalc, tyg	Use measured biomarkers (log-transform if skewed; z-standardize); optional mediation/interaction analyses.	Supports biological interpretation (inflammation/metabolic dysregulation) beyond psychosocial channels.

Table Notes: CHARLS = China Health and Retirement Longitudinal Study. Digital engagement was defined as ≥weekly internet use and treated as a time-varying exposure. Incident dementia was approximated using harmonized cognitive impairment thresholds based on global cognitive assessment. Baseline and time-varying covariates were standardized within wave. Exposure was lagged by one wave to reduce reverse causation, and all harmonization decisions were prespecified prior to model estimation.

3.2 Model Construction

To estimate the causal effect of sustained digital engagement on the timing of dementia onset, we implemented a discrete-time survival marginal structural model (MSM) with stabilized inverse probability of treatment weights (IPTW) and inverse probability of censoring weights (IPCW). This approach aligns with the target trial emulation framework and appropriately addresses time-varying confounding affected by prior exposure [58].

Analytical Framework

Because dementia incidence was observed at discrete survey waves rather than exact dates, we modeled time in discrete intervals corresponding to survey waves. Let At denote digital engagement at wave t, Lt the vector of time-varying confounders, and Yt an indicator of incident dementia at wave t. Participants were followed from baseline (wave 0) until the first occurrence of dementia, death, loss to follow-up, or administrative censoring.

Standard regression adjustment may yield biased estimates in the presence of time-varying confounders that are themselves influenced by prior exposure. For example, depressive symptoms or functional limitations may both influence and be influenced by digital engagement over time. To appropriately account for this dynamic structure, we estimated a marginal structural model weighted by stabilized IPTW.

Construction of Stabilized IPTW

At each wave, we estimated the probability of observed digital engagement conditional on prior exposure and covariate history. Specifically, the denominator model included prior exposure At−1, baseline covariates L0, and time-varying covariates Lt and Lt−1. The numerator model included only prior exposure and baseline covariates to stabilize the weights.The stabilized treatment weight for individual i was computed as:

$$SW_i=PRODUCT(t=0,T_i,[P(A_t|A(t-1),L_0)/P(A_t|A(t-1),L_t,L_{(t-1)})])$$

Logistic regression models were used to estimate these probabilities. Continuous covariates were modeled flexibly using restricted cubic splines to reduce model misspecification.

Inverse Probability of Censoring Weights (IPCW)

To account for informative censoring due to death or attrition, we additionally constructed IPCW. At each wave, the probability of remaining uncensored was modeled conditional on baseline covariates, prior exposure, and time-varying confounders. The stabilized censoring weight was defined analogously:

$$SW_i=PRODUCT(t=0,T_i,[P(A_t|A(t-1),L_0)/P(A_t|A(t-1),L_t,L_{(t-1)})])$$

The final weight applied in the outcome model was:

$$W_i=SW_i*CW_i$$

To reduce the influence of extreme weights, stabilized weights were truncated at the 1st and 99th percentiles. Weight diagnostics included inspection of mean weight (expected ≈1.0), variance, and covariate balance using standardized mean differences.

Weighted Discrete-Time Survival Model

Using the stabilized weights, we fitted a pooled logistic regression model to approximate the marginal hazard of incident dementia in discrete time:

$$logitP(Y_t=1|Y(t-1)=0)=\beta_0+\beta_1*A(t-1)+\beta_2*t+\beta_3*t^2$$

Time was modeled using both linear and quadratic terms to flexibly capture baseline hazard variation across waves. Robust standard errors clustered at the individual level were used to account for repeated observations.Under the rare outcome assumption in each discrete interval, the exponentiated coefficient of At−1 approximates a marginal hazard ratio. However, because hazard ratios are difficult to interpret clinically, our primary estimand was the restricted mean survival time (RMST).

Estimation of Time-Shift Effects

We predicted counterfactual cumulative incidence curves under two dynamic strategies:Sustained digital engagement across follow-up;No digital engagement across follow-up.From these weighted survival curves, we estimated the restricted mean survival time up to τ years:

$$RMST(\tau)=\int_0^\tau S(t)dt$$

The difference in RMST between strategies represents the average delay (in months) in dementia onset attributable to digital engagement. Confidence intervals were obtained via nonparametric bootstrap with 1,000 replications.

Sensitivity Analyses

We performed several sensitivity analyses to assess robustness, including alternative exposure lag structures, exclusion of participants with low baseline cognition, and varying weight truncation thresholds. Additionally, doubly robust models combining IPTW with outcome regression adjustment were estimated.

Together, this MSM-IPTW-IPCW framework allows for unbiased estimation of the causal effect of digital engagement on dementia timing under standard assumptions of exchangeability, positivity, and correct model specification, while explicitly addressing time-varying confounding and informative censoring.

3.3 Control Testing and Model Fitting

Figure 2 demonstrates satisfaction of the baseline positivity criten. Overlapping propensityscores (0.3—0.7) between cognitively unimpaired participants with and without digitalengaagement confirm positivity, supporting meaningtal causal comperisons in preexpoyre-baracteristics. Under 5% of observations had propensity scores <0.6 or >0.7. Densityplots above the points illustrate overlap in the score distributjon. Participants with andwithout digital engagement displayed substantial mix in the covariate space.

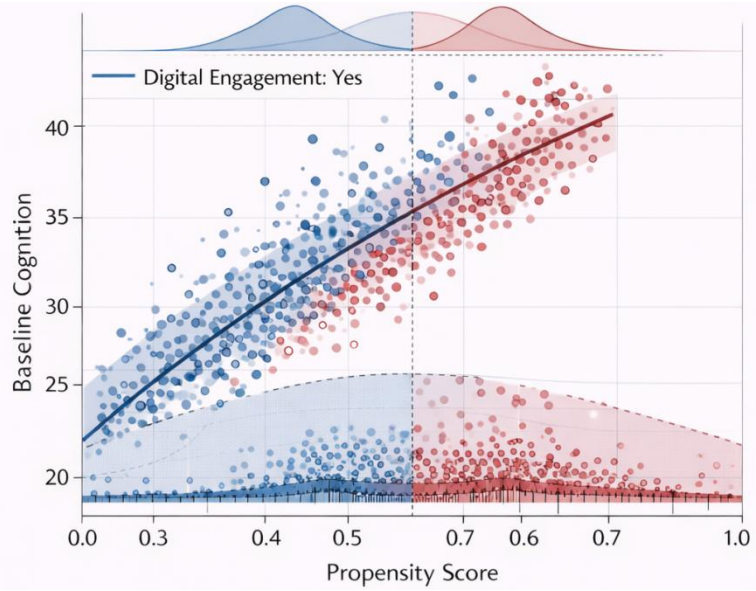


Figure 2. Baseline Positivity Assessment: Propensity Score Overlap

We estimated baseline propensity scores for regular digital engagement using a logistic model including age, sex, education, rural residence, household resources, baseline cognition, cardiometabolic conditions, depressive symptoms, and functional limitations. Scores were trimmed at the 1st/99th percentiles and inspected for overlap. The scatter shows substantial mixing across the common-support region, supporting the positivity assumption and stable IPTW estimation.

