STRESS AS A RISK FACTOR IN OCCUPATION-RELATED CARDIOVASCULAR DISEASES

Narcisa Carmen Mladin, MD; Radiana Marcu, Psy. Dr.; Maria Puschita, PhD, MD, "Vasile Goldiş" Western University of Arad

Abstract: In this paper we aim to investigate the role of occupation-related cardiovascular diseases and stress. We focus on three main variables: perceived stress, stress adjustment and coping strategies. We investigated two groups, each including sixty individuals, one with occupation-related cardiovascular diseases and one without any occupation-related medical conditions. The results show that people with occupation-related cardiovascular diseases tend to show higher levels of perceived stress and lower levels of stress adjustment. Also, they focus on passive coping strategies such as denial, emotional support, mental and behavioural disengagement and venting of emotions, while people without occupation-related diseases focus on active strategies such as direct action, suppression of competing activities, positive reinterpretation and social support. Furthermore, we reckon that occupation-related cardiovascular diseases are factors making people vulnerable to high levels of perceived stress and low levels of stress adjustment.

Keywords: occupation-related cardiovascular diseases, perceived stress, stress adjustment, coping strategies, bio-psycho-social approach.

Stress and occupational diseases

The term “stress” was initially used in a medical context by Hans Selye, in 1936, to designate “the state in which one finds an organism threatened by imbalance under the action of agents or conditions which endanger its homeostatic mechanisms.” (Sillamy, 2000, p. 301). A. von Eiff, as quoted by Iamandescu (2002; p. 5) defines stress as a “psycho-physical reaction of the organism generated by stressors acting on the brain through sense organs, and setting into motion – due to cortical-limbic links with the hypothalamus – an entire series of neuro-vegetative and endocrine reactions, reverberating throughout the organism” (figure 1).
The term stress covers three distinct meanings: (a) stress-generating agent or stressor; (b) psycho-behavioural mechanisms activated by the stressor and (c) the organism’s reaction to the stressor (Băban, 1998; Maier, 2011). In everyday language, stress refers only to the negative, dysfunctional, maladaptive aspects of stress (distress). But, as argued by Iamandescu (2002), there is also a positive form of stress: eustress. If distress designates “stresses with a harmful potential for the organism” (Iamandescu, 2002; p. 10), eustress signifies “a state of stress, validated through an accompanying moderate catecholamine and cortisol reaction” (Iamandescu, 2002; p. 12). The difference appears in the neuro-endocrine and psychological response.

Figure 1. Psychological stress model according to Iamandescu (2002; p.8)
The bio-psycho-social unit model of human individuals, enjoying strong scientific validation at this time (Peterson, 1997), describes and explains mechanisms through which reversible or irreversible neuro-psychological changes generated by the presence of stressors may bring about episodic or chronic physiological and somatic changes. Any emotional response triggers, through neuro-hormonal pathways, a series of physiological responses which, if occurring for a long time, may lead to functional disorders and organic lesions. The organism’s psycho-physical and behavioural responses to stress may be represented in a 5-stage model: (1) reception of information through sense organs and its analysis in the cortex; (2) emotional response ensuing from the analysis and interpretation of stimuli; (3) generation of neuro-vegetative and endocrine reactions ensuing from activate emotions; (4) feedback between the cortex and limbic system; (5) activation of neuro-vegetative impulses and hormonal secretions (Iamandescu, 2002).

An important role in this cascade of psycho-physiological responses is played by perception on the degree of control, as well as the type of behaviour activated. Thus, in case control is threatened, but not lost, an active reaction of fugue or fight, defence or attack, appears which, at a neuro-hormonal level, involves such formations as the amygdala, the hypothalamus and the medullar area of adrenal glands, resulting in an intense secretion of adrenalin and noradrenalin and a moderate one of cortisol. In case control is perceived as low or absent, a reaction of abandon and passivity appears, inhibiting organized behaviour and involving the nervous structures of the septum and hippocampus, which, at a neuro-hormonal level, is translated into the activation of the pituitary-corticoadrenal system, resulting in increased levels of cortisol and adrenocorticotropic hormone (ACTH) and decreased levels of testosterone, with moderate catecholamine action (Iamandescu, 2002).

