Affective Determinants of Health (ADoH): A Conceptual, Evidentiary, and Mechanistic Framework

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Author Note

This is the first of several possible translational investigations into public health, policy, population management, clinical, academic, and other potential implications of the emerging field of affective determinants of health.

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Abstract

Persistent negative feelings such as stress, loneliness, fatigue, pain, irritability, anxiety, and depressed mood are more than temporary discomforts. When they endure, even at subclinical levels, they predict disease, disability, and premature mortality as strongly as many traditional medical and social risks. Yet decades of evidence have not yet been organized into a framework that recognizes these states as determinants of health in their own right. This article advances the concept of Affective Determinants of Health (ADoHs), a category parallel to Social Determinants of Health (SDOH). We define affectivity as the systemic capacity to feel, affects as its phenomenal instances, and affective determinants as the subset of affects that meet criteria for determinant status.

A novel contribution of this framework is the recognition that persistent affects function not only as indicators of strain, suffering, and distress, but as stressors themselves, producing cumulative biological and psychosocial strain. To explain their operation, we introduce a Three-Channel Model of biological, behavioral, and coping pathways and describe the construct of cumulative affective load, which parallels allostatic load. By consolidating diverse evidence, we argue that systematic recognition of ADoHs as transdiagnostic determinants can improve early detection, prevention, and intervention across clinical care, population health, workplaces, education, and policy. Recognizing ADoHs highlights both the costs of neglect and the opportunity to address the affective foundations of health, equity, and human functioning.

Keywords: affective determinants of health, affectivity, social determinants of health (SDOH), emotion, health disparities

Public Significance Statement

Persistent feelings such as stress, loneliness, fatigue, pain, irritability, anxiety, and depressed mood are not just symptoms of illness or discomforts of daily life. When they last over time, even below diagnostic thresholds, they increase the risk of serious health problems, disability, and early death. This article proposes that these states be recognized as Affective Determinants of Health (ADoHs), in parallel with how social determinants like income, education, and neighborhood conditions are now acknowledged. Framing them as determinants clarifies their predictive power, mechanisms of action, and modifiability. It also highlights opportunities for intervention across healthcare, workplaces, schools, and communities. Naming and addressing ADoHs makes visible an insufficiently consolidated and operationalized domain of health risk and prevention, with potential to reduce suffering, improve equity, and strengthen the foundations of public health.

Introduction

Affectivity has been the subject of extensive theoretical proposals and empirical research. Drawing from core affect models (Russell, 1980), constructivist perspectives (Barrett, 2017), embodied accounts (Damasio & Damasio, 2023), component process theories (Scherer, 2009), enactive and phenomenological approaches (Maiese, 2023; Colombetti, 2014), as well as control-process models (Carver & Scheier, 1990) and predictive frameworks such as Affective Inference Theory (Velasco & Loev, 2021), this paper integrates these insights into a unified definitional framework and argues that certain affective experiences (affects) constitute risk factors independent of their status as symptoms of disorders or diseases.

The idea that affective states can act as determinants of health and functioning is not without precedent. Dolezal and Lyons (2017) argued that health-related shame operates as an affective determinant, showing how it influences healthcare access, adherence, and wellbeing in ways that parallel social determinants. Similarly, *Affective Determinants of Health Behavior* (Williams, Rhodes, & Conner, 2018) highlighted how affective attitudes, anticipated emotional responses, and related constructs influence health behaviors. These contributions foreshadow the present thesis but focus either on a single affect or on health-related behaviors. Our framework extends this groundwork by identifying a broader class of affects that rise to determinant status and by situating them within a comprehensive definitional and mechanistic model.

The eight affects emphasized here—stress, anxiety, depressed mood, irritability, loneliness, fatigue, pain, and malaise—are among the most common complaints in primary care, behavioral health, and population surveys (Kroenke & Mangelsdorff, 1989; Kessler et al., 2003, 2005; Holt-Lunstad et al., 2015; Cohen, Janicki-Deverts, & Miller, 2007; Ferrari et al., 2013; WHO, 2017; Servaes, Verhagen, & Bleijenberg, 2002; Von Korff et al., 1992; Stringaris & Goodman, 2009; Vidal-Ribas et al., 2016; Kumari, Head, & Marmot, 2004). Their prevalence, persistence, and well-documented associations with morbidity, mortality, and functional costs make them especially strong candidates for determinant status, as elaborated below.

