Involvement of tissue monoamine and plasma FFA concentrations in the responses of the pigeon to changes of photoperiod[1]

S. Saarela, E. Hohtola, A. Haim[2] and O. Vakkuri

Department of Zoology, Zoophysiological Laboratory, University of Oulu, SF-90100 Oulu 10 (Finland), and University of Haifa, Oranim, P.O. Kiryat Tivon (Israel), 29 December 1980

Summary. A deviation of the length of photophase from the rhythm of 12L:12D induced monoamine accumulation in diencephalon and pancreas. A short photophase increased plasma FFA. The results suggest that photoperiodism is a stronger modulator of catecholamines and lipid metabolism than changes in ambient temperature (Ta).

The role of catecholamines (CA) in the temperature regulation of birds has been studied intensively in recent years. It has been shown that, unlike the situation in mammals, CA's do not stimulate regulatory heat production. Rather, both exogenous CA's and even endogenously released noradrenaline (NA) induce hypothermia in the cold[3,4]. This response appears to be modulated by seasonal acclimatization, thyroxine treatment and photoperiodicity[4-6]. In fact the short photoperiods may be a stronger acclimatizing factor in arctic animals than low ambient temperatures (Ta)[7,8]. Since CA concentrations and their turnover rate seem to change during temperature stress[9], and after thyroxine treatment[10], it was decided to study whether tissue CA and plasma FFA concentrations are affected by changes in the length of photophase. Such influences might elucidate the obtained thermoregulatory action of photoperiodicity[6,11].

Materials and methods. Adult pigeons *(Columba livia)*, adapted to laboratory conditions (12L:12D photoperiodicity, 22±1 °C Ta), were acclimated to the following lengths of circadian photophase during 21 days: 0, 4, 20 and 24 h. In addition, natural summer and winter conditions were simulated by acclimation of pigeons to long photophase (20 h) and warm ambient temperature (Ta = 32 °C), and to a short photophase (4 h) and cold ambient temperature (Ta = 2 °C). In order to find out which one, photoperiodism or Ta, is more important as a stimulus for tissue CA metabolism, pigeons were acclimated also to warm Ta and short photophase, and to cold Ta and long photophase.

The decapitation of pigeons was performed in the middle (±1 h) of photophase. Pigeons maintained in continuous light or darkness were decapitated at the same time. A previous study[12] showed that mere handling stress caused no significant changes in plasma CA within 5 min.

Tissue CA concentrations were assayed fluorometrically[13,14]. The procedure and modifications have been described earlier[9]. This method gives a signal to blank ratio of 2 when about 1 ng of A or NA is recovered for the fluorescence development. The measured signal to blank values were always 2 or better. Furthermore, subsequent radioenzymatic assays of pigeon plasma CA gave values similar to the present method. For maximum specificity, the values were always corrected for cross-fluorescence, which was practically zero for NA/A and less than 13% for A/NA. Plasma FFA was determined colorimetrically[15].

Results and discussion. A deviation of the length of photophase from the 12L:12D-rhythm (control) induced serotonin (5-HT) and NA accumulation in diencephalon and pancreas (fig. 1). Simultaneous temperature acclimation did not seem to be an additive factor in these tissues. However, combination of cold-acclimation with short photophase induced an increase in adrenal NA concentration, and warm-acclimation suppressed the influence of short or long photophase. Reduced plasma NA and increased adrenal NA concentrations may reflect an increased NA turnover in the low Ta/short photophase conditions, a result consistent with our previous observations[9,10]. A short photophase induced also a reduction of plasma A concentration. This

