Psychological conditions are increasingly being recognized as having an impact on the physical health of the individual. In this module, we consider some of those factors.
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References

  ► Chapter 9: Affective states and health
  ► Chapter 11: Experimental psychosocial interventions

There is growing body of literature linking emotions to the occurrence of chronic diseases. Much of this research has to do with the development of and mortality due to heart diseases and the links to negative emotions, such as anger, hostility, aggression, anxiety, depression and others.
Psychologists and psychiatrists, really from the beginning of the last century but picking up in the 1940s, tried to explore some of the relationships between psychological issues and health, in particular, disease outcomes. Their early theories were largely personality theories. They speculated that there was a unique personality that was associated with different physiologic disorders. They studied large populations, in a real epidemiologic sense, to try to tease out these relationships. Weak organ theories then followed. They were relating stress to the existence of a weak organ, and they suggested that stress would produce a breakdown in that weak organ system. These theories were often referred to as diathesis-stress models of health.
Type A behavior was one of the very early emotion-based formulations of how social determinants might be influencing health. It was developed by Rosenman and Friedman during the late 1950s and through the 60’s; it remained a popular concept even up until the 80s. Rosenman and Friedman were actually cardiologists who were also researchers. They noticed, Friedman and Rosenman, that the furniture in their cardiology practice’s waiting room was wearing out, so they sent it out be re-upholstered. The fellow who did the work came back and said, “There was a really interesting wear pattern on your furniture.” “Most furniture,” he said, “wears out on the back of the seat and the back of the furniture itself. But your furniture is wearing out on the very front edge of the seat.”

The two physicians started thinking about why that might be. They noticed, as they started observing their patients more closely, that there was a lot of impatience and, when the patient was really challenged, a lot of hostility. They also noted hyper-aggressiveness, a sense of time urgency and, in many cases, pressured speech and a tendency to interrupt in conversations.

The early assessment measures for type A behavior really were based on audiotaped structured interviews during which the interviewer would adopt a challenging behavior in the structure of the questions. Trained staff would review the audiotapes and rate the participant’s behavior. It was a fairly arduous process.
to actually do the assessment and then score the audiotapes from these interviews, so people later tried to come up with self-report scales, briefer ways of trying to characterize what was going on.
The risk associated with type A behavior was confirmed during a number of very large-scale epidemiologic studies of both patients and populations at risk for developing heart disease. It seemed to be a fairly stable concept during the 1960s and through the early 80s, and people embraced it. But in the 1980s there were some large-scale cohort studies which failed to confirm type A behavior as a risk factor. Since then, other studies that have tried to look at it have failed to confirm that the concept is a real risk factor. It did not matter whether the researchers used the original interview method or used some of the self-report scales that were developed later; the concept did not hold up as a risk factor.
The question you should be asking yourself is: “Why was there such a shift? Why did a construct that seemed to be so consistent in being validated as a risk factor for coronary heart disease across multiple epidemiologic studies pretty suddenly shift to negative findings in additional later studies?” There are probably a number of reasons. One of them may be that there were measurement difficulties stemming from the use of taped, structured interviews. It may be that the training of raters of those audiotapes shifted over time, so that we were not really picking up on the same behaviors and scoring them in the same way as those working for Friedman and Rosenman had done in the early years.

It is also possible that as researchers shifted to self-report measures to try to measure the construct, they were not measuring the same construct that was originally assessed with the audiotaped interviews. But even when people did try to replicate the audiotape method, there was a failure to validate the construct. It just was not a significant predictor of coronary heart disease in those studies.

Thus, people began to think that we should be measuring the underlying emotional construct that really account for the risk in what was earlier described as type A behavior. People began to look at anxiety and anger and other emotions that they thought were associated with this behavior pattern.
They also looked at sub-components of these emotions. For example, anger can be broken down into “anger-in,” where people try to control their anger and not express it, versus “anger-out,” where people become overtly angered. Later, researchers explored the thought that it might be the case that there is really a constellation of negative affect associations linking emotion and coronary heart disease and, perhaps, other health outcomes as well. That position really characterizes our current thinking about emotions and the development of coronary heart disease.
We remain challenged to understand the associations that appear to exist between emotions and health. These challenges fall into two basic categories. First, there is not a single clear biological pathway to explain the associations that we observe. Second, as there were with type A behavior, there are significant methodological issues, particularly with regard to measuring emotions.
There is still some question about how emotions work to influence health outcomes. One proposed path is the physiological, cognitive and behavioral responses to those emotions. It is thought that emotions may have direct physiological effects on the development of disease or maintenance of health through biological alterations that occur. When people are angry or anxious, for example, their blood pressure may rise, chemicals may be released in the brain. These may occur because of the cumulative effects of repeated emotional experiences or because of an acute emotional episode.

It is also thought that emotions can influence health by motivating or inhibiting health-relevant behaviors. For example, when people are angry or anxious or depressed, they may smoke or consume alcohol; they may eat more or exercise less.
Similarly, there are at least 3 methodological, or measurement, issues in understanding potential associations between emotions and health. First, many of these negative emotions – anger, hostility, anxiety, depression, others – are related to one another. For example, anger and hostility are sometimes expression of an underlying depression, and anxiety and depression often co-exist. Thus, it is difficult sometimes to figure out which is the emotion that is the real risk factor for the health outcome. Second, they are somewhat subjective constructs with regard to measurement. There are no lab tests for anxiety or depression; they are typically diagnosed using interviews and clinical judgment. There are, for some of the constructs, screening tools that provide preliminary assessments, but these tools – which are typically formatted as yes-no or agree-disagree questions – are not sufficiently sensitive (that is, they generate a lot of false positives and false negatives) if you are looking for a definitive determination.

