Variation in mortality risk of people released from prison

The body of research examining mortality of people who are or have been imprisoned has made clear that there are specific high-risk periods during which mortality is especially increased, particularly that due to unnatural causes such as suicide and drug overdose. The first weeks of imprisonment are of concern in relation to suicide deaths, with a substantial minority of prison suicides occurring soon after reception to custody.1 Drug overdose deaths are increased in the initial 2–4 weeks following release from prison, with risk decreasing with the passing of time.2 Suicide deaths can also be increased during this period.3

Zheng Chang and colleagues4 report on mortality among people who have been released from Swedish prisons, finding increased mortality risk among released prisoners with substance use disorders. Released prisoners with other psychiatric disorders did not show increased mortality following adjustment for socio-demographic and criminological factors and substance use disorders. Although we agree with Chang and colleagues that substance use disorders are an important confounder in examining mortality of people with psychiatric disorders, we note that their analysis did not separately assess mortality during the high-risk weeks immediately after prison release. Furthermore, adjustment for subsequent periods of imprisonment following the first observed release from prison does not seem to have been made. Throughout follow-up, it is likely that a substantial proportion of the cohort was reimprisoned, perhaps multiple times. Mortality risk for these individuals would have varied over time, with high-risk periods immediately following imprisonment, very low risk of death during the remainder of imprisonment, and increased risk again following release from prison. Examining these specific periods of time in comparison with other times in custody and at liberty would perhaps provide a very different picture of the risk of death in relation to psychiatric disorders and imprisonment.

Chang and colleagues4 call for further studies with extended follow-up to assess the effect of treatment for substance use disorders on mortality of released prisoners. Findings from longitudinal cohort studies have shown that opioid substitution therapy, such as methadone or buprenorphine maintenance, saves lives in prison and post-release.1,5 There is an urgent need to scale up access to opioid substitution therapy in correctional and community settings globally to address mortality of people with opioid use disorders, particularly in the high-risk weeks after prison release.

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Authors’ reply

Our study in 47 326 prisoners over a median follow-up of 5 years showed high rates of mortality after prison release.6 These high rates replicate findings from many previous studies,7 and so the main focus of our investigation was to determine psychiatric risk factors, with careful adjustments for confounds and additional sibling designs to control for familial factors.

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The letter by Sarah Larney and colleagues raises two specific points. First, they mention that we did not study the high-risk weeks immediately after prison release. Although, as described above, this was not one of the principal aims of the study, the survival graphs (figure 1 in the Article) present these findings. We found that the risk continued beyond the immediate period after release, and the effects of substance use disorders and psychiatric disorders looked reasonably proportional from the survival curves.

Second, they raise the issue that some of the sample would have been re-incarcerated and this might influence the contribution of psychiatric risk factors. This is an important point, and we have re-examined the data accordingly. We found that 16 766 (35%) of the cohort of released prisoners (n=47 326) was re-incarcerated, and that their mortality differed slightly from that of others (6.7% [1120 of 16 766] in the re-incarcerated sample vs 5.7% [1754 of 30 560] in released prisoners who were not re-incarcerated during follow-up).

From an epidemiological perspective, we think that it is reasonable to model the total effects of substance use disorders and psychiatric disorders, even if part of the effects can be viewed as mediated via re-incarceration. Nevertheless, we have done a new sensitivity analysis that censored individuals at re-incarceration. This new analysis showed similar results (for all-cause mortality, alcohol use: adjusted hazard ratio 1.81, 95% CI 1.61-2.04; drug use: adjusted hazard ratio 1.81, 95% CI 1.56-2.01) to those reported in the paper. Nevertheless, re-incarcerated individuals might be a particular high-risk subgroup and future research can usefully examine the differential effects of risk factors in them.

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Giving up the disease model

The defence of the biomedical model in the editorial in the June, 2015, issue of The Lancet Psychiatry shows that conflict about this issue is conceptual and not just about the use of language. Like the editors, I have reservations about “the simple relabelling of mental illness as mental distress”. However, your wishful speculation that the findings of neuroscientific research will translate into clinical practice implies that you think that mental health disorders are brain diseases. As a general member of the Division of Clinical Psychology (DCP), I do not want to encourage disciplinary conflict in mental health, but your view opposes the DCP public affirmation of the “need to move towards a system which is no longer based on a ‘disease’ model”.

As a doctor, I agree with the DCP’s position, as does the Critical Psychiatry Network (CPN).

The DCP has deliberately chosen to put the word “disease” in inverted commas in this quote. In everyday language, the terms illness and disease can be used interchangeably. However, a technical distinction has been made in the scientific literature.

Illness is the personal experience of symptoms and suffering, whereas disease is the underlying biological pathology. To quote Eric Cassell, “Illness is something an organ has; disease is something a…[person] has.”

The problem is that your editorial, in these terms, encourages the reduction of mental illness to brain disease. I agree that clinicians have a wide range of views on this matter. In particular, many psychiatrists say they are more eclectical in their approach to the underlying causes of mental illness than solely relying on biological factors. However, few psychiatrists sufficiently acknowledge, as do DCP and CPN, that minds are enabled by, but not reducible to, brains. In fact, medical training tends to encourage the adoption of the biomedical model in psychiatry. I agree that medical training can have advantages for mental health work, especially for the management of psychosomatic disorders and medically unexplained symptoms. However, the disadvantage is the difficulty doctors have in giving up the “disease” model as proposed by the DCP. It is a pity that The Lancet Psychiatry—at least as reflected in its editorial—does not seem to be able to take this issue forward.

I declare no competing interests.

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1 The Lancet Psychiatry. This year’s model. Lancet Psychiatry 2015; 2: 427.