Our aim is fourfold: to define affectivity as the systemic capacity to feel; to delineate affects as its phenomenal instances; to specify criteria for identifying which affects qualify as ADoHs based on epidemiological and mechanistic significance; and to introduce a three-channel model explaining their operation through biological/allostatic, behavioral, and coping-related pathways. In doing so, we introduce the concept of cumulative affective load, which parallels the concept of allostatic load.

By providing this conceptual language, we aim not to displace prior work but to consolidate it. Just as the field of social determinants of health (SDOH) unified diverse contextual factors under a single framework, ADoHs can consolidate diverse affective experiences under a framework that is simultaneously phenomenological, mechanistic, and population-relevant.

Special Focus: Historical Lineage of Affectivity

From Aristotle's reflections on passions to William James's embodied psychology and beyond, affectivity has long been regarded as central to human life (Aristotle, trans. 1991; James, 1884). Early modern philosophers debated its role in reason and action; Descartes (1649/1989) framing passions as mechanistic processes of mind–body interaction, and Spinoza (1677/1996)

presenting affects as essential to *conatus*, the striving for persistence. Though sidelined during the rise of behaviorism and cognitive science (Hilgard, 1980), psychology re-engaged affectivity with systematic accounts such as Frijda's (1986) laws of emotion. Modern perspectives further reasserted its importance: control-process theories link affect to goal pursuit (Carver & Scheier, 1990), evolutionary psychiatry emphasizes adaptive functions (Nesse, 2019), and stress physiology and neuroscience confirm health relevance (McEwen, 2017; Damasio, 2010). Selye's (1956/1976) formulation of stress as biological strain, and McEwen's (1998, 2017) elaboration into allostatic load, demonstrated that chronic affect-laden strain can be biologically toxic. This lineage contextualizes the recognition of affective determinants of health today.

Defining Affectivity

Affectivity can be defined as a whole-body (albeit predominantly neural) system of processes that generates a felt sense of what matters, in what ways, and to what degrees, based on interoceptive, somatosensory, exteroceptive, mnemonic, cognitive, and imaginal inputs processed concurrently, though not always equally (Craig, 2002; Damasio & Damasio, 2023; Simmons et al., 2013; Barrett & Bliss-Moreau, 2009; Barrett & Satpute, 2013; Schacter et al., 2008). Affective phenomena thus include both the systemic processes and the conscious, valenced experiences that emerge from them (Barrett & Bliss-Moreau, 2009; LeDoux & Hofmann, 2018; Russell, 1980). This system initiates, directs, modifies, and ceases attention and motivated behavior with varying phenomenal intensity and temporal persistence (LeDoux & Hofmann, 2018; Wrzus & Luong, 2016; Yap et al., 2017). Its essence lies in the felt orientation of the organism to its internal milieu and environment (Russell, 1980; Posner, Russell, & Peterson, 2005; Zych & Gogolla, 2021).

For the purposes of this framework, affects are the phenomenal instances of affectivity. This usage encompasses emotions, moods, and other transient or persistent feeling-states, while avoiding contested distinctions (e.g., Barrett, 2016; Lindquist et al., 2012; Adolphs, 2016) and we do not seek here to resolve any such debates. By foregrounding their shared property—conscious, valenced experience that orients attention and behavior—we provide a transdiagnostic vocabulary useful for clinical, public health, and policy applications.

Characteristics of Affects

Affects share several principal characteristics. They are subjectively experienced, constituting the phenomenal "what-it-feels-like" dimension of mental life (Nagel, 1974; LeDoux & Brown, 2017). They are temporally flexible, occurring in anticipation, during encounters, or retrospectively as relief or regret (Nesse, 1990; Dolan, Dayan, & Daw, 2022). All involve phenomenal valence, experienced as positive, negative, or mixed (Russell, 1980; Cacioppo, Gardner, & Berntson, 1999). They vary in intensity, which is phenomenally distinct from arousal (Ventura-Bort, Wendt, & Weymar, 2021), though arousal is a frequent concomitant (Barrett & Bliss-Moreau, 2009; Russell, 1980).

