Final
Transcript of Lucy Jobbins
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Introduction and background

I'm a PhD candidate at the University of Oxford on the topic of sleep and circadian rhythms in dementia

This has already been covered a little bit, but the sleep-wake cycle is controlled by two main processes.

Homeostatic v. Circadian rhythm

So this is the homeostatic sleep drive and then circadian rhythms, which we've talked about quite a lot. So the homeostatic sleep drive is essentially an increased pressure to go to sleep the longer that you've been awake.

When you sleep, that decreases. And then, of course, the circadian rhythm that we've looked at, which is essentially your internal alignment to the sleep-wake cycle. And circadian rhythms are part of the non-image forming function of the eye and are primarily driven by the intrinsically photosensitive retinal ganglion cells that express melanopsin. And these are most sensitive to light at 480 nanometers. But that's not to say they're not sensitive to light at other nanometers as well.

And what we know is that light can have a direct effect on numerous different aspects of physiology and behaviour, and so sleep and circadian rhythms are one example of those, along with other responses like hormones, mood, pupil responses, etc.

Historical perspective on dementia

Dementia was first described over 100 years ago now by Alois Alzheimer and one of his first patients, Augusta, had quite significant circadian disruption in terms of being awake the whole night and then sleeping throughout the day. So this misalignment has been known about since we started thinking about dementia.

And dementia is characterised by a relatively long progression. So often, things that happen before people have symptoms occur in the brain, maybe 10, 20 years before the symptoms ever take place.

And alongside that, you have quite significant worsening sleep and circadian disruption across that disease trajectory.

Sleep and circadian rhythm in dementia- progressive disruption

So, what do we know so far about sleep and circadian disruption in dementia? So we know that as people get worse, they're waking up more, they're taking longer to fall asleep.

Sleep duration may be increased or decreased, often depending on how you're defining sleep. And if we think about sleep stages measured from polysomnography, this slide shows a recording from polysomnography, which is sensors, electrodes on the head to measure your brain activity, and you get these sleep stages from it. So people with dementia tend to have a shift towards what we say are lighter stages of sleep, so more of the N1 sleep and a shift away from stages of sleep like N3 or deep sleep and REM sleep as well.

And this all culminates in worse sleep efficiency.

Open questions - reasons for change in circadian rhythm and cognition: biological, social, protein build-up?

But there's a lot that we don't know. So in terms of things like sleep microstructure, so patterns of activity in the brain that are important for memory consolidation, if there are changes in things like people's circadian rhythms, and if that's driven by a change in their light exposure, whether that is an internal biological change that's happening, or driven by a social change in terms of if you're diagnosed with dementia, then you don't really want to go outside as much because there's so much risk in terms of getting lost.

We also don't know too much about how this relates to people's cognitive functioning or their biomarkers for dementia, things like amyloid and tau proteins, and then how this changes within a home-based environment.

So it's not very common for people to measure people's brain activity whilst they're asleep in the home. And that's what I am aiming to do with my PhD study, comparing people with mild cognitive impairments, very early stages of dementia to healthy older adults using polysomnography, actigraphy, looking at light exposure, their cognition and dementia biomarkers in their blood.