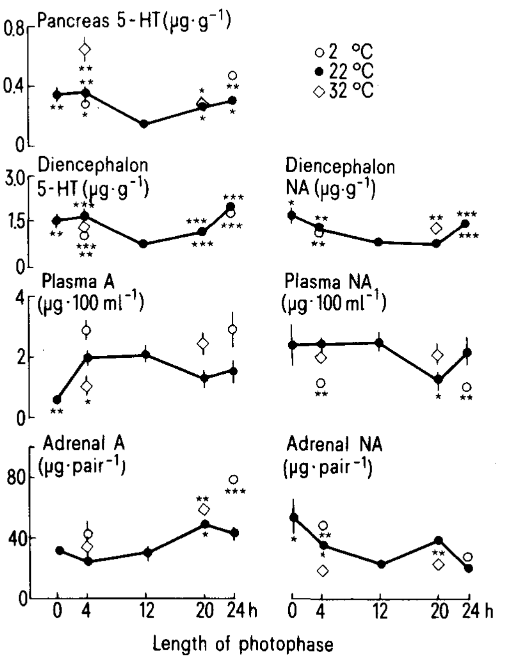

Figure 1. Monoamine concentrations of pigeon tissues after acclimation periods to different lengths of photophase (as an abscissa) and the ambient temperatures 2 °C ○, 22 °C ●, 32 °C ◇. Each symbol refers to the mean for 4-6 pigeons except in the group 24L:0D/22 °C where N = 3. Standard errors of the mean larger than symbol diameter are indicated by vertical bars, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ compared with the control group 12L:12D/22 °C.

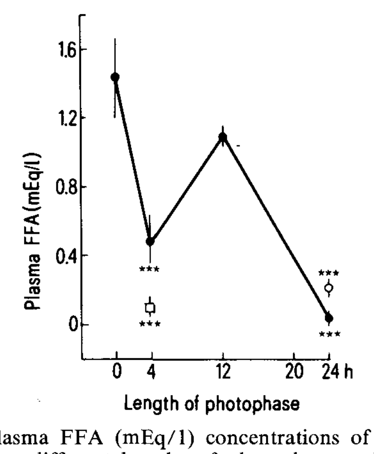

Figure 2. Plasma FFA (mEq/l) concentrations of pigeons after acclimation to different lengths of photophase and the ambient temperatures 2 °C ○, 22 °C ●, 32 °C □. Vertical bars refer to standard errprs. *** $p < 0.001$ compared to control (12L:12D/22 °C).

may be explained by the inhibitory effect of melatonin on adrenocortical glucocorticoids[16] known to stimulate adrenal PNMT.

Acclimation of pigeons to continuous darkness increased plasma FFA concentration by 30% compared to control pigeons (12L:12D/22 °C), but long photophase diminished it to 5% of controls (fig. 2). An increased plasma FFA concentration has been found also after cold exposure of chicks[17] and pigeons[12]. However, cold-acclimation could not reverse the reducing effect of long photophase on plasma FFA concentration in the pigeon in this work. This may mean that photoperiodism has a stronger influence than Ta on lipid metabolism in birds.

Our results support the suggestion that photoperiodism can modulate CA and lipid metabolism, perhaps even more than changes in Ta. These changes may be involved in the adjustment of tissue metabolism leading to thermoregulatory adaptation of birds in nature. Whether this cascade is triggered by the pineal gland and melatonin release, has yet to be discovered.