Affects bias action tendencies and shape behavior. Control-process theory conceptualizes affect as feedback about goal progress (Carver & Scheier, 1990, 1998), while Affective Inference Theory situates affect within predictive processing, proposing that feelings are inferences about

action viability and expected outcomes (Velasco & Loev, 2021). Both perspectives converge on affect's role as action-guiding.

Affects also exert cognitive and physiological consequences, influencing decision-making, memory, and stress regulation (Clore & Huntsinger, 2007; Dolcos et al., 2017; McEwen, 2017). Expression—facial, postural, or vocal—may occur but is neither necessary nor reliable (Anderson & Guerrero, 1996; Zych & Gogolla, 2021). They can be linguistically labeled with varying granularity across individuals and cultures, influencing regulation (Lindquist & Barrett, 2008). Finally, affects can be categorized as discrete families (Ekman, 1992; Roseman, 2011) or located within dimensional spaces of valence and arousal (Russell, 1980; Posner et al., 2005; Loev, 2022). Affects may be continually experienced although not necessarily at the foreground of attentional awareness at any given moment.

We are mindful of the *homunculus fallacy*; our claim is not that affects "do" things as if they were little actors, but that they are embodied processes and lived dynamics (Dennett, 1991; Colombetti, 2014). They are instantiated in physiological regulation and simultaneously experienced as the felt sense of tension, vitality, or burden through which those processes are lived (Damasio, 2010). This experiential dimension is constitutive, not additive: the feeling is the way physiological regulation becomes consciously meaningful. Thus, to say that "affects exert effects" is shorthand for distributed processes that are mechanistic and experiential at once, emerging from and contributing to the functioning of bodily and psychological systems.

How Some Affects Become Enduring

Some affects become persistent because the same mechanisms that make them adaptive—orienting organisms to restore safety, connection, or equilibrium—can also trap them. Neural plasticity sensitizes circuits to repeated activation (Baliki, Geha, Apkarian, & Chialvo, 2008). Slow-acting mediators like cortisol and cytokines sustain tone across hours and days (McEwen, 2017). Biased attention and appraisal loops reinforce vigilance: loneliness heightens social threat sensitivity (Cacioppo & Hawkley, 2009), irritability strengthens hostile attribution bias (Stringaris & Goodman, 2009). Some background affects are dispositional, reflecting temperament or neurobiological set-points (Kagan & Snidman, 2004; Rothbart, Ahadi, & Evans, 2000). Others are acquired through trauma, recalibrating systems toward persistence (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Teicher & Samson, 2016). Gene–environment interactions amplify vulnerability (Caspi et al., 2003). Evolutionary pressures favor persistence over premature disengagement (Nesse, 2019). When corrective signals are absent, affects remain sensitized, transforming adaptive signals into enduring burdens that qualify as determinants.

Some affects are truly persistent, continuing as a background tone even without constant awareness. Core affective states may manifest as enduring mood (Russell, 2003), action tendencies can outlast episodes (Frijda, 1986), and neural processes shape behavior outside awareness (LeDoux & Brown, 2017). Philosophical accounts likewise highlight background affectivity as constitutive of meaning and motivation (Maiese, 2023). Others are *effectively* persistent: episodic states frequent or severe enough that, upon reflection, individuals report them as characteristic of their existence. Instruments such as the PHQ-9 and GAD-7 capture this retrospective recognition (Kroenke, Spitzer, & Williams, 2001; Spitzer, Kroenke, Williams, &

Löwe, 2006). For example, a person may deny continuous anxious affect yet endorse moderate anxiety on a two-week lookback because episodes recur often.

Research confirms that such retrospective recognition corresponds to genuine persistence: loneliness endures by heightening attentional bias toward social threat cues (Cacioppo & Hawkley, 2009), irritability shows temporal stability and predicts long-term impairment even when episodic (Stringaris & Goodman, 2009), and chronic pain involves network reorganization that sustains its burden (Baliki, Geha, Apkarian, & Chialvo, 2008; Apkarian, Hashmi, & Baliki, 2009). Subsyndromal depression exemplifies effective persistence, with recurrent low mood predicting impairment even absent continuous presence (Judd et al., 1996). Both persistent and effectively persistent forms forecast risk and are amenable to intervention, including psychotherapy for subthreshold states (Han et al., 2022).

