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Physical Effects of Daily Stressors Are Psychologically Mediated, Heterogeneous, and Bidirectional

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Prior research shows that daily stressors lead to greater psychological distress. A separate body of research links daily stressors to physical symptoms such as backaches and stomach problems. We integrate these literatures by positing an interconnected causal system, whereby stressors lead to psychological distress which, in turn, leads to physical symptoms. Our integrated approach also includes causal effects in the opposing directions: Psychological distress can increase physical symptoms and physical symptoms can increase psychological distress. Put simply, causal effects are bidirectional. This finding illuminates the concept of feedback loops, which have never been investigated in the stress literature until now. We find that reverberating feedback between stressors and distress equilibrates after just one and a half loops and that feedback between stressors and physical symptoms does not actually reach a full loop. Because of this, feedback loops have only minor consequences for physical symptoms by the end of the day. Finally, we discuss the aforementioned phenomena with between-person differences at the forefront, showing how some people are as much as four times as reactive as the average person, some people are not reactive at all, and other people are reactive in reverse directions (e.g., distress leads to fewer physical symptoms). We empirically support these claims using daily diary data from three separate studies that together represent diverse ages, geographic regions, relationship statuses, and racial identities. Once established, we consider the implications of our integrated causal feedback system in relation to existing knowledge and highlight critical areas for future study.

Keywords: daily stress, nonrecursive structural equation models, physical symptoms, psychological distress

Daily life is stressful. Someone could be racing to work to finish something for a deadline. . .and then get into a minor car accident. Or someone could be having a relaxing evening at home. . .and then get into an argument with their romantic partner. Every person in every walk of life experiences stressors like these (Almeida et al., 2020). Unfortunately, stressors have long-term implications for people's psychological and physical health. Psychologically, stressors can lead to clinical levels of depression and anxiety, whereas physically they can lead to cardiovascular disease, infection, and cancer (Cohen et al., 2007). It is no surprise that stressors are considered a major risk factor for morbidity and mortality (Epel et al., 2018).

But what about the short-terms effects of daily stressors? How do stressors impact psychological and physical well-being in people's everyday lives? If we think about stressful events in the context of particular people in their particular lives, we begin to see how complicated, nuanced, and meaningful the answer to these questions could be. Consider the characters portrayed in Table 1.

Clearly, stressors have a profound impact on Jasmine and James' daily psychological and physical health. Even so, the scientific literature on the psychological and physical effects of daily stressors does not capture the complexity of these processes because it remains siloed. Whereas one study might capture the fact that daily stressors worsen Jasmine's mood, another study might show how James' distress worsens his physical state on a given day. Because of this, we do not know whether daily stressors lead to physical symptoms because of mood. Without knowing whether mood operates as a mediator in the link between stressors and physical symptoms, we do not know whether interventions like mindfulness (e.g., Chiesa & Serretti, 2009), reappraisal (e.g., Lewis et al., 2018), or social support (e.g., Bolger et al., 2000) could help alleviate stress-

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The authors have uploaded all data, code, and model objects to the following OSF repository: https://osf.io/86xmu/.

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Table 1
Character Illustrations of the Effects of Daily Stressors

Jasmine	James
Jasmine works a financially stable job, is in a secure romantic relationship, and is highly in-tune with her emotional and physical self. Stressors generally lead Jasmine to be in worse moods, and her bad mood can lead her to experience physical symptoms, in her case often headaches. For Jasmine, stressors lead to headaches *because of* her distress; when she doesn't get upset by stressors, the stressors tend not to give her headaches. However, when Jasmine does get upset, the effects of stressors can escalate. When in a stressor-induced bad mood, Jasmine is likely to initiate more stressors for herself; she might get into an argument with her boyfriend, take on too much work, or poorly plan her commute so that she ends up in traffic. These increased stressors further exacerbate Jasmine's psychological distress, so that she not only has a headache by the end of the day, but also tension in her muscles.	James is in a supportive romantic relationship but has an unstable financial situation and therefore frequently experiences stressors. However, James is not so mood-reactive to these daily stressors. And for him, a bad mood actually has a reversed effect on his physical state—James' negative mood often leads him to overlook or ignore his physical symptoms. Interestingly though, when James' mood is disrupted for another reason, such as when he perseverates over an embarrassing social interaction, he tends to focus on everything that bothers him in his home. He cleans a lot, organizes things, and initiates repairs when in those bad moods. Put simply, James' nonstressor related bad moods lead him to cause more stressors for himself. He is then even less likely to experience physical symptoms because he gets so caught up in his disrupted mood states and consequent stressors.

induced physical problems in the context of people's everyday lives.

Our examples also showed bidirectional causal effects between stressors and their outcomes, for example how stressors not only altered Jasmine's mood but also how her negative mood led to more stressors. By realizing that causal effects between stressors and their outcomes can be bidirectional, our example illuminated the concept of feedback loops, which we saw when James' negative mood led to more stressors which led to an even worse mood. Although some researchers have pondered about feedback effects theoretically, the fact that feedback loops haven't been studied empirically means that we do not actually know anything about how feedback loops work. If feedback loops occur, how and when do people return to their baseline states? Which type of feedback loop matters more, that between stressors and mood or that between mood and physical symptoms? And what is the additive effect of these feedback loops on physical symptoms by the end of the day?

Finally, the effects of daily stressors differed between Jasmine and James. Although between-person differences are a major feature of the stress literature, prior work has only focused on explaining the types of people who are more and less reactive; for example, that older people may be more reactive than younger people (Mroczek & Almeida, 2004). But what about the magnitude of those differences between people? Are highly reactive people twice as reactive as the average person? Four times so? Knowing this would help determine whether stress-reduction interventions should address the population in general, or whether interventionists should use their resources to target the highly reactive people most in need. Perhaps more importantly, no one has considered the range of between-person differences. If the range from least- to most-reactive person includes a few people who actually show no reactivity, then we should consider whether lack of reactivity is itself a maladaptive outcome. If the range includes reversed reactivity, like how James' worse mood led him to report fewer physical symptoms, then new theory would be needed to explain those reversed effects. Analyzing the magnitude and range of between-person differences in stress reactivity can therefore provide a helpful roadmap for theory, detailing how much people differ from one another and whether and how many people show null and reversed effects.

In this article, we synthesize existing work by building an integrative theoretical and empirical framework that resolves these prior discrepancies and generates new ideas. Throughout the paper, we ask and answer the following four questions using data collected over the course of three decades that together represent a large portion of the U.S. population:

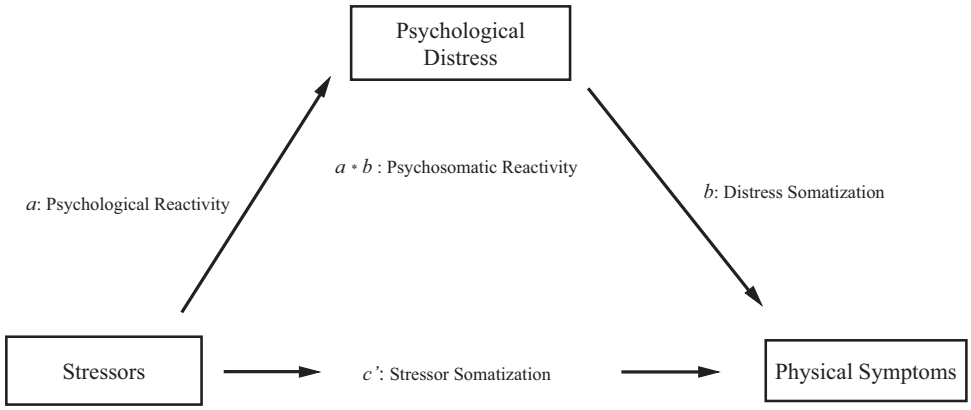
1. For the average person, do daily stressors lead to daily physical symptoms because of the negative moods they induce?
2. Are causal effects of daily stressors bidirectional for the average person?
3. If they are, then what is the magnitude and nature of feedback loops for the average person?
4. How much do these effects differ between people and what is the range of those differences?

In addition to answering these questions through taking a novel perspective on existing literature, we also implement an underutilized statistical approach that enables us to answer them empirically. Like all statistical models, we present one that rests on assumptions and has potential limitations. But because the availability (or lack thereof) of statistical tools constrains the questions that can be asked (Sharpe, 2013), we envision this model as one that will stimulate new questions that would otherwise not be considered.

For the Average Person, Do Daily Stressors Lead to Daily Physical Symptoms Because of the Negative Moods they Induce?

Stress research burgeoned in the midst of the cognitive revolution in psychology (Hobfoll, 2004), which led to the key insight that psychological factors explain why stressors lead to poorer physical health: Stressors disrupt psychological states, which in turn disrupt physical health (Cohen et al., 2016). In the daily stress literature, this mediational hypothesis has been tested at the between-person level (for one exception, see Ong et al., 2013 who investigate the within-person process from stressors to positive

Figure 1
Mediational Daily Stress Framework



affect to sleep). Chronically mood-reactive people exhibit lower levels of heart rate variability (Sin et al., 2016), show increased levels of biomarkers for inflammation (Sin et al., 2015), are at increased risk for chronic physical health conditions (Piazza et al., 2013), and even die younger (Mroczek et al., 2015). Together, this research supports the intuition that daily stressors impact health for people who are consistently more mood-reactive to daily stressors relative to people who are less reactive.

But what about the within-person mediation process as depicted in Figure 1? Regardless of people's chronic levels of reactivity, do the negative moods induced by stressors alter their sense of physical well-being in daily life? Physical symptoms are the most commonly experienced form of illness in daily life (Verbrugge et al., 1999). They motivate people to seek care from physicians (Stone, 2000) and ultimately predict chronic conditions and functional impairment decades later (Leger et al., 2015). It is therefore important to establish whether interventions that target negative mood, such as mindfulness (Chiesa & Serretti, 2009), have the potential to alleviate the physical manifestations of stressors in people's everyday lives.

Before delving further into the literature, we'd like to point out that the existing literature on daily negative emotions and mood has used the term psychological distress to describe these constructs (see Almeida, 2005 for a review). From this point forward, we use that terminology to be consistent with prior literature.

Despite the abundant between-person literature on distress mediation (Cohen et al., 2016), and the fact that distress mediation mirrors our intuitive experiences as people in everyday life, existing research on the daily psychological and physical effects of stressors has not taken a mediational approach. As seen in Figure 2, prior work has separately analyzed the affective and physical effects of the same daily stressors (Almeida, 2005). Because of this, the literature implicitly assumes that psychological reactivity and stressor somatization constitute separate direct effects. In concrete terms, if Jasmine had to wait an extra 20 minutes for her train at the end of the workday, the delay could give her a headache even if she was not bothered by the extra wait time. Perhaps the prior literature assumes this because researchers think that stressors impact physical symptoms via mechanisms below the level of emotional awareness. This is possible in light of the fact that there is no known medical explanation for most daily physical symptoms (De Gucht et al., 2004).

To test between these various possibilities, we apply statistical within-person mediation to daily diary data of stressors, psychological distress, and physical symptoms. Note that daily diary studies consist of participants providing evening summaries of events and feelings throughout the day. They trade off some level of experimental control (and causal inference) in exchange for high levels of ecological validity and real-world impact (Bolger et al., 2003). Analyzing this type of data will enable us to learn whether the direct effect between stressors and physical symptoms persists once distress mediation is accounted for at the within-person level of analysis.

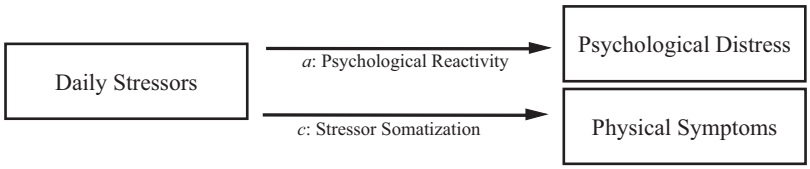
Are Causal Effects of Daily Stressors Bidirectional for the Average Person?

In addition to testing the mediation hypothesis, we will also acknowledge previously overlooked literature on causal effects in opposing directions, as depicted in Figure 3.

There has been some work suggesting that psychological distress can lead to more stressors. One possible mechanism is that negative mood can lead to biased recall of stressors. We discuss this possibility in depth in the Notes on Measurement section found in the Supplemental Materials (https://osf.io/86xmu/). For now, we briefly point out that daily diary studies assessing the number (rather than intensity) of stressors ask whether something happened or not, which has been shown to be unbiased in a one-day time frame (see Tennen et al., 2006). Therefore, the effect from distress to stressors in daily diary studies likely reflects selective exposure. Not only are people more likely to initiate interpersonal conflicts when in bad moods (Almeida, 2005; Neff, 2012), but they also tend to make poorer decisions overall. We saw this with Jasmine, who was riskier and could have used counterproductive heuristics when navigating her commute and financial decisions (Eeckhoudt et al., 2011; Gear et al., 2017; Hockey et al., 2000; Mitchell & Phillips, 2007).

