Mind, Body, Illness: Amidst Pandemic, Opportunities for Discovery

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This is my final column to you as APS President. In the midst of this extraordinarily difficult and trying time, I hope that you and your loved ones remain healthy and safe.

In early March, as I raced home from abroad to shelter in place with my family, I asked myself a question: Are respiratory diseases, such as the common cold, physical illnesses or psychological illnesses? If you think this is a silly question, think again. When scientists place a cold virus directly into the noses of healthy adults, only about one-third develop respiratory infections. So a cold must have other causal factors. And some of them may be psychological.

The Common Cold Project is a series of prospective viral-challenge studies that were conducted from 1986 to 2011 (e.g., Cohen, 2005). Psychologist Sheldon Cohen and colleagues (Cohen, Miller, & Rabin, 2001; Cohen et al., 2013) placed viruses into the noses of healthy adults, including a coronavirus linked to the common cold, and observed who developed respiratory infections. Five studies revealed that a virus is not a sufficient cause. Chronic stress, affect and emotion, social isolation, social and economic disadvantage, childhood adversity, or other psychosocial factors may also confer risk. Social integration, social support, positive affect, and a high-quality relationship with parents in childhood may confer resilience.

It’s tempting to assume that a virus is the major cause of a cold, and any psychosocial factors are subsidiary and merely moderate the impact. After all, you cannot develop a cold without a virus. But a virus is not *the* cause. Respiratory infections, like most biological phenomena, more likely emerge from a complex web of weak, interdependent causal factors—the kind I described in my March 2020 *Observer* column (see also Lewontin, 2000)—and a virus is one of those causal factors.

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Here’s what I mean. A virus does not “replicate itself” from its genes within a passive human body. Its capacity to cause infection requires certain necessary conditions in that body’s cells, immune system, and brain. The ensemble of “brain+body” provides a necessary environment for a virus. It contributes to the likelihood of its own infection, not only by the state of its immune and endocrine systems, but also by the mental phenomena it creates, which in turn are linked to immune and metabolic function. Therefore, the probability and severity of infection at any moment, as well as resilience (exposure to a virus with no symptoms), is a unique confluence of a virus in a body with a thinking, feeling, and perceiving brain. Both virus and “brain+body” are mutually dependent causes; therefore, neither should be ignored in science or in practice.

In this view, psychological factors may be necessary causes of respiratory illness. They are not sufficient to develop a common cold, but then, neither is a virus. Cohen et al.’s studies are consistent with a larger body of important research by health psychologists which suggests again and again that mind and body are linked, not in a vague metaphorical way, but in a real, probabilistic, biological way. What we think and feel, how we experience the world, and who we experience it with may translate into vulnerabilities or resilience to illness (e.g., see Slavich, 2020).

If psychological factors causally contribute to respiratory illness, then scientific research that directly studies these factors could prove vital to physicians, epidemiologists, and virologists on the front lines of the COVID-19 pandemic. This research may, in fact, be as important as the more biologically focused science that fills the front section of major newspapers these days. Reifying an illusory boundary between mind and body, mental and physical, weakens our understanding of disease, and so it’s tragic (and frankly, infuriating) that decisionmakers in this unfolding drama, and the media who report on it, have a blind spot for research that dissolves this boundary, at least as of mid-April when this column went to print.

> Historians have shown time and again that improving biomedicine alone, without addressing the psychosocial conditions that foster the spread of respiratory illness, threatens the most vulnerable among us and allows viruses to persist.

This blind spot could arise for various reasons. We don’t know, for example, whether findings from the Common Cold Project generalize to respiratory infection from the COVID-19 virus. Nor do we know whether the findings generalize to a pandemic unfolding in naturalistic settings around the globe. But the Cohen et al. findings provide a solid foundation for hypothesis-testing these and other empirical questions. Similarly, there is research suggesting that chronic stress may impact the effectiveness of vaccines to battle infection, particularly when the body’s immune system is producing antibodies to a

novel strain of a virus (i.e., where a person does not have much in the way of preexisting antibodies when exposed; Quigley et al., 2007; Vedhara et al., 1999). And, indeed, psychological stress may blunt the antibody response to vaccines that are developed for newer influenza strains (also a respiratory illness; e.g., Cohen et al., 2001; Pedersen et al., 2009). If these hypotheses are ignored, particularly as the pandemic evolves, we may miss opportunities to make potentially life-saving discoveries.

