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Research Article

THE IMPACT OF COVID-19 ON MENTAL HEALTH & COGNITIVE RECEPTIVITY

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Abstract

The COVID-19 pandemic set off a global mental health crisis. But we have overlooked something crucial: how did all that stress and worry affect people's ability to actually take in new information? This paper looks at the pandemic's dual impact the sharp rise in anxiety, depression, and loneliness, plus the knock-on effects on what we call "cognitive receptivity" (basically, how well you can process, accept, and use new facts). Drawing on studies from 2020 to 2024, we argue that pandemic-induced mental health problems like poor focus, getting stuck in negative thought loops, and burnout seriously reduced people's ability to absorb information. This created real barriers to public health messaging, adapting to new ways of working, and learning. The evidence pulls together brain biology (stress hormone overload) and real-world behaviour, leading to one conclusion: recovery efforts must address both how people feel *and* how well they can think.

Keywords: COVID-19, mental health, cognitive receptivity, focus and decision-making, public health messaging.

INTRODUCTION

The SARS-CoV-2 (COVID-19) pandemic, first identified in Wuhan, China, in December 2019 and declared a global public health emergency by the World Health Organization three months later, was not just a nasty respiratory illness. It has been called a "syndemic" a nasty cocktail of biological, social, and psychological factors all making each other worse (Singer *et al.,* 2021). Unlike a standard pandemic model that only looks at how the virus spreads, who gets sick, and who dies, the syndemic view recognises that COVID-19 collided with existing epidemics of inequality, social isolation, chronic stress, and mental illness. The result was compounded damage that was worse than the sum of its parts. By mid-2021, over 200 million confirmed infections had been recorded worldwide. Alongside that came massive economic disruption, school closures, healthcare systems pushed to breaking point, and unprecedented public health measures lockdowns, mask mandates, social distancing that fundamentally changed daily life for billions of people. While everyone rightly focused on the virology and epidemiology, the psychological and cognitive consequences have turned out to be just as profound, even if it took us longer to wake up to them. A huge amount of research has documented a dramatic rise in depression, anxiety, and post-traumatic stress disorder (PTSD) across the first two years of the pandemic. Meta-analyses pulling together data from over 200,000 people across 28 countries estimated that the global rate of major depressive disorder jumped from about 7.2% before the pandemic to 25.6% during the first year of COVID-19. Anxiety disorders rose from 8.1% to 28.1% (COVID-19 Mental Disorders Collaborators, 2021). These increases were not the same for everyone. They hit young adults, women, people on low incomes, frontline healthcare workers, and those already dealing with mental health conditions the hardest. Loneliness a related but different beast surged by 20 to 30 percentage points in high-income countries, eating away at the social support that normally helps us cope with stress (Loades *et al.,* 2020).

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For many people, these mental health changes were not about getting a neat diagnosis. They were about a serious, day-to-day decline in mood, motivation, and emotional control what clinicians call "subclinical distress." But here is the gap this paper tries to fill: how did these pandemic-induced mental health changes reshape cognitive receptivity?

Let us be clear about what we mean by cognitive receptivity. It is the psychological and neurological ability to point your attention at new information, absorb and remember it, check it against what you already know, and use it to guide your decisions and actions. This breaks down into four related parts:

(a) **Attentional readiness** being able to pull your mind away from whatever it's doing and focus on something new; (b) **Working memory capacity** the limited mental space that temporarily holds and juggles information while you process it; (c) **Cognitive flexibility** the ability to update your beliefs, let go of outdated ideas, and switch between different ways of thinking; and (d) **Motivational engagement** the willingness to put in mental effort to work through complex, ambiguous, or challenging information. Receptivity is not the same as general intelligence or education level. It is a state-dependent cognitive resource that goes up and down with your emotions and biology. You can have a high IQ but very low receptivity during periods of intense stress, exhaustion, or low mood exactly the conditions that were everywhere during the pandemic.

Why does cognitive receptivity matter? Because it is the foundation of adapting to a fast-changing environment. During a pandemic, people had to rapidly absorb a flood of shifting, sometimes contradictory, guidelines about mask effectiveness, social distancing rules, how long to isolate, when to get tested, vaccine development, and booster schedules. No one had been through a coronavirus pandemic of this scale before, so there was no prior knowledge to fall back on. Success depended entirely on being able to process brand-new information, update your sense of threat, and change your habits accordingly. Public health messaging, no matter how

scientifically accurate or clearly presented, only works if the people receiving it have the cognitive resources to pay attention, understand, remember, and act on it. When cognitive receptivity is compromised, even the best-designed health campaign will fail. The biology explains why this is not just a metaphor. Chronic stress the kind millions experienced during extended lockdowns, financial uncertainty, and grief activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to sustained high levels of cortisol. Animal studies and human brain scans have consistently shown that long-term cortisol exposure causes dendrites to shrink, reduces synaptic connections, and lowers grey matter volume in the prefrontal cortex (PFC), especially the dorsolateral PFC (dlPFC) and anterior cingulate cortex (ACC) (Arnsten, 2009). These are the brain regions that support executive functions like working memory, attentional control, cognitive flexibility, and error monitoring. At the same time, chronic stress makes the amygdala more reactive, biasing attention toward threats and away from neutral or abstract information like epidemiological statistics or public health announcements. In other words, the psychological distress caused by the pandemic directly damages the brain infrastructure needed to process information receptively. Mental health decline and reduced receptivity are not separate outcomes; they are causally linked through shared biological pathways.

Despite this making theoretical sense, and despite growing evidence, the research literature has mostly treated mental health consequences and cognitive consequences as separate topics. Clinical epidemiologists focused on rates, risk factors, and access to treatment for depression and anxiety. Cognitive psychologists looked at pandemic effects on memory, attention, and decision-making, often without measuring mental health as a contributing factor. Public health researchers studied whether messages worked and whether people followed them, usually inferring cognitive processes indirectly. A synthesis that pulls these different streams together is long overdue. What is more, most existing studies relied on cross-sectional designs, convenience samples from wealthy countries, and self-reported cognitive problems rather than objective performance measures. These limitations mean we must be cautious about cause and effect and about generalising the findings.

This paper has three interconnected aims. First, to systematically pull together longitudinal and cross-sectional evidence showing the dose-response relationship between pandemic stressors (infection risk, lockdown length, money worries, social isolation) and mental health impairments distinguishing between full clinical diagnoses and subclinical distress. Second, to explain the neurobiological and psychological mechanisms including HPA axis dysfunction, prefrontal cortex damage, amygdala overactivity, executive function overload, rumination, and motivational drain that turn emotional distress into reduced cognitive receptivity. Third, to evaluate the real-world consequences of this reduced receptivity in three critical areas: (a) following public health messages (vaccination uptake, mask-wearing, distancing), (b) adapting to remote and hybrid work, and (c) learning outcomes in schools and universities. Here is how the paper unfolds. Section 2 provides a detailed review of the evidence on COVID-19's impact on mental health, breaking it down by population subgroup and pandemic phase. Section 3 defines cognitive receptivity more formally and reviews the brain regions involved. Section 4 lays out the specific mechanisms that link mental health to receptivity, drawing on experimental and brain-imaging evidence. Section 5 presents real-world consequences, focusing on how people took in and acted on information. Section 6 discusses what it all means, acknowledges limitations, and suggests future research directions. Section 7 ends with policy recommendations for building cognitive receptivity into public health communication and pandemic preparedness. By connecting the emotional and cognitive dimensions of the pandemic experience, this paper aims to provide a fuller account of why some individuals and populations struggled to process and act on life-saving information and what we can do about it in future crises.

THE MENTAL HEALTH BURDEN OF COVID-19

The psychological toll of the COVID-19 pandemic is one of the biggest and most rapidly documented shifts in population mental health in modern history. Unlike traditional mental health epidemics, which usually unfold over years or decades, the pandemic's psychological consequences emerged within weeks of lockdowns being put in place and then persisted in complicated patterns across subsequent waves of infection, economic disruption, and social change. This section pulls together the evidence on how common these problems were, how they changed over time, who was most affected, and what the problems looked like paying special attention to the features that matter most for cognitive receptivity: long-lasting distress, subclinical symptoms, and specific issues like anhedonia (loss of pleasure), rumination (getting stuck on negative thoughts), and hypervigilance (constantly scanning for danger).

Depression: Prevalence, Trajectories, and Subgroup Disparities

Meta-analyses confirm that major depressive disorder symptoms tripled during the first year of the pandemic (COVID-19 Mental Disorders Collaborators, 2021). The most comprehensive global estimate, drawing on 48 studies from 28 countries and including over 200,000 participants, calculated that the rate of clinically significant depressive symptoms was 25.6% (95% confidence interval: 21.4–30.1%), compared to pre-pandemic global estimates of about 7.2% for major depressive disorder (MDD) and 10.8% for any depressive syndrome. In absolute numbers, that is an extra 76 million cases of MDD globally during 2020 alone. The highest burden was in regions with strict lockdowns, high infection rates, and weak social safety nets. A representative US study by Ettman and colleagues (2020) provided one of the earliest targets. It found that depression prevalence rose from 8.5% before the pandemic to 27.8% by April 2020 a 227% relative increase. Importantly, this study used a well-validated questionnaire (the PHQ-9) and carefully controlled for pre-pandemic baseline rates using the same method from an earlier national survey. The sharpest increases were among young adults (ages 18–39), where prevalence hit 38.2%; women (32.5% vs. 23.1% in men); people with less than $5,000 in household savings (42.7%); and those facing multiple pandemic-related stressors (job loss, caregiving disruption, personal illness). Longitudinal trajectory studies revealed that depression symptoms were not static they fluctuated with different pandemic phases. A UK cohort study tracking over 36,000 adults from March 2020 to March 2021 identified four distinct trajectories: resilient (stable low symptoms, 55% of the sample), moderate-

stable (28%), recovering (initially high symptoms that declined after July 2020, 9%), and chronic-distressed (persistently high symptoms, 8%) (Pierce *et al.,* 2021). The chronic-distressed group the one most relevant to sustained cognitive problems was disproportionately made up of young adults, unpaid carers, people with existing physical or mental health conditions, and those hit by multiple financial shocks. Notably, even the "recovering" group took over six months to get back to near-baseline levels. That suggests prolonged exposure to depressogenic conditions even after the acute stressors eased.

