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Occupational Depression

Depression is an important topic. It is a major contributor to the global burden of disease and disability (James et al., 2018). Our goal for the beginning of this chapter is to trace, however briefly, the history of humankind's acquaintance with the disorder. Our forebears have known about depression for millennia. We commonly recognize it in a person when that individual is persistently sad, derives little or no pleasure from the things that ordinarily give pleasure, retreats from getting in touch with friends, and so forth. We will provide a more formal definition of the condition later in the chapter. Although the focal concern of this chapter is work-related depression, by way of an introduction, we briefly address some of the historical background regarding our knowledge of depression in general.

Brief History of Depression

Our brief history commences with the beginning of the recorded word, in Mesopotamia between 2900 and 2300 BCE. The context is *The Epic of Gilgamesh*. King Gilgamesh had been affected by two of life's inevitabilities. First, he is grieving the loss of his friend, Enkidu. The second is his fear of his own eventual death and the fruitless search for a way to avoid it.

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Urshanabi said to him, to Gilgamesh:
 “Why are thy cheeks wasted, is sunken [thy face],
 Is so sad thy heart, [are worn thy features] ?
 (Why) should there be woe in [thy belly],
 [Thy face be like that] of a wayfarer from afar,
 With cold and heat be seared [thy countenance],
 [As in quest of a wind-puff] thou roamest over the steppe”
 [Gilgamesh] said [to him], to [Urshanabi]: “[Urshanabi, why
 should my] cheeks
 [not be so wasted], [So sunken my face],
 [So sad] my [heart], so worn my features?
 [(Why) should there not be] woe in [my belly],
 [My face not be like that of a wayfarer from afar],
 Not be so seared [my countenance with cold and heat],
 [And in quest of a wind-puff should I not roam over the
 steppe]

(Speiser, 1955, p. 91)

We note the sad heart, the somatic symptom of a stomachache, and the dour countenance. Later we will address the difference between depression and mourning, a subject about which Sigmund Freud famously wrote.

Depression, as a human condition, is also evident in several verses in the Hebrew Bible. In Ecclesiastes 1:8, we read: “All things toil to weariness; man cannot utter it, the eye is not satisfied with seeing, nor the ear filled with hearing” (The Jewish Publication Society, 1917). The verse underlines fatigue, anhedonia, and an indifference to life, hallmarks of depression.

Verses 1 and 2 of Psalm 40 are also instructive. The Jewish Publication Society (1917) translates the verses as follows:

1. I waited patiently for the LORD; and He inclined unto me, and heard my cry.
2. He brought me up also out of the tumultuous pit, out of the miry clay; and He set my feet upon a rock, He established my goings.

A more interpretative translation of the verses comes from the Tyndale Bible.

1. I waited patiently for the LORD to help me, and he turned to me and heard my cry.
2. He lifted me out of the pit of despair, out of the mud and the mire.

Tyndale House (1996)

The more interpretative translation refers to King David being in the pit of despair, an indication of his melancholic suffering. We observe despair/sorrow in Psalm 13:3, “How long shall I take counsel in my soul having sorrow in my heart by day?” (The Jewish Publication Society, 1917).

Scattered through the writings of Hippocrates, the Greek physician who lived during the fifth century BCE, is the term “melancholia.” In his *Aphorisms*, Hippocrates (1931) described melancholia as “fear and sadness that last a long time.” The term is thought to be largely synonymous with depression and is derived from the Greek words for black [μέλαινα] bile [χολή]. Humorists like Hippocrates and his followers believed that sufferers of depression had an excess of black bile, black bile being one of the four humors, which also include yellow bile, blood, and phlegm. Good health required balance among the humors. Although that belief is considered useless today, Hippocrates and his followers, however, did something of great importance. Hippocrates advanced the idea that physical disorders and melancholia had natural causes (Jackson, 1986). The naturalist hypothesis of Hippocrates was then overlooked for centuries. In the Middle Ages, people commonly believed that some diseases, including melancholia, had supernatural causes, for example, the devil’s handiwork or God’s punishment (Jackson, 1986).

We observe evidence in Shakespeare’s *Hamlet* that Elizabethans understood melancholia.

. . . I have of late—but
wherefore I know not—lost all my mirth, forgone all
custom of exercises; and indeed it goes so heavily
with my disposition that this goodly frame, the
earth, seems to me a sterile promontory, this most
excellent canopy, the air, look you, this brave
o’erhanging firmament, this majestic roof fretted
with golden fire, why, it appears no other thing to
me than a foul and pestilent congregation of vapours

*Thank you, Mr. Jacob Hendon, ISS’s
middle school English teacher*

The Hippocratic humoral theory of melancholia, with its idea that an imbalance in bodily humors accompanies melancholy, lived on through the Renaissance. The English scholar Robert Burton, who published *The Anatomy of Melancholy* in 1621, subscribed to a humoral view of depression. The view was commonly held throughout Europe (Sadowsky, 2021).

By the end of the nineteenth century, the mental disorder melancholia was commonly viewed as the province of psychiatry. From the standpoint of clinical science, it is important to reliably distinguish melancholia from other mental disorders. Important milestones in psychiatric classification were achieved in the late nineteenth and early twentieth centuries. In the 1890s, the German psychiatrist Emil Kraepelin developed the beginnings of a nosological system for psychiatry by differentiating manic-depressive psychosis and dementia praecox, which is recognized today as schizophrenia (Jackson, 1986). He used both the symptoms and, based on longitudinal follow-up, the course of the disorder (e.g., whether deteriorating over time or not) to help establish the diagnostic categories. Manic-depressive psychosis included depressed states and manic states (circular insanity), in other words, alterations of depressive and manic periods (today's bipolar disorder). He brought "most melancholic disorders together, eventually to be named manic-depressive insanity" (Jackson, p. 193). As for symptoms of depression, Kraepelin included cognitive slowness, indecisiveness, limiting one's thoughts to life's dark side, exhaustion, and suicidal ideation. Kraepelin was able to discern levels of severity in melancholia from straightforward depression to a depressive condition so severe that it is accompanied by hallucinations and delusions. Kraepelin came to regard these disorders as frequently having a basis in heredity (Jackson, 1986), a far cry from an imbalance involving black bile. He, however, could not pinpoint anatomical markers of these disorders.

In 1904, the Swiss American psychiatrist Adolf Meyer advanced nosology further by distinguishing (unipolar) depression as a category that is separate from Kraepelin's manic-depressive nosological category (Jackson, 1986). Meyer favored the term "depression" over its overripe synonym "melancholia" (Sadowsky, 2021). Meyer tended to regard mental disorder as a pattern of maladaptive responses to the circumstances of life, the patterning depending on "constitution and life experiences" (Jackson, 1986).

What is clear thus far from this brief survey is that from the beginning of recorded time melancholia or depression has been widely observed. But what causes it? As science matured, the idea that the basis for melancholia lies in excessive black bile was recognized as a dead end.

"Depression is rage turned inward," Dr. Jennifer Melfi

Beginning in the late 1890s and continuing onward into the next century, the psychoanalytic movement had much to say about mental disorders, including depression, and came to dominate psychiatry for much of the twentieth century. The German psychoanalyst Karl Abraham (1968/1911) observed the similarities of grief or sadness, on one hand, and depression, on the other. He then suggested an important difference. Abraham advanced the view that depression is unconsciously motivated.

Sigmund Freud (1953–1974/1917) graciously allowed Abraham's observation to be the point of departure for the influential paper "Mourning and Melancholia," although Freud had made the grief–depression comparison even earlier than Abraham.¹ In "Mourning and Melancholia," Freud wrote that "although mourning involves grave departures from the normal attitude to life, it never occurs to us to regard it as a pathological condition" (p. 243). The mental features of melancholia include "a profoundly painful dejection, cessation of interest in the outside world, loss of the capacity to love, inhibition of all activity, and a lowering of the self-regarding feelings to a degree that finds utterance in self-reproaches" (p. 244). Freud noted that individuals who mourn a person they loved share many of the same features as the melancholic, but with one notable exception, namely, disturbed self-regard.

Freud observed that in mourning, eventually the individual who suffered a loss, as grief-stricken as the individual is at first, gradually comes to regain an interest in the world. He called that transition "the work of mourning." The person suffering from melancholia may have, indeed, suffered a loss of varying degrees of personal importance, ranging from

¹ Noted by James Strachey, the editor of *The Standard Edition of the Complete Psychological Works of Sigmund Freud*.

the death of a parent with whom the melancholic has had an ambivalent relationship to a romantic relationship that ended when one partner jilted the other to a personal slight from a close friend. In melancholia, Freud pointed out that the degree of symptom manifestation and duration is greatly out of proportion to the magnitude of the loss. In Freud's estimation, melancholics regard themselves as "worthless, incapable of any achievement and morally despicable."

Freud outlined what he regarded as a major cause of depression by attempting to solve the riddle of that surmised disproportion. He wrote, "If one listens patiently to a melancholic's many and various self-accusations, one cannot in the end avoid the impression that often the most violent of them are hardly at all applicable to the patient himself, but that with insignificant modifications they do fit someone else, someone whom the patient loves or has loved or should love" (p. 248). Freud viewed the self-reproaches of melancholics as reproaches against a love object that have been unconsciously displaced onto the self. To reproach another who is (or should be) much loved (e.g., a mother or a father) is impermissible. Murderous rage against a love object gets displaced, becoming suicidal ideation. The treatment for melancholia is then psychoanalysis, an important goal of which is to make what has been unconscious conscious.

The quotation (Figgis et al., 2004) that heads this section comes from an episode of a popular TV crime series about a mobster named Tony Soprano who operates in northern New Jersey. Unknown to the soldiers and capos in Tony's crime family and the crime families of his rivals, he sees a psychiatrist named Jennifer Melfi for treatment of his mental health problems. The episode we quoted was first broadcast in 2004, more than 87 years after the publication—in German—of *Mourning and Melancholia* and continues to be broadcast, evidence of the staying power of Freud's ideas regarding a principal cause of depression.

Challenge to Freud's Explanation of Depression

Freud's theory of depression and other mental disorders (e.g., schizophrenia) has not been without critics. The Austrian British philosopher of science Karl Popper (1963) advanced the view that psychoanalytic theory should not even be regarded as a scientific theory. Popper rejected

the tendency of adherents of psychoanalysis to interpret many kinds of human interactions as evidence for the truth of psychoanalytic theory. The first author, to help undergraduates attending his research course, created an illustrative example of what Popper meant. Here is an excerpt from that course.

A psychoanalyst would find consistent with psychoanalytic theory the following observations. A pair of overprotecting parents living in the Park Slope section of Brooklyn raises a very hostile boy. The analyst asserts that the parents' overprotectiveness unconsciously generates belligerence in the offspring, who directs hostility to other neighborhood children, suspecting that they "are out to get him." Another pair of overprotecting parents from the same neighborhood has a nervous boy. The analyst claims that the parents unconsciously and excessively communicate to the child that the other children in the neighborhood are dangerous delinquents, causing the boy to be meek and avoidant. Yet another pair overprotecting parents living in the same neighborhood has a normal boy and somehow that gets explained by the analyst. The psychoanalyst can somehow explain what takes place in every kind of parent-child constellation.