Opposing causal effects might also occur in the relation between psychological distress and physical. People randomly assigned to receive S. *typhi* capsular polysaccharide, a vaccine that stimulates inflammation, report more distress than those injected with a saline control serum (Wright et al., 2005). Similarly, reducing physical

Figure 2
Existing Daily Stress Framework



symptoms such as pain can reduce psychological distress (Gebhardt et al., 2016). In daily diary studies, physical symptoms on one day correspond to changes in psychological states the next day (Charles & Almeida, 2006; Diefenbach et al., 1996) and the same day (Eckenrode, 1984; Larsen & Kasimatis, 1991). Physical symptoms seem to put people in worse moods.

Considered alongside the mediation framework, it could be that the relation between stressors and distress, and that between distress and physical symptoms, operate bidirectionally. Unfortunately, no prior work has unified these seemingly contradictory causal effects. As we will see in the next section, combining these literatures raises intriguing new questions.

What Is the Magnitude and Nature of Feedback Loops for the Average Person?

So far, we discussed how stressors in daily life impact people's daily well-being; experiencing stressors heightens distress which can initiate physical symptoms. If we incorporate the opposing paths, then feedback loops emerge.

In Figure 4, we see that stressors can increase psychological distress (a_1), and that those stress-induced negative moods can in turn initiate more stressors (a_2) which could lead to even more distress (a_1 again). Concretely, imagine that Jasmine got into an argument with a friend and that the conflict upset her. Because she was upset, she shouted at her romantic partner later in the day, which further exacerbated her distress. We also see in Figure 4 that feedback could occur between distress and physical symptoms. Stress-induced distress might increase physical symptoms (b_1) which, in turn, heighten psychological distress (b_2). Those heightened negative mood states could go on to create more physical symptoms (b_1). Concretely, imagine that James' headache irritated him so much that he also ended up with muscle tension by the end of the day.

Beyond not knowing whether feedback loops occur empirically, we also do not know how and when people equilibrate to their baseline states. How many times through a feedback loop does it take for those effects to die down? Moreover, we do not know which kind of feedback, that between stressors and psychological distress or that between psychological distress and physical symptoms, matters more. And what are the additive effects of feedback on physical symptoms by the end of the day? Because the field lacks adequate tools to answer these questions, the concept of feedback loops has only been discussed as a theoretical possibility (see Almeida, 2005), one without any specification about how they would actually work.

If we consider the methodologies available to the field at this point, the most obvious tool to address feedback loops is ecological momentary assessment (EMA). Yet the problem with this approach is that existing theory has yet to specify the time lags of the causal processes, as well as whether and how much those time lags would vary from one person to the next. In this case, a short time lag seems optimal because it would allow us to test associations over short (e.g., between time t and time $t + 1$) as well as longer (e.g., between time t and time $t + 2$) time frames. A short time lag would also mirror our intuition that the time it takes for a stressor to elicit a bad mood, or for a bad mood to bring about a headache, would be relatively short. However, in our view, pinging participants so frequently would disrupt the causal processes that we are interested in studying. Constant measurement would shape the way people experience their days and would also likely bother participants so much that their negative moods could be heightened. This could lead participants to ignore some surveys. Moreover, because constant pinging would be expensive, generalizability would almost certainly be limited by necessitating a small and therefore nonrepresentative sample.

We believe that a statistical approach for causal estimation of all the paths in Figure 4 is therefore optimal (see Kline, 2013). If we contemporaneously measure stressors, distress, and physical symptoms once the effects have died down, then we can use nonrecursive structural equation models (SEM) to estimate feedback loops that occurred earlier in the day. Conceptually, the way nonrecursive SEMs work is that stressors, distress, and physical symptoms are measured once the feedback system has equilibrated. The analyst then uses path tracing rules to investigate any sequence of causal effects they are interested in (Kenny, 1979). For example,

Figure 3
Opposing Causal Paths

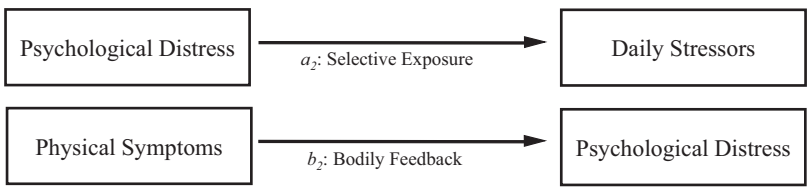
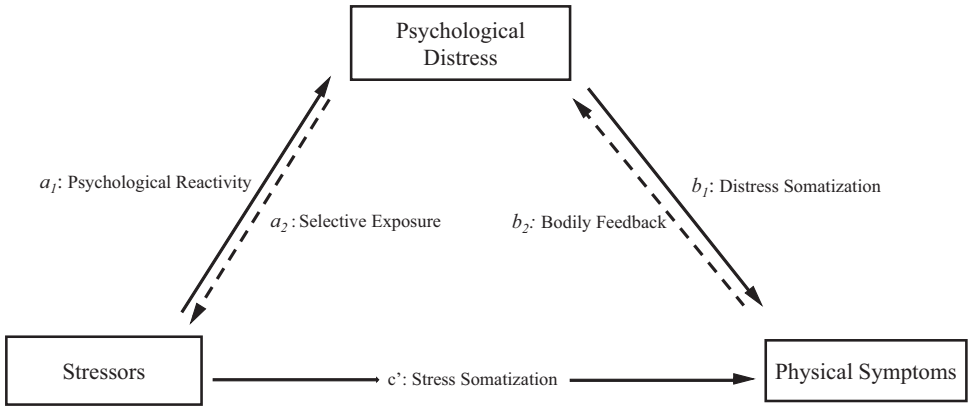


Figure 4
Bidirectional Causal Effects of Stressors, Psychological Distress, and Physical Symptoms



we can see how much stressors lead to psychological distress by looking at the estimate for the a_1 path. To see one full loop effect, that is, whether stressor-induced moods lead to even more stressors, we simply multiply a_1 by a_2. To see whether those effects lead to more distress, we multiply $a_1 \times a_2 \times a_1$. To understand feedback effects, we have to determine how many times through the loop it takes for the effect to diminish to zero. At that point, equilibrium has been reached.

An additional benefit of nonrecursive SEM is that they represent fully causal models. Because the reverse effects are accounted for, the mediation path from stressors to distress to physical symptoms represents the true forward-feeding causal process, assuming the model assumptions are met (Pearl, 2010). Because of their ability to address interesting questions and make causal inferences, these models are used in econometrics (e.g., van Giesen & Pieters, 2019), biology (e.g., Kokkonen et al., 2019), genetics (e.g., Saborío-Montero et al., 2020), and sociology (e.g., Hornstra et al., 2020; Li & Fang, 2019). Our empirical analyses will show how the stress literature can also benefit from their use.

In this article, we are especially interested in phenomena that start with stressors and end with physical symptoms. We want to know whether feedback loops initiated by stressors have downstream consequences for physical symptoms. For example, whether spiraling between stressors and distress throughout the day further increases physical symptoms, beyond the effect from stressors to physical symptoms via distress. Knowing whether this happens matters because the findings would imply a new type of intervention for stress reactivity: that people need to be self-aware of their stressor-induced moods, as well as their mood-induced physical symptoms, so as not to initiate even more stressors and even worse moods that land them with more physical problems by the end of the day.

How Much Do the Effects of Stressors Differ Between People, and What Are the Ranges of Those Differences?

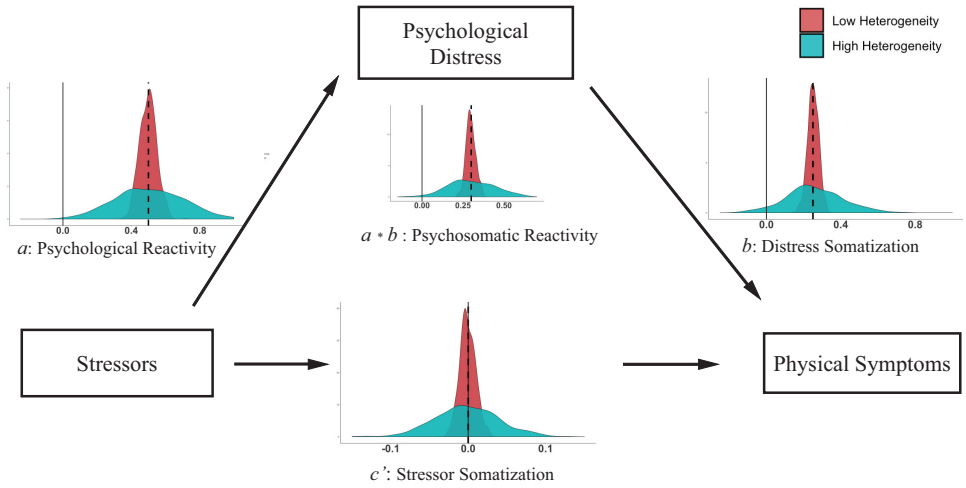
In line with every article we have cited so far, our discussion has focused on how stressors influence distress and physical symptoms on average. But a fundamental tenet of stress theory is that stress reactivity depends on the person (e.g., Boyce & Ellis, 2005; Cacioppo, 1998; Lazarus, 1993). For example, older people (Mroczek & Almeida, 2004), people with higher levels of neuroticism (Bolger & Zuckerman, 1995), and those with nonsupportive social relationships (Uchino, 2006) are most reactive. Daily diary data are unique in that they enable researchers to estimate these between-person differences via multilevel models that give a slope for each individual person. This fact has previously been leveraged to show the percent of between-person differences accounted for by a certain variable, such as sense of purpose (Hill et al., 2018).

Our goal is to build on this prior work by quantifying the magnitude and range of those between-person differences in relation to the average person. We do not investigate specific moderators because we do not aim to build a theory around the most important one. Instead, we build a metatheory by spending time analyzing the distributions of each effect. We not only empirically test for "meaningful heterogeneity" using newly established criteria (Bolger et al., 2019) but also analyze how much more reactive some people are compared with the average person, as well as whether some people show reversed effects relative to the average person. Since reversed effects imply that the distribution has crossed zero, we also reveal whether some people are not reactive at all.

To illustrate this point, Figure 5 depicts distributions of effects for each path. The x axes represent the standardized slopes between the predictors and the outcomes, whereas the y axes represent the relative proportion of people with each slope; that is, these are density curves. If we first look at the psychological reactivity path, we arbitrarily assigned a slope of .4 to the average person. We also depict two heterogeneous distributions that differ in their magnitude and meaning. In the red distribution, there are between-person differences in psychological reactivity, but those differences are small and the slopes are positive for every person. The blue distribution is not only heterogeneous, but even more so. In this case, some people are more than twice as reactive as the average person. We also see that a few people exhibit slopes of 0; their moods are not disrupted by stressors in daily life. Both distributions would align with existing findings—psychological reactivity is moderated—but the meanings are quite different.

If we turn to the distress somatization path in Figure 5, we see something else. Here, the average person arbitrarily has a slope of

Figure 5
Distributions of Causal Effects



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.2. The red distribution, although heterogeneous, implies that everyone experiences at least a little bit of distress somatization. Alternatively, the blue distribution has other implications beyond the fact that between-person differences exist. Here, some people are far more reactive than average, and other people have reversed effects. For 20% of people in this sample, like James, distress leads to *fewer* symptoms.

Understanding the magnitude and range between-person differences is essential for stress theory to progress. We need to know whether the effects we know so well, for example, that stressors put people in worse moods, are universal effects. Are the slope positive for everyone? If it is not, then we need knew theory to explain how that could be. Without doing this, stress theory remains stifled by assumptions that may not represent the true causal process for every person in every walk of life (Haaf & Rouder, 2019).

Bring It All Together: An Integrated Causal Feedback System

We started by thinking about how daily stressors might impact Jasmine and James. In reviewing the existing literature, we arrived at four theory-driven hypotheses that capture Jasmine and James' daily experiences. These are depicted in Figure 6. Our first hypothesis was that stressors lead to physical symptoms because of the psychological distress they induce throughout the day, at least for the average person. Our second hypothesis was that causal effects between stressors and distress and between distress and physical symptoms operate in opposing directions. This meant that causal effects would be bidirectional. Bidirectionality led to our third hypothesis, which was that feedback loops can occur and that they impact physical symptoms by the end of the day. Finally, our fourth hypothesis was that some people are far more reactive than the average person, almost no one will be nonreactive, and that reversed effects are unlikely. Note that our model implies that physical symptoms cannot induce stressors unless those physical symptoms disrupt people's psychological states. Next, we test these hypotheses with data.

Overview of the Present Studies

We use data from three daily diary studies to empirically test our integrated causal feedback system. Fully understanding "life as it is lived" requires appreciating the contexts that embed study participants: the varied stressors they face given their life circumstances and particular sociocultural climate (Bolger et al., 2003). Study 1 includes a relatively small sample of highly educated, financially stable, and racially diverse couples who filled out night-time reports of stressors, distress, and physical symptoms for 35 nights. Study 2 includes a geographically, but not racially, diverse sample of individuals at varying life stages for a relatively short diary study that took place in the evening (7–8 days; MIDUS). Finally, Study 3 uses data from a relatively large, racially and socioeconomically diverse sample of cohabiting romantic partners who responded to nightly diaries for 42 consecutive nights.

