Psychosocial environment and health

The assumption that there is a direct influence of the psychosocial environment on health is of increasing interest in partially explaining health inequalities. Recent works have shed light on mechanisms linking the psychosocial environment and biology. As an example, in 2010 a special volume of annals of the New York academy of sciences focussed on ‘The biology of disadvantage: socioeconomic status and health’. This volume summarizes evidence from the literature regarding the strong link existing between environment, in the broadest sense of the term, and biology.

Biological mechanisms

Taking the example of cancer, several biological mechanisms are involved in cancer development and progression, which occurs over a chronic time period, and specific risk factors have been identified (i.e. alcohol, tobacco, chemicals agents). Causal relationships between environmental factors and biological mechanisms involved in cancer have been well demonstrated in animals. In human, evidence is more difficult to obtain, therefore available studies are sparse and mainly based on correlations. However, an increasing literature suggests links between psychosocial factors, like stress, depression or social isolation, and cancer progression through activation of neuro-hormonal systems and epigenetic mechanisms. Evidence of the role of psychosocial factors in cancer initiation is more equivocal, although recent articles linking environment, DNA hypomethylation and cancer development suggest that the environment may modify DNA and gene expression.

Biological mechanisms and the reduction of social inequalities in cancer

If the psychosocial environment can influence cancer progression, and even the onset of disease at a given level of exposure to a risk factor, this implies that people exposed to a stressful psychosocial environment are at a higher risk of developing cancer at an earlier age and more rapidly. Furthermore, if the way in which the environment is embodied is more biologically damaging for people with low socioeconomic status (SES), this might, in part, explain social inequalities observed in cancer incidence and mortality. In fact, it has been shown that measures of allostatic load, a measure combining biological parameters of multiple regulatory systems (cardiovascular, metabolic, inflammatory parameters), follow a SES gradient. This may have consequences for epidemiological research and public health policy aiming at reducing health inequalities in cancer.

In epidemiological research, the assumption that some individuals are biologically more at risk of developing cancer raises questions about the way in which risk factors are considered. Classically, in cancer their effect is related to dose and duration, like for alcohol or tobacco consumption. For a fixed dose and duration, the effect is assumed to be the same in each group of a given population. If we assume that people who live in a poor psychosocial environment are more at risk of biological damage and therefore more susceptible to developing cancer, the risk associated with smoking could be higher for these individuals, as is the case for individuals with a lower SES.

At the individual level, the impact of risk factors should be considered according to the way in which the environment has been embodied by measuring for example key biological mediators contributing to the allostatic load and by analysing the interaction between risk factors and allostatic load. At the population level, for which data from biological samples are sparse, the effects of exposure to risk factors should be analysed by considering their interaction with social characteristics. Studies including clinical, biological, psychosocial data are thus urgently needed if we want to explore how the psychosocial environment impacts cancer risk and progression.

Regarding public health policy, assuming that the psychosocial environment can participate in producing social inequalities via biological mechanisms, modulating the effect of risk factors raises important questions:

In prevention, should interventions aiming to reduce the biological burden caused by environmental embodiment be
encouraged? As biological damage due to embodiment is cumulative, and because some exposures occurring during sensitive periods such as pregnancy or early childhood may have irreversible biological effects, early interventions could be prioritized, or be conceptualized using a lifecourse approach. The nature of these interventions remains to be seen, but they may include social, behavioural, societal, even pharmacological approaches (β-blockers, antidepressant medications) rather than focus only on fighting risk factors. Whatever form these interventions take, allostatic load could be used as short-term outcome to measure the effect of interventions on health.

In relation to screening, should populations with a high allostatic load be considered as high risk for cancer and thus targeted for screening?

Regarding treatment, behavioural and pharmacological approaches that target neuroendocrine pathways that support cancer initiation and progression may be tested in addition to curative treatment to increase chance of treatment success, as suggested in recent works.

Should surveillance, which is nowadays mainly scheduled according to clinical criteria, in particular according to tumour characteristics, integrate these dimensions? Indeed, cancer among people who embodied unhealthy environments could be more at risk to progress or less at risk to succeed with therapies.

A new model, taking into account the way people have embodied their environment over time, making use of measures of allostatic load, could be developed for managing cancer. In the light of the growing early evidence regarding the link between environment and biology, increasing research from data using biological and psychosocial data for analysing health inequalities is needed.

References

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