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 working 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...
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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, be-
fore 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 ge-
netic disease—most smokers do not get lung cancer
[10]. Similarly, most employees exposed to apparently
stressful workplaces do not become psychiatrically un-
well. In both situations, individual factors, genes and
others, such as temperament must also play a crucial

Although there seems to be some support for the sta-
tus 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 dis-
orders, 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 prevent-
able, but providing occupational physicians with an
evidence base for early interventions might reduce the
probability of further episodes such as occurred at
France Telecom.

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