



Somatosensory pain is not reliably modulated by weak acoustic stimuli☆☆☆



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ABSTRACT

Background: Pain induced by electrical stimuli has been found in previous research to be reduced by brief, weak electrical pulses, termed prepulses, presented 40 to 60 ms prior to the painful electrical stimulus.

Methods: The present experiment investigated the generality of this effect by presenting weak acoustic stimuli simultaneously with, or 80 or 1000 ms prior to, painful electric shocks. In the second half of the experimental session, each participant (N = 119) was told that the acoustic stimuli would either increase or decrease the pain induced by the electric shock, to investigate automatic and controlled cognitive processes in the modulation of pain.

Results: Acoustic stimuli presented simultaneously with painful stimulation increased pain slightly (4 mm on a 100 mm scale). Acoustic stimuli presented 80 and 1000 ms prior to painful stimuli had no effect on pain. Information that acoustic stimuli would increase pain did so in females, but only when the acoustic stimulus was presented 80 ms prior to the painful stimulus.

Conclusions: The effect of the acoustic stimuli and of information was weak. Failure to replicate previous findings of decreased pain by weak prepulses was most likely due to the sensory modality of the prepulse stimuli. It is recommended that further studies of pain modulation by brief stimulation use electrical and not acoustic prepulse stimuli.

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1. Introduction

The present study investigated whether weak acoustic stimuli presented immediately before or simultaneously with a painful electric shock could reduce that pain. The potential impact of informing subjects that the acoustic stimuli would increase or decrease pain was also investigated.

Prepulse inhibition offers a well-established method with which to study effects of automatic and controlled processes (Elden and Flaten, 2002; Filion et al., 1993). Prepulse inhibition refers to the finding wherein a weak stimulus (prepulse) inhibits (or under some circumstances facilitates) the response to a subsequently presented reflex-eliciting stimulus (Dawson et al., 1999; Flaten and Blumenthal, 1996; Graham, 1975). Inhibition of the reflex is hypothesized to be caused by the automatic processing of the prepulse initiating an inhibitory process that dampens the response to subsequent high-intensity stimuli (Graham, 1975). Automatic processes can be studied by presenting the prepulse at SOAs from 0 to about 100 ms prior to a reflex-eliciting

stimulus, i.e., before voluntary processing of the prepulse can have any effect. In this way, processes like the detection, analysis, and identification of the prepulse can be studied (Elden and Flaten, 2003). Controlled attention can be studied at longer SOAs, by informing the participants that they should attend to or not attend to the prepulse (Elden and Flaten, 2002; Filion et al., 1993).

Pain elicited by electric shock to the arm can be reduced by weak electric shocks just above sensory threshold, presented 40 or 60 ms prior to a painful shock and to the same location on the arm (Blumenthal et al., 2001). Blumenthal et al. (2001) showed that a prepulse could reduce reported pain, not just reflexes. Likewise, the perceived intensity of a reflexogenic stimulus has been shown to be reduced by a weak acoustic prepulse (Swerdlow et al., 2007). If prepulses reduce pain, they can be used to reduce pain induced by medical and dental procedures like injections, blood sample collection, and other procedures where the pain is necessary, acute, and short-lived.

Blumenthal et al. (2001) the prepulse and the painful stimulus were electrical, i.e., in the same sensory modality. Prepulse inhibition has, however, been observed with cross-modal stimuli (Elden and Flaten, 2003; Hill and Blumenthal, 2005). Flaten (2002) and Stitt et al. (1980) observed inhibition of tactile (airpuff and tap to the glabella, respectively) startle by an acoustic prepulse at SOAs of 50 to 400 ms. Likewise, Sanes and Ison (1979) observed inhibition of electrically elicited startle by an acoustic prepulse presented 50 to 200 ms prior to the electrical stimulus. Powers et al. (1997) had similar findings to those of Sanes and Ison

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(1979), and also found that electrical prepulses inhibited electrically elicited startle at SOAs of 50 ms and longer.

In the present study, stimulus onset asynchronies (SOAs) of 0, 80, and 1000 ms were used to observe the temporal development of cross-modal prepulse inhibition of pain. Prepulses were weak acoustic stimuli, and pain was elicited by electric shock to the arm, as in Blumenthal et al. (2001). The effect of automatic processing on pain was investigated at the 0 and 80 ms SOA, and the effect of controlled processing at the 1000 ms SOA. Since the speed of neural transmission of the acoustic signal is faster than that of pain, the effective SOAs in the brain were longer than the programmed SOAs. The acoustic signal reaches the auditory cortex within 20 ms after stimulus onset (Woldorff and Hillyard, 1991). The time from stimulus onset of painful stimuli to the hand until pain-related event-related potentials are recorded in the brain is about 200 ms (Kanda et al., 2000). Thus, the actual SOA in the group receiving a programmed SOA of 0 ms may be closer to 180 ms, and in the 80 ms SOA condition the true SOA in the brain should be about 260 ms. Studies using acoustic prepulses and somatosensory reflex-eliciting stimuli have shown inhibition of reflexes at short SOAs of about 100–400 ms (Flaten, 2002). Thus, inhibition of pain was expected at the 0 and 80 ms SOAs.

The effects of controlled processing on pain were further investigated by informing the participants that the prepulses would either increase or decrease pain. Information that a treatment will reduce pain has often been found to decrease pain, termed placebo analgesia (Aslaksen et al., 2011; Benedetti et al., 2003; Johansen et al., 2003; Wager et al., 2004). Reduced attention to the painful stimulus has been suggested to play a role (Colloca et al., 2013). Therefore, half the subjects were informed that the prepulses would reduce their pain, and the other half were told that the prepulses would increase pain, in a second block of trials presented after a first block where no information about the prepulses was provided.