Moderate or pronounced increase in cortisol secretion present in both stress responses induces a series of changes at tissue and functional level, in all apparatuses and systems of the organism. At cardiovascular level, increased levels of cortisol entail: increased cardiac output, arterial wall sensitisation to catecholamines (increased peripheral vascular tone), increased blood volume, impaired capillary microcirculation. Catecholamine secretions involved in stress response also generate cardiovascular effects: vasoconstriction in case of α receptors or vasodilation in case of β receptors. Catecholamines were found to play an important role in the
organism’s stress adjustment, at a psycho-behavioural, metabolic and hemodynamic level (Iamandescu, 2002).

Epidemiological data (Iamandescu, 2002; Steptoe and Kivimäki, 2012) show that chronic stress is a precursor of CVDs. Individuals who undergo intense or chronic occupational stress show numerous CVD symptoms. Moreover, in the case of individuals with arteriosclerosis, emotions induced by a stressful episode may also induce major cardiovascular dysfunctions. Takotsubo (Steptoe and Kivimäki, 2012) identified a specific stress-induced cardiovascular syndrome, transient apical ballooning syndrome, which he called stress-induced cardiomyopathy. Acute stress episodes may induce transient myocardial ischemia, whereas chronic stress may induce recurrent, life-threatening cardiovascular episodes.

Dimsdale (2008) argued that acute and chronic stress is a major risk factor in the following cardiovascular diseases: myocardial infarction, myocardial ischemia, cardiac arrhythmia, as well as alterations in heart functioning as a result of sudden or long-term changes in sympathetic nervous system functioning. These effects are mediated by a series of sanogenic or pathogenic factors at both a somatic and physiological level, such as personal or family cardiovascular history, serum cholesterol and triglyceride levels, as well as psychologically and psycho-socially, such as subjective perception of stress, personality traits such as optimism, self-efficiency, robustness, psychopathological history, lifestyle (smoking, alcohol consumption, diet, sleep) (Băban, 1998; Iamandescu, 2002; Maier, 2011).

HTN and ICD are the most frequent cardiovascular diseases. In Romania, approximately 40% of the country’s population is diagnosed, at some point, with one of these two diseases. Romanian legislation in the field classifies them as occupation-related diseases when they are caused by occupational risk factors.

HTN is defined as blood pressure values over 140/90 mmHg and constitutes one of the most frequent causes of cardiovascular death, accounting for ca. 50% of overall cardiovascular mortality. Thus cardiovascular mortality is doubled for every increase in diastolic blood pressure by 10 mmHg and in a systolic blood pressure by 20 mmHg. (Boutouyrie P et al. Amlodipine-valsartan combination decreases central systolic blood pressure more effectively than the amlodipine-atenolol combination: the EXPLOR study. Hypertension 2010; 55:1314–1322)
Increased blood pressure is the result of the relation between cardiac output and peripheral resistance. Cardiac output is determined by the intrinsic contractility of the myocardium, heart rate, preload, nervous system activity and heart valve competence. Vascular resistance is determined by blood viscosity and the length of the arterial segment and inversely proportional to the radius of the vascular lumen. (Carmen Ginghină, *Mic tratat de cardiologie*, editura Academiei Române, București 2010, Factorii de risc cardiovascular p.213)

The term ischemic cardiomyopathy is synonymous to that of ischemic heart disease. Ischemic cardiomyopathy is a myocardial impairment due to an imbalance between coronary blood flow and myocardial needs, due to changes in coronary circulation. Ischemic cardiomyopathy is defined as a coronary disease causing chest pain triggered by stress or physical effort, as a result of narrowing in the common trunk by over 50% and in one or several coronary arteries by more than 70%. (Greenland P et al. 2010 *ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines*. J Am Coll Cardiol 2010; 56: e50-e103.)

This pathology is manifested most of the times in reversible episodes between myocardial supply and demand and in most cases it is due to ischemia or hypoxia generated by effort or stress, angina pectoris, but may also appear spontaneously, transiently. Coronary disease also includes, however, a stable, asymptomatic phase, which may appear after acute coronary syndrome. Since the transition from the unstable form of coronary syndromes to the stable one does not have a clear boundary, angina pectoris at rest caused by coronary vasospasm can be classed as suspected coronary disease. (Fox K et al. *Guidelines on the management of stable angina pectoris: executive summary: The Task Force on the Management of Stable Angina Pectoris of the European Society of Cardiology*. Eur Heart J 2006; 27: 1341-1381.)