There was more to the classroom discussion of Popper's views, but the foregoing example provides a flavor for what Popper thought of psychoanalytic theory. It explains everything, but it explains everything post hoc. Professionals allied to the psychoanalytic movement have failed to make predictions (hypotheses) in advance of collecting observations. By making such advance predictions, then collecting observations, these professionals could test the truth value of the predictions. Popper (1963) underlined the idea that it is important for science that scientific theories be able to generate hypotheses that are falsifiable. By that he meant that scientists need to show that theories generate hypotheses that can be shown, in principle, to be incompatible with the observations collected in a research endeavor. In plain English, the scientist proposing a hypothesis "goes out on a limb." The scientist collects data that can be consistent with the hypothesis or contradict the hypothesis. Theories that generate hypotheses that are demonstrated to be consistent with the collected observations gain status in the scientific community. Theories

that generate hypotheses that are inconsistent with observations lose status. The problem with psychoanalytic theory, according to Popper, is that it fails to generate falsifiable, that is, testable, hypotheses at all (in Chapter 3 we return to Popper's ideas regarding hypothesis-testing, but in a different context). The American psychiatrist Aaron Beck was to our knowledge the first researcher to put psychoanalytic theory to the test.

Aaron Beck and Albert Ellis trained to become and indeed became practitioners of psychoanalysis. They both, however, evolved into apostates, rejecting psychoanalytic theories. Both men independently went on to develop cognitive therapies that contrast with psychoanalysis. Their therapies were nonetheless designed to help individuals with depression and other mental health problems. Ellis (1958), an American psychologist, broke away from psychoanalysis first. He viewed maladaptive behavior patterns as the result of irrational thinking and beliefs that have been learned. He developed a psychotherapy called rational therapy that later evolved into rational emotive therapy and that further evolved into rational emotive behavior therapy. Ellis's approach to psychotherapy employs something like a Socratic dialogue and disputation to help individuals discover what is false or misleading in their thinking, correct that thinking, and thereby get clients to lead happier, more productive lives. Ellis's approach influenced later cognitively oriented therapies for depression and other mental health problems (see Chapter 5).

Like Ellis, Beck was a psychotherapist. Beck, however, differed from Ellis in an important way. Beck was also a researcher. He and his colleague Marvin Hurvich (1959) conducted a study in which the dreams of depressed and nondepressed patients were transcribed and evaluated by two raters. The authors hypothesized that, compared to the dreams of the nondepressed, the dreams of depressed patients would be characterized by disappointment, sorrow, loneliness, and rejection. Beck also conducted a follow-up study with a larger sample, again finding similar results (Beck & Ward, 1961). But Beck and his colleagues made an easy prediction, namely, that, compared to the nondepressed, depressed individuals would report more depressive features in their dreams. The dreams in the depressed samples were consistent with what depressed individuals experience in their waking thoughts and feelings.

The test of psychoanalytic theory should be more nuanced. To understand that nuance, we should turn to the theory's axioms bearing on dreams.

If we restrict ourselves to the minimum of new knowledge which has been established with certainty, we can still say this of dreams: they have proved that *what is suppressed continues to exist in normal people as well as abnormal, and remains capable of psychical functioning*. Dreams themselves are among the manifestations of this suppressed material. . . . (Freud, 1953–1974/1899, p. 608).

According to Freudian theory, during waking life an unconscious mental censor protects against id-related unconscious content, such as unacceptable sexual and aggressive thoughts and feelings, entering a person's consciousness. During sleep, however, the censor relaxes somewhat, allowing the unacceptable content to emerge, slightly disguised by “dream work,” and that content plays a part in dreams. If dreams are the king's highway to the unconscious, then dreams should, consistent with psychoanalytic theory, reveal content that underscores anger or hostility directed at a loved one or another important figure in a person's life. An expression of that anger would ordinarily be deemed unacceptable in everyday family and social life. A Popperian hypothesis that would follow from psychoanalytic theory would be that, compared to the dreams of the nondepressed, the dreams of depressed individuals would show more other-directed angry and hostile imagery, which was not what Beck found.

Beck (personal communication to ISS, 2014) reported that he and his colleague Hurvich unexpectedly found *less* evidence of hostile content in the dreams of the depressed patients than in the dreams of the nondepressed patients, contradicting the abovementioned Popperian hypothesis that would follow from psychoanalytic theory, a hypothesis they did not include in their 1959 and, for that matter, Beck and Ward's 1961 paper. The papers were published before Beck's transition away from psychoanalytic orthodoxy. Beck's research and clinical observations led to his break with psychoanalytic orthodoxy. Beck went on to develop a cognitively oriented therapy designed to help individuals suffering from depression (Beck, 2019), a therapy we describe in Chapter 5.

Helplessness and Hopelessness

The purpose of the learned helplessness theory of depression, developed by psychologist Martin E. Seligman and behavioral neuroscientist Steven F. Maier, was to explain how individuals may come to regard themselves as powerless and become passive in the face of adversity (for reviews, see Seligman, 1975, and Peterson et al., 1993). The theory emerged from a series of experiments conducted in the 1960s (e.g., Overmier & Seligman, 1967; Seligman & Maier, 1967). The experiments showed that dogs that experienced uncontrollable and inescapable electric shocks did not attempt to escape even when given the opportunity to do so. They had learned to be helpless.

Seligman and Maier extrapolated these findings, suggesting that similar processes could occur in humans. According to their theory, when individuals repeatedly face negative situations that they perceive as beyond their control, they may learn that they have no agency over their environment. This belief in the inability to change or influence outcomes can lead to a state of passivity and depression.

The learned helplessness theory emphasizes the importance of three factors:

- Contingency: the perceived lack of relationship between one's behavior and the outcome of events.
- Cognition: the development of a belief that outcomes are uncontrollable.
- Behavior: a resulting passive or depressive response due to the belief that one cannot influence outcomes.

Learned helplessness theory has been influential in understanding cognitive aspects of depression, underlining how beliefs about control and efficacy can influence health (e.g., De Raedt & Hooley, 2016; Grahek et al., 2019; Moscarello & Hartley, 2017). The theory led to further research into cognitive-behavioral mechanisms and therapies designed to counteract these learned beliefs by fostering a sense of control and efficacy in individuals.

The original learned helplessness theory underwent significant refinement over the years, leading to a more nuanced understanding of how learned helplessness relates to depression and other psychological conditions (e.g., anxiety). Key developments in this evolution included

an “attributional reformulation” (Abramson et al., 1978). Building on the initial theory, so-called *attributional styles* were introduced as an important factor in the learned helplessness model. This reformulation proposed that the way individuals explain the causes of negative events—whether they assess these causes as internal or external to themselves, stable or unstable, and global (pan-situational) or situation specific—influences their likelihood of developing learned helplessness. People who habitually interpret setbacks as permanent, pervasive, and due to personal failings are more likely to become depressed.

Abramson et al. (1989) revised the attributional reformulation of the theory, introducing the *hopelessness theory of depression*: “According to the hopelessness theory, a proximal sufficient cause of the symptoms of hopelessness depression is an expectation that highly desired outcomes will not occur or that highly aversive outcomes will occur coupled with an expectation that no response in one’s repertoire will change the likelihood of occurrence of these outcomes” (p. 359). Hopelessness theory refined the learned helplessness model, notably by offering a more detailed framework for understanding how cognitive factors contribute to the development of depression, emphasizing the role of pessimistic expectations about the future (Alloy et al., 2006; Liu et al., 2015). We connect to this attributional reformulation in Chapter 3 when we explore how burnout connects to a depressive cognitive style.

Early Linkages of Work to Psychological State

The Italian physician Bernardino Ramazzini (1700/1750), the father of occupational medicine, is perhaps the first person to systematically link work to disease. The first edition of his magnum opus was published in Latin as *De Morbis Artificum Diatriba* [Diseases of Workers] in 1700 and translated into English in 1750. He may have been the first writer to link specific occupations to specific illnesses. For example, he understood something about musculoskeletal disorders. He found that clerks, because of their constant sitting and bookkeeping activities, experience a great deal of fatigue including fatigue of the arm. Ramazzini found that sedentary workers are subject to lumbago. He also found that melancholia plagues scholars. He wrote, “we find, that studious Persons, though naturally of jovial merry Temper, do, in Process of Time, become

melancholy and heavy” (p. 271). Ramazzini also found that stomach-aches are prevalent in men of learning “because the animal spirits are diverted, and taken up in the intellectual Service; or these Spirits are not conveyed to the Stomach with sufficient Influx” (p. 270). “Animal spirits” were thought to be important to the smooth running of the viscera but cause problems when they are diverted to the brain. He also suggested that fatigue falls heavily on scholars.

The Scottish philosopher and economist Adam Smith makes a surprise visit to these pages. He was the author of *An Inquiry into the Nature and Causes of the Wealth of Nations*, which is also known by its shorter title *The Wealth of Nations*, a text that builds a case for a laissez-faire economy, free markets, and the division of labor. Published in 1776, at the dawn of the Industrial Revolution, Smith describes a pin factory in two different circumstances. In one, each worker in the factory builds an entire pin from scratch. In the other circumstance, wealth-building economies of scale are obtained with a division of labor. In that latter case one worker repeatedly makes one facet of the pin (e.g., “draws out the wire”) and passes the work to the next worker, who performs the next facet of pin-making (e.g., straightens the wire), and so on until a final worker places the head on the pin. Smith describes 18 distinct operations. Each worker repeats over and over the operation with which the worker was tasked.

Smith understood that having workers repetitively engage in simple tasks all day would have a deleterious effect on the workers’ psychological functioning (Schonfeld & Chang, 2017). He wrote that the laborer “generally becomes as stupid and ignorant as it is possible for a human creature. The torpor of his mind renders him, not only incapable of relishing or bearing a part in any rational conversation, but of conceiving any generous, noble, or tender sentiment” (p. 303, Vol. 2). While Smith did not use the term “melancholia,” the mental torpor of mind to which he referred is either melancholia or a condition that is adjacent to melancholia. Although Smith was generally opposed to governments interfering with the economy (except in the case of government regulation of banks), he wrote that this is the fate of “the laboring poor, that is, the great body of the people . . . unless government takes some pains to prevent it” (p. 303, Vol. 2).

Not surprisingly, we turn to Smith’s antithesis, Karl Marx. He was concerned with the impact of industrialization on the industrial worker.

Marx (1967/1844) advanced the concept of alienation, which has important implications for psychology. The concept has several different but related senses. In one sense, alienation refers to industrial workers losing the ability to direct their own lives, including their labor. In another sense, alienation can mean that a worker can feel estranged from fellow workers and other human beings. A worker has become a commodity, an easily replaceable and salable part of a giant industrial flywheel. In yet another sense of the term “alienation,” industrial workers, unlike the artisans they replaced, are distanced from the goods they create.

Marx, like Smith, underscored the psychological impact of the brutalization of the division of labor in industrial production. Work, Marx noted, is drained of intrinsic satisfaction. With increasing exertion, monotony, and repetitiveness, “the poorer [the worker] and his inner world become.” Although neither Marx nor Smith used the term “melancholia,” we find an echo of that concept in their writing about the psychological impact of industrialization and the division of labor on the worker.

In his book *The Division of Labor in Society*, Émile Durkheim (1984/1893), the French sociologist, underlined the benefits of the division of labor. For example, the division of labor, he argued, creates mutual dependency and social integration. He also observed that industries, because they expand markets beyond local areas, can become national or even international in scope. He wrote that because consumers are dispersed, the producer “can no longer figure out to himself [the market’s] limits” with production lacking “any check or regulation,” which can lead to miscalculations—upward or downward—of the size of demand (p. 305). This condition results in “crises that periodically disturb economic functions” (p. 305). Durkheim called the lack of regulation in the economy “anomie.” He argued against unfettered capitalism and supported the idea of government regulation of the economy to mitigate the deleterious effects of wild swings in the economy.

