Social inequalities in health:  
Biological, cognitive and learning theory perspectives 

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ABSTRACT

Increasing social inequalities in health have been ascribed to unequal distribution of resources, and to exposure factors. We propose that these differences also may be explained by principles from cognitive stress theory. There seems to be consensus in the stress literature that the stress response is not predicted from the external situation. The acquired expectancies to stimuli and response outcome are determining the response. These expectancies are learned. Based on available reinforcement contingencies and resources, the individual learns positive response outcome expectancies ("coping"), or that nothing helps ("helplessness"), or that everything goes wrong ("hopelessness" – negative outcome expectancy). These expectancies are associated with social position and social success, in man and animals. High levels of coping are associated with high social position, and low stress values. Low level or lack of coping is associated with high and maintained stress levels, which may produce somatic changes. Lack of coping also affects choice of life style. Social inequalities in health, therefore, may depend on the learning history of the individuals. Expectancies of failure produce direct somatic effects via maintained high stress levels, and via learning of life styles and motivation for change.

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INTRODUCTION

We propose that principles from learning theory and cognitive psychology may contribute to an explanation of social inequalities in health in Western Europe and other industrialised societies. This influence, we propose, acts both through direct somatic effects (sustained activation) and via reinforcement factors for choice of lifestyle. Social inequalities seem to increase in Western Europe, despite broad political consensus for eliminating them and at least some success in reducing traditional differences in social class and poverty. There are still social inequalities in incidence, prevalence, and morbidity for traditional somatic diseases, and for subjective or unexplained health complaints like muscle pain, fatigue and undifferentiated gastrointestinal complaints (1-4).

There are two main hypotheses for these conditions. One is that lower social classes have fewer resources at hand for maintaining and improving their health. The other hypothesis is that lower social classes are exposed to more harmful agents throughout life (1). A likely compromise is that these two factors interact. In this paper we will propose a third hypothesis, based on learning theory and cognitive aspects of modern stress theory. We suggest that social differences in education and rewards produce differences in life style and health, and differences in “stress” due to different learning contingencies. In our opinion, social differences in reported stress are a product of differences in the expectancy to cope with the difficulties. Social differences rely more on cognition than on exposure, it is the acquired expectancies to available strategies that determine the somatic response to a “stressor”. This hypothesis is not necessarily an alternative to earlier hypotheses. We claim that our approach combines earlier hypotheses in a psychobiologically consistent theory of inequalities in health.

STRESS THEORY

In the literature assuming that social inequalities are caused by “stress” (1,5), a prevalent position is to combine all environmental factors as “stress” no matter whether they are physical, social, psychological or psychosocial. Within the realm of stress literature, there is reasonable consensus that the response to an environmental factor cannot be predicted by the exposure alone (5). The external stimuli do not determine the response, it is necessary to know the learning history of the individual. What does the situation mean to this particular individual, what are the expectancies attached to the situation, what actions or coping strategies are available to the individual, and what does the individual expect from her or his actions? Bluntly, it is not what happens to you, but what you make out of it.
that matters.

These acquired expectancies depend on previous experience, which depends on individual characteristics as well as social class and education. Formally, expectancies may be classified as attached to the stimulus situation, or to the outcomes of available response programmes and solutions. This represents a cognitive reformulation of learning theory (6). In this context, classical conditioning is defined as stimulus expectancy, and instrumental conditioning as response outcome expectancy (6). Positive response outcome expectancy (“coping”) reduces the stress level in man and animals. Lack of positive expectancies (“helplessness” (24)) or negative outcome expectancies (“hopelessness” (5)) are associated with high stress levels, sometimes sustained high levels (5). In what we refer to as Cognitive Activation Theory of Stress (CATS (5,7)), this is explained by stress being an alarm reaction that occurs when there is a discrepancy between what is expected (set value) and what exists (actual value). When the discrepancy is neutralised, the alarm is subdued and eliminated (5,7). Our main hypothesis, therefore, is that when coping fails and negative expectancies are acquired this may lead to poor health, via two mechanisms. First, there may be a sustained stress response that can cause somatic changes. Second, learning and reward factors lead to differences in lifestyle. Both mechanisms depend on response outcome expectancies. Since these are systematically distributed in society, both routes seem to us to be important determinants for social inequalities in health.

**MECHANISM 1: ALLOSTATIC LOAD, SUSTAINED ACTIVATION AND PATHOLOGY**

Physiologically, the stress response (the alarm) is a neurophysiological activation with an increase in arousal and wakefulness (5,7). This is not only a brain event, but also leads to changes in the hormonal level, the vegetative nervous system, and even in immunological variables (5,7,8). This means that “stress” responses may be measured by psychological, behavioural, and physiological methods.