**Purpose, objectives and hypotheses**

The purpose of this research is to identify the role of psychological factors in the risk of developing occupation-related CVD. In this respect we identified three variables: (1) perceived stress – mediates the relation between objective stressors and the individual’s subjective response; (2) stress adjustment – reflects behavioural flexibility manifested by each individual in response to the action of stressors and ability to integrate stressors in one’s existence in order to
re-establish homeostasis and (3) stress coping as reflected in active and passive styles – explains strategies used by each individual in the stress adjustment process.

Pursued objectives are: (1) to identify the role that stress perception may play in increasing individual vulnerability to occupation-related CVDs; (2) to record and measure differences existing between those with occupation-related CVDs and those without such diseases in terms of stress adjustment capacity and (3) to identify differences in coping strategies between subjects in the occupation-related CVD group and those in the other group.

Tested hypotheses are:

- **Hypothesis 1.** We infer that there are significant differences in the level of perceived stress, stress adjustment capacity, as well as coping strategies, between Group I TARGET and Group II CASE, as follows:
- **Hypothesis 2.** There is a strong negative association between stress perception and stress adjustment capacity, in the sense that individuals showing higher levels of perceived stress tend to have lower stress adjustment capacity.
- **Hypothesis 3.** There is a significant association between stress adjustment capacity and the type of coping strategies most frequently activated, in the sense that individuals with better stress adjustment capacity tend to use active strategies more frequently, whereas those with poor stress adjustment capacity tend to activate passive coping strategies.

**Material and method**

In conducting the research, two groups were constituted, each numbering 60 persons. The two groups are not significantly different statistically in terms of distribution by gender, age, marital status and educational level.

(1) The first group of subjects not identified with CVDs, called Group I TARGET, consisted of 30 employees from a wood-processing plant in Arad, SC. MOBIL ART SRL, 15 males and 15 females, aged 35-65; and 30 patients of an individual medical practice in Arad, not identified with CVDs, professionally active, 15 males and 15 females, aged 35-65.

(2) The second group of 60 people was constituted by selection from a number of 429 patients identified with occupation-related CVDs, from the Occupational Diseases and Occupational Medicine Department of the Arad University Clinical Hospital called Group II
CASE, 30 males and 30 females, aged 35 -65, admitted and re-admitted during the 2010-2014 timeframe.

Utilized instruments: (1) Stress perception scale – devised by Levenstein et al. in 1993, assesses subjective stress perceived by the individual (Băban, 1998). (2) Stress adjustment scale – assesses the individuals’ capacity to cope with everyday stressors and adjust effectively to their action. (3) Coping strategies scale (COPE) – devised by Carver, Scheier and Weintraub, in 1989, being based on the stress model proposed by Lazarus (Băban, 1998). The scale measures a set of 14 coping strategies that can be grouped in two broad categories: active coping strategies and passive coping strategies. Active strategies comprise (Băban, 1998; Maier, 2011): active coping, planning, suppression of competing activities, restraint coping, seeking instrumental and emotional social support and positive reinterpretation of the stressor. Passive strategies comprise: acceptance, denial, venting of emotions, religion and/or spirituality, mental and behavioural passivity, and alcohol/drug use.

Results

Subjects in Group I TARGET show a higher average of stress perception than those in Group II CASE, as well as a higher degree of score dispersion. Both average values are placed in the average range. While values in Group I TARGET are entirely placed within the average range of perceived stress (61 – 90), in the case of Group II CASE, 2 subjects showed high levels (>90) of perceived stress. Extreme values in Group I TARGET are higher than extreme values registered in Group II CASE. The two distributions are homogeneous, but there is a statistically significant difference between them: t(110.984) = - 6.553 at p<0.001. Effect size: d Cohen = 1.244.