The idea of anomie has implications for human psychology. Durkheim (1951/1912) later connected anomie to the risk of suicide. Using official data collected in several European countries, he found that the number of suicides increases during both downward *and* upward swings in the business cycle. He advanced the view that poverty per se does not increase the rate of suicide.

If therefore industrial or financial crises increase suicides, this is not because they cause poverty, since crises of prosperity have the same result; it is because they are crises, that is, disturbances of the collective order. Every disturbance of equilibrium, even though it achieves greater comfort and a heightening of general vitality, is an impulse to voluntary death. (p. 246)

Psychiatry has long linked suicidal ideation, attempted suicide, and completed suicide to depression. What, according to Durkheim, is occurring at the level of the individual that leads to suicide? In Durkheim's view, individuals who have attained wealth may develop an "inextinguishable thirst" for more and more pleasures and drive themselves into a "state of perpetual unhappiness." By contrast, "[i]n the case of economic disasters," something else comes into play, according to Durkheim. Life has dramatically changed. The individual experiences a need to repress wants and engage in an unaccustomed level of self-control. This "reduced existence" is intolerable.

The experience of modern industrial workers is important to look at. The Ford assembly line could be a case study. With the refinement of the assembly line at Ford plants in Michigan, an underside emerged, namely, that of the "increasing dehumanization of workers" (Wallace, 2003). Wallace described the plants as run like a "totalitarian state in miniature." There was even an organization in the plant, one with the anodyne name of "Service Department," that line workers nicknamed "Ford's Gestapo." It was staffed by ex-convicts, former prizefighters, and informants. Workers who aroused suspicions that they were unhappy with Ford were beaten by Service Department thugs. Ford workers despised, and feared, the Service Department (Cruden, 1932). The fast-paced assembly line allowed for little time for rest or toilet breaks (Linder & Nygaard, 1998). Ford concealed the true accident rates at its plants by sending accident victims to Ford's own hospital (Cruden, 1932). Many men at the plant suspected, although an understandable exaggeration, that one man per day was killed in an industrial accident; on one of the days that Cruden worked, six men were accidentally killed—deaths at the plant would be difficult to conceal. Cruden reported that feelings of cynicism, discontent, and distress were prevalent among Ford's factory workers. During the Great Depression, many Ford workers were trapped in their jobs because alternative work was difficult to obtain.

Part of the impact of work also involves the experience of involuntary unemployment. In 1933, Marie Jahoda, one of the first women to attain renown in the male-dominated social sciences, published, along with her husband Paul F. Lazarsfeld and their colleague Hans Zeisel, a trail-blazing book entitled *Marienthal: The Sociography of an Unemployed Community* (1971/1933). The book detailed their research on the residents of a small Austrian community in which *all* adults were unemployed throughout the late 1920s and early 1930s.

Jahoda et al. found that over time community members became less and less engaged in everyday activities. The research team documented the community members' growing apathy. Between 1929 and 1931, on average the number of books residents borrowed from the town library decreased. Local membership in the leading political party declined. Life history interviews with residents revealed an aimlessness in their lives. An observer may see "heartrending outbursts of despair." The team detailed "the paralyzing effects of unemployment." Thus, Jahoda and her colleagues documented the psychological costs of involuntary unemployment, which, taking place during the Great Depression, had the look and feel of psychological depression.

The Diagnosis of Depression

To pave the way for our look at research bearing on the relation of work to depression, we consider two ways in which depression has been thought of. We begin with one of those two ways, a diagnosis. According to modern psychiatry's most favored definition of depression, which is found in the *Diagnostic and Statistical Manual (DSM-5)* of the American Psychiatric Association (APA, 2013) and its most recent revision (APA, 2022), to be diagnosed with major depressive disorder (MDD),² individuals must meet several symptom criteria. First, they must experience at least one of two symptoms nearly every day for almost two consecutive weeks. The two required symptoms are either anhedonia or depressed mood. Anhedonia refers to a person being unable to experience

² MDD is also called major depression, clinical depression, or a major depressive episode.

pleasure in everyday life—a nice cup of coffee or a walk in the park. We are not describing sexual pleasure, just the ordinary, everyday variety of pleasure that people normally enjoy. The other required symptom, depressed mood, refers to feelings of dejection, hopelessness, and/or sadness. The cloak of sadness occurs even without having suffered the loss of a loved one.

In addition, to meet the criteria for a diagnosis of MDD, an individual must show evidence of experiencing at least four of seven other symptoms. If the individual shows evidence of both anhedonia and depressed mood, the person needs to manifest at least three of the other seven symptoms to meet the criteria for a diagnosis.

These seven other symptoms include change in appetite, disturbed sleep, psychomotor alterations, fatigue, feelings of worthlessness, cognitive impairment, and thoughts of death. Except for thoughts of death, the other symptoms must be experienced almost every day over the same two-week period anhedonia and/or depressed mood are experienced.

Change in appetite refers to a dietary change that leads to dramatic weight loss or gain. Disturbed sleep refers to difficulty falling or staying asleep (insomnia). Disturbed sleep could also refer to sleeping way too much (hypersomnia). Psychomotor alterations include psychomotor agitation, restlessness, or fidgeting. They could also reflect the opposite, in which the affected individual moves unusually slowly (psychomotor retardation). The fatigue symptom involves the loss of energy or feeling tired much of the time. Feelings of worthlessness include feeling like a failure, having let down members of one's family, or experiencing excessive and inappropriate guilt. Cognitive impairment refers to the individual experiencing difficulties thinking, concentrating, or paying attention. The individual may also be unusually indecisive.

The seventh symptom involves “recurrent thoughts of death” (APA, 2013). These thoughts can reflect the affected person's idea of being better off if no longer alive. Individuals are considered positive for this symptom regardless of having a specific plan to end their lives or attempt suicide. This last symptom counts toward a diagnosis of MDD even if it is experienced for a day or two during the two-week period. The symptom is too important to ignore.

There is one additional criterion for a diagnosis. The symptoms must have caused “clinically significant distress or impairment in social, occupational, or other important areas of functioning” (APA, 2013).

Finally, the pattern of symptoms cannot be attributed to the effects of substance use or a medical condition. The *DSM-5*'s approach to diagnosing major depression comports with the diagnostic criteria in the latest edition of the *International Classification of Diseases* (<https://icd.who.int/browse/2024-01/mms/en#1563440232>; World Health Organization, 2024).

A diagnosis of depression or any other mental disorder is made by way of a clinical interview. Sometimes a person close to the individual could provide background information to the clinician. The *DSM-5* observes that “[o]ften insomnia or fatigue is the presenting complaint, and a failure to probe for accompanying depressive symptoms will result in underdiagnosis” (p. 162). Although not part of the diagnosis, other symptoms, including anxiety, complaints about physical health, irritability, and tearfulness, often co-occur with depression (American Psychiatric Association, 2013; World Health Organization, 2024). Depression is also associated with higher risk of mortality from illness or accident. A diagnosis can be further refined using specifiers, for example, major depressive disorder with atypical features. The latter specifier “atypical” does not refer to a low prevalence. Rather it means that among these depressed individuals, several other patterns emerge including a mood that can occasionally brighten, a significant increase in appetite or weight, hypersomnia, a sensitivity to interpersonal rejection that impairs social relations, and leaden feelings in the limbs.

Another Way to Think About Depression

The philosopher of science Carl Hempel (1961) noted that in the sciences categorical classification often gives way to a dimensional approach to phenomena. Besides the diagnostic conceptualization of depression, there is another way to conceptualize it, a dimensional way. Instead of regarding depression as an entity that is either present or absent, one can also conceptualize depression as a continuum, like temperature. To meet criteria for a diagnosis, an individual must manifest at least five symptoms for specified durations. But what about an individual who manifests fewer than the five symptoms or experiences those symptoms but less often than nearly every day during a two-week period? That individual does not meet criteria for a diagnosis. Nevertheless, something is going

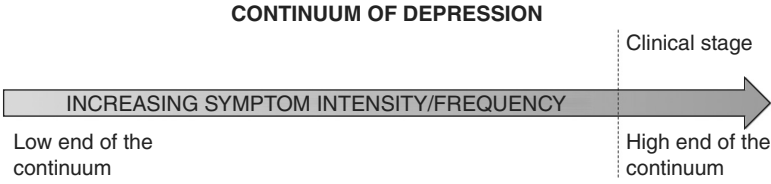


Figure 1.1 The relation of depression as a continuum to depression as a diagnosis.

wrong for the individual. But what? If we think of depression as a continuum, we have a better idea of the “what.”

Using a general population sample, Dohrenwend et al. (1980) found that several psychological symptom scales were “strongly intercorrelated relative to their reliabilities.”³ These scales assessed such symptoms as dread, sadness, hopelessness, poor self-esteem, guilt, enervation, confused thinking (e.g., trouble concentrating), and anxiety. Moreover, the symptoms each scale assesses are, except for anxiety, markers of depression. Anxiety, however, is part of that underlying dimension and is a condition that is associated with depression (APA, 2013). Later studies at the frontiers of research on psychopathology have provided evidence that depression *can* be conceptualized as a continuum (Haslam et al., 2012; Kotov et al., 2017; Liu, 2016; Pickles & Angold, 2003). The research, moreover, suggests that depression and anxiety are part of the same continuum of internalizing psychopathology⁴ (Schonfeld et al., 2019). Individuals who manifest very high scores on psychological symptom scales that reflect that continuum are likely to meet criteria for a diagnosis of MDD. But people with intermediate positions on the continuum also suffer their own commensurate measure of psychological distress.⁵ Figure 1.1 illustrates the relation between depression as a continuous factor and depression as a clinical diagnosis.

3 The reliability mentioned here reflects the extent to which the kernel of a scale, as opposed to the measurement error a scale score also carries, reflects something dependable. A scale’s reliability coefficient sets an upper limit on a correlation coefficient. See Chapter 2 for further discussion of reliability.

4 There is also an externalizing dimension of psychopathology that includes aggressive behavior, risky sexual behavior, and so forth (Haslam et al., 2012).

5 Some authors regard MDD and depressive symptoms measured as a continuous factor to be different entities (Kendler & Aggen, 2023).

Assessing Depression in the Research Context

We have established that researchers treat depression in either of two ways. The first is that of a diagnosis that is present or absent. A diagnosis is ascertained in a research context with the help of a clinical interview, often a structured interview with some limited ways for the interviewer to diverge from the interview text when a research participant's answers are unclear. Several semistructured diagnostic interviews are available for research purposes; three of the most prominent are the Schedule of Affective Disorders and Schizophrenia or SADS (Endicott & Spitzer, 1978; Fawcett, 2013); the Structured Clinical Interview for *DSM-III* or SCID (Riskind et al., 1987), and the Composite International Diagnostic Interview or CIDI (World Health Organization, 1990). We also underline that the interviews have been subject to revisions over time or have variants that address current disorders or disorders that have occurred over a lifetime.

The second way to treat depression is as a continuous dimension. In this case, the number of assessment instruments grows. We can employ psychological symptom scales that can be completed either as interviews or questionnaires. Among these is an alphabet soup of assessment instruments including the BDI, CES-D, DASS, GHQ, HADS, HRSD, HSCL, MMPI-D, ODI, PHQ-9, POMS, SCL-90-R, and so forth. Each assesses depression on a continuum either as a stand-alone scale (like the PHQ-9) or as part of a package that includes one or more other psychological symptom scales (like the DASS). Researchers have conducted psychometric research to validate each scale. Validating a scale refers to adducing evidence sufficient to show that the scale measures the target construct, in this case depression as a continuous factor. Although clinical interviews have been used in research on the impact of adverse working conditions, depressive symptom scales have been employed more often in such research.