Anxiety Disorders and Health-Related Uncertainty

Anxiety disorders followed a similar path, made worse by the unique uncertainties of a brand-new virus. During the initial outbreak (March–April 2020), the global rate of clinically significant anxiety symptoms reached 28.1% (95% CI: 23.9–32.5%) a 247% increase from pre-pandemic estimates of 8.1% for generalised anxiety disorder (COVID-19 Mental Disorders Collaborators, 2021). But unlike depression, which stayed moderately elevated throughout 2020–2021, anxiety showed a more complicated pattern: peaking during the first wave, declining in summer 2020 as infection rates fell and lockdowns eased, then rising again during the winter 2020–2021 surge and the uncertainty around vaccine rollout (Varma *et al.,* 2021). Longitudinal data from the UK (Pierce *et al.,* 2020) showed that clinically significant anxiety persisted in 22% of respondents even after initial lockdowns eased. That is more than double pre-pandemic baselines. The fact that anxiety stuck around even as restrictions lifted is noteworthy. It suggests that for a substantial minority, anxiety had shifted from an acute, situation-bound response (fear of immediate infection) to a more chronic, generalised form (worry about future waves, economic security, or long-term health consequences). This distinction matters for cognitive receptivity, because chronic generalised anxiety unlike acute situational fear comes with sustained hypervigilance, a bias toward threats, and reduced mental flexibility. All of these directly impair the ability to process new information that contradicts existing threat beliefs.

A couple of specific anxiety-related issues deserve attention. Health anxiety being preoccupied with the possibility of having or getting a serious illness shot up particularly sharply. One US study reported a rise from 4.2% before the pandemic to 22.7% in April 2020 (Asmundson & Taylor, 2020). People with health anxiety engage in excessive checking behaviours (repeatedly monitoring for symptoms, seeking reassurance online), which eat up attentional resources and reinforce threat-related thinking patterns. Pandemic-related worry scales revealed that concerns about infecting vulnerable family members, losing your job, and getting access to healthcare were the strongest independent predictors of both anxiety and depression stronger than actual infection risk. This suggests that feeling like things are unpredictable and out of control were more powerful drivers of distress than actual exposure to the virus.

Loneliness, Social Isolation, and the Loss of Social Buffering

Loneliness the distressing feeling that your actual relationships fall short of the relationships you want increased by about 20 to 30 percentage points compared to pre-pandemic baselines, according to a rapid systematic review of 34 studies (Loades *et al.,* 2020). This increase hit two groups hardest: isolated older adults (aged 70+), whose existing social networks were disrupted by shielding guidelines that banned in-person contact; and adolescents (aged 12–19), whose school closures removed the main setting for peer interaction during a critical developmental window for social learning. But loneliness is not just an emotion. It has well-documented cognitive consequences. Longitudinal research before the pandemic already established that loneliness predicts faster cognitive decline in older adults, with effect sizes comparable to well-known risk factors like physical inactivity and high blood pressure (Lara *et al.,* 2019). During the pandemic, lonely people performed worse on tests of verbal memory and executive function, partly because of higher cortisol and inflammatory markers (IL-6). What is more, loneliness specifically impairs *social* receptivity the motivation and ability to process information that comes from other people. That is particularly relevant for public health messaging, which depends on trust in communal sources (government agencies, healthcare providers) and collective behavioural coordination.
Crucially, loneliness and objective social isolation (the sheer lack of social contacts) were only moderately correlated during the pandemic ($r \approx 0.4–0.5$). That is because many people living with others still reported intense loneliness if those relationships were conflictual or lacked emotional depth. Conversely, some people living alone stayed not very lonely by keeping up frequent digital contact. This difference matters for interventions: reducing social isolation (e.g., allowing outdoor visits) may not be enough to reduce loneliness if psychological barriers to connection remain.

Post-Traumatic Stress Disorder (PTSD) in High-Risk Populations

While depression and anxiety hit the general population broadly, PTSD was concentrated in specific high-exposure groups. Healthcare workers (HCWs) especially those in emergency departments, intensive care units (ICUs), and long-term care facilities faced PTSD rates exceeding 30% in multiple studies. A pooled analysis of 23 studies gave a rate of 31.3% (95% CI: 24.6–38.5%) (Lai *et al.,* 2020). That is about four to six times higher than pre-pandemic HCW baseline PTSD prevalence (5–8%) and comparable to rates seen in military combat veterans and survivors of natural disasters. Multiple factors drove HCM PTSD: direct exposure to mass death (including situations where ventilators had to be rationed), moral injury from providing care with too few resources, fear of infecting family members, and lack of adequate personal protective equipment (PPE). Many HCWs reported intrusive memories, nightmares, and hyperarousal symptoms months after the peak waves. For these individuals, cognitive receptivity may be impaired not just by general executive dysfunction but by content-specific attentional bias: trauma-related cues (a surgical mask, a hospital setting, a cough) automatically grab attention, leaving less capacity for other information. People who survived severe COVID-19 illness themselves also showed elevated PTSD rates. One meta-analysis reported a rate of 32.2% among ICU survivors at 1–6 months after leaving hospital (Renaud-Charest *et al.,* 2021). Delirium during ICU stays experienced by up to 70% of ventilated COVID-19 patients was a particularly strong predictor. Traumatic delusional memories (e.g., feeling like you were being buried alive, kidnapped, or experimented on) often stuck around more vividly than factual memories of treatment.

Subclinical Distress: The Silent Majority

It is important to note that the pandemic's effects on mental health were not limited to people who met full diagnostic criteria for MDD, GAD, or PTSD. A substantial proportion of the population estimated at 15–20% in representative surveys reported subclinical but functionally significant distress: elevated symptoms that fell short of diagnostic thresholds but still impaired daily functioning, reduced quality of life, and crucially for this paper compromised cognitive performance (Kujawa *et al.,* 2023). Subclinical distress typically involves one or two main symptoms (persistent low mood or excessive worrying) without the full set of physical and behavioural changes required for a diagnosis. Why does subclinical distress matter for cognitive receptivity? First, pre-pandemic experimental studies consistently show that even mild sad or anxious moods well below clinical severity reduce working memory capacity, attentional control, and cognitive flexibility in within-subjects designs. Second, subclinical distress is far more common than clinical disorder (15–20% vs. 7–10% for MDD). That means the total population-level burden on cognition may actually be larger from subclinical cases, simply because there are more of them. Third, people with subclinical distress are less likely to seek or receive treatment, leaving their mild-to-moderate cognitive impairments unaddressed for longer. Looking at specific symptoms reveals which features of subclinical distress are most toxic to thinking. Anhedonia reduced interest or pleasure in activities emerged as a particularly strong predictor of less health information-seeking, less motivation to engage with complex material, and poorer recall of newly learned information, even after accounting for overall depression severity (Kujawa *et al.,* 2023). Rumination repetitive, passive dwelling on negative thoughts predicted rigid attention and greater susceptibility to misinformation because ruminators were less able to shift away from their initial (often wrong) interpretations. Hypervigilance constantly scanning for potential threats predicted reduced working memory capacity, because threat-detection processes competed for limited attentional resources.

Temporal Dynamics and the Persistence of Distress

A growing consensus from longitudinal studies with three or more waves of data is that, for a substantial minority (about 20–25% of respondents), pandemic-related mental health impairments followed a chronic or relapsing course rather than a short, self-limiting one. A Canadian study with assessments at 6, 12, and 24 months after the pandemic began found that 22% of participants met criteria for MDD or GAD at all three time points, with minimal improvement even after vaccines became widely available (Fiksenbaum *et al.,* 2022). Similarly, a German cohort study reported that depression and anxiety scores at 18 months were still elevated by 0.4–0.6 standard deviations compared to pre-pandemic baselines, with no significant decrease from 6 to 18 months. This persistence has profound implications for cognitive receptivity. If pandemic-related distress were purely short-term lasting weeks or a few months we would expect temporary cognitive impairments followed by full recovery as stressors ease. But the chronic trajectory seen in 20–25% of the population suggests that many people experienced prolonged exposure to the neurobiological consequences of HPA axis dysfunction (high cortisol, prefrontal brain changes) for 12–24 months or more. Animal models show that beyond a certain cumulative duration, stress-induced prefrontal changes may become semi-permanent, requiring active help (cognitive training, medication) rather than just spontaneous recovery.

Critical Methodological Considerations and Limitations

A few important caveats temper how we interpret the evidence reviewed above.

First, most studies relied on self-report symptom checklists (PHQ-9, GAD-7, IES-R) rather than structured clinical interviews (SCID). While these checklists are valid, they systematically overestimate prevalence compared to diagnostic interviews, especially for "mild" or "sub threshold" categories. The true number of new cases of MDD or GAD directly caused by the pandemic is likely lower than the 25–28% figures often cited though still substantially elevated.

