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In addition to being the stimulus for vision, there is increasing evidence that light influences biology and behaviour through other mechanisms. Laboratory studies, clinical evidence, and epidemiological studies are elucidating effects on hormone regulation, neurotransmitter function, and revealing behavioural and health effects not previously recognized. For instance, daytime light exposure influences both immediate social behaviours and night-time sleep quality. The evidence suggests that good health requires a minimum daily dose of light, although we do not yet know what the dose ought to be. The presentation will give an overview of recent research, give an indication of research gaps, and summarize possible implications for practical applications through workplace design, architecture, and individual light hygiene habits.

Biography:

Dr. Veitch is a Senior Research Officer in the National Research Council of Canada Institute for Research in Construction, where she leads research into the effects of the built environment on health and behaviour. Among her leadership roles in professional organizations, she chaired the International Commission on Illumination's TC 6-11 'Systemic Effects of Optical Radiation on Humans' to the first consensus report on the effects of ocular light on human physiology and behaviour, published in 2004. She is a Fellow of the Canadian Psychological Association, the American Psychological Association, and the Illuminating Engineering Society of North America.



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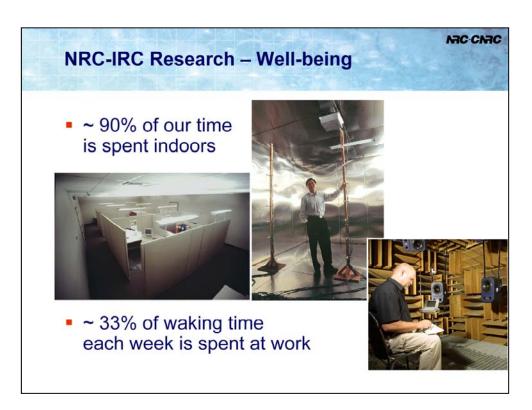


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Producing:

Technical knowledge - journal and conference papers Decision tools - software and design guidance Technology - patents and licenses



Research into health is centered in the Indoor Environment Research Program, which has three sub-programs. The subprogram staff also work together on multidisciplinary consortium projects.

Ventilation and Indoor Air Quality:

e.g., Managing emissions of VOC's - **Material Emissions Chamber** - used in the creation of Material Emissions Database and Software (IAQuest)

- database provides list of materials with their properties

- software developed to model the IAQ in a specific area (using parameters from the database)

Acoustics:

Anechoic Chamber- used in speech intelligibility tests, part of work on speech security and classroom acoustics

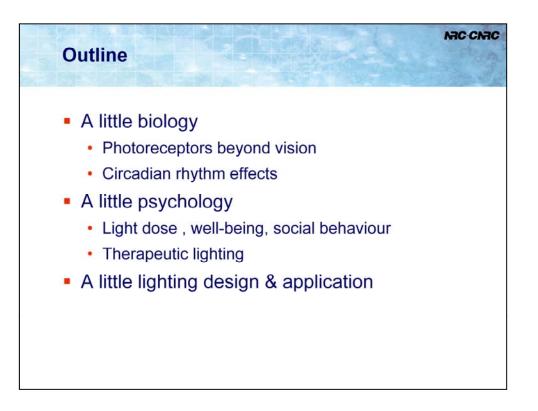
Lighting:

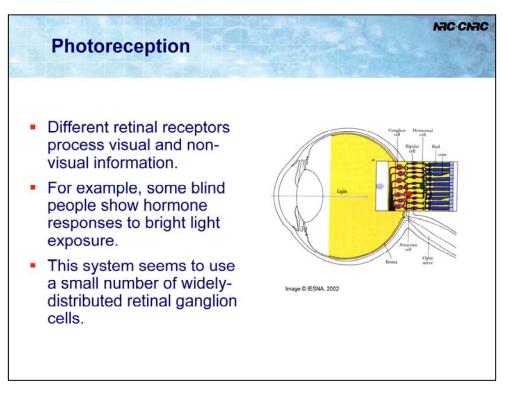
- Lighting quality and individual control over lighting lab and field experiments
- Design tools: Daysim, Daylight 1-2-3, Skyvision