"The Stress of Life"

Research on life stress has provided a foundation for research on the social origins of depression. Building on research conducted by Walter B. Cannon 30 and 40 years earlier, Hans Selye, in 1956, published a

book entitled *The Stress of Life* and published a revised edition in 1976. Selye's research, which was concerned with how an organism responds physiologically to environmental stimuli, particularly stimuli most of us would term "stressful," had implications for depression research to come. He pioneered research on the hypothalamic–pituitary–adrenal (HPA), or stress, axis (see Schonfeld & Chang, 2017). Although Selye was not specifically interested in job-related stress, his research, nonetheless, influenced investigators who were interested in studying job stress.

In 1957, Norman G. Hawkins, Roberts Davies, and Thomas H. Holmes, in a paper they published in a tuberculosis and pulmonary disease journal, noted that "[f]or twenty-five hundred years or more some physicians have believed that tuberculosis was frequently initiated by unhappy or stressful experiences" (p. 768). In 1964, Richard H. Rahe, Holmes, and their colleagues published a paper in which they carried out research that employed Hawkins et al.'s measure of social stressors. Although not a flawless study from a methodological standpoint (e.g., recall bias), Rahe and colleagues found that social stressors preceded the diagnosis of tuberculosis, cardiac disease, dermatologic disease, and inguinal hernia and antedated unwed pregnancies.

Several methodologically stronger studies (Friedman et al., 1958; Kasl & Cobb, 1970; Levi, 1972; Cobb & Rose, 1973) demonstrated that we can investigate the impact of job stressors on health. Meyer Friedman, Ray Rosenman, and Vernice Carroll found that, independent of risk factors such as diet and exercise, severe job stress in the form of highly excessive workloads adversely affected serum cholesterol levels and blood clotting time in male accountants. Stanislav Kasl and Sydney Cobb linked involuntary unemployment in male factory workers to elevations in blood pressure. Lennart Levi showed that a change from hourly wages to piecework was followed by increases in adrenaline and noradrenaline in women invoicing clerks; a reversion to hourly wages was attended by a decline. In another all-male sample, Sydney Cobb and Robert Rose linked the intensity of air traffic to hypertension and peptic ulcer in air traffic controllers.

A challenge that emerged was to show that methodologically strong research could demonstrate a link between working conditions and psychological symptoms. Katharine Parkes (1982) met that challenge.

Capitalizing on a natural experiment, Parkes showed that compared to rotations into surgical wards, when student nurses rotated into medical wards, which put “greater affective demands” on them, they manifested higher levels of depressive symptoms.

Perhaps the first person to use a phrase that is a variant of the term “occupational depression” was Maureen Oswin (1978). Oswin, a British nurse, identified a phenomenon she called “professional depression.” Although these were uncontrolled, qualitative observations, Oswin convincingly linked professional depression to adverse working conditions at an understaffed, long-term-care hospital that served severely handicapped children. She reported on working conditions that included a lack of support from the hospital administration, meals arriving late from the central kitchen, crises during feeding, and having to deal with children who were difficult to feed. The work was complicated by a continuing shortage of nurses that was sustained by the stressfulness of the job. Many years later, the authors of this book developed a measure of occupational depression entitled the Occupational Depression Inventory (ODI; Bianchi & Schonfeld, 2020; Schonfeld & Bianchi, 2022). The ODI is described in some detail in Chapter 3 and is included in the Appendix.

Stressful Life Events

The foundation of research on stressful job conditions, conditions that could potentially lead to clinical depression or elevations in depressive symptoms, derives from research aimed at identifying the stressful conditions of life in general. The year 1967 is a reasonably good starting point for this disquisition. During that year, Thomas H. Holmes and Richard H. Rahe, both psychiatrists, built on their earlier work (Rahe et al., 1964) and published a paper describing their new Social Readjustment Rating Scale (SRRS). The SRRS could be administered by interview or questionnaire. Researchers can use the instrument to ascertain whether an individual experienced any of 43 life events. The events were derived from clinical experience and reflect the influence of Adolf Meyer on Holmes and Rahe. Meyer advanced the goal of helping psychiatrists better understand an individual’s life circumstances. The events on

the SRRS included marriage, vacation, divorce, getting fired, death of a loved one, and so forth and how much of a life adjustment an individual would, on average, make upon experiencing the event. Aside from estimating how much readjustment each event requires, some of the events in question are ordinarily positive and others, stressful.

The paper by Holmes and Rahe proved to be influential. In 2006, Bruce P. Dohrenwend wrote that following the publication of their SRRS, “a tremendous increase has occurred in the construction of such measures and in quantitative research on relations between inventoried life events and health” (p. 477). By January 2024, the paper was cited more than 7,700 times.

Influenced by Holmes and Rahe, other investigators began to create measures to assess stressful life events (SLEs). Some of those investigators examined SLEs’ link to depression, as a diagnosis. Others examined SLEs’ link to depressive symptoms, as assessed by psychological symptom scales. For example, Barbara Snell Dohrenwend (1973) linked SLEs to a composite measure of psychosomatic, anxiety, and depressive symptoms. She also linked the risk of exposure to SLEs to lower social statuses.

George W. Brown and Tirril Harris (1978), in their groundbreaking book, *The Social Origins of Depression*, concerned themselves with social factors that increase the risk of clinical depression, as diagnosed by clinical interview. In a study of women who lived in the Camberwell borough of London, Brown and Harris examined the link between depression and SLEs, but only SLEs that likely occurred independently of the women’s personalities. An example of such an event is the woman’s husband getting unexpectedly laid off. The question of SLE independence was important because the authors were concerned about the confounding of event, personality, and mental disorder. They excluded events that a research participant, by virtue of her personality, could arguably have contributed to.

Brown and Harris also extended the concept of SLEs to the idea of “difficulties,” which represented disagreeable conditions that lasted four or more weeks (e.g., a damp apartment or a son who uses illicit drugs). They, surprisingly, did not find that SLEs or difficulties alone predicted clinical depression. The authors, however, did find that an SLE or difficulty occurring in combination with what they termed a “vulnerability factor” increased the risk of depression. An example of a vulnerability

factor⁶ is the absence of a *confidant*, that is, another person—a husband or a friend—with whom the woman had a close and confiding relationship, someone in whom the woman could tell anything about herself without feeling embarrassed. Another vulnerability is lower social class with its attendant woes, such as meager income or substandard housing. Interestingly, the vulnerability factors on their own did not lead to an episode of depression; however, they served as the ground on which an SLE or difficulty landed, and the combination of an SLE or difficulty with a vulnerability increased the risk of clinical depression.

An SLE and a vulnerability factor can be termed risk factors, in this case risk factors for depression. The risk factors had a combined impact on depression that was different from—in this case greater than—the sum of the singular impacts of each factor. In research parlance, a combined effect of the impact of which is greater than the sum of the individual effects of each factor—a kind of synergy of misfortune—represents an interaction. The idea of an interaction also emerges in the demand–control model of the impact of working conditions on the health of workers, a model we discuss later in this chapter.

In addition to studies linking SLEs to depression as a diagnosis, other research that grew out of Holmes and Rahe's initial work linked SLEs to elevations in depressive symptoms. For example, Monroe et al. (1986), in a study of married Pennsylvania women having low depressive symptom levels at baseline, found that SLEs and reduced social support at baseline predicted depressive symptoms one year later. Importantly, the investigators statistically controlled for baseline

6 Brown, a sociologist, and Harris, a psychoanalytically oriented psychotherapist, also included among the vulnerability factors, early loss of a woman's mother. Given their combined interest in Freud, John Bowlby, and Aaron Beck, they developed a theory about the impact of an SLE on a woman who had suffered maternal loss at age 11 or younger, the psychological residua of that loss creating a base with which the adult experience of an SLE would resonate to provoke an episode of depression. They, however, revised their theory given later evidence they collected. In a study of the impact of SLEs in women who experienced early maternal loss (through death or separation), they (Harris et al., 1986) found that if the quality of care following the loss (e.g., the father pulled himself together to take good care of his daughter), the woman was no more vulnerable to a depressive episode following an SLE than a woman who did not experience such a loss and but later was subject to an SLE. Harris et al. found that inadequacy of care following early maternal loss is the vulnerability factor, not the loss per se.

symptoms in view of the potential for any confounding of the reporting of SLEs and symptoms.

Studies of general life stress have provided a backdrop for research on the relation of working conditions to heightened depressive symptom levels. Working conditions can be episodic like SLEs. Or they can be ongoing, like Brown and Harris's (1978) difficulties. Individuals exposed to episodic or ongoing stressors at work, just as individuals exposed to such stressors outside of work, can be put at risk.

An example of Holmes and Rahe's influence (by way of Dohrenwend [1979; Dohrenwend et al., 1987]) on research on working conditions comes from Schonfeld's (2001) study of job stressors affecting women who were first-year New York City public school teachers.⁷ For SLEs, Schonfeld assessed the frequencies of episodically occurring classroom stressors (e.g., a fight between students, a student threatening the teacher). He also assessed ongoing stressors (e.g., an overcrowded classroom, a vandalized classroom going unrepaired) loosely paralleling Brown and Harris's (1978) concept of difficulties. Schonfeld mixed a few positively worded items (e.g., praise from an administrator) among the stressor questions to reduce the risk of "response sets"⁸ and ensure that the teachers paid careful attention.

Because official reports of stressful school conditions have been defective (e.g., violence against teachers being woefully underreported; Bloch, 1978; Schonfeld, 1992), Schonfeld designed special self-report stressor measures instead of relying on official records. These measures were "neutral self-reports," a just-the-facts type of measure (Kasl, 1987). In many earlier studies, researchers had respondents inject their feelings into stressor frequency measures, creating an artifact that likely increased the magnitude of the correlation between the stressor scales

7 The first author was for six years a mathematics teacher in a New York City public school. During that period, he did not anticipate that one day he would conduct research on the impact of job stress on teachers. His psychology-related research interests at the time he was a teacher lay in cognitive development as he pursued a master's degree in night school. But during that period, he became aware of the considerable burden of job stress affecting many teachers in his school. The stress came in the form of assaults, threats of assault, and generally discourteous student behavior.

8 A response set is a tendency for a research participant to respond to scale items in an excessively consistent way, such as a tendency to respond to items about the occurrence of episodic events "Not at all" or "Once per week," no matter the actual frequency of the events identified in the items.

and psychological symptoms. Concerned that teacher distress could still bias the reporting of job stressors (e.g., a distressed teacher overreporting student misbehavior), Schonfeld (1996, 2001) found that the neutrally worded stressor reports obtained during the fall and spring terms were minimally confounded with depressive symptoms he assessed during the summer pre-employment period.

Consistent with his hypothesis bearing on the importance of control in the work environment, Schonfeld (2001) found that increasing exposure to episodic and ongoing stressors predicted immediate and later elevations in depressive symptoms, with no evidence of reverse causation.⁹ Schonfeld also found that the supportiveness of friends and relatives outside of work, as assessed during the pre-employment period, was related to fewer symptoms. In another test, the women were categorized as belonging to three approximately equal-sized groups: the teachers (1) most exposed to work-related stressors, (2) least exposed, and (3) with intermediate levels of stressor exposures (Schonfeld, 2000). As shown in Figure 1.2, the depressive symptom levels of the three groups were indistinguishable during the pre-employment period. After school began, the most exposed teachers manifested a sharp rise in depressive symptoms. The least exposed showed a decrease in symptoms (as if a safe, well-run work environment is therapeutic). Those with intermediate exposures remained at intermediate symptom levels.

