Research suggests pain varies by menstrual cycle phase, with pain generally being enhanced during the luteal phase relative to follicular. While the mechanisms underlying these changes are not fully understood, it is hypothesized that endogenous opioids and cytokines may play a role. The current study aimed to investigate the effects of the menstrual cycle on emotional modulation of pain and physiological pain responses (i.e., NFR, HR, SCR).

**Participants**
- Healthy Female Participants: N = 44
- Participant Characteristics: White, non-Hispanic (72.7%), married (77.3%), employed (78.9%), never regular smoker (64.4%), mean years of education = 15.22 (SD = 1.71); age range = 18-50 years (SD = 8.30)

**Exclusion Criteria:**
- History of chronic or acute pain, injury, or health condition that might affect pain sensitivity
- Participation in any research or medication study within 2 months of study inclusion
- Current use of anxiolytic and/or antihypertensive medication
- Current use of tranquilizers or over-the-counter pain medication
- History of substance abuse or dependence
- Current or past history of psychiatric illness
- History of cardiovascular, neurological, circulatory, and/or hearing problems
- Chronic pain condition (e.g., back pain, chronic headache)
- Current use of opioid and/or antidepressant medication
- Current acne, infections, or dermatological conditions

**Procedure**
- Participants were instructed to attend two laboratory testing sessions during the follicular and luteal phases of their menstrual cycle.
- The main effect of Picture Content was significant for Pain Ratings, NFR, and SCR but not SCR
- The main effect of Picture Content was not significant for Valence Ratings and Arousal Ratings
- The main effect of Phase was significant for Pain Ratings, NFR, and SCR
- The main effect of Phase was not significant for Valence Ratings and Arousal Ratings
- Planned simple effects tests did not indicate differences between phases in Pain Ratings

**Conclusions**
- The menstrual cycle does not appear to influence emotional modulation of pain and spinal nociceptive processes.