

Acute psychosocial stress reduces pain modulation capabilities in healthy men



Nirit Geva^a, Jens Pruessner^b, Ruth Defrin^{a,*}

^a Department of Physical Therapy and Sagol School of Neuroscience, Sackler Faculty of Medicine, Tel-Aviv University, Tel Aviv, Israel

^b Douglas Mental Health Institute, Department of Psychiatry, McGill University, Montreal, Quebec, Canada

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ABSTRACT

Anecdotes on the ability of individuals to continue to function under stressful conditions despite injuries causing excruciating pain suggest that acute stress may induce analgesia. However, studies exploring the effect of acute experimental stress on pain perception show inconsistent results, possibly due to methodological differences. Our aim was to systematically study the effect of acute stress on pain perception using static and dynamic, state-of-the-art pain measurements. Participants were 29 healthy men who underwent the measurement of heat-pain threshold, heat-pain intolerance, temporal summation of pain, and conditioned pain modulation (CPM). Testing was conducted before and during exposure to the Montreal Imaging Stress Task (MIST), inducing acute psychosocial stress. Stress levels were evaluated using perceived ratings of stress and anxiety, autonomic variables, and salivary cortisol. The MIST induced a significant stress reaction. Although pain threshold and pain intolerance were unaffected by stress, an increase in temporal summation of pain and a decrease in CPM were observed. These changes were significantly more robust among individuals with stronger reaction to stress ("high responders"), with a significant correlation between the perception of stress and the performance in the pain measurements. We conclude that acute psychosocial stress seems not to affect the sensitivity to pain, however, it significantly reduces the ability to modulate pain in a dose–response manner. Considering the diverse effects of stress in this and other studies, it appears that the type of stress and the magnitude of its appraisal determine its interactions with the pain system.

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1. Introduction

Since Selye's definition of stress as a nonspecific body response to any demand for change [47], the concept of stress has undergone constant evolution. The current accepted view of stress maintains that not all conditions that trigger physiological response are stressful/threatening, and that stress is a cognitive perception of uncontrollability and/or unpredictability that is expressed in a physiological and behavioral response [31]. Hence, the evaluation of stress should consider not only the physiological aspects manifested by the response of the hypothalamic pituitary adrenocortical (HPA) axis and the sympathetic adrenomedullary system (SAM) [18,24,31], but also, and especially, the cognitive perception of stress [3].

* Corresponding author at: Department of Physical Therapy, Sackler Faculty of Medicine, Tel-Aviv University, Tel Aviv 69978, Israel. Tel.: +972 3 6405431; fax: +972 3 6409223.

E-mail address: rutidef@post.tau.ac.il (R. Defrin).

Besides the interaction of the stress systems with each other, the interactions with other regulatory systems are likely to further contribute to the overall experience of stress, but many have not been fully established. The acute stress response is regarded as a protective response aimed to mobilize the body's resources to deal with threatening circumstances. Anecdotes on the ability of individuals to continue to perform under stressful conditions, despite injuries causing excruciating pain, suggest that acute stress may induce analgesia/hypoalgesia. Indeed, acute stress in animals, such as inescapable foot shock or exposure to predators, induces hypoalgesia [9,14,29]. In human subjects as well, various models of acute mental stress such as public speaking and erythematic tasks induce hypoalgesia, manifested in increased pain threshold [6,23,59] and reduced pain ratings [23,59].

However, the opposite effect was also reported. Hyperalgesia following acute stress occurred in animals [30,42,45] and in human subjects manifest in decreased pain threshold [10,11,16,43], decreased pain tolerance [10], and increased pain ratings [33].

In other studies, acute mental stress had only a partial effect, for example, in patients but not healthy subjects [12,46], or in women but not men [1,8].

Two factors may explain the controversial effect of stress on pain perception. First, most of the aforementioned studies used pain thresholds and pain ratings, that is, “static measurements.” These measurements evaluate pain response at a particular moment in time and may, therefore, vary greatly within and between subjects [38,50]. Second, most of the aforementioned studies recorded autonomic variables as stress indices but did not measure individuals’ perception of stress. Therefore, there is no certainty that all the participants were under stress according to the current definition [3,31].

“Dynamic measurements,” such as conditioned pain modulation (CPM) and temporal summation of pain (TSP), seem more suitable to test the stress–pain interaction as they evaluate traits of the pain system, particularly of central pain modulation [4,27,57]. We found only 4 studies in which acute stress effect was evaluated using dynamic measurements, showing either reduced pain modulation [39] or no effect [13,16,43]. However, only in the former study was a stress response ascertained based on physiological and cognitive indices. Therefore, the aim of the current study was to systematically explore the effects of acute stress on pain perception, by 1) using dynamic and static pain measurements and 2) ascertaining a stress response by measuring subjective emotional perception of stress as well as HPA-axis and SAM responses.

