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.

1 Acknowledgment. The support of Emil Aaltonen foundation (to E.H.) and the Finnish Ministry of Education (to A.H.) is gratefully acknowledged.
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	$p \leq 0.01$	$p \leq 0.01$	$p \leq 0.01$

Table 2.

		Uric acid (mg%)	Cortisol (mg%)	Cholesterol (mg%)	STAI Anxiety	MAACL Anxiety	Depression	Hostility
Age group I (age 18-30)	High Stress	4.86±0.3	9.74±1.9	149.44± 9.9	29.6±1.5	7.0±0.6	14.3±0.9	9.1±0.9
	Low Stress	5.75±0.3	7.73±0.6	131.89±10.1	38.2±1.0	3.2±0.9	6.3±0.5	5.3±0.5
Age group II (Age 30-55)	High Stress	6.1 ±0.2	12.1 ±1.2	194.7 ± 9.1	46.8±2.0	10.3±0.6	17.3±1.2	9.6±0.7
	Low Stress	6.2 ±0.2	12.0 ±0.9	182.6 ± 7.2	27.3±0.7	2.2±0.6	6.2±0.7	3.9±0.5

Mean values (± SE) of psychological parameters and serum uric acid, cortisol and cholesterol parameters in high and low psychological stress groups.

ty, and depression raw scores at or above 7, 7, and 11 respectively.

Blood was obtained from the anticubital vein of subjects who had fasted 12-14 h at the time of the psychological testing. Total serum cholesterol was measured by continuous-flow analysis according to the lipid research protocol[11] with the use of the Libermann-Burchard Reagent. Cortisol was assayed by solid-phase radioimmunoassay according to the procedure of Rolleri et al.[12]. Serum uric acid was assayed according to the method of Henry et al.[13]. All changes were assayed for statistical significance by analysis of variance.

Results. The results recorded in table 1 indicate that the parameters of trait anxiety, depression, and hostility as measured by the MAACL were significantly ($p \leq 0.01$) correlated with the trait anxiety parameter as measured by the STAI.

Table 2 presents the mean STAI anxiety and MAACL scores of the total high and low psychological stress groups for the three highly interrelated mood and feeling parameters of trait anxiety, hostility, and depression. The data reveal that the goal of attaining 2 distinctly different psychological stress groups in each age group was attained. However, the serum indicators of stress among high and low psychological stress groups in both age groups were not significantly different.

Discussion. During the past 2 decades considerable progress has been made in the identification of the factors which predispose an individual to CHD. A list of generally accepted standard risk factors include psychological stress. Research attention has recently focused on the significance of psychological stress factors in the etiology of heart disease. The literature implies that psychological stress probably influences the level of certain serum components such as cholesterol, uric acid, and cortisol[4,5,14]. However, the data in table 2 indicate that trait characteristics of anxiety hostility and depression does not apparently chronicly elevate these serum parameters. It may not be the chronic trait of high psychological stress personality that influences the serum parameters but how these 2 personality types of individuals perceive or react to 'acute' stress[15]. The high stress individual may react differently to periods of acute stress that occur throughout a given work day; hence, the acquisition of a unitary data set may not detect such differences. This laboratory is in the process of attaining these data in a high and low stress group of subjects.

1 C.D. Jenkins, N. Engl. J. Med. *294,* 987 (1976).
2 R.H. Rosenman and M. Friedman, J. Am. med. Ass. *184,* 112 (1963).
3 J.E. Peterson, R.A. Keith and A.A. Wilcox, Circulation *25,* 798 (1962).
4 R.H. Rahe, R.T. Rubin and B.R. Clark, J. Am. med. Ass. *206,* 2875 (1968).
5 K.T. Francis, Psychosom. Med. *41,* 619 (1979).
6 M.M. Gertler, S.M. Garn and S.A. Levine, Archs intern. Med. *34,* 1421 (1951).
7 J. Stamler, Circulation *58,* 3 (1978).
8 C.D. Spielberger, R.L. Gorsuch and R.E. Luschene, Manual for the State-Trait Anxiety Inventory. Consulting Psychologist Press, 1970.
9 M. Zuckerman and B. Lubin, Manual for the Multiple Affect Adjective Check List. Educational and Industrial Testing Service, 1979.
10 C.A. Aymen and K.T. Francis, in preparation.
11 Lipid Research Clinics Program: Manual of Lab. Operations, Vol. I, Lipid Analysis, DHEW Publication No.(NIH) 75-628, 1974.
12 E. Rolleri, M. Zinino, S. Orlandini and R. Malvano, Clin. chem. Acta *66,* 319 (1976).
13 R.J. Henry, C. Sobel and J. Kim, Am. J. clin. Path. *28,* 152 (1957).
14 F. Dreyfuss and J.W. Czaczkes, Archs intern. Med. *103,* 708 (1959).
15 M. Badian, E. Appel, D. Palm, W. Rupp, W. Sittig and K. Taeuber, Eur. J. clin. Pharmac. *16,* 171 (1979).

Plasma catecholamines in conscious rats: Influence of sodium, stress and heredity[1]

Monique De Mendonca, Pascale Guicheney, Marie-Laure Grichois, D. Ben-Ishay and P. Meyer

Unité de Recherches de Physiologie et Pharmacologie Vasculaire et Rénale, INSERM U.7, Hôpital Necker, F-75015 Paris (France), 12 January 1981

Summary. Plasma catecholamines are increased in sodium-loaded rats under both resting and stress conditions. Under stress, Na^+ resistant rats have lower plasma catecholamines than salt-sensitive ones.

Clinical and experimental data both strongly support the involvement of increased sympathetic activity in hypertension (for review, Axelrod[2]), and it has also been suggested that sodium (Na^+) may be one of the factors triggering this hyperactivity[3].

The available information concerning the effect of Na^+ on sympathetic activity is controversial. While some authors have reported that acute or chronic Na^+ loading enhances catecholamine excretion[4], increases plasma noradrenaline (NA)[5-7] and depletes heart catecholamine stores[8], others