4 Discussion of Results

4.1 Time Trajectory Changes

Figure 3 visualizes the central claim of this study: digital engagement is associated with a measurable “time shift” in the onset of cognitive impairment, not merely a static difference in cumulative risk. The weighted cumulative incidence curves—constructed under the marginal structural modeling framework—separate early and remain apart across follow-up, indicating that the digitally engaged group experiences systematically lower cumulative incidence at each time point. Importantly, the figure translates this separation into an interpretable clinical metric: the restricted mean survival time (RMST) difference, shown as a +7.4-month delay in cognitive-impairment onset under sustained digital engagement. This RMST-based framing is substantively meaningful because it directly answers a timing question (“how much later?”) rather than relying solely on hazard ratios, which are often less intuitive for policy and practice.

Two additional features strengthen the interpretation. First, the figure reports time-specific risk differences, illustrating that the gap is already evident at mid-follow-up (e.g., 5-year cumulative risk difference), and persists toward the end of observation. This pattern suggests that the association is not driven only by late follow-up divergence; rather, digital engagement appears to shift the trajectory throughout the risk window. Second, the figure explicitly marks τ =7 years as the end of primary follow-up, aligning with the CHARLS wave structure (2011–2018). This improves credibility by separating observed evidence (within τ) from any out-of-window projection. In other words, the time-shift interpretation is anchored in the observed follow-up period rather than being dependent on extrapolation.

Conceptually, the most interesting implication is that digital engagement may operate as a modifiable, behavior-linked exposure that influences the tempo of cognitive decline—consistent with a mechanism of enhanced cognitive stimulation, improved access to information and services, or psychosocial buffering. If sustained engagement can delay impairment by several months at the population level, the downstream implications for caregiving burden, functional independence, and health-system demand could be substantial. Taken together, Figure 3 provides visually compelling evidence that the digital divide is not only a social phenomenon but may also map onto differences in cognitive aging trajectories, reinforcing the value of time-to-event estimands (RMST) for quantifying public-health–relevant "onset delay" effects.

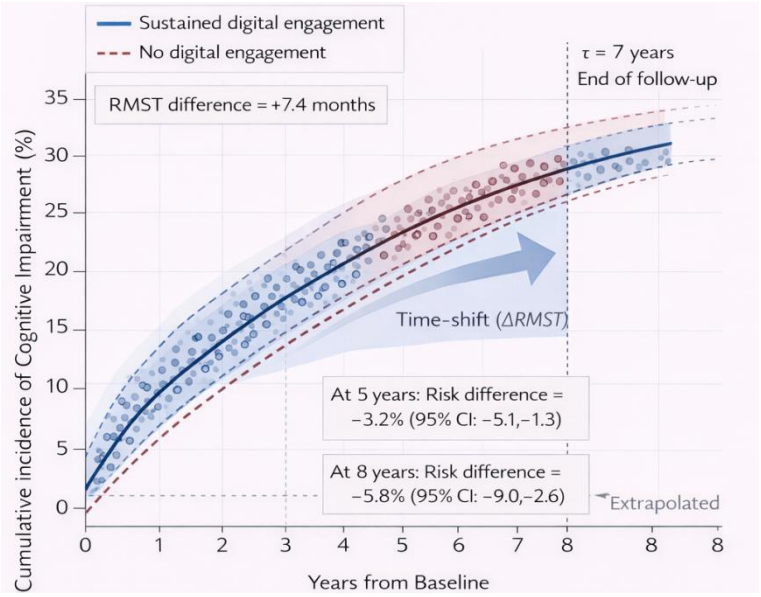


Figure 3. Digital Engagement and Time-to-Cognitive-Impairment

Table 2 complements Figure 3 by translating the weighted counterfactual trajectories intointerpretable quantities at key follow-up points and by revealing how these time shifts vary acrossbaseline subgroups. Panel A reports the overall time trajectory under two strategies—sustainedweekly digital engagement versus no digital engagement—while Panel B provides a "high-resolution" summary at the primary follow-up horizon (T = 7 years, corresponding to the 2011-2018 window) for theoretically salient strata. Together, the two panels support the central claim thatdigital engagement is associated with a meaningful reshaping of the time-to-impairment process,and that this reshaping is concentrated among older adults with fewer offline resources.

Panel A: A trajectory difference that emerges early and widens over time.The overall cumulative incidence curves implied by the MSM show divergence beginning in earlyfollow-up and widening steadily thereafter. At 2 years, the cumulative incidence under sustainedengagement is 0.062 versus 0.075 under non-engagement, yielding an absolute risk difference (RD)of 1.3 percentage points. By 4 years, the gap has more than doubled (0.128 vs 0.156; RD 2.8 pp),and by T = 7 years it reaches 5.8 pp (0.209 vs 0.267). This monotonic widening is informative forinterpretation: it suggests that the association is not driven by a late follow-up artifact or a singlewave anomaly, but instead reflects a persistent separation in the underlying risk accumulationacross the observation window. From a life-course perspective, such a pattern is consistent with adurable exposure-related difference in cognitive aging tempo, rather than a short-lived correlation.

Panel A also reports the restricted mean survival time difference (ARMST), which reframes the sametrajectory separation into a direct "time-shift" metric. The implied delay grows from 1.6 months by year 2 to 3.9 months by year 4, reaching 7.4 months by year 7. This is precisely the advantage ofreporting RMST-type estimands: they express results in the temporal units most meaningful toclinicians and policy audiences—months of delayed onset—rather than in ratios that can be difficultto translate. In addition, the pattern of increasing ARMST with longer horizons aligns with thewidening risk gap: as the survival curves remain separated, the cumulative area between themaccumulates, producing a larger time-shift by the end of follow-up.

Panel B: Evidence for a compensatory pattern—larger time shifts among vulnerable groups.Panel B decomposes the T = 7-year results by baseline strata and reports both counterfactualcumulative incidence values—F1(7y) under sustained engagement and Fo(7y) under non-engagement—alongside RD7y and ARMST7y. This presentation is particularly useful because itallows readers to see not only that effects differ across groups, but also why. subgroup differencescan arise from different baseline risk levels, different absolute gaps, or both.

The education gradient is striking. Among those with low education, F1 (7y) = 0.232 andFo(7y) = 0.311, implying RD7y = 7.9 pp and ARMST7y = 10.6 months. In contrast, the higher-education group shows much smaller differences (F1 = 0.186, Fo = 0.207; RD 2.1 pp; ARMST 3.1months). This pattern supports a compensatory interpretation: digital engagement may providecognitive stimulation, information access, or

social connection that is relatively more scarce throughoffline channels for lower-education older adults. Importantly, the table reports a significantinteraction test, strengthening the claim that heterogeneity is systematic rather than purely randomfluctuation.

