



Photoperiodism and Circadian Rhythms in Relation to the Hazards of Environmental pollutants

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The duration, intensity and wavelengths of light to which vertebrates and invertebrates are exposed vary widely over a 24-hour period as well as throughout the year. Species, by means of their behavioral patterns, differentially control in part the photoperiodic environment to which they are exposed. It is essential that mammals, probably including humans, adjust their physiology with changes in the makeup of the photoperiod. Numerous body functions undergo variations recurring at about 24-hour intervals in the presence or absence of known environmental changes with similar periods. This applies to continuous but rhythmic phenomena, with a peak and trough repeating itself every 24-hours, as well as to discrete events occurring about once a day. The time intervals separating these consecutive periodic events are similar but often not identical. Such periods are called circadian. Rhythms have been reported in cell growth, hormonal levels, and so on. Rhythms are generally resistant to a variety of chemical substances including stimulants and depressants. Photoperiodism and circadian rhythms, in relation to the hazardous effects of environmental pollutants, as pesticides; which may directly or indirectly affect or alter physiological processes in living things, are summarized

1. Introduction

Many species utilize environmental lighting cues to synchronize homeostatic mechanisms which best maximize survival of the individual and the species (Vaughan et al., 1985). Alternation of light and darkness as a result of the earth's rotation has an obvious periodicity. In humans, there is an obvious light-sensitive receptor, the eye. Nervous connections have been demonstrated between the eye and the pineal gland which is a neuroendocrine transducer, transferring information from environmental light to the pineal gland via a neural pathway and this information is converted to an endocrine signal (melatonin) by the gland (Ebadi, 1984). The importance of the pineal gland began with different studies that showed the effects of concentrations of environmental light and some physiological functions and the pineal gland (Quay 1963; Relkin, 1972 and Vriend, 1983).

The pineal cyclically produces several chemical substances including norepinephrine (NE), serotonin (5HT), histamine, and melatonin (McClintic, 1978). One or more of these substances may affect a part of the brain or the pituitary, producing cyclical alteration of body function.

2. Photoperiod and Generation of the Pineal Melatonin Rhythm

Circadian rhythms have been reported to have impacts on cell growth, mitosis, hormonal levels, body temperature, central nervous system functions and other biological activities (Calabrese, 1978). The pineal gland is a haven for chronobiological investigation. Virtually any parameter one measures in the gland over 24-hour period exhibits a rhythm. Whereas many of these rhythms may be mutually dependent, others seem to be unrelated to other 24-hours fluctuations in the pineal (Reiter, 1988). The stimulus, which signals the nighttime rise in melatonin, seems to originate in the SCN of the hypothalamus (Moore & Klin, 1974).

Perhaps the most thoroughly investigated of the pineal rhythms are those relating to the synthesis of melatonin (Arendt, 1985 and Reiter, 1986). The postganglionic sympathetic neurons in the superior cervical ganglia are essential for many of the rhythms that can be measured in the pineal gland. During the day, action potentials in the sympathetic nerve terminals in the pineal gland are minimal; with the onset of darkness this activity increases with a subsequent rise in the synthesis and release of the neurotransmitter, NE (Groos & Meijer, 1985).

3. Modification of Pineal Melatonin Circadian Rhythm by Pesticides

Circadian rhythms in pineal indole metabolism, especially 5-HT, NAS, and melatonin, are regulated primarily by the activity of the enzyme

NAT (Binkely, 1981; Ebadi, 1984 and Quay, 1964). Previously, lindane, parathion and carbaryl were found to stimulate rat pineal NAT activity above those in the control animals, thereby causing enhanced pineal and serum melatonin levels (Attia et al., 1990, 1991a,b). Low daytime levels of pineal NAT activity were unaffected by treatment of rats with a sublethal doses of carbaryl. On the otherhand, the marked rise in the nocturnal activity of the N-acetylating enzyme seen in control rats was significantly augmented in animals given carbaryl (fig. 1)(Attia et al.,1990); thus, at 0100 the carbaryl-treated animals had statistically higher ($p < 0.001$) pineal NAT activity compared to that in pineals of control rats. Pineal HIOMT values in the control rats were similar at both 2000 and 2300 but at 0100 the carbaryl-treated animals had statistically higher ($p < 0.01$) pineal HIOMT activity compared to that in pineals of control rats at the same time (fig. 1)(Attia et al., 1990).