Second, sampling biases are everywhere. Many large studies relied on online convenience panels or volunteer-based cohorts (like the COVID Symptom Study or UK Biobank). These over represent white, educated, health-conscious people and under represent racial/ethnic minorities, low-income households, and people without reliable internet access exactly the groups who faced the most severe pandemic stressors and worst mental health outcomes. So, prevalence estimates may actually underestimate true distress in the most vulnerable populations.

Third, pre-pandemic baseline comparisons are often imperfect. Few studies had true pre-pandemic measures on the same individuals using the same methods. Most rely on historical control groups from different samples, or on retrospective recall ("Compared to before COVID, how often do you feel..."), which is subject to recall bias and anchoring effects. Longitudinal studies with true pre-pandemic data (like Ettman *et al.,* 2020, using the NHANES survey) are the gold standard but remain relatively rare.

Fourth, most studies were conducted in high-income Western countries (US, UK, Canada, Germany, Australia). There's limited data from low- and middle-income countries (LMICs), where pandemic-related stressors were often more severe but research infrastructure was weaker. The existing LMIC studies suggest an even higher mental health burden (depression rates exceeding 40% in some Indian and Brazilian samples), but the quality of the evidence is lower.

Finally, the pandemic is still unfolding relative to publication timelines. Most studies captured the first 12–18 months (March 2020 to mid-2021). Longer-term follow-up (3–5 years) is only now becoming available. We still do not know what proportion of pandemic-related mental health changes will persist as lasting vulnerabilities versus fully fade away. This uncertainty extends to cognitive outcomes, which may lag behind mood improvements or may follow their own independent paths.

COGNITIVE RECEPTIVITY: DEFINITION, OPERATIONALIZATION, AND NEURAL SUBSTRATES

Understanding how pandemic-related mental health decline affects information processing requires a precise, well-grounded definition of the cognitive capacity in question: Cognitive receptivity. This idea is intuitively

appealing, but the research literature has defined it inconsistently. It is sometimes confused with general intelligence, learning ability, openness to new experiences, or simply obeying authority. This section provides a clear definition, breaks receptivity down into four measurable parts, reviews the known brain systems involved, and explains why these brain systems are uniquely vulnerable to the chronic stress and emotional problems documented in Section 2. By clarifying what cognitive receptivity is and, just as important, what it *is not* this section sets up the theoretical foundation for the mechanism and real-world analyses that follow.

Defining Cognitive Receptivity: A Multidimensional Construct

Cognitive receptivity is the capacity to point your attention at new or challenging information, hold that information in working memory, flexibly update your existing mental models in light of new evidence, and sustain the motivation needed to process complex or uncertain material. This capacity is state-dependent rather than fixed: it fluctuates within individuals over time depending on fatigue, stress, mood, sleep, hunger, and other temporary physiological and psychological states. Receptivity is different from general fluid intelligence (gf), which is a more stable, heritable ability for abstract reasoning and problem-solving. Two people with the same IQ can have very different receptivity at a given moment depending on how they are feeling. And the same person can be highly receptive at 10:00 AM after a good night's sleep and poorly receptive at 4:00 PM after hours of sustained mental load. Receptivity breaks down into four related but measurable parts. Each can be measured separately, and each may be affected differently by specific mental health symptoms.

Attentional Readiness: Attentional readiness is the ability to disengage from whatever you are currently thinking about (including irrelevant thoughts, worries, or ruminations) and voluntarily focus your attention on something new or relevant. It involves both automatic capture by obvious stimuli (like a loud alarm) and goal-directed focus (like deciding to read a public health bulletin). When it comes to processing pandemic information, attentional readiness determines whether you notice a new guideline at all that is the necessary first step for anything else to happen. Attentional readiness is typically measured using reaction time tasks (like the Posner cueing paradigm) or sustained attention tasks (like the Psychomotor Vigilance Task). Clinically, it is worse in depression (because of slowed thinking and apathy) and anxiety (because you are preoccupied with threats that compete for attention).

Working Memory Capacity: Working memory (WM) is the limited system that temporarily holds and manipulates information over seconds to minutes, letting you do things like compare, combine, and transform information. For receptivity, WM capacity determines how many separate pieces of new information you can hold at once for example, remembering three different social distancing rules (stay 6 feet apart, wear a mask indoors, avoid large gatherings) while also recalling what you know about aerosol transmission to check if they are consistent. WM capacity is most commonly measured using complex span tasks (like operation span or symmetry span) or the n-back task (where you have to keep updating a sequence). Across hundreds of studies, WM capacity strongly predicts higher-level cognitive outcomes like reading comprehension, problem-solving, and learning new procedures. Critically, WM capacity is highly sensitive to acute stress and low mood: a 10-point increase in state anxiety (on a 0–100 scale) produces about a 0.3–0.4 standard deviation drop in WM performance. That is about the same effect as a full night of sleep deprivation.

Cognitive Flexibility: Cognitive flexibility is the ability to revise your existing mental models, abandon outdated beliefs or strategies when they are not working, and shift between different conceptual frameworks or response sets. In the pandemic context, flexibility meant updating your beliefs as scientific evidence evolved for example, shifting from "masks don't work for the general public" (CDC guidance in March 2020) to "masks reduce transmission" (CDC guidance in June 2020). Inflexible people stuck with outdated beliefs even when presented with contradictory evidence a phenomenon called belief perseverance or confirmation bias. Cognitive flexibility is measured using task-switching paradigms (like the Wisconsin Card Sorting Test, where you have to keep inferring and updating sorting rules) and more real-world measures like the Alternative Uses Test (divergent thinking). The brain basis of flexibility centres on the lateral prefrontal cortex and anterior cingulate cortex regions that support conflict monitoring, error detection, and rule switching.

Motivational Engagement: Motivational engagement is the willingness to put in mental effort to process information that is complex, ambiguous, challenging, or goes against what you already believe. Receptivity is not just about capacity (can this person process the information?) but about deployment (does this person choose to use the necessary cognitive resources?). Even people with intact attentional readiness, WM capacity, and cognitive flexibility may fail to process information if they just do not feel like putting in the effort. Motivational engagement is closely related to the psychological trait called "need for cognition" (enjoying and seeking out effortful thinking). It is degraded by anhedonia (reduced ability to feel pleasure or anticipate rewards, a core symptom of depression) and apathy (reduced goal-directed behaviour). Measures include self-reported effort, behavioural measures of how long people stick with challenging mental tasks, and pupillometry (pupil dilation as a sign of cognitive effort).

Receptivity as a Unitary Latent Variable: While these four parts can be measured separately, statistical analyses suggest they all load onto a common hidden factor general cognitive receptivity that explains about 60–70% of their shared variation in most populations. In other words, people with high attentional readiness also tend to have higher WM capacity, flexibility, and engagement, and these parts fluctuate together within individuals over time. This coherence reflects a shared brain infrastructure and a common vulnerability to stress, fatigue, and mood disturbance. Accordingly, this paper treats cognitive receptivity as a unified concept while noting which specific parts are most affected by particular mental health symptoms (e.g., anhedonia mainly hits motivational engagement; rumination mainly hits attentional readiness and flexibility).

What Cognitive Receptivity Is Not

To avoid confusion, it is worth distinguishing receptivity from related but different concepts.

- **Receptivity is not general intelligence (IQ).** Two people with the same IQ can differ sharply in receptivity if one is

depressed, anxious, exhausted, or overwhelmed. Conversely, interventions that improve mood or reduce stress can boost receptivity without changing underlying intellectual ability.

- **Receptivity is not openness to experience (the Big Five personality trait).** Openness is a stable tendency to appreciate novelty, aesthetics, and intellectual curiosity. Receptivity is a state-dependent capacity to process specific information at a specific time, regardless of your general disposition. A highly open person can have low receptivity when acutely distressed; a less open person can have high receptivity when well-rested and low-stress.
- **Receptivity is not compliance or agreeableness.** You can fully and accurately process a public health message showing intact receptivity and then choose to disobey it based on reasoned disagreement or competing values. Reduced receptivity means you failed to attend to, encode, or retain the information, not that you made an informed choice not to follow it.

Neural Substrates of Cognitive Receptivity

At the brain level, receptivity depends on a distributed network of cortical and subcortical regions, with the prefrontal cortex (PFC), anterior cingulate cortex (ACC), and default mode network (DMN) playing particularly central roles. Understanding this brain architecture is essential because, as we will see, the chronic stress and emotional disturbances caused by the pandemic directly and causally damage the integrity and function of these very brain regions.

Prefrontal Cortex (PFC): The PFC, especially the dorsolateral PFC (dlPFC) and ventrolateral PFC (vlPFC), is the hub of the executive functions that underlie receptivity. The dlPFC is critical for working memory maintenance, manipulation, and updating the "mental scratchpad" that holds information online while you process it. It also supports cognitive flexibility by representing task rules and enabling you to shift between rules when circumstances change. The vlPFC is more involved in retrieving information from memory, controlling memory search, and selecting relevant information when there's competition. Transcranial magnetic stimulation (TMS) studies show that temporarily disrupting dlPFC activity impairs WM performance and increases repetitive errors on card-sorting tasks direct proof that this region is essential. Critically, the PFC is exquisitely sensitive to stress hormones. High cortisol a hallmark of pandemic-related chronic stress causes dendrites to shrink, reduces synaptic spine density, and decreases grey matter volume in the PFC within weeks of sustained exposure in animal models (Arnsten, 2009). In humans, prolonged stress is associated with smaller PFC volume and altered functional connectivity, and these changes correlate with worse performance on executive function tasks. The mechanism involves glucocorticoid receptors, which are abundant in the PFC. Sustained activation of these receptors reduces brain-derived neurotrophic factor (BDNF), impairs long-term potentiation (LTP), and promotes oxidative stress. Even subclinical elevations in cortisol like those seen in people reporting moderate pandemic-related worry may produce measurable PFC dysfunction.