Assessing workers' characteristics before they take a job is an important idea borrowed from epidemiology. Epidemiologic researchers are concerned about selection processes leading to wrong conclusions (Berkson, 1946). In the preceding example, there is evidence that the three groups of teachers were initially equivalent on depressive symptom levels and differed only on the working conditions they were exposed to. What if hiring managers at the Board of Education (now the Department of Education) had assigned distressed teachers to the most chaotic schools and nondistressed teachers to better run schools. If the groups of teachers were not initially equivalent on levels of depressive symptoms and the investigator had not known about the lack of initial equivalence

9 Reverse causation in this case would mean that depressive symptoms would lead to more student-related stressors, say, by way of a depressed teacher being weak at classroom management (Schonfeld & Ruan, 1991). Later in the chapter there is a more extended discussion of reverse causation.

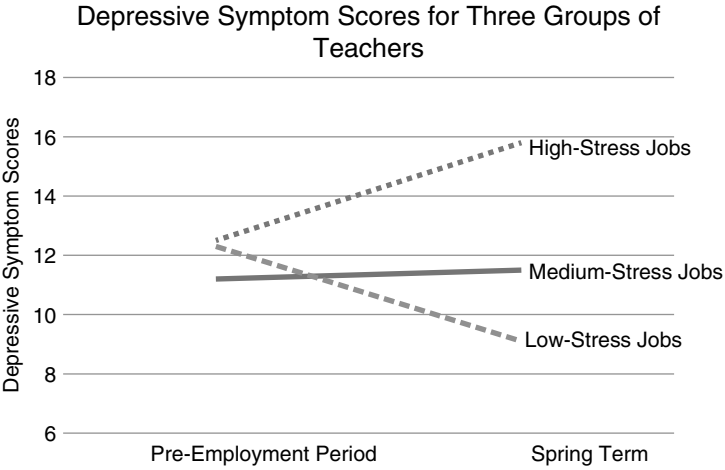


Figure 1.2 Teachers in low-, medium-, and high-stress classrooms. *Note:* The figure shows first-year women teachers' ($n = 184$) mean symptom scores on the Center for Epidemiologic Studies Depression Scale (CES-D). The sample was restricted to teachers who did not change schools between the fall and spring. The teachers were assessed before they joined the workforce (the pre-employment period) and then again during the spring term. Working conditions were assessed during the fall term. Although the pre-employment mean differences were not significant, the spring-term differences were highly significant ($p < .001$).

on the depressive-symptoms outcome variable, the investigator could have drawn conclusions based on misleading information. It is important for researchers to consider how selection processes may influence study results, including results on the impact of working conditions on depressive symptoms in teachers and other workers. Keep in mind that there are examples from the history of science that bear out the problem of selection (including research on autism¹⁰).

10 Psychiatrist Leo Kanner, who conducted pioneering research on autism, made a momentous mistake based on selection processes. He erroneously concluded that parents of autistic children are, on average, psychologically distant. But that may even have been true of the parents who brought their children to see him during the Great Depression and the beginning of World War II; it was a time when many Americans were in dire straits economically. However, mainly parents who were well-heeled enough to travel distances from their homes, disabled children in tow, could visit Kanner at Johns Hopkins University. Those observations, based on the self-selected

A lesson for longitudinal researchers who study job-related depressive symptoms is to assess and statistically control for workers' characteristics, including pre-employment depressive symptoms, when evaluating the magnitude of the link between working conditions and later symptoms. In much of the research on the impact of working conditions on depressive symptoms, we don't have, or ethically want, the ability to randomly allocate workers to more and less stressful jobs, although doing so would ensure (when using large samples) group equivalence on background characteristics. Moreover, most longitudinal research on job stress begins with workers already on the job. In that case, although not a perfect solution, it is helpful to assess key background characteristics (e.g., age, sex, initial symptom levels) along with working conditions at a study's starting point (baseline) to statistically control for variation in those characteristics when assessing the relation of baseline job conditions to later depressive symptoms. Tables 1.2 and 1.3 summarize studies that bear on the relationship of working conditions to depressive disorders or symptoms. The content of those tables is limited to studies that control for baseline disorders or symptoms when linking baseline working conditions to later disorders or symptoms.

The Demand–Control (DC) Model of Job Stress

In 1979, Robert Karasek published an important paper in the journal *Administrative Science Quarterly*. Like the article by Holmes and Rahe, Karasek's set in motion a cascade of research. This new surge in research, however, concerned the relation of working conditions to what he termed "mental strain," a label that embraced a range of problems including depressive symptoms, psychophysiologic (PP) symptoms (e.g., headaches, stomachaches, and so forth), and job dissatisfaction. He identified two

parents who traveled to see him, led to many psychiatrists and psychologists to conclude, wrongly, that psychological distance in parents, particularly mothers, is a key etiological factor in the development of autism (see Schonfeld & Farrell, 2010, for a fuller description). Of course, Kanner's error may look easy to identify from the vantage point of today. However, the ease with which an investigator can commit a selection-based error should not be overlooked. It took years to figure out that psychological distance in a mother does *not* cause autism. And a great deal of personal hurt to parents was done before the psychological-distance theory was put to rest.

factors that potentially affect mental strain. One he called job demands, which refers to the size of an individual's workload. The workload Karasek referred to was psychological, not physical, workload (e.g., lifting packages). Job demands or psychological workload did not only include the quantity of work but also included the extent of job complexity and the pace of work. The other factor concerned how much autonomy or control a worker has over job-related tasks.

Karasek had several intellectual forerunners. The research conducted by Friedman et al. (1958) anticipated the baleful effect of severely excessive workloads. The idea of job stressors in terms of workload also has some debt to Holmes and Rahe (1967) and Dohrenwend and Dohrenwend (1974). Also relevant to the impact of workload is that during the period from 1969 to 1977, researchers at the Institute for Social Research at the University of Michigan conducted three Quality of Employment Surveys involving representative samples of U.S. workers (Quinn & Staines, 1979). A landmark government report on the first survey (Quinn et al., 1970), largely conducted during November and December 1969, showed that one of the "foremost" job-related problems was "demands that the worker perform excessive amounts of work or work too fast" (p. 371). In addition, there were problems of monotony at work, namely, that the worker had to "do things that are very repetitious." The authors found that the intensity of work-related demands was concurrently related to reduced job satisfaction and higher levels of depressive and PP symptoms. Parallel findings emerged for workplace monotony.

Several other predecessors of Karasek were concerned with job incumbents' autonomy or control at the workplace. In 1951, the journal *Human Relations* published a paper by Eric L. Trist and Kenneth W. Bamforth. The paper concerned changes that had taken place in the British mining industry and their impact on miners.¹¹ The changes involved an increased division of labor and reduced autonomy for miners. Before the changes in job structure, the work of British miners had been more artisanal. The changes led to increased job dissatisfaction. Social science researchers at the University of Michigan, including Quinn and colleagues (1970),

¹¹ Trist was Bamforth's mentor at the Tavistock Institute of Human Relations in London, a prestigious social science organization. Bamforth had been a miner before doing a fellowship at Tavistock. His experience represented a crack in the case-hardened British class system.

became concerned with the idea of control. Quinn et al. investigated how much “job autonomy,” or, control, workers had in their positions. The study team found that autonomy was concurrently related to lower levels of depressive and PP symptoms and greater job satisfaction. The Swedish social psychologist Bertil Gardell (1971) found that the extent to which pulp and paper workers exerted control over their work tasks was directly related to better mental health. Interest in lack of control extended to psychoanalytic thinkers concerned with depression. The American psychoanalyst Edward Bibring (1953) described depression as “a state of helplessness and powerlessness of the ego” regardless of the factors that may have caused the disorder (p. 24).

Karasek’s (1979) innovation was to take a combined look at workload and control (see Table 1.1). He used the term “decision latitude” in his model of job stress. The term is imbued with the idea of control. According to Karasek, decision latitude comprises two components (1) decision authority and (2) skill discretion. Decision authority refers to the extent to which the worker exerts control over meaningful job-related decisions. Skill discretion involves the diversity of job-related skills the worker can exercise. It also includes the extent to which opportunities are available to the worker to learn new skills. Mausner-Dorsch and Eaton (2000), however, advanced that view that, compared to skill discretion, decision authority is more important for mental health.

In Karasek’s (1979) demand–control (DC) model of job stress, high levels of job demands “place the individual in a motivated or energized state of ‘stress,’” a state that has the potential to devolve into mental strain (p. 287). However, according to the theory, decision latitude “modulates the release or transformation” of that energy into “the energy of action” (p. 287). According to the theory, there is a kind of synergy

Table 1.1 Karasek’s original demand–control model.

		Job Demands	
		Low	High
Control/ Decision Latitude	Low	Passive jobs	High-strain jobs
	High	Low-strain jobs	Active jobs

between high workload and high decision latitude that benefits the worker. Karasek labeled jobs that are characterized by high demands and high control as “active jobs.” In Karasek’s theory, individuals holding active jobs tend to have greater opportunities for accomplishment and satisfaction as well as better mental health. An example of an active job would be that of an electrical engineer, who works hard by planning the circuitry for a new building. In an active job, the arousal precipitated by a job’s demands, given the worker’s control over the tasks at hand, becomes energy directed at problem-solving and goal attainment, with little arousal remaining that could become psychological strain (Karasek & Theorell, 1990).

However, for workers who face high workloads, but have little control/decision latitude, the theory holds that the worker will be at increased risk for “job strain.” Jobs with high workloads and little latitude have been labeled “high-strain” jobs. An example of such a job is a worker on an assembly line. In the DC model, workers holding jobs having high workloads but with little latitude to modulate the workload’s impact experience elevated levels of arousal, which gets “transformed into damaging, unused residual strain” (p. 33; Karasek & Theorell, 1990).

Apropos of high-strain jobs, it could be helpful to consider what is happening from a biological standpoint. High levels of psychosocial stressors exert effects on mental health through both the central and the autonomic nervous systems. For example, a situation in which a worker has little decision latitude when faced with high workloads or excessive job-related interpersonal conflict can challenge those systems, leading to effects such as greater catecholamine and cortisol release. Schonfeld and Chang (2017) wrote, “Stressors are interpreted by the hippocampus and amygdala and, when chronically occurring (as in high-strain jobs), can create an imbalance in allostatic (i.e., adaptive) systems such as the hypothalamic–pituitary–adrenal, or stress, axis” (p. 79). Elevated allostatic loads can have an adverse effect on mental health *and* contribute to heart disease risk by way of increased blood pressure and atherogenesis¹² (McEwen, 2004; Peters et al., 2017).

¹² Atherogenesis refers to the forming of plaques on the innermost layer of artery walls.

To put the DC model in perspective, consider jobs characterized by high workloads combined with (1) high levels of decision latitude versus (2) low levels of decision latitude. Workers employed in high-workload, high-latitude jobs are likely to manifest fewer depressive and PP symptoms as well as greater job satisfaction. By contrast, workers employed in high-workload, low-latitude jobs are likely to manifest more depressive and PP symptoms as well as reduced job satisfaction. Thus, in the DC model, jobs that combine high workloads with high decision latitude have beneficial effects; by contrast, jobs that combine high workloads with low decision latitude have detrimental effects.

Karasek (1979) termed the type of job that combines low workload with little decision latitude a “passive job.” An example of a passive job would be a watchman in a warehouse. Passive jobs, according to Karasek, lead to helplessness and, not surprisingly, passivity. The job that combines low workload with high levels of decision latitude would be termed a low-strain job. Karasek and Theorell (1990) mordantly called that type of job, if it exists, a “low-stress utopia.” The authors, nonetheless, identified some occupations, based on three U.S. national surveys, as low-strain jobs. Examples they provided include lineman and natural scientist (although it would be interesting to learn how those job incumbents would respond to the low-stress/low-strain label).