The following studies contain secondary data analyses, which constitute both strengths and weaknesses. Interested readers, or those new to daily diary methods, may read a detailed summary of the measures, as well as their strengths and limitations, in the Supplemental Materials (https://osf.io/86xmu/) section *Notes on Measurement*.

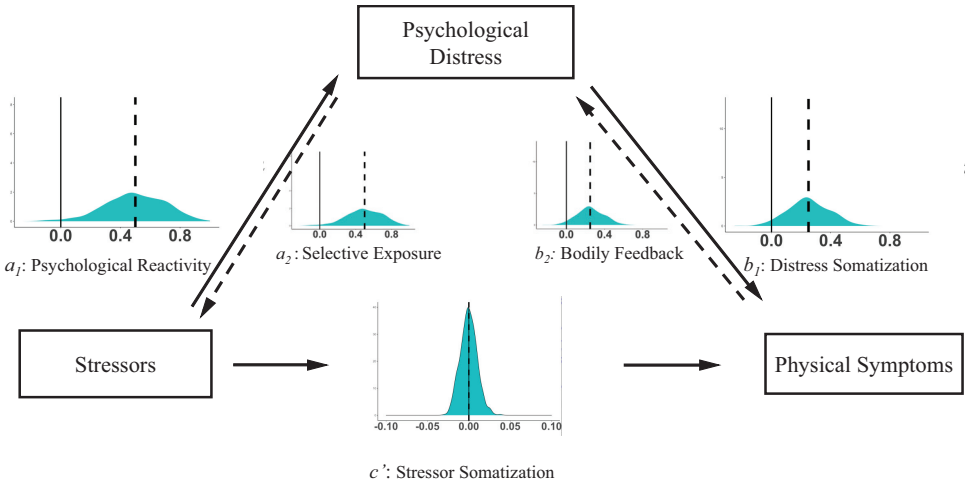
Data Analysis

All code, measures, and analyses can be found at the following OSF repository, which we hope aids future researchers in implementing nonrecursive SEMs: https://osf.io/86xmu/

Analytic Setup

All analyses were implemented with multilevel structural equation modeling with Bayesian estimation, as implemented in the brms package in the R statistical software (Bürkner, 2017). Bayesian approaches are increasingly popular in the psychological sciences (van de Schoot et al., 2017) and, when employed with

Figure 6
An Integrated Causal Feedback System for Daily Stressors



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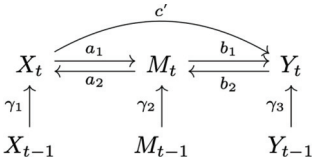
noninformative priors, give rise to similar estimates and uncertainty intervals as models using frequentist approaches. A benefit of Bayesian models is that they allow us to make probability statements about values of model parameters. This is not possible in frequentist approaches, which result in probability statements about likely data values in hypothetical exact replications of the research study (Gelman et al., 2013). Also, by emphasizing the entire probability distribution of parameters that are consistent with the sample data, Bayesian models align with rising concerns regarding the shortcomings of binary significance testing (Dienes, 2011; Wagenmakers, 2007).

Prior to conducting analyses, all variables were within-person standardized to allow ease of interpretation across persons and studies. This type of centering also prevents confounding of between- and within-person levels of analysis (Bolger & Laurenceau, 2013).

Statistical Model

Analyzing the paths of the model (see Figure 7) requires estimating a system of structural equations. For simplicity, we display below the path diagram for one person and note that the complete model allows each person to have their own slopes and mediated effects:



This diagram corresponds to the following system of structural equations, which we estimate for each person:

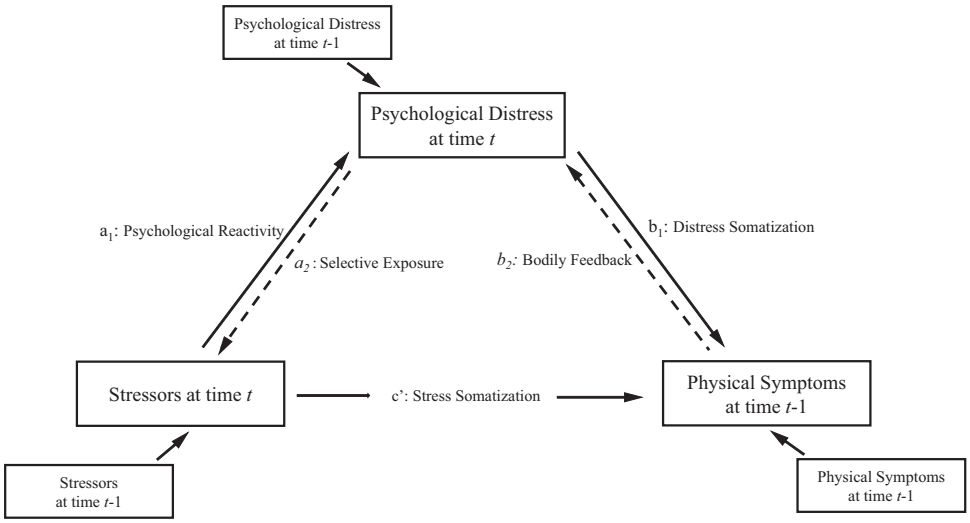
$$\begin{bmatrix} X_t \\ M_t \\ Y_t \end{bmatrix} = \begin{bmatrix} 0 & a_2 & 0 \\ a_1 & 0 & b_2 \\ c' & b_1 & 0 \end{bmatrix} \begin{bmatrix} X_t \\ M_t \\ Y_t \end{bmatrix} + \begin{bmatrix} \gamma_1 & 0 & 0 \\ 0 & \gamma_2 & 0 \\ 0 & 0 & \gamma_3 \end{bmatrix} \begin{bmatrix} X_{t-1} \\ M_{t-1} \\ Y_{t-1} \end{bmatrix} + \begin{bmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \varepsilon_3 \end{bmatrix}$$

This model gives rise to posterior distributions (distributions capturing the range of possible effect sizes for a given parameter) for: (a) population estimates for average (fixed) effects that represent the average person's level of a_1 (psychological reactivity), a_2 (selective exposure), b_1 (distress somatization), b_2 (bodily feedback), and c'(stressor somatization), (b) population estimates for the variance (random effect) of a_1, a_2, b_1, b_2, and c', (c) model-predicted estimates of the slopes for each person in the sample, which represents sample-level heterogeneity, and (d) covariances between the paths. In this model, a_1, b_1 *and* c' are assumed to be multivariate normal with covariance matrix T and ε_1 and ε_2 are bivariate normal with covariance matrix Σ (Bolger & Laurenceau, 2013).

In discussing the results, we focus on (a) population estimates for the average (fixed) effects and (b) model-predicted sample estimates for heterogeneity. We focus on population estimates of the average effects because these have been used to represent the experience of the hypothetical average person (Bolger et al., 2003). We focus on model-predicted sample estimates for heterogeneity because these represent the data of actual individuals.

Since the model does not directly output an estimate for the average person's mediated effects (i.e., psychosomatic reactivity), we compute them using the Monte Carlo simulation approach discussed in Bauer et al., (2006) as well as MacKinnon et al. (2004). In brief, we simulate the bivariate normal distribution of the a_1 and b_1 paths 1,000 times, each time drawing 50,000 samples and multiplying the paired estimates of a_1 and b_1 together. This gives rise to a distribution for the expectation of a_1 times b_1. The mean of this distribution estimates the expectation of a_1 times b_1 and is equivalent to what we would obtain using the formula for the expectation of a_1 times b_1 discussed in Kenny et al. (2003). This is psychosomatic reactivity for the average person. The standard deviation of this distribution estimates the standard error of the expectation of a_1 times b_1, that is, whether the effect for the average person is statistically reliable. To obtain estimates for the model-predicted sample estimates of psychosomatic reactivity, we multiply each individual's model-predicted value for a_1 by their model-predicted value for b_1.

Figure 7
Data Structure for Model Identification



We use the same Monte Carlo simulation approach to estimate the effects of the feedback loops. In terms of estimating the effects of the feedback loops on physical systems, we focus on the two that we think could be meaningful: 1) feedback between stressors and distress, $a_1 * a_2 * a_1 * b_1$, and 2) feedback between distress and physical symptoms, $a_1 * b_1 * b_2 * b_1$. We obtain these estimates for the average person using the same method as that used for the indirect effect—with Monte Carlo simulation. For example, we estimate the effect of the first feedback loop (cycling through the stressor-distress loop once) by simulating the trivariate normal distribution of the a_1, a_2, and b_1 paths 1,000 times, each time drawing 50,000 samples and multiplying the estimates of a_1 by a_2, by a_1 by b_1. This gives rise to a distribution for the expectation of these paths multiplied together, and the mean of this distribution estimates the expectation of the feedback loop. The standard deviation of this distribution is an estimator of the standard error of the indirect effect for the average person. Once estimated, we can add these feedback effects to the original, mediated effect to derive a more complete estimate of the effect of stressor on symptoms, one that includes feedback loops. To obtain model-predicted sample estimates for the feedback loops (i.e., sample-level heterogeneity), we multiply each individual's model-predicted value for each of the paths. We first test one loop through-stressor-distress path and one loop though the distress-physical symptom path. If they are large, we continue adding paths until the additional effects are so small as to be meaningless. This allows us to determine how many loops it takes through the system to equilibrate.

Caveats About the Model

Although powerful once estimated, nonrecursive SEMs require data with three specific qualities. The first quality is that the assumptions of the model are met. In addition to the typical assumptions for SEM, nonrecursive SEM has two additional assumptions. The first is equilibrium, which means that changes in the system that underlie feedback have already manifested (Heise, 1975; Kenny, 1979). In our case, we need to assume that any feedback effects have died out by the end of the day. This is reasonable given that people go to sleep soon after filling out the diary. The second additional assumption is stationarity, which means that the underlying causal structure does not change over time. In our case we have no reason to believe that over the time period of a diary study, which is a snippet of a person's life, one path exists for the beginning of the study and not the end. There is no way to test these assumptions beyond theory and good judgment (Kline, 2013), and we think it reasonable to assume them in the context of daily stressors and their daily effects. To the extent that these assumptions are met, the causal effects of interest are estimated without bias (Pearl, 2010).

The second quality of nonrecursive SEMs is that instrumental variables must exist to enable model identification. Identification refers to having the right kind of data to estimate each path. In the case of nonrecursive SEM, the model is only identified when each predictor has its own instrumental variable, one that *uniquely* predicts each construct in Figure 6. For example, there needs to be a variable that predicts stressors but does not predict psychological distress. This may seem difficult, until we realize the nature of daily diary data. With many repeated measurements of the same constructs, have the opportunity to use time-lagged effects, as shown in Figure 7. We know from prior research that time-lagged effects of the same construct are generally related, while time-lagged effects between constructs are not. Concretely, we leverage the fact that psychological distress at time $t - 1$ predicts psychological distress at time t, and that psychological distress at time $t - 1$ does *not* predict stressors at time t. Similarly, stressors at time $t - 1$ predict stressors at time t whereas stressors at time $t - 1$ do *not* predict psychological distress at time t, and so on. There must also be no third variable that causes stressors and psychological distress at time $t - 1$ and time t other than the ones we have specified in the model. We can assume this on the basis of a putative direct relation, for example, between how someone feels today and how they feel tomorrow. Instrumental variables have been shown to lead to optimal estimation in nonrecursive models when those instruments are strong and reliable (Wong & Law, 1999).

Table 2
Models for Studies 1–3

Study	Paths	Statistical tests	Inferences made
1	a_1, b_1, c'	Mediation	Daily stressors lead to psychological distress, which in turn elicits somatic symptoms
		Heterogeneity	People differ from one another in the magnitude of each path and the mediated effect
2	a_1, b_1, c'	Mediation	Daily stressors lead to psychological distress, which in turn elicits somatic symptoms
		Heterogeneity	People differ from one another in the magnitude of each path and the mediated effect
3	a_1, a_2, b_1, b_2, c'	Mediation	Daily stressors lead to psychological distress, which in turn elicits somatic symptoms
		Heterogeneity	People differ from one another in the magnitude of each path and the mediated effect
		Bidirectional Causality	Psychological distress leads to stressors and somatic symptoms lead to psychological distress
		Feedback	Effects can reverberate; experiencing more distress leads to more physical symptoms, which leads back to distress

The final quality of these models is that they must be computationally feasible to estimate. This has only recently become possible with user-friendly software for Bayesian estimation. Bayesian estimation is better than frequentist estimation for complex models because of their algorithmic approach to generating posterior distributions rather than estimating maximum likelihood parameters (Gelman & Hill, 2006). In short, the distributional approach to parameter estimation enables complex multilevel models to converge. Because we implement noninformative priors, the parameter estimates are the same as they would be in frequentist versions of the models.