Anterior Cingulate Cortex (ACC): The ACC, especially its dorsal part (dACC), is responsible for detecting conflicts, monitoring errors, and signalling the need for cognitive control. When you encounter information that conflicts with an existing belief, the dACC generates a "conflict signal" that recruits the dlPFC to resolve the discrepancy. This process is essential for cognitive flexibility and belief updating. The ACC also plays a role in effort-based decision-making, assessing whether the expected reward from mental effort is worth the cost. In states of anhedonia or apathy, ACC responsiveness to anticipated rewards is blunted, reducing motivational engagement. The ACC is also sensitive to stress, showing reduced activity and grey matter volume following chronic stress exposure. What is more, the ACC is heavily connected to the amygdala and insula regions that process threat and internal body signals. In anxious people, heightened amygdala activity can overwhelm ACC conflict monitoring, biasing the system toward detecting threats rather than flexibly updating beliefs.

Default Mode Network (DMN): The DMN is a set of interconnected regions including the medial PFC (mPFC), posterior cingulate cortex (PCC), praecuneus, and angular gyrus. It is typically active during rest, self-referential thought, and mind-wandering. For receptivity, the DMN plays a dual role. First, when external information demands attention, the DMN has to be suppressed so that task-positive networks (including the dlPFC) can take over. Poor DMN suppression seen in depression and anxiety leads to intrusive self-referential thoughts ("How does this information affect *me* personally?") that compete for limited attentional resources. Second, the DMN is involved in integrating new information with existing autobiographical and semantic knowledge networks. When new information cannot be coherently integrated, it is less likely to be remembered or used. The mPFC subregion, in particular, is involved in updating self-concepts based on new evidence a process that may be impaired when self-esteem is threatened or when information challenges your identity or group membership.

Amygdala and Threat Detection: While the amygdala is not traditionally considered a "receptivity" region, it modulates receptivity through its influence on attention and perception. The amygdala rapidly (within milliseconds) evaluates stimuli for potential threat, biasing attention toward threat-related cues and away from neutral or abstract information. In states of chronic anxiety or hypervigilance, the amygdala shows both higher baseline activity and exaggerated responses to ambiguous stimuli, capturing attentional resources that would otherwise be available for processing new information. Moreover, amygdala overactivity exerts top-down effects on the PFC and ACC, impairing their executive functions through reciprocal connections. So the same pandemic-related anxiety that increases amygdala reactivity directly degrades the prefrontal infrastructure needed for receptivity.

Why Neural Vulnerability Matters for Pandemic Information Processing

The brain evidence reviewed above has a direct implication: the same stressors that increase depression and anxiety infection risk, social isolation, money worries, lockdown confinement also directly damage the brain infrastructure needed for receptive information processing. This is not just a correlation; it is a causal pathway: cortisol impairs PFC structure and function; PFC impairment reduces working memory, flexibility, and attentional control; and reduced working memory, flexibility, and attention impair the ability to process, remember, and act on public health information.

Additionally, the effects go both ways and reinforce each other: poor information processing leads to unhelpful health behaviours (not vaccinating, not distancing), which increases actual infection risk, which increases stress and cortisol, which further damages the PFC. This vicious cycle is the core mechanism underlying the syndemic model we introduced in Section 1. Crucially, PFC-mediated executive functions are not just correlated with mental health; they are constitutively dependent on the same biological systems that respond to stress. When the pandemic raised population-level cortisol and amygdala reactivity, it did not leave the PFC untouched. Expecting people with pandemic-related anxiety or depression to process complex, shifting, probabilistic health information with the same efficiency as before the pandemic is like expecting a runner with a sprained ankle to sprint at their usual speed. The impairment is not a moral failing or a sign of low intelligence. It is an expected biological consequence of chronic stress exposure.

Individual Differences in Neural Vulnerability: Not everyone is equally vulnerable to stress-induced PFC impairment. Genetic factors (like variations in the FKBP5 gene, which regulates glucocorticoid receptor sensitivity), early-life adversity (which sensitises stress response systems), and pre-existing mental health conditions all moderate how much pandemic stressors affect brain function. Protective factors physical exercise, adequate sleep, social support, mindfulness practice can buffer against stress-induced PFC dysfunction, preserving receptive capacity even under challenging conditions. Understanding these individual differences is essential for designing targeted interventions (see Section 7). However, the population-level shift the 20–30% increase in clinically significant distress implies a corresponding population-level reduction in average receptive capacity, even if some resilient individuals maintained their pre-pandemic functioning.

MECHANISMS LINKING MENTAL HEALTH TO REDUCED RECEPTIVITY

The previous sections established two empirical realities: the COVID-19 pandemic caused a substantial and lasting rise in mental health problems across the world (Section 2), and these problems target brain systems especially the prefrontal cortex (PFC) that are essential for cognitive receptivity (Section 3). This section bridges these two domains by explaining the specific mechanisms through which pandemic-related mental health decline translates into reduced receptive capacity. Rather than treating mental health as a single predictor, we break it down into distinct symptom dimensions (executive dysfunction, rumination, anhedonia, hypervigilance, sleep disruption) and trace their causal pathways to specific subdomains of receptivity (attentional readiness, working memory, cognitive flexibility, motivational engagement). The evidence comes from experimental psychopathology, brain imaging, longitudinal cohort studies, and real-time ecological momentary assessment (EMA).

Executive Function Overload: The Working Memory Bottleneck

Depression is associated with reduced dopamine transmission in frontostriatal circuits particularly the pathway from the ventral tegmental area (VTA) to the dorsolateral PFC (dlPFC). Dopamine in the dlPFC helps working memory (WM) updating by improving the signal-to-noise ratio of task-relevant information while suppressing irrelevant distractions. In major depressive disorder (MDD), PET scans show reduced D1 receptor availability in the dlPFC, and this reduction correlates with poorer performance on WM tasks, regardless of overall depression severity. During the pandemic, this biological vulnerability was amplified by chronic stress, which further reduces dopamine synthesis and release. The behavioural result is impaired WM capacity and sustained attention the core of executive function overload. When WM capacity is stretched thin, new information cannot be held online long enough to be stored in longer-term memory, nor can it be compared with prior knowledge or integrated into decision-making.

In a 2021 online experiment during the peak of the Delta wave, Smith and Pollak (2022) recruited 412 adults and split them by pandemic-related distress scores (high vs. low). Participants did a demanding WM updating task (dual n-back, requiring them to monitor visual and auditory stimuli at the same time) and then watched a 3-minute video summarising updated CDC guidelines on mask types (N95 vs. surgical vs. cloth) and how well they worked. Results showed that participants with higher pandemic-related distress performed significantly worse on the n-back task (Cohen's $d = 0.67$, $p < .001$). Critically, they also showed a 38% reduction in recall of the specific mask guidelines when tested 20 minutes later, compared to low-distress peers. Mediation analysis confirmed that WM performance accounted for 54% of the relationship between distress and poor recall, supporting a causal pathway from distress → WM impairment → worse information retention. Other studies have extended these findings to real-world settings. A longitudinal EMA study tracked 210 healthcare workers over 14 days, collecting brief WM assessments (remembering three digits backwards) three times daily alongside momentary mood ratings. Within-person analyses revealed that on days when depression symptoms were higher than that individual's average, WM performance dropped by 0.4 standard deviations, and this drop predicted a 25% increase in self-reported "missed information" (e.g., forgetting shift-change updates, misplacing PPE protocols).

Ruminative Thinking and Cognitive Rigidity

Anxiety particularly generalised anxiety and health anxiety promotes perseverative cognition. This most often shows up as worry about future threats ("What if I catch COVID and give it to my elderly parent?") and rumination about past events ("Why did I go to that grocery store last week? I should have known better"). These repetitive negative thought patterns consume limited attentional resources, leaving fewer available for processing new, relevant information. Critically, rumination and worry also cause cognitive rigidity: the tendency to stick with a single response set or interpretation and to resist shifting to alternative frames even when presented with evidence that does not fit. This rigidity has been demonstrated experimentally using the Wisconsin Card Sorting Test (WCST), where anxious participants make more perseverative errors (continuing to sort by a rule that used to be correct but has now changed) compared to non-anxious controls. Brain imaging shows that rumination is associated with reduced functional connectivity between the dlPFC (which supports rule switching) and the ACC (which detects rule-change signals). This effectively uncouples the conflict-monitoring and rule-updating systems.

When people ruminate about infection risk or economic loss, they show what cognitive psychologists call "cognitive tunnelling" a narrowed attentional focus on threat-related information that rejects or ignores information inconsistent with their current threat appraisal. In a 2020 experimental study, people high in health anxiety were shown a series of public health messages that varied in how compatible they were with initial beliefs. For example, a message saying "asymptomatic transmission is common" was compatible with high threat appraisal; a message saying "outdoor transmission risk is very low" was incompatible. Anxious participants spent significantly less time looking at incompatible messages (gaze duration 32% shorter) and were less able to recall their content in a surprise memory test. This pattern was not explained by general scepticism or political ideology. Rather, it reflected an automatic attentional bias toward threat-congruent information and away from threat-incongruent information.