Consortium projects:

e.g., Cost-effective open plan environments (COPE)

IRC studied the impact of various design factors-lighting, acoustics (speech privacy), and ventilation and thermal conditions-on occupant satisfaction in open-plan offices. We used our **Indoor Environment Research Facility** for behavioural and engineering experiments, as well as a large field study of 779 workstations in 9 buildings in 5 cities (and other IRC lab facilities).



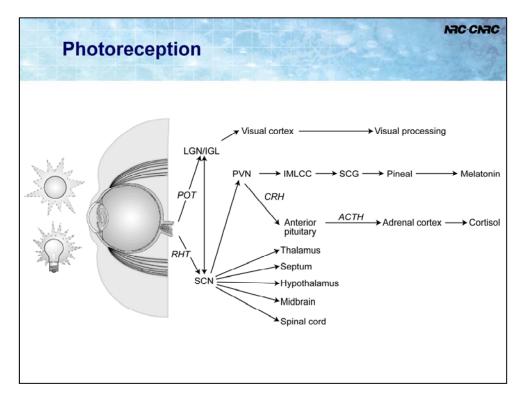


Biologists thought they knew all about light detection when they had identified rods (which operate in black-and-white and at low light levels) and cones (which detect colour and fine details, and operate only at higher light levels). The other cell types shown aggregate and begin to process visual signals before connecting together into the optic nerve.

The discovery of new photoreceptors set the photobiology world on its ear in 2001! We thought we knew everything there is to know about the retina: that rods and cones detect light and send signals to the brain that are decoded to produce visual perception.

However, it was found that some blind people (depending on the type of blindness) showed hormonal responses to bright light exposure. Scientists track the release of the hormone melatonin, which happens at night, in the dark; it can be suppressed by acute light exposure. Certain blind people can't detect the bright light - they can't tell you if they're sitting in light or dark - but yet, their melatonin could reliably be suppressed by light exposure at night, just like in sighted people.

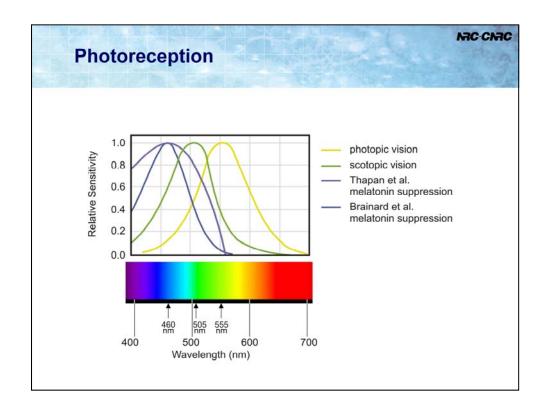
Following anatomical research and other functional studies, we now know that there is a separate set of retinal receptors, a special subset of the ganglion cells (intrinsically photoreceptive retinal ganglion cells, ipRGCs), that detects light and sends signals to the brain. It seems to be a small number of the cells, spread all over the retina.



The signals from these photoreceptors travel a complex route to various brain structures. This figure is from CIE 158:2004, and © CIE, 2009.

Schematic diagram of eye-brain pathways. Light received by the eye is converted to neural signals that pass via the optic nerve to two pathways, one visual and one non-visual. POT = Primary optic tract. RHT = Retino-hypothalamic tract. LGN/IGL = Intergeniculate leaflet of the lateral geniculate nucleus of the hypothalamus. SCN = Suprachiasmatic nucleus of the hypothalamus. PVN = Paraventricular nucleus of the hypothalamus. IMLCC = Intermediolateral cell column. SCG = Superior cervical ganglion. CRH = Corticotropic releasing hormone. ACTH = adrenocorticotropic hormone

Melatonin is the hormone that has received the greatest amount of attention. It's a key molecule in keeping physiological systems on schedule - it seems to start some things up, and slow others down - and it's released in darkness. Because of the dominance of research on this hormone, some people talk about "circadian effects of light", but really there is more to it than this.