One point needs to be underscored regarding Karasek’s job categories. Higher socioeconomic status tends to be related to greater decision latitude and lower risk of an unfavorable effort–reward imbalance (Hoven et al., 2015; see a later section). Job categories are largely stratified along socioeconomic lines, for example, blue collar, white collar, highly educated professional, and upper management, which makes research on unraveling the impact of working conditions on depressive symptoms and disorders challenging. Are the job conditions affecting depressive symptoms in workers? Or is the socioeconomic status of job incumbents, with its attendant benefits and problems (e.g., housing conditions), affecting symptom levels? Or some combination of the two? Moreover, we need to be concerned about the influence of individuals’ mental health affecting (1) the jobs they self-select, (2) hiring managers’ job assignments or placement decisions, and/or (3) biases in the job incumbents’ descriptions of their working conditions.

A New Development: The Demand–Control–Support (DCS) Model of Job Stress

Karasek and his colleagues (1981) extended the DC model to apply to the development of cardiovascular disease (CVD). A few years later, in connection to research on CVD, Jeffrey V. Johnson, Ellen M. Hall, and Töres Theorell (1989), when studying risk factors for CVD, introduced another dimension to the DC model, namely, social isolation, which gave rise to the “iso-strain” or demand–control–support (DCS) model. The expanded model fueled additional research.

Johnson, Hall, and Theorell’s (1989) addition of social connections to the DC model has been important. Social connections already had a long history in the annals of research. The research on the health effects of social connections dates to Émile Durkheim (1951/1912), who, as mentioned earlier, linked suicide risk to upward and downward turns in the business cycle. Durkheim also found elevated suicide risk in adults who are not married. In Durkheimian thought, marriage is reflective of greater social integration. The line of research on the health-giving effects of social relationships was rediscovered and expanded with Gove (1973), Berkman and Syme (1979), and Cohen and Wills (1985). Much of the research concerned the protectiveness of family, community, and friendship ties, making it reasonable to hypothesize that socially supportive relationships with coworkers and managers are beneficial to the mental and physical health of workers. Why would supportive social relationships exert beneficial effects on mental health? At least two theoretical ideas have emerged. Supportive social relationships (1) encourage an individual, either explicitly or tacitly, to engage in adaptive health-related behaviors (e.g., encourage individuals not to smoke or give up smoking) and/or (2) have an evolutionary basis for exerting a healthful influence on the neuroendocrine axis, especially in the context of responding to stressors in the workplace and elsewhere (Schonfeld & Chang, 2017).

Moreover, research on support distinguishes between perceived and received support (Cohen & Wills, 1985; Schonfeld, 1991). Generally, the perceived availability of current support is more important to the individual’s well-being. Perceived support is the pre-existing context that is available to a worker when confronted by job-related stressors. Received

support tends to be confounded with prior need for support owing to previous encounters with stressful situations. In a study of new teachers, Schonfeld (2001) showed that perceived support was related to reduced levels of depressive symptoms.

The research on the DCS factors has only occasionally revealed a Karasek type of demand–control interaction. A triple interaction involving demands, control, and support is exceedingly rare. Research has tended to find that the three factors are more likely to have an additive rather than an interactive, that is, synergistic, impact on depressive symptoms. Table 1.2 bears on the impact of the DCS factors but is restricted to evidence abstracted from high-quality longitudinal studies, namely, studies that rigorously examined the impact of earlier working conditions (assessed at “Time t ” or baseline) on later depressive symptoms or disorders (assessed at “Time $t + 1$ ”) while controlling for Time t depressive symptoms or disorders. We excluded studies that failed to control for baseline depressive conditions. We also excluded studies that examined the relation of (1) change in working conditions from baseline to Time $t + 1$ to (2) change in depressive symptoms or disorders from baseline to Time $t + 1$ because such studies do not establish the temporal priority of the putative cause, working conditions, over the hypothesized effect, depressive conditions. The table demonstrates that generally workload, control, and support have an impact on depressive symptoms and disorders in the expected directions, although not perfectly consistently.

Reverse Causality

Barbara Snell Dohrenwend and Bruce Dohrenwend (1981) developed several hypothetical models of how stressful life events (SLEs) are related to changes in health symptoms. One of those models was the event proneness model, in which individuals experiencing high levels of health symptoms provoke the occurrence of SLEs. The Dohrenwends also included in the event proneness model the idea that while symptoms may provoke SLEs, those SLEs can, in turn, provoke more symptoms. The model reflects a causal relationship that is the reverse of the ordinarily expected idea that stressors provoke symptoms.

Table 1.2 High-quality longitudinal studies that bear on the relation of DCS workplace factors to depression, anxiety, and psychological distress. Parts of this table are from Schonfeld and Chang's (2017) / Springer Publishing Company, *Occupational Health Psychology: Work, Stress, and Health* by permission of the Springer Publishing Company. A key to the abbreviations is on bottom of the table.

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Åhlin et al. (2019)	Sweden	3,947 (58% ♀)	Series of 2-yr. lags over 8 yrs.	Demographic factors + each individual as own control	Effort (demands) → ↑ SCL depression Control, support ↔ depression	No evidence of reverse causality. 2-yr. lag may be too long to detect some effects.
Bromet et al. (1988)	U.S.	325 ♂s working in power plant	1 yr.	Baseline disorder	ψWL but not DL → affective disorder by SADS and SCL-90; Unanticipated ψWL x DL interaction.	Used the GSI score from the SCL-90. Workers with less DL and greater ψWL, surprisingly, had fewer symptoms than workers with “active jobs.” IVs treated as continuous variables.

Bültmann et al. (2002)	Netherlands	> 8,800 individuals (26% ♀)	1 yr.	Age, educ., marital status, GHQ	In ♂s, high ψ WL and emotional demands, low supervisor and coworker support, and high conflict with coworkers → ↑ GHQ but DL not significant; In ♀s, high ψ WL and emotional demands → ↑ GHQ but DL not significant	Dichotomized GHQ. Trichotomized ψ WL, demands, DL. Dichotomized support.
Burr et al. (2021)	Germany	2,212 workers; 1/2 ♀	5 yrs.	Age, gender, SES, baseline symptoms	Additive effects for WL and control → ↑ PHQ-9 scores; No interaction.	WL, Control, PHQ-9 dichotomized. Did not control for workers who changed jobs.
Clays et al. (2007)	Belgium	> 2,800 workers in variety of sectors (69% ♂)	6 1/2 yrs.	Baseline CES-D	Combination of high ψ WL, low DL, and low support → incidence of ↑ CES-D scores in ♂s ψ WL, DL, and low support do not → CES-D scores in ♂s	Long follow-up likely attenuated effects. CES-D treated dichotomously. But individuals above CES-D cutoff at baseline were excluded from analyses.

(Continued)

Table 1.2 (Continued)

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
d'Errio et al. (2011)	Italy	2,046 unionized BC (80%) and WC workers (23% ♀)	5 yrs.	Age, sex	High WL → ↑ antidepressant meds prescriptions in BC workers but lowered risk in WC workers. No effect for control; No DC interaction	Excluded workers taking antidepressants at baseline. Antidepressant meds are proxy for depression. No control for job change over 5 yrs.
De Lange et al. (2004)	Netherlands	668 workers (69% ♂) The SMASH study	4 waves of data collection of 4 yrs.	Age, gender, and DV assessed in the previous wave	ψWL affected → ↑ CES-D and EE (MBI); Control → ↑ job satisfaction; Supervisor support → ↓ EE Job satisfaction → ↑ supervisor support EE → ↓ supervisor support EE → ↑ ψWL	Predictor variables and DVs were treated as continuous factors. Used structural equation modeling that controlled for measurement error. Also found significant but weaker reverse causal effects for EE.

Fandiño-Losada et al. (2013)	Sweden	4,427 workers (55% ♀) The PART Study	3 yrs.	Sociodemo., time 1 depressive symptoms	Prospective study: excluded workers with time 1 depressive dx. Poor work support → ↑ depression in ♀s ψWL, SkD, DA ↔ depression in ♀s DA ↔ depression in ♂s ψWL, low SkD → ↓ depression in ♂s	Dx was probable depression based on standardized questionnaire. In ♂s, finding that high ψWL and low SkD → lower depression risk was unexpected. Among ♂s, number of cases at time 2 was low ($n = 31$; 1.5% of ♂s).
Finne et al. (2014)	Norway	3,212 workers from 48 public and private organizations (61% ♀)	2 yrs.	Age, sex, skill level Workers with high baseline symptoms excluded	Role conflict → ↑ symptoms Support from supervisor → ↓ symptoms Fair leadership → ↓ symptoms Control → ↓ symptoms only when average of baseline and follow-up used. No effect for WL	10-item symptom measure combined depressive and anxiety symptoms. Only used dichotomized (high–low) version of the symptom measure

(Continued)

Table 1.2 (Continued)

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Juvani et al., (2018)	Finland	42,862 (80% ♀) workers from 10 towns	Until disability pension, up to 4 yrs	Sociodemo., location, alc., smoking, hx mental and physical disorders	T1 high strain → ↑ disability pension and depression See Table 1.3	
Kawakami et al. (1992)	Japan	460 ♂, blue collar workers	4 times over 4 yrs.	Sociodemo., medical, Zung	Low control & problematic interpersonal relations → ↑ Zung High ψ WL not significant	Job's incompatibility with worker's skills → ↑ Zung. Zung scale was dichotomized.
Kivimäki et al. (2003a)	Finland	>5,600 mostly ♀ employees of 10 hospitals	2 yrs.	Sociodemo., health behaviors	High ψ WL and low DA → ↑ GHQ scores Relational justice → low GHQ scores	Trichotomized continuous IVs. Dichotomized GHQ.

Marchand et al. (2005)	Canada	National sample of > 6,000 workers (46% ♀) NHPS study	4 time points over 6 yrs.	Sociodemo.	Low DA → severe distress (6-item scale); ψWL and support were not significant	Dichotomized continuous DV. Survival analysis to the first episode of severe distress. IVs had low reliability (e.g., ψWL, support) and were dichotomized. Used single items to assess varieties of workload.
Mino et al. (1999)	Japan	310 machine workers (53% ♀)	2 yrs.	Sociodemo., excluded those with elevated baseline GHQ.	ψWL → ↑ GHQ	Did not control for depression at baseline. Dichotomized CES-D.
Niedhammer et al. (1998)	France	>11,500 workers (27% ♀) at national gas co.	1 yr.	Absenteeism owing to MH problems but not CES-D	High ψWL, low DL, and low work-related support → ↑ CES-D	
Niedhammer et al. (2015)	France	National sample 4,717 workers (≈ 50% ♂)	4 yrs.	Sociodemo., nonwork support and adversity, childhood adversity	ψWL → GAD not MDD Emotional demands → ↑ GAD Control ↔ GAD or MDD	Prospective study. One of few studies to look at GAD dx.

(Continued)

Table 1.2 (Continued)

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Nigatu and Wang (2018)	Alberta, Canada	2,180 (55% ♀)	4 waves, at 1-yr intervals	Sociodem., job type, history of MDD	Combo of high D/C ratio and high ERI → ↑ MDD Combo of high D/C ratio + WFC → ↑ MDD high D/C ratio alone marginally significant predictor of MDD	High D/C ratio is demand/control ratio is dichotomous. Not often used. WFC and ERI also dichotomized.
Paterniti et al. (2002)	France	>8,000 workers (75% ♂) GAZEL study	3 yrs.	Occupational grade, hostility, Type A behavior	DL, ψ WL → ↑ CES-D in ♂s ψ WL → ↑ CES-D in ♀s Work support → ↓ CES-D	IVs were measured during year 2. DV in regression was time-1–time-2 CES-D change score. Better to treat baseline CES-D as IV and time 2 CES-D as the DV.