The mechanisms that sustain persistence—neural sensitization, slow-acting mediators, biased appraisal loops, dispositional set-points, trauma recalibrations, and evolutionary pressures—explain how certain affects become recurrent or chronic. At the same time, these persistent affects can also be understood phenomenologically as *evaluative stances*: embodied orientations through which the world is disclosed in value-laden terms. From this perspective, stress reveals the world as over-demanding, anxiety as uncertain and potentially threatening, depression as emptied of possibility, irritability as filled with provocations, loneliness as socially barren, pain as injurious, fatigue as energetically unaffordable, and malaise as inhospitable or unwell. This phenomenological construal is consistent with enactivist and existential accounts of affectivity (Maiese, 2023; Ratcliffe, 2008; Colombetti, 2014). Such stances not only emerge from mechanistic processes but also reinforce them, because they reorganize meaning, bias attention, and shape behavior in ways that sustain their own conditions. Framing persistence in this dual manner—mechanistic and phenomenological— suggests they both *arise from* and *become* feedback loops that recalibrate health trajectories.

From Affects to Determinants

Although all affects belong to the broad system of affectivity, only a subset functions as determinants of health. Fleeting feelings and occasional transient moods can produce acute physiological changes, but evidence suggests they rarely accumulate into long-term morbidity or mortality risk unless they recur frequently or consolidate into persistent states (Segerstrom & Miller, 2004; Cohen, Janicki-Deverts, & Miller, 2007; Pressman & Cohen, 2005). Persistent or recurrent affects can assume a determinant-level role in shaping population outcomes.

The criteria for determinant status parallel those of social determinants of health: epidemiological significance, mechanistic plausibility, and modifiability (Marmot & Wilkinson, 2006; WHO, 2008; Braveman, Egerter, & Williams, 2011). Evidence from transdiagnostic interventions such as Barlow and colleagues' Unified Protocol shows that persistent affective states can be modified by targeting shared mechanisms of emotion regulation, avoidance, and maladaptive coping, further underscoring their determinant status (Farchione et al., 2012; Barlow et al., 2017). Determinant-level affects consistently predict disability, morbidity, and premature mortality. By definition, their influence is *transdiagnostic*, cutting across multiple disorders, diseases, and outcomes. They manifest across diverse outcomes; and they are modifiable by

psychosocial, medical, peer, community, digital, and other interventions (e.g., Lattie, Cohen, Winquist, & Mohr, 2019; Masi, Chen, Hawkley, & Cacioppo, 2011; Pfeiffer, Heisler, Piette, Rogers, & Valenstein, 2011; Shekelle et al., 2024; Zhang et al., 2024).

Several exemplars illustrate this class. Chronic loneliness predicts cardiovascular disease and all-cause mortality at magnitudes comparable to other risks (Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015). Persistently depressed mood ranks among the leading global contributors to disability (Ferrari et al., 2013; WHO, 2017). Chronic stress is implicated in immune, metabolic, and cardiovascular dysregulation (Cohen, Janicki-Deverts, & Miller, 2007). Fatigue, illness-related or not, forecasts outcomes beyond diagnostic boundaries (Servaes, Verhagen, & Bleijenberg, 2002). Irritability predicts suicide risk, aggression, and occupational impairment (Stringaris & Goodman, 2009; Vidal-Ribas, Brotman, Valdivieso, Leibenluft, & Stringaris, 2016). Malaise, often dismissed as nonspecific, nonetheless predicts absenteeism and cardiometabolic risk (Kumari, Head, & Marmot, 2004). Anxiety predicts depression, substance misuse, and medical morbidity (Craske et al., 2017). Malaise refers to a diffuse, subjective sense of diminished wellness or vitality, encompassing feelings of being unwell, weak, or sick. Like the construct of "poor subjective health" used in epidemiology, malaise captures a broad felt state that may arise independently or in the context of medical or psychiatric conditions. Its presence, whether syndromal or subclinical, predicts functional decline and cardiometabolic risk (Kumari, Head, & Marmot, 2004).

