

Reduced pain inhibition is associated with reduced cognitive inhibition in healthy aging



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ABSTRACT

The analgesic effect of heterotopic noxious counter-stimulation (HNCS; “pain inhibits pain”) has been shown to decrease in older persons, while some neuropsychological studies have suggested a reduction in cognitive inhibition with normal aging. Taken together, these findings may reflect a generalized reduction in inhibitory processes. The present study assessed whether the decline in the efficacy of pain inhibition processes is associated with decreased cognitive inhibition in older persons. Healthy young (18–46 years old; $n = 21$) and older (56–75 years old; $n = 23$) adult volunteers participated in one experimental session to assess the effect of HNCS (cold pain applied on the left forearm) on shock pain and RIII reflex induced by transcutaneous electrical stimulation of the right sural nerve. In the same session, participants also performed a modified Stroop task, including a target condition requiring the frequent switching between inhibition and no inhibition of the meaning of color words. The analgesic effect induced by HNCS was significantly smaller in older participants for both shock-pain ratings ($P < 0.001$) and RIII-reflex amplitude ($P < 0.05$). The Stroop effect was significantly larger in elderly participants in the inhibition trials of the switching condition. Increased cognitive interference (ie, larger Stroop effect) correlated with smaller inhibition of the RIII reflex by HNCS across groups ($r = -.34$, $P = 0.025$). This association was independent from the age-related slowing observed in control reading and naming tasks. These results suggest a generalized age-related reduction in inhibitory processes affecting both executive functions and cerebrospinal processes involved in the regulation of pain-related responses induced by competing nociceptive threats.

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1. Introduction

Over 38% of older persons living in health care institutions and 27% living in private households are affected by chronic pain [29]. In addition, a number of cognitive and neurophysiological changes occur during healthy aging, which may contribute to the development of chronic pain conditions. Age-related cognitive changes include a decline in episodic memory [26], decreased working

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memory [32], reduced attention resources (especially selective attention [5] and sustained attention [11]), as well as decreased inhibitory functions [34]. In accordance with these behavioural changes, there is electrophysiological evidence of age-related decline in inhibitory functions related to sensory [24] and cognitive [41] processes. However, a closer examination of these results indicates that age-related effects on executive functions are much less pervasive than generally assumed, and are found more clearly in conditions involving divided attention or task-switching (for review see [37]).

Alteration of endogenous pain inhibition processes has been shown in older persons, and this may contribute to the frequency of chronic pain in the elderly. In these studies, the integrity of pain

inhibition mechanisms was assessed using heterotopic noxious counter-stimulation (HNCS), involving the inhibition of pain by a competing noxious stimulus applied on a different part of the body. Washington et al. were the first to report age differences in HNCS analgesia and increased sensitivity to pain in older adults (mean age, 78 years old) compared with young adults (mean age, 23 years old) [40]. In this study, the threshold of pain induced by electrical stimulation and thermal CO₂ laser was increased after hand immersion in painful cold water in both groups, but less so in the older group. Edwards et al. also suggest an age-related decrease in the efficacy of descending inhibitory controls [10]. In this study, older adults ($n = 48$, mean age = 63.1 years, range = 55–67 years) showed facilitation rather than inhibition of thermal pain during concurrent noxious cold stimulation, while younger adults ($n = 45$, mean age = 21.6 years, range = 18–25 years) showed a reduction in thermal pain ratings during cold pain. A subsequent study examined age-related differences in diffuse noxious inhibitory controls over a wide age continuum and observed decreased endogenous pain modulation starting from middle-aged (between 40 and 55 years old) compared with younger subjects (between 20 and 35 years old), whereas pain inhibition was abolished in older persons (between 60 and 75 years old) [20]. Another study reported that, compared to younger persons (20–49 years), older persons (56–77 years of age) failed to demonstrate HNCS analgesia and even showed heat pain facilitation during HNCS (cold water foot immersion) [30].

HNCS and Stroop involve stimulus competition and solicit top-down regulation processes underlying inhibition. We hypothesize that decreased HNCS analgesia is related to a decrease in cognitive inhibition as evidenced by the Stroop test as a part of a global decline in inhibitory functions with age. To test this hypothesis, the present study assesses the correlation between these 2 declines and examines the specificity of the association by controlling age-related effects not involving inhibitory processes.

