# 1 My texts

For example, in flies expression of the core clock genes *tim* and *per* are downregulated by their own protein products [3]. Although it is not yet well understood how cycradian process modulates the transcriptome, it has been demonstrated that neurons exhibit circadian rhythms in ion channel gene expressions [1–3].

---

# 2 From papers

TuBu synchronizes R5 SWA and increases sleep, while not showing SWA itself [4, 6]

TuBu neurons convey sensory and circadian information [4]

Our data suggest that compound delta oscillations specific to the sleep-regulating R5 network are generated by circadian drive transduced via TuBu neurons. [4]

As our molecular clocks are not precisely running on a 24 h cycle, so-called clock neurons need light input to constantly reset the molecular clocks (Dunlap 1999) [6] (Suarez et al 2021)

Optigenetic activation of DN1p clock neurons leads to oscillatory activity in ring neurons, demonstraing the circadian influence of generating sleep need at the level of neural networks (Guo et al 2018) [6] (Suarez et al 2021)

Circadian time influences sensory processing. Secondly, the circadian time could determine when and to what extent sensory processing leads to the accumulation of sleep need or subjective tiredness [6] (Suarez et al 2021)

period (per) and timeless (tim) - expressed at higher levels in the early night compared to the early day; cryptochrome (cry) and Clock (Clk) mRNA - the opposite. Core clock genes are expressed and cycle specifically in Drosophila clock neurons and glia. While Clk expression is restricted to clock neurons and glia, other core clock genes per, tim and Cycle (Cyc) are expressed in more cell types. No cell type expresses Clk without expression of other core circadian genes is consistent with the notion that Clk is a circadian master regulator [2]. (Dopp et al 2024)

Cell types involved in process S: the four annotated clusters with the highest amount of sleep drive correlates were cell populations associated with sleep homeostasis. 121 correlates by dFB

neurons. Similarly, Oct, Tyr and non-PAM DAN neurons each had more than 100 sleep drive corelates. In contrast, the related dopaminergic subtype of PAM neurons only had 14 correlates. (Dopp et al 2024)

Flies with disrupted glial clock showed significantly reduced rebound sleep after SD compared to control flies (Dopp et al 2024)

In Drosophila, the core molecular clock components are coexpressed only in a restricted set of 150 neurons, which serve a function similar to the mammalian superchiasmatic nucleus (SCN) in regulating circadian rhythms in behavioral activity. [3] (Dubowy and Sehgal 2017)

A molecular clock in a subset of DN1 is sufficient to drive morning anticipatory activity in LD cycles, and, in certain temperature conditions, can drive evening anticipation as well (Y. Zhang et al. 2010).

Sleep homeostasis is often conceptualized as a continuous build-up of sleep need over periods of wakefulness and dissipation over periods of sleep, such that the same mechanisms should be invoked both when flies are spontaneously waking and during periods of forced wakefulness (sleep deprivation). However, recent work in Drosophila has called this view into question. [3] (Dubowy and Sehgal 2017)

One likely function of the circadian system is to suppress the onset of sleep during times of the day when sleep pressure is high but when sleep would be dangerous or maladaptive, thereby delaying it until the appropriate time. For nearly four decades, the sleep field has conceptualized sleep through the two-process model, which posits that sleep is governed by interactions between homeostatic and circadian control processes. In this model, sustained wakefulness produces a homeostatic sleep pressure and increased slow-wave sleep, while a circadian system sets the thresholds for sleep pressure that correspond to sleep or wakefulness. [5] (Shafer and Kenee 2021)

R5 neurons stimulate downstream neurons in the dorsal fan-shaped body (dFB), which are sufficient to produce sleep (Donlea et al., 2014; Donlea et al., 2011; Liu et al., 2016). [1] (Andreani et al 2022)

R5 neurons promote sleep in response to deprivation by activating the sleep promoting dFB (Liu et al., 2016). [1] (Andreani et al 2022)