Plaisier et al. (2007)	Netherlands	>2,600 workers (42% ♂) The NEMESIS study	2 yrs.	Sociodemo., physical health	Prospective study, excluded workers with disorder at baseline. ψWL but not DL → ↑ Depressive and anxiety disorders; No effect for job security. Combined work and nonwork support → ↓ Depressive disorders	Incidence study. Used diagnostic instrument CIDI to detect disorders. ψWL, DL, and support were continuous scales (I. Plaisier, personal communication, Nov. 11, 2014).
Rugulies et al. (2006)	Denmark	>4,100 workers (½ ♂) The DWECS study	5 yrs.	Sociodemo., health behaviors; job changing	Low supervisor support and low influence (akin to control) → ↑ depressive symptom levels in ♀s but not ♂s No effect for ψWL.	Long follow-up likely attenuated effects (although there were statistical controls for job changes). Controlled concurrent alc., often a comorbid disorder, potentially weakening effects of IVs. DV was dichotomous. Predictors either dichotomous and/or based on single item.

(Continued)

Table 1.2 (Continued)

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Shields (2006)	Canada NHPS study	>12,000 (≈50% ♂)	2 yrs.	Age, SES, personal stress, mastery	Strain → ↑ depression in ♂s Low coworker support → ↑ depression in ♀s	Incidence study. Depression dx by CIDI module.
Stansfeld et al. (1999)	U.K.	>7,900 civil servants (2/3 ♂) Whitehall II	5 yrs.	Sociodemo., NA, hostility, baseline GHQ or excluded those with elevated baseline GHQ	ψWL → ↑ GHQ Low DA → ↑ GHQ Low collective support → ↑ GHQ Low info from supervisor → ↑ GHQ SkD ⇏ GHQ	Findings held when controlling for baseline GHQ or excluding those with high baseline GHQ.

Too et al. (2021)	Australia	1,279 (50% ♂)	4 waves over 12 yrs	Sociodemo., verbal IQ, neuroticism, early common mental disorders	Cumulative exposure to poor job quality over first 3 waves → ↑ common mental disorder at wave 4.	Cumulative exposure = number of waves in which 2 or more of high demands, low control, and high insecurity occurred. Demands, control, and insecurity were dichotomized. Common mental disorders assessed by pattern of responses on the Goldberg scale
Virtanen et al. (2007)	Finland	>3,300 working adults (50% ♀)	3 yrs.	Hx psycho-pathology	High ψ WL and high ψ WL $\times$ low control → affective disorder as reflected antidepressant meds prescriptions	Depression assessed by prescription for antidepressant meds. Many depressed do not take meds, and individuals with anxiety disorders sometimes take antidepressant meds.

(Continued)

Table 1.2 (Continued)

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Wang (2004)	Canada	Same sample as Marchand et al.	4 time points over 6 yrs.	Physical health; childhood trauma; stressful life events	Low SkD, high ψ WL, low support $\rightarrow$ newly incident episode of depression	Survival model to first incidence of diagnosed depression. IVs had low reliability (e.g., ψ WL, support) and were dichotomized. Controlled contemporaneous physical health with depressive episode, likely attenuating IV-DV relation.

Note: Abbreviations: ψ WL, psychological workload or psychological demands; ϕ female; δ male; BC, blue collar; WC, white collar; DL, decision latitude; DA, decision authority; SkD, skill discretion; MH, mental health; NA, negative affectivity; Sociodemo, Sociodemographic variables; alc., Excessive alcohol use; CES-D, Center for Epidemiologic Studies Depression Scale; SCL-90, Symptom Checklist; MDD, major depressive disorder; GAD, generalized anxiety disorder; GSI, Global Severity Index of the SCL-90; SADS, Schedule for Affective Disorders and Schizophrenia; GHQ, General Health Questionnaire (a psychiatric symptom measure); Goldberg, Goldberg's Depression and Anxiety scales, subscales of the GHQ; Zung, Zung depression scale; hx, history; ERI, effort-reward imbalance; WFC, work-family conflict. Baseline DVs refer to the baseline versions of the dependent variables. IVs refer to the principal independent variables.

Mentioned earlier, Schonfeld (2001) in his study of New York City teachers found no evidence of reverse causality; the causal effect went exclusively from work stressors to depressive symptoms and not the other way around. In a study of Swedish workers, Åhlin et al. (2019) found no reverse causal effects for the impact of depressive symptoms on control, workload, support, and so forth, but their two-year lags may have been too long to detect such effects (details summarized in Table 1.2). In a four-year study with one year between waves of data collection, De Lange et al. (2004) in a study of Dutch workers (see Table 1.2) found no reverse effect of depression on demands, control, and support; the team, however, found emotional exhaustion, a core symptom dimension of burnout (see Chapter 2) that was assessed at the Time 1 baseline, predicted more demands and less support at Time 2, controlling for baseline demands and support.

In research on life stress, there is evidence that depressed individuals are at risk of generating new stressors (Hammen, 2020), and the research also suggests “that stress generation leads to further depression” (p. 333), thus echoing the Dohrenwends’ earlier event stress proneness model. Most of the studies of workplace stress and health focus, rightfully so, on the impact of job stressors on depressive conditions and other health effects. It would still be helpful to develop a more rounded picture of the stress process by building tests of reverse causal hypotheses into analyses. The idea of reverse causal effects re-emerges in Chapter 2.

The Effort–Reward Imbalance (ERI) Model

In addition to the DCS model, several alternative models have been developed to explain the impact of work-related stressors. Among these models are the job-demands-resources, stimulus–response, conservation of resources, allostatic load, and hindrance–challenge models (Cunningham et al., 2023) as well as the person–environment fit model (Schonfeld & Chang, 2017). While this chapter is concerned with documenting the impact of adverse working conditions on depressive symptoms and disorders, none of these alternative models has generated as much high-quality longitudinal research as the DCS model. However, one other theoretical

model, the effort–reward imbalance (ERI) model, runs a close second. The ERI model is therefore the only other model of job stress covered in this chapter.

In 1996, the German sociologist Johannes Siegrist published an article describing the ERI model of job stress. The ERI model rests on a long tradition in sociology and social psychology, a tradition that recognizes that the foundation of social life is group membership and exchange. Work is a socially sanctioned exchange. In exchange for a worker's efforts fulfilling the demands of a job, the worker receives benefits. What a worker applies to a job is an amalgam of effort and motivation. The motivation can be extrinsic, like pay and advancement opportunities, and/or intrinsic—enjoyment of the work itself. Leading up to the ERI model are important ideas associated with Marcel Mauss, George Homans, and Alvin Gouldner. Mauss (1954/1925), who was Émile Durkheim's nephew, showed that the giving of gifts is virtually universal. Gift-giving, in turn, creates a sense of indebtedness in the receiver, giving rise to an obligation to reciprocate. Gift-giving between groups builds alliances and social solidarity. For Homans (1958), virtually all social behavior is based on “an exchange of goods, material and non-material” (p. 597). When each person in a dyad finds the behavior of the other person reinforcing, they continue to engage with each other. When the behaviors in the dyad are not reinforcing, the dyad weakens. Gouldner (1960) advanced the view that reciprocity is a universal behavioral norm. Norms are “rules or expectations that determine and regulate appropriate behavior within a culture, group, or society” (Open Education Sociology Dictionary, 2024). Reciprocity is observed in all human societies, like, for example, the incest taboo. Gouldner wrote that the norm of reciprocity has two components: “(1) people should help those who have helped them, and (2) people should not injure those who have helped them” (p. 171). An implication of Gouldner's view is that a violation of the norm of reciprocity is destabilizing.

Siegrist (1996) adapted his intellectual forebears' ideas to the work role. Accordingly, Siegrist held that disruptions to the “continuity of crucial social roles,” like the work role, adversely affect the individual's self-regulatory functioning, sense of mastery, and self-esteem, giving rise to psychological distress. Layoffs, job insecurity, jobs with no opportunities for advancement, and work in which meager rewards are incommensurate with the worker's effort cause distress. Siegrist (2004) wrote

that “effort at work is spent as part of a contract based on the norm of social reciprocity where rewards are provided in terms of money, esteem, and career opportunities including job security” (p. 1484). Contracts, however, are often asymmetric, to the detriment of the worker. Siegrist (1996) wisely addressed the often-posed question of why workers stay in jobs in which they face a chronic imbalance between effort and reward. He observed that many workers remain in jobs that pay low wages and offer few tangible benefits because they face high costs in quitting when there are few opportunities to obtain jobs with better conditions (e.g., pay, work–life balance).

Like the high-quality longitudinal research generated by the DC and DCS models, the ERI model has also stimulated high-quality research. Table 1.3 summarizes much of that research. As in the previous table, Table 1.3 demonstrates the adverse impact of stressful job conditions on depression and depressive symptoms in workers. Moreover, the research documented in both Tables 1.2 and 1.3 is consistent with earlier meta-analyses¹³ and reviews that show the adverse effect of stressful working conditions on depressive symptoms (Bonde, 2008; Nieuwenhuijsen et al., 2010; Stansfeld & Candy, 2006; Theorell et al., 2015).

Workplace Bullying

Workplace bullying is another important stressor with adverse mental-health effects on workers. By bullying, we refer to a situation characterized by “one or more perpetrators with a power advantage over their victim systematically directing hostile behaviors toward their target over a sustained period of time” (Schonfeld & Chang, 2017, p. 164). In Nielsen and Einarsen’s (2012) theoretical model, repeated exposure to workplace bullying leads to prolonged cognitive and physiological activation that results in impaired mental and physical health. In their “stress-as-offense-to-self” model, Semmer et al. (2007) advanced the view that being a target of psychological aggression at work wears down

¹³ Meta-analyses provide a way of averaging study results. We discuss meta-analyses in a footnote in Chapter 2 and again in the text of Chapter 5.

Table 1.3 High-quality longitudinal studies that bear on the relationship of the ERI model of workplace factors to depressive symptoms. Parts of this table are from Schonfeld and Chang's (2017) / Springer Publishing Company. *Occupational Health Psychology: Work, Stress, and Health* by permission of the Springer Publishing Company. A key to the abbreviations is on bottom of the table.

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Godin et al. (2005)	Belgium	<2,000 (½ ♂) SOMSTRESS study	1 yr.	Sociodem., threat from global economy, job dissatisfaction, workplace instability	In ♂s combo of no ERI at T1 but ERI at T2 → ↑ T2 depression, anxiety, somatization, and fatigue. In ♀s, combo of ERI at T1 and T2 → ↑ T2 depression, anxiety, somatization, and fatigue.	Dimensions of SCL-90 treated dichotomously. Excluded workers with above-cutoff SCL-90 scores T1 scores. Part of the effect is concurrent.
Halonen et al. (2019)	Sweden	3239 (47.5% ♀)	3 waves, waves 2 yrs. apart	Sociodemo., chronic disease, and physical WL. Excluded those with high depression scores	T1 ERI → ↑ T2 depression symptoms Part of study of depression symptoms mediating relation of ERI to neck/shoulder pain. Depression symptoms were a mediator.	Depression symptoms were continuous. ERI turned into a dichotomy.