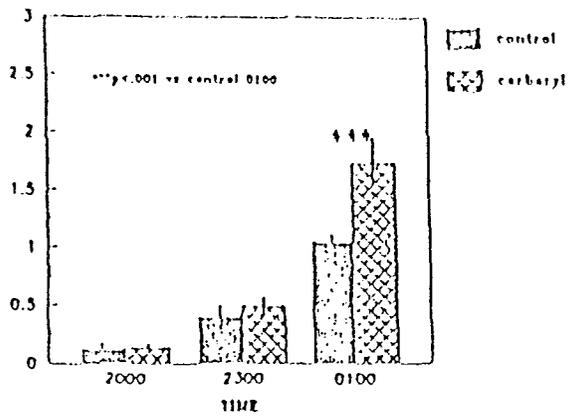
Although pineal levels of melatonin tended to be higher in the carbaryl-treated rats at night, the increases were not statistically significant (fig.2)(Attia et al., 1990). As with NAT activity, carbaryl treatment stimulated nocturnal levels of serum melatonin (fig.2)(Attia et al., 1990); hence, at 2300 the serum melatonin levels were higher ($p < 0.001$) than in the control rats. Serum melatonin levels did not differ at 0100 (Attia et al., 1990).

These pesticides failed to augment the nocturnal rise in NAT activity when tested animals were injected with propranolol, a β -adrenergic receptor antagonist, one hour before lights off (Attia et al., 1991c; 1995a,b). The failure of the pesticide i.e., parathion to enhance pineal NAT activity after propranolol treatment indicates that the parathion-induced increase in pineal NAT activity may involve an action of the pesticide on β -adrenergic receptors since their blockade obviously prevented the action of parathion, (fig.1) (Attia et al., 1995a). Besides the inability of parathion to modify NAT activity in propranolol-treated rats, the pesticide also had no statistically significant action on either pineal or serum melatonin when it was administered to propranolol-injected animals, (fig.2) (Attia et al., 1995a). The significant observation herein is that propranolol treatment prevented parathion from inducing a reduction in 5-HT; this is consistent with the observation that NAT activity was not stimulated by parathion when the β -adrenergic receptors were blocked by propranolol. Thus, since NAT activity was not augmented and its conversion to 5-hydroxyindole acetic acid (5-HIAA) was restored (Attia et al., 1995a).

Similar findings were obtained in earlier studies. Carbaryl, when administered alone, augmented the nocturnal rise in pineal NAT activity above levels in control animals (fig.1) (Attia et. al, 1995b). When rats were treated with the beta-adrenergic receptor agonist propranolol, the nighttime increase in pineal NAT induced by carbaryl was prevented. Carbaryl

treatment by itself, and the combined treatment of propranolol and carbaryl led to significant increase in pineal HIOMT, the melatonin forming enzyme (fig.1)(Attia et. al, 1995b). Pineal and serum melatonin levels were suppressed by propranolol administration and carbaryl was without effect on either parameter when administered to propranolol-injected rats. (fig.2) (Attia et. al, 1995b). The pineal response to these toxins appears to be one of a generalized stimulation in pineal metabolic activity, however, mediated by the sympathetic input to the organ.

PINEAL NAT ACTIVITY



PINEAL HIOMT ACTIVITY

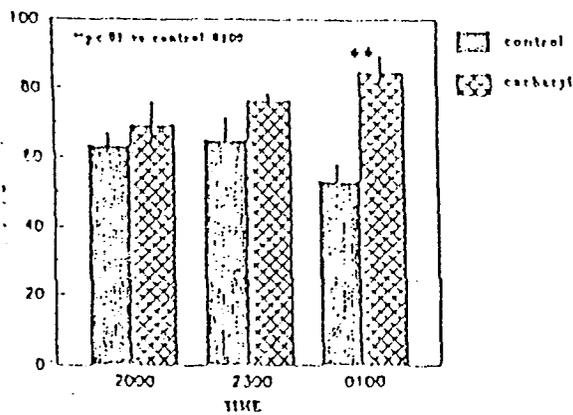
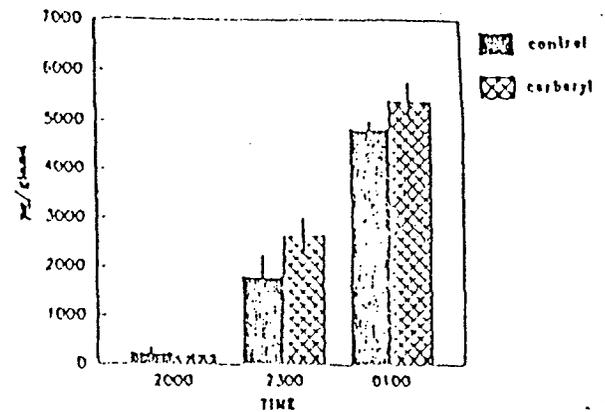


Fig. 1. Effect of carbaryl on pineal NAT and HIOMT activity. Animals were killed at the end of light phase (2000) or at either 2 (2300) or 4 (0100) hr after light off. Data are means \pm SEM.