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2 A.H., University of Haifa, Oranim, P.O. Kiryat Tivon, Israel.
3 R. Hissa, J.C. George, D.E. Stevens and S. Saarela, Acta physiol. scand. *110,* 175 (1980).
4 S. Saarela, Acta Univ. Oul. A 100 (1980).
5 S. Saarela and R. Hissa, Comp. Biochem. Physiol. *56C,* 25 (1977).
6 A. Haim, S. Saarela and R. Hissa, J. therm. Biol. *4,* 167 (1979).
7 J.C. Hart, Rev. Can. Biol. *16,* 133 (1957).
8 G.C. West, Comp. Biochem. Physiol. *42A,* 867 (1972).
9 S. Saarela, R. Hissa, E. Hohtola and E. Jeronen, J. therm. Biol. *2,* 121 (1977).
10 S. Saarela, Gen. Pharmac. *11,* 521 (1980).
11 A. Haim, S. Saarela and R. Hissa, Comp. Biochem. Physiol. *63A,* 547 (1979).
12 E. Jeronen, P. Isometsä, R. Hissa and A. Pyörnilä, Comp. Biochem. Physiol. *55C,* 17 (1976).
13 J. Diamant and S.O. Byers, J. Lab. clin. Med. *85,* 678 (1975).
14 M. Schlumpf, W. Lichtensteiger, H. Langemann, P.G. Waser and F. Hefti, Biochem. Pharmac. *23,* 2437 (1974).
15 K. Itaya, J. Lipid Res. *18,* 663 (1977).
16 M. Motta, O. Schiaffini, F. Piva and L. Martini, in: The Pineal Gland, p.279. Ed. G.E.W. Wolstenholme and J. Knight. Churchill Livingstone, Edinburgh 1971.
17 B.M. Freeman, Comp. Biochem. Physiol. *54A,* 141 (1976).

The relationship between high and low trait psychological stress and serum indicators of stress

K.T. Francis

Division of Physical Therapy, Room 106, Tidwell Hall, University of Alabama in Birmingham, Birmingham (Alabama 35294, USA), 13 October 1980

Summary. Psychological stress as measured by the parameters of trait anxiety, hostility, and depression was compared in 2 different age groups (age 18-30 and 30-55) with serum indicators of stress. There was no significant difference between high and low psychological stress subjects in either age group with any of the serum indicators.

Recently research attention has been directed at the role of 'psychological stress' in the development of coronary heart disease (CHD). The term psychological stress includes the behavioral parameters of anxiety, hostility, and depression. The positive relationship between these behavioral variables and CHD have been well documented[1,2]. However, the exact relationship between these behavioral variables and biochemical correlates of stress and the subsequent development of CHD still remains vague and elusive.

Considerable evidence has now accumulated indicating that 3 biochemical parameters are closely linked with stress. These parameters are serum cholesterol[3,4], serum uric acid[4,5], and serum cortisol[5]. Elevations of 2 of these parameters (uric acid and cholesterol) have been implicated as risk factors in the development of atherosclerosis[6,7]. Even though these 2 different categories - psychological stress and serum parameters - have individually been shown to be related to the pathological state of CHD, there is exiguity of information pertaining to the relationship between these 2 variables. In view of the lack of this information the following study was undertaken.

Method. 18 normal healthy male and female subjects age 18-30 (age group I) and 39 normal male subjects age 30-55 (age group II) with no previous history of CHD were selected from a population of faculty and students from the University of Alabama in Birmingham and from businessmen belonging to local organizations in the Birmingham area. The subjects were selected from a group of 93 volunteers on the basis of their scores on 2 psychological stress examinations. The 2 written exams used as a screening device were the state-trait anxiety inventory (STAI) developed by Speilberger et al.[8] and the multiple affect adjective check list (MAACL) developed by Zuckerman et al.[9]. The STAI is a brief self-report that measures both state and trait anxiety. The MAACL, like the STAI, provides a measure of both state and trait levels of anxiety. In addition the MAACL measures levels of depression and hostility. Both the STAI and the MAACL have an extensive bibliography of research in which evidence of validity has been presented[8,9]. Based on the scores obtained on the STAI and MAACL, subjects were categorized into a high psychological stress group and low psychological stress group. Raw scores of 38 or above on the STAI anxiety index was used for assignment to the high stress group[10]. The STAI score also had to be confirmed by MAACL trait anxiety, hostili-

Table 1. Correlational analysis (R scores) and significance levels between psychometric variables of anxiety, hostility and depression

	MAACL		Speilburger STAI anxiety
			0.795
		Anxiety	$p \leq 0.01$
		0.702	0.660
	Hostility	$p < 0.01$	$p < 0.01$
	0.719	0.713	0.716
Depression	$p \leq 0.01$	$p \leq 0.01$	$p \leq 0.01$