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Alterations in endogenous pain modulation in endurance athletes: An experimental study using quantitative sensory testing and the cold-pressor task

Jonas Tesarz [a,*], Andreas Gerhardt [a], Kai Schommer [b], Rolf-Detlef Treede [c], Wolfgang Eich [a]

[a] Department of General Internal Medicine and Psychosomatics, Medical Hospital, University of Heidelberg, Germany
[b] Department of Sports Medicine, Medical Hospital, University of Heidelberg, Germany
[c] Chair of Neurophysiology, Center for Biomedicine and Medical Technology Mannheim (CBTM), University of Heidelberg, Germany

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ABSTRACT

There is evidence for long-term alterations in pain tolerance among athletes compared with normally active controls. However, scientific data on pain thresholds in this population are inconsistent, and the underlying mechanisms for the differences remain unclear. Therefore, we assessed differences and similarities in pain perception and conditioned pain modulation (CPM) at rest in endurance athletes and normally active controls.

The standardised quantitative sensory testing protocol (QST) of the 'German-Research-Network-on-Neuropathic-Pain' was used to obtain comprehensive profiles on somatosensory functions. The protocol consisted of thermal and mechanical detection as well as pain thresholds, vibration thresholds, and pain sensitivity to sharp and blunt mechanical stimuli. CPM (the diffuse-noxious-inhibitory-control-like effect) was measured using 2 tonic heat pain test stimuli (at the temperature exceeding a subjective pain rating of 50/100) separated by a 2-min cold-pressor task (CPM-TASK; conditioning stimulus). Pain ratings were measured with a numerical rating scale. Endurance capacity was validated by assessment of maximum oxygen uptake (VO_2max). Participants included 25 pain-free male endurance athletes (VO_2max > 60 mL/min * kg) and 26 pain-free normally active controls (VO_2max < 45 mL/min * kg) matched based on age and body mass index.

Athletes were significantly less sensitive to mechanical pain but showed higher sensitivity to vibration ($P < 0.05$). In athletes, CPM was significantly less activated by the conditioning stimuli ($P < 0.05$) when compared with normally active controls.

Our data show that somatosensory processing in athletes differs in comparison with controls, and suggest that the endogenous pain inhibitory system may be less responsive. This finding may explain the paradoxical propensity of athletes to develop chronic widespread pain.

1. Introduction

Pain is a common phenomenon in athletes [3,22,26,51,60,67]. This is paradoxical, as physical activity is part of most multimodal pain treatment programmes. Thus, on the one hand, physical activity might be the origin of a variety of pain syndromes in athletes who engage in rigorous physical activity [3,22,26,51,60,67], whereas on the other hand, physical activity also represents an important therapeutic concept in pain syndromes [20,21,43,55]. Therefore, increased knowledge concerning the role of physical activity on pain perception and processing may impact the medical care of pain patients in general, and athletes in particular.

There has been consistent evidence that after an episode of intense exercise, pain perception is reduced for a limited period of time, i.e., 'acute exercise-induced analgesia' [29,31]. It has been theorised that physical activity activates some generalised endogenous pain-modulatory mechanisms, e.g., conditioned pain modulation (CPM; formerly termed 'diffuse noxious inhibitory control') [5,29], baroreflex-mediated analgesia [7,30], stress-induced hypoalgesia [29], or attentional factors [29,31]. Although different mechanisms have been proposed [29,30], CPM is of special interest, as alterations in this system have been reported for a variety of chronic pain conditions [19,27,28,36,40,41,44,63,71]. Moreover, a deficit in this system is associated with chronic widespread pain (CWP) [44], which is frequently reported in athletes (prevalence 31% [23]).

* Corresponding author. Address: Department of General Internal Medicine and Psychosomatics, Medical Hospital, University of Heidelberg, Im Neuenheimer Feld 410, D-69120 Heidelberg, Germany. Tel.: +49 6221 56 37862; fax: +49 6221 56 8450.
E-mail address: jonas.tesarz@med.uni-heidelberg.de (J. Tesarz).

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To date, research has focused on pain perception during physical activity rather than the potential long-term consequences of regular exposure to physical activity on pain processing at rest. In particular, the endogenous pain inhibitory system is a little-researched issue in athletes and, to date, no data have been published about CPM.

Researchers have postulated that long lasting physical activity may alter pain perception at rest and have often concluded that athletes possess higher pain thresholds and a higher pain tolerance in general [50,53]. A recent meta-analysis confirmed significantly higher pain tolerance in athletes at rest and specific alterations in pain thresholds [57]. But, although some studies have reported elevated pain tolerance or pain thresholds [16,18,56], there are also data demonstrating normal [49] or even lower [45] pain thresholds in athletes. This ambiguity may be because different pain induction methods with non-standardised and non-validated testing paradigms have been used [10,11,16,18,45,49,50,66]. The situation is aggravated because the definition of an athlete in most pain studies has been characterised arbitrarily, and to date, there are almost no pain studies in which physical fitness has been assessed objectively [57].