Carbaryl and pineal melatonin biosynthesis

PINEAL MELATONIN



SERUM MELATONIN

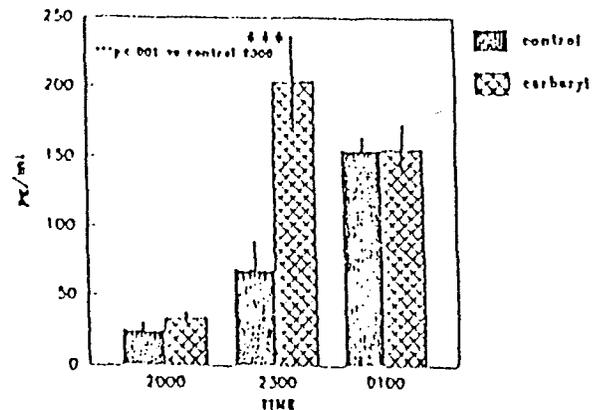
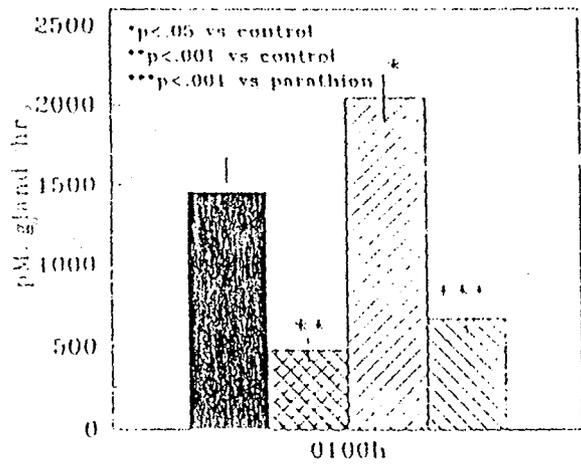
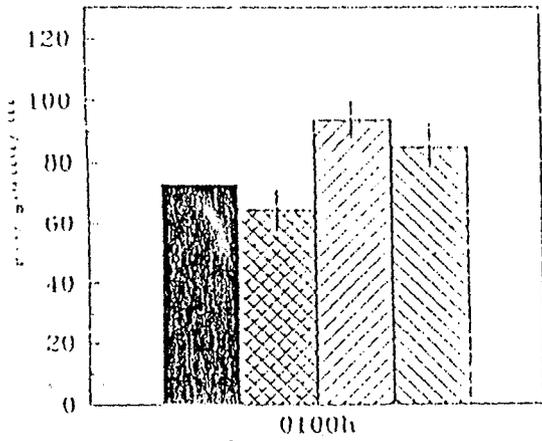


Fig. 2. Effect of carbaryl on pineal and serum melatonin levels. Animals were killed at the end of light phase (2000) or at either 2 (2300) or 4 (0100) hr after light off. Data are means \pm SEM.

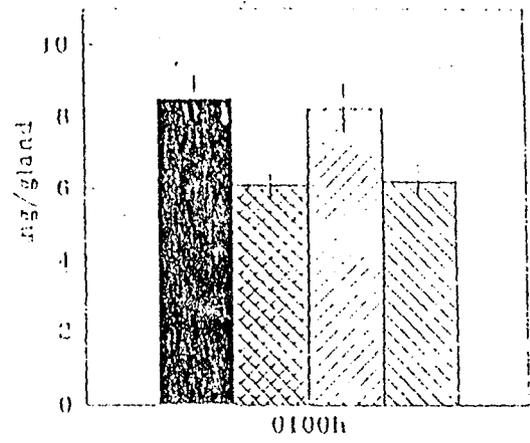


■ control
 ▨ propranolol
 ▩ parathion
 ▧ parathion+propranolol

PINEAL HOMET



Effect of parathion and propranolol on nocturnal pineal and HOMET activities. Animals were killed 4 h (01:00 h) after lights off. Data are means \pm S.E.M.



