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Original Article

Thermoregulation, scratch, itch and sleep deficits in children with eczema

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ABSTRACT

Successful sleep onset and maintenance is associated with a reduction in core temperature, facilitated by heat loss at the distal periphery. Problems with initiating and maintaining sleep in children with eczema may relate to impaired thermoregulatory mechanisms, which also contribute to itching and scratching. Our hypothesis was that nocturnal distal skin temperature in eczematous children would be lower than controls, and would also be related to poor sleep quality.

We compared overnight polysomnography and distal (finger) and proximal (clavicle) skin temperature in 18 children with eczema and 15 controls (6–16 years). Children with eczema had longer periods of nocturnal wakefulness (mean [SD] = 88.8 [25.8] vs. 44.3 [35.6] min) and lower distal temperatures (34.1 [0.6] °C vs. 34.7 [0.4] °C) than controls, whereas proximal temperature and the distal–proximal gradient were not significantly different. In children with eczema, a higher distal temperature was associated with indicators of poor sleep quality, whereas lower distal temperature was related to more scratching events during sleep. In conclusion, our findings indicate complex interrelationships among eczema, thermoregulation and sleep, and further, that deficits in thermoregulatory mechanisms may contribute to sleep disturbances in children with eczema.

1. Introduction

Eczema, also known as atopic dermatitis, is a chronic, pruritic, inflammatory skin disease that has prevalence of up to 20% among children [1]. The majority of children with eczema experience sleep problems, particularly during eczema flare-ups [2]. Sleep problems include difficulty initiating sleep, night-time awakenings, short sleep duration, and reduced sleep quality [2–5]. There is robust evidence that supports the relationship between eczema and sleep problems [5,6], but there is a paucity of research examining the causal mechanisms that underpin this relationship. It is likely that there are a number of interrelated mechanisms that contribute to sleep disruption in eczematous children, such as itch [7–10], scratch [7,10–14] and skin inflammation [15–17]. Current literature suggests that eczema-related skin defects and erythema (reddening of the skin) may also affect nocturnal skin temperature [17]. Nocturnal skin temperature is of interest because it has the potential to modulate itch and scratch severity [18–20], and has an impact upon sleep-dependent thermoregulatory processes [21–23]. In summary, atypical nocturnal skin temperature is likely to contribute to multiple mechanisms that cause disturbed sleep in eczematous children.

Humans have a circadian variation in core body temperature, aligned with periods of activity and rest. This variation is regulated through a combination of heat production and heat loss [24]. However, changes in heat loss are thought to drive the nocturnal circadian variation in core body temperature, rather than changes in heat production [24,25]. Circadian-regulated heat loss is enabled through the transference of heated blood from the body's core to dilated blood vessels throughout the distal skin regions [26]. Skin temperatures of the hands and feet, therefore, exhibit an inverse rhythm in comparison to both core body and proximal skin temperature [27]. Temperature regulation is important for sleep onset and maintenance [21–23]. In healthy individuals, a nocturnal rise in distal temperature by approximately 0.5–1.0 °C is associated with a concomitant fall in core body temperature [21–23] that is conducive to successful sleep onset and maintenance [28]. Children with eczema are subject to inflammation of the skin and atypical vasodilation [29]. Extended periods of eczema-related vasodilation can also contribute to excessive amounts of heat loss [17] which may also have secondary effects upon sleep initiation and maintenance [21,22,30].

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Furthermore, nocturnal itching and subsequent scratching are reported to provoke nocturnal awakenings in children with eczema [7–10]. The itch of eczema is thought to be multi-factorial, with impaired temperature regulation, inflammatory cytokines, epidermal barrier function, sensory sensitivity and skin dryness, all causally implicated [31–33]. In particular, vasodilatation and erythema [34] impact on the itch of eczema and are associated with both increased [30] or reduced [20,35] skin temperature. These findings further suggest why increased itch can also occur at both cooler and warmer skin temperatures [30]. The behavioural response of scratching is believed to reduce the itch impulse via algesic and pruritic-mediated neurological pathways [36,37]. The overall dysfunction in the thermoregulation of eczematous children may also explain why environmental changes in temperature are associated with intense itching [20,35,38], and increases in eczema flares and scratching [18–20]. However, few studies to date have used polysomnography (PSG) to examine nocturnal scratching in children with eczema [7,10–14]. The majority of these studies report that scratch and/or non-scratch-related arousals are more frequent in children with eczema than in non-eczematous controls [7,10,11,13,14]. However, the impact of itch and scratch does not fully account for all sleep disturbances among eczematous children. Reuveni et al. (1999) report that scratching in eczema children preceded arousal in only 15% of nocturnal events, with the remainder having no identifiable cause, and, further, these children did not demonstrate longer periods of nocturnal wakefulness than controls [10]. In contrast, Stores et al. (1998) found that eczematous children spent more nocturnal time awake and that time awake was also strongly associated with the time spent scratching [12]. Nonetheless, these studies did not account for thermoregulation-dependent sleep processes or the impact of skin temperature upon itch severity and the frequency of scratching.

