Ultraviolet light and life – by Lindsay Hazley

My research and 25 years' experience of breeding and rearing tuatara in artificial environments have proved the importance of ultraviolet wave lengths of light within the natural photoperiod for the survival of newborn tuatara.

I have followed the direction of research into sudden infant death syndrome (SIDS) with interest. The purpose of this paper is to offer another potential research angle for understanding what causes SIDS.

Several research study papers on the subjects of SIDS, melatonin and ultraviolet light (University of Adelaide, Australia; Massachusetts Institute of Technology, Cambridge, MA, USA; University of Barcelona, Spain; Concordia University, Montreal, Canada) have provided reinforcement to a hypothesis I have had for six years but been unable to qualify with scientific evidence, other than my own discoveries and experiments with tuatara.

Humans evolved outdoors and today we are living in artificial environments where some genetic lines may be struggling.

Technology has provided a new, predominantly indoor, environment behind plastics and glass, with indoor entertainment aids such as television.

Statistics show the incidence of SIDS is high in areas of cold climate and air pollution. Mothers are reluctant to take their babies outdoors, fearing for their wellbeing in such environmental conditions.

The incidence of SIDS is very high among Maori. The European lifestyle of predominantly indoor living may not suit their genetic evolution. The purpose of reproduction is to produce offspring that will better cope with the changing environment conditions of the planet. If these changes are too rapid, genetic adaptability may not be possible, as is evidenced by the numerous animal species now extinct.

In the mid-1960s, newborn babies were religiously taken outdoors for five to 10 minutes each day, rain hail or snow – presumably for thee fresh air – and this practice was not questioned.

SIDS was unheard of then, but it could be said technology did not yet allow the correct identification of such deaths.

During the last 30 years maternity homes have discontinued this practice. It is interesting to note Holland and Sweden have the lowest incidence of SIDS and continue to use the old practice.

SIDS could also have a genetic factor, particularly when we hear of couples losing their first child to SIDS. If it was not for the aid of monitoring equipment their second child could have also died. Certain genetic combinations may produce offspring with a weakness in circadian breathing who need the natural photoperiod to stimulate breathing.

Continuous breathing is a new function for a newborn baby, with the brain requiring strong environmental signals (day and night) confirming that it is outside the womb, independent of its mother.

Premature infants, in particular, have problems with breathing due to poor lung development and often have to be revived in incubators. The practice in the incubator of continual lighting to accelerate growth does not give the infant's brain the environment cue of being born. So when the infant in the incubator becomes tired, why breathe? The baby's brain thinks oxygen will still be provided through thee mother's placenta.

The biological time clock of a newborn has to receive stimulation from the natural photoperiod, unfiltered by glass or plastic. The eyes register the day-night photoperiod that establishes the suppression of melatonin release by day and secretion by night. Overseas research questions the importance of melatonin for the establishment of circadian organisation within the brain.

In the case of SIDS, I suggest that the brain's circadian (automatic) function of breathing is stimulated by the production of melatonin. Sturner and colleagues summarise autopsies done on 32 infants whose deaths were attributed to SIDS and 36 whose cause was non-SIDS. The results showed SIDS infants to have significantly lower melatonin levels, which suggests that diminished melatonin levels, which suggests that diminished melatonin production may possibly be characteristic of SIDS. This could represent an impairment in the physiological circadian organisation. In a study by Sparks and Hunsaker, SIDS infants were reported to have smaller pineal glands than those of children dying of other causes.

In most life forms, the biological time clock is a small organ of 1mm in size called the suprachiasmatic nucleus (SCN). The foetus receives melatonin via the mother's blood and infants are born with a functionally immature SCN-pineal gland axis that does not establish significant hormonal rhythmicity until about 12 weeks of age (49 to 52 weeks post-conception) to establish thee light-dark cycle. Neonatal lighting conditions of continual light to accelerate growth which further delays the appearance of melatonin rhythm in premature infants have also been questioned.

The SCN receives light from the eyes via the retinohypothalamic tract and stimulates the generation and entrainment of many circadian rhythms, including sleep, temperature, and cortisol, and melatonin release. My theory is that it's not simply 'visible' light that traverses this pathway, but only the high frequency wave lengths of the photoperiod, within the UVA band.

Monochromatic UV light (360nm; half bandwidth, 8.8nm) has been shown to suppress pineal melatonin release in rats and to induce phase shift in circadian rhythms. In the rat it induces the hypothalamic SCN expression of the transcription factor Fos, a known cellular correlate of light-induced phase shifts of the hypothalamic SCN pacemaker.

The Tuatara experiment

Tuatara are of extreme scientific interest in that their ancestry dates back 225 million years. Tuatara have a pineal eye, which has been of great scientific interest.

Melatonin has been found in this small eye, indicating that it has a gland function. With tuatara their pineal eye and gland may receive the light rather than the eyes having a neural pathway to the pineal as in most mammals.

The problems I encountered rearing captive tuatara born in an artificial environmental led me to look into the quality of light.

