Recently, General Motors announced the slashing of 30,000 jobs in North America. The move will incur savings to the ailing carmaker of $7 billion a year (1). In 2004, Daimler-Chrysler agreed to a pay-freeze in exchange for job security in Germany. This year, the company announced it will lay off 8,500 workers in the very heartland of Mercedes-Benz around Stuttgart. Two days after the announcement by General Motors, the bankrupt car parts maker Delphi had to defer a $488 million executive bonus plan following a storm of criticism by unions and shareholders (2). Aren’t these times to become depressed? Thus, why wonder that Rugulies et al. (3) in this issue report on a twofold increased risk for depressive symptoms in Danish men experiencing job insecurity and in women with low influence at work or low supervisor support? However, in the case of the study by Rugulies et al., the economic conditions in Denmark at the time were quite different from the situation in most industrialized countries today: At the collection of the baseline data in 1995, few citizens doubted the Danish cradle-to-grave welfare system. Why should Danish men perceiving job insecurity be at increased risk of severe depressive symptoms 5 years later? The outcome scale measuring depression in this study was a subscale of the 36-item Short-Form Health Survey (a health-related quality-of-life instrument), asking respondents to recall the past 4 weeks (4). The job insecurity items inquired about both worries of losing a job and whether participants considered it difficult to find another job in case of unemployment. Given that depression is one of the most prevalent forms of mental illness in the industrialized world (5) and given the undulating course of depressive spells throughout life (6), could respondents with a worry-prone personality and absence of depressive symptoms in 1995 be more likely to encounter a depressive spell at follow-up in 2005? A recent report from the Whitehall II study found that more than 80 percent of the difference in depression scores in subjects with job insecurity was explained when introducing pessimism, vigilance, primary deprivation, financial security, social support, job satisfaction, and job control into the equation (7). Most data, however, support the view that individual-level factors ranging from genes (8) to personality traits or social resources amplify the effect of exposures, such as job insecurity or life events, on the risk for depression. In line with this, a recent study among middle-aged Australian managers identified the combination of job insecurity with excessive job strain as a particularly toxic synergism with odds ratios of 13.9 for depression (9).

The second important finding arising from the Danish study is a gender difference in work-related psychosocial risk factors for depression. Women reporting low support by their supervisors and women perceiving low influence at work were twice as likely to experience severe depressive symptoms during the follow-up as did study participants enjoying decision latitude and supervisor support. How can these gender differences whereby job insecurity was predictive in men but social support was predictive in women be reconciled? A straightforward explanation is that there are gender differences in perceived sources of stress. It has been well described that men are more likely to be stressed by issues related to the domain of work, whereas women are more likely to be stressed by relationship concerns. The reason for this differential perception of stressors remains elusive. Is job insecurity as a psychosocial stressor linked to the social function of providing nutrition and security for the family—regardless of gender—or do such differences arise from genetic determination of male and female brains? Whatever the case, job insecurity in men and lack of social support in women appear to be perceived as chronic threats to social integrity.
What are the biologic consequences of such chronic psychosocial stress? Data suggest that prolonged psychosocial stress may eventually alter the functioning of the hypothalamic-pituitary-adrenal axis with flatter circadian cortisol slopes arising from more sustained cortisol levels throughout the day (10). The high prevalence and the possible chronic nature of job insecurity or low supervisor support in the Danish study suggest that it is unlikely that these stressors elicit profound responses of the sympathetic nervous system or shifts in the circadian cortisol secretion. It is, however, conceivable that self-perceived job insecurity may elicit a mild biologic stress reaction, such as withdrawal of parasympathetic tone (11). If the parasympathetic nervous system retreats, the organism no longer functions at its physiologic optimum (12). Instead, the balance is shifted toward a chronic stress response with increased allostatic load, as evidenced by higher risk of insulin resistance and elevated levels of low density lipoprotein cholesterol or C-reactive protein. Lowered parasympathetic tone can be assessed by a decrease in heart-rate variability (12). Taking specific heart-rate variability measures as a proxy for automatic balance, the Whitehall study showed that lower heart-rate variability at rest was associated with increased waist circumference and systolic blood pressure, decreased high density lipoprotein cholesterol, and increased tryglicerides and fasting and 2-hour postload glucose levels (13).