Psychobiologically, the stress response is an alarm required for improved performance whenever there is a discrepancy between set values and actual values (7). This means that the stress response is an adaptive response, increasing the chances of survival, increasing performance, and a necessary homeostatic mechanism. This explains why we find the response in fish, birds, and mammals, and at all ages. If the system is challenged beyond capacity, this may be detrimental to health and performance. McEwen (10) refers to the challenge of this system as an “allostatic load”. This may tax the system beyond capacity. This occurs whenever an individual is in a situation where goal and reality are very different, and there is no way of developing or maintaining any positive expectancy. The stress response (the alarm) may then be sustained (5,7,8), or repeated without proper recovery (9-11). Such sustained activation has been related to pathology through specific pathophysiological mechanisms for hypertension, diabetes, cardiovascular attacks, brain signal molecules, and gastrointestinal disturbances (5,8). The most important psychobiological mechanism may be that normal resting periods or phases of restitution do not alternate normally with the activation processes (11,12). Such conditions are well known in sports. In order to strengthen a muscle or the cardiovascular system, training periods must be alternated with resting periods. Lack of restitution after work can start a vicious circle that in the long run can cause serious health problems, for instance cardiovascular disease or chronic muscle pain (11,12).

**MECHANISM 2: SUSTAINED ACTIVATION, LIFESTYLE, AND PATHOLOGY**

The other mechanism implies that the reward factors and the learning factors play an important role for the choice of behaviour (13). This means that learning of response outcome expectancies influences the choice of lifestyle and health behaviour. Development of “helplessness” and “hopelessness” affects motivation for life change. A “helpless” individual has learned to believe that there are no relations between actions and reward. This individual is less likely to develop and comply with instructions for new behaviours than someone that has learned that what he or she does really matters. This is probably even more true for hopelessness, where the expectancy is that everything the individual does leads to disastrous results. It does not make any sense to this individual even to try, there is no hope of success.

This is a “cognitive-behaviour path” that affects health through health behaviour (13). There is a general trend to move from traditional psychosomatic medicine towards behavioural medicine (14). The main thesis is that factors that control behaviour also control health. It is the choice of health damaging behaviour that damages health, for instance by choosing to inhale toxic substances or physical inactivity. The cognitive brain mechanisms that determine the choice of behaviour follow the rules from learning theory: What expectancies of outcome have been learned, what are the rewarding factors for the individual, what are the chances of success, and how much energy is to be invested in one particular behaviour.

**DISEASE AND ILLNESS**

To us, it seems necessary to distinguish between somatic disease and illness. We use the term subjective health complaints (15,16) for complaints where there is no apparent somatic reason for the complaint. In the extreme form and with long duration, these conditions come under the psychiatric diagnosis “somatoform disorder”. Comorbidity with other psychiatric diagnoses
Learning, therefore, is a crucial element in the Cognitive Activation Theory of Stress, and in our concept of mechanisms for social inequalities in health.

Learning depends on resources and reinforcement. Under experimental conditions these factors can be manipulated. We can arrange a situation where there is no possibility of solving the problem no matter what the individual does. The individual will then learn a response outcome expectancy called “helplessness” in the classical description by Overmier and Seligman (24). This causes both behavioural and biological changes. The animal learns that nothing helps, the animal becomes inactive, but has high catabolic activation (5). In this condition ulcers are developed in rats, cardiac arrests in pigs, and there is reduced resistance to infections, including cancer-causing virus (25,26).

Both man and animals can learn a third type of expectancy called “hopelessness” (5,7). This means that there is a connection between the responses and the outcome, but the outcome is negative. No matter what the animal or the person attempts, the outcome is negative or non-preferable. This is then a negative response outcome expectancy. This again causes sustained activation, and can produce the same organic diseases as for helplessness (25,26).

Social inequalities in health, therefore, may derive from sustained stress responses giving somatic pathology directly. It may also derive from learned behaviour causing behaviour that affects health indirectly. The man or animal that has acquired a negative response outcome expectancy like hopelessness, and those that do not see any connection between responses and outcome (helplessness), will adjust behaviour according to these expectancies. There is no reason to be physically active if it does not help, there is no reason to reduce fat content in food if I do not have any reasonable expectancy of this affecting my life. Negative outcome expectancies affect all abilities to respond to new challenges, which may be an important factor for the health problems in Eastern Europe (27).

Both of these non-preferable response outcome expectancies are related to depression and anxiety, both have been offered as models for depression and anxiety (5,7,24). Helplessness includes more anxiety, hopelessness more depression, and is, therefore, a better model for depression (7). Both expectancies relate to evaluation of one’s own health as poor, which is a strong predictor of mortality (28). It is important to emphasise that this is not necessarily a psychiatric depression, it is a negative expectancy of what will happen, therefore, related to pessimism, mood change, and inability to respond to new challenges.

**Ontogenesis**

The response expectancies can be influenced by experience all through life, but early experiences may be crucial for all subsequent learning (5). The relationship between mother and child represent a coping structure.
for both parties (29). It is reasonable to assume that offspring that learn positive outcome expectancies early in life will be more active and be more resistant to stress throughout life (5).