Because of these criteria, we ran two preliminary models for each study, the first of which examined cross-lagged effects between stressors and psychological distress and the second of which examined cross-lagged effects between psychological distress and physical symptoms. In assessing the strength of these relations, and lack thereof, we found that the nonrecursive model was not identified in Studies 1–2 (see the Supplemental Materials for these results: https://osf.io/86xmu/). Therefore, we impose a constraint on the models in Studies 1 and 2 that a_2 and b_2 be set to 0. This means that only our first and third hypotheses are tested in Studies 1 and 2. Because those studies do not include the opposing causal paths, it is possible that the estimates of a_1 and a_2 in those studies will be biased upward. Whether upward biases are meaningful in the first two studies can be assessed when the results of all three studies are combined and the consistency across studies is evaluated. Table 2 summarizes which hypotheses are tested in which studies.

A Conceptual Overview of the Results Section

Before delving into the results, we would like to emphasize a few things. First, we report effects for the average person. In technical terms, these are the fixed effects. Second, we report whether this estimate for the average person is statistically reliable. In technical terms, we do this by reporting the 95% *credibility* intervals for the fixed effects, which help us make claims about statistical reliability. Finally, we report the 95% *heterogeneity* interval, which represents the distribution of model-predicted effects for each person in the sample. We report the person who is at the 2.5th and 97.5th percentiles of the heterogeneity distribution as representatives of low- and high-reactors. These estimates are not about statistical significance but are instead about between-person differences. Finally, we report the percent of the sample whose model predictions are in the same direction as hypothesized in the Introduction.

Because we are interested in the heterogeneity interval, we use guidelines set forth by Bolger et al. (2019) to test whether heterogeneity is significant and meaningful for each of the paths. These criteria include: (a) whether the standard deviation of the effect is at least 25% of the average effect for the "typical" person, (b) whether the credibility interval for the standard deviation of the average effect excludes zero, and (c) whether model-fit is improved by including heterogeneity in the model. These analyses can be found in the Supplemental Materials (https://osf.io/86xmu/).

Finally, we would like to point out that we take the *segmentation* approach to mediated effects (Memon et al., 2018). The segmentation approach tests each path in a causal chain. For example, the mediation part of the model refers to stressors leading to psychological distress which leads to physical symptoms. We therefore estimate the reliability of the *a* path (psychological reactivity), the *b* path (distress somatization), and the product of these two paths (psychosomatic reactivity). Although some researchers use the *transmittal* approach, in which only the reliability of the indirect effect (i.e., psychosomatic reactivity) is tested, the segmentation approach is considered superior in that it encourages consideration of the full causal chain of effects.

Ethics Approval

For the three studies, all procedures accorded with Institutional Review Boards at Columbia University, the University of Wisconsin-Madison, and the University of Michigan, for Studies 1 (SASSY), 2 (MIDUS), and 3 (Conflict), respectively.

Study 1

As an initial test of hypotheses 1 and 3, we use data collected from racially diverse individuals living in a highly urban area with financial stability and functional interpersonal relationships.[1]

[1] The other published paper using this dataset (Stadler et al., 2012) investigated the relation between interpersonal intimacy among romantic couples and somatic symptoms in daily life.

Table 3
Model Results for Study 1

	Average person [95% Credibility interval]	95% Heterogeneity interval	People with slopes > 0
Psychological stress reactivity (a_1)	**0.20** [0.16, 0.25]	[−0.08, 0.48]	92%
Distress somatization (b_1)	**0.13** [0.09, 0.16]	[−0.03, 0.30]	94%
Psychosomatic reactivity (a_1* b_1)	**0.03** [0.034, 0.035]	[−0.01, 0.12]	92%
Stressor somatization (c')	0.02 [−0.02, 0.06]	[−0.20, 0.27]	53%

Note. Bolded effects are those whose intervals do not cross zero.

Individuals in this study represent the experience of those living in a densely populated metropolitan area—New York City—which poses a unique set of daily stressors and norms governing responses to stressful events. Living in a metropolitan area like New York City introduces, on average, greater job stress intensity and frequency than rural areas (Gellis et al., 2004), which alters stress reactivity at the neurological level (Lederbogen et al., 2011). At the same time, this heightened stress environment may be buffered by the fact that participants in this sample were financially stable and in highly committed relationships (McEwen & Gianaros, 2010; Ozbay et al., 2007; Uchino, 2006). We encourage readers to consider the ensuing results in light of the peculiarities of this sample while also acknowledging that the sample reflects the lived experience of a portion of the U.S. population.

Method

Participants and Procedures

The data for this study were collected as part of a larger project investigating romantic couples' daily stress experiences over 35 consecutive days (Stadler et al., 2012). To participate in the study, both partners had to be native English speakers, over 18 years old, in a committed heterosexual relationship, and with access to high-speed Internet and a working e-mail address to fill out the daily diary online. To ensure retention, participants were paid up to $145 per person, including a $35 bonus for completing at least five morning and five evening entries per week. We only analyze data for evening responses. Each partner received a separate daily e-mail asking them to fill out an online diary every evening within 1 hour of going to bed. Participants were asked to complete the diary entries separately and not to share or discuss their answers with their partners.

Initially, data were collected from 164 participants who lived in the New York metropolitan area between June 2006 and February 2009; data from three people were excluded from analyses because they filled out fewer than six diary entries. All remaining participants (N = 161) completed at least nine diary entries totaling 4624 diary days (on average, 28 days of the 35-day period; range = 9–35 diary days). Participants were generally young (M age = 31 years, SD = 9) and slightly less than half (43%) were married. The ethnic makeup of the sample was representative of the U.S. population; 55% were White, 17% were African American, 14% were Hispanic, 12% were Asian, and 2% identified in an "other" category.

Measures

Daily Stressors. Each evening, participants were presented with a list of 15 "troublesome things" that could have occurred in the previous 24 hours, including a financial problem, too much work or school, receiving negative feedback, interpersonal conflict, and other stressors (see Supplemental Materials: https://osf.io/86xmu/). These items were generated in a pilot study that identified the most common stressful events experienced in daily life (for further details, see Bolger, DeLongis, Kessler, Schilling, et al., 1989). Participants in this sample reported, on average, 1.3 stressors per day over the course of the diary (between-person standard deviation = .79, range = .1–4.7).

Daily Psychological Distress. Psychological distress was assessed with an eight-item measure generated from the Profile of Mood States (McNair et al., 1981). Participants reported on a 0–4 scale, from 0 = *not at all* to 4 = *extremely*, how much they felt each of these 8 moods in the moment that they filled out the diary. On average, participants reported a mean distress level of 1.7 over the course of the diary (between-person standard deviation = .39, range = 1.09–2.86).

Daily Physical Symptoms. Physical symptoms were measured each evening with a shortened version of the symptoms checklist of Larsen and Kasimatis (1991) and an additional insomnia item. The checklist of symptoms contained six items including back ache, muscle tension, and poor sleep. The symptoms variable indicates the number of symptoms a participant reported in the previous 24 hours, ranging from 0 (no symptoms) to 6 (all symptoms). Participants in this sample reported, on average, .47 symptoms per day over the course of the diary (between-person standard deviation = .51, range = .00–3.33).

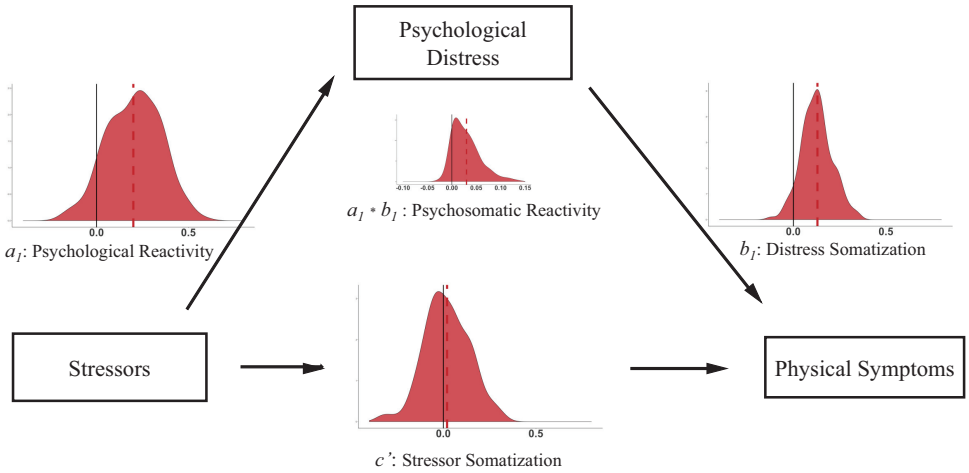
Results

Average Effects

All results can be seen in Table 3.[2] We first discuss effects for the average person. In terms of psychological mediation, the average person exhibited psychological reactivity (a_1 = .20 [.16, .25]), such that days with one standard deviation more stressors than the person's own average were associated with psychological distress that was 20% of a standard deviation above the person's own average. Similarly, the average person exhibited distress somatization (b_1 = .13 [.09, .16]); days on which the average person experienced a level of distress one standard deviation above their own average were associated with about 10% of a standard deviation greater-than-average physical symptoms. Psychosomatic reactivity was also statistically reliable (indirect effect = .03 [.034, .035]), although

[2] We ran secondary models that included covariates: (a) couple-level means and (b) weekend dummy variables. The results remain unchanged when controlling for shared levels of stressors, distress, and physical symptoms within couples and when controlling for weekend versus weekday effects.

Figure 8
Study 1 Results



Note: Effects for the average person are the dashed line, and model-predicted effects for each person in the sample are the density. See the online article for the color version of this figure.

practical interpretation is not feasible. Interestingly, the average person did not display stressor somatization (c' = .02 [−.02, .06]), implying that stressors had no effect on symptoms beyond their effects through psychological distress for the average person.

Heterogeneity of Effects

Although these effects emerged for the average person, there was also considerable between-person heterogeneity in each of the paths, as seen in Figure 8. The results from testing the criteria in Bolger et al. (2019) can be found in the Supplemental Materials (https://osf.io/86xmu/). Almost all criteria were met for all paths.[3]

In terms of psychological reactivity, 93% of the sample exhibited effects above 0, which means that for almost everyone, stressors led to more psychological distress. This is in line with our original hypothesis. Nonetheless, the person at the very low end of the distribution exhibited a slight reversal (2.5th percentile of a_1 = −.08), with more stressors actually reducing distress. The person at the high end of the distribution was more than twice as reactive as the average person (97.5th percentile of a_1 = .48).

There was also considerable heterogeneity in distress somatization, but again and as expected, an overwhelming majority of the sample (94%) exhibited effects above zero. The person at the low end of the distribution showed a slight reversal (2.5th percentile of b_1 = −.03) with more distress leading to fewer symptoms. All the while, the very reactive person was three times as reactive as the average person (97.5th percentile of b_1 = .28).

The estimate for the indirect effect, psychosomatic reactivity, was also heterogeneous, although notably 92% of the sample exhibited positive indirect effects. The person at the bottom of the distribution had a very slight reversal (2.5th percentile = −.01). At the other end of the distribution, the highly psychosomatic person, had an effect four times as large as the average person (97.5th percentile = .12).

Finally, we consider heterogeneity in the stressor somatization path. Its size is approximately half a scale point (.47), which is slightly smaller than the heterogeneity of the overall stressors-to-symptoms link prior to including distress as a mediator, models in which physical symptoms were the outcome and stressors were the predictors. This means that although we were successful in explaining the fixed effect of stressors on symptoms, we were unsuccessful in explaining the random effect. Within-person mediation, like conventional mediation, is often framed as a method to explain fixed effects (Bauer et al., 2006; Bolger & Laurenceau, 2013; Kenny et al., 2003); to our knowledge there has been no focus on the degree to which the heterogeneity in an effect is explained by heterogeneity in the indirect paths. Our results imply that other within-subject mediators would be required to do this, or that between-subjects predictors would be necessary. We return to this point in the General Discussion.

Discussion

Study 1 was a preliminary test of mediation and between-person differences. The results provide support for the postulated within-person mediation process such that daily stressors led to distress, which, in turn, led to physical symptoms for the average person. The estimate for psychosomatic reactivity (the indirect effect) was statistically reliable as well. Moreover, the direct effect between stressors and physical symptoms (stressor somatization) was essentially zero on average. Taken together, these results imply the indispensable role of distress in the relation between daily stressors and physical symptoms, at least for the average person.

At the same time, people varied considerably in the magnitude of each effect, with a few people actually exhibiting reversals. Of course, the majority (more than 90%) of people exhibited slopes in the expected directions. Nonetheless, these reversals raise intriguing questions about, for example, what kind of person experiences fewer physical symptoms when in a bad mood. In the other directions, the highly reactive person was at least twice as reactivate as the average person.

[3] The third criteria, improved model fit with the inclusion of the random effect, was not met for the distress somatization path.