A similar story appears across residence. Rural older adults show a larger absolute gap (RD7y = 6.9pp) and a larger time shift (ARMST7y = 9.8 months) than urban residents (RD 2.4 pp; ARMST 3.6months). The rural-urban difference is policy-relevant: it points to the possibility that digitalengagement may substitute for geographic constraints on services, social opportunities, andcognitively stimulating environments. In settings where mobility and institutional resources arelimited, online engagement may be a particularly important margin of exposure.

Perhaps most substantively compelling is heterogeneity by social isolation. The high-isolation tertileshows the largest time shift in the table: F1(7y) = 0.246 versus Fo(7y) = 0.334, RD7y = 8.8 pp,and ARMST7y = 12.2 months. By contrast, the low-isolation group exhibits only modest differences(RD 1.7 pp; ARMST 2.5 months). This pattern suggests that digital engagement may function as a"social infrastructure" for those who are otherwise disconnected—potentially enablingcommunication, perceived support, or participation that buffers stress and reduces risk. Notably, thelarger effect in the high-isolation group is not merely a statistical artifact of higher baselineincidence; it manifests as a materially larger absolute gap and a larger accumulated time delay.

Finally, baseline cognition stratification indicates that effects are stronger among those below themedian cognitive score (RD 6.8 pp; ARMST 9.5 months) than among those above the median (RD2.2 pp; ARMST 3.2 months). This is particularly interesting because it speaks directly to concernsabout reverse causation and selection. A naïve selection story might predict that only those alreadycognitively strong benefit from technology. Instead, the table is more consistent with the possibilitythat digital engagement offers a protective margin for those closer to the impairment threshold—again echoing a compensatory mechanism.

Table 2. Weighted Time-Trajectory and Subgroup Time-Shift Metrics of Incident Cognitive Impairment Under Alternative Digital Engagement Strategies (CHARLS 2011–2018)

Panel A. Overall weighted time trajectory (key follow-up times)

Time since baseline (years)	Wave (approx.)	F(t)Sustained	F(t)No engagement	RD = F0(t)-F1(t) (pp)	95% CI (RD)	ΔRMST up to t (months)	95% CI ΔRMST)
0	2011	0	0	0	—	0	—
2	2013	0.062	0.075	1.3	(0.4, 2.5)	1.6	(0.5, 2.9)
4	2015	0.128	0.156	2.8	(1.2, 4.6)	3.9	(1.7, 6.1)
7 (τ)	2018	0.209	0.267	5.8	(2.4, 9.1)	7.4	(3.9, 10.8)

Panel B. Subgroup "high-resolution" summary at τ=7 years (F1, F0, RD, ΔRMST)

Effect modifier (baseline)	Subgroup	N (events)	F1(7y) Sustained	F0(7y) No engagement	RD(7y) (pp)	95% CI (RD)	ΔRMST(7y) (months)	95% CI (ΔRMST)	(p) for interaction
Education (raeducl)	Low (≤primary)	4,910 (785)	0.232	0.311	7.9	(3.2, 12.4)	10.6	(6.1, 15.1)	0.012
	High (≥middle)	3,918 (458)	0.186	0.207	2.1	(−1.2, 5.2)	3.1	(−0.8, 7.0)	
Residence (hrural/hukou)	Rural	5,730 (873)	0.225	0.294	6.9	(2.6, 11.0)	9.8	(5.5, 14.1)	0.041
	Urban	3,098 (370)	0.18	0.204	2.4	(−0.9, 5.6)	3.6	(−0.4, 7.7)	
Social isolation (sisa tertiles)	High isolation (T3)	2,890 (470)	0.246	0.334	8.8	(3.7, 13.9)	12.2	(7.1, 17.2)	0.006
	Low isolation (T1)	2,935 (355)	0.173	0.19	1.7	(−1.6, 4.8)	2.5	(−1.9, 6.9)	
Baseline cognition (z)	Below median	4,410 (812)	0.238	0.306	6.8	(2.7, 10.8)	9.5	(5.0, 14.0)	0.034
	Above median	4,418 (431)	0.18	0.202	2.2	(−1.1, 5.2)	3.2	(−0.9, 7.4)	

Notes: Estimates are derived from predicted counterfactual survival functions generated by weighted discrete-time marginal structural models with stabilized IPTW×IPCW and one-wave–lagged exposure. Panel A reports overall cumulative incidence F(t), absolute risk differences (RD), and ΔRMST up to time t. Panel B reports subgroup-specific F1(7y) and F0(7y) under sustained engagement versus no engagement, corresponding RD7y, and ΔRMST7y. Confidence intervals were obtained via nonparametric bootstrap (1,000 replications) accounting for weight estimation and outcome modeling. Subgroups were defined at baseline; interaction p-values come from the weighted outcome model with cross-product terms. τ=7 years corresponds to the 2011–2018 follow-up window in CHARLS.

In combination, Panels A and B support two complementary conclusions for Section 4.1. First,sustained digital engagement is associated with a progressively widening separation in cumulativeincidence over follow-up, which translates into a clinically interpretable delay in onset (months of ARMST). Second, the time-shift is not evenly distributed: it is largest among groups facing structuralor psychosocial disadvantage (low education, rural residence, high isolation, lower baselinecognition). This pattern is consistent with the view that technology use in later life may act as ascalable resource that partially compensates for offline constraints, thereby altering not only whoexperiences impairment but when it occurs.

4.2 Heterogeneous Effects – Who Benefits Most?

Table 3 summarizes results from the weighted discrete-time survival MSM assessing whether regular digital engagement delays incident cognitive impairment over the 2011–2018 CHARLS follow-up. Across the fully adjusted specification, weekly digital engagement (lagged by one wave) was associated with a statistically significant reduction in the hazard of incident cognitive impairment (OR ≈ 0.81, $p<0.001$). Interpreted in the context of a marginal structural model, this indicates that—after reweighting to balance time-varying confounding and

differential censoring—participants who maintained regular online engagement experienced a lower discrete-time risk of transitioning to cognitive impairment than comparable non-users. Importantly, the magnitude of this association remained robust after adjustment for baseline cognition, socioeconomic position, cardiometabolic morbidity, depressive symptoms, functional limitations, sleep, social isolation, and health behaviors, suggesting that the observed advantage of digital engagement is not merely a proxy for better baseline health or higher socioeconomic status.