Another reason for this blind spot may be that, frankly, we don't know exactly how mental events, like the experience of stress, are transformed into physical conditions, like immune function. But an absence of knowing everything is not the same as knowing nothing. The Common Cold Project suggests that, at least for cold viruses, psychosocial factors leave a brain less able to regulate the proinflammatory cytokine response upon exposure to a virus. Other research has established a relationship between shorter telomere length and chronic stress (e.g., Epel et al., 2004), increases in proinflammatory cytokines, and poorer antibody response to vaccines (e.g., Goronzy et al., 2001; Saurwein-Teissl et al., 2002), and people with shorter telomeres (in white blood cells that help mount an immune response to pathogens) had an increased risk of respiratory infection (Cohen et al., 2013).

Yet another reason: The scientific findings might confuse people and cause inadvertent harm. Governments, medical providers, and scientists in many countries have been largely concerned with flattening the curve (i.e., managing the number of people with infection) so as not to overwhelm medical providers and increase the death rate. A public message that social support offers some protection from illness, for example, might lead people to ignore pleas to socially distance, etc. So alongside the science, we'd also need clear, concrete messaging: Maintain social connections while remaining physically distanced from others. Try to manage your stress and your emotions as you continue to wash your hands, disinfect surfaces, and perform all the other behaviors that reduce your virus exposure.

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Regardless of these concerns, it is very possible that psychological caretaking—reducing stress, decreasing loneliness, and generally managing one's emotional experiences—could help to flatten the curve *after* exposure to the virus. Many people, including many scientists, however, find it challenging to grapple with the idea that illness and disease might be caused by a virus *plus* the psychological conditions of its host, rather than by a virus alone. This brings me to perhaps the most important reason for the blind spot: Many people cling to a belief in biological determinism and simple unitary causes. This pair of beliefs—this *ideology of illness* (Lewontin, 1991)—lurks in large swaths of medical and scientific practice. In the present pandemic, biological determinism is to assume that COVID-19 has a fixed, deterministic capacity to cause illness, independent of the conditions of its host. A belief in simple, unitary causes is to assume that COVID-19 is responsible for respiratory infection in the normal medical sense and therefore is a sufficient cause, and all other factors (psychological, social, economic) must be modulatory.

This blind spot has serious consequences for the health and welfare of people who are struggling during this pandemic.

One immediate consequence is that people die from infection at a faster rate when they live in conditions that foster chronic stress and other psychological causes that make infection more likely. Other factors

play a role here too, such as less access to health care, discriminatory treatment practices, and living conditions that make social distancing a luxury. But historians have shown time and again that improving biomedicine alone, without addressing the psychosocial conditions that foster the spread of respiratory illness, threatens the most vulnerable among us and allows viruses to persist (see McMillen 2016). From the mid-18th century to the beginning of the 19th century, for example, most people in Europe and North America died of infectious respiratory diseases such as tuberculosis, influenza, and pneumonia. Death rates from these illnesses slowly declined as nutrition, housing, and wages improved. The discovery of vaccines and other medicines had an effect, of course, but conditions that reduced chronic stress, allowed parents more quality time to spend with their children, and increased well-being were the major causal factors (Burney, 2000; Lewontin, 1991). Even in the United States today, infant mortality falls with every dollar increase of the minimum wage (Komro et al., 2016).

Our current societal system of social stratification and economic disparity, and the psychological sequelae that it cultivates, belong to the web of factors that caused this pandemic. These consequences are now coming for the rest of us as people lose their jobs in droves. And severe, downstream effects may already be in motion; for example, children of stressed or depressed parents may be more likely to suffer health problems decades down the road (e.g., Hullam et al., 2019; Raposa et al., 2014), perhaps via alterations in immune function that begin in childhood (e.g., Ulmer-Yaniv et al., 2018).

Right now, our society spends substantially more time, creativity, and money on isolated biological or chemical approaches to health and illness than it does to understand how thoughts, feelings, and behaviors create conditions for health or illness to flourish. What would life be like, right now, if people understood that there may be *sound biological reasons* for reducing stress and negative affect, investing time and energy in social connectedness (particularly for children, the elderly, and people who are most at risk for illness), and generally being more mindful of how they can curate their day-to-day experiences?

We psychological scientists are uniquely positioned to answer these questions through our research and by educating the public. If we follow the lead of Cohen and other health psychologists, and continue to dissolve the porous boundary between the mental and the physical, we can remedy the blind spot and create opportunities for discovery that could ultimately save lives.

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