Although Study 1 provided compelling preliminary support for the mediation and between-person difference hypotheses, it is not without limitations. The most obvious limitation is that the opposing causal paths were not accounted for, so the estimates here may be biased upward. We will see in Study 3 that this upward bias was trivial at most. Nonetheless, another limitation is that the sampling procedure in this study led to a very particular sample of adults: a convenience sample of participants who volunteered to come as a couple into a laboratory and then complete the daily diary study for compensation. The sample was therefore potentially shaped by selection bias. The restricted age range of the sample could also limit generalizability, as well as the fact that the sample itself was relatively small. Finally, this study had a potential measurement flaw; the evening diary asked about stressors and physical symptoms in the previous 24 hours while the distress measure referred to *right now*. It would seem at first glance that the mediator precedes the predictor as well as follows the outcome. However, in our view, distress at the end of the day and immediately before bed is naturally a summary measure. People spontaneously reflect on their day each evening (Newman & Nezlek, 2019; Szőllősi et al., 2015), remembering both good and bad things that happened (Connolly & Alloy, 2018; Kahneman et al., 2004). Because participants in this sample were couples, they likely discussed the course of their day with each other in the evenings (Hicks & Diamond, 2008). Therefore, how someone feels right now, before going to bed, likely approximates a day-end summary, at least more so than any other distress measure throughout the day. Nonetheless, we know that measuring distress in the moment before sleep is a less precise summary than explicitly asking for a summary and may therefore be biased. We are fortunate to have two other studies to compare results that will lend confidence to our interpretations in this study.

Study 2 therefore incorporated data from a much larger sample collected from participants who were geographically representative of the U.S. population, constituted a diverse age range, and measured stressors, distress, and physical symptoms as a summary.

Study 2

In a conceptual replication of Study 1, we used the second wave of the Midlife Development in the United States (MIDUS; Ryff et al., 2007) and the National Study of Daily Experiences (NSDE) project.[4] Individuals in this sample constitute a geographically representative sample of the U.S. population. The sample included participants from different walks of life, with considerable age, occupation, and health-status diversity. Thus, although we cannot paint one picture of participants' daily lives and the stressors they may face, we can say that the data reflect experiences of a wide range of adults in the United States. A list of recent findings from the MIDUS study can be found at their website: https://aging.wisc.edu/recent-midus-findings/.

Method

Participants and Procedures

MIDUS II (N = 4963) was a 10-year follow-up on MIDUS I (N = 7108) to examine adult's age-related changes in physical and mental well-being in the United States. After completion of MIDUS II, a subsample of participants was recruited for the NSDE II (N = 2022). The NSDE II was a daily diary study that occurred via phone over the course of eight consecutive evenings.

Initially, data were collected from 2,022 participants between 2004 and 2006; data from 149 people were excluded from analyses because they filled out fewer than six diary entries. All remaining participants (N = 1873) responded to at least six evening calls totaling 14,372 diary days (on average, 7.7 days of the 8-day period; range = 6–8 days). 342 additional participants were removed during the modeling phase due to having no variation in the variables of interest. The final sample therefore consisted of 1531 individuals. Participants were age-diverse (M = 56 years, SD = 12) and slightly under half (43%) were male. The ethnic makeup of the sample was not diverse: 86% were White/Caucasian, 3% were Hispanic, 10% were African American, .05% were Asian, and 2% identified in an "other" category.

Measures

Daily Stressors. Each evening, participants received a phone call and answered a series of questions about the stressful events they experienced that day. The items represent seven of the most common stressful things that happen on a daily basis, such as getting into an argument with someone or experiencing racial discrimination (see Supplemental Materials: https://osf.io/86xmu/). Information about how these items were generated can be found elsewhere (for further details, see Almeida et al., 2009). Participants in this sample reported, on average, .51 stressors per day (between-person SD = .43, range = .00–3.71).

Daily Psychological Distress. Psychological distress was assessed with a 20-item measure according to Item Response Theory that measures psychological distress (Kessler et al., 2002). Participants reported on a 0–4 scale from 0 = *none of the time* to 4 = *all of the time* how much they felt each of these 20 distress items throughout that day. On average, participants reported .19 on the distress scale (SD = .24, range = .00–2.19).

Daily Physical Symptoms. Physical symptoms were measured each evening with a 28-item adapted version of the symptoms checklist of Larsen and Kasimatis (1991). The checklist of symptoms contained items including back ache, muscle tension, and sore throat. The symptoms variable indicates the number of symptoms a participant reported throughout the course of the day, ranging from 0 (*no symptoms*) to 28 (*all symptoms*). Participants in this sample reported, on average, 1.8 symptoms per day (between-person SD = 1.18, range = .00–15.00).

Results

Average Effects

All results for this study are in Table 4.[5] As in the first study, the average person exhibited psychological reactivity (a_1 = .41

[4] Hundreds of articles have been written using the diary portion of the MIDUS study (see http://midus.wisc.edu/findings/index.php). None of this existing work considers psychological distress as mediating the relation between stressors and physical symptoms at the daily level nor does it analyze the range, magnitude, and subsequent meaning of between-person differences.

[5] As with Study 1, controlling for day of the week had almost no effect on the results. The parameter estimates for the model controlling for weekend versus weekday can be found in the Supplemental Materials at https://osf.io/86xmu/.

Table 4
Model Results for Study 2

	Average person [95% credibility interval]	95% heterogeneity interval	People with effects > 0
Psychological stress reactivity (a_1)	**0.41** [0.39, 0.43]	[0.17, 0.58]	100%
Distress somatization (b_1)	**0.18** [0.16, 0.21]	[−0.08, 0.42]	90%
Psychosomatic reactivity (a_1* b_1)	**0.07** [0.070, 0.076]	[−0.03, 0.18]	90%
Stressor somatization (c')	**0.08** [0.06, 0.10]	[−0.14, 0.31]	73%

Note. Bolded effects are those whose intervals do not cross zero.

[.39, .43]), such that days with one standard deviation more stressors than the person's own average were associated with psychological distress that was 40% of a standard deviation above the person's own average. Interestingly, the average person in this study was twice as reactive as the average person in Study 1. Also, as before, the average person exhibited distress somatization (b_1 = .18 [.16, .21]); days on which the average person experienced a level of distress one standard deviation above their own average they experienced 20% of a standard deviation greater-than-average physical symptoms. This estimate is also almost twice that of Study 1. Psychosomatic reactivity for the average person was again in the hypothesized direction (indirect effect = .07 [.073, .076]). Interestingly, and in contrast to Study 1, the average person exhibited stressor somatization (c' = .08 [.06, .10]), although the effect was small. This implies that stressors had almost no effect on symptoms beyond their effects through psychological distress for the average person.

Heterogeneity of Effects

As shown in Figure 9, there were considerable differences between people in the magnitude of these effects. In terms of psychological reactivity, 100% of the people in the sample exhibited effects larger than 0; there were no reversals (see *Caveats About the Model*). Nonetheless, the person at the very low end of the distribution showed an effect about half that of the average person, (2.5th percentile of a_1 = .17) whereas the very reactive person was 40% more reactive than the average person (97.5th percentile of a_1 = .58). Whereas an overwhelming majority exhibited these positive slopes in Study 1, the full sample did in Study 2. This implies that psychological reactivity could be a near-universal phenomenon among U.S. adults.

There was also considerable heterogeneity in distress somatization such that the person at the low end of the distribution showed a slight reversal (2.5th percentile of b_1 = −.08) and the person at the high end of the distribution showed an effect about twice that of the average person (97.5th percentile of b_1 = .42). This range is similar to that in Study 1, as a notable majority (90%) had effects in the expected direction. At the same time, these results lead us to wonder about the 10% of people who showed reverse effects, those for whom distress led to fewer symptom reports.

In terms of psychosomatic reactivity, there were a few people with small reversals (2.5th percentile = −.03) whereas the highly psychosomatic person had an effect more than twice the size of the typical person (97.5th percentile = .18). Similar to Study 1, 90% of the sample had indirect effects above 0.

Once again, between-person differences in the stressor somatization path is difficult to interpret (see Study 1 Discussion). We therefore rely on the distributions of effects for the other paths and note that distress mediation does not perfectly explain the relation between stressors and physical symptoms for every person in the sample.

Discussion

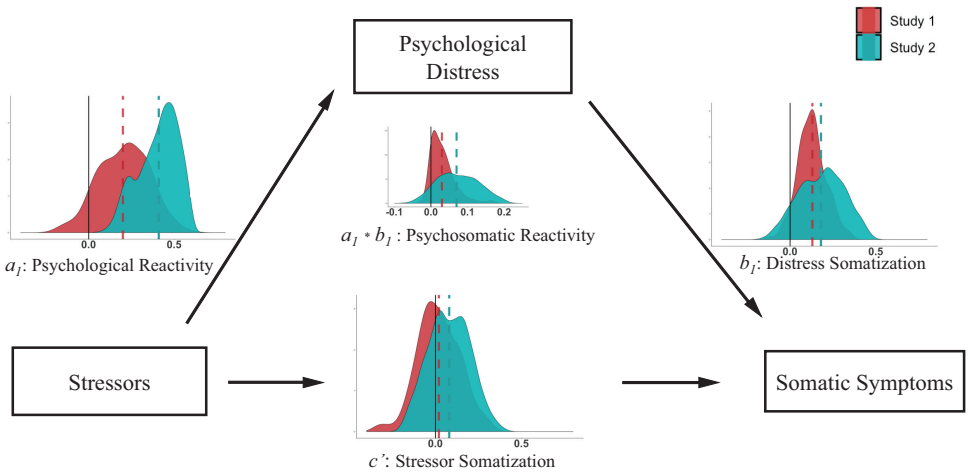
Again, the mediation hypothesis was supported by data. This time, the sample was considerably larger and more diverse in terms of relationship status, age, and geographic location. Moreover, all three questionnaire instructions accurately referred to the desired time period: today.

In this study, stressors were associated with psychological distress, which was, in turn, associated with physical symptoms for the average person. The estimate for the indirect effect, psychosomatic reactivity, also emerged reliably for the average person. In contrast to Study 1, the direct effect of stressors on physical symptoms (i.e., stressor somatization) was statistically nonzero for the average person, although its magnitude was relatively small. Taken together, these results imply the primary role of distress in the relation between daily stressors and physical symptoms, at least for the average person. In terms of the separate paths, while Study 1 found that 8% of people had reversals from stressors to psychological distress, 0% of the people in this sample did, perhaps because of the superior instructions in this study. Moreover, we learned that 10% of the people in this sample experienced reversals in distress somatization, with more distress leading to fewer physical symptoms. These findings are even larger than those in Study 1, where 6% of that sample showed reversals.

Although Study 2 certainly contributes to our confidence in the results from Study 1, it had its own limitations. In terms of sampling, the most notable limitation is that the sample was overwhelmingly (86%) white. Also, because Study 2 was an epidemiological study, there was a tradeoff between sample size and the number of repeated measures. Each person only provided eight evening reports of stressors, distress, and physical symptoms. The variance estimates are therefore less reliable. Finally, as with Study 1, we assumed that causal effects only operate in the directions depicted in Figure 9. A better approach would be to model bidirectional causal effects by estimating a nonrecursive SEM (Kline, 2013). In this way, we could assess the extent to which excluding the opposing causal paths biased the estimates here and in Study 1. However, in both data sets we analyzed so far, the nonrecursive SEM approach was not available to us because of the relatively small number of participants in Study 1 and the small number of days in Study 2 (see *Caveats About the Model*).

Study 3 addresses these limitations by using data from a sample approximately twice as large as that of Study 1 and with more than five times the number of repeated measurements as that of

Figure 9
Results for Studies 1 and 2



Note: Effects for the average person are the dashed lines, and model-predicted effects for each person in the samples are the densities. See the online article for the color version of this figure.

Study 2. This sample was also racially diverse when compared with Study 2. Thus, in Study 3, we can use a nonrecursive within-subject SEM to evaluate our assumed causal directions in Studies 1 and 2, as well as test our remaining two hypotheses.

Study 3

Individuals in this study lived in both urban and suburban areas of Detroit, MI, in the late 1980s.[6] Detroit at this time was characterized by economic hardship and political turmoil, and whether or not participants were directly involved in the industrial decline, violent crime and racial tension were high, implying that participants were exposed to a unique set of stressors and strains that permeated the sociopolitical discourse of daily life in Michigan (Digaetano & Lawless, 1980). The particular context in which these data were collected should inform interpretation of their ensuing results.

Method

Participants and Procedures

The data for this study were collected as part of a larger project that used survey sampling procedures and investigated stress and coping in romantic couples (Bolger, DeLongis, Kessler, Schilling, et al., 1989); 778 couples were contacted from the Detroit metropolitan area, and a subset of these participants opted to participate in the diary study. To participate in the study, both partners had to be native English speakers, over 18 years old, and in a committed heterosexual relationship. Respondents were not paid for their participation, although a $5 gift was sent along with the first diary booklet. Participants completed the diary each evening and mailed their booklets back to the study team at the end of the week. Participants were asked to complete the diary entries separately and not to share or discuss their answers with their partner.