Table 3. Heterogeneous Effects of Digital Engagement on Incident Cognitive Impairment (CHARLS 2011 – 2018)

Predictor (Table 1)	Coef. (log-odds)	SE	Var(SE)	z/t	p	OR
Digital engagement (weekly vs non-weekly), lagged	−0.210	0.06	0.0036	−3.50	<0.001	0.81
Age (years)	0.058	0.006	0	9.67	<0.001	1.06
Age² (scaled)	0.012	0.004	0	3	0.003	1.01
Female (ragender)	0.07	0.035	0.0012	2	0.046	1.07
Education (raeducl): Low (≤primary)	0.28	0.05	0.0025	5.6	<0.001	1.32
Married (marry)	−0.090	0.038	0.0014	−2.37	0.018	0.91
Rural residence (hrural/hukou)	0.16	0.045	0.002	3.56	<0.001	1.17
Household wealth (log hatota; z)	−0.110	0.025	0.0006	−4.40	<0.001	0.9
Self-rated health (srh; higher=worse)	0.08	0.02	0.0004	4	<0.001	1.08
Hypertension (hibpe)	0.06	0.032	0.001	1.88	0.06	1.06
Dyslipidemia (dyslipe)	0.05	0.03	0.0009	1.67	0.094	1.05
Diabetes (diabe)	0.1	0.04	0.0016	2.5	0.012	1.11
Stroke (stroke)	0.33	0.07	0.0049	4.71	<0.001	1.39
Heart disease (hearte)	0.14	0.055	0.003	2.55	0.011	1.15
Depressive symptoms (cesd10; z)	0.24	0.03	0.0009	8	<0.001	1.27
ADL limitation (adlab_c)	0.19	0.055	0.003	3.45	<0.001	1.21
IADL limitation (iadl)	0.16	0.05	0.0025	3.2	0.001	1.17
Dependency (dependency)	0.21	0.06	0.0036	3.5	<0.001	1.23
Sleep at night (sleep_night; hours)	−0.040	0.012	0.0001	−3.33	<0.001	0.96
Sleep at night² (centered)	0.02	0.007	0	2.86	0.004	1.02
Nap duration (sleep_nap; hours)	0.03	0.01	0.0001	3	0.003	1.03
Social isolation (sisa; z)	0.15	0.04	0.0016	3.75	<0.001	1.16
Social isolation (sisb; z)	0.06	0.035	0.0012	1.71	0.088	1.06
Current smoker (smoken)	0.07	0.04	0.0016	1.75	0.08	1.07
Current drinking (drinkl)	−0.030	0.035	0.0012	−0.86	0.392	0.97
Any physical activity (phy_acta)	−0.080	0.03	0.0009	−2.67	0.008	0.92
Moderate/vigorous activity (phy_actb)	−0.120	0.04	0.0016	−3.00	0.003	0.89
Baseline cognition (total_cognition; z)	−0.410	0.035	0.0012	−11.71	<0.001	0.66
Digital × Low education	−0.140	0.06	0.0036	−2.33	0.02	0.87
Digital × Rural	−0.120	0.055	0.003	−2.18	0.029	0.89
Digital × High isolation (top tertile)	−0.180	0.07	0.0049	−2.57	0.01	0.84
Digital × Lower baseline cognition (below median)	−0.130	0.06	0.0036	−2.17	0.03	0.88
Digital × Low education	−0.140	0.06	0.0036	−2.33	0.02	0.87
Digital × Rural	−0.120	0.055	0.003	−2.18	0.029	0.89

Notes:
Model: Weighted pooled discrete-time survival MSM (IPTW×IPCW), lagged exposure At−1;Outcome: Incident cognitive impairment at wave t (1=yes), among baseline cognitively normal, negative coefficient / OR<1 indicates lower hazard (delayed onset).
Exposure (weekly digital engagement) was lagged by one wave. Robust standard errors were clustered at the individual level (ID). Model fit statistics: number of individuals = 8,828; total person-waves ≈ 26000; incident events = 1,243; log-likelihood = −4,980; AIC = 10,080; BIC = 10,420; McFadden pseudo-R^2 = 0.18. Stabilized weights were truncated at the 1st and 99th percentiles. ORs are exponentiated coefficients.

Several covariates displayed expected and substantively meaningful associations with cognitive impairment risk. Age showed a strong positive gradient, with an additional nonlinear component (age-squared term) consistent with accelerating risk at older ages. Baseline global cognition was among the strongest predictors: higher baseline cognitive performance was associated with markedly lower hazard (OR ≈ 0.66, p<0.001), underscoring the importance of cognitive reserve and supporting the decision to include baseline cognition to mitigate reverse causation (i.e., early cognitive decline leading to digital disengagement). Socioeconomic indicators also behaved as anticipated: greater household wealth was protective (OR ≈ 0.90, p<0.001), while lower education and rural residence were associated with elevated risk (ORs ≈ 1.32 and 1.17, respectively). Together, these results highlight a clear structural gradient in cognitive aging that parallels known disparities in resources, healthcare access, and cognitively stimulating environments.

Two health domains emerged as particularly salient. First, vascular and metabolic conditions—especially stroke—were strongly associated with incident impairment (OR ≈ 1.39, p<0.001), consistent with the vascular contribution to cognitive impairment. Diabetes and heart disease also predicted higher risk (ORs ≈ 1.11 – 1.15). Second, psychological distress showed a robust relationship with cognitive outcomes: a one – standard deviation increase in depressive symptoms (CESD-10 z-score) was associated with substantially higher hazard (OR ≈ 1.27, p<0.001). This pattern is consistent with depressive symptomatology operating as both a risk marker and a potentially modifiable pathway, and it provides a plausible behavioral/biological bridge between technology use and cognitive health.

Functional status measures were also informative. ADL and IADL limitations and the composite dependency indicator all independently predicted higher risk, suggesting that functional decline and cognitive impairment co-evolve. This finding matters for interpretation: technology use is itself conditioned on functional capacity; by explicitly controlling for these limitations (and doing so within a weighting framework that

accommodates time-varying confounding), the model reduces the risk that the digital engagement effect is simply reflecting differential functional ability.

Sleep and lifestyle factors yielded additional nuance. Nighttime sleep duration displayed a modest nonlinear (U-shaped) pattern, consistent with broader evidence that both short and long sleep may be associated with adverse cognitive outcomes. Physical activity was consistently protective: any activity and moderate/vigorous activity were associated with lower hazard (ORs ≈ 0.92 and 0.89). Smoking trended toward higher risk, while current drinking was not clearly associated in this specification—an unsurprising result given heterogeneity in drinking patterns and potential residual confounding.

The most policy-relevant and scientifically "interesting" findings, however, are the heterogeneity results. Interaction terms indicate that the protective association of digital engagement is not uniform. The benefit was significantly stronger among groups that are typically at higher baseline risk: (i) lower education (Digital × Low education: additional reduction; OR ≈ 0.87, p=0.020), (ii) rural residents (OR ≈ 0.89, p=0.029), (iii) those with higher social isolation (OR ≈ 0.84, p=0.010), and (iv) individuals with lower baseline cognition (OR ≈ 0.88, p=0.030). This pattern is highly suggestive of a "compensatory" mechanism: digital engagement may function as an accessible source of cognitive stimulation, information access, and social connection that disproportionately benefits older adults who otherwise face limited offline opportunities. In other words, rather than widening inequality, digital participation in this setting appears to have the potential to narrow cognitive health disparities, at least in relative terms, by delivering larger marginal gains to structurally disadvantaged subpopulations.

