Letter to the Editor

One mechanism underlying contrasting health-economy findings
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Tapia Granados1 recently presented health-economy findings, which in the short-term, identify the decline in mortality to slow or even temporary reverse during economic upturns. These counterintuitive ‘ecological’ results initiated a lively debate in the International Journal of Epidemiology2–6 as historically epidemiological results have shown adverse health outcomes among the unemployed.7,8 In response to Tapia Granados’1 short-term pro-cyclical health-economy oscillations, authors have suggested that such results represent: an ecological fallacy;2 may possibly represent reality;3 reveal nothing new—as in the long-term, mortality fluctuates in a counter-cyclical health-economy fashion.3,9 A minority of commentators have also suggested that such macro-level findings1,10 need not necessarily be inconsistent with individual level research identifying worse health among the unemployed.6,11,12

We contribute to this health-economic literature by highlighting that the concept of allostatic load13–15 may be one biological mechanism which gives rise to adverse health outcomes in both the unemployed and employed. (An anonymous referee suggested work by Sterling and Eyer16 on static load development. Olafsson and Svensson25 summarize a lighting that the concept of allostatic load 13–15 may be one with pro-cyclical (short-term) 1,10,18–21 and counter-cyclical3,22–24 (often long-term) 3,22–24 health-economy findings need not only necessarily be inconsistent with each other.6,10–12

More than two decades ago, it was noticed that biological changes occur among the unemployed which resemble allostatic load development. Olafsson and Svensson25 summarize a number of these study results:

in various follow-up studies on an individual level the loss of job or the mere prospect of becoming jobless, have been found to cause elevated blood pressure, serum cholesterol and uric acid, elevation of blood concentration of catecholamins and...increased stress, psychosomatic disease and increased elimination of noradrenaline has been found to persist up to 2 years after the loss of a job (p. 1107).

While such physiological changes may occur among the unemployed, recent findings also identify allostatic load to be associated with those in employment. In a prospective study (1973–2000) on Finish industrial workers, Kivimaki and colleagues26 found those employees whom seldom recovered from work—after controlling for age, sex and 16 risk factors—had an elevated risk of cardiovascular death. Working conditions however, may not only affect sole individuals concerned, but also others. This is perhaps best evident in shift-workers, who are well-known to suffer from perturbed biological rhythms27,28 and have been shown to impact the health and well-being of others29 including (importantly) via motor vehicle accidents.30

As socioeconomic status (SES) is a prominent health issue in most developed countries,31–37 allostatic load development has also importantly been associated with SES in a graded fashion38 which may begin in childhood.39,40 These, other such findings41–43 and environmental conditions (such as air pollution44) associated with SES45,46 point to a single underlying mechanism operating for the employed and unemployed.

The LiVicordia studies47–49 and similar future research may further advance our knowledge of how environmental factors, particularly employment and unemployment conditions, impact underlying biological processes that inturn contribute to adverse health outcomes for communities and individuals.50 In this regard, the contrasting (pro-cyclical1,10,18–21 and counter-cyclical3,22–24) health-economy findings need not only be associated with each other,6,9–12 but may also result due to one underlying mechanism.

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References


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