Initially, data were collected from 336 participants who lived in the Detroit Metropolitan area between 1985 and 1986; data from seven people were excluded from analyses because they filled out fewer than six diary entries and an additional 40 people were excluded during the analytic stage for having no variability in physical symptoms. All remaining participants (N = 290) completed at least six diary entries totaling 11,192 diary days (on average, 34 days of the six-week diary; range = 6–42 diary days). Participants (M = 42 years, SD = 12) were all married.

Measures

Daily Stressors. Each evening, participants were presented with a list of 21 "troublesome things" that could have occurred in the previous 24 hr including a financial problem, extra work at school or work, arguments with various people, and more (see Supplemental Materials: https://osf.io/86xmu/). These items were generated from pilot testing of stressor items in a sample of 64 married couples (Kessler et al., 1988). Participants in this sample reported, on average, 1.1 stressors per day (between-person SD = .73, range = .00–5.19).

Daily Psychological Distress. Psychological distress was assessed with an 18-item measure generated from the Affects Balance Scale (Derogatis, 1975) designed to measure anxiety (e.g., nervous, tense, afraid), hostility (e.g., irritable, angry, resentful), and depression (e.g., helpless, worthless, depressed). Participants reported on a 0–4 scale from 0 = *not at all* to 4 = *a lot* how much they felt each of these 18 distress items in the previous 24 hr. On average, participants reported 1.35 on the distress scale (between-person SD = .35, range = 1.01–3.60).

[6] Two other published papers using this dataset focus on the effects of daily stress on negative mood without considering physical symptoms (Bolger, DeLongis, Kessler, Schilling, et al., 1989) or on stress contagion among romantic partners (Bolger, DeLongis, Kessler, Wethington, et al., 1989).

Table 5
Model Results for Study 3

Model	Average person [95% credibility interval]	95% heterogeneity interval	People with slopes > 0
Psychological stress reactivity (a_1)	**0.35** [0.32, 0.38]	[−0.04, 0.67]	96%
Selective exposure (a_2)	**0.37** [0.34, 0.40]	[−0.04, 0.69]	96%
Distress somatization (b_1)	**0.15** [0.13, 0.18]	[−0.09, 0.48]	85%
Bodily feedback (b_2)	**0.13** [0.10, 0.15]	[−0.10, 0.43]	82%
Stressor somatization (c')	0.02 [−0.01, 0.05]	[−0.13, 0.19]	58%
Psychosomatic reactivity($a_1 * b_1$)	**0.05** [0.048, 0.050]	[−0.04, 0.17]	83%
Stressor-psychological distress feedback ($a_1 * a_2 * a_1 * b_1$)	**0.01** [0.011, 0.012]	[−0.01, 0.05]	80%[a]
Psychological distress-physical symptoms feedback ($a_1 * b_1 * b_2 * b_1$)	**0.006** [0.005, 0.006]	[0.00, 0.03]	83%[a]

Note. Bolded effects are those whose intervals do not cross zero.
[a] Per our discussion, although these effects are positive for many people, they are small in magnitude.

Daily Physical Symptoms. Physical symptoms were measured each evening with a checklist of 10 physical symptoms, including stomach problems and muscle tension (see Supplemental Materials: https://osf.io/86xmu/). The symptoms variable indicates the number of symptoms a participant reported in the previous 24 hr, ranging from 0 (*no symptoms*) to 10 (*all symptoms*). Participants in this sample reported, on average, .65 symptoms per day (between-person *SD* = .58, range = 0–4).

Results

Average Effects

All results can be seen in Table 5.[7]

Mediation Paths (a_1, b_1, and c'). The effects for the average person in Study 3 are similar to those in Studies 1 and 2. The average person exhibited psychological reactivity (a_1 = .35 [.32, .38]), such that days with one standard deviation more stressors than the person's own average were associated with increases in distress that were about 35% of a standard deviation above the person's own average. Similarly, the average person exhibited distress somatization (b_1 = .15 [.13, .18]); days on which the person experienced a level of distress one standard deviation above their own average were associated with about 15% of a standard deviation greater-than-average physical symptoms. Interestingly, as in Study 1 but not in Study 2, the average person did not display stressor somatization (c' = .02 [−.01, .05]), implying that for the average person, stressors had no effect on symptoms beyond their effects through psychological distress. In line with this causal chain of events, the average person exhibited psychosomatic reactivity (indirect effect = .05 [.048, .050]).

Opposing Causal Paths (a_2 and b_2). In addition to the forward-feeding causal process from stressors to distress to physical symptoms, this model also estimated reverse causal paths. In line with our hypothesis, the average person experienced more stressors when they were distressed (a_2 = .37 [.34, .40]), such that a one-*SD* increase in psychological distress relative to the person's own average was associated with an increase in stressors that were about 35% of a standard deviation above the person's own average. The other opposing effect, from symptoms to distress, also occurred for the average person (b_2 = .13 [.10, .15]); days on which the person experienced a physical symptoms one standard deviation above their own average were associated with about 10% of a standard deviation increase in distress. Because causal effects between stressors and distress, as well as between distress and physical symptoms, operated in both directions, we next consider reverberating feedback through these paths.

Stressor-Distress Feedback ($a_1 * a_2 * a_1 * b_1$). We next consider feedback in the relation between stressors and distress for the average person. In this case, stressors worsened the person's distress which, in turn, led them to have more stressors. We compare psychological reactivity, a_1 = .35, to the process in which psychological reactivity turned back around and heightened stressors, $a_1 * a_2$ = .13. This effect was nontrivial in size, meaning that stress-induced psychological distress led the average person to initiate more stressors for themselves. That is one loop. When we investigated whether that effect reverberated back and led to even more psychological distress, we found only a very small effect; $a_1 * a_2 * a_1$ = .04. The effect is now only 13% of the one that we started with. Just one more reverberation (back to stressors) and the effect was basically zero, $a_1 * a_2 * a_1 * a_2$ = .02. The average person equilibrated after just one and a half loops through stressor-distress feedback.

Considering this, it was not surprising that stressor-distress feedback had very small downstream effects on psychologically mediated physical symptoms by the end of the day. Remember that we want to know whether feedback had any additive effects on physical symptoms. We saw that the mediated effect was .05 for the average person. If we then loop though the stressor-mood path one time before taking the b_1 path, we have to add ($a_1 * a_2 * a_1 * b_1$ = .01[.011, .012]) to the mediated effect. Put concretely, experiencing a standard deviation increase in stressors was already estimated to lead to .05 more symptoms via distress, and looping around the feedback path increased its effect by 20% to .06 (.05 + .01). Just one more loop through this system, $a_1 * a_2 * a_1 * a_2 * a_1 * b_1$, decayed it to a trivially small value (less than .001). We conclude that, although stressor-induced distress worsened physical symptoms, the reverberation between stressors and distress did not

[7] We ran a secondary model that included as covariates (a) couple-level means and (b) weekend dummy variables. The results remain unchanged when controlling for shared levels of stressors, distress, and physical symptoms among couples and when controlling for weekend versus weekday effects. These results can be found in the Supplemental Materials at https://osf.io/86xmu/.

escalate to have meaningful downstream consequences for physical symptoms.

Psychological Distress-Physical Symptoms Feedback: (a_1 * b_1 * b_2 * b_1). We also consider feedback in the relation between stressors and psychological distress. In this scenario, we compare distress somatization, b_1 = .15, with the path in which that somatization turns back around and heightens psychological distress, b_1 * b_2 = .02. This effect was trivial in size. Physical symptoms like headaches may have worsened mood, but not in a way that the worsened mood went on to lead to more physical symptoms. Not even one feedback loop emerged.

Given this, we were not surprised that distress-physical symptom feedback had almost no downstream consequences on physical symptoms. For the average person, the additive effect of distress-physical symptom feedback was trivial (a_1 * b_1 * b_2 * b_1 = .006 [.005, .006]; the effect of stressors on physical symptoms only increased from .05 (the mediated effect) to .056. This means that the effect of stressors on symptoms that reverberated through distress and stressors had meaningless additive effects.

Heterogeneity of Effects

Mediation Paths. As with the first two studies, between-person differences emerged for all of the paths according to the criteria in Bolger et al. (2019). Looking closely at the distributions, there were very few reversals. This can be seen visually in Figure 10. In terms of psychological reactivity, the person at the very low end of the distribution exhibited a slight reversal (2.5th percentile of a_1 = −.04). This suggests that a small number of people (4%) experienced less distress on days with more stressors than their own average. In contrast, the highly reactive person exhibited twice as much reactivity as the average person (97.5th percentile of a_1 = .69), similar to Studies 1 and 2.

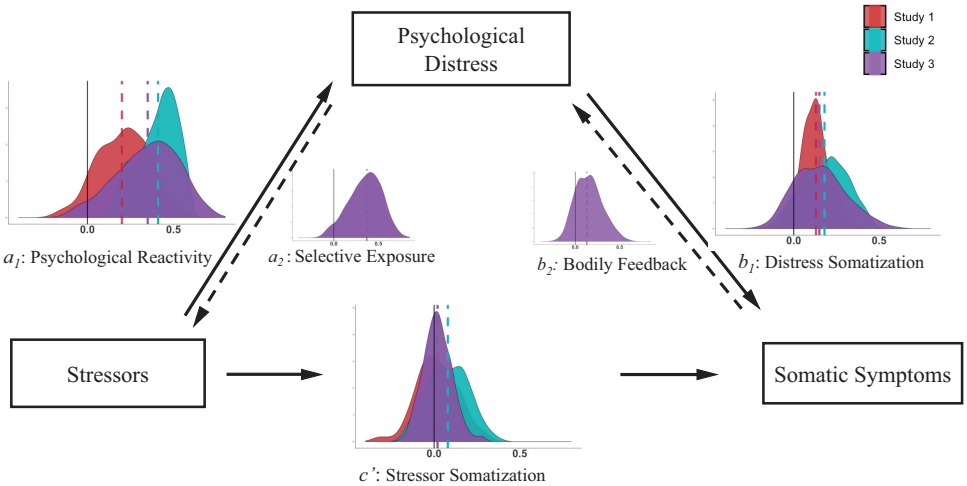
There was also considerable heterogeneity in distress somatization, such that the person at the low end of the distribution showed a slight reversal (2.5th percentile of b_1 = −.09) and the person at the high end of the distribution was more than twice as reactive than the average person (97.5th percentile of b_1 = .48). In fact, distress somatization was reversed for 15% of the sample. The greater number of reversals relative to the first two studies may have occurred because reverse causality was controlled for.

Also similar to the first two studies, psychosomatic reactivity was heterogeneous, with a few people showing effects around zero (2.5th percentile = −.04) and others exhibiting effects about five times that of the average person (97.5th percentile = .17). Perhaps because of these reversals in the distress somatization path, the indirect effect was reversed for about 17% of the sample. As before, we are reluctant to interpret the magnitude of between-person differences in the direct effect between stressors and physical symptoms (see Study 1 Discussion).

Stressor-Psychological Distress Feedback (a_1 * a_2 * a_1 * b_1). We saw that stressor-distress feedback equilibrated after one and a half loops for the average person. Given the magnitude of the parameters, we draw the same conclusion for about half of the sample; equilibrium was reached after about one and a half to two loops for the people with reactivity effects larger than the typical person.

Therefore, when considering whether the feedback loop through stressors and distress impacted downstream physical symptoms for every person in the sample, we conclude *not really*. We find that the distribution of feedback effects through the stressor-distress paths on later physical symptoms ranged from −.01 (for the person at the 2.5th percentile) to .05 (for the person at the 97.5th percentile). In practical terms, the very small reversed effects when looping through the stressor-distress paths represent the people with reversals in some part of the causal chain (either a_1 or a_2). At the high end of the distribution, the person with the largest feedback effect (.05) may seem to have had meaningful reverberation, given that the indirect effect was .05 for the average person. However, when considering that this person probably also had a large indirect effect to begin with, then the additive effect of looping through the stressor-distress path was relatively small.

Figure 10
Results for Studies 1–3



Note: Effects for the average person are the dashed lines, and model-predicted effects for each person in the samples are the densities. See the online article for the color version of this figure.

Distress-Physical Symptoms Feedback: ($a_1 * b_1 * b_2 * b_1$). Similar results emerged for between-person differences in feedback through the distress-physical symptom path. Here, the feedback effects ranged from 0 to .03, for the 2.5th and 97.5th percentiles respectively. This means that for some people, the feedback effect was actually zero and for others, those who are already high responders in the distress-symptom path, relatively small additions from looping through this feedback loop just one time emerged.

Discussion

Study 3 tested all of our hypotheses simultaneously, not only replicating results from Studies 1 and 2 but also addressing our hypotheses of bidirectional causality and feedback loops. As with the first two studies, the mediation hypothesis was confirmed for the average person. In looking at bidirectional causality, our second hypothesis was confirmed as the average person, and most people in the sample, not only exhibited selective stressor exposure (the opposite of psychological reactivity) but also bodily feedback (the opposite of distress somatization). In sum, stressors disrupted distress which disrupted physical symptoms even when opposing effects were accounted for, and those opposing effects occurred for the average person.