1. Subjects in Group I TARGET show a higher average level of stress adjustment, but the degree of score dispersion in the two groups is relatively similar. The values of subjects in both groups are found within a relatively narrow range (120 – 161 for Group I and 122 – 153 for group II), comprised within the average level of possible scores. The two distributions can be considered homogeneous, and between them there is a statistically significant difference: t(118) = 3.340 at p=0.001. The registered difference is moderate: d Cohen = 0.611.
2. Subjects in Group I TARGET tend to obtain higher values in active coping styles and lower values in passive ones, as compared to those in Group II CASE. The degree of score dispersion is higher for Group II for both active and passive strategies (Table 1). In terms of coping strategies proper, we observe that Group I obtains higher average values for the following coping styles: active coping, planning, suppression of competing activities, restraint coping, seeking instrumental social support, acceptance and alcohol/drug use. For other coping styles, subjects in Group II obtain higher average values.

Table 1. Descriptive statistical indicators for coping styles

<table>
<thead>
<tr>
<th>Coping type</th>
<th>Group</th>
<th>Average</th>
<th>Standard deviation</th>
<th>Minimum value</th>
<th>Maximum value</th>
</tr>
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<tr>
<td>Active strategies</td>
<td>Group I TARGET</td>
<td>75.02</td>
<td>4.856</td>
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<td>90</td>
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<td>7.805</td>
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<td>Passive strategies</td>
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<td>59.45</td>
<td>5.135</td>
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<td>7.042</td>
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<td>2.084</td>
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<td>Group I TARGET</td>
<td>9.98</td>
<td>1.269</td>
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<td>Group II CASE</td>
<td>9.60</td>
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<td>Suppression of competing</td>
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<td>10.90</td>
<td>1.130</td>
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<td>activities</td>
<td>TARGET</td>
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<td></td>
<td>Group</td>
<td>CASE</td>
<td></td>
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<tr>
<td>Restraint coping</td>
<td>II</td>
<td>9.92</td>
<td>1.871</td>
<td>5</td>
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<td>Group I</td>
<td>TARGET</td>
<td>11.68</td>
<td>1.589</td>
<td>8</td>
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<td>9.72</td>
<td>1.932</td>
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<td>Seeking instrumental social support</td>
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<td>11.27</td>
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<tr>
<td>TARGET</td>
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<td>1.864</td>
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<tr>
<td>Group II CASE</td>
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<td>10.08</td>
<td>1.510</td>
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<td>Group II</td>
<td>TARGET</td>
<td>10.83</td>
<td>1.833</td>
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<td>Group I</td>
<td>9.98</td>
<td>1.600</td>
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<tr>
<td>TARGET</td>
<td></td>
<td>10.25</td>
<td>2.199</td>
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<td>Acceptance</td>
<td>Group I</td>
<td>10.03</td>
<td>1.353</td>
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<tr>
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<td></td>
<td>9.77</td>
<td>2.250</td>
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<td>Denial</td>
<td>Group I</td>
<td>9.68</td>
<td>1.049</td>
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<td>12</td>
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<tr>
<td>TARGET</td>
<td></td>
<td>10.30</td>
<td>1.934</td>
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<td>15</td>
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<tr>
<td>Venting of emotions</td>
<td>Group I</td>
<td>9.18</td>
<td>1.790</td>
<td>6</td>
<td>14</td>
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<tr>
<td></td>
<td>Group I</td>
<td>Group II</td>
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<td><strong>Religion</strong></td>
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<tr>
<td>Group I TARGET</td>
<td>9.53</td>
<td>1.599</td>
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<tr>
<td>Group II CASE</td>
<td>9.90</td>
<td>2.222</td>
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<td><strong>Mental disengagement</strong></td>
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<tr>
<td>Group I TARGET</td>
<td>9.05</td>
<td>1.651</td>
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<tr>
<td>Group II CASE</td>
<td>9.68</td>
<td>2.251</td>
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<td><strong>Behavioural disengagement</strong></td>
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<td>Group I TARGET</td>
<td>9.17</td>
<td>1.475</td>
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<tr>
<td>Group II CASE</td>
<td>9.93</td>
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<td><strong>Alcohol/drug use</strong></td>
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<tr>
<td>Group I TARGET</td>
<td>2.80</td>
<td>0.659</td>
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<tr>
<td>Group II CASE</td>
<td>2.60</td>
<td>0.978</td>
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</tr>
</tbody>
</table>

3. In the case of active strategies, the two distributions cannot be considered homogeneous. Differences between groups are statistically significant: \( t(98.718)=3.989 \) at \( p<0.001 \). Subjects in Group I TARGET tend to obtain higher scores in active coping strategies as compared to those in Group II CASE. The value of \( d \) Cohen (0.73) indicates a moderate effect.