What do the new photoreceptors do? They send signals about the presence of light to other brain structures. One of the keys to identifying these cells as being different from rods and cones has been to identify the action spectrum - that is, the response of these cells to different wavelengths of light. The response that has been chosen is the suppression of the hormone melatonin by light exposure at night. Two independent labs, using slightly different techniques, published the first curves at the same time in 2001. It's clear now that the peak response is around 460 nm - in the blue region of the spectrum - and that it's not the same as the response of the visual system (nor of any of its component photoreceptors, not shown). (The range 447 - 477 nm is the key area.)

Two proposed action spectra for melatonin suppression: Results from Brainard et al. (2001) and results from Thapan et al. (2001). Also shown: the scotopic and photopic visual efficiency functions, from Gregory (1977).

These findings are important because:

1. It further demonstrates that there's a separate sensory system for nonvisual effects.

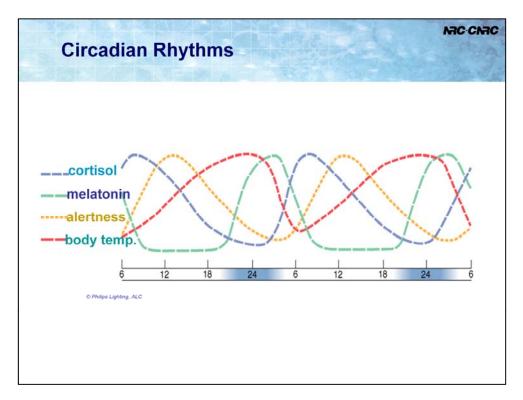
2. It tells us that short-wavelength illumination is most potent for influencing the processes to which this signal extends.

References

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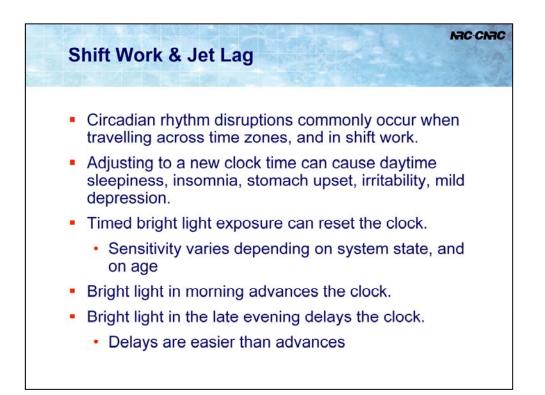
Thapan, K., Arendt, J., & Skene, D. J. (2001). An action spectrum for melatonin suppression Evidence for a novel non-rod, non-cone photoreceptor system in humans. *Journal of Physiology*, 535(Pt 1), 261-267.



This shows in a simplified way how various circadian rhythms vary. Note that the shapes as well as the timing of different events vary. We don't know everything about how these various cycles interact, nor are they they only ones we know of.

One important linkage that is very robust is the pattern of melatonin peak followed shortly by the nadir of core body temperature. This pairing is consistent even when the melatonin peak us shifted (e.g., by bright light exposure).

(This slide was given to me by Gerrit van den Beld of Philips Lighting.)

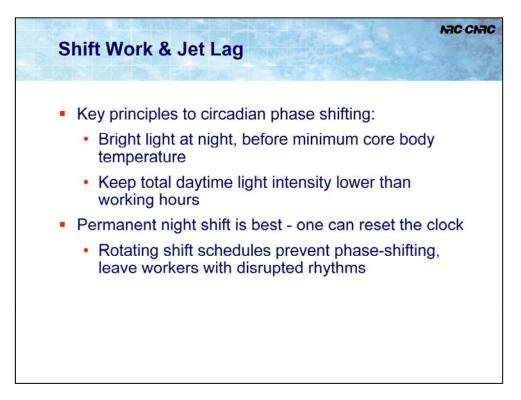


In this next set of slides, I'll talk about how these mechanisms might affect people who are basically healthy.