Hoven et al. (2015)	11 Euro. countries	2798 (59% ♂)	2 yrs.	Sociodemographics, low symptom levels at baseline	T1 ERI → ↑ T2 depression symptoms ERI mediates relation of social position to depression symptoms	Not clear that all participants remained in same job over the 2 yrs. of the study.
Juvani et al. (2018)	Finland	42,862 (80% ♀) workers from 10 towns	Until disability pension, up to 4 yrs	Sociodemo., location, alc. smoking, hx mental and physical disorders	T1 ERI → ↑ disability pension depression T1 ERI + high strain: job → ↑ disability pension depression (greater effect)	ERI and high strain were dichotomized. Add organizational injustice and effect was larger.

(Continued)

Table 1.3 (Continued)

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Kivimäki et al. (2007)	Finland	>4,800 hospital employees (Hosp. Personnel Study) + >18,000 municipal employees (10 Town Study) Mostly ♀s	2–4 yrs.	Sociodemo., employment grade.	ERI → ↑ GHQ and depression dx in the 10 Town Study. ERI → ↑ Depression dx in Hospital Personnel Study.	ERI comprised a single item making the findings surprising ordinarily given weaknesses of a 1-item scale. Dichotomized GHQ used in both studies. Self-reported doctor dx of depression and GHQ used in both studies. Excluded individuals with disorder or borderline disorder at baseline.
Lamy et al. (2013)	France	1,209 ♀ nurses and 908 ♀ nursing assistants	2 yrs	Age, work unit specialty, schedule Excluded ♀s with high T1 CES-Ds	ERI → ↑ depression symptoms	The T1 cutoff on the CES-D for excluding ♀s from the analyses was high, 20. Lower score like 16 would be preferable.

Matthews et al. (2022)	U.S.	1,599 workers (51% ♀); representative of workforce	9 yrs.	Sociodemo., alc., LTPA, job control, family problems. Excluded people with baseline depression	ERI (either as categorical or continuous variable) → ↑ risk dx of depressive episode	Family problems include excessive conflict, demands, criticism. MDE dx with CIDI. Over 9 yrs. job conditions could change. Healthy worker effect: over 9 yrs. people with unrewarding jobs could have left job or dropped out of study.
Niedhammer et al. (2015)	France	National sample 4,717 workers (≈ 50% ♂)	4 yrs.	See Table 1.2	Low reward → GAD and MDD	Prospective study. One of few studies to look at GAD.
Nigatu and Wang (2018)	Alberta, Canada	2180 (55% ♀)	4 waves, at 1-yr intervals	Sociodemo., job type, history of MDD	ERI alone → ↑ MDD Combo of high D/C ratio and hi ERI → ↑ MDD Combo of WFC and hi ERI → ↑ MDD	High D/C ratio is demand/control ratio is dichotomous. Not often used. WFC and ERI also dichotomized.

(Continued)

Table 1.3 (Continued)

Study team	Country	Sample	Time lag	Control variables	Major findings	Comments
Rugulies et al. (2013)	Denmark	Representative workforce sample 2,710 (51% ♀)	5 yrs.	Sociodemo, smoking, self-rated health, LTPA, alc.	Although ERI is most unfavorable in highest occupational grades, ERI → ↑ severe depression symptoms, with stronger impact in lower occupational grades.	Excluded individuals with high levels of depression symptoms at T1. Also controlled for continuous measure of T1 depression symptoms. Can't control for change in job conditions over time.
Stansfeld et al. (1999)	U.K.	>7,300 civil servants (2/3 ♂) Whitehall II	5 yrs.	T1 GHQ, employment grade, age	ERI → ↑ GHQ, poor social functioning, and worse general mental health	Dichotomized continuous DV. Trichotomized ERI. Excluded baseline cases of hi GHQ.

Note: Abbreviations. ERI, effort-reward imbalance; sociodemo., sociodemographic variables at baseline; ♀, female; ♂, male; LTPA, leisure time physical activity; ERI, effort-reward imbalance; GAD, generalized anxiety disorder; CES-D, Center for Epidemiologic Studies Depression Scale; hx, history; alc., excessive alcohol use; WFC, work-family conflict; D/C ratio, demand/control ratio; GHQ, General Health Questionnaire (a psychiatric symptom measure); CIDI, Composite International Diagnostic Interview; MDD, major depressive disorder; MDE, major depressive episode; T1, Time 1 and T2, Time 2. Baseline DVs refer to the baseline version of the dependent variable. IVs refer to the principal independent variables.

the victim's feelings of self-worth. Self-worth is an important personal resource, whereas reduced self-worth is a symptom of depression.

Kivimäki et al. (2003b), in a longitudinal study of more than 5,400 Finnish workers, found that job-related bullying is related to increased risk of clinical depression. Nielsen et al. (2012) in another longitudinal study, this one involving Norwegian workers ($n = 1,775$), found that having been a victim of workplace bullying is related to later increases in depressive and anxiety symptoms. In their meta-analysis, Nielsen and Einarsen (2012) found that bullying predicts later adverse mental health outcomes, including increased depressive, anxiety, and burnout symptoms. They also noted that earlier poorer mental health in workers predicts them becoming targets of bullying later. It is possible that aggressive coworkers and customers detect vulnerability in already-affected workers, which in turn arouses those potential aggressors to bully the affected workers. The issue of whether witnessing workplace bullying affects health in a substantial manner remains open to question (Nielsen et al., 2024).

Underestimates

The research summarized in Tables 1.2 and 1.3 demonstrates that workplace stressors affect depression risk, whether reflected in a diagnosable disorder or higher scores on a symptom scale. Two factors, however, suggest that the results in the two tables are underestimates of the impact of job stressors on depression. First, Altman and Royston (2006) wrote that “dichotomising a variable at the median reduces power by the same amount as would discarding a third of the data” (p. 1080). Of course, dichotomizing by pitting the highest tertile or highest quartile against everyone else also reduces a study's power to detect effects.¹⁴ The authors recommend avoiding converting continuous variables into binary categories because of the distortion such conversions may produce. Many of the scales used in the studies in the tables,

¹⁴ Altman and Royston are also careful to note that sometimes when researchers hunt for an “optimal” cutoff, they risk obtaining an overestimate of the relationship between two variables, which is not the case in the studies summarized in Tables 1.2 and 1.3. Those studies tended to use arbitrary cutoffs, such as the highest quartile versus everyone else.

however, have been converted from continuous to categorical measures (dichotomized, trichotomized, etc.). It is a procrustean but popular procedure in research on depression, probably because of the ease with which results can be presented. It is often done to establish landmarks. Take, for example, a study of blood pressure; researchers are interested in identifying risk factors for hypertension. If we have two groups comprising individuals with high and low blood pressures, someone whose pressure is just below the threshold for joining the high blood pressure category is treated as equivalent to someone whose blood pressure is very good for the person's age. Consider depressive symptoms, the subject of this chapter. Researchers may regard certain cutoffs like the score of 16 on the Center for Epidemiologic Studies Depression Scale (CES-D) as an important landmark denoting high levels of distress or depression risk (Radloff, 1977). However, studying the impact of working conditions on depression by employing binary categories is problematic. A worker obtaining a depression score (1) below a cutoff (between 0 and 15 on the CES-D) or (2) at or above a cutoff (from 16 to 60) ignores all the variation scores below and above the cutoff.

In the previous paragraph, we considered endpoint variables like blood pressure and depressive symptoms. Consider now predictor variables like age. We can study the correlation of age with general physical health. What if we have a sample of adults between ages 18 and 90 and use the median to divide the sample into two groups that we call young and old? The size of the correlation between the predictor age and health is going to be smaller than if we consider age in all its variation. A similar idea applies to the predictor variables we have been discussing, predictors such as ERI and the intensity of job demands. By taking continuous measures of predictor variables and chopping them into, say, binary variables, information is lost. For example, workers in the highest quartile of job demands are pitted against everyone else. If the highest quartile is used as a marker of high workloads, the worker with a score just above that marker is considered categorically different from a worker whose score is just below that quartile marker. And the worker whose score is just a hair's breadth below the highest-quartile marker is considered a member of the same category as a worker with a relatively undemanding job. In dichotomizing (or trichotomizing) predictors (job stressors) and outcomes (depressive symptoms) the loss of information is great, thus distorting the size of the effect, usually downward.

Second, there is a selection phenomenon known as the “healthy worker effect.” While the idea behind the phenomenon has long been known to epidemiologists, the term itself was coined by McMichael (1976). McMichael used the term to refer to the fact that employed individuals tend to have more favorable standardized mortality ratios (an epidemiologic measure of mortality risk) than the general population. People who have jobs are ordinarily healthy enough to work. The idea, however, does not have to be limited to mortality experience, which is why the “healthy worker effect” bears on the studies summarized in Tables 1.2 and 1.3. Individuals who are included in a worker sample are likely to be healthier than those who are not included. Furthermore, individuals included in a longitudinal study and who made it to the study’s endpoint are likely to be healthier than same-age coworkers who dropped out or retired, sometimes because of the deleterious health impact of job stressors. Thus, chances are that the workers who took part in the foregoing studies were healthier than those who did not. The healthy worker effect likely reduces the size of the estimate of the impact of job stressors on depressive symptoms (e.g., Christ et al., 2007) as well as symptoms of burnout (e.g., Schaufeli, 1995).

Conclusions

Depression is a health-related problem that contributes heavily to the global burden of disease and disability (James et al., 2018). It has been recognized from the dawn of the recorded word, beginning with the *Gilgamesh* saga, the Bible, and Hippocrates. That recognition continued through the time of Shakespeare. Although it has gone by different names, thinkers as diverse as Adam Smith, Karl Marx, and Émile Durkheim have recognized it in one way or another. Depression, of course, became the subject of psychiatry, as we have represented in the work of Emil Kraepelin, Adolf Meyer, and Sigmund Freud. Investigators have come to regard depression in two different ways. One way is as a diagnosis. The other way is as a phenomenon that exists on a continuum, like temperature, with individuals who are at the high end of the continuum expected to meet criteria for a diagnosis.

The evidence presented in this chapter shows that adverse working conditions contribute to depressive symptoms and disorders. These

working conditions include excessive workloads, little control over job tasks, unsupportive coworkers, imbalances disfavoring workers in terms of the efforts they put into a job and the rewards they receive from it, and bullying. Moreover, the effects described in the chapter tend to be underestimates.

Postscript

To be perfectly clear, we do *not* claim that stressful working conditions are the only contributors to depressive symptoms and disorders. We recognize that other factors contribute to depressive conditions. For example, stressful life events occurring outside the workplace can also increase the risk of clinical depression and elevated levels of depressive symptoms.

We recognize that heredity also plays a role in the development of both MDD and depressive symptoms. Sullivan et al.'s (2000) meta-analysis of twin studies shows that monozygotic in comparison to dizygotic twins¹⁵ are more concordant for major depression, evidence that genes play a role in the disorder's emergence. Although we could not find a meta-analysis purely devoted to depressive symptoms in adults, Bergen et al.'s (2007) meta-analysis of research on adolescents and young adults indicates that there is greater similarity in depressive symptom scores between pairs of monozygotic twins compared to the similarity between pairs of dizygotic twins. Such a finding suggests that genes contribute to the emergence of depressive symptoms. While genetic influences are certain to exist, Levinson (2013) wrote "Of the three major psychiatric disorders in adults—schizophrenia, bipolar disorder, and major depressive disorder—major depression has proven to be the most challenging for modern genetic methods. This is perhaps not surprising, given that major depression is the most common and the most influenced by non-genetic factors" (p. 396). Those nongenetic factors include stressful working conditions.

¹⁵ Monozygotic, or identical, twins share 100 percent of the same genes. By contrast, dizygotic, or fraternal, twins share 50 percent of the same genes, like ordinary siblings.

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