Fear of pain refers to an increase in anticipatory fear due to threat of being subjected to painful stimulation. Increased fear of pain has been shown to increase reports of pain, and to be negatively correlated with placebo analgesia (Lyby et al., 2010). Fear of pain was thus assessed prior to the experimental procedure.

The following predictions were made: 1) Pain should be reduced by the acoustic prepulses. 2) Pain should be reduced more in the 80 ms SOA group compared to the 0 and 1000 ms SOA groups. 3) In the second half of the session, pain should be reduced in the Decrease group and should be increased in the Increase group; and 4) Subjects high in fear of pain in the Increase group should display more pain to painful stimuli presented after the prepulses.

2. Materials and methods

2.1. Participants

All procedures were approved by the local Institutional Review Board at Wake Forest University. Introductory Psychology students ($N = 136$) were recruited with an online system (Sona), and received course credit for participation. Participants were dismissed if they reported any history of hearing loss, cardiovascular problems, seizure disorder, anxiety disorder, or hyper- or hypo-sensitivity to pain, or if they were currently pregnant or using stimulant medications, or if they had used pain relieving medication in the past day. Data from 13 participants were lost due to equipment malfunction or experimenter error, and 3 participants chose to terminate their session early. One participant reported pain levels below 10 in one of the Blocks, and was removed from the data. The remaining 119 participants ranged in age from 18 to 22 years, and nearly equal numbers of males ($N = 60$) and females ($N = 59$) were included. Two male experimenters collected all data. The experimenter in the room with the participant throughout the session was a nationally certified Emergency Medical Technician.

2.2. Apparatus and stimuli

Electric shocks (150 V, 0.5 ms duration) were produced with a Biopac MP150 Work Station activating an STM100 Stimulator and a STIM ISOB shock amplifier, controlled by AcqKnowledge 4.0 software. Shocks were applied with removable 1 3/8 inch disk electrodes (Biopac EL503) taped to the skin on the lateral surface of the biceps of the non-dominant arm, approximately 3 cm apart. Eyeblink activity was measured from In Vivo Metric E220X electrodes (4 mm diameter recording area) filled with conducting paste and attached to the skin below the eye ipsilateral to the shock electrodes, overlying the orbicularis oculi (Blumenthal et al., 2005). A ground electrode was placed on the ipsilateral temple. The EMG signal was amplified with a Biopac EMG 100 amplifier, and the signal was then sampled (1000 Hz), filtered (28–500 Hz passband), rectified, and smoothed with a 5 sample boxcar filter.

Sounds (80 dB broadband noise, 5 ms rise/fall time, 40 ms duration) were produced with Audacity software, amplified by a Presonus HP4 headphone amplifier, and presented via Sennheiser 220X headphones. Sound intensity was calibrated by measuring the loudness of a 5 s long sound with a Quest 215 sound level meter. A 40 ms duration segment of that sound file was then used to create the 80 dB sound pulses used in this study. The timing of sound and shock presentation was controlled by a SuperLab 4.0 program, with sound onset preceding shock onset by 0, 80, or 1000 ms, with 40 participants each in the groups that received the 0 and 1000 ms SOAs, and 39 participants in the group that received the 80 ms SOA.

Self-report questionnaires were used to collect information about personality parameters that may influence pain sensitivity or reactivity. These included the following:

- 1) the Emotionality–Fearfulness items from the Emotionality, Activity, Sociability Temperament Survey (Buss and Plomin, 1984).
- 2) the Fear of Pain Questionnaire III (McNeil and Rainwater, 1998).
- 3) the Fear Survey Schedule III (Arrindel et al. (1984).
- 4) the Fearlessness, Stress Immunity, and Social Potency subscales of the Psychopathic Personality Inventory (Lilienfeld and Andrews, 1996).
- 5) the Thrill and Adventure Seeking subscale of the Sensation Seeking Scale (Zuckerman, 1979), a questionnaire that was administered to each participant in two alternate forms: Experience, and Intentions for the Future.
- 6) the Harm Avoidance subscales of the Triphasic Personality Questionnaire (Cloninger, 1987).

2.2.1. Procedure

Upon arriving at the lab, participants read and signed an informed consent form, and completed a health history questionnaire. Qualifying participants then filled out the battery of questionnaires described above. The experimenter then attached two shock electrodes to the arm. The skin below the eye on the same side was cleaned with a cotton swab dipped in rubbing alcohol, and two EMG recording electrodes were attached below the eye, with a ground electrode on the ipsilateral temple. The experimenter then explained the procedure, and showed the participant how to use the VAS rating scales. On each trial, the participant rated, on a separate sheet of paper, the pain intensity and unpleasantness of the shock on a 100 mm line. The Pain line had anchors of “No Pain” and “Unbearable Pain”, whereas the Unpleasantness line had anchors of “Not Unpleasant” and “Extremely Unpleasant”. Each rating took approximately 5 s or less.

The experimenter stayed in the room with the participant throughout the session, removing the previous rating form on each trial and giving the participant a new rating form.

Trials were presented in two blocks of 8 trials each, with intertrial intervals randomized to 15, 20, or 25 s, and a break between blocks of approximately 1 min. On 4 of the 8 trials in each block, the shock was presented alone, and on the other 4 trials the shock was preceded by the acoustic prepulse at a lead interval of 0, 80, or 1000 ms. Two different

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