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We show that central and peripheral circadian gene expression were exacerbated when birds were housed together, leading to increased desynchronization of correlated brain and liver expression. Advanced activity onset was also strongly correlated to gene expression in the hypothalamus but did not affect downstream melatonin expression. We highlight that social context strongly affects gene expression and circadian misalignment, exacerbating responses to external stressors.

We found ALAN exposure increased nocturnal activity and caused earlier activity onset, which was intensified by social interactions. Furthermore, ALAN exposure decreased *per3* expression in the hypothalamus uniformly, but *bmal1* expression was only disrupted in social birds in central (hypothalamus) and peripheral (liver) clocks. The interaction of ALAN’s effects across social conditions was supported by the relationship of circadian hypothalamic genes on activity onset, which has been debated whether the two are connected (*7, 20, 21*). Hypothalamic expression in the early morning of *bmal1*, *cry1*, and *per2* significantly predicted activity onset. In social groups, these genes' expression was more severely affected by ALAN, correlating with greater alterations in activity patterns. This supports the argument for an association between behavioral and core clock shifts due to ALAN.

ALAN disrupts circadian rhythms in both behavioral patterns and gene expression, but there has been debate in the field as to whether these two are connected (*7, 20, 21*). We found ALAN exposure increased nocturnal activity and caused earlier activity onset, which was intensified by social interactions. Furthermore, ALAN exposure decreased *per3* expression in the hypothalamus uniformly, but *bmal1* expression was only disrupted in social birds in central (hypothalamus) and peripheral (liver) clocks. The interaction of ALAN’s effects across social conditions was supported by the relationship of circadian hypothalamic genes on activity onset. Hypothalamic expression in the early morning of *bmal1*, *cry1*, and *per2* significantly predicted activity onset. In social groups, these genes' expression was more severely affected by ALAN, correlating with greater alterations in activity patterns. This supports the argument for an association between behavioral and core clock shifts due to ALAN.

The central clock in the SCN performs the crucial role of synchronizing peripheral clocks across an organism, with desynchronization leading to health deficits (*22, 23*). ALAN, and other stressors, have the capability of disrupting peripheral rhythms even if behavioral rhythms or the core clock are untouched, desynchronizing the organism’s system (*24, 25*). We found that ALAN reorganized the relationship of circadian genes differently in birds that were isolated compared to grouped. Different patterns emerged between the two treatments and in the early night we saw increased desynchronization of central and peripheral clocks particularly for social birds. Therefore, in social settings, ALAN not only disrupts both core and peripheral clocks but also leads to a greater degree of desynchronization in their relationship compared to isolated birds.

Contrary to our hypothesis, we observed no significant differences in melatonin levels across treatment groups, suggesting that the mechanism by which ALAN and social interactions affect circadian rhythms may not directly involve melatonin suppression. There are mixed findings on ALAN’s ability to suppress melatonin (*6-8, 26*). Some prior studies reported ALAN-induced melatonin disruption in birds, housed individually or socially (*6, 26, 27*). However, we previously found no suppression of melatonin in bird housed individually (*7*) and another study in humans found negative effects from ALAN independent of melatonin excretion (*8*). These discrepancies may be due to species, light intensity, and individual variation, demonstrating the complex interplay of physiological responses to environmental change.

The synchronization of circadian rhythms, facilitated by social cues, is a critical aspect of social coordination. However, within flies, an individual with a disrupted timing can propagate this disruption throughout an entire group, allowing them to adopt the altered rhythm (*17*). This mechanism may explain why in our study social birds under ALAN showed greater disruption in circadian gene expression, desynchronization, and activity patterns compared to their isolated counterparts. The specific mechanism of social information propagation or disturbance, e.g., visual, acoustic, etc., is worth of future investigation (cite SOCIAL CIRC LIT). We speculate that individuals within a social group who are particularly sensitive to ALAN might influence the circadian rhythm of the entire group.

Our study's results are the first of our knowledge to demonstrate a molecular shift in circadian regulation due to social interactions in vertebrates. This study emphasizes the role of social context in understanding the effects of environmental disturbances like ALAN. The exacerbated responses observed in social settings suggest that collective behaviors might amplify responses to light pollution. This has significant implications for understanding the ecological impacts of ALAN, as social dynamics and group living are common across many taxa. Furthermore, these findings highlight the importance of incorporating social contexts into research designs to obtain more relevant insights into the biological impacts of environmental stressors.

Our investigation into the effects of ALAN on zebra finches reveals that social interactions significantly amplify circadian disruptions. These findings contribute to a growing body of evidence indicating the profound impact of light pollution on biological rhythms and highlight the need for further research into mitigating these effects. Future studies should explore the underlying mechanisms of social amplification of ALAN effects and assess the ecological consequences of disrupted circadian rhythms in group-living species. The findings of this study shed light on the complex interplay between social conditions in response to ALAN exposure and the importance of accounting for social context in experimental lab settings as results may otherwise be less applicable to natural life.