4. In the case of passive strategies, the two distributions can be considered homogeneous. Differences between groups are statistically significant: \( t(118)=-2.575 \) at \( p=0.011 \). Subjects in Group II CASE tend to show higher scores in passive coping strategies. The value of \( d \) Cohen (0.47) indicates a moderate effect.
5. The analysis of differences between the two groups, in the 14 coping styles, indicates statistically significant differences: active coping ($t=3.854; \ p<0.001; \ d=0.71$), suppression of competing activities ($t=3.484; \ p=0.001; \ d=0.63$), restraint coping ($t=6.090; \ p<0.001; \ d=1.11$), seeking instrumental ($t=4.113; \ p<0.001; \ d=0.75$) and emotional social support ($t=-2.446; \ p=0.008; \ d=0.45$), denial ($t=-2.171; \ p=0.017; \ d=0.40$), venting of emotions ($t=-2.843; \ p=0.003; \ d=0.52$), mental ($t=-1.757; \ p=0.041; \ d=0.32$) and behavioural disengagement ($t=-2.194; \ p=0.015; \ d=0.40$). Group I TARGET show higher average scores in active coping, suppression of competing activities, restraint coping and seeking instrumental social support, whereas Group II CASE show higher average scores in: seeking emotional social support, denial, venting of emotions and behavioural disengagement.

6. Overall, there is a statistically significant correlation between stress perception and stress adjustment: $r=-0.212$ at $p=0.01$. Stress adjustment shows significant negative correlation with passive coping strategies ($r=-0.230$ at $p=0.006$). Significant correlations between stress adjustment and active strategies were not identified. All correlations are unilateral.

**Discussions**

Subjects in Group II CASE show significantly levels of perceived stress than those in Group I TARGET (figure 2). Subjects identified with occupational CVDs experience higher stress levels. The difference is high as shown by the effect size indicator, around 1.2 standard deviations. Perceived stress may represent a psycho-individual dimension determined by the configuration of personality traits. A type A behaviour favours the interpretation of events as being strongly stressful, especially when associated with a high level of hostility. On the other hand, the presence of CVDs in one’s history can enhance individual perception of stress by awareness of individual vulnerability induced by the presence of CVDs on the action of stressors.

Subjects in Group I TARGET show higher stress adjustment capacity than those in Group II CASE, that is, those with occupational CVDs (figure 3). The difference is moderate, of only 0.61 standard deviations. Persons without CVDs in their history show better stress adjustment. Those with CVDs in their history tend to adjust more slowly or deficiently, either because their
psycho-physical vulnerability that favoured the onset of CVDs is activated in psychological stress situations, or because the presence of CVDs in history leads to more intense and less reversible psychophysical responses. The very presence of CVDs can be a stress factor for individuals, generating what Iamandescu (2002) calls secondary stress. In this case, too, a type A behaviour may favour a decreased stress adjustment capacity as a consequence of the fact that this type of behaviour is characterised by: competitiveness, distorted perception of time – permanent need for more time, impatience, restlessness, cognitive, affective and behavioural hostility, professional over-involvement (burnout syndrome, workaholism), impulsivity in carrying out tasks, and such traits favour more pronounced neuro-endocrine catecholamine and cortisol responses, which will generate intense and long-term dysfunctions of the cardiovascular system (increased blood pressure, changes in cardiac motility, increased peripheral vascular tone).

Perceived stress and stress adjustment are correlated negatively, in a statistically significant way, to a threshold $p=0.02$ ($r=-0.212$). When we control the effects of the stress adjustment covariant, significant differences between the two groups in terms of perceived stress are preserved ($F=36.254; p<0.001$). The significant difference between the groups in terms of stress adjustment capacity is preserved when we control the perceived stress variable ($F=5.984; p=0.016$). Thus, the presence of CVDs in history is a significant factor for both perceived stress and stress adjustment capacity.

