dependent[3,9-11,13,18]. The finding that the potency of GABA 'binding' decreased from higher to lower CNS structures is in accord with its relative potency in iontophoretic studies[3,4]. The potency of glycine 'binding' was not so clearly related to its iontophoretic potency. Results with strychnine provided evidence for the lack of specificity of this agent[3,22,23]. Its inhibition of the binding of glycine and GABA indicates that it interacts with Na^+-dependent binding mechanisms involved in the uptake of these amino acids.

22 A. R. Freeman, J. Neurobiol. *4*, 567 (1973).
23 D. A. Pollen and C. Ajmone Marsan, J. Neurophysiol. *28*, 342 (1965).

Periodicity in body temperature in man

J. Empson

Department of Psychology, The University, Hull HU6 7RX (England), 8 September 1976

Summary. Body temperatures and self-assessed mood scales were recorded for 7 weeks or more by male volunteers. Auto-correlations of temperatures from the 21 subjects show evidence of a significant peak at the 20-day-interval. The self-assessed measure of 'Alertness' (from 18 of them) also shows some evidence of rhythmicity, at the 22-day-period.

It is well-known that there are regular variations in body temperature in the human female, accompanying the menstrual cycle. Little is known about any analogous phenomenon in the male, despite the fact that there is some evidence of periodic (20-day) changes in urinary 17-ketosteroid excretion[1], and of a 20-day cycle in pitch perception[2]. The present study arose from the author's work on the 90-min Rapid Eye Movement sleep cycle (REMS/SWS cycle), using penile skin temperature as a measure of the erections accompanying REM sleep[3,4]. Frequency analysis showed little evidence of stable patterns of REM sleep over 17 weeks in 1 subject. However, taking average temperature values for each night, frequency analysis showed evidence of periodicity at 21 days. It was this tentative 'finding' which stimulated this subsequent experiment.

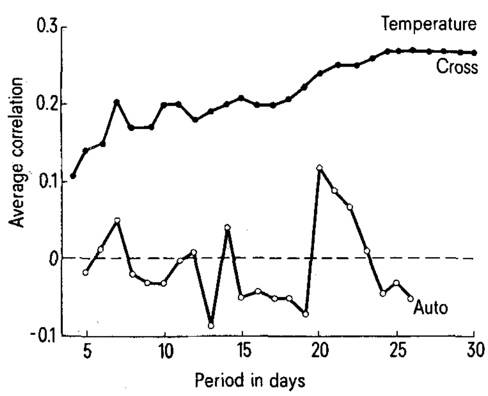

Fig. 1. Average auto-correlogram and cross-correlogram for temperature time series (N = 21).

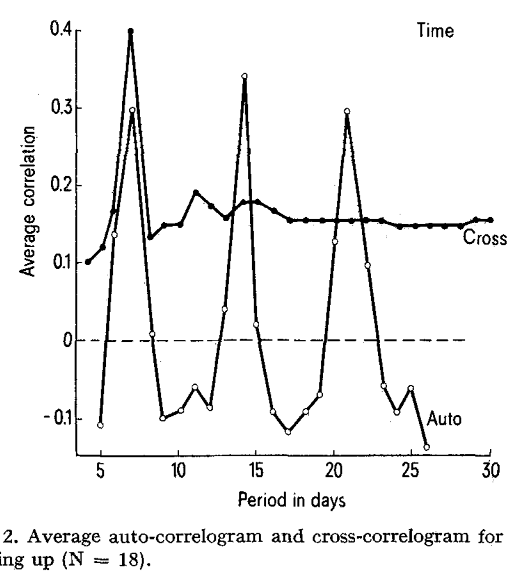

Fig. 2. Average auto-correlogram and cross-correlogram for time of getting up (N = 18).

Method. 21 male subjects, including undergraduate, postgraduate and staff members of the University of Hull, recorded their body temperature daily, for periods varying from 49 to 102 days. They were asked to continue for at least 7 weeks, and for as long as possible after that. 1 subject (the author) provided all-night average armpit skin temperatures. The others took their oral temperature with a clinical thermometer every morning, before getting up, noting the time at which the recording was made. 18 of them also provided estimates of their mood, and the previous night's sleep quality, using 4 10 cm analogue scales[5]. The 3 mood scales were labelled at each end with the adjectives found to be most highly loaded on the extremes of 3 major bipolar factors in mood, recently assessed in a British university population[6]. These scales could best be described as being 'Alertness-Dullness', 'Anxiety-Confidence and 'Elation-Quietness'. Subjects were instructed to complete the analogue scales as soon as they had got up.

Results. 18 subjects provided measures on each of 6 scales – temperature, time of recording, 3 mood scales and sleep quality. 3 others provided temperature measures only. Each number series was analyzed, using both auto-correlation and sinusoidal cross-correlation. (The analogue scale series were each ranked before analysis, as it is highly unlikely that subjects would be using strictly interval scaling in their self-assessments.)

Figure 1 shows the average correlograms for temperature, and it is apparent that there is a peak in the auto-cor-

1 F. Halberg, A. Rev. Physiol. *31*, 675 (1969).
2 V. T. Wynn, J. Physiol. Lond. *220*, 627 (1972).
3 P. Ohlmeyer, H. Brilmayer and H. Huellstrung, Pflügers Arch. ges. Physiol. *248*, 559 (1944).
4 C. Fisher, J. Gross and J. Zuch, Archs gen. Psychiat. *12*, 29 (1965).
5 R. C. Aitken, Proc. R. Soc. Med. *62*, 989 (1969).
6 R. Meddis, Br. J. Soc. Clin. Psychol. *11*, 178 (1972).

relogram at the 20-day period. 17 of the 21 subjects showed a positive value at this point, statistically significant at the $p < 0.01$ level (sign test, two-tailed). Auto-correlation at the 21-day period is similarly statistically significant at the $p < 0.05$ level, with 15 out of 21 showing a positive value. There are no other significantly positive auto-correlations in the spectrum. Cross-correlation fails to show any clear peak. This indicates that the fluctuations producing positive auto-correlations at 20 and 21 days are by no means sinusoidal.