■ control
 ▨ propranolol
 ▩ parathion
 ▧ parathion+propranolol

SERUM MELATONIN

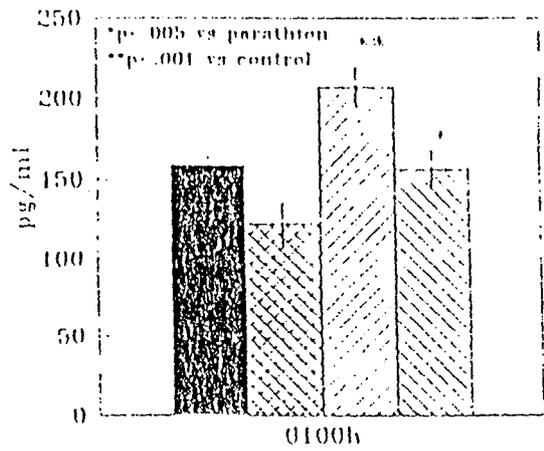
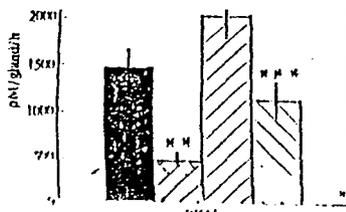
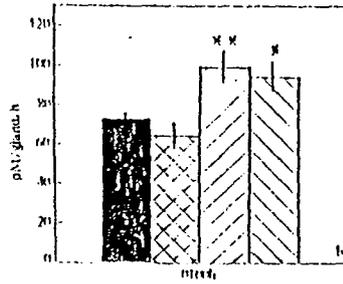


Fig. 2. Effect of parathion and propranolol on nocturnal pineal and serum melatonin levels. Animals were killed 4 h (01:00 h) after lights off. Data are means \pm S.E.M.

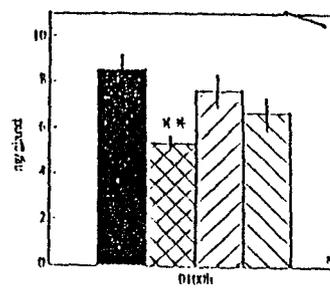


Pineal NAT

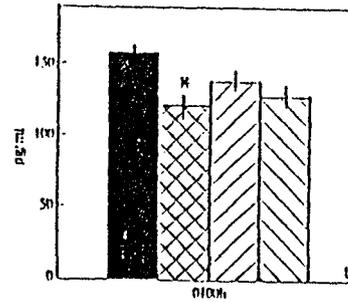


Control
 Propamadol
 Carbaryl
 Carbaryl + propamadol

Fig. 1. Effect of carbaryl and propamadol on nocturnal pineal NAT and HIOMT activities. Animals were killed 4 hours after lights off (0100h). Data are $\bar{x} \pm s. n.$ a. * $P < 0.05$ vs. control; ** $P < 0.01$ vs. control; *** $P < 0.005$ vs. carbaryl; b. * $P < 0.01$ vs. control; ** $P < 0.001$ vs. control.



Serum melatonin



Control
 Propamadol
 Carbaryl
 Carbaryl + propamadol

Fig. 2. Effect of carbaryl and propamadol on nocturnal pineal and serum melatonin levels. Animals were killed 4 hours after lights off (0100h). Data are $\bar{x} \pm s. n.$ a. ** $P < 0.001$ vs. control; b. * $P < 0.05$ vs. control.

3. Concluding Remarks

Carbaryl-stimulated NAT and HIOMT activities above those in the control animals. These changes were apparent only at 5 hr after darkness suggesting that carbaryl stimulates the process required for increased melatonin production. At 0100, pineal NAT and HIOMT activities of carbaryl-treated rats were stimulated more than 60% over control levels; a similar augmentation of NAT has been reported for rats exposed to a sublethal dose of lindane, another common pesticide (Attia et al., 1990). Parathion was found to previously stimulate rat pineal NAT activity and pineal and serum melatonin levels (Attia et al., 1991a).

Significant findings in this study are that parathion enhances both pineal NAT activity and serum melatonin levels above that in the control animals but failed to enhance them when the rats were treated with the beta-

adrenergic receptor blocker propranolol. Whereas neither propranolol nor parathion altered pineal 5-HTP levels, parathion by itself significantly reduced pineal 5-HT values. Similar findings were obtained in earlier studies (Attia et al., 1991). Results suggest that parathion modifies pineal 5-HT metabolism via an effect on either the sympathetic innervation to the pineal gland or on the beta-adrenergic receptors in the pinealocyte membrane. Further investigation of implications of these pollutants, is to be considered.

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A Reliability-Based Determination of Economic Life of Marine Power Plants

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The reliability-based life approach is utilized. Selective failure modes of marine power plants are used for illustration. A case study of the Egyptian Commercial Fleet owned by the Public Sector Company was analyzed and used to establish a demonstration of the expected economic life based on local operating and maintenance conditions. The data acquired is analyzed and failure trend is derived for each failure mode. Probabilistic techniques are used to randomly generate numbers and times of occurrence of different failure modes. The reliability analysis is performed on the life span expected by the manufacture to predict the total number of failures, dependent failures, and cost of failures. Total expenditure due to random failure and cost of scheduled maintenance together with the annual income are utilized (using the time value of money) to determine the economic life of the plant. Conclusions are derived and recommendations for the enhancement of this work in the future are made.