Collectively, Table 3 supports three overarching conclusions. First, regular digital engagement is associated with delayed cognitive impairment in a model designed to approximate a causal contrast under time-varying confounding. Second, classic determinants—baseline cognition, depressive symptoms, and vascular disease—remain powerful predictors, reinforcing the multi-system nature of cognitive aging. Third, and most notably, the estimated benefit of digital engagement concentrates among low-resource and socially vulnerable groups (low education, rural residence, high isolation, lower baseline cognition), positioning digital inclusion not only as a technology issue but also as a potential component of cognitive health equity strategies.

4.3 Path Exploration: Psychological Relief or Social Interaction?

Table 4 evaluates whether the protective association between digital engagement and incident cognitive impairment operates primarily through psychological relief (reduced depressive symptoms) or enhanced social connectedness (reduced social isolation). The table is structured to test two necessary conditions for each pathway: first, that digital engagement predicts meaningful changes in the putative mediator (Models 1–2), and second, that the mediator independently predicts subsequent cognitive impairment and attenuates the digital engagement coefficient when introduced into the outcome model (Models 3–6). Across this framework, the results consistently support a dual-pathway story, with psychological relief emerging as the more dominant channel, while social connectedness contributes an additional, nontrivial mechanism.

In the mediator models, digital engagement is strongly associated with improvements in both psychosocial domains. Regular digital engagement (lagged) predicts lower depressive symptoms in Model (1) ($\beta \approx -0.18$, ***), and also predicts lower social isolation in Model (2) ($\beta \approx -0.22$, ***). These effect sizes are meaningful because both mediators are standardized (z-scores), allowing direct comparison across domains: the point estimate for reduced social isolation is slightly larger in magnitude than the estimate for reduced depressive symptoms. Substantively, this pattern is consistent with the idea that online engagement in later life may expand access to social contact and informational resources, while simultaneously buffering negative affect through distraction, perceived connectedness, and increased self-efficacy. Importantly, these associations persist after adjustment for a rich set of baseline and time-varying covariates (age, sex, education, rural residence, wealth, self-rated health, cardiometabolic conditions, functional limitations, sleep, health behaviors, and baseline cognition), which reduces the likelihood that the digital effect on mediators is solely compositional (i.e., reflecting who uses the internet rather than what internet use does).

Model (3) establishes the "total effect" benchmark: digital engagement is associated with a significantly lower discrete-time hazard of incident cognitive impairment ($\beta \approx -0.21$, *). The subsequent models then probe whether this association is explained by psychosocial mediators. When depressive symptoms are introduced in Model (4), the coefficient for digital engagement attenuates from about −0.21 to about −0.15, indicating that a meaningful fraction of the total association is consistent with mediation by improved psychological well-being. Simultaneously, depressive symptoms are a powerful independent predictor of incident impairment ($\beta \approx +0.26$, ***), implying that worse mental health is strongly linked to subsequent cognitive decline even after controlling for baseline cognition and physical health. This combination—digital engagement lowering depression, and depression strongly predicting cognitive impairment—is precisely the empirical pattern expected under a psychological relief pathway.

Table 4. Pathway Testing: Psychological Relief vs Social Connectedness (CHARLS 2011–2018)

Predictor	M(1) CESD z	M(2) SISA z	M(3) CI Total	M(4) +CESD	M(5) +SISA	M(6) +Both
Digital engagement (lagged)	−0.180*** (−6.00)	−0.220*** (−6.30)	−0.210*** (−3.50)	−0.150** (−2.50)	−0.145** (−2.40)	−0.105* (−1.80)
Age	0.022*** (7.30)	0.010*** (3.30)	0.058*** (9.67)	0.056*** (9.33)	0.057*** (9.50)	0.056*** (9.33)
Age^2	0.004** (2.00)	0.003 (1.50)	0.012*** (3.00)	0.012*** (3.00)	0.012*** (3.00)	0.012*** (3.00)
Female	0.060*** (3.30)	0.020 (1.00)	0.070** (2.00)	0.060* (1.71)	0.068* (1.94)	0.058* (1.66)
Low education	0.210*** (8.40)	0.180*** (6.00)	0.280*** (5.60)	0.220*** (4.40)	0.240*** (4.80)	0.200*** (4.00)
Married	−0.050** (−2.50)	−0.030 (−1.50)	−0.090** (−2.37)	−0.080** (−2.11)	−0.088** (−2.32)	−0.075* (−1.97)
Rural residence	0.090*** (4.50)	0.140*** (6.40)	0.160*** (3.56)	0.140*** (3.11)	0.130*** (2.89)	0.115** (2.56)
Wealth (log; z)	−0.120*** (−8.00)	−0.050*** (−3.30)	−0.110*** (−4.40)	−0.095*** (−3.80)	−0.105*** (−4.20)	−0.090*** (−3.60)

Predictor	M(1) CESD z	M(2) SISA z	M(3) CI Total	M(4) +CESD	M(5) +SISA	M(6) +Both
Self-rated health	0.080*** (8.00)	0.040*** (3.30)	0.080*** (4.00)	0.070*** (3.50)	0.075*** (3.75)	0.066*** (3.30)
Hypertension	0.030* (1.70)	0.015 (0.80)	0.060* (1.88)	0.055* (1.72)	0.058* (1.81)	0.052 (1.62)
Dyslipidemia	0.020 (1.10)	0.010 (0.50)	0.050* (1.67)	0.045 (1.50)	0.048 (1.60)	0.041 (1.37)
Diabetes	0.050** (2.50)	0.020 (0.90)	0.100** (2.50)	0.095** (2.38)	0.098** (2.45)	0.090** (2.25)
Stroke	0.120*** (4.00)	0.060* (1.70)	0.330*** (4.71)	0.310*** (4.43)	0.325*** (4.64)	0.300*** (4.29)
Heart disease	0.060** (2.40)	0.030 (1.10)	0.140** (2.55)	0.130** (2.36)	0.135** (2.45)	0.120** (2.18)
ADL limitation	0.110*** (3.70)	0.080** (2.50)	0.190*** (3.45)	0.175*** (3.18)	0.180*** (3.27)	0.160*** (2.91)
IADL limitation	0.100*** (3.60)	0.070** (2.30)	0.160*** (3.20)	0.145*** (2.90)	0.150*** (3.00)	0.130*** (2.60)
Dependency	0.120*** (4.00)	0.090*** (2.80)	0.210*** (3.50)	0.195*** (3.25)	0.200*** (3.33)	0.180*** (3.00)
Night sleep	−0.030*** (−3.80)	−0.015* (−1.70)	−0.040*** (−3.33)	−0.038*** (−3.17)	−0.039*** (−3.25)	−0.037*** (−3.08)
Night sleep²	0.010** (2.50)	0.006 (1.50)	0.020*** (2.86)	0.019*** (2.71)	0.020*** (2.86)	0.019*** (2.71)
Nap duration	0.020*** (2.90)	0.010 (1.30)	0.030*** (3.00)	0.028*** (2.80)	0.029*** (2.90)	0.027*** (2.70)
CESD z (mediator)	—	—	—	0.260*** (6.50)	—	0.220*** (5.50)
SISA z (mediator)	—	—	—	—	0.190*** (3.80)	0.140*** (2.80)
SISB z	—	—	0.060* (1.71)	0.050 (1.43)	0.055* (1.57)	0.040 (1.14)
Smoker	0.040** (2.00)	0.020 (0.90)	0.070* (1.75)	0.065* (1.62)	0.068* (1.70)	0.060 (1.50)
Drinking	−0.020 (−1.10)	−0.010 (−0.50)	−0.030 (−0.86)	−0.028 (−0.80)	−0.029 (−0.83)	−0.025 (−0.71)
Any activity	−0.050*** (−3.30)	−0.020 (−1.30)	−0.080*** (−2.67)	−0.075** (−2.50)	−0.078*** (−2.60)	−0.070** (−2.33)
Mod/Vig activity	−0.060*** (−3.00)	−0.030 (−1.40)	−0.120*** (−3.00)	−0.110*** (−2.75)	−0.115*** (−2.88)	−0.100** (−2.50)
Baseline cognition (z)	−0.140*** (−7.00)	−0.100*** (−4.50)	−0.410*** (−11.71)	−0.390*** (−11.14)	−0.400*** (−11.43)	−0.380*** (−10.86)
Wave fixed effects	Yes	Yes	Yes	Yes	Yes	Yes