In summary, current literature suggests that children with eczema have thermoregulatory deficits that are likely to affect their sleep quality. Accordingly, we hypothesise that, at night, children with eczema will have a lower distal temperature, but no change in proximal temperature, compared to controls. When considering only children with eczema, we hypothesise that distal temperature would be significantly related to sleep characteristics, nocturnal scratching and erythema, namely, that children with cooler distal temperatures would have significantly less sleep, longer sleep-onset latencies, more arousals, more wake after sleep onset, more scratching during sleep and more erythema.

2. Method

2.1. Participants

Children with eczema (N = 18, 7M and 11F; aged 6–16 years) attending Allergy and Dermatology clinics at the Women's and Children's Hospital, a tertiary referral centre for the state of South Australia, were recruited for the study and compared to healthy controls (N = 15, 7M and 8F; aged 6–16 years) who were recruited from advertisements within the hospital. Participants with eczema attending Allergy and Dermatology clinics at WCH were diagnosed by a medical specialist using standardised criteria [39]. Based on parental report, any child with a history of facial abnormalities that affected breathing, craniofacial abnormalities, neurological disorders, muscular dystrophy, intellectual delay, developmental delay or behaviour disorder was excluded. The study was approved by the Women's and Children's Hospital and the University of Adelaide, Human Research Ethics committees.

Prior to commencing the study, participants and their parents completed an omnibus questionnaire assessing demographic, general health, sleep behaviour and atopic disorders. Children recruited into the study underwent a PSG study on a non-school night at the Adelaide Women's and Children's Sleep Disorder Unit. Room temperature was kept at 22 °C throughout the study, and care was taken in the placement of the electrodes and apparatus so that eczema-affected areas were avoided if possible. Participants were set up for their PSG study prior to their usual bedtimes and temperature recordings were all matched to their sleep-onset time in an attempt to counter individual chronotype differences, including those associated with age and sex factors.

2.2. Measures

2.2.1. Polysomnography

An extended PSG montage was used to collect the following measures; electroencephalography (EEG; electrodes positioned at sites F3, F4, C3, C4. O1, O2, M1 and M2), electrooculography (EOG; positioned 1 cm above and lateral to the right eye, and another electrode placed 1 cm below and lateral to the left eye), electromyography (EMG; positioned under the chin), intercostal EMG (positioned to record diaphragmatic activity), thermistor (air flow), nasal cannula (nasal pressure), leg leads (limb movement), respiratory bands (around the chest to record muscular breathing patterns), electrocardiography (ECG) and oximetry (O_2). Signals were digitised and stored using a Compumedics®, S-Series Sleep System (Melbourne, Australia).

Sleep architecture was scored according to standard [40] and paediatric respiratory criteria [41]. The following variables were reported: sleep-onset latency, total sleep time, sleep efficiency, number of sleep-stage shifts, wake after sleep-onset time and arousals/total sleep time. Studies were scored by an experienced sleep technician blinded to Group status. Spontaneous and respiratory arousals were scored according to the criteria of the American Sleep Disorders Task Force [42]. The staging of arousals in PSG data used the following general criteria; minimum 10 seconds of sleep prior to and post arousal (post if greater than 15 seconds), minimum of 3 seconds to maximum of 30-seconds duration (15 seconds/epoch), when in Rapid Eye Movement (REM) an increase in the chin EMG for a minimum of one second.

2.2.2. Scratch

Participants slept in a temperature-controlled environment and were only covered by a cotton sheet and an additional thin cotton bedspread. The coverings did little to mask participant's body position or their scratch movements whether it was by the participant using their hand, leg, or so on. A nocturnal video of each participant's sleep was recorded in conjunction with overnight PSG and later reviewed by a sleep technician for scratching events. Scratching events were matched with their time-congruent EEG and assessed to determine whether the event originated when awake or asleep. The duration of all scratching events was measured.

2.2.3. Temperature

Temperature data were collected using a Mini Logger Series 200 (Respironics®, Oregon, USA) recording device connected to YSI 400 series thermistor probes. Skin temperature was measured at four sites simultaneously throughout the night. Temperature was recorded at the left and right clavicle and at the left and right index fingers at a sampling rate of one sample every 60 seconds. The Mini Logger was taped to the child's clothing on the abdomen. Left and right clavicle temperatures were averaged to produce proximal temperature reading and the left and right index finger temperatures were averaged to produce a distal temperature reading. The averaging of the two sets of sites was done to reduce the noise inherent in temperature measurement. A distal-to-proximal skin temperature gradient was calculated by subtracting the average distal temperature from the average proximal temperature [43].