Figure 2 shows the results of frequency analysis on the *time at which subjects made their recordings*. The cross-correlogram shows a clear peak at 7 days, but no peak at 20 or 21 days. The auto-correlogram shows statistically significant peaks at the 7-day interval and subsequent harmonics.

The 'Alertness-Dullness' auto-correlogram showed small peaks at 21 and 23 days, and a significantly positive value at the 22-day interval ($p < 0.05$; sign test, two-tailed). Analyses of the other self-reported mood and sleep quality scales showed no clear evidence of periodicity.

Discussion. The correlograms shown in figure 2, of time of day, require some interpretation. In general, if the auto-correlation at 1 period is highly positive, then, inevitably, auto-correlations at every subsequent harmonic of that period will be similarly highly positive, unless there is a phase shift. The apparent peak in the 'Time' auto-correlogram at 21 days, like the slightly larger one at 14 days, is probably artefactual and produced by the high auto-correlation at the 7-day interval. (There would be no question of phase shift, since all subjects were almost certainly strongly entrained by our calender week.) Cross-correlation shows no hint of a peak at 21 days. It is therefore unlikely that the observed 20-day periodicity in temperature was mediated through variations in the time at which the recordings were made.

It is tempting to speculate that the reported periodicities in steroid excretion[1], and pitch perception[2], like the variations in temperature, and perhaps even mood, reported here, are manifestations of some stable intrinsic rhythm in the human male. It will be necessary to carry out prolonged series of measurements before anything can confidently be said about either the shape of the temperature cycle, or its phase relation to any other cycles. The evidence presented here, however, supports the notion of a temperature cycle of about 20 days period length.

Inhibition of growth in developing oocytes of the desert locust

S. S. Tobe[1]

Department of Zoology, University of Toronto, 25 Harbord Street, Toronto (Ontario, Canada M5S 1A1),
3 August 1976

Summary. The location of the terminal oocytes (T) in the ovary of the desert locust dictates whether the penultimate oocytes (T-1) will enter vitellogenesis – as long as the T oocytes are retained within the ovarioles, vitellogenesis in T-1 oocytes is prevented. When the T oocytes are ovulated into the oviduct, growth of the T-1 oocytes (new T) resumes. Inhibition of vitellogenesis in T-1 oocytes is not due to low rates of JH biosynthesis since high rates of JH biosynthesis were observed in animals in which T oocytes were retained in the ovarioles.

Oocyte growth in many insects is a highly coordinated synchronous process, with batches of oocytes maturing at the same rate for oviposition at the same time. In these insects, only the terminal oocytes in individual ovarioles reach maturity during any given reproductive cycle. This implies that the penultimate and younger oocytes are prevented from completing maturation. This inhibition usually involves the arrest of growth in the penultimate oocytes (T-1), as long as the terminal oocytes (T) are retained within the female reproductive system. Thus, in females bearing oothecae, in females retaining mature oocytes and in females reared in the absence of males, the ultimate effect of this inhibition is to prevent the growth and maturation of T-1 and younger oocytes[2]. There are at present two types of inhibition of oocyte maturation which have been postulated to operate in insects. The first of these, which has been hypothesized to function in houseflies, ovoviviparous cockroaches and the Hemipteran, Iphita limbata, suggests that a humoral factor is released from the reproductive system which then acts upon the corpus allatum (CA), preventing the release of the juvenile hormone (JH)[3–6]. Accordingly, egg production is reduced or stopped, since the maturation of oocytes in most insects requires the presence of 'active' CA[2]. The second hypothesis does not involve the release of JH from the CA but rather postulates that a factor is released from the ovaries which acts directly upon the follicular epithelium to prevent the uptake of vitellogenin by the maturing oocytes; such a mechanism has been postulated in Rhodnius prolixus[7,8].

In the desert locust Schistocerca gregaria, T-1 oocytes do not enter vitellogenesis in normal females as long as either maturing or mature T oocytes are present[9,10]. Thus it seemed likely that some type of inhibition operated to prevent T-1 oocytes becoming vitellogenic. Highnam[11] suggested that the presence of mature oocytes at least partially restricted the activity of the CA. The hypothesized mode of action of this inhibitory factor in S. gregaria

1 I thank S. Honsl for technical assistance and Prof. K. G. Davey and Dr G. E. Pratt for critically reading the manuscript. This work was supported by National Research Council of Canada Operating, grant A9408.
2 F. Engelmann, in: The Physiology of Insect Reproduction. Pergamon Press, Oxford 1970.
3 T. S. Adams, A. M. Hintz and J. G. Pomonis, J. Insect. Physiol. *14*, 983 (1968).
4 T. S. Adams, J. Insect. Physiol. *16*, 349 (1970).
5 F. Engelmann, Arch. Anat. microsc. *54*, 387 (1965).
6 K. K. Nayar, Proc. Ind. Acad. Sci. B *47*, 233 (1958).
7 G. E. Pratt and K. G. Davey, J. exp. Biol. *56*, 223 (1972).
8 E. Huebner and K. G. Davey, Can. J. Zool. *51*, 113 (1973).
9 K. C. Highnam, O. Lusis and L. Hill, J. Insect. Physiol. *9*, 827 (1963).
10 S. S. Tobe and G. E. Pratt, J. exp. Biol. *62*, 611 (1975).
11 K. C. Highnam, Roy. Ent. Soc. Lond., Symp. No. 2, 26 (1964).