Many people experience uncomfortable symptoms such as daytime sleepiness, nighttime insomnia, gastro-intestinal distress, irritability, mild depression and confusion, when their circadian rhythms are disrupted by travel, shift work, or sleep disorders.

For people working shifts that rotate rapidly, these can be chronic problems.

Other consequences are an increased error rate, memory disruptions, and cognitive confusion. The accident rate for people driving home from night shifts is higher than for other drivers.

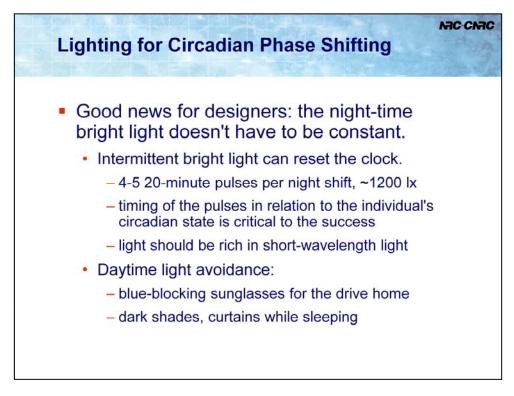


Charmane Eastman and colleagues have presented a compromise schedule for permanent night shift workers. The bright light at work starts relatively early in the shift at the start of the week, then gradually pushes later and later. Behavioural compliance by the worker is part of the scheme; if they don't avoid light when the shift ends, it won't work.

Eastman, C. I., & Martin, S. K. (1999). How to use light and dark to produce circadian adaptation to night shift work. Annals of Medicine, 31, 87-98.

The authors, who are not alone in this recommendation, say that from a phase-shifting perspective, a permanent night shift is best (even more so if you can keep the schedule during days off and vacation, but few do). They are especially critical of rapidly-rotating shifts because the worker is never appropriately phase-shifted. This is especially dangerous for critical positions such as nuclear control rooms, or critical-care nursing, where errors are potentially fatal.

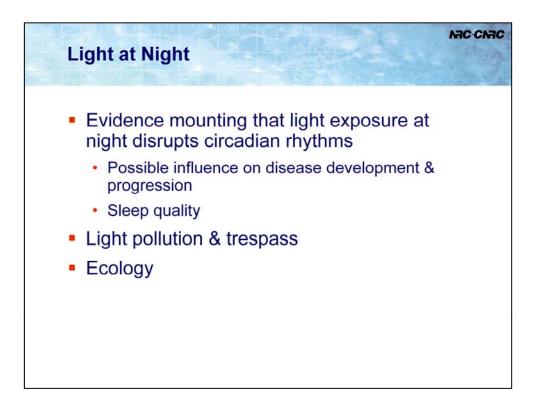
Note: this phenomenon is the same as the experience of jet lag, for which there are also solutions based on timed light exposure and light avoidance.



Thus, success at phase shifting isn't a matter of technology alone, but of the behaviour of the individual, too.

In designing spaces for night-shift work, it would make sense to build in a way to deliver high-intensity light for short periods. Don't rely on the occupants to use a light box on their breaks; they are unlikely to persist in it. How about a brighter area in the lunchroom?

Remember, that does is **light at the eye**, not on the table.



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I'll turn now to an area that is more controversial: the suggestion that we don't generally have sufficient daily light exposure.