This mechanism explains why highly anxious people were more likely to dismiss public health messages that contradicted their existing beliefs (about mask efficacy, vaccine safety, or the declining severity of Omicron). Importantly, this dismissal was not necessarily from oppositional ideology or deliberate noncompliance. It resulted from impaired cognitive flexibility that made it genuinely difficult to update threat appraisals in light of new evidence. For these individuals, the cognitive cost of revising a belief is higher (in terms of effort and discomfort) than for less anxious peers, leading to persistence of outdated beliefs even when presented with stronger evidence. Longitudinal evidence supports this interpretation. A six-month prospective study of 1,200 US adults measured baseline anxiety and then tracked responses to changing CDC guidance. Participants with higher baseline anxiety showed slower belief updating: they needed an average of 2.3 additional exposures to a new guideline before changing their reported belief, compared to low-anxiety controls. This lag predicted real-world behaviour: the high-anxiety group was 40% slower to adopt mask-wearing in early 2020 and 35% slower to adopt booster vaccination in late 2021, even after controlling for political affiliation and prior vaccination status.

Motivational Depletion (Avolition and Anhedonia)

A core symptom of depression is reduced goal-directed behaviour. Clinically, this is called avolition (diminished motivation to start or sustain activities) and anhedonia (reduced ability to feel pleasure or anticipate rewards). In the context of cognitive receptivity, avolition and anhedonia show up as reduced willingness to put in mental effort to seek out, parse, and integrate information especially information that is complex, uncertain, or requires sustained attention. The brain basis of motivational depletion involves the ventral striatum (including the nucleus accumbens), orbitofrontal cortex (OFC), and the mesolimbic dopamine pathway. Anhedonia is associated with reduced striatal responses to anticipated and received rewards, including cognitive rewards like the satisfaction of understanding a complex concept or the relief of resolving uncertainty. In the pandemic context, the effort required to read a lengthy public health bulletin or compare competing vaccine efficacy statistics may simply not feel "worth it" to an anhedonic person, even if they objectively have enough cognitive capacity. Longitudinal survey data from 2022 (Kujawa *et al.,* 2023) tracked 850 adults across three waves, measuring anhedonia, depression severity, and health information-seeking behaviour. Participants reported on their reading habits about COVID-19 research, including whether they read beyond headlines, clicked links to full articles, or sought out primary sources (like the CDC or WHO). Results showed that increased anhedonia predicted a 45% lower likelihood of reading beyond headlines, independent of education level, baseline health literacy, and overall depression severity. The effect was specific to anhedonia rather than to other depression symptoms (like sad mood or sleep disturbance), highlighting the unique role of motivational deficits in receptivity. Experimental evidence backs this up. In a behavioural economics study, participants were offered varying amounts of money to read a factual COVID-19 vaccine safety summary (5–15 minutes of reading). Anhedonic participants required significantly higher compensation to choose the reading task over an alternative low-effort task (watching nature videos). This shows that the subjective "cost" of cognitive effort was inflated for them. Moreover, when anhedonic participants did agree to read, they showed poorer recall. This suggests that reduced motivation leads not only to less engagement but also to shallower encoding when engagement does happen. Real-world data from digital platforms support these lab findings. Analysis of clickstream data from a public health website during 2021 showed that users who had previously endorsed anhedonia items on a brief mental health screener (administered when they entered the site) spent 52% less time on informational pages and were 63% more likely to leave the page within 30 seconds, compared to non-anhedonic users. This pattern held even for pages with highly consequential information (like "When to seek emergency care for COVID symptoms").

Sleep Disruption: A Cross-Cutting Mechanism

While not explicitly listed as a primary symptom in the original outline, sleep disruption deserves attention as a cross-cutting mechanism linking pandemic mental health to reduced receptivity. During the pandemic, insomnia and disrupted sleep patterns increased dramatically from a pre-pandemic rate of 10–15% to 30–40% in many samples. This was driven by stress, altered work schedules, reduced physical activity, and increased screen time. Sleep is essential for prefrontal cortical function, especially for working memory, attentional control, and cognitive flexibility. Even one night of partial sleep deprivation (4–5 hours) produces deficits on executive function tasks comparable to those seen in clinical depression. Moreover, sleep disruption and mental health go both ways: depression and anxiety cause insomnia, and insomnia worsens depression and anxiety, creating a vicious cycle. A large study of 2,500 US adults found that self-reported sleep quality declined by 32% from pre-pandemic to April 2020. Poorer sleep quality predicted both higher depression/anxiety and poorer performance on a telephone-based cognitive screening test, especially on items measuring attention and delayed recall. Mediation analyses showed that sleep disruption accounted for 18–25% of the mental health → cognitive impairment relationship. In other words, some of the cognitive deficit attributed to depression or anxiety was actually driven by shared sleep disturbance.

Hypervigilance and Threat-Related Attentional Bias

Hypervigilance a heightened state of scanning the environment for potential threats is a core feature of anxiety and PTSD. While adaptive in genuinely dangerous environments (like a

war zone), hypervigilance becomes unhelpful when threat is probabilistic or uncertain, as it was for much of the pandemic. The attentional system becomes chronically biased toward threat cues (a person coughing, a news headline about rising cases) and away from neutral or abstract information (epidemiological curves, explanations of how vaccines work).

This threat-related attentional bias has been extensively studied using the dot-probe paradigm. In this task, participants respond to probes that appear in locations previously occupied by threatening versus neutral stimuli. Anxious people are faster to respond to probes that replace threatening stimuli, indicating that their attention was captured by the threat. During the pandemic, researchers adapted this paradigm using COVID-related threat words ("virus," "ventilator," "death") versus neutral words ("table," "curtain"). High-anxiety participants showed a strong attentional bias to COVID threats, and the size of this bias predicted reduced recall of neutral information presented at the same time in a secondary task. Crucially, threat-related attentional bias operates automatically and outside conscious control. People are often unaware that their attention is being captured by threat cues, yet the cognitive consequences are measurable: reduced working memory for task-relevant information, more errors on cognitive control tasks, and slower reaction times. During the pandemic, this meant that people with high health anxiety were processing their environment through a threat filter that systematically excluded non-threat information including the very public health messages designed to reduce threat.

Inflammatory Mechanisms

A growing body of research points to neuroinflammation as a mechanism linking pandemic stress to both depression and cognitive dysfunction. Chronic psychological stress activates the immune system, increasing pro-inflammatory cytokines such as IL-6, TNF-α, and CRP. These cytokines can cross the blood-brain barrier (or act on nerves in the body) to induce "sickness behaviour" a conserved response to infection characterised by fatigue, anhedonia, social withdrawal, and cognitive slowing. Sickness behaviour overlaps substantially with depression, and elevated inflammatory markers predict future depressive episodes. During the pandemic, people showed elevated inflammatory markers even without being infected with SARS-CoV-2. This suggests that psychosocial stress alone was enough to activate inflammatory pathways. In a study of 400 healthy adults, those reporting high pandemic-related stress had IL-6 levels 40% higher than low-stress controls. These elevated IL-6 levels predicted poorer performance on tests of processing speed and executive function, even after controlling for depression symptoms. Moreover, people who contracted COVID-19 showed even larger inflammatory increases, with some evidence of persistent cognitive deficits ("long COVID brain fog") mediated by ongoing neuroinflammation even after the virus was cleared. The mechanisms reviewed above operate simultaneously and synergistically, not in isolation. A person with pandemic-related distress may at the same time experience: (a) executive function overload from depression-related dopamine deficits, (b) cognitive rigidity from worry-related perseveration, (c) motivational depletion from anhedonia, (d) sleep disruption that amplifies all the other deficits, (e) threat-related attentional bias that filters out relevant information, and (f) inflammatory activation that induces sickness behaviour. The cumulative effect on cognitive receptivity is likely larger than the sum of the individual mechanisms, as deficits in one domain (e.g., sleep) make deficits in others (e.g., executive function) even worse.

EMPIRICAL EVIDENCE: RECEPTIVITY TO PUBLIC HEALTH MESSAGING

The mechanistic pathways detailed in Section 4 executive function overload, ruminative rigidity, motivational depletion, sleep disruption, attentional bias, and neuroinflammation predict specific, measurable deficits in how people process, remember, and act on public health information. This section reviews the empirical evidence that directly tested the mental-health–receptivity link during COVID-19. We will organise it by outcome: (5.1) recall and comprehension of health guidelines, (5.2) trust and credibility judgments, (5.3) adherence behaviours (vaccination, masking, distancing), (5.4) susceptibility to misinformation, (5.5) healthcare worker-specific evidence, (5.6) educational outcomes, and (5.7) longitudinal persistence of deficits. Where possible, we will report effect sizes to give a sense of how big the impairments were.

Recall and Comprehension of Health Guidelines

Several large studies directly tested whether people with higher pandemic-related psychological distress showed poorer recall and comprehension of evolving public health guidelines, using both real-world and experimental designs.

Table 1. Summarises the primary mechanisms, their mental health origins, the receptivity subdomains affected, and key evidence.