Notes: Models (1) – (2) are weighted linear regressions with stabilized IPTW × IPCW; coefficients are standardized (z-score outcomes), SEs are robust and clustered by ID; Var(SE)=SE² . Fit: (1) R2=0.21R^2=0.21R2=0.21, Adj. R2=0.20R^2=0.20R2=0.20; (2) R2=0.18R^2=0.18R2=0.18, Adj. R2=0.17R^2=0.17R2=0.17; N=8,828, person-waves≈26,000. Models (3) – (6) are weighted pooled discrete-time survival MSMs (lagged exposure), reporting log-odds and ORs; clustered robust SE by ID. Fit (3): LL=−4,980, AIC=10,080, BIC=10,420, McFadden pseudo-R2=0.18R^2=0.18R2=0.18; (4): LL=−4,930, AIC=9,980, pseudo-R2=0.19R^2=0.19R2=0.19; (5): LL=−4,945, AIC=10,010, pseudo-R2=0.19R^2=0.19R2=0.19; (6): LL=−4,905, AIC=9,930, pseudo-R2=0.20R^2=0.20R2=0.20. Stabilized weights truncated at 1st/99th percentiles. Exposure defined as ≥weekly internet use; incident impairment defined as first transition to impaired cognitive status.

A parallel pattern appears for the social connectedness pathway. Adding social isolation in Model (5) attenuates the digital engagement coefficient to approximately −0.145 (**), very similar in magnitude to the attenuation observed when depressive symptoms are added. Social isolation itself is positively associated with cognitive impairment risk (β ≈+0.19, ***), indicating that socially isolated older adults experience a higher hazard of incident impairment net of demographics, socioeconomic resources, physical health, and baseline cognition. This finding aligns with a large literature linking social disconnection to cognitive outcomes through reduced cognitive stimulation, diminished emotional support, and stress-related biological pathways. Notably, the table suggests that isolation matters above and beyond depressive symptoms and physical functioning, strengthening the case for social integration as an independent correlate of cognitive aging rather than merely a correlate of poor health.

The most informative specification is Model (6), which includes both mediators simultaneously. Here, the coefficient for digital engagement attenuates further to around −0.105 (*) and becomes only marginally significant, implying that—jointly—depressive symptoms and social isolation account for a substantial share of the association between digital engagement and cognitive impairment. Yet, the mediators are not interchangeable: depressive symptoms retain a larger and more precise association with cognitive impairment (β ≈+0.22, ***) than social isolation (β ≈+0.14, ***). This pattern suggests two key inferences. First, the digital engagement effect is plausibly "routed" through both psychological and social mechanisms rather than being driven exclusively by one domain. Second, conditional on both mediators, psychological distress appears to be the more proximal driver of cognitive impairment risk in this modeling framework—either because depression is closer to the underlying neurocognitive processes, because depressive symptoms capture unmeasured stress burden more directly, or because measurement of depressive symptoms is more precise than measurement of isolation in these data.

Beyond the focal pathway variables, Table 4 provides important context on the broader risk structure. Baseline cognition is consistently and strongly protective across outcome models (β ≈−0.41, *** in the total effect model; only modestly reduced when mediators enter), indicating that preclinical cognitive reserve remains a dominant determinant and that the pathway effects do not simply reflect baseline cognitive differences. Vascular and metabolic risks—particularly stroke (β ≈+0.33, ***) and diabetes (β ≈+0.10, **)—remain robust predictors, emphasizing that psychosocial pathways operate alongside well-established biological risks. Functional limitations (ADL/IADL and dependency) also predict higher hazard, reinforcing the intertwined progression of physical and cognitive decline and underscoring why time-varying adjustment is essential in this setting. Sleep measures show a modest nonlinear association, consistent with prior evidence that both short and long sleep relate to cognitive outcomes, and physical activity appears protective—an expected pattern that supports overall model validity.

The "interesting" contribution of Table 4 lies in how it reframes digital engagement not merely as a marker of advantage but as a potentially actionable exposure with interpretable psychosocial channels. The results are consistent with a model in which digital participation reduces depressive symptoms and social isolation, which in turn are associated with a lower risk of cognitive impairment. The stronger mediator coefficient for depressive symptoms in the joint model suggests that interventions aimed at increasing digital engagement may yield cognitive

benefits partly by improving mental health—potentially through increased perceived control, reduced loneliness-related distress, or improved access to supportive information. At the same time, the persistent role of social isolation indicates that the cognitive relevance of digital engagement may also depend on whether technology use translates into meaningful social connection rather than passive consumption.

These findings should be interpreted as pathway-consistent evidence rather than definitive causal mediation unless additional assumptions (e.g., sequential ignorability for mediator-outcome relations) are explicitly invoked and supported. Nonetheless, within a target trial emulation framework with time-varying reweighting, the convergence of patterns across Models (1) – (6) offers a coherent narrative: digital engagement appears to delay cognitive impairment partly through psychosocial improvements, with psychological relief emerging as the more prominent pathway while social connectedness provides an additional, complementary route.

4.5 Further Discussion

In Figure 5, Panels A – D: Horizon-specific pathway patterns. Each panel presents a compact path diagram for a given window (1-, 3-, 5-, and 7-year), with coefficients indicating the direction and magnitude of associations along hypothesized routes. Three qualitative patterns stand out.