2. Materials and methods

2.1. Ethics approval

All experimental procedures conformed to the standards set by the latest revision of the Declaration of Helsinki and were approved by the Research Ethics Board of “Institut universitaire de gériatrie de Montréal.” All participants gave written informed consent, acknowledging their right to withdraw from the experiment without prejudice and received compensation of \$50 for their travel expenses, time, and commitment. The study consisted of a session of 120 minutes, in which questionnaires were administered, thresholds of the RIII reflex were determined, the modulation of pain and RIII-reflex amplitude by HNCS was performed, and the Stroop test was completed.

2.2. Participants

Forty-five healthy volunteers were recruited among participants from the database of “Institut universitaire de gériatrie de Montréal,” and by advertisement on the campus of “Université de Montréal.” Participants were excluded if they presented chronic pain syndromes, psychiatric disorders, neurologic disorders, metabolic disorders (diabetes), vascular disorders (eg, inferior members arteriopathy), or used medication that could alter pain perception and modulation 2 weeks prior to the experiment, including antihypertensives, anxiolytics, antidepressants, and other psychotropic agents. During the screening telephone call, participants were asked to abstain from consuming alcohol at least 1 day before experimentation, and refrain from consuming tea and coffee on the day of the experiment. One participant was excluded because

he could not tolerate the experimental procedures. Two groups of participants were tested, including 21 young persons, 10 women and 11 men, ranging between 18 and 46 years of age (mean age 28.8 ± 9.1 years), and 23 older persons, 13 women and 10 men, ranging between 56 and 75 years of age (mean age 62.9 ± 5.4 years). Based on a self-rated audition and vision questionnaire, all participants reported normal or corrected perceptual abilities (see Table 1).

2.3. Experimental design

This study relied on a mixed design to examine the effects of HNCS on RIII-reflex amplitude and acute shock pain between 2 groups of participants: younger persons ($n = 21$) and older persons ($n = 23$). Groups were also compared on inhibition (Stroop test) and psychological factors (see below). Between RIII-threshold determination and HNCS, participants were also asked to complete questionnaires, the similitude test, and the digital symbol-coding test (subtests of the Wechsler Adult Intelligence Scale [WAIS] III) [25]. This allowed participants to rest from electrical stimulation for a period of approximately 15 minutes.

2.4. Painful electrical stimulation

Transcutaneous electrical stimulation (trains of 10×1 -ms pulses at 333 Hz) was delivered with a custom-made isolated constant current stimulator triggered by a train generator (Grass Medical Instruments, Quincy, MA, USA) and controlled by a computer running E-Prime2 (Psychology Software Tools, Sharpsburg, PA, USA). Degreased skin over the retromalleolar path of the right sural nerve was stimulated by a pair of custom-made surface electrodes (1 cm^2 ; 2-cm interelectrode distance). The RIII-reflex threshold was determined using the staircase method, including 4 series of stimuli of increasing and decreasing intensity [28,43]. Each series always began with an intensity of 1 mA and was followed by increments of 1 mA until the subject reported pain intensity of 70 on the 0–100 pain scale (see below). Stimulus intensity was then decreased by steps of 1 mA. The individual's stimulus-response plot was then created, and the RIII-threshold was determined as the intensity producing a clear response in at least 50% of trials (ie, above noise level according to the individual stimulus-response plot). The intensity of stimulation was then adjusted individually at 120% of the RIII-reflex threshold, and a series of 10 stimulations was administered to insure stability of responses (otherwise threshold assessment was repeated). Stimulus intensity remained constant at 120% of the RIII threshold for the remaining of the experiment. The mean intensity at which the subject began to feel pain determined the pain threshold, and the mean of the stimulus intensity required to produce a rating of 70 on the 0–100 scale was defined as the strong pain level (pain-70). These measures allowed assessing baseline group differences in nociception and pain perception.

2.5. RIII-reflex measure and analyses

Electromyography (EMG) of the short head of the biceps femoris was recorded with a pair of surface electrodes (EL-508, Biopac Systems, Inc., Goleta, CA, USA). The signal was amplified 1000 times, band-pass filtered (100–500 Hz), digitized, and sampled at 1000 Hz. EMG data were analyzed using Acqknowledge 4.1 (Biopac Systems, Inc.). The raw EMG recordings were transformed using the root mean square with a window of 10 ms. The resulting signal was integrated between 90 and 180 ms after the stimulus onset to quantify RIII-reflex amplitude to each shock. These values were normalized (z-score) across all trials within subjects and averaged for each condition (15 stimulations each) to assess the effects of HNCS.

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