Data from man is less controlled than data from animals, but there is no reason to doubt that coping is an important factor for children (29). A coping child learns that it can cope with challenges, and is rewarded for approaching new and challenging tasks because it expects success. Such reward experiences are not distributed at random across social class. We suggest that this results in social inequality in the distribution of learning and coping, hence in expectancies, and eventually in health risk factors. Success in learning, or failure, will be of crucial importance for the future coping efforts and learning. Social inequalities, therefore, may be established in pattern in childhood, and augmented through additional life experiences resulting in the differences in health.

COPING AND SOCIAL STATUS

In man and animals, a social order is established that reduces insecurity, and reduces hostility and conflicts. This has biological survival value in most or all species. To illustrate this, we may look at a species living in an extreme environment, where the survival value is easy to observe. The Arctic grouse (ptarmigans – lagopus lagopus lagopus) establishes a social structure during winter (30). The dominant male has low cortisol, high testosterone, high secondary sexual characteristics, and lower body temperature than males of lower status. This implies that after social order has been established and aggression is reduced, the catabolic stress will diminish in the entire flock, but most in the dominant male. Energy is conserved and improves chances of survival in Arctic winters. In summer the flock disperses and couple up over large territories, improving the chicks’ chance of survival (30).

We find similar conditions in higher species. The general finding is that the dominating or “alpha” male has high testosterone, low cortisol, and low catecholamine levels. In the literature the explanations are often tied to the stress concept. The frustrated losers or suppressed individuals have raised cortisol and catecholamines because they are more “stressed” (31). Our explanation is that the psychoendocrine differences are due to the high positive coping level in the successful male. The dominant male has a high level of positive response outcome expectancies, and therefore, low catabolic activation (32). Similar findings exist regarding social organisation in children. Conflict solving and established social order reduces psychoendocrine and psychoimmune activation (29).

HEALTHY WORK

Karasek and Theorell (33) have presented the most well known model for the relationship between factors in working life and health. The health effects depend on demands and control. In particular, the level of influence on decision making is important (“decision latitude”). We argue that the most important variable is not control or decision latitude per se, but the type of expectancy linked to the situation, and the decision strategies available (34).

For the present argument, the crucial issue is the unequal distribution of the chances to develop positive expectancies. The early experience matters, and it moderates the abilities of the individual, as well as later to the characteristics of the job and the jobs available for the individual. Social class influences the chance of developing positive expectancies (coping), negative (hopeless), or the no relation (helpless) expectancy. Reward factors also differ with job characteristics and social class. A particularly relevant point seems to be disproportions between effort and reward, which seems to constitute a particular health risk (35). A poor learning climate is also a risk factor for ill health, and may to some degree be alleviated with work environment reform (36).

LEARNING LIFE STYLE: REINFORCEMENT CONTINGENCIES AND EXPECTANCY

We believe that it is easier to convince a coping individual of the benefits of a healthy life style than someone that does not really expect good results from his or her acts. This accentuates social differences. Success breeds success, the coping individual has more to loose, and a better expectancy for results of his or her actions. The coping individual has by definition a high level of perceived internal control of reinforcement (37). The social inequality in the distribution of internal versus external control of reinforcement (37,38) is to some extent only describing facts of life. The director has more influence than the manual worker. The locus of control influences the choice of behaviour and trust in results, and, therefore, acceptance and compliance to health improving programs. Our experience is that those who do not participate in physical activity are the ones that need it the most (39,40). Non-participants in physical activity report age, gender, weak physical condition, and high levels of subjective health complaints to be reasons for their non-participation, and we found these resistance factors most pronounced in persons with low status jobs with high levels of sickness leave (22).

CONCLUSION

In our view, social inequalities in health depend to a large extent on social inequality arising from the distribution of learned expectancies of relationships between acts and results. High levels of what we refer to as helplessness (no relation between acts and consequences) or hopelessness (expectancy of negative
results of acts) influence the health via two main pathways.

The direct somatic route is mediated through sustained stress responses, what we refer to as “sustained activation”. This may lead to somatic pathology via known pathophysiological mechanisms. The other route is indirectly via effects on life style. This route is perhaps the most important, and the most deadly. Both paths for health depend on cognitive brain mechanisms that are developed through differences in reward conditions and the individual’s resultant learning.

Both paths give opportunities for preventive measures. Interventions may be directed towards the organisation of work, for instance via improved learning climate and learning opportunities. Interventions may also be directed towards the individual, strengthening the opportunities of learning positive expectancies. Both aspects are important for developing a healthy work environment and an education system that at least gives more people a chance to develop positive expectancies.

There is an unsolvable paradox that increased efforts to improve health may increase the social inequalities in health. Some of us profit from new information, some do not. More emphasis should be directed specifically towards the individuals that do not normally benefit from information, either because of low education, or low motivation for change (22).

REFERENCES