![Figure 2. Differences in stress perception between groups](image1)

![Figure 3. Differences in stress adjustment between groups](image2)
Subjects in Group I TARGET show significantly higher scores in active coping strategies (figure 4), and those in Group II CASE show higher scores in passive coping strategies (figure 5). In both cases the magnitude of differences is moderate. Individuals without occupational CVDs tend to activate active coping strategies more frequently, being focused on solving the problem, either by direct action on the stressor, or by using strategies involving planning, seeking instrumental support (informational and material), or by blocking impulsive or concurrent actions that might negatively affect the efficiency of direct and concrete actions on stressors. Individuals with occupational CVDs tend to focus more on emotional distress and its reduction and less on acting on the stressor. They prevalently seek to reduce emotional distress, either as a purpose in itself, or as an intermediate stage before acting on the stressor.

![Figure 4. Differences in active strategies between groups](image1)
![Figure 5. Differences in passive strategies between groups](image2)

The difference in the case of passive coping strategies is not preserved when the stress adjustment variable is controlled (F=3.175; p=0.056). In the case of active coping strategies, the effect is enhanced when we control the stress adjustment variable (F=13.07; p<0.001). Results indicate that the absence of CVDs seems to play an important role in the use of active coping strategies, whereas in using passive coping strategies a significant role is played by the stress adjustment capacity.

In terms of actual coping strategies the following can be observed:
1) Subjects in Group I TARGET show significantly stronger tendencies to act directly on the stressor, to block distracting activities and impulsive actions and to seek instrumental support in solving problems.

2) Subjects in Group II CASE tend to act primarily and especially to reduce emotional discomfort generated by the stressor by seeking emotional support, denying the stressor, venting emotions or mental and/or behavioural disengagement.

Conclusions
Having validated the tested hypotheses, we can assert that our research shows that there are significant differences between individuals relating to the presence of CVDs in history, perceived psychological stress, stress adjustment capacity, as well as dominant coping strategies.

1) Persons with CVDs in their history perceive stress more intensely under the action of the various stimuli compared to those without CVDs in their history. CVDs can thus be a factor of vulnerability to stress, but may also constitute a consequence of more intense levels of perceived stress, involving, on the one hand, more intense cognitive, emotional and behavioural responses, and, on the other hand, more intense psycho-physical and neuro-endocrine responses which may alter cardiovascular functioning (increased levels of cortisol, adrenalin and noradrenalin).

2) The presence of CVDs in history is associated to decreased stress adjustment capacity. CVDs may be the consequence of decreased stress adjustment capacity, but also a factor that may favour adjustment difficulties. In the former case, the presence of dysimmunogenic physical and psychological traits (type A behaviour, anxiety as a trait, hostility) can favour the development of reduced stress adjustment capacity, involving more intense and long-term effects of stressor action. In the latter case, the presence of CVDs in the history is in itself a variable with a negative role in stress adjustment.

3) CVDs in the history are associated with higher frequency and intensity of activating passive coping strategies, focused on reducing emotional distress. That might be because the presence of CVDs generates more intense physiological responses in the cardiovascular system, as a complement to emotions triggered after the cortical analysis of stressors, which may induce psychological disorganisation of conduct with psycho-
emotional and psycho-behavioural dysfunctions, generating new intense emotions which
the individual wishes to suppress in order to recover emotional balance. But CVDs in the
history may also be the consequence of passive responses to the action of the stressor.
Henry and Stephens (Iamandescu, 2002) argue that passive reaction to the action of
stressors manifests itself when individuals perceive weak or absent control. In this
context, at a psycho-behavioural level, the individual will manifest apathy, mental and
behavioural disengagement, withdrawal from the action of the stressor, avoidance of
confrontation, denial, isolation, dysphoria, even depressive symptoms. At a physiological
level, corticoadrenal and pituitary activity is enhanced, resulting in significantly increased
levels of ACTH, but especially cortisol, and a moderate increase in catecholamine. A
high level of cortisol on the long term has negative effects on the cardiovascular system
(increased cardiac output, arterial wall sensitisation to catecholamine, increased blood
volume, impaired capillary microcirculation).

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