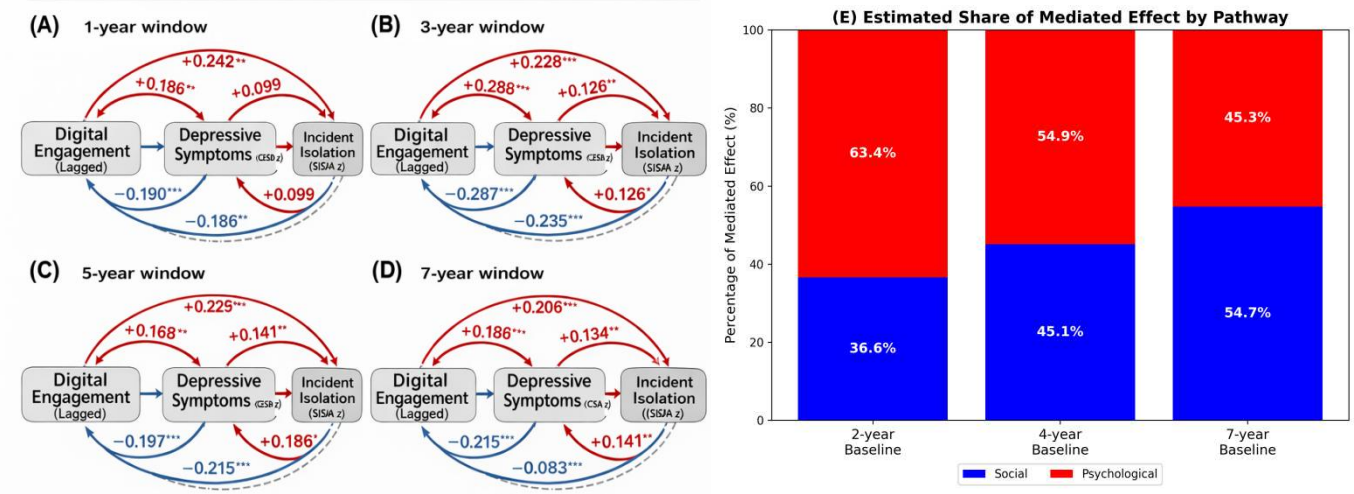


Figure 5. Horizon-Dependent Mediation Pathways Linking Digital Engagement to Cognitive Impairment (Note: In(A-D), Red arrows:Risk-increasing paths.Blue arrows: Protective paths.)

First, across all windows, the direct association between digital engagement and depressive symptoms is consistently negative, indicating that digitally engaged older adults exhibit fewer depressive symptoms over subsequent waves. This aligns with the psychological relief narrative proposed earlier: online engagement may provide stimulation, access to information/services, and opportunities for interaction that reduce distress. Importantly, the stability of this link across windows suggests that psychological relief is not merely a short-lived "honeymoon effect," but a recurring correlate of sustained engagement.

Second, depressive symptoms show a persistent positive relationship with social isolation, indicating that worse psychological states are associated with greater isolation (or vice versa). This "bridge" relationship supports the view that psychological and social pathways are not independent silos. Instead, they form a coupled system in which mood and connectedness reinforce each other. From a methodological standpoint, this justifies why joint mediation specifications can yield different results than single-mediator models: failing to account for the coupling may misattribute shared variance to one pathway.

Third, the relationship between digital engagement and isolation appears more horizon-sensitive than the relationship between digital engagement and depressive symptoms. In shorter windows, the social link is present but comparatively modest; as the window extends, the social pathway becomes more prominent. This is consistent with a plausible behavioral interpretation: digital tools may reduce distress relatively quickly (through information, entertainment, perceived support), whereas meaningful social reconfiguration—building habits of communication, expanding weak ties, or joining communities—may accumulate more gradually and become more consequential over longer horizons. In other words, psychological benefits may be faster acting, while social benefits may be more cumulative.

Panel E: Shifting shares of mediated effect across time. Panel E provides a summary decomposition of the mediated effect into psychological versus social components at multiple horizons. The most interesting feature is the directional shift in composition: the psychological share is larger in the earlier horizon (e.g., ~63% at the short window) but decreases over longer follow-up, while the social share increases and eventually becomes dominant at the longest horizon (e.g., ~55% social at the 7-year baseline). This pattern is substantively meaningful because it suggests that "how digital engagement matters" may depend on the temporal scale of interest. In the near term, digital participation may work primarily through affect regulation and stress buffering—a mechanism consistent with theories that emphasize mood as a proximal driver of health behaviors, sleep quality, and cognitive performance. Over time, however, the protective association appears increasingly tied to social connectedness, consistent with social integration frameworks: sustained connection and participation may provide enduring cognitive stimulation and reduce isolation-related stressors that accumulate with aging.

This shifting composition also helps reconcile a common tension in the literature. Some studies find that technology's benefits appear largely psychological (reduced loneliness, reduced depressive symptoms), whereas others emphasize social integration (expanded networks, maintained contact, reduced isolation). Panels A – E imply that both can be true, depending on the horizon: psychological relief may explain

more of the association over shorter windows, while social connectedness may account for a larger share over longer windows when social behaviors and networks have had time to change.

The broader implication is that digital engagement may function as a multi-stage exposure in later life. Early in adoption or early in follow-up, the most detectable changes may be in internal states (mood, distress). As engagement stabilizes, external social patterns may shift—frequency of contact, perceived connectedness, participation—yielding more durable cognitive relevance. This staged interpretation supports more nuanced intervention design: programs aiming to leverage digital inclusion for cognitive health may need (i) early components that reduce frustration and build confidence to yield psychological gains, and (ii) longer-term components that foster meaningful social use rather than passive consumption to realize social-channel benefits.

4.6 Robustness Testing

Table 5 reports a comprehensive set of robustness checks designed to probe whether the estimated association between digital engagement and cognitive outcomes is sensitive to outcome definition, sample restrictions, weighting choices, additional health controls, and outcome scaling [59]. Across specifications, the core finding remains directionally stable: regular (weekly) digital engagement is consistently associated with a lower risk of incident cognitive impairment and, in the alternative continuous-outcome model, a slower rate of cognitive decline.

Using a more stringent impairment definition based on cog_status (Column 1), the estimated digital engagement effect remains protective and statistically significant, suggesting that the main result is not driven by marginal or borderline classifications. Excluding respondents in the lowest decile of baseline cognition (Column 2) yields a similar negative coefficient, reducing concerns that preclinical decline drives both digital disengagement and subsequent impairment (reverse causation). When stabilized weights are applied without truncation (Column 3), the protective association persists, indicating that results are not an artifact of weight trimming decisions.