2. Methods

2.1. Subjects

Participants were 29 healthy male subjects (mean age 33 ± 10 years). We included only male subjects in order to avoid the confounding effect of sex, as sex may affect the manner with which subjects respond to pain and to stress. The subjects were recruited by advertisements posted at the university. Exclusion criteria were: acute or chronic pain, present or previous pathology in the hands (testing site), bruises or any other skin lesions on the hands, diseases causing potential neural damage (eg, diabetes), systemic and mental illnesses (eg, anxiety disorders, depression, bipolar disorder), and communication disabilities. Informed consent was obtained from all the subjects. The experiment was approved by the Helsinki committee of Sheba Medical Center and institutional review board of Tel-Aviv University.

2.2. Equipment

2.2.1. Recording and processing of physiological signals

The physiological signals were recorded, sampled, and stored using a personal computer with the PMD-100 system (Medasense Biometrics Ltd., Ramat Yishai, Israel) through a finger probe. A 1-lead electrocardiogram signal was sampled with a frequency of 500 Hz, and a reflectance-mode photoplethysmogram signal from the right-hand index finger was sampled with the same frequency. Skin conductance (measured in micro-Siemens, μS) was measured using 2 electrodes positioned on the volar pads of the distal phalanx in the middle and ring fingers of the right hand, and was sampled with a frequency of 31.25 Hz. The recorded signals were synchronized and processed off-line using MATLAB R2010 scientific software (The MathWorks, Inc., Natick, MA, USA).

2.2.2. Thermal stimulators

Heat stimuli were delivered using 2 Peltier-based computerized thermal stimulators (TSA II, Medoc Ltd., Ramat Yishai, Israel) with 3×3 cm contact probes. According to the principles of the Peltier

element, a passage of current through the Peltier element produces temperature changes at rates determined by an active feedback system. As soon as the target temperature is attained, probe temperature actively reverts to a preset adaptation temperature by passage of an inverse current. The adaptation (baseline) temperature was set to 35 °C. The probes were attached to the testing site by means of a Velcro band.

2.3. Measurements of the stress response

2.3.1. Perceived stress

Perceived stress was evaluated using a visual analogue scale (VAS). The VAS consisted of a 10-cm line with 2 anchor points at its extremes, set as “no stress” = 0 and “worst imaginable stress” = 10.

2.3.2. State anxiety

Anxiety was evaluated with the short form of the State-Trait Anxiety Inventory [49]. This questionnaire contains 10 items, and subjects are asked to rate the degree to which they experienced each symptom of anxiety at that moment, on a 4-point Likert-type scale (1 = not at all, to 4 = very much so). This measure of state anxiety has been used extensively in previous research and has consistently demonstrated good psychometric properties, especially under conditions of stress [37].

2.3.3. Autonomic variables

The SAM system responds to stress by secreting noradrenaline, thereby increasing sympathetic tone, resulting in changes in heart rate, blood pressure, respiration, skin conductance, etc. The sympathetic response was thus investigated by recording the change in heart rate (HR) and in galvanic skin response (GSR) using the PMD-100 system. HR and GSR were recorded continuously at a rate of 500 Hz during the experiment, and values were extracted off-line for relevant time points, as described below.

2.3.4. Salivary cortisol

The HPA axis is strongly activated by psychosocial stress and secretes the stress hormone cortisol [22]. Saliva samples of cortisol were collected with Salivates (Sarstedt, Rommels-dorft, Germany). Participants were asked to place a roll of cotton in their mouths, chew on it for a minute until it became saturated, and place it in a Salivate. The samples were stored at -20°C until assayed. Cortisol levels were assayed using a commercial enzyme-linked immunosorbent assay kit (Assay Design, Ann Arbor, MI, USA). Measurements were performed in duplicate, according to the kit’s instructions, with the reagents provided. Cortisol levels were calculated using MATLAB-7, according to standard parametric calibration curves based on the data from the kit [54].

2.4. Quantitative sensory testing

2.4.1. Pain threshold and pain tolerance

Pain threshold and pain tolerance were measured with the method of limits, using a thermal stimulator. For each threshold measurement separately, subjects received 4 successive ramps of gradually increasing temperature, starting from a baseline temperature of 35 °C, at a rate of 2 °C/s (interstimulus interval of 30 seconds). For pain threshold, the subjects were asked to press a switch when a pain sensation was first perceived. For pain tolerance, the subjects were asked to press a switch when they could no longer withstand the pain. Pressing the switch resulted in an automatic recording of the threshold temperature and reset the probe temperature to baseline value. Pain threshold and pain tolerance were computed separately by averaging the readings of 4 successive stimuli in each measurement [20].

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