Moreover, several longitudinal studies have now identified depression and lack of social support as risk factors for cardiovascular disease (14). It is believed that the biologic pathways linking these psychological states to adverse health outcomes involve factors that propagate the progression of atherosclerosis. Risk factors for atherosclerosis comprise imbalances in blood lipids, type II diabetes, hypertension, increased levels of C-reactive protein, and higher resting heart rate. Except for the latter, these factors have also been used to operationalize allostatic load as a measure of the biologic burden or wear and tear of chronic stress or adverse health behavior (15, 16). Higher scores on an allostatic load measure predicted higher mortality and poorer mental and physical functioning in retired Americans (16).

At the moment the worker is laid off or during the immediate fear of being laid off, he or she will somehow survive, thanks to the mobilization of resources during the stress response. However, as suggested by the allostatic load model, the burden of chronic work-related psychosocial risk factors may sink in as wear and tear on the organism’s homeostatic systems over decades (17). Thus, the consequences of today’s job insecurity may not surface until several decades later, when affected persons die earlier or lose their capacity to work. In the face of the demographic change with an aging working population, today’s job insecurity in men or low supervisor support in women may eventually surface throughout the day (10). The high prevalence and the possible chronic nature of job insecurity or low supervisor support in the Danish study suggest that it is unlikely that these stressors elicit profound responses of the sympathetic nervous system or shifts in the circadian cortisol secretion. It is, however, conceivable that self-perceived job insecurity may elicit a mild biologic stress reaction, such as withdrawal of parasympathetic tone (11). If the parasympathetic nervous system retreats, the organism no longer functions at its physiologic optimum (12). Instead, the balance is shifted toward a chronic stress response with increased allostatic load, as evidenced by higher risk of insulin resistance and elevated levels of low density lipoprotein cholesterol or C-reactive protein. Lowered parasympathetic tone can be assessed by a decrease in heart-rate variability (12). Taking specific heart-rate variability measures as a proxy for automatic balance, the Whitehall study showed that lower heart-rate variability at rest was associated with increased waist circumference and systolic blood pressure, decreased high density lipoprotein cholesterol, and increased tryglicerides and fasting and 2-hour postload glucose levels (13).

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According to the Harvard Business Review, the American Institute of Stress currently estimates that American businesses are losing $300 billion annually as a result of lowered productivity, absenteeism, and increased health-care costs attributable to stress (18). If the industrial world continues to ignore data such as the findings by Rugulies et al. (3) and those from the Whitehall study or the Australian investigators, the room to maneuver may rapidly shrink as it did for the captain of the Titanic steering toward the ice field. In these times, job insecurity or low social support at work should no longer be viewed as insignificant soft factors affecting a minority of vulnerable individuals. The hardwiring of our brains enforces biologic system alterations as a response to these perceived threats that may chronically severely affect population health. Already, the US reports a lower life expectancy as should be expected from the gross domestic product. The risk factors identified in the Danish study may add to the burden of social inequality.

Which agenda should future research address? Among the many questions raised in the report by Rugulies et al. are the following: What is the dose-response relation between job insecurity and depressive symptoms? What is the lag time between the onset of job insecurity and the onset of depressive symptoms? Is there a duration-effect relation something like job-insecurity pack-years? How do we refine our current job strain measures to incorporate the changing work environment in order to elucidate the interactions between job insecurity and job strain or job insecurity and violated reciprocity (effort-reward imbalance)? What are the effects of job insecurity-mediated depressive symptoms on the bottom line, for example, on employee productivity, job performance, absenteeism, and the loss of talent and knowledge due to increased job turnover or health-related early retirement? What are the biologic mechanisms possibly linking the job insecurity or low supervisor support to long-term health outcomes (12)? What is the role of gene-environment interactions? The task of successfully managing public health in an aging population mandates a high priority to these research questions. However, research should not be confined to the realm of social epidemiology. Researchers should seek to exchange with business schools in order to enumerate the potential economic burden of these psychosocial icebergs and to identify strategies toward ameliorating the fall-out. In the future, job insecurity will become a reality for most employees. The public health agenda is how society will cope with it.

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REFERENCES