The third hypothesis that we tested in this study concerned feedback loops. In terms of feedback between stressors and psychological distress, we found that stressor-induced negative moods did turn back to lead people to initiate more stressors. The phenomenon whereby those added stressors led to even more distress was very small in size and did not turn back to lead to even more stressors. We concluded that the average person equilibrated after just one and a half loops through stressors and distress. We also found this for most people in the sample. In the other feedback path, there was not even one loop. Although distress led to physical symptoms, and physical symptoms led to distress, distress-induced physical symptoms didn't turn back to heighten negative mood. We understand this is a lot to think about at first, so we highlight our conclusion that people equilibrate back to normal fairly quickly in terms of distress and physical symptom. Together, this means that stressor-distress feedback is a larger phenomenon than distress-physical symptom feedback. Nonetheless, neither kind of feedback had meaningful effects on downstream physical symptoms. This was contrary to our hypothesis.

Our fourth hypothesis of between-person differences was also confirmed, in that the results from this study echoed our prior findings. The overwhelming majority of people exhibited effects in the hypothesized directions, although the range of effects was quite large.

As with the first two studies, there are methodological limitations. The most obvious is that participants filled out diary booklets over the course of the week and mailed the responses to the research team. What if participants lied about filling them out each day and instead back-filled their responses? A deep discussion of this issue can be found in the Did Participants Comply? subsection of the Supplemental Materials (https://osf.io/86xmu/), which reviews research suggesting that such lying is highly unlikely (Green et al., 2006).

The remaining limitations are not unique to this study but are unique to daily diary studies in general. A deep discussion of the measures and limitations can be found in the Supplemental Materials (https://osf.io/86xmu/), but we summarize them here. First, memory researchers increasingly emphasize the biases and heuristics in the moment of recall that can shape participant reports of events, distress, and health experiences. In terms of events, in the Can Participants Accurately Recall the Day? subsection of the Supplemental Materials, we review literature suggesting that such biases are rare for recall of discrete events (Tennen et al., 2006). In terms of distress, the same section in the Supplemental Materials draws upon recent research showing that averages of distress assessed from EMA throughout the course of the day converge with evening summaries (Neubauer et al., 2020). We also discuss in the Supplemental Materials how physical symptoms per day are clinically relevant and far less biased than longer time periods of physical symptom recall (Larsen, 1992). A final issue of variability in the instructions across datasets (i.e., that Study 2 asked about *today* whereas Studies 1 and 3 mostly asked about *the previous* 24 hr) are also discussed in the Supplemental Materials, where we highlight data showing that similar time periods are considered by participants regardless of the instructions used (Stone et al., 2020). The Supplemental Materials also highlight the unique strengths of converging operationalization and diverse sampling used in Study 3 as well as the other two studies.

Comparisons Across Studies

We hope it is clear by now that each study has its own strengths and weaknesses, providing unique evidence for the effects of daily stressors across samples and in different time periods. We believe it is the three studies considered as a whole that lends the most insight.

There was remarkable similarity in effects for the average person across studies. Psychological reactivity (.20, .41, and .35), distress somatization (.13, .18, and .15), and psychosomatic reactivity (.03, .08, .05) were not only statistically reliable in each study, but also of similar magnitudes for the average person. The stressor somatization path (.02,.08,.02) was also remarkably consistent for the average person, and it was statistically indistinguishable from 0 in Studies 1 and 3. The convergence across studies is not only important because it increases the evidence for these parameter estimates, but also because it ensures that our constraining the opposing causal paths in Studies 1 and 2 introduced nonnoticeable biases. As a stringent confirmation of nonbiased estimates, we ran the same model from Studies 1 and 2 (the constrained model) on the data in Study 3 and found that the estimates were only slightly larger relative to the model that controlled for reverse causality. In terms of heterogeneity, the percent of people with slopes in the hypothesized directions were very similar across studies, with 92%, 100%, and 96% having positive effects for psychological reactivity, 94%, 90%, and 85% for distress somatization, and 53%, 73%, and 83% for psychosomatic reactivity in Studies 1, 2, and 3, respectively.

It is also worthwhile to consider similarities and differences in the magnitudes and ranges of slopes for individuals across all three studies, especially since all of the parameters were expressed in standard deviation units. Using Hanel et al.'s (2019) overlap metric, we calculated the percent overlap between each of the heterogeneity distributions. The results can be found in Table 6.

Clearly, the largest discrepancy occurs between the psychological reactivity distributions for Studies 1 and 2. This may have occurred because participants in Study 1 constitute a highly

Table 6
Overlap Between Study Distributions

	Study 1 overlap with Study 2	Study 1 overlap with Study 3	Study 2 overlap with Study 3
Psychological reactivity	39%	67%	83%
Distress somatization	82%	82%	91%
Psychosomatic reactivity	70%	70%	90%
Stressor somatization	80%	80%	79%

specific group of people: financially stable members of relatively happy romantic relationships living in a highly urban area. In contrast, participants in Study 2 were diverse in terms of socioeconomic status, relationship status, and geographic region. This suggests, as does prior research (Almeida, 2005; Uchino et al., 1996), that happy couples who are financially stable are generally less stress or reactive than other people. In line with this notion, Study 3 participants were also in relatively happy romantic partnerships and the distribution there overlapped considerably with that of Study 1. Considered alongside the fact that the distributions in Studies 2 and 3 overlapped, it may be that increased reactivity could be primarily attributable to financial and living status (urban versus not). Although we do not have the data to test that claim, it is an interesting question for future research. Otherwise, the overlaps in the distributions are quite large, especially in light of the different measures used in each study, the different sampling procedures (self-selection versus true population sampling), the different time periods, and the different demographics of the participants. We hope that Table 6 may encourage readers to dig deeper into the small amounts of differences that did emerge between the distributions for each study.

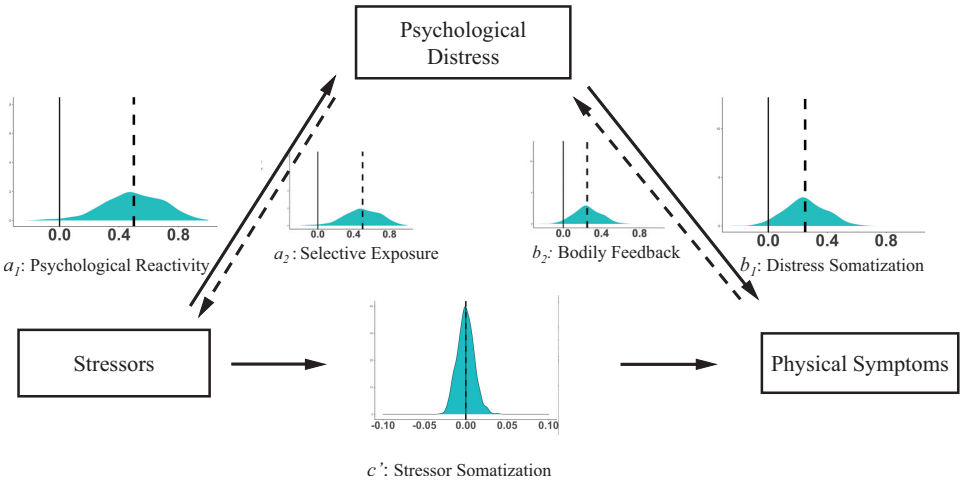
General Discussion

We zoomed into the effects of stressors in everyday life by simultaneously testing four theory-driven hypotheses, which we depict again in Figure 11 below. As expected, stressors increased psychological distress, which worsened physical symptoms for the average person across all three studies. Also as hypothesized, the short-term psychological and physical outcomes of daily stressors had opposing effects in Study 3; not only did stressors alter distress but distress also altered stressors. Not only did distress alter physical symptoms but physical symptoms also altered distress. Because we statistically controlled for those opposing causal effects in Study 3, we were more confident in the mediation parameters that we estimated in the first two studies. Next, the bidirectional causal effects enabled us to test our third hypothesis, which was that feedback loops between stressors and distress and between distress and physical symptoms would contribute to even more physical symptoms by the end of the day. This hypothesis was not supported by the data. Finally, our fourth hypothesis was confirmed in that statistically meaningful between-person differences were found for each path. Notably, the effects were in the expected directions for almost everyone in every sample. Nonetheless, the few null and reversed effects that did emerge raise interesting questions for future work, as we will discuss.

Daily Stressors Lead to Physical Symptoms Because of Stress or -Induced Distress for the Average Person

According to the Stage Model of Stress and Disease, cross-disciplinary researchers are united by their common assertion that stressors alter psychological states which, in turn, disrupt physical health (Cohen et al., 2016). Although psychologists have

Figure 11
An Integrated Causal Feedback System for Daily Stressors



Note. See the online article for the color version of this figure.

examined this at the level of acute stressors (for a review see Blascovich, 2013), at the level of event-based stressors (for a commentary see Cohen et al., 2007), and at the level of chronic stressors (for a review see Juster et al., 2010), less work has focused on how *daily* distress mediates the relation between daily stressors and daily physical health. When daily stressors have been the focus (Mroczek et al., 2015; Ong et al., 2013; Piazza et al., 2013; Sin et al., 2015, 2016), research has typically taken a between-person approach. Similar to the aforementioned research on other timescales of stressors, the daily stressor work has focused on whether between-person differences in psychological reactivity explain between-person differences in physical health. Although this is a valid approach, it is not without limitations (Kenny et al., 2003). Most notably for stress and health, between-person differences in personality have been shown to confound the relation between self-reported stressors and physical health outcomes. Specifically, people with persistent negative affect (neuroticism) are those who report more stressors and physical symptoms (Watson & Pennebaker, 1989). By leveraging within-person mediation, we partialed out neuroticism as a confound and instead investigated negative affect as it fluctuates within an individual. Our results imply that, regardless of chronic negative affect, stressors are associated with changes in state negative affect for each person, and that those individualized changes in negative affect are associated with heightened physical symptoms.

This finding translates into actionable interventions that could help the average person navigate the ups and downs of daily life. Mindfulness therapy is increasingly popular, both in the psychological sciences and the general population, because it aims to improve health via a brief and effective psychological intervention. Mindfulness-based interventions have successfully reduced physical ailments associated with stressors (Ali et al., 2017; Würtzen et al., 2015) and a meta-analysis asserts the efficacy of such approaches (Chiesa & Serretti, 2009). Our results undergird the potential for mindfulness to reduce the effect of daily stressors on physical symptoms, as mindfulness tends to reduce negative affect throughout the day (Brockman et al., 2016). We think a pressing avenue for future mindfulness-based interventions is to specify the time-scale of stressors; acute versus event-based versus daily versus chronic (Epel et al., 2018). Because we focused on psychological mediation for daily stressors in particular, we highlight that daily stressors are unique in that they do not require a huge amounts of life readjustment (Holmes & Rahe, 1967), but they do require constant and vigilant attention. Perhaps mindfulness therapies that are targeted to daily stressors can emphasize their chronicity and deceptive triviality.

The Effects of Daily Stressors Operate in Opposing Directions for the Average Person

Because the bulk of the stress literature has focused on the forward-feeding mediational process from stressors to psychological distress to physical health, opposing causality has mostly been ignored. We think this is, in part, because of the methods that are currently available to stress researchers. Because experiments are considered the best method for determining causal directionality, researchers tend to focus on only one causal direction at a time to make statements like, "stressors cause psychological distress." This means that either stressors are manipulated and psychological distress is measured *or* that psychological distress is manipulated and stressors are measured (for an exception in a different context, see Smith, 1982). When nonexperimental data is used, as in daily diary research, the contemporaneous measurements lead to correlational statements. By nature, correlations lead researchers to make assertions like, "we do not know whether stressors cause psychological distress *or* whether psychological distress causes stressors." Because we leveraged nonrecursive structural equation modeling, we were able to blend experimental and correlational ways of thinking; for example, we thought about effects from stressors to distress *and* from distress to stressors.

Although Some Feedback Occurs, Feedback Loops Did Not Impact Physical Symptoms for Almost Anyone

Thinking about bidirectional causality led us to the concept of feedback loops, which we view as the most significant contribution of our work. Prior to running analyses, we pondered the following questions: Do feedback loops actually occur throughout the course of the day? If they do, then what is the magnitude of feedback? Which kind of feedback, that between stressors and distress or that between distress and physical symptoms, is more important? How many iterations through the feedback loops does it take for a person to equilibrate back to normal? And our most pressing question: Do feedback loops contribute to more stress-induced physical symptoms by the end of a day?