To overcome some of these shortcomings, this study assessed for the first time pain perception and endogenous pain modulation in athletes using a comprehensive standardised quantitative sensory testing protocol (QST [47]) and an objective evaluation of 'physical fitness.' The aim of this study was (1) to examine whether there are differences in pain perception at rest between athletes and normally active controls, and if so, (2) to determine if endogenous pain-modulating mechanisms are involved. It was predicted that athletes are characterised by specific sensory profiles and that the endogenous pain modulation of athletes is significantly different compared with normally active controls.

2. Methods

2.1. Study population

In the present study, 25 endurance athletes and 26 normally active controls were included. Athletes were recruited from regional sport clubs. Healthy normally active controls were recruited via flyers posted in the local community. Inclusion criteria were as follows: male sex, age 18-35, and without pain. The study sample was restricted with respect to sex and age, as QST and CPM are sex- [9,46] and age-dependent [8,48]. Athletes trained for at least 3 h/wk for more than 3 years and were characterised by a maximal oxygen consumption (VO_2max) >60 mL/min $*$ kg. Controls were age- and BMI-matched, did not engage in regular physical activity, and had a VO_2max < 45 mL/min $*$ kg.

Study participants were screened using a questionnaire, physical examination, and electrocardiogram to rule out acute or chronic pain; in addition, data concerning regular medication use, diseases affecting sensory processing (e.g., diabetes, polyneuropathy) or contraindications to treadmill testing were used to screen patients. Subjects were excluded if they reported any history of injury of the hand dorsum or arm, as this was the area tested in our paradigm. Participants were advised not to take any medication 24 h prior to the investigation and to refrain from intensive or prolonged training on the day prior to each test.

2.2. Instruments

2.2.1. Assessment of athletic performance

Maximal oxygen consumption (VO_2max, mL/min $*$ kg) was measured in a ramp protocol on a motor-driven treadmill (Quasar med, H/P/Cosmos, Traunstein, Germany). After warming-up for 2 min at 4 km/h at an incline of 1.5%, the test began at a speed of 7.2 km/h, and the speed was increased by 0.6 km/h over 30 s until volitional exhaustion. Oxygen consumption was measured using a metabolic card (Ergostik, Geratherm Respiratory GmbH, Bad Kissingen, Germany). VO_2 max related to body weight was considered to be the highest VO_2 over a period of 30 s during the test. Prior to each test, both sensors were calibrated according to the manufacturer's instructions. During the treadmill test, a continuous 12-lead ECG was recorded.

Specifications of physical activity were also captured using a questionnaire that included a detailed self-report of the type, frequency, intensity, and duration of physical activities.

2.2.2. Assessment of pain perception

Somatosensory function was assessed using the comprehensive QST protocol, which was developed as part of the German Research Network on Neuropathic Pain (DFNS) [47]. It covers all relevant aspects of the somatosensory system, including large and small fibre functions, and signs of central sensitisation (dynamic tactile allodynia, punctate mechanical hyperalgesia, and paradoxical heat sensations). In this manner, detailed profiles of somatosensory function for the tested body areas were obtained. The dorsum of the dominant hand was tested.

To familiarise participants with the test procedure, all tests were first conducted over an area that was not tested later during the QST session.

The tests for thermal detection thresholds (warm detection threshold, WDT, and cold detection threshold, CDT), thermal pain thresholds (heat pain threshold, HPT, and cold pain threshold, CPT), and paradoxical heat sensations (PHS) were conducted using a TSA 2001-II (MEDOC, Israel) thermal sensory testing device [72]. All thresholds were obtained using ramped stimuli (1°C/s, 32°C baseline, 0°C and 50°C cut-offs, 8 cm^2 thermode), which were terminated when participants pressed a button. The mean of 3 consecutive measurements was calculated. Thermal sensory limen (TSL), a test with alternating warming and cooling ramps, was used as a provocative test to induce PHS.

The mechanical detection threshold (MDT) was measured with a standardised set of modified von Frey filaments (Optihair$_2$-Set, Marstock Nervetest, Germany), which exert forces between 0.25 and 256 mN [13]. The contact area was of uniform size and shape (round, 0.5 mm diameter). The threshold was the geometric mean of 5 series of ascending and descending stimulus intensities.

The mechanical pain threshold (MPT) was measured using a set of weighted pinprick stimulators with a flat contact area of 0.25 mm diameter, which exert forces between 8 and 512 mN [4]. Again, using the method of limits, the threshold was the geometric mean of 5 series of ascending and descending stimulus intensities.

Mechanical pain sensitivity (MPS) was tested using the same weighted pinprick stimuli as that for MPT. To obtain stimulus response function, these 7 pinpricks were applied in balanced order 5 times each. The participant was asked to rate each stimulus for pain on a 0 to 100 numerical rating scale (0 indicating 'no pain,' and 100 indicating 'most intense pain imaginable'). The geometric mean of the 35 pain ratings was the final value for MPS. Stimulus response functions for dynamic mechanical allodynia (DMA) were determined using a set of 3 light tactile stimulators [4,34]. They were intermingled with the pinprick stimuli in a balanced order, and participants were asked to give a rating on the same numeric rating scale.

The vibration detection threshold (VDT) was determined with a Rydel-Seiffer tuning fork (64 Hz, 8/8 scale), which was placed over the bony prominence of the processus styloideus radii of the dominant hand 3 times. Subjects indicated the time at which they no longer experienced vibratory sensations.