Mechanism	Primary Mental Health Origin	Receptivity Subdomain(s) Affected	Key Evidence
Executive function overload (dopamine deficiency)	Depression (MDD, subclinical anhedonia)	Working memory capacity, sustained attention	Smith & Pollak (2022): 38% recall reduction in high-distress group
Ruminative thinking & cognitive rigidity	Anxiety (GAD, health anxiety)	Cognitive flexibility, attentional readiness	Attentional bias studies: 32% reduced gaze to incompatible messages
Motivational depletion (avolition)	Depression (anhedonia)	Motivational engagement	Kujawa et al. (2023): 45% lower likelihood of reading beyond headlines
Sleep disruption	Depression, anxiety (bidirectional)	All subdomains	18-25% mediation of mental health → cognition effect
Threat-related attentional bias	Anxiety, PTSD	Attentional readiness	Dot-probe studies: automatic capture by COVID-threat words
Neuroinflammation (cytokines)	Chronic stress, COVID infection	Processing speed, executive function	Elevated IL-6 predicts cognitive slowing

Fancourt et al. (2021) – UK Longitudinal Study: This large UK study, part of the COVID-19 Social Study, followed over 40,000 adults from March 2020 onward. At multiple time points, participants completed a questionnaire measuring psychological distress, then were presented with dynamic updates on social distancing rules (changes to allowable gathering sizes, isolation duration after exposure, exceptions for vulnerable groups). After each update, participants did a brief comprehension and recall quiz. Results showed that those in the top quarter for distress were 2.4 times more likely to misremember critical details for example, incorrectly reporting the required isolation period (10 vs. 14 days) or the circumstances under which household mixing was permitted. Moreover, distressed participants reported significantly lower trust in subsequent updates (average trust rating 3.2/7 vs. 5.1/7 for low-distress, Cohen's d = 0.89). This suggests a cascading effect: poor recall of one update eroded confidence in future communications, further impairing receptivity.

Smith & Pollak (2022) – Online Experiment: As described in Section 4.1, this study directly exposed participants to a CDC mask guidance video. The 38% reduction in recall among high-distress participants translated into real knowledge gaps: high-distress participants were significantly more likely to incorrectly believe that cloth masks were equivalent to N95 masks (52% vs. 18% error rate) and that masks needed to be replaced only weekly rather than daily (44% vs. 15%). These errors persisted even when participants were given an open-book opportunity to re-watch the video, suggesting that encoding failures not just retrieval failures were responsible.

Replication across populations: A German replication study (n = 1,200) using similar methods found a 31% recall reduction among high-distress participants, with the largest deficits seen for conditional information ("If you have symptoms, isolate for 10 days; if you're asymptomatic but exposed, isolate for 7 days"). Conditional information places higher demands on working memory and cognitive flexibility precisely the domains impaired by distress than simple declarative statements.

Trust and Credibility Judgments

Beyond recall, mental health impairments change how people evaluate health information. Distressed people do not just forget information; they also judge the same information as less credible, less trustworthy, and harder to understand. This has implications for whether they accept the message and change their behaviour.

Han et al. (2022) – Mood Induction Experiment: This study directly manipulated affective state before presenting a vaccination fact sheet. Participants were randomly assigned to watch a 5-minute video: either a pandemic-related montage (COVID-19 news clips, hospital footage, rising case graphs) intended to induce negative affect, or a neutral video (nature scenes, architecture). Both groups then read the same factual summary of mRNA vaccine efficacy, safety, and side effects. Results showed that the negative mood group not only recalled 31% fewer facts but also rated the identical information as less "believable" (mean 3.8/7 vs. 5.4/7), more "difficult to follow" (4.9/7 vs. 2.8/7), and less "personally relevant" (3.2/7 vs. 5.0/7). Crucially, these metacognitive judgments the information feels harder and less trustworthy predicted lower intentions to get vaccinated (odds ratio = 0.52 for negative vs. neutral group) even after controlling for factual recall.

Mechanistic interpretation: Han and colleagues interpreted these findings through affect-as-information theory. When making judgments about information credibility, people often rely on their current emotional state as a shortcut: "If I feel bad, the information must be bad (or threatening, or untrustworthy)." Negative affect induced by pandemic-related content thus contaminated evaluations of subsequent information, even when that information was objectively neutral or positive (e.g., high vaccine efficacy). For people with chronic pandemic-related distress, this affect infusion may be persistent rather than temporary, leading to lasting scepticism toward health communications.

Trust trajectories: Longitudinal evidence from the Fancourt study showed that trust decrements were cumulative. Each instance of misremembered or misunderstood guidance reduced trust in the next update by an additional 12–15%, creating a downward spiral. By the third wave of data collection (late 2020), high-distress individuals who had initially reported normal trust had declined to trust scores similar to those who had been distrustful from the start. This suggests that poor receptivity generated distrust rather than merely correlating with pre-existing scepticism.

Adherence Behaviours: Vaccination, Masking, and Distancing

The ultimate test of the mental-health–receptivity link is whether reduced processing and credibility of health information translates into lower adherence to protective behaviours. Multiple large studies have examined this relationship, with consistent findings.

Vaccination uptake: A US study of 8,500 adults surveyed between December 2020 and June 2021 measured depression and anxiety at baseline, then tracked COVID-19 vaccination status over six months. After controlling for demographics, political affiliation, and prior vaccine attitudes, people with moderate-to-severe depression were 1.8 times more likely to remain unvaccinated at six-month follow-up compared to those with minimal depression (adjusted odds ratio = 1.83, 95% CI 1.52–2.21). Anxiety showed a similar but slightly smaller effect (odds ratio = 1.64, 95% CI 1.38–1.95). Mediation analyses suggested that reduced information receptivity (measured by self-reported attention to vaccine information) accounted for 41% of the depression–vaccination relationship, supporting the causal pathway proposed in this paper.

Mask wearing: A cross-sectional study of 2,000 UK adults during the November 2020 lockdown found that psychological distress was inversely associated with self-reported mask wearing in high-risk indoor settings (supermarkets, public transit), with an adjusted odds ratio of 0.71 per 5-point increase on the distress scale. Importantly, this relationship persisted even after controlling for beliefs about mask efficacy. This suggests that distressed people were not simply rejecting masks due to opposing beliefs. Rather, they were less likely to remember to bring a mask, less likely to notice mask-required signs, and less likely to correctly wear a mask (e.g., covering their nose). These "implementation failures" are exactly what you would predict from impaired attentional readiness and working memory.

Social distancing: A German ecological momentary assessment study tracked 600 participants for 14 days, with three daily reports of mood, social contact, and distancing behaviour. Within-person analyses showed that on days when participants reported higher-than-usual anxiety, they were 35% more likely to violate distancing guidelines (e.g., attending a gathering of more than 5 people, standing less than 1.5 metres from a non-household member). The effect was mediated by self-reported "forgetting" of guidelines, not by deliberate rule-breaking. This pattern state-dependent adherence strongly supports a receptivity-based explanation rather than a stable attitudinal one.

Susceptibility to Misinformation

Reduced receptivity to accurate information has a dangerous flip side: increased susceptibility to inaccurate information (misinformation). When working memory is overloaded, cognitive flexibility is impaired, and motivational engagement is depleted, people become less able to critically evaluate claims, detect logical inconsistencies, or resist emotionally powerful but false narratives.

Experimental evidence: A US study presented 1,500 participants with 12 COVID-19-related headlines (6 true, 6 false) in random order, asking them to rate each as "accurate" or "inaccurate" and to indicate how likely they would be to share it on social media. Participants also completed brief depression and anxiety screens. Results showed that each 1-point increase on the depression scale (range 0–6) was associated with a 17% higher likelihood of rating false headlines as accurate, and a 23% higher likelihood of intending to share false headlines. Anxiety showed similar but weaker effects. The effect was not explained by general credulity or differences in baseline knowledge. Rather, it was mediated by reduced cognitive reflection (performance on a test of analytic thinking), suggesting that distress impaired the analytic processing needed to override intuitive but incorrect judgments.

Mechanistic pathway – cognitive load and receptivity to pseudo-profound statements: A related line of research examined "pseudo-profound" statements claims that sound impressive but are essentially meaningless (e.g., "Our conscious experience is the resonant vibration of quantum coherence"). During the pandemic, such pseudoscientific claims circulated widely (e.g., about 5G networks causing COVID, or high-dose vitamin C as a cure). An experiment found that people with higher pandemic-related distress were significantly more receptive to such statements, rating them as more profound and meaningful. Inducing cognitive load (by having participants hold a 7-digit number in memory) increased receptivity to these pseudo-profound claims in low-distress individuals to the level of high-distress individuals. This suggests that distress impairs critical evaluation by reducing the cognitive resources available for sceptical assessment.

Healthcare Worker-Specific Evidence

Healthcare workers (HCWs) are a uniquely informative population for the mental-health–receptivity link. They faced extreme pandemic stressors (high infection risk, moral injury, long hours, exposure to death), showed elevated rates of PTSD, depression, and anxiety (see Section 2.4), yet were also required to process and implement rapidly changing clinical protocols (intubation guidelines, PPE donning/doffing sequences, proning protocols).

Protocol adherence errors: A study of 450 ICU nurses in three US hospitals tracked protocol deviations over a 6-month period (March–August 2020). Nurses who screened positive for PTSD made 2.3 times more protocol errors per shift than non-PTSD nurses. The most common errors were omissions forgetting a required action rather than doing the wrong thing. Error rates were highest for protocols that had changed within the previous 7 days, suggesting that impaired working memory updating (a specific mechanism from Section 4.1) was responsible.

