Light shed on a reason for our discontent

Do some people get depressed just because it's winter, and cheer up in the spring? Dr Simon Wessely reports on the latest medical thinking

Throughout history writers have observed that some people become melancholic at certain times of the year, and not others. In particular there have been frequent suggestions that some people are consistently depressed in winter, only to cheer up again with the coming of spring.

This is a romantic notion that accords with our general view of the contrasts between night, associated with blackness and all that conveys, and day. The word gloomy describes both low mood, and low light.

One of the earliest medical descriptions of this phenomenon was given by the doctor accompanying Robert Peary's Arctic polar explorations at the end of the 19th century. He observed that the lack of sunlight had dramatic effects on the mood, not only of the explorers, but also the local Inuit (Eskimos).

However, it was not until the past decade that the concept of seasonal variations in mood received serious attention. Since then, psychiatrists have described patients whose mood is invariably worse in winter than summer.

These sufferers report that their appetite and sleep are not reduced, as in conventional depression, but increased. They also have profound exhaustion. The name "Seasonal Affective Disorder" (Sad) has been coined to describe them. Perhaps because of its catchy acronym and the universal desire to ascribe psychiatric syndromes to events outside of ourselves, the label has caught on.

That mood might be influenced by changes in seasons is not far-fetched. Rhythm and the blues go together not just in music. Certain parts of the brain involved in the control of mood show endogenous rhythmic variations. These variations are reflected in the characteristic variation that occurs during the 24-hour cycle in the production of certain hormones.

It is thought that the body's internal biological clock, or circadian rhythm, is controlled from these centres. This daily variation is entirely normal, and is reflected in the daily changes in sleep, appetite, mood and energy that we all experience. Changes in mood, sleep, appetite and energy are characteristic of depression. Depressed patients also frequently show changes in the normal daily variation of certain hormones. Hence it is frequently assumed that depression is related to a fault in the body's own biological clock. If daily variations can occur, why not seasonal variations as well?

> The lack of sunlight has dramatic effects on the mood of Inuit

Various hypotheses have been suggested as the cause of Sad. The simplest is that the energy transmitted by light has a direct role in maintaining mood. The brighter the light, the better the mood. However, how this might occur is obscure.

Another idea is the "phase shift" hypothesis. The production of one hormone, melatonin, is particularly sensitive to changes in light. It is secreted by the brain almost entirely at night, and is barely detectable during the day. Does this hormone lower mood? If so, could Sad be associated with a shift in the normal rhythm of melatonin secretion? Alternatively, does the direct suppression of melatonin secretion by light have an anti-depressant effect?

However, papers in the current issue of the *British Journal of Psychiatry* have cast doubt on some of these ideas. Researchers led by Dr Stuart Checkley of the Institute of Psychiatry in London have shown that Sad patients do not differ in their physiological responses to light — light has the same effect on melatonin secretion in them as in normal people. The studies also found that the normal circadian rhythm of melatonin is unaffected in Sad. Hence the two major hypotheses for Sad, even if plausible, may not be accurate.

Some psychiatrists will not be surprised by these findings. Not everyone accepts that Sad is a distinct syndrome, suggesting that the seasonal pattern is just coincidence. Most studies have taken place on selected patients, who may be far from typical of depressed patients. Sad might be just another way of permitting some depressed patients to seek help without the stigma and blame associated with psychiatric disorder No one can be blamed for becoming depressed simply because of lack of sunlight. Some studies showed that only those who had heard of Sad claimed seasonal shifts in their mood.

However, this criticism was not confirmed by another study in the same issue of the *Journal* showing that normal people, and especially women, living in Pennsylvania, and not seeking treatment, also reported seasonal variation in symptoms.

Furthermore, there can be no doubt the biological clock is disturbed in depression, so it is reasonable to presume that, at least in some cases, seasonal changes in the outside environment affect mood.

If Sad is due to a lack of light, then the obvious treatment is light. Many groups, including those at the Institute of Psychiatry, have shown that artificial light does help mood. The necessary equipment is reminiscent of a Heath Robinson cartoon, but it seems to work. The light needs to be bright but does not have to be given for more than two hours a day.

However, three cautions must be given. First, it is almost impossible to design a proper placebo controlled trial of light therapy. To be certain that a treatment is effective, subjects must be "blind" as to which treatment is active, and which the placebo. For obvious reasons, blindness is a contradiction in terms when designing a trial of light therapy.

Secondly, as with any treatment, there are side effects. If light treatment is given to excess it can cause cataracts and can affect the skin. Too bright a light, such as that used to promote tanning, carries a small but definite risk of skin cancer.

Finally, the researchers are at pains to point out that new treatments such as light therapy are designed to supplement existing treatments known to work, such as anti-depressants and psychological therapies, and not to replace them.

● *The author is senior lecturer in Psychological Medicine at the Institute of Psychiatry*