In the industrialized world, total daily light exposure (from all sources) is low . A study of106 people in San Diego, ages 40-64, found that the median person spent 4% of each 24 hr in illumination greater than 1000 lx, and more than 50% of the time in illuminance levels from 0.1 to 100 lx (an additional 38.6% of the time was below 0.1 lx, consistent with sleeping, driving at night, etc.). The data were collected in August and September, so this isn't a winter effect. This is remarkable given that of all places in North America you would expect southern California to be a place where people spend lots of time outdoors, and have lots of sunlight to experience! San Diego is the 81st percentile in US hours of sunshine. Other places probably show even lower light exposure.

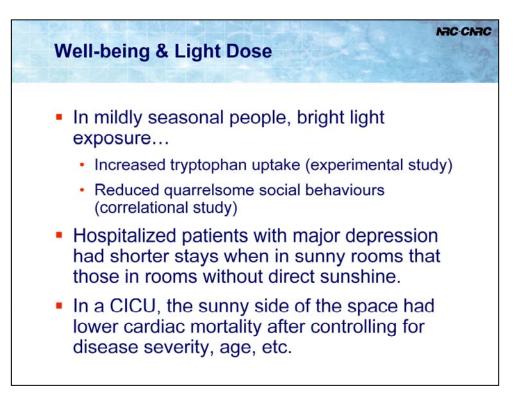
The questionnaire results showed a moderate correlation between atypical SAD mood symptoms and time in bright light (r=-.27). This suggests that inadequate light exposure is associated with depressed mood, but doesn't establish a causal link.

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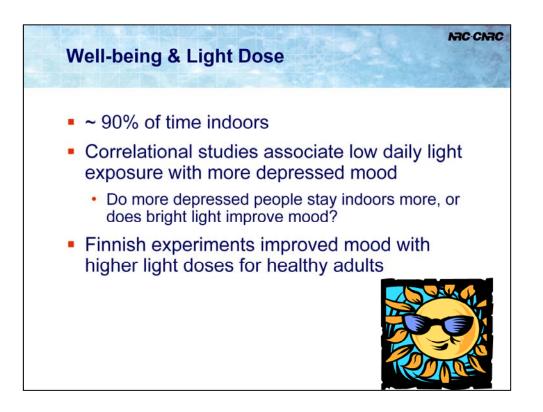


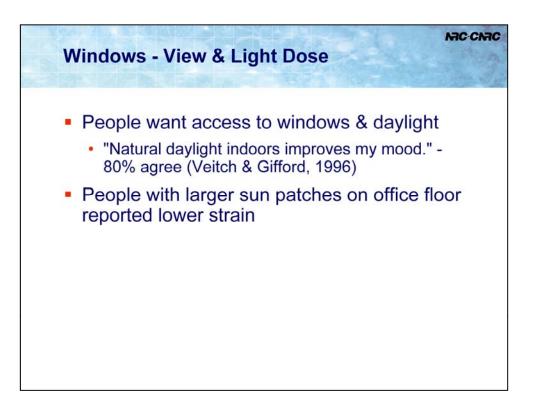
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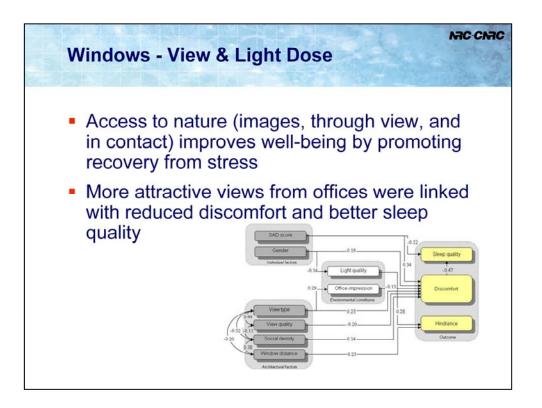
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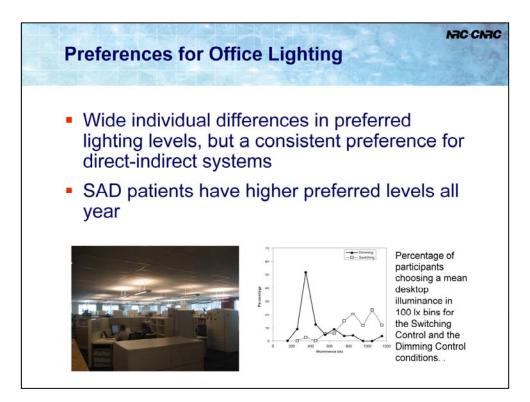
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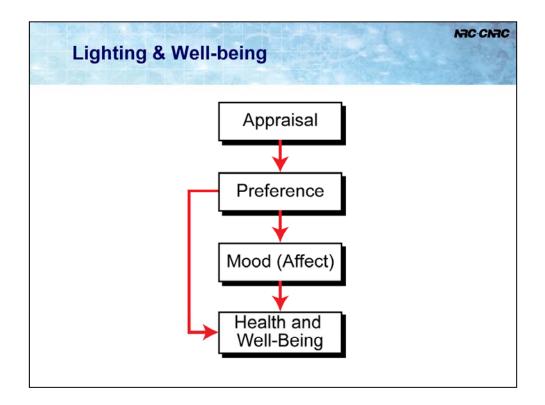