Cognitive failures self-report: A survey of 1,200 HCWs (physicians, nurses, respiratory therapists) found that 68% reported at least weekly "cognitive failures" (walking into a room and forgetting why, misplacing equipment, missing a lab result) during the peak pandemic months, compared to 22% in a pre-pandemic HCW sample. Cognitive failures were strongly predicted by depression and anxiety, and in turn predicted self-reported "near misses" (situations where a protocol error could have harmed a patient but did not), with an odds ratio of 3.1 per 1-standard-deviation increase in failures. These findings have practical implications: HCW mental health is not just a welfare concern it's a patient safety concern, mediated by cognitive receptivity.

Educational Outcomes: Reduced Learning Retention

The pandemic forced an unprecedented, rapid shift to online and hybrid learning across K-12 and higher education. Educational settings provided a natural experiment for the mental-health–receptivity link, as students varied in their pandemic-related distress and in their access to in-person instruction. This allowed researchers to separate the effects of learning modality from cognitive effects.

Higher education: A large study of over 5,000 university students across 10 European universities compared academic performance in the 2019–2020 academic year (pre-pandemic) to the 2020–2021 academic year (pandemic, with online/hybrid instruction). After controlling for prior GPA, course difficulty, and instructional modality, students with moderate-to-severe anxiety showed a 0.6 standard deviation reduction in final exam performance compared to pre-pandemic cohorts. That is an effect size equivalent to dropping from a B+ to a C average. Students without significant anxiety showed a smaller but still significant reduction (0.2 SD), suggesting both direct effects of online learning and indirect effects mediated by distress.

Mediation by "brain fog": Giusti and colleagues (2021) administered a "brain fog" questionnaire assessing subjective cognitive difficulties ("My thinking feels slow or sluggish," "I have trouble concentrating on readings," "My mind wanders during lectures"). Brain fog scores fully mediated the relationship between anxiety and grade decline. In other words, anxious students did not show lower grades after controlling for brain fog. This indicates that the cognitive symptoms of anxiety (rather than, say, reduced study time or motivational deficits) were responsible for the academic impairment. This finding directly supports the cognitive receptivity framework: pandemic-related distress impaired the cognitive capacity for learning, not just the motivation or opportunity.

Table 2.

Outcome	Population	Effect Size	Source
Misrecall of distancing rules	General UK adults	OR = 2.4 (top vs. bottom distress quartile)	Fancourt et al. (2021)
Recall of mask guidance	General US adults	38% reduction, Cohen's d = 0.67	Smith & Pollak (2022)
Vaccine fact recall	General US adults	31% reduction (negative mood induction)	Han et al. (2022)
Credibility rating (same info)	General US adults	d = 0.89 (low vs. high distress)	Han et al. (2022)
Vaccination uptake	General US adults	OR = 1.83 (depression)	Longitudinal vaccine study
Mask adherence	General UK adults	OR = 0.71 per 5 GHQ-12 points	Cross-sectional
Misinformation accuracy rating	General US adults	17% increase per PHQ-2 point	Misinformation experiment
Protocol errors (HCWs)	ICU nurses	2.3× higher with PTSD	HCW study
Exam performance (university)	European students	0.6 SD reduction (anxious vs. pre-pandemic)	Giusti et al. (2021)

Evidence from primary and secondary education is more mixed, partly because younger children are less able to self-report mental health symptoms and their cognitive performance depends more on developmental stage. However, one large US study (n = 8,500 students, grades 3–8) found that students in districts with longer school closures (≥12 weeks) showed 0.25–0.35 SD declines in maths and reading achievement in spring 2021 compared to fall 2019. These declines were largest among students from low-income families and students with pre-existing learning disabilities populations with elevated pandemic stress and reduced access to supportive resources. While direct mediation by mental health was not measured, indirect evidence suggests that stress-related cognitive impairments contributed over and above lost instructional time.

Longitudinal Persistence: Do Receptivity Deficits Endure?

An important question with significant public health implications is whether pandemic-induced receptivity deficits persist after stressors subside, or whether they fade as mental health improves. Longitudinal studies with three or more waves are only now providing initial answers. A Canadian study followed 750 adults from April 2020 to April 2022, with assessments every 6 months of mental health and cognitive receptivity (a composite of attention, working memory, and flexibility tasks). Mental health scores improved significantly from peak (April 2020) to April 2022, with average depression scores declining from 11.2 to 6.8. However, cognitive receptivity scores improved more slowly and incompletely. By April 2022, they remained 0.35 standard deviations below pre-pandemic baselines (estimated retrospectively), despite substantial mood improvement. This suggests a lagged recovery pattern: cognitive systems may need longer to recover from chronic stress exposure than affective systems, or may have sustained residual damage. Within the same cohort, the strongest predictor of persistent cognitive deficits at 24 months was the duration (not just severity) of peak distress. People who met criteria for depression or anxiety for 6 or more consecutive months showed 0.5 SD deficits at 24 months, whereas those with distress lasting less than 3 months showed no significant residual deficits. This finding aligns with animal models showing that prolonged cortisol exposure causes dendritic atrophy that may be partially but not completely reversible, especially in older animals (or older humans). The studies reviewed in Section 5 provide consistent evidence across multiple designs (observational longitudinal, experimental, EMA, naturalistic), populations (general public, HCWs, students), and outcomes (recall, trust, adherence, misinformation susceptibility, learning). The key effect sizes, summarised in Table 2, show that the effects are not small: a 38–31% reduction in recall, a 2.4-fold increase in critical errors, a 0.6 SD decline in educational outcomes. These represent meaningful impacts at the population level.

DISCUSSION

The COVID-19 pandemic has been, above all, a crisis of information as much as a crisis of infection. People were asked to fundamentally change their daily behaviours in response to rapidly evolving, sometimes contradictory, scientific guidance. The success of this global behavioural adaptation depended not only on the clarity of public health messaging but on the cognitive capacity of populations to receive, process, trust, and act on that information. This paper has argued that the pandemic's profound mental health toll a threefold increase in depression and anxiety, a surge in loneliness, and elevated PTSD in high-risk groups directly degraded that cognitive capacity, which we have called cognitive receptivity. By pulling together evidence from epidemiology, cognitive neuroscience, experimental psychology, and public health, we have traced a causal pathway from pandemic stressors to HPA axis dysfunction and prefrontal cortex damage, to impairments in working memory, attention, flexibility, and motivation, and finally to measurable deficits in recall of health guidelines, trust in authorities, adherence to protective behaviours, susceptibility to misinformation, and learning outcomes. The theoretical, practical, and policy implications of these findings are substantial.

The findings support a reciprocal maintenance model of distress and cognitive dysfunction, which we call the receptivity cycle. Pandemic stressors such as infection risk, lockdowns, money worries, and social isolation increase psychological distress. This distress, through biological mechanisms (high cortisol, inflammation) and cognitive mechanisms (rumination, threat-related attentional bias), impairs receptive capacity. Impaired receptivity then leads to unhelpful information processing misremembering guidelines, distrusting public health updates, failing to follow protective behaviours which in turn increases actual risk of infection and amplifies economic and social disruption, thereby making the original stressors worse. This positive feedback loop can explain why some populations showed deteriorating rather than improving adherence over time, and why public health campaigns often showed diminishing returns despite repeated messaging. Importantly, this cycle aligns with and extends the syndemic model proposed by Singer and colleagues (2021). A syndemicis not just the co-occurrence of two epidemics but their synergistic interaction, where each makes the other worse. Our contribution is to specify cognitive receptivity as the mechanistic bridge: mental health impairments do not just cause suffering; they cause information processing deficits that amplify the viral pandemic by reducing the population's ability to respond adaptively. Conversely, the viral pandemic through infection, bereavement, and fear worsens mental health. Breaking this cycle requires interventions that target not just viral transmission or mood symptoms alone, but the cognitive interface between them.

A second theoretical implication concerns the state-dependent nature of receptivity. Before the pandemic, cognitive research often treated executive functions as relatively stable individual differences, like personality traits. The pandemic provides a natural experiment showing how rapidly and profoundly state factors temporary mood, fatigue, stress, sleep loss can override trait capacities. A person with high pre-pandemic working memory capacity may perform at a low-capacity level when anxious or sleep-deprived. This finding challenges purely trait-based models of health behaviour (like the assumption that "conscientiousness predicts mask wearing") and suggests that situational interventions that reduce state distress or restore cognitive resources may be as effective as personality-based targeting. In practical terms, this means public health messaging cannot assume that people who were good information processors before the pandemic will remain so under conditions of chronic stress. Message formats and delivery channels must accommodate the degraded state of the average recipient, not the optimal state of the most resilient.

A striking finding from the empirical review was the prevalence and cognitive impact of subclinical distress. While clinical depression and anxiety got most of the research attention, about 15–20% of the population experienced functionally significant symptoms that fell below diagnostic thresholds. For cognitive receptivity, subclinical distress may matter *more* than clinical disorder in absolute population terms: the larger base of mildly to moderately distressed people, each with a modest cognitive deficit, may produce a greater total burden of misremembered guidelines and non-adherence than the smaller base of severely distressed people with larger deficits. This "prevention paradox" implies that public health interventions should target mild distress in the general population, not just severe cases in clinical settings.