Importantly, determinant status extends to sub-clinical but enduring levels across all major exemplars. Non-syndromal fatigue and malaise predict absenteeism and cardiometabolic risk (Kumari, Head, & Marmot, 2004; Servaes, Verhagen, & Bleijenberg, 2002). Low-to-moderate but persistent loneliness and anxiety contribute to cardiovascular risk and functional decline (Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015; Craske et al., 2017). Sub-threshold depressed mood forecasts disability and increased service utilization even outside major depressive episodes (Judd, Paulus, Wells, & Rapaport, 1996). Chronic stress at modest levels predicts dysregulated immune and metabolic function (Cohen, Janicki-Deverts, & Miller, 2007). Persistent irritability, even when not syndromal, is associated with heightened risk of suicidality and long-term impairment (Stringaris & Goodman, 2009). Likewise, recurrent pain of moderate intensity, including musculoskeletal pain, predicts functional decline and disability (Von Korff, Ormel, Keefe, & Dworkin, 1992). Taken together, these findings underscore that it is persistence and recurrence—rather than severity alone—that confer determinant-level significance.

The Three-Channel Model

Predictive validity identifies which affects qualify as determinants, but explanatory frameworks clarify how they exert effects. A Three-Channel Model organizes evidence across biological, behavioral, and coping domains, highlighting points of intervention and hypotheses about cumulative burden.

Biological/allostatic channel
Persistent affects disrupt physiological regulation in ways paralleling allostatic load (McEwen, 1998, 2017). Stress, anxiety, and pain dysregulate the hypothalamic–pituitary–adrenal axis, immune signaling, and cardiovascular tone (Segerstrom & Miller, 2004; Institute of Medicine,

2011). Loneliness alters immune gene expression, including pro-inflammatory profiles (Cole et al., 2011). Depression and fatigue are likewise linked to systemic inflammatory changes and metabolic dysregulation, while irritability and malaise often co-occur with heightened physiological reactivity, sustaining low-grade toxic strain. From the perspective of Affective Inference Theory, repeated inferences of insufficiency or threat keep physiological systems on chronic alert, producing cumulative biological effects. Persistent affects disrupt physiological regulation in ways paralleling allostatic load (McEwen, 1998, 2017). In this sense, they do not merely signal the presence of stress but act as stressors themselves, sustaining biological strain through chronic activation.

Behavioral/psychosocial channel

Because affects are motivational, persistent negative states bias action in maladaptive directions. Stress, depression, and fatigue reduce activity, disrupt sleep, and erode adherence to healthy routines (Katon, 2011; Tice, Bratslavsky, & Baumeister, 2001). Anxiety increases vigilance and avoidance, limiting opportunity and functioning (Craske et al., 2017). Loneliness and irritability foster withdrawal and conflict, degrading support networks (Holt-Lunstad et al., 2015; Zohar, 1999). Pain contributes to physical inactivity and social disengagement, while malaise amplifies absenteeism and diminished role functioning. Across these affects, treatment non-adherence emerges as a critical pathway: depression, stress, and related burdens reliably predict lower adherence to medical regimens and preventive behaviors (DiMatteo, Lepper, & Croghan, 2000; Grenard et al., 2011). In this way, everyday behaviors become distorted, sustaining and magnifying health risk.

Coping/regulatory channel

Coping strategies motivated by a desire to avoid, diminish the intensity of, or reduce the duration of unpleasant affects often provide short-term relief but can impose long-term harm. Substance use, compulsive eating, and reliance on sedatives or stimulants exemplify maladaptive relief (Cooper, Frone, Russell, & Mudar, 1995). Poor nutrition, sedentarism, and compulsive distraction (Meier & Reinecke, 2020; Roberts, Yaya, & Manolis, 2014), as well as rumination, suppression, or rigid avoidance (Gross & John, 2003; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), entrench cycles of burden. These patterns cut across conditions: fatigue may invite overreliance on stimulants, pain may prompt opioid use, stress and anxiety may drive compulsive distraction, depression and malaise foster withdrawal and avoidance, and irritability increases maladaptive confrontation. Thus, the coping channel shows how various determinants perpetuate risk through regulation strategies that can backfire.

Although analytically distinct, the three channels are mutually reinforcing. Biological dysregulation increases vulnerability to maladaptive behavior; behavioral shifts amplify coping demands; and maladaptive coping worsens physiological strain and functioning. These feedback loops magnify risk and can prolong the persistence of a given affect. For example, loneliness may prompt excessive alcohol use and social media consumption, which disrupt sleep, thereby heightening irritability and alienating behaviors, further sustaining the lonely state. In this way, interactions across channels can entrench a single determinant over time. In practice they are causally entangled, linked by bidirectional feedback and effects. Hypotheses and interventions would anticipate cross-channel spillover.

Special Focus: Cumulative Affective Load

Like allostatic load in biology (McEwen & Stellar, 1993; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; McEwen, 1998; McEwen & Gianaros, 2010), cumulative affective load captures the lived burden of multiple affects—stress, fatigue, loneliness, irritability, pain—occurring together. In this framework, each persistent affect represents a stressor itself, with cumulative risk arising from their interaction. Distinct affects retain unique pathways yet interact synergistically, creating toxic combinations. Importantly, cumulative affective load can be assessed through self-report and behavioral measures, offering a clinically accessible complement to biomarker-based allostatic load (Kumari, Head, & Marmot, 2004; Holt-Lunstad et al., 2015; Stringaris & Goodman, 2009).

Whereas allostatic load emphasizes cumulative physiological strain, cumulative affective load highlights the subjective and behavioral dimensions through which strain is lived. It is not proposed as a replacement but as a complementary construct, extending the explanatory reach of allostatic load by incorporating the affective burdens that erode resilience and accelerate decline. We propose that cumulative affective load may forecast the emergence of allostatic load and operate concurrent with it, as sustained affective pressures and biological dysregulation tend to develop in parallel and reinforce one another.

Integration

The framework can be understood as a progressive sharpening of focus: affectivity as the systemic capacity for felt prioritization; affects as the phenomenal instances of this system; affective determinants of health as the subset that rise to determinant status; and the Three-Channel Model as the explanatory mechanism.

Notably, in lived experience, determinants rarely occur in isolation, and distinct ADoHs frequently exacerbate one another across the same channels. Stress, anxiety, depression, irritability, fatigue, loneliness, pain, and malaise often co-occur and interact (Kessler et al., 2005; Kroenke et al., 2010). Chronic pain interacts with depression and fatigue (Bair et al., 2003; Finan, Goodin, & Smith, 2013). Loneliness amplifies depressive symptoms and anxiety (Cacioppo et al., 2010; Wang et al., 2018). Irritability contributes to conflict, reinforcing isolation and stress (Pickles et al., 2010; Stringaris & Goodman, 2009).

Cumulative affective load helps explain these dynamics. Allostatic load describes accumulating physiological strain from repeated stress; cumulative affective load describes the accumulating burden of interacting affective determinants. Distinct appraisal profiles (Roseman, 2011) suggest that loneliness, fatigue, and irritability each contribute unique risks even when co-occurring. The framework therefore emphasizes both the distinctiveness of each determinant and their potential to combine into a cumulative burden with wide-ranging effects.

Context and Broader Influences

Affective determinants of health do not emerge in isolation from broader social, developmental, and structural conditions. Social determinants, adverse childhood experiences, trauma, and

inequities—including racial and economic disparities—shape the onset, intensity, and expression of affective states. Adverse childhood experiences are linked to chronic stress, depression, and pain syndromes later in life (Felitti et al., 1998; Hughes et al., 2017). Experiences of discrimination and inequity increase anxiety, irritability, and physiological stress dysregulation (Williams, Lawrence, & Davis, 2019). Social gradients in health influence exposure to negative affective states (Marmot, 2005). While trauma and structural adversity strongly amplify risk, research also shows that persistent affective determinants can arise from the accumulation of everyday stressors. Daily diary and epidemiological studies demonstrate that chronic minor hassles—work strain, interpersonal conflict, caregiving burdens—predict sustained negative affect, dysregulated physiology, and long-term health decline (Almeida, 2005; Bolger, DeLongis, Kessler, & Schilling, 1989; Zohar, 1999; McEwen, 1998).

ADoHs therefore not only mediate and moderate the effects of broader conditions but also generate and perpetuate them. Persistent depression, fatigue, and pain reduce productivity and shorten work-life expectancy, contributing to economic insecurity (Kessler et al., 2006; Pedersen et al., 2019; Von Korff et al., 1992). In parents, depressive and irritable states diminish caregiving quality and increase conflict, thereby creating adverse home environments that become exposures for children (Heim et al., 2008; Teicher & Samson, 2016). Depressive symptoms in adolescence forecast lower educational attainment, seeding later socioeconomic disadvantage (Judd et al., 1996). Loneliness and irritability can destabilize social ties, leading to erosion of protective networks (Cacioppo & Hawkley, 2009). In this way, affective determinants act as stressors that shape the very structural and developmental risks often treated as upstream. Clarifying these bidirectional dynamics is essential for explanatory accuracy and policy relevance.

Implications

Population health

Acknowledging affective states as determinants allows them to be measured and monitored alongside smoking, hypertension, and social disadvantage—not only as correlates of stress, but as stress exposures in their own right. Incorporating ADoHs into large-scale surveys and surveillance systems would clarify their contributions to morbidity, mortality, disability, and health-span. Routine inclusion of measures for stress, loneliness, fatigue, and related affects would enable better risk stratification, helping to identify high-burden subgroups before clinical conditions manifest. Population-level tracking would also illuminate interactions with other determinants, such as how economic insecurity amplifies fatigue or how social isolation accelerates pain-related disability, providing a fuller picture of cumulative risk.

Research

Despite their prevalence, ADoHs remain undermeasured. Depression and anxiety screening has gained traction (USPSTF, 2016, 2023; Siu et al., 2016; O'Connor, Rossom, Henninger, Groom, & Burda, 2016), but stress, fatigue, pain, irritability, loneliness, and malaise are seldom assessed in standardized form (Kumari et al., 2004; Holt-Lunstad et al., 2015; Institute of Medicine, 2011; Stringaris & Goodman, 2009). Key gaps include identifying which affects, in what combinations and durations, best predict outcomes. Closing these gaps requires longitudinal designs, EHR integration (Taylor, 2006), and linkage with biological and social data (McEwen, 2017; Williams

et al., 2019). Advanced methods such as machine learning, network modeling, and digital phenotyping can detect nonlinear dose–response and interaction effects (Bzdok & Meyer-Lindenberg, 2018; Insel, 2017; Robinaugh et al., 2020).

Clinical care

In clinical settings, ADoHs can be integrated into routine workflows with minimal burden. Brief self-report or digital assessments of stress, fatigue, pain, loneliness, irritability, and depressed mood provide actionable data for early detection and stepped care (Donovan & Blazer, 2020; Katon, 2011). Incorporating these measures into annual wellness visits, chronic disease management, remote monitoring, and behavioral health encounters expands the set of modifiable risks clinicians can address. Embedding ADoH metrics in electronic health records not only enhances prognostic accuracy (Taylor, 2006) but also allows longitudinal monitoring and proactive outreach when thresholds are exceeded. Evidence from digital and peer-supported interventions shows that even low-intensity strategies, applied early, reduce burden (Lattie et al., 2019; Pfeiffer et al., 2011). Recognition of ADoHs therefore equips clinicians to move beyond reactive treatment toward anticipatory prevention and transdiagnostic care.

Workplaces and education

Affective determinants influence performance, safety, and development across the lifespan. In workplaces, stress, fatigue, and irritability contribute to absenteeism, presenteeism, turnover, and error rates, all of which carry substantial productivity and cost implications (OECD, 2015; WHO, 2020). Organizations that address psychological safety and reduce affective burden not only mitigate risk but also foster engagement and resilience (Kivimäki & Steptoe, 2018). In education, fatigue, anxiety, loneliness, and irritability undermine attention, learning, and social development. Conceptualizing these states as determinants underscores the importance of early screening, supportive environments, and resilience-building interventions in schools (WHO, 2020). Addressing ADoHs in work and educational contexts therefore functions as both a performance strategy and a long-term public health investment.

Policy

Just as codification of social determinants reshaped global health priorities, recognition of affective determinants could guide investment, reimbursement, and regulation. Insurance coverage and payment models could incentivize ADoH screening and early intervention; community health planning could incorporate local affective risk profiles; and workplace standards could address fatigue, stress, and psychological safety as systematically as physical hazards (WHO, 2013; Institute of Medicine, 2013). International bodies that embraced SDOH as a framework for equity could similarly adopt ADoHs, strengthening policy coherence across health, education, and labor sectors (Marmot, Allen, Boyce, Goldblatt, & Morrison, 2020). At the policy level, recognition of affective determinants repositions them not as soft or ancillary concerns but as foundational drivers of health and equity, warranting coordinated, cross-sector action.

Conclusion

The evidence for recognizing a class of affective phenomena that operate as *transdiagnostic risk factors* for disease, disability, and premature mortality is compelling. To label these states as

Affective Determinants of Health (ADoHs) is not a mere rebranding, but a necessary consolidation that highlights their collective importance alongside social, behavioral, and biological risks.

The significance of this recognition lies in its practical implications. Early detection and ongoing monitoring of ADoHs, paired with effective prevention and intervention strategies, can reduce suffering, lower costs, improve equity, and save lives. Determinant status does not hinge on diagnostic severity; sub-clinical but enduring forms of stress, loneliness, fatigue, irritability, pain, depressed mood, and malaise are epidemiologically consequential, making their recognition essential.

While this article has focused on persistent negative affects that operate as risks, the broader field of ADoH could also encompass positive and protective affects. Experiences such as hope, gratitude, social connectedness, and vitality are likewise enduring states that predict resilience, recovery, and long-term health (Fredrickson, 2001; Cohn et al., 2009; Pressman & Cohen, 2005; Stellar et al., 2015). Their omission here reflects the need to first establish the determinant status of burdensome affects, which have historically been overlooked despite robust evidence of risk. Yet the same framework that demonstrates how stress, loneliness, and fatigue undermine health can also explain how protective affects foster adaptation and flourishing. Future work may therefore extend ADoH beyond risk to include promotive factors, ensuring a balanced science of affective determinants that addresses both vulnerability and resilience.

Whether a formal field of ADoH research and practice should be consolidated remains an open question, subject to institutional priorities and disciplinary boundaries. Nevertheless, the time has come to recognize the affective determinants of health reviewed here, and potentially others as research may reveal, as central to the challenges and opportunities of modern healthcare and policy. Systematic recognition and integration of ADoHs into research, clinical care, public health, workplaces, education, and policy not only promises to transform individual outcomes and collective wellbeing, but also to advance equity, reduce costs, and reshape how health systems confront the affective foundations of human functioning.

Special Focus: Costs of Persistent Subclinical Affects

Persistent affects exert measurable health and economic costs even at low-to-moderate, subclinical levels. Key studies demonstrate this across all eight exemplars:

- **Stress.** Modest chronic stress predicted immune dysregulation and cardiovascular morbidity, with downstream health costs (Cohen, Janicki-Deverts, & Miller, 2007).
- **Loneliness.** Meta-analytic evidence shows persistent loneliness increases premature mortality risk on par with smoking and obesity (Holt-Lunstad, Smith, Baker, Harris, & Stephenson, 2015).
- **Fatigue.** Non-syndromal fatigue in cancer survivors predicted absenteeism and reduced role functioning beyond diagnostic categories (Servaes, Verhagen, & Bleijenberg, 2002).

- **Pain.** Recurrent moderate musculoskeletal pain forecasted functional decline and work disability, not only severe pain syndromes (Von Korff, Ormel, Keefe, & Dworkin, 1992).
- **Irritability.** Subthreshold irritability in youth predicted long-term social and occupational impairment (Stringaris & Goodman, 2009).
- **Anxiety.** Subclinical anxiety increased risk for depression, substance use, and medical morbidity, with added healthcare burden (Craske et al., 2017).
- **Depressed mood.** Persistent but subsyndromal depressive symptoms forecasted disability and greater service utilization (Judd, Paulus, Wells, & Rapaport, 1996).
- **Malaise.** Moderate malaise scores predicted cardiometabolic risk and absenteeism in the Whitehall II study (Kumari, Head, & Marmot, 2004).

Together, these findings underscore a central point: Determinant status does not depend on diagnostic severity. Even modest but enduring burdens reliably forecast health decline, disability, and economic loss, making their recognition critical for prevention and policy.

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