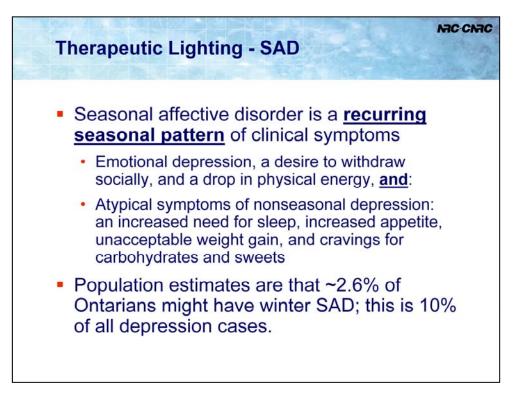




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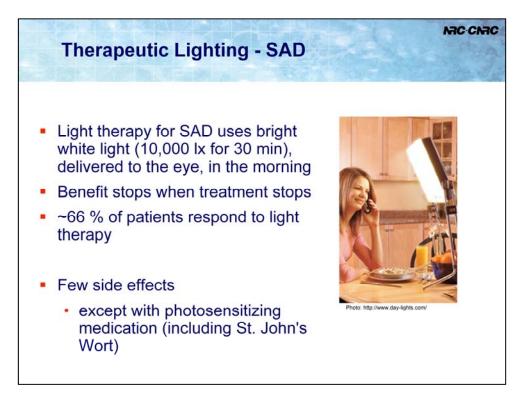
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We hear a lot about SAD and light treatment. I want to emphasize that this is not a very frequently occurring disease.

When I say we might need more light exposure, I don't mean we all should use the light treatment I'm going to discuss here.

N.B. The evidence for increasing incidence with latitude is very weak. Going up north is not a sure-fire way to get this disorder.



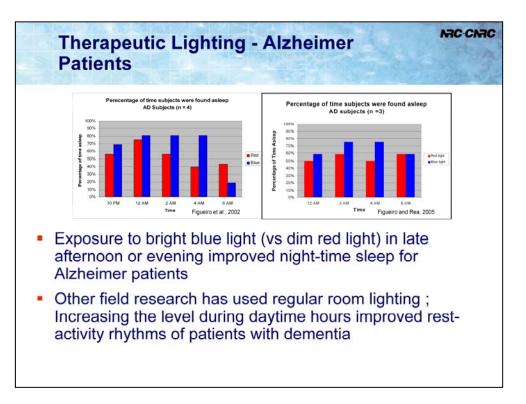
Michalak, Lam, & Levitt, 2002: "Light therapy has been shown to produce relatively limited or mild side effects, with the most common being headache, eye strain, nausea or agitation. Rare reports of hypomania or mania as a result of light therapy have occurred. Consequently, patients with bipolar disorder should be monitored closely during treatment. There are no absolute contraindications to light therapy, and no evidence exists that it associates with ocular or retinal damage. Nevertheless, patients with ocular risk factors (for example, retinal disease, diabetes, macular degeneration, photosensitizing medications, such as lithium, St. John's Wort, and phenothiazine antipsychotics) should have a baseline ophthalmological consultation prior to starting light therapy and should undergo periodic monitoring."