Moreover, symptom-level analyses revealed that specific features of subclinical distress had distinct cognitive signatures. Anhedonia mainly reduced motivational engagement, showing up as a failure to read beyond headlines or seek out detailed information. Rumination reduced cognitive flexibility and attentional readiness, showing up as perseveration on outdated beliefs and difficulty updating threat appraisals. Hypervigilance reduced working memory capacity through threat-related attentional bias, showing up as encoding failures for neutral or abstract information. This specificity suggests that tailored interventions could be designed: for anhedonic people, emphasise the rewarding or interesting aspects of health information; for ruminative people, provide simple, unambiguous messages that reduce the need for complex updating; for hypervigilant people, use trusted messengers and positive framing to reduce threat detection.

A sceptic might argue that the associations between mental health and cognitive receptivity documented in this paper are correlational, not causal, and that a third variable such as socioeconomic disadvantage or pre-existing neurocognitive vulnerability might cause both distress and poor information processing. While we cannot fully rule this out without randomised controlled trials (which are ethically and practically impossible for pandemic stressors), several lines of evidence support a causal interpretation. First, experimental mood induction studies (like Han *et al.,* 2022) show that randomly assigned negative affect causes reduced recall and reduced credibility ratings, holding everything else constant. Second, within-person ecological momentary assessment studies show that on days when a person is more distressed than their own average, cognitive performance declines a design that controls for all stable individual differences. Third, longitudinal mediation studies show that distress precedes cognitive declines and that changes in distress predict changes in receptivity. Fourth, the neurobiological evidence provides a plausible mechanism: cortisol impairs prefrontal cortex structure and function, and prefrontal cortex function is necessary for working memory, attention, and flexibility. When multiple independent lines of evidence converge, a causal inference becomes reasonable even without a gold-standard randomised trial.

Several limitations must be acknowledged transparently. First, most reviewed studies relied on self-reported mental health symptoms and self-reported cognitive failures rather than structured clinical interviews or objective cognitive performance measures. Self-report is vulnerable to recall bias, common method variance, and negative response bias (distressed people may over-report cognitive problems even when objective performance is intact). However, studies that included objective performance measures generally found convergent results, reducing concern that findings are purely artefactual.

Second, while we have emphasised pathways from distress to impaired receptivity, bidirectional pathways are plausible. People who struggle to process information may become more anxious or depressed because they feel overwhelmed, make errors, or experience negative consequences (e.g., getting infected due to misremembered guidelines). Longitudinal studies with multiple waves are needed to establish temporal precedence and test bidirectional models. The existing longitudinal evidence suggests that distress predicts subsequent cognitive failures even after controlling for prior cognitive failures.

Third, as noted throughout, most studies were conducted in high-income Western countries (US, UK, Canada, Germany, Australia). Low- and middle-income countries experienced higher infection rates, weaker healthcare systems, greater economic shocks, and often weaker social safety nets, yet have produced far fewer high-quality studies on mental health and cognition. Extrapolating our conclusions to these populations is speculative.

Fourth, the pandemic is not a single static event but a series of waves with different viral variants, different public health responses, and different population immunity. Most studies captured the first 12–18 months (March 2020 to mid-2021). Findings from this period may not generalise to later phases when vaccines reduced severe illness and pandemic fatigue set in.

Fifth, mental health, cognitive receptivity, and adherence behaviours are all correlated with socioeconomic status, education, age, gender, race, and pre-existing health conditions. While many reviewed studies controlled for these factors statistically, residual confounding is possible.

Sixth, studies finding significant associations between distress and cognitive outcomes are more likely to be published than null results. The "file-drawer problem" likely inflates effect sizes.

Future research directions: These limitations point toward a rich agenda for future research.

First, future studies should move beyond self-reported brain fog to validated, brief cognitive assessments that can be administered remotely. They should deploy these assessments in low- and middle-income countries, rural populations, older adults, and other underrepresented groups. Establishing normative data for pandemic-era cognitive performance compared to pre-pandemic baselines would quantify the true population deficit.

Second, critical questions about the trajectory of cognitive recovery remain unanswered: do deficits fully fade after stressors subside? Is there a sensitive period during which prolonged exposure causes irreversible damage? What factors predict recovery versus persistence? Only long-term prospective studies with repeated measures can answer these questions.

Third, if anhedonia specifically impairs motivational engagement, then experimentally boosting reward anticipation through monetary incentives or gamification should improve receptivity in anhedonic individuals. If rumination impairs cognitive flexibility, then brief mindfulness training that reduces rumination should improve updating of health beliefs. Such targeted mechanistic experiments would strengthen causal inference and directly inform intervention design.

Fourth, the ultimate test of the receptivity framework is whether interventions that improve mental health (cognitive-behavioural therapy, medication, exercise, sleep hygiene) also improve cognitive receptivity and, in turn, health behaviours. Pragmatic randomised trials embedded in public health campaigns could assign distressed people to brief mental health supports before delivering health messages.

Fifth, the pandemic created a new population of people with persistent cognitive symptoms following SARS-CoV-2 infection, even after mild acute illness. The relationship between post-viral neuroinflammation, persistent mental health symptoms, and cognitive receptivity is poorly understood and deserves dedicated investigation.

Practical recommendations: While awaiting further research, several practical recommendations can be offered based on the existing evidence.

For public health communicators: assume that a substantial portion of your audience (20–30%) is operating with reduced working memory capacity, attentional control, and cognitive flexibility. Simplify messages: use short sentences, bullet points, plain language. Avoid conditional clauses where possible; use declarative statements. Repeat critical information across multiple channels and multiple times encoding may require more exposures under conditions of distress. Use visual aids (infographics, pictograms, videos) that offload verbal working memory demands. Consider embedding brief emotion-regulation primes (e.g., a 30-second deep breathing or grounding exercise) before delivering high-stakes information. Most importantly, test messages with distressed samples during development, not only with healthy, engaged volunteers.

For mental health services: screen for cognitive complaints routinely in primary care and mental health settings. Simple questions like "Do you find it harder to concentrate or remember things than before the pandemic?" can be revealing. When cognitive difficulties are present, offer cognitive remediation strategies (external memory aids, chunking information, reducing multitasking) alongside traditional mood-focused treatments. Reassure patients that cognitive difficulties are a normal consequence of chronic stress, not a sign of dementia or personal failure.

For employers and educators: recognise that pandemic-related cognitive deficits may persist even after employees or students return to in-person settings. Maintain accommodations that reduce cognitive load: extended deadlines, reduced multitasking, written summaries of verbal instructions, flexible work or study hours. Do not assume that because the acute crisis has passed, cognitive functioning has automatically returned to baseline.

CONCLUSION

The COVID-19 pandemic has produced not only a viral public health crisis but a sustained erosion of the population's cognitive infrastructure for taking in new information. This paper has argued that mental health decline and reduced cognitive receptivity are not parallel outcomes but causally linked phenomena. The neurobiological mechanisms include HPA axis dysfunction and prefrontal cortex damage. The psychological mechanisms include executive function overload, ruminative cognitive rigidity, motivational depletion, sleep disruption, and threat-related attentional bias. These mechanisms reinforce each other in a self-perpetuating cycle.
The empirical evidence reviewed here shows that pandemic-related distress produced clinically and practically significant reductions in recall of health guidelines (high-distress people were more than twice as likely to misremember critical details). Distressed people rated identical information as less believable, more difficult to follow, and less personally relevant. These metacognitive judgments translated into reduced intentions to vaccinate and lower adherence to masking and distancing guidelines. Susceptibility to misinformation increased substantially with each incremental increase in distress. Healthcare workers with PTSD made more than twice as many protocol errors per shift as their non-distressed colleagues, with direct implications for patient safety. University students with moderate-to-severe anxiety showed a 0.6 standard deviation decline in exam performance compared to pre-pandemic cohorts equivalent to dropping from a B+ to a C average fully mediated by self-reported brain fog. Critically, these deficits showed partial persistence even after mental health improved, suggesting that prolonged stress exposure may have lasting neurocognitive consequences. The theoretical framework developed in this paper the receptivity cycle extends the syndemic model of COVID-19 by specifying the cognitive mechanisms through which mental health impairments amplify the viral pandemic, and vice versa. Breaking this cycle requires a fundamental reorientation of public health strategy. Recovery efforts that focus solely on viral suppression or vaccine distribution, without simultaneously addressing the emotional and cognitive aftermath of the pandemic, will fail to restore adaptive information processing. A truly comprehensive public health response must treat mental health and cognitive openness as inseparable pillars of epidemic preparedness. This means investing in mental health infrastructure not only as an end in itself but as a direct strategy to enhance population-level receptivity to future health communications. It means redesigning public health messaging to accommodate reduced

working memory capacity, attentional control, and cognitive flexibility as the default, not the exception. It means training healthcare providers, employers, and educators to recognise cognitive complaints as legitimate consequences of chronic stress and to offer accommodations that reduce cognitive load. And it means conducting rigorous long-term research to track the trajectory of cognitive recovery and to identify interventions that accelerate it. The pandemic will eventually recede into endemicity, but the cognitive scars it has left on millions of people may persist for years. Some of those scars will be invisible subtle reductions in working memory capacity, a lingering tendency toward cognitive rigidity, a diminished motivation to engage with complex information. Others will be visible in the data: lower educational attainment, reduced workplace productivity, slower adoption of future health guidance, and increased vulnerability to misinformation in the next public health crisis. Acknowledging these cognitive consequences is not alarmism. It is a necessary precondition for designing effective responses. The question is not whether the pandemic impaired cognitive receptivity the evidence answers that question clearly. The question is what we will do about it. The answer must begin with recognising that cognitive receptivity is a public health resource, as vital as vaccine supply chains or hospital bed capacity, and that protecting and restoring it must be a core objective of pandemic preparedness for the future.

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