As we mentioned, bidirectional causal paths between stressors and distress and between distress and physical symptoms emerged. Although we found that stressor-induced distress led to more stressors (that is, one loop emerged through stressors and distress), we did *not* find that distress-induced physical symptoms led to more distress (that is, one loop through distress and physical symptoms did not emerge). Given these findings, one avenue for interventions could be to help people realize the concrete effects of stressors to prevent the stressor-distress loop. Concretely, when someone like Jasmine is in a bad mood because of a minor fender-bender in the morning, she should be careful in her interpersonal interactions so as not to initiate a fight. Recognizing *why* a person is distressed (because of an exogenous stressor) could help people refrain from making things worse for themselves (Goldring & Bolger, 2021).

Because there was a meaningful feedback effect between stressors and distress, we wanted to know when a person equilibrates. We found that just one and half loops through the system were sufficient for a person to stop escalating. This is a nice finding in that it implies that, over the course of one day, a person might initiate some number of stressors for themselves because an initial stressor put them in a bad mood, but that the distress-induced stressor will have only a small additive effect on distress. Although the concept of equilibrium was previously vague, we now know that people reach it relatively quickly, at least in terms of concrete outcomes like heightened daily stressors. As we discuss in the Future Directions section below, other stress outcomes might not equilibrate as quickly, especially when their time-course is quicker.

Because the stressor-distress paths equilibrated quickly, and because the distress-physical symptom paths did not even reach a full loop, neither had meaningful effects on physical symptoms. Before running our analyses, we thought that feedback loops might not only happen, but also that they would cause more

physical symptoms by the end of the day. Because that hypothesis was not confirmed, we know that reverberating feedback does not have to be a major target for interventions in terms of physical symptoms outcomes. In other words, our results suggest that trying to prevent people from spiraling in the aftermath of a stressor will not have a meaningful impact on physical symptoms.

The Magnitude and Range of Between-Person Differences Sheds Light on Stressor Reactivity

Of course, we not only investigated all of our hypotheses for the average person but also considered the magnitude and range of between-person differences. Until recently, it has been difficult to quantify whether between-person differences really matter. How different do people have to be for those differences to be substantively important? According to Bolger et al. (2019), those differences have to be (a) at least half the magnitude of the effect for the average person, (b) statistically significant (i.e., between-person differences are not a statistical artifact of the data), and (c) substantial enough to contribute to model fit. We confirmed in almost all paths for all three studies that these criteria were met. Now, the notion of between-person differences in stressor reactivity is not an abstract assumption in the field. It is an empirical statement.

Another long-held assumption that we investigated was the universality of each causal path among U.S. adults. Our representative sampling across three different studies which, together, constitute a diverse range of ages, socioeconomic statuses, geographic regions, and racial identities, enables us to take these distributions seriously. The overwhelming majority of people in every sample exhibited paths in the directions we expected. As Haaf and Rouder (2019) would say, these phenomena are close to reaching universality, at least in the United States.

Nonetheless, our hypotheses were not born out for everyone. We did not see 100% of people in all three studies with effects in the expected directions. We also did not see trivial differences between people. Instead, we saw some people being as much as four times as reactive as the average person, some people being nonreactive, and others having effects in reverse of our hypothesized directions.

If we first think about between-person differences in the mediated effect, we realize that the mediation hypothesis was true for almost everyone, but not quite. For some people, the effect was slightly reversed and for others it was very close to zero. One idea proposed by Kleinman (1977) is that in some cultures, expressing emotionality is non-normative, which leads people to express their internal turmoil by complaining about physical problems such as upset stomachs. Indeed, people from Asian (Karasz et al., 2007; Ryder et al., 2008; Yen et al., 2000) and Hispanic (Angel & Guarnaccia, 1989; Canino, 2004) cultures typically endorse physical symptoms after stressor exposure. We think that because we measured psychological distress with self-report questionnaires, individuals for whom it is culturally inappropriate to say they are "on edge" or "feeling anxious" may be the ones who did not endorse those items and therefore did not show psychological mediation. This implies that interventions that target psychological states may be ineffective for people who are not used to, or do not want to, communicate about their feelings. Instead, we may need to bypass psychological states altogether by implementing interventions such as breathing exercises (Han et al., 2000) for those people. Of course, another implication of the self-report distress measures is that some individuals may simply lack emotional awareness. Those people would be unable to accurately report their distress following a stressor, which would imply an unmediated effect even if it were there. In either case, our results suggest that the mediation framework, while applicable for most, did not perfectly fit the data of every individual.

We can understand this even better when we consider the magnitude and range of effects for each path in the mediation process separately. Against our hypothesis, some people were not psychologically reactive to daily stressors at all, and others showed reversals. In considering the meaning of nonreactivity, we realize that depressed individuals exhibit blunted reactivity to stressors (Burke et al., 2005). The corollary is that nonreactivity is probably as maladaptive as overreactivity (Selye, 1976). Moreover, across samples, 3% of people showed reversed effects. More stressors led to less distress. Although rare, this phenomenon happened; those estimates are based on pooled information across many people. It could be that having a positive mindset about stressors reduced negative affect (Crum et al., 2013), although this remains an area for future work. And on the opposite end of the distribution, some people were more than twice as psychologically reactive as the typical person. This finding implies that interventions focused on reducing stress-induced psychological distress could be twice as effective if they spent time focusing on those highly reactive people.

More surprising were our findings about between-person differences in distress somatization. Before running the models, we intuited that reversals might occur and showed how James had fewer physical symptoms when psychologically distressed. But no existing theory supported that intuition. On average across samples, 10% of people exhibited reversed effects of distress somatization. In Study 3 (when bidirectional effects were controlled for), it was 15% of the sample. We posit that for some people, psychological distress constitutes the full manifestation of stressor reactivity. Although stressors make people feel more anxious, depressed, and on-edge, for some people they do not go on to induce headaches and muscle tension because their psyche is already heightened. Because of this, distress not only fails to lead to physical symptoms, but it actually reduces the likelihood of them. Of course, empirical research is now needed to support this theory. In thinking about null and magnified distress somatization, we again conclude that future research should focus on applying meaning to the full range of effects that we found; that some people exhibit nontrivial reversals, some are not reactive at all, and others are as much as three times as reactive as the average person.

In all cases, we think that daily diary approaches may be effective in identifying individuals who are most in need of stress interventions. Interventions could use a week-long diary among a large population sample to identify the people who are most stress-reactive. Then, the intervention could be administered among those individuals. Doing so could be more effective than trying to target individuals on the basis of their personality, relationship status, and chronic stress environments. Although those moderators explain differences between people, they would be less accurate than directly identifying each person's amount of chronic reactivity, as the daily diary approach does.

Finally, we encourage other researchers to take a similar metatheoretical approach to between-person differences. Using daily diary data to investigate the magnitude and range of slopes for every person illuminated stress theory and intervention. Our findings not only generated new questions that remain unresolved by existing theory, but also imply that between-person differences in other stress contexts could be null, reversed, or extremely amplified. We are excited to see whether that theory is borne out across other stress domains.

Future Directions

Although we focused on the downstream effects of daily stressors, our integrated causal feedback system depicted in Figure 11 can answer many more questions. Using path tracing rules, other researchers could test questions like: Does psychological distress, regardless of whether it is initiated by stressors or not, lead to physical symptoms? In other words, start the causal chain at psychological distress and look at downstream consequences. Another question could be: To what extent do physical symptoms influence stressors via mood? In other words, start the causal chain at physical symptoms and end with stressors. Our model has the capacity to answer these questions. Future readers can leverage our model to develop theories about how and why such processes might occur.

Moreover, other constructs could be inputted into the boxes in Figure 11 that would answer other unresolved questions about stress. For example, one question in the stress literature concerns the instantaneous relation between stress and coping. According to Lazarus (2006), stressors induce negative emotions that immediately trigger coping responses such as cognitive reappraisal and psychological avoidance. These coping responses quickly feed back to reduce negative emotions as part of an adaptational process. Because this process occurs so quickly, Lazarus never empirically tested his instantaneous interaction hypothesis. Moreover, how long it takes for a person to equilibrate back to a baseline levels of emotions remains unknown. Because the answers to these questions are obfuscated by the quick time-course of stress-induced emotions and coping, the nonrecursive SEM could be used to approximate it. In that context, researchers would estimate the feedback loop between negative emotion and coping to quantify the number of loops through the system that it takes for equilibrium to be reached. Once known, the field would gain empirical evidence for Lazarus' long-held assumption, as well as more information about the basic process of stress and coping.

Yet another expansion of our framework could be to examine the mediating role of positive affect. By now it is clear that negative and positive affect constitute separable dimensions, each with their unique implications for physical health (Clark & Watson, 1988; Richardson, 2017; Watson, 1988). Whether stressors reduce positive affect, which increases physical symptoms, could be tested by collecting daily diary data on positive affect and running the same model that we did with positive affect. In fact, Study 2 in the present paper comes from the publicly available MIDUS dataset that includes daily measures of positive affect. Someone would simply need to use these measures and replace negative affect with positive affect in the code found on our OSF repository. Of course, a considerable amount of theoretical integration and hypothesis generation would need to be done in that work. Once achieved, the ensuing model could also provide additional insight into the buffering hypothesis (e.g., Nelson & Bergeman, 2020), which states that persistent positivity in the face of stressful events can buffer against health problems.

Of course, nonrecursive SEMs are not without their limitations. To test these questions about stress and coping and about stress and positive aspect, the models must meet certain assumptions and analytic software must exist to reliably estimate their complex distributional structures. Most prohibitively, the model must meet be identified through the use of instrumental variables, variables that are typically difficult to conceptualize and collect (see the Statistical Model section). Nonetheless, we point out that daily diary data is uniquely suited to meet these demands when combined with Bayesian estimation for model convergence.

A critical methodological avenue for future work will be to generate cut-off points on the distributions of between-person differences that map onto meaningful effect sizes for each person in the sample. Consider that our assessment of between-person differences was based on zero as the cutoff point; we decided that slopes in the hypothesized directions were above zero, those with null results were at zero, and those with reversed effects were below zero. But is a slope of .02 in standard deviation units meaningful? Or should that be considered a null effect? The reason these questions are difficult to answer is that within-person standard deviation units mean different things for different people. For some, .02 standard deviation units in physical symptoms could be 1 symptom, it could be .01 of a physical symptom, or it could be any other number of symptoms. Therefore, generating a range of 'meaningless effects' rather than a cutoff point at zero is difficult; the task is to translate within-person changes to between-person comparisons. In sum, although we think our approach using zero as a cutoff point for the range of between-person differences is justified, it could be improved.

Finally, we noted in the discussion of Study 1 that although the indirect effect shrunk to zero for the average person, it did not shrink the heterogeneity to zero. We saw this again in the remaining two studies; the average direct effect was zero while people at the low ends of the distributions had highly negative slopes and those at the high ends of the distributions had highly positive slopes. What does this mean in practical terms? It means that we did an excellent job explaining the link between stressors and physical symptoms for the average person, but that our mediator did not do an excellent job explaining between-person differences in the link between stressors and physical symptoms. This opens the door for future researchers to not only determine what other mediators could be at play, either between- or within-subject, but also how to analytically estimate mediation heterogeneity.

Limitations

First and foremost, the data and models presented here assess causal links through the use of within-subject comparisons rather than through experimental manipulation. This was justified through already existing experimental support for the causal arrows in our model, our implementation of within- rather than between-person mediation, as well as our application of nonrecursive SEMs in Study 3. Nonetheless, the fact that we were only able to test the full model in Study 3 implies that other research should replicate those findings. Second, we modeled the number

of physical symptoms rather than the severity of the symptoms. This was done partially to map our results to an extensive body of existing literature that explores antecedents of the number of physical symptoms reported in daily life (Almeida, 2005), and in light of the extremely high correlation between the number and severity of physical symptoms (Mroczek & Almeida, 2004). Nonetheless, there is a conceptual distinction between the number of physical symptoms that a person reports and the severity of those symptoms and future work could test the hypotheses we outlined in the context of severity. Additionally, stressors and distress only account for a small amount of the variability in physical symptoms. Although interested readers may draw upon the data and code in our Supplemental Materials (https://osf.io/86xmu/) to investigate this quantitatively, our brief analysis found that only about 10% of the variance in physical symptoms is explained by stressors for the average person. Clearly, factors other than stressors lead to headaches, muscle tension, and other physical symptoms in daily life.

A number of measurement limitations should also be considered, for example the self-reported nature of the data and the use of different instructional phrases (i.e., in *the previous* 24 hr vs. *today*) on the inferences we made. We discuss these and other measurement considerations in detail in the Supplemental Materials (https://osf.io/86xmu/), and we encourage readers to consider our findings in light of what the measures can and cannot tell us.

Conclusion

We have proposed an integrated causal feedback system for daily stressors that enabled us to resolve discrepancies, fill gaps, and contribute new ideas to the stress literature. We asked and answered four key questions about the effects of daily stressors: (a) does psychological distress mediate the association between stressors and physical symptoms?; (b) do causal effects operate bidirectionally?; (c) do feedback loops occur and do they impact physical health over the course of a day?; and (d) how much do people differ from one another in the magnitude of these effects? We hope that our answers to these questions encourage other researchers to study integrated causal systems that can similarly advance the field of stress science.

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