

tion; c) the ($z \rightarrow z'$) axis is directed from the inferior to the superior face of the nucleotide base molecule. The differentiation of the 2 faces of a nucleotide base molecule has a functional significance, because in the DNA system, 2 complementary nucleotide bases interconnect so that locking at the common molecular plan, a nucleotide base is directed with the superior face, and another with the inferior face to the observer.

It is possible to imagine some nucleotide bases symmetrical in mirror with the natural nucleotide bases, by substituting in the natural nucleotide bases the carbon, nitrogen, oxygen and hydrogen atoms with their correspondent antiatoms. In the antinucleotide base molecules, the rotational direction of the positrons in the ring becomes: $C^-(1) \leftarrow N^+(2) \leftarrow C^-(3) \leftarrow N^+(4) \leftarrow$ and the ($z \rightarrow z'$) axis changes its direction (figure 1, b). Because the antinucleotide bases are not present in nature, the asymmetry of the nucleotide bases is unique in nature.

The asymmetry of the amino acids molecules can be defined by 3 orthogonal axes: a) The ($x \rightarrow x'$) axis goes from C_α -asymmetric carbon to the chemical group situated at the end of the root R (figure 2, a). b) The ($y \rightarrow y'$) axis goes from ($-NH_3^+$) group to ($-COO^-$) group (figure 2, a). c) The ($z \rightarrow z'$) axis is perpendicular in origin to the above-mentioned plan. The peak of the ($z \rightarrow z'$) axis is directed in the reverse direction of the hydrogen atom, which is bound to the C_α -carbon atom (figure 2, a). By directing a L-amino acid and a D-amino acid molecules, so to have their ($x \rightarrow x'$) axes and their ($y \rightarrow y'$) axes parallel, then the 2 ($z \rightarrow z'$) axes are antiparallel (figure 2, a and b).

Living organisms are strictly selective for L-amino acids³⁻⁵. A polypeptide chain, formed by L-amino acids, usually (due to the minimum energy) has the form of a right-handed α -helix⁶. The D-amino acids, usually make polypeptide chains having the shape of a left-handed α -helix. The available data indicate that the 2 strands of DNA form a right-handed double helix⁷. The concordance between the direction of rotation of a polypeptide chain, built by L-amino acids, and the polynucleotide strands of DNA, certainly play an important functional role. For example, it favours the coupling between the polypeptide signal molecules in DNA and their corresponding receiver genes. This indicates that the predilection of the living organisms for L-amino acids is due to the unique asymmetry of the nucleotide bases.

- 1 L. Pauling, General Chemistry, p.183. W.H. Freeman, San Francisco 1970.
- 2 A. Julg, *Chimie Quantique*, p.179. Dudod, Paris 1967.
- 3 R.P. Feynman, *Lecture on Physics*, vol.I, p.821. Wesley, Massachusetts 1965.
- 4 F. Bades and F. Kerek, *Stereochimie*, p.220. Stiintifică, Bucuresti 1974.
- 5 L. Stryer, *Biochemistry*, p.14. W.H. Freeman, San Francisco 1975.
- 6 A. Lehninger, *Biochemistry*, p.864. Worth Publishers, New York 1975.
- 7 E. Harbers, D. Götz and W. Müller, *Introduction to Nucleic Acids*, p.53. Reinhold Book Corporation, New York 1968.

Circadian change of sweating rate measured locally by the resistance hygrometry method in man

H. Tokura, T. Ohta and M. Shimomoto¹

Laboratory of Physiology, Department of Clothing Sciences, Nara Women's University, Nara 630 (Japan), 11 September 1978

Summary. There existed circadian change in the sweating rate locally measured from the anterior of the left thigh: the sweating rate showed a remarkable decline during the period 2.00–5.00 h, while at other times throughout the day it generally remained high. This reduction seemed to be independent of sleep or sleeplessness.

In thermoneutral ambient temperature zones, there exists a circadian change of total dry heat loss due to radiation, convection and conductance, and this circadian change is primarily responsible for the creation of the circadian core temperature rhythm in man^{2,3}. However, it remains to be known to what extent the sweating rate could contribute to the circadian rhythm in core temperature under high ambient temperatures. Although studies have been reported about the change of sweating rate associated with nocturnal sleep⁴⁻⁶, the day-night change of threshold for sweating in esophageal temperature of man exercising on a bicycle ergometer in a 25°C ambient⁷, and the circadian variation of sweating responses to heat stimulation⁸, to our knowledge no attempts have been made yet to record the sweating rate consecutively for 24 h. Therefore, in the present experiment we endeavoured to measure the sweating rate under constant warm ambient temperature (32°C) and relative humidity (50–55%) to determine whether or not man has circadian rhythm in his sweating rate. The experiment was executed in August and September 1977 in a climatic chamber with natural sunlight coming through the window. The ambient temperature and relative humidity were controlled constant at 32±1°C and 50–55%, respectively. 3 males (2: 19 years, 1: 38 years) and 1 female (22