In the first three years, I was experiencing a 50 to 80 percent mortality rate with tuatara reared in captivity. The main cause of death was a chronic calcium deficiency. Dietary supplementation had little to no effect.

Assistance from the Lotteries Grants Board enabled the purchase of equipment to measure the ultraviolet (UVA and UVB) light emission of the enclosure's horticultural quality fibreglass roof. The result was no UV passed through the roof to the animals.

In a newborn tuatara the pineal eye is visible under the skin and becomes covered over with less transparent scales after four to six months.

In 1992 I set up an experiment with 10 newborn tuatara. An ultraviolet bulb was suspended one metre above them (UVA 1.5 watts square metre), time-clocked to be on for six hours each day. The tuatara would bask under the UV for one to two hours each day.

This bulb is the same type used in hospitals to treat jaundice in newborn babies. (Incidentally, a search by Southland Hospital showed no baby treated for jaundice with UV had ever been recorded of dying of SIDS).

After six months, four of the tuatara were removed and placed in the enclosure with no further UV. This was to determine if the reptiles' calcium deficiency was not just simply a vitamin D deficiency, but the possibility the pineal is stimulated by UV to establish correct parathyroid function. (Vitamin D formed in the skin is modified by enzymic hydroxylation by the UV wave lengths 290 to 320 nm).

I postulated that if the four tuatara I had removed from the UV showed calcium deficiencies, it would indicate UV is responsible for vitamin D calcium metabolism.

Now, six years later, these four tuatara, with no further UV, have shown no symptoms of calcium deficiency, indicating something is happening in the pineal that stimulates parathyroid function.

All newborn tuatara now get six months of UV with result of 100 percent rearing success.

Juvenile tuatara aged over three years with chronic calcium deficiencies did not improve when exposed to the UV. This may indicate that a newborn has receptors in the brain that require stimulation by the photoperiod (UV). If these receptors do not receive the environmental cues, they close down without the stimulus from the photoperiod.

Right up to puberty the brain goes through stages of receptivity. It is recognised that schoolchildren at a certain age are receptive to learning spelling instruction. If they do not receive this teaching at this period they will always have difficulty with spelling.

It is interesting to note that the pineal in humans becomes calcified at puberty.

Conclusions

Our planet is continually changing and too rapid an environmental change may not allow enough time for genetic adaptation.

Many medical disorders we face today could well be attributed to the artificial environment we were exposed to at birth.

When a new life form enters the world, not all biological functions are necessarily automatic. Certain functions (circadian rhythms) within the brain requires stimulation from the photoperiod.

Most mammalian life forms have a neural pathway from the visual eye to the pineal gland. The pineal gland is often referred to as our biological time clock and its most recognised function is melatonin secretion by night and suppression by day.

I have found it is the ultraviolet wave lengths (UVA) that traverse this pathway to the pineal.

Artificial environments deprive the pineal, particularly at birth, of access to vital stimulation when the brain's receptors are active and require programming.

Not a lot of study has been done on the pineal. From a non-medical person's view, could the function of the pineal be to programme the brain to a point of sexual maturity? Once a life form is reproductive, the genetic line can continue, so the pineal has served its function and may have a lesser role, if any.

I feel my theory offers no danger to a baby's well-being and it is easy to prove its worth. Cot death researchers could, with couples who have lost their first baby to SIDS and intend to have another child, experiment by going back to the old practice of exposing children outdoors for five to ten minutes each day. Usual sleep monitoring should be continued. If no sleep disorders show, the problem of SIDS may well be solved. Sounds too easy, doesn't it?

The influence of light – and not just the brightness, but the quality of light – may have a major influence on some of the medical disorders we are facing today.

Lindsay Hazley is the curator of the photography/tuatara at Southland Museum.

References

Amir S, Robinson B. Ultraviolet light entrains rodent suprachiasmatic nucleus pacemaker. Neuroscience 1995; 69 (4): 1005-1011

Boyce P, Kennaway DJ. Effects of light on melatonin production. Biol Psychiatry 1987; 22: 473-478.

Firth BT, Cockrem JF, Cree A, et al. Plasma melatonin in the tuatara (Sphenodon punctatus): diurnal rhythm and response to light and heat [abstract]. Proc Aust Endocr Soc 31:S10.

Gsarcia-Patterson A, Puig-Domingo M. Webb SM. Thirty years off human pineal research. J Pineal Research 1996; 10: 1-6 (ISSN 0742-3098)

Kennaway DJ, Globle FC, Stamp GE. J Clin Endocrinology Metabolism; 81 (4)

Sparks LF, Hunsaker JC. The pineal gland in sudden infant death syndrome: preliminary observations. J Pineal Res 1988; 5: 111-118

Sturner WQ, Lynch HJ, Deng MH, et al. Melatonin concentrations in the sudden infant death syndrome. Forensic Science International, 45 (1990) 11171-180

Tosini-G AU, Menaker-M So. TI circadian rhythms in cultured mammalian retina. Science 1996 (Washington, DC); 272 (5260): 419-421