Supplemental Information

Correlational Analyses

In terms of cross-sectional correlations, higher levels of baseline pain intensity and interference were associated with higher levels of baseline PTSS (rs = .35 and .36, ps < .001, respectively), more severe insomnia (r = .22, p < .05 and r = .48, p < .001, respectively), and poorer sleep quality (r = -.20, p < .05 and r = -.40, p < .001, respectively), ps < .05. Higher levels of baseline PTSS were associated with poorer sleep quality (r = -.36, p < .001) and more severe insomnia (r = .49, p < .001). Higher pain interference at follow-up was associated with higher levels of follow-up pain intensity (r = .52, p < .001) and 3-month PTSS (r = .25, p < .05). Longer sleep duration was associated with better sleep quality (r = .20, p < .01). Correlations between objectively assessed sleep duration and most of self-report variables were small and non-significant (rs ranged from |.02| to |.16|, ps > .05).

With regards to longitudinal correlations, higher levels of pain intensity, pain interference, and PTSS at baseline correlated with higher pain intensity, pain interference, and PTSS at follow-up (rs ranged from .24 to .72, ps < .05). Similarly, more severe insomnia and poorer sleep quality at baseline were associated with higher pain intensity, pain interference, and PTSS at follow-up (rs ranged from |.23| to |.43|, ps < .05).

Additional Analyses: Child PTSD Symptom scale (CPSS-5) subscales

The total CPSS-5 score was used in the analyses to be in keeping with previous research on PTSS and chronic pain in youth (e.g., Neville et al., 2018; Noel et al., 2018; Noel et al., 2016). The CPSS-5 includes four subscales (i.e., intrusion, avoidance, changes in cognition and mood, arousal and hyperactivity) that map into DSM-5 PTSD diagnostic criteria. Additional

correlational analyses were conducted, and the total PTSS score in the second model was replaced with the CPSS-5 hyperarousal symptoms subscale.

- The Intrusion subscale was correlated with sleep quality (r = -.28, p = .001) and insomnia (r = .38, p < .001), but not sleep time (p > .05).
- The Avoidance subscale was correlated with sleep quality (r = -.29, p = .001) and insomnia (r = .42, p < .001), but not sleep time (p > .05).
- The Changes in Cognition and Mood subscale was correlated with sleep quality (r = -.31, p < .001) and insomnia (r = .48, p < .001), but not sleep time (p > .05).
- The Arousal and Hyperactivity subscale was correlated with sleep quality (r = -.31, p = .002) and insomnia (r = .39, p < .001), but not sleep time (p > .05).

Overall, the strength of associations is similar to the correlations between the CPSS-5 total score and sleep variables, which is consistent with our previous research (Noel et al., 2018). For parsimony and in keeping with previous research we examined the total CPSS-5 score.

The Arousal and Hyperactivity subscale was correlated with sleep quality (r = -.37, p = .002) and insomnia (r = .50, p < .001), but not sleep duration (p > .05). It also correlated strongly with the total CPSS-5 score (r = .90, p < .001). In replacing the CPSS-5 total score in the second model with the Arousal and Hyperactivity subscale scores, we found that the model demonstrated good fit $\chi^2(29) = 33.06$, p = .28, $\chi 2/df$ = 1.14, CFI =.988, RMSEA = .032, pClose = .706, and arousal and hyperactivity symptoms significantly predicted subjective sleep disturbance. However, the relationship between subjective sleep and pain interference at follow up was no longer significant. We hypothesize that while arousal and hyperactivity play an important role in subjective report of sleep disturbances, it is the combined clusters of PTSS that drives worsening of pain interference over time.

Additional Analyses: Insomnia Symptoms and Sleep Quality

Additional analyses were conducted, in which the latent subjective sleep disturbance variable was replaced with, first, sleep quality scores and, then, with insomnia severity scores.

The model with sleep quality (i.e., not containing insomnia severity scores) demonstrated good fit, $\chi^2(21) = 19.36$, $p = .56$, $\chi 2/df = 0.92$, CFI > .999, RMSEA < .001, pClose = .860. PTSS at baseline predicted sleep quality scores, however sleep quality no longer significantly predicted pain interference at 3 months ($p > .05$). Similarly, the model with insomnia severity scores (i.e., excluding sleep quality) demonstrated good fit, $\chi^2(19) = 12.42$, $p = .867$, $\chi 2/df = 0.65$, CFI > .999, RMSEA < .001, pClose = .973. PTSS at baseline predicted insomnia severity, however insomnia no longer significantly predicted pain interference at 3 months ($p > .05$). Therefore, we hypothesize that broadly disturbed sleep, including symptoms of insomnia and a subjective perception that sleep quality has been impaired underlie worsening of pain interference over time.

Additional Analyses: Wake After Sleep Onset and Sleep Efficiency

Additional analyses were conducted, where sleep duration was replaced with, first, wake after sleep onset and then sleep efficiency. The model with wake after sleep onset demonstrated good fit, $\chi^2(25) = 20.43$, $p = .73$, $\chi 2/df = 0.82$, CFI > .999, RMSEA < .001, pClose = .942. However, wake after sleep onset was unrelated to any outcome at follow-up, while the effect of subjective sleep on pain interference was still significant, albeit at a trend level (p=.064). Further, wake after sleep onset was significantly associated only with child sex ($\beta = .20$, $p < .05$). Whereas sleep duration (the objective sleep variable included in the original model) was significantly associated with baseline PTSS and child sex. The model with sleep efficiency demonstrated good fit, $\chi^2(26) = 21.46$, $p = .72$, $\chi 2/df = 0.83$, CFI > .999, RMSEA < .001, pClose

= .944. Subjective sleep continued to mediate the relationship between PTSS at baseline and follow-up levels of pain interference.