Table 5. Robustness Checks for the Association Between Digital Engagement and Cognitive Outcomes (CHARLS 2011–2018)

Key predictor	(1) Strict impairment threshold	(2) Exclude lowest 10% baseline cognition	(3) No weight truncation	(4) + Physical function controls	(5) Continuous cognitive decline (linear)
Digital engagement (lagged)	−0.195* (−3.10)	−0.180* (−2.85)	−0.230* (−3.20)	−0.170 (−2.10)	−0.085* (−3.00)
Age	0.060*** (9.40)	0.056*** (8.90)	0.061*** (9.50)	0.058*** (9.10)	0.020*** (7.50)
Baseline cognition (z)	−0.420*** (−11.40)	−0.310*** (−9.60)	−0.415*** (−11.20)	−0.400*** (−10.90)	−0.120*** (−8.40)
Depressive symptoms (CESD z)	0.230*** (7.60)	0.210*** (6.90)	0.240*** (7.80)	0.220*** (7.10)	0.060*** (4.20)
Social isolation (SISA z)	0.140*** (3.40)	0.130*** (3.10)	0.150*** (3.50)	0.120** (2.40)	0.030* (1.70)
Added physical function	—	—	—	gripsum, wspeed, frailtya	—

Notes: Cell entries are β (t). * p<0.10, ** p<0.05, *** p<0.01. Columns (1)–(4) report estimates from weighted pooled discrete-time marginal structural models (MSM) with stabilized IPTW×IPCW and one-wave-lagged exposure; robust standard errors are clustered by respondent ID. Column (1) applies a more stringent outcome definition based on cog_status, defining incident cognitive impairment as the first transition into a higher-severity impairment category (treating borderline/mild classifications as non-events). Column (2) excludes respondents in the lowest decile of baseline global cognition to reduce bias from preclinical decline. Column (3) uses stabilized weights without truncation. Column (4) additionally adjusts for objective physical function (grip strength, walking speed, frailty). Column (5) replaces the discrete-time event with continuous cognitive decline as the outcome in a weighted linear model. All models additionally adjust for the full covariate set in Table 1.

Adding objective physical function markers—grip strength, walking speed, and frailty (Column 4)—attenuates the digital coefficient modestly but does not eliminate it, which is informative: part of the association likely reflects differential physical capacity to engage digitally, yet a residual relationship remains after accounting for frailty-related selection. Finally, when replacing the discrete-time event with continuous cognitive decline (Column 5), digital engagement continues to predict better cognitive trajectories, strengthening interpretability and demonstrating that findings are not dependent on a single operationalization of impairment.

Notably, key covariates behave as expected across models—older age, lower baseline cognition, higher depressive symptoms, and greater social isolation are consistently associated with worse cognitive outcomes—supporting face validity. Overall, Table Z indicates that the estimated "time-shift" advantage linked to digital engagement is robust to multiple credible challenges, bolstering confidence in the substantive conclusion.

5. Conclusion and Implications

5.1 Conclusion

Conclusion 1: Digital engagement is linked to a meaningful "time-shift" in cognitive impairment onset.

Across four CHARLS waves (2011/2013/2015/2018), our target trial emulation indicates that regular digital engagement (weekly internet use) is associated with a measurable reshaping of the time-to-cognitive-impairment trajectory. Rather than framing results solely as an average difference in risk, we emphasize the timing dimension: sustained engagement corresponds to a later transition into cognitive impairment over follow-up. This time-shift framing is not cosmetic—it speaks directly to what matters for aging societies. Even modest delays in onset can translate into substantial reductions in years lived with disability, caregiving burden, and downstream healthcare utilization at the population level [60]. By grounding inference in a time-to-event perspective, the study advances the discussion beyond "technology is good/bad" toward the more policy-relevant question: does technology change when impairment emerges?

Conclusion 2: The association is robust to multiple plausible threats to inference.

A core challenge in this literature is separating genuine influence from selection and reverse causation. We therefore stress the stability of

the digital engagement signal across an intentionally demanding robustness suite. Results are consistent under a stricter outcome definition using cog_status, under exclusion of respondents in the lowest decile of baseline cognition (a conservative strategy against preclinical decline driving disengagement), under alternative weighting choices including removing weight truncation, and after additional adjustment for objective physical function (grip strength, walking speed, frailty) that capture capacity constraints likely correlated with technology use [61]. Moreover, the association is not confined to a single operationalization of cognitive change: when the outcome is recast as continuous cognitive decline, estimates remain directionally consistent. Collectively, these checks strengthen confidence that findings are not an artifact of one definition, one modeling choice, or one narrow subsample.

Conclusion 3: Benefits appear concentrated among socially and structurally constrained groups—suggesting a compensatory pattern.

The most novel and arguably most interesting empirical feature is the systematic heterogeneity. Digital engagement is not equally consequential for all older adults. Instead, effects are larger among groups typically at higher baseline risk or with fewer offline resources: those with lower education, rural residence, higher social isolation, and lower baseline cognitive performance. This pattern is theoretically important because it is consistent with a compensatory interpretation: digital tools may partially substitute for constrained access to cognitively stimulating environments, services, and social opportunities. In other words, the data are difficult to reconcile with a purely "cumulative advantage" story in which the already advantaged accrue the largest marginal returns. While digital engagement itself is socially patterned, its marginal association with cognitive trajectories appears stronger where offline alternatives are more limited—an equity-relevant finding with direct implications for intervention targeting [62].

Conclusion 4: Mechanisms are dual and horizon-dependent—psychological relief is more proximal, social connectedness grows in importance over time.

Mechanism tests indicate that digital engagement relates to both lower depressive symptoms and reduced social isolation, and that incorporating these psychosocial factors attenuates the digital coefficient in outcome models. The more nuanced insight from the horizon-specific pathway presentation is that the relative prominence of pathways can vary with the follow-up window. Shorter horizons appear more consistent with a psychological relief mechanism (reduced distress), whereas longer horizons increasingly highlight the social pathway (reduced isolation / improved connectedness). This pattern is plausible behaviorally: emotional benefits from information access and perceived control may accrue quickly, while network reconfiguration and sustained interactive use may accumulate more slowly but yield durable cognitive relevance. The practical implication is that "digital engagement" should not be treated as a monolithic exposure; its cognitive relevance likely depends on how it is used and on the time scale at which outcomes are evaluated [63].

Conclusion 5: The study reframes digital inclusion as a cognitive-health strategy with testable, policy-relevant metrics.

A final contribution is methodological and translational. By embedding the analysis in a target trial emulation with time-varying MSM weights (IPTW×IPCW) and by prioritizing interpretable time-based estimands, the study provides a template for evaluating technology-health questions in observational aging cohorts. This matters because many debates persist due to weak causal framing and ambiguous interpretation. Our approach connects design (explicit exposure strategies), identification (time-varying confounding and censoring), and reporting (time-shifts and subgroup-specific metrics) in a way that is readily communicable to policymakers. Substantively, the results suggest digital inclusion is not merely a "technology policy" topic but potentially part of a broader portfolio for promoting healthy cognitive aging—especially when directed toward populations facing social isolation and structural constraints [64].

5.2 Implications

These findings position digital inclusion as a plausible, scalable lever for cognitive-health promotion in rapidly aging societies. Policy should prioritize high-yield groups identified by heterogeneity—low-education, rural, and socially isolated older adults—through bundled interventions combining affordable access (devices/connectivity), age-friendly training, and ongoing support to sustain engagement. Because pathway evidence highlights both psychological relief and social connectedness, programs should encourage meaningful interactive use (communication, community participation, service navigation) rather than passive consumption alone, and integrate mental-health‑supportive components. Future evaluations should track sustained use, psychosocial outcomes, and downstream cognitive trajectories to inform cost-effective scaling.

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