



Health and stress

(week 2)

Course code: Dent325

The impact of stress on Cardiovascular Disease

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Outlines

- Source of stress
- Response to stress
- Stress and heart disease
- Stress predictors
- Research data

Objectives

- Count the sources of stress
- Count and compare the general responses of stress.
- Analyze the available research data

Source of stress

- Stress is a normal part of life.
- physical causes like not getting enough sleep or having an illness.
- Emotional, worrying about not having enough money or the death of a loved one.
- less dramatic causes like everyday obligations and pressures that make you feel that you are not in control.

Common responses to stress include:

- Aches and pains
- Decreased energy and sleep
- Feelings of anxiety, anger, and depression
- Impatience
- Forgetfulness

- People respond to stressful situations differently.
- Some react strongly to a situation.
- Others are relaxed and unconcerned. Luckily, you can decrease the effect of stress on your body.
- Identify situations that cause stress. Although difficult, try to control your mental and physical reactions to these stressful situations.

Stress Can Increase Your Risk for Heart Disease



- the high levels of some stress hormones e.g. **cortisol** from long-term stress can increase blood cholesterol, triglycerides, blood sugar, and blood pressure (common risk factors for heart disease).
- stress can cause changes that promote the buildup of plaque deposits in the arteries.
- Even minor stress can trigger heart problems like poor blood flow to the heart muscle (a condition in which the heart doesn't get enough blood or oxygen).
- long-term stress can affect how the blood clots (makes the blood stickier and increases the risk of stroke).

The major manifestations most often studied

- Include angina pectoris, myocardial infarction, and sudden death.
- It is necessary to **maintain a separation among these** since the risk factor may be clearly different.
- Thus, indices of neuroticism, anxiety, and distress relate prospectively to **angina**, while they appear to be only the consequence of MI.
- E.g: distinguishing between **sudden death** (the first manifestation of coronary heart disease) and **coronary heart disease mortality** in general, which reflects death certificate diagnosis irrespective of presence of previous morbidity.
- The latter **may be sensitive to many variables that influence case fatality, including socioeconomic status, medical care, depression**, and so on.

Stress predictors (research data)

- Prospective studies “include multivariate adjustments for established risk factors” particularly age, blood pressure, serum cholesterol, smoking status have yielded the most convincing **“stress” predictors**.
- It was noted that for MI, a certain cluster of symptoms and complaints (poor sleep, exhaustion, inability to relax) appears as a confirmed risk factor.

- For example, **social mobility** seemed to point to promising classes of environmental exposures
- The concept is defined by a multitude of attributes: hard-driving effort, striving for achievement, competitiveness, aggressiveness, haste, impatience, restlessness, alertness, hurried motor movements.
- It is conceptualized as the result of a **predisposition, stimulated by appropriate environmental challenges.**
- **European** male patients with heart disease report excess stresses in the work setting, while **American** male patients report excess stresses in the family setting.

Mechanisms Underlying the Relationship between Work Stress and Coronary Heart Disease

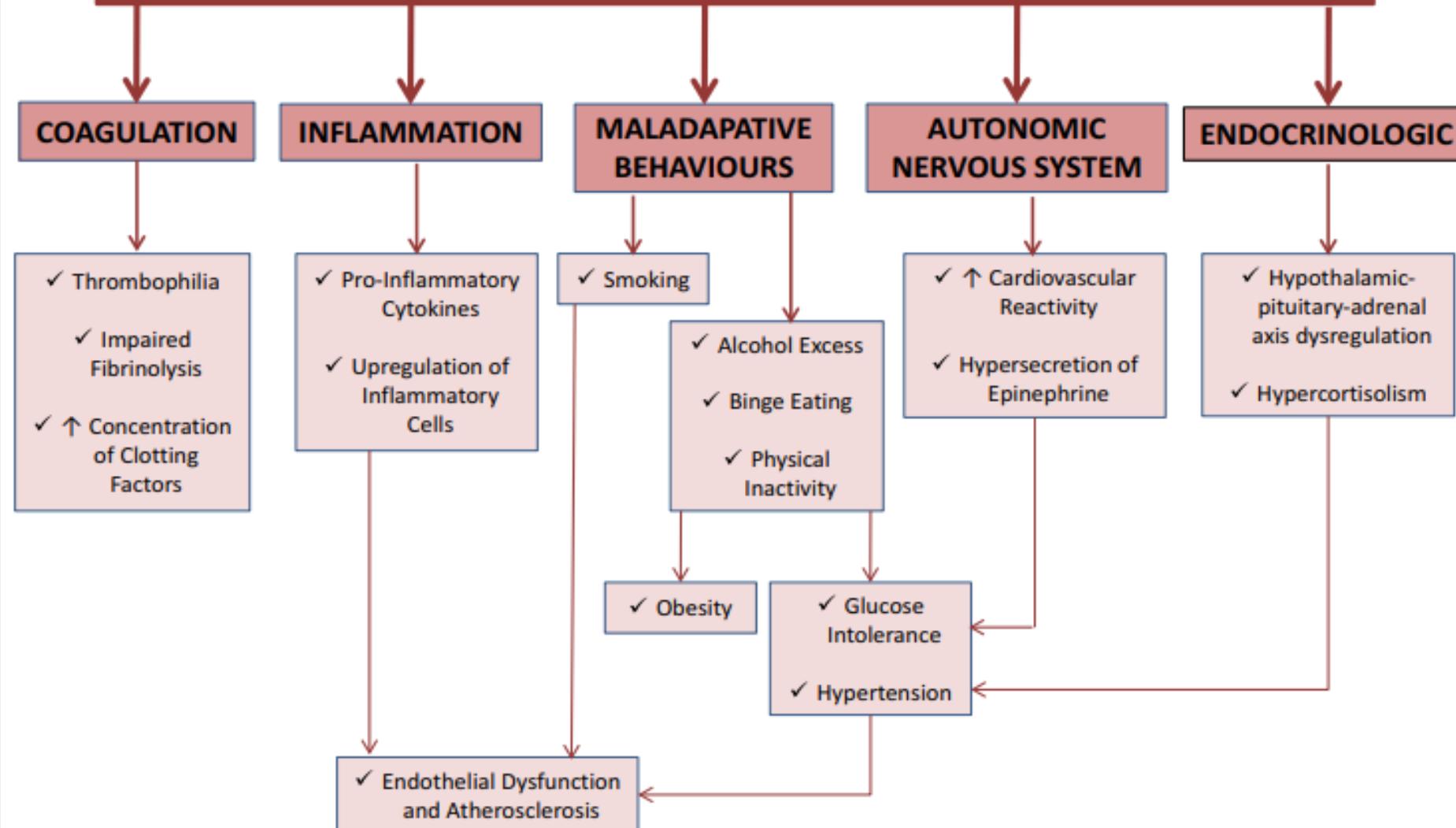


Figure 3. Potential mechanisms by which work-related stress may lead to coronary heart disease.

References

- Margolis *et al.*, 1983, Type A Behavior: An Ecological Approach, *Journal of Behavioral Medicine*, Vol. 6, No. 3.
- Sara *et al.*, 2018, Association Between Work-Related Stress and Coronary Heart Disease: A Review of Prospective Studies Through the Job Strain, Effort-Reward Balance, and Organizational Justice Models, **Journal of the American Heart Association** Volume 7, Issue 9.