Do stressful working conditions cause psychiatric disorders?

Twenty-five employees of France Telecom, a European telecommunications giant, have committed suicide within less than a 2-year period. The company’s trades unions claimed that the number of suicides was a result of stressful working conditions. These events alerted the media globally—but doubts have been raised about whether it was working conditions that caused the 25 incidents and whether the Telecom suicides were greatly in excess of expected.

Without taking any strong position to this particular case, it may be worth considering the question at a more general level. Suicide clusters are a recognized phenomenon whose ‘causes’ are not always related to excess of mental disorders in the group, but to a culture where suicide somehow becomes contagious. In terms of primary prevention in the workplace, however, targeting mental health, rather than aspects of culture, might be the more feasible strategy.

The attributed cause of suicide at France Telecom was stressful working conditions, but is there scientific evidence, even at a more general level, to prove that work stress actually causes psychiatric disorders or drives people to kill themselves? If there is such a link, is this specific to a particular type of psychiatric disorder or, alternatively, does too-demanding work increase the risk of a range of disorders, the particular vulnerabilities of each exposed individual determining which disorder the intolerable stress triggers?

Research on work stress has long traditions. The conceptual models in this field suggest that excessive job demands in combination with low control over work or low social support at work [1], the experience of imbalance between high effort spent at work and low reward received [2], and unfair treatment of employees by the management [3] are particularly stressful. If prolonged, they have adverse effects on physical and mental health. A recent meta-analysis of published epidemiological data calculated summary estimates for the effects of work stress: the average risk of depression across all studies weighted by the number of participants was 1.2- to 1.4-fold [4].

However, robust associations do not guarantee causality. The arguments against cause-and-effect are: first, the observed association between work stress and mental health might be spurious, as poor mental health in adolescence affects selection into jobs with poorer work characteristics and is associated with increased risk of psychiatric disorders in adulthood. It is worth noting that the median age of first onset for mental health problems is as early as age 11 for any anxiety or impulse-control disorders, 20 for any substance use disorders and 30 for any mood disorders, i.e. at ages when exposure to work life has been modest or lacking [5].

Second, reverse causality is difficult to eliminate in the majority of studies as they assess work stress with self-reports. A recent experimental study of facial expressions showed a strong negative bias in recognition of happiness among depressive individuals [6]. Indeed, depressed mood is associated with people having a negative view of their surroundings, including the working environment, and it is a strong predictor of subsequent psychiatric disorder. Prodromal or unrecognized depression may therefore artificially inflate any association between self-reported work stress and mental disorders. Clearly, it is methodologically challenging to disentangle the extent to which a self-report reflects objective work environment versus subjective interpretation.

Randomized controlled trials, providing a gold standard to deal with such methodological uncertainties, are hardly feasible in this case, because people cannot be randomly allocated to long-term stressful work conditions for practical and ethical reasons. However, quasi-experimental studies, although lacking the explicit randomization, may still provide substantial advantages over the mainstream questionnaire-based studies on work stress. One example for this is the study by Virtanen et al. [7] who used routinely collected monthly bed occupancy figures (the sum of inpatient days divided by the number of beds in the ward) as a proxy measure of stressful working conditions for hospital ward personnel. This objective proxy measure avoids the problems related to self-reports, such as subjectivity and recall biases.

The researchers found a dose–response relationship between bed occupancy rate and subsequent psychiatric illness absence among staff. A greater long-term overcrowding was associated with a greater risk of subsequent sick leave due to depressive disorders, but not with sick leave from other psychiatric disorders [7]. If we accept that the degree of overcrowding reflects work stress, then those data provide evidence to support objectively assessed work stress as a risk factor for depressive symptoms.

However, with two measurement points in that study temporality remains uncertain. A further report by Virtanen et al. [8] addresses this issue by following changes in occupancy rates and commencing
antidepressant treatment among these employees over an extended period of 4 years. Longitudinal multilevel analyses of the data showed a higher likelihood of starting a new antidepressant treatment after each 6-month periods of overcrowding. Importantly, the authors confirmed that the use of antidepressants did not predict overcrowding in wards, thus excluding the possibility of reverse causality bias, that is, that depressive employees at ward, through poorer performance, caused the overcrowding.

Demonstrating a causal effect without opportunities to conduct randomized trials is challenging, but not necessarily impossible. The causal link between smoking and lung cancer, for example, was mainly demonstrated with accumulated observational evidence. However, before the cause-and-effect was widely accepted a series of alternative hypotheses were considered and rebutted point by point (e.g. the effect of an ageing population, recall bias, selection of study groups, confounding variables, other aetiological factors etc) [9]. This then justified the public health policy and efforts that have now been successful in reducing smoking prevalence in many countries.

The analogy between smoking-lung cancer and work stress-depression does not stop here. If everyone smoked 20 cigarettes/day, we would think lung cancer was a genetic disease—most smokers do not get lung cancer [10]. Similarly, most employees exposed to apparently stressful workplaces do not become psychiatrically unwell. In both situations, individual factors, genes and others, such as temperament must also play a crucial role [11].

Although there seems to be some support for the status of stressful working conditions as a causal risk factor for depressive symptoms, more and better evidence is needed before any firm conclusions can be drawn and alternative explanations be excluded. In psychiatric disorders, multiple correlated risk factors precede clinical disease and cluster among those at high risk. This makes it difficult to ascertain the independent effect of any one exposure on disease onset, including work stress. Nevertheless, this scientific challenge is important to take up. Methodological problems related to selection bias might be overcome using, for example, birth cohort data [12].

A better understanding of the relationship between work and mental health is urgently needed. From this might emerge new strategies for prevention and treatment. Not all suicide and depression are preventable, but providing occupational physicians with an evidence base for early interventions might reduce the probability of further episodes such as occurred at France Telecom.

Funding

BUPA (originally, the British United Provident Association) Foundation, UK; National Institute on Aging, National Institutes of Health, USA, to M.K.; South London Maudsley National Health Service Foundation Trust/Institute of Psychiatry, King’s College London, National Institute for Health Research Specialist Biomedical Research Centre to M.H., M.H.

Mika Kivimäki, Matthew Hotopf and Max Henderson

Department of Epidemiology and Public Health, University College London, London; Department of Psychological Medicine and Psychiatry, Institute of Psychiatry, King’s College London, London, UK
e-mail: m.kivimaki@ucl.ac.uk

References