years) served as subjects. Males wore trunks only, and female wore also a low cut sleeveless shirt. Sweating rate was continuously measured from the anterior of the left thigh from noon of one day to noon of the next day by the resistance hygrometry method⁹. A 1 l/min air flow rate passed through the sweat measuring system. The subjects entered the climatic chamber about 1 h (11.00 h) before the recording started. Each sat on a chair from 12.00 to 23.00 h quietly and then lay down without coverlet on a bed with cotton mattress from 23.00 to 7.00 h. From 8.00 to 12.00 h they again sat on a chair. In 3 cases, rectal temperature was recorded every minute with 2 subjects, using copper-constantan thermocouples. Continuous recordings of the sweating rate for 24 h were carried out 6 times with 4 subjects. Representative results of one of the male subjects are depicted in figure 1. As seen in figure 1, the sweating rate decreased definitely during the period 2.00–5.00 h. This reduction during these times of day occurred without exception in all observations with 4 subjects. The average values during the period 2.00–5.00 h were 0.07 ± 0.05 mg/min/cm² (mean±SD), which were significantly lower than those (0.12 ± 0.08 mg/min/cm²) during other times of the day ($p < 0.003$). Furthermore, sleep or sleeplessness seemed to have little influence on this

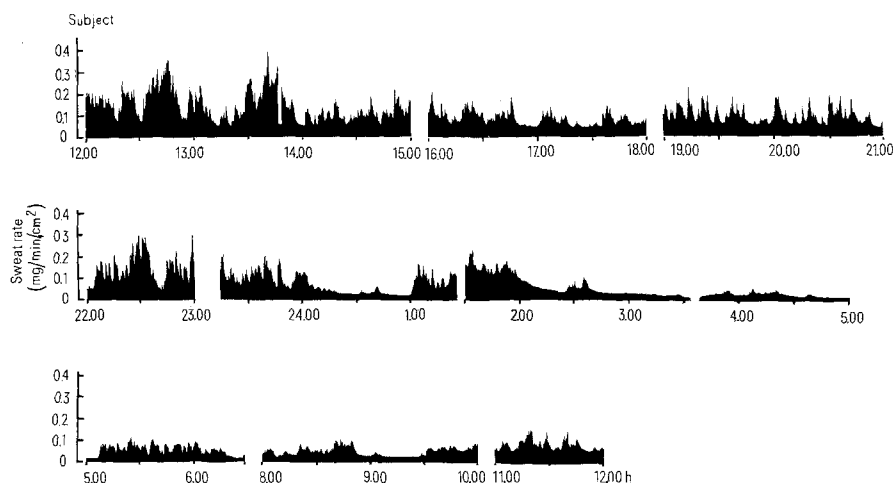


Fig. 1. A 24-h recording of the sweating rate taken from the anterior of the left thigh in a resting male subject under constant conditions (T_a 32 °C, R.H. 50-55%).

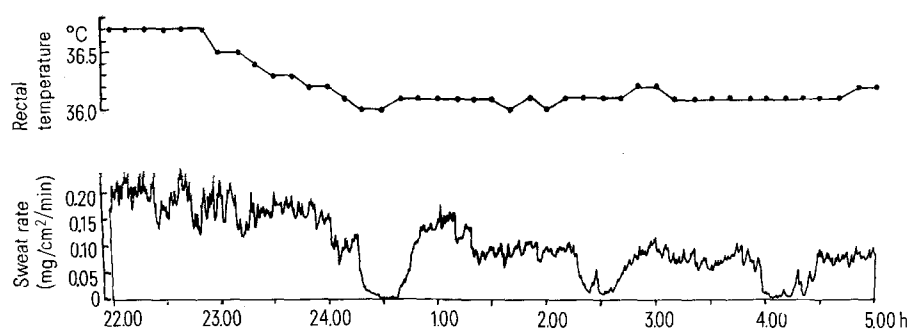


Fig. 2. Rectal temperature (top) and sweating rate (bottom) during the period 22.00-5.00 h in a resting male subject under constant conditions (T_a 32 °C, R.H. 50-55%).

decrease of the sweating rate, because although 2 subjects could not sleep at all (according to their subjective judgment), the values of their sweating rate during the period 2.00-5.00 h were significantly lower (0.10 ± 0.05 mg/min/cm²) than those (0.15 ± 0.07 mg/min/cm²) during other times of the day ($p < 0.01$). It is also definitely shown that the circadian rhythms of core temperature and plasma cortisol in man do not depend upon sleep and sleeplessness^{10,11}. This nocturnal reduction of the sweating rate from 2.00 to 5.00 h supports the results reported by some investigators⁴⁻⁶ and is not inconsistent with Takagi's assertion⁵ that thermogenic sweating rate is not associated with depth of nocturnal sleep. During the periods 12.00-2.00 h and 8.00-12.00 h, the sweating rate generally remained high although abrupt and temporary declines occurