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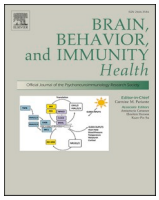

An Extension to the stress-buffering model: Timing of support across the lifecourse

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ABSTRACT

Children and adolescents exposed to severe stressors exhibit poorer health across the lifespan. However, decades of research evaluating the Stress-Buffering model suggests that social support can attenuate stressors' negative impacts. Psychoneuroimmunology research in this area has shifted from asking whether support buffers stress to when and why support would succeed (or fail) to confer protection. This article takes a lifecourse perspective and proposes that timing of support may shape support's protective value by defining the type of protection that is provided and its operating mechanisms. Specifically, it considers three temporal scenarios: support that occurs during, after, or before stressor exposure. When support intervenes at the same developmental stage as the stressor (concurrent support), buffering effects occur wherein support prevents the development of intermediary mechanisms that reflect or increase disease risk; when support is present at a developmental stage before stressor exposure (prior support), banking effects occur such that support intervenes indirectly by fortifying the individual with resilience-promoting characteristics that in turn prevents the development of intermediary mechanisms; finally, when support arrives at a developmental stage after stressor exposure (later support), counteracting effects occur such that support offsets the impacts of intermediary mechanisms on diseases. It further posits that a match between timing of support and the linkage of interest (e.g., the stressor-mechanism path vs. the mechanism-disease path) is necessary for successful protection. The present paper discusses these postulations, reviews nascent evidence, and proposes future directions.

An Extension to the Stress-Buffering Model: Timing of Support across the Lifecourse

Children and adolescents exposed to severe stressors (e.g., violence) are vulnerable to many diseases (e.g., cardiovascular diseases) in adulthood (Chen et al., 2022, 2023; Dube et al., 2009; Joseph et al., 2022). Though robust, these associations are not definitive. The seminal Stress-Buffering Model posits that social support during times of stress can buffer its negative impacts (Cohen and Wills, 1985), and at least three-decades of evidence accumulated, supporting it (e.g., Chen et al., 2017; Hostinar et al., 2014; Robles, 2021; Uchino et al., 2011). Building on this foundation, the next generation of psychoneuroimmunology (PNI) research on social support has started to shift from asking *whether* support buffers stress to *when* and *why* support would succeed (or fail) to confer protection. Taking a lifecourse perspective, this article proposes that timing of support can shape support's protective magnitude. Briefly, it considers three temporal scenarios, distinguished by the developmental stage when support occurs relative to stressor exposure (during, before, or after). It hypothesizes that the timing of support affects the type of protection that is offered (buffering, counteracting, and banking respectively) and the mechanistic action (prevents, offsets, and fortifies respectively). It further posits that a match between timing of support and the linkage of interest (e.g., the pathway connecting a stressor to an intermediary biomarker like low-grade inflammation vs. the pathway connecting an intermediary biomarker to a disease outcome like myocardial infarction) is necessary for successful protection, enabling more precise predictions about support's protective value across the lifecourse.

Given scant empirical research on support's timing, the current hypothesis is speculative. Therefore, rather than proposing a comprehensive model, the aim is to specify a working hypothesis that generates testable predictions, intended to offer a novel perspective for future PNI research. This article first provides brief overviews of the stressor-disease and stress-buffering models. Then, it proposes that timing can shape support's functions and provides preliminary evidence associated with each timing scenario. Future directions are then discussed.

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1. The childhood/adolescence stressor-disease association

Numerous models have proposed how childhood and adolescence stressors can impact physical health e.g., (Miller et al., 2011; Cohen et al., 2016; Repetti et al., 2002; Nusslock and Miller, 2016; Kuhlman et al., 2017). As depicted in Fig. 1A, a typical model begins with the notion that a childhood/adolescence stressor – a life event that poses excessive demands or harm, elicits substantial adaptation, or interrupts major goals (Cohen et al., 2019)—gives rise to psychological stress—the appraisal that perceived demand exceeds resources (Lazarus, 1966). In turn, this appraisal elicits short-term affective, behavioral, and physiological responses (Miller et al., 2009) involving changes in neuroendocrine (secretion of cortisol; Dickerson and Kemeny, 2004) and autonomic outflow (release of norepinephrine). As stress responses reflect transient changes, they alone are not pathogenetic (Miller et al., 2009). Rather, consistent with chains of risk theories (Umberson et al., 2014), an acute stressor can increase the probability of future stressors and/or increase the probability of lasting stress appraisals. The initial stressor and the future stressors/stress it begets may result in repeated activations, which can lead to longer-lasting changes in behavior and physiology (McEwen, 1998), particularly if experienced during childhood/adolescence when plasticity is heightened. For example, repeated stress may calibrate the tendencies of innate immune cells (monocytes) or brain networks (cortico-amygdala) so they have heightened responses to threat (Nusslock and Miller, 2016). Repeated stress may also elicit health-compromising behaviors, such as poorer sleep and increased substance use. Over time, these altered physiology and behavior, referred to as intermediary mechanisms, increase disease risk, eventually leading to a clinical disease (a medical condition that impairs one's functioning capacity).

Fig. 1B provides a more detailed example of how an acute severe stressor may contribute to cardiovascular disease via the immune pathway: children exposed to parental death may experience future stressors like eviction from their family home (Thompson et al., 1998), which contribute to, and are exacerbated by, heightened threat vigilance that increases the probability of stress appraisals (Luecken and Appelhans, 2005). These repeated exposures and activations signal low survival odds to the developing immune system, shaping it to enter a heightened state of preparedness (Belsky et al., 2012; Lam et al., 2022), characterized by a selective mobilization of pro-inflammatory monocytes into circulation (Miller et al., 2011). These cells accumulate in damaged blood vessel walls and mount excessive cytokine responses to danger signals and oxidized lipoproteins they encounter (Miller et al., 2011). Over time, those excessive responses contribute to the growth and instability of atherosclerotic plaques (Nathan and Ding, 2010). If one ruptures, the ensuing thrombus blocks blood flow, and eventuates myocardial infarction.

This long chain of events can be parsed into three parts (Miller et al., 2009): (a) the "stressor-mechanism path", which refers to the association between a stressor and an intermediary mechanism ("a path" in Fig. 1A; the link between parental death and altered monocytes/increased circulating inflammation in Fig. 1B); (b) the "mechanism-disease path", which refers to the association between an intermediate mechanism and a clinical disease ("b path" in Fig. 1A; the link between altered monocytes/increased circulating inflammation and atherosclerosis/myocardial infarction in Fig. 1B); and (c) the "stressor-disease path", which refers to the association between a stressor and a clinical disease ("c path" in Fig. 1; the link between parental death and atherosclerosis/myocardial infarction in Fig. 1B).

2. The stress-buffering model

The Stress-Buffering Model posits that social support—a social network's provision of psychological and material resources — experienced

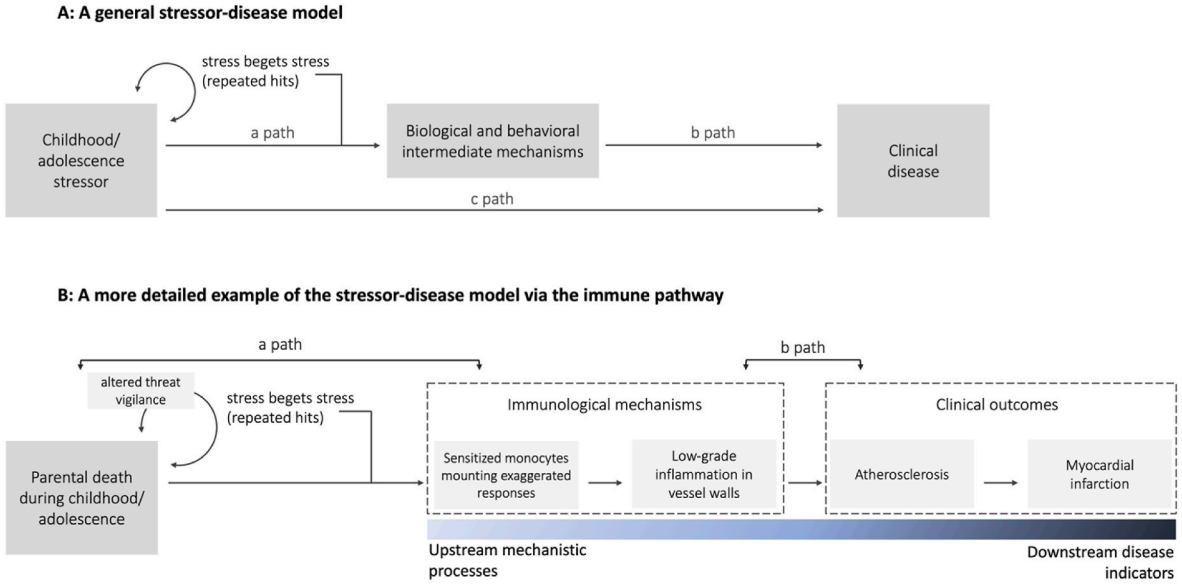

Fig. 1. Panel A depicts a general stressor-disease model. A childhood/adolescence stressor elicits psychological stress—the appraisal that perceived demand exceeds resources—which in turn elicits stress responses—short-term activations of affective, biological, and behavioral systems (not depicted for brevity). This stressor is thought to operate via (a) increasing future stressor exposures or likelihood of a lasting stress appraisal; and (b) biological and behavioral systems adapting to stress in ways that accrue disease risk, which over time manifest as clinical diseases. Panel B provides a more detailed example: parental death increases future stressors, which contribute to, and are exacerbated by, heightened threat vigilance. Both parental loss and the subsequent exacerbated stressors mobilize pro-inflammatory monocytes into circulation, which accumulate in damaged blood vessel walls, contributing to atherosclerotic processes like growth of plaques. If one of these plaques rupture, forming a thrombus that blocks blood flow, a myocardial infarction precipitates. This long chain of events can be parsed into three parts: (a) the "stressor-mechanism path", which refers to the association between a stressor and an intermediate mechanism (labeled as the "a path"); (b) the "mechanism-disease path", which refers to the association between an intermediate mechanism and a clinical disease (labeled as the "b path"); and (c) the "stressor-disease path", which refers to the association between a stressor and a clinical disease outcome (labeled as the "c path"). Note that for ease of interpretation, outcomes are categorized as intermediary biomarkers vs. clinical disease, but underlying this binary distinction is a continuum from upstream mechanistic processes to downstream disease indicators.

during times of stress can mitigate stress-related behavioral and biological adaptations thought to, over time, confer disease risk (Cohen and Wills, 1985). This model spurred decades of empirical research, reviewed by previous work (Hostinar et al., 2014; Robles, 2021; Uchino, 2009; Chen et al., 2017; Gunnar, 2017). Among research that focuses on youth stressors, the general conclusion is that stress-buffering was observed in observational studies of real-world stressors (e.g., poverty; Chen et al., 2011), laboratory studies that induced stressors (ruling out that protection is due to low stressor frequency confounding with high support; Gunnar, 2017), and experimental studies that manipulated social support (providing causal evidence that support led to protection; Miller et al., 2014; Perry et al., 2021). Furthermore, support attenuated the paths linking stressor/stress to multiple systems: autonomic (e.g., sympathetic nervous system [SNS] reactivity; Wade et al., 2020), immune (e.g., poorer antibody responses following vaccination; Snyder et al., 1990), neural (e.g., amygdala reactivity; Eisenberger et al., 2007), and endocrine (e.g., cortisol reactivity; Hostinar et al., 2014). Evidence was also observed across the causal chain connecting stressor to disease: stress-buffering was observed for relative upstream biomarkers like transcription of inflammation-related genes and low-grade inflammation as well as relatively downstream ones like infectious disease incidence and mortality (Robles, 2021; Cohen et al., 2020; Chiang et al., 2018; Lam et al., 2025).

Moreover, support can be differentiated by its function (Cohen and Wills, 1985), such as emotional (comfort and reassurance) and instrumental (tangible assistance) as well as based on whether support is perceived (potential access to resources) or received (receipt of resources; Uchino, 2009). To this end, evidence has been observed across different functional categories (Guan et al., 2016; Jaffee et al., 2017) whether it be perceived or received (e.g., Hostinar et al., 2014; Millwood and Manczak, 2023). Finally, evidence has been observed across different assessments of support, including measures that are relatively subjective (e.g., self- or other-reported; Hazel et al., 2014) or objective (e.g., observer-coded support behaviors; Brown et al., 2020), and across different methodologies, including retrospective and real-time measures (e.g., Chiang et al., 2018; Lam et al., 2024). In sum, this model is well-established conceptually and empirically. Yet, support's protective magnitude varies from study to study, and in some cases, support fails to confer protection (e.g., Berge et al., 2015; Gee et al., 2014). Identifying factors underlying the heterogeneity in support's protective value can refine theories and inform interventions, specifying when to intervene and how.

3. The proposed hypothesis

Numerous factors may explain the heterogeneity in stress-buffering magnitude: support's responsiveness (the match between the support strategy and the recipient's needs e.g., Cohen and Wills, 1985), aspect of support (e.g., perceived availability of support may be more beneficial than received support as the latter may elicit feelings of indebted and distress; Gleason et al., 2008; Uchino et al., 2011; Uchino et al., 2018) and developmental stage (e.g., relative to children, adolescents may benefit more from parental support that allows autonomy vs. that emphasizes physical proximity; Chen et al., 2017). Here, the article focuses on timing of support as a factor that has implications for how protection occurs. Indeed, the original Stress-Buffering Model (Cohen and Wills, 1985) discusses two timepoints at which support may intervene, which shapes how buffering occurs: when support prevents a stress appraisal, it buffers the path connecting stressor to stress responses, whereas when support attenuates stress responses, it buffers the path connecting stressor to intermediary mechanisms. This article takes a different approach by considering support's function as it occurs across the lifecourse, rather than the course of a stressor.

To first define terms and scope for this article, support is considered as a trait-like (vs. situational) construct that reflects the overall emotional and instrumental resources available to one by their social network (Uchino, 2009). Therefore, a broad set of interpersonal processes that give rise to supportive relationships (e.g., providing warmth) and attributes that are indicative of long-term supportive processes (e.g., being securely attached) are considered. This is consistent with Feeney and Collins' definition of social support as "an interpersonal process that unfolds over time" (Feeney and Collins, 2015), providing a rationale for considering support across longer periods. Furthermore, unlike other definitions of support (e.g., provision of psychological and material resources *intended to benefit an individual's ability to cope with stress*; Cohen, 2004), this definition allows support to occur in the absence of stressors (Feeney and Collins, 2015) and for support processes to be nonspecific to stressors, enabling differential hypotheses about how support operates when it concurs with a stressor vs. when it precedes a stressor or occurs after a stressor has ended. Note that although support may occur in the absence of a stressor, as in the original Stress-Buffering Model (Cohen and Wills, 1985), support's *functions and impacts* are always considered in the context of a stressor, and thus this article considers interaction effects with support being a moderator, rather than main effects of support on health.

In addition, this article focuses on stressors that occur during childhood or adolescence (before age 18) and how timing of support relative to stressor exposure is related to biological or disease outcomes. The types of stressors covered include severe acute stressors, such as exposure to violence, natural disasters, and parental death. The focus on acute stressors allows isolation of different timing scenarios, such as enabling hypothesizing about the effects of later support (support that occurs after the stressor has ended) without concerns for conceptual redundancy with concurrent support.

Finally, timing of support is defined relative to the stressor exposure (during, before, and after) in the unit of developmental stage (roughly, childhood [before age 13], adolescence [age 13–17], young adulthood [age 18 to 39], middle adulthood [age 40 to 65], and older adulthood [over age 65]; Repetti et al., 2011; Healthy People, 2020; Beyer and Lazzara, 2020; Arnett et al., 2014). Accordingly, the presence of support refers to high level of emotional or instrumental support experienced from at least one important other during a given developmental stage, referred to as high aggregated level support below. As depicted in Fig. 2 and summarized in Table 1, this conceptualization of timing results in three scenarios.

1. Concurrent support–the presence of high aggregated level of support during the same developmental stage as the stressor exposure—is hypothesized to confer *buffering* effects wherein support prevents development of the stressor-mechanism path.
2. Prior support–the presence of high aggregated level of support during the developmental stage(s) preceding stressor exposure – is hypothesized to confer *banking* effects wherein support intervenes indirectly by fortifying the individual with resilience-promoting biological characteristics that in turn intervenes at the stressor-mechanism path.
3. Later support–the presence of high aggregated level of support during the developmental stage(s) after the stressor exposure—is hypothesized to confer *counteracting* effects wherein support intervenes at the mechanism-disease path by offsetting the impacts of already established intermediary mechanisms on disease outcomes.

4. Concurrent support

Support at the same developmental stage as the stressor confers buffering effects by preventing intermediary mechanisms from developing or becoming established. Mechanistically, concurrent support mitigates the likelihood of a stress appraisal or attenuate stress responses (Cohen and Wills, 1985). Theories on social regulation of emotion provide insights on potential interpersonal processes underlying such protection (Reeck et al., 2016; Zaki and Williams, 2013). For example, the caregiver of a child exposed to a community violent event may

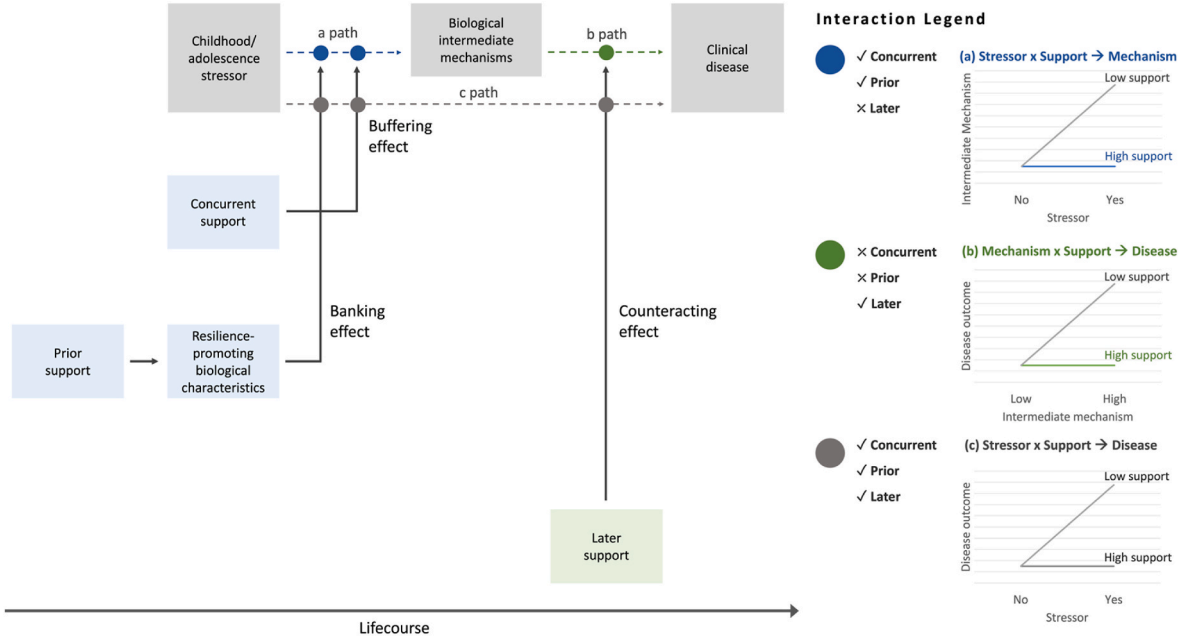

Fig. 2. Timing of support is proposed to affect the type of protection offered and the mechanistic action. The three timing scenarios are overlaid on the general stressor-disease mediation model. Support is postulated to attenuate the stressor-disease path ("c path"), but whether this occurs via attenuation of the stressor-mechanism path ("a path") or the mechanism-disease path ("b path") depends on timing of support. Concurrent support—the presence of high aggregated level of support during the same developmental stage as the stressor exposure—is hypothesized to confer buffering effects wherein support prevents development of the stressor-mechanism path ("a path"). Prior support—the presence of high aggregated level of support during the developmental stage(s) preceding stressor exposure—is hypothesized to confer banking effects wherein support intervenes indirectly by fortifying the individual with resilience-promoting biological characteristics that in turn intervenes in the stressor-mechanism path ("a path"). Later support—the presence of high aggregated level of support during the developmental stage(s) after the stressor exposure—is hypothesized to confer counteracting effects wherein support intervenes at the mechanism-disease path ("b path") by offsetting the impacts of already established intermediary mechanisms on disease outcomes.

utilize attentional deployment (e.g., distract the child from the violent scene) to prevent a stress appraisal or utilize cognitive change (e.g., highlight that the police have arrived to keep the community safe) or response modulation (e.g., offer comfort) to reduce stress responses. These interpersonal processes should reflect high levels of support over time, which has been linked to reduced threat-related neural activations (Coan et al., 2006) and increased regulation-related neural activations (Eisenberger et al., 2007) despite stressor exposure. Thus, concurrent support should (1) attenuate the link connecting stressors and intermediate mechanisms ("a path" in Fig. 1), manifested as a stressor × support interaction on intermediary mechanism (Fig. 2a); and (2) thereby, also attenuate the link connecting stressors and diseases ("c path" in Fig. 1), manifested as a stressor × support interaction on disease outcomes (Fig. 2c).

Family is a major part of youth's social lives, and protection arising from family support is observed across development. In childhood, stress associated with Hurricane Sandy was linked with post-hurricane reduction in electroencephalogram-assessed reward sensitivity only when parents exhibited a parenting style that focuses on punishment, but not when parents exhibited a parenting style that has been linked to parental warmth (Gao et al., 2021; Kessel et al., 2019). Furthermore, parental warmth reduced alterations to cortisol's circadian pattern among youth exposed to interpersonal conflicts (Lippold et al., 2016). In adolescence, parental support continues to attenuate the link between conflicts and cortisol activities (Hanson and Chen, 2010). Extending to immune processes, adolescents with greater demands had immune cells that mounted more exaggerated inflammatory response to challenges only among youth with less (vs. more) supportive family relationships (Levine et al., 2017).

As a result of curbing the stressor-mechanism path, concurrent support should also attenuate the stressor-disease path. Indeed, young children who were indirectly exposed to violence (maternal intimate partner violence) had increased risk for asthma two years later only among those who had low, but not high, caregiver support (Suglia et al., 2009). In sum, there is evidence that concurrent support may buffer the stressor-mechanism and stressor-disease paths.

5. Prior support

Social support has largely been examined in negative contexts (Gable et al., 2012; Leatham and Duck, 1990), despite conceptual models highlighting support's utility in positive and mundane contexts (Feeney and Collins, 2015). For example, Feeney and Collins proposed that support, in the absence of adversity, can promote full participation in life opportunities by providing a secure base for exploration, increasing capitalization, and nurturing desires to seize growth opportunities (Feeney and Collins, 2015). In turn, these support processes may promote neural and physiological functioning, such as increased activation of neural areas associated with reward and positive affect (Eisenberger and Cole, 2012), connecting support experienced in non-negative contexts to thriving, a broad construct that includes better physical health (Feeney and Collins, 2015). The current hypothesis similarly postulates that support, in the absence of adversity, can shape biological systems in enduring manners; however, it is extended to propose that these biological benefits may be "banked" to subsequently buffer the negative impact of a future stressor experienced at a later developmental stage. That is, while Feeney and Collins' model (2015) details a main effect of how support in the absence of stress may lead to better health, the current hypothesis speculates that the biological characteristics imparted by support in the absence of stress can attenuate a future stressor's impacts.

Mechanistically, prior support fortifies youth by shaping autonomic and immune processes in ways that promote resilience, which in turn prevent later stressors from eliciting the biological cascade. In the

Table 1
Definition, examples, hypothesized mechanisms, and predicted results by timing of support.

	Concurrent Support	Prior Support	Prior Later Support
Definitions and examples	Definition: the presence of high aggregated level of support during the same developmental stage as the stressor exposure. Example: Exposure to gun violence during childhood + high level of caregiver support during childhood.	Definition: the presence of high aggregated level of support during the developmental stage preceding stressor exposure. Example: Exposure to gun violence during adolescence + high level of caregiver support during childhood.	Definition: the presence of high aggregated level of support during a developmental stage after the stressor exposure. Example: Exposure to gun violence during childhood + high level of romantic partner support during adulthood.
Hypothesized mechanisms	Buffering effect: support prevents development of the stressor-mechanism path by mitigating the likelihood of a stress appraisal or attenuating stress responses (e.g., via reduced threat reactivity and increased coping capacities).	Banking effect: prior support intervenes the stressor-mechanism path, like concurrent support. However, it intervenes *indirectly* by fortifying the individual with resilience-promoting biological characteristics (e.g., higher resting PNS activity, decreased NF-κB signaling) that in turn interfere with a future stressor's ability to promote intermediate mechanisms.	Counteracting effect: Assumes that the stressor-mechanism path is established such that later support cannot prevent or reverse its development due to declining plasticity. Later support intervenes the mechanism-disease path by offsetting the impacts of mechanisms on disease outcomes (e.g., via increased release of oxytocin).
Predicted results	1. Concurrent support attenuates the stressor-mechanism path (stressor x support → mechanism in Fig. 2a). 2. Thus, concurrent support should also attenuate the stressor-disease path (stressor x support → disease in Fig. 2c).	1. Prior support should attenuate future stressor-mechanism path (stressor x support → mechanism in Fig. 2a). 2. Thus, prior support should also attenuate future stressor-disease path (stressor x support → disease in Fig. 2c). 3. These protective patterns should emerge via banked characteristics.	1. Later support would not be as effective in attenuating the stressor-mechanism path. 2. Later support should offset the mechanism-disease path (mechanism x support → disease in Fig. 2b). 3. Thus, later support should also attenuate the stressor-disease path (stressor x support → disease in Fig. 2c).

autonomic pathway, parental warmth has been associated with resting (trait-like) parasympathetic nervous system (PNS) activities (Calkins et al., 2008; Köhler-Dauner et al., 2022), which facilitate "rest and digest". In turn, youth with higher resting PNS activity exhibited lower stress reactivity (Rahal et al., 2023) and better mood repair capabilities (Yaroslavsky et al., 2016). In the immune pathway, early positive relationships may modulate the expression of inflammation-related genes thought to mediate inflammatory cytokine production. When innate immune cells detect pathogen-associated molecular patterns, transcription factors like nuclear factor kappa B (NF-κB) become activated. This activation leads to the transcription of specific genes into messenger RNA, which is then translated into proteins, such as interleukin-6, that facilitate inflammatory responses (Webster et al., 2002). As such, dampened NF-κB signaling may interfere with a later stressor's ability to sensitize monocytes towards producing excessive inflammatory responses. This speculation is based on disparate literature. Specifically, youth experiencing greater parental warmth exhibited lower expression of genes with NF-κB response elements (Robles et al., 2018). This reduction in gene expression is expected to limit the subsequent production of proinflammatory cytokines by decreasing the translation of associated proteins (Ghosh et al., 1998). If these connections are causal and corroborated in larger samples, a future stressor's capacity to sensitize monocytes may be limited by the warmth-induced downregulation of NF-κB signaling. Additionally, systems may work in tandem as PNS can downregulate NF-kB signaling via acetylcholine (Pinheiro et al., 2015). Indeed, manipulated increase in PNS activity also attenuated endotoxin-stimulated production of inflammation markers (Lehrer et al., 2010).

If these postulations are true, then (1) prior support should attenuate future stressor-mechanism path (Fig. 2a); (2) and the future stressor-disease path (Fig. 2c); but (3) these protective patterns should emerge via banked characteristics. First, prior support may curb the stressor-mechanisms link. A longitudinal study assessed maternal attachment in a sample of young children (aged 2.5 years) and exposed them to a stressor (aversive social cues) in adolescence (aged 13–15 years; Rogers et al., 2022). Adolescents with early secure (vs. insecure) attachment 10 years ago displayed better behavioral regulatory responses and neural activation patterns that suggest less attention to aversive cues, providing preliminary evidence that prior support may attenuate the link between future stress and mechanisms. Importantly, adolescents with early secure attachment displayed better regulatory capabilities when they were exposed to the stressor alone, rather than when a parent was present. By contrast, adolescents with early insecure attachment exhibited poorer responses when they were exposed to the stressor alone, but not when a parent was present. These findings are consistent with the third postulation that protection conferred by prior support emerged because early support fortifies individuals with resources that enabled them to *independently* respond to future stressors in a regulated manner, not because of continued parental presence during these challenges. With regards to the second postulation, there isn't direct evidence due to lack of studies that examined clinical disease outcomes. However, greater parental warmth during childhood prevented the development of a clinical psychiatric disorder (post-traumatic stress disorder) following urban violence exposure in adulthood (Lima et al., 2014).

While this section sought research on support in the absence of stress during childhood attenuating the impacts of future stressors in adolescence or beyond, the small literature that examined early support either assessed it in a stressful context (e.g., institutionalization; Wade et al., 2020; Dauvermann et al., 2021; Hagan et al., 2011), examined main, rather than, stress-moderating effects (Köhler-Dauner et al., 2022; Lima et al., 2014), or did not assess biological or clinical outcomes (Hazel et al., 2014). Therefore, although there is indirect evidence from disparate literatures that prior positive relationships may shape autonomic and immune systems, which in turn have been found to modulate stress responses, studies have not formally examined the presented postulations.

6. Later support

What if the window for prior or concurrent support is missed, can support at a later developmental stage still change the events connecting the stressor to diseases? Later support may confer counteracting effects, wherein support offsets the impacts of mechanisms on diseases. Mechanistically, it's assumed that the stressor-mechanism path has already been set into motion such that support that arrives after may not be able to prevent or reverse its development. This is consistent with the notion that biological plasticity generally decreases with age (Lam et al., 2022). However, although biomarkers reflecting altered physiology are often used as outcomes in empirical research, they are not solely deterministic of diseases. Therefore, support at a later stage may still confer protection by intervening along the mechanism-disease path.

For instance, childhood exposure to violence calibrates monocytes towards pro-inflammatory tendencies, which gives rise to low-grade

inflammation, damaging tissues in ways that lead to cardiovascular diseases in mid/late-adulthood (Nathan and Ding, 2010). Having a supportive romantic partner in adulthood likely will not reverse monocytes' tendencies because plasticity decreases across the life-course. However, support may increase the release of oxytocin (Crockford et al., 2014), which, as demonstrated by experiments, reduced systemic release of inflammation markers following endotoxin challenge (Szeto et al., 2017). Notably, among rats with myocardial infarction, oxytocin reduced macrophages (monocytes that migrated into circulation) and apoptosis in the infarct region, reflecting improved functioning of the injured heart (Jankowski et al., 2010). As such, via increased oxytocin, later support may offset the impact of altered monocytes on downstream low-grade inflammation by reducing macrophages in circulation, and thus attenuates the link between altered monocytes and cardiovascular disease. In other words, later support may confer protection, not by reducing intermediate mechanisms (e.g., not by reversing monocytes' pro-inflammatory tendencies), but by promoting processes, like increased oxytocin, that can offset the impacts of mechanisms on disease. However, these proposed linkages are speculative as they are based on animal models yet to be thoroughly examined in humans.

If true, then (1) later support would not be as effective in attenuating the link connecting stressors to mechanisms ("a path" in Fig. 1); (2) later support should offset the link connecting mechanisms and diseases ("b path" in Fig. 1), manifested as a mechanism × support interaction on disease outcomes (Fig. 2b); and (3) as a result, later support should attenuate the link connecting stressors and diseases ("c path" in Fig. 1), manifested as a stressor × support interaction on disease outcomes (Fig. 2c).

There is preliminary support for the first postulation that later support would not buffer the stressor-mechanism link. Young adults (mean age 22) who experienced childhood adversity (before the age of 16) exhibited exaggerated neural activity in frontolimbic regions related to emotional processing, learning, and memory. Later friendship (age 22) was examined as a buffering factor, but protection was largely not observed such that young adults with high vs. low friendship quality similarly exhibited the altered exaggerated neural reactivity (König et al., 2023). These findings are consistent with the proposition that early adversity sets into motion intermediary mechanisms such that later support may no longer prevent their development.

No studies have examined the second postulation that later support would attenuate the mechanism-disease link (intermediary mechanism × later support interaction on disease). However, indirect evidence has emerged: institutionalization (vs. never institutionalized) was associated with exaggerated SNS reactivity at age 12, which in turn was associated with downstream psychosocial adjustments with peers at age 16. This mediation from early institutionalization to SNS reactivity to adjustment problems was apparent only among adolescents with low, but not high, friendship quality at age 12. Importantly, protection by friendship quality emerged because friendship attenuated the longitudinal link between SNS reactivity and adjustment problems, rather than the link between institutionalization (stressor) and SNS reactivity (Tang et al., 2022). Although psychosocial well-being rather than physical illnesses was assessed, findings are consistent with the idea that later support may offset the impacts of intermediary mechanisms on downstream outcomes rather than buffer stressor's impact on intermediary mechanisms.

With respect to the third postulation, childhood abuse was associated with increased mortality risk in adulthood; however, greater social support during mid-adulthood reduced the mortality risks associated with childhood abuse (Chiang et al., 2018), supporting the third postulation that supportive relationships decades after stressor has ended can still intervene the stressor-disease path.

In sum, preliminary evidence suggests that later support may not buffer the stressor-mechanism link but may counteract the mechanism-disease link, thereby attenuating the stressor-disease link. Note that although this hypothesis emphasizes a match between timing of support and path assessed for successful protection, such matching likely does not occur in a categorical sense, but along a continuous spectrum as depicted in Fig. 3.

7. Discussion and future directions

This working hypothesis gives rise to testable predictions about the mechanistic processes underlying protection when support occurs during, before, or after a stressor. It also provides future directions. First, studies are necessary to empirically test the tenets summarized in Table 1 because presented evidence emanate from stitching together disparate literature that do not directly examine support's timing. As a result, these studies did not assess support at the unit of developmental stage as theorized; rather, measurements of support typically referred to an undefined timeframe (Cohen and Hoberman, 1983) or shorter timeframes (e.g., a month; Gottlieb, 1978). If the support assessed was not reflective of the overall level of support for a given developmental stage, the conclusions drawn from these studies may be inaccurate. To directly test the proposed hypotheses, future research will need to utilize some combinations of retrospective assessments of stress and social support measured per developmental stage together with longitudinal studies that can prospectively track both stress and social support. For instance, it will be beneficial to develop assessments that measure support across different timeframes (e.g., separate ratings for childhood vs. adolescence), similar to existing stress assessments that are sensitive to exposure timing (e.g., Slavich and Shields, 2018; Wolfe and Kimerling, 1997). In addition, because testing the current hypothesis requires examining interaction effects, future research will need to recruit sufficiently large samples to ensure adequate statistical power for detecting the postulated attenuation patterns. Specifically, to detect a fully attenuated interaction (Cohen's $d = .17$ or equivalent $r = .08$) or partially attenuated interaction (Cohen's $d = .08$ or equivalent $r = .04$) using mixed models with .80 power, sample sizes of about 260 and 1400, respectively, are necessary (Sommet et al., 2023). Therefore, large longitudinally datasets that repeatedly assessed support across multiple developmental stages, like Add Health and the Dunedin Study, may be leveraged to create aggregated support ratings per stage to test the current hypothesis.

Second, in addition to testing mechanistic processes, as depicted in Fig. 3, the current hypothesis further postulates that concurrent and prior support may be equally effective at buffering the stressor-mechanism path because they both operate via reducing intermediary mechanisms. However, if intermediary mechanisms are established and declining plasticity over time means reversing altered physiology is not feasible, as time between stressor exposure and support increases (i.e., later support), support's protective value may decrease for the stressor-mechanism path and increase for the mechanism-disease path. Yet, most PNI research on social support relies on intermediary biomarkers as the dependent variables, leading to support's protective role being examined largely for the stressor-mechanism link (i.e., stressor × support interactions), but not for the mechanism-disease link (i.e., mechanism × support interactions). Although there is merit to intervening relatively upstream of the causal chain, future research that examines whether support can offset the impacts of already established mechanisms on disease outcome may reveal another window of opportunity and potential intervention targets for individuals who are already exhibiting altered physiology.

Third, future studies should examine whether the postulated protective effects of social support operate in the form of moderation as theorized, rather than mediation. For example, although it is hypothesized that support intervenes by reducing the stressor's *impacts*, some forms of support (e.g., interpersonal emotion regulation strategies like situation selection; Reeck et al., 2016) may directly reduce stressor frequency, and thereby promotes better health. Future research should examine both interaction and mediation scenarios within the same study

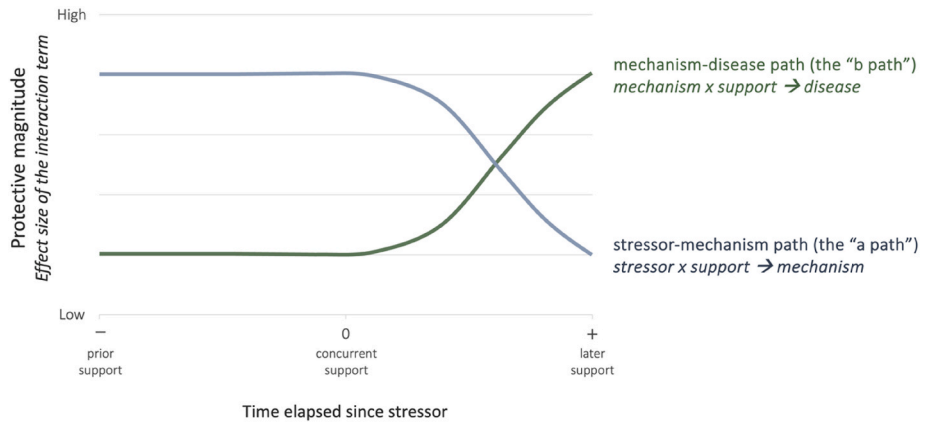

Fig. 3. The protective magnitude of support varies as a function of timing of support (prior support and concurrent support vs. later support) and the target path of interest (stressor-mechanism path vs. mechanism-disease path). Because both concurrent and prior support intervene by mitigating stress appraisal or responses, they are postulated to be equally effective at attenuating the path connecting the stressor to intermediary mechanisms (the "a path" in Fig. 1), manifested as stressor × support interactions on intermediary mechanisms (Fig. 1a). However, as time between stressor exposure and support increases, the path connecting stressors to mechanisms becomes more established while biological plasticity decreases. As such, the ability for support at a later developmental stage to prevent the development of the stressor-mechanism path diminishes. Instead, later support's protective value may increase in offsetting the path connecting intermediary mechanisms to diseases (the "b path" in Fig. 1), manifested as mechanism × support interactions on disease outcomes (Fig. 1b). Here, protective magnitude refers to the effect size of the corresponding interaction term.

to better isolate these pathways to health.

Fourth, this hypothesis could be extended to and tested with behavioral mechanisms. It is unclear whether health behaviors would operate in a similar fashion to biological processes. Whereas biological processes are increasingly difficult to alter as people age due to decreased plasticity (Lam et al., 2022), behaviors may be modifiable across the lifespan. However, although behaviors are theoretically modifiable, empirically, adolescent behaviors tend to persist into adulthood (Paavola et al., 2004). Moreover, intervention effects on modifying health behaviors (e.g., smoking cessation interventions) often do not persist (Prochaska et al., 2004), suggesting that while theoretically reversible, behaviors may be practically resistant to change. Thus, the application of the current hypothesis to health behaviors and subsequent disease outcomes would need to be tested in future research.

Fifth, after testing these basic postulations, future research may refine the hypothesis by examining its parameters. For example, with two exceptions (Suglia et al., 2009; Rogers et al., 2022), the reviewed studies largely assessed *perceived* relationship attributes. As differential associations of perceived vs. received support with health outcomes have been documented (Gleason et al., 2008; Uchino et al., 2011, 2018), it would be beneficial to examine whether one aspect of support is more sensitive to timing than the other. Other questions that test the hypothesis's parameters may include whether the kind of interpersonal processes that are most effective would vary by timing (e.g., protection by concurrent support may require stressor-specific support, whereas protection by prior and later support do not as they occur in the absence of stressor), whether the number of supportive others matters (e.g., having one vs. many close figures), and whether the current postulations are applicable only to certain type of stressors (e.g., acute vs. chronic).

Finally, there are boundaries to the proposed hypothesis. First, since timing is considered by developmental stage, the prior support scenario is not possible when adversity is exposed in childhood. Second, there is likely an upper limit as to how late support can emerge and still offset the impacts of altered physiology. As clinical diseases typically manifest in middle adulthood (Driver et al., 2008), support that arrives during older adulthood likely will not confer protection.

To conclude, this article aims to encourage PNI research on social support to consider factors underlying support's protective value, highlighting timing of support as one such factor. The current hypothesis has implications for future mechanistic research, which may inform interventions.

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Data availability

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