The cause of SAD is **not known.** It's not necessarily a lack of light. After all, pneumonia is cured by penicillin, but the cause of pneumonia is not a lack of penicillin!

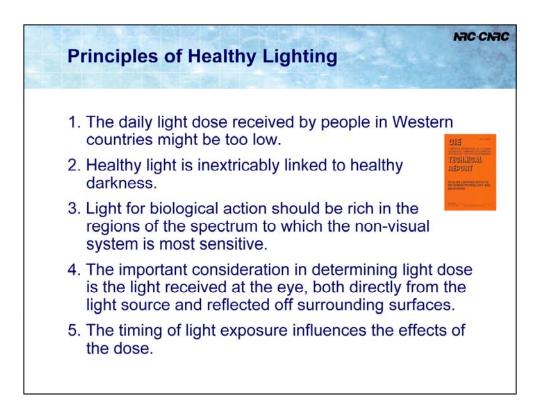


Alzheimer patients often suffer from disrupted sleep and night-time restlessness. This is a big problem for caregivers.

It's likely the case that institutionalized AD patients have very very low light exposure - so the problem may not entirely be the disease.



In both cases, night-time waking was lower for the blue-light condition than the red. The sample size is small, but that makes the demonstration more convincing as it's hard to get statistical significance for a small sample.

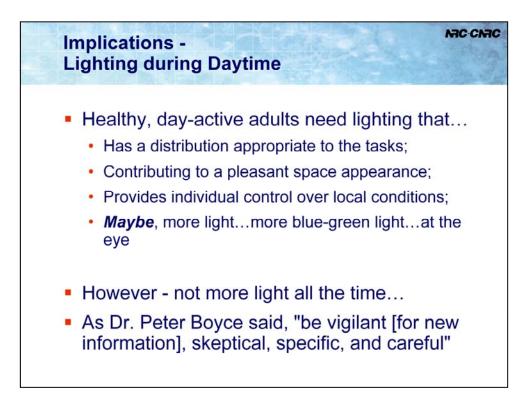


Although we can't say what the necessary daily dose for light might be, there's evidence that

a. Most people get very little light exposure on a daily basis, even if they live in climates where it would be possible.

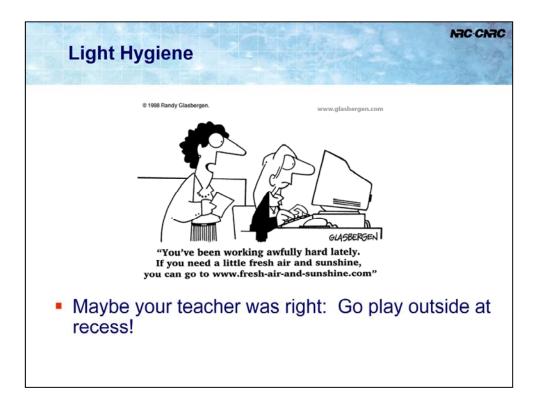
b. Lower light exposure is associated with poorer mood.

These are not causally related in the literature, but there is an evidentiary path that could lead us to recommend that people get more light exposure during waking hours than they currently receive.



What does all this mean for lighting practice?

The implications are not at all clear for daytime lighting, because we don't know the necessary light dose. Remember all those brain structures the light signals go to - we don't know much about most of them.





We do have some clear guidance for night-shift workers. Happily, we know we don't need to increase their light exposure hugely, all the time.

For all of us, at night we should try to avoid light for at least part of the time.