The other issue that comes to mind with regard to these emotional states is whether you are dealing with a chronic condition or a more transient, time-limited condition. For example, anxiety can be either a temporary, transient state – perhaps in reaction to an event or circumstance – or it can be a more chronic condition for the individual. Similarly, depression can be temporary – perhaps as a reaction to a life event, such as the death of a loved one or an acute health event, like a heart attack – or it can be a more chronic condition. Psychologists talk about this as “state” (temporary, reactive condition) versus “trait” (chronic condition, perhaps a
personality trait). This distinction is important because it interacts with the measurement issues (are you assessing the trait or the state?) and with your conceptualization of the pathways and mechanisms of influence.
We cannot talk about psychosocial determinants of health without spending some time talking about stress. It is defined as “a state of mental or emotional strain or tension resulting from adverse or very demanding circumstances.” We talk about it a lot in relation to health, and it is often conceptualized as being a mediator for other, more up-stream, determinants. For example, think of the effect of poverty on health being, at least in part, related to the stress generated by being poor and not having sufficient resources to take care of basic daily needs for one’s family. Another example is the hypothesis that living beyond your means creates stress which impacts health. You can probably come up with many, many other examples.

There are two components of the stress-health relationship. The first is the magnitude of the stressor – how large it is. This is based, of course, on an assumption that stressors can be reliably characterized on a continuum from ‘small’ to ‘large’. The second is the capacity of the individual to manage the experience. This is based on an assumption that individuals appraise stressors differently and that the way you perceive the stressor affects your capacity to deal with it with more or less strain. This explains, at least theoretically, why, when faced with the same situation, some individuals are relatively unaffected while others collapse under the strain.
As with many of these psychosocial determinants, stress is a very subjective construct, one that depends on the perception of the individual and one that is hard to empirically define and measure. It is difficult – if not impossible – to predict when demands will exceed an individual’s capacity to cope, or which individuals will have sufficient coping skills and under what circumstances.

What we do know is that people cope with stress in different ways and that coping strategies can mediate or mitigate the effect of the stress on the body (and therefore on health). Coping strategies can be positive (e.g., talking with friends, exercising) or negative (e.g., smoking, excessive use of alcohol or other substances).
Programs seeking to modify psychological factors are relatively common. Examples include programs at multiple levels – programs targeting individuals or families; programs offered at the community level; programs working within social networks or workplaces; and even programs that purport to address the issue at the larger population level. These programs are often focused on stress or time management, anger management, anxiety reduction, coping skills, recognizing and dealing with depression, and the like.
There are any number of ways to approach psychosocial interventions. Many are focused on modifying specific health-related behaviors that are risk factors for disease onset or recurrence. Unfortunately, the targeted behaviors have most often been treated as discrete, voluntary, and individually modifiable lifestyle choices, detached from the social context in which behaviors arise. Ignoring the social context and the underlying reasons for those behaviors potentially limits the long-term effectiveness of the intervention. For example, if the individual is over-eating because of stress and you do not either deal with the stress or recognize it and provide them with a more positive, productive coping mechanism, your intervention may not be effective in the long term.

Interventions designed to bolster social support or to provide specialized types of support have been primarily conducted at the individual level. These interventions have ranged from support groups (led by professionals or by lay leaders) to support mobilization interventions, but overall they have shown mixed results when the outcome was an improved health outcome.

Disease management interventions target psychosocial aspects of illness to enhance the patient’s ability to cope with the disease or prevent symptom recurrence. The emphasis has been on providing specific coping strategies designed to address particular problems encountered in the course of the disease. A number of programs have focused on adherence to medical recommendations, including medication adherence.

Distress mitigation interventions aim to reduce distress, with a wide variety of
techniques including but not limited to cognitive behavioral therapy, relaxation training, and patient education. The primary target of change is the experience of stress or distress.
Control and efficacy enhancement interventions are designed to modify the individual’s sense of control over events, thus reducing the experience of stress. The goal is to provide a sense of mastery and control. Typically these have been focused at the individual level but the same idea can be applied to increase collective efficacy at the community level. Collective efficacy interventions have typically placed an emphasis on increasing community resilience through the mobilization of existing resources and community empowerment.

Some have viewed workplaces as an excellent venue to address psychosocial issues, particularly with regard to occupational stressors. These interventions address worksite conditions, attempting to improve productivity in the workplace, reduce turnover and absenteeism, and improve mental health in employees.
We believe it is important to think of psychosocial factors in the context of the socio-ecological model. We tend to think of them as individual level factors. As we have discussed, however, it is possible to think of them in terms of other levels – the use of social networks to provide social support and mitigate their effects; the delivery of policy and behavioral interventions within workplaces to reduce or mitigate occupational stress; the consideration of increasing community efficacy and resiliency as a way to mitigate some of the stress experienced by community members. Interventions for what might be considered an individual risk factor do not need to target the individual directly.
Thought questions

- What did you read or hear in this discussion that was new information for you?
- What surprised or challenged you?
- What did you agree with or disagree with and why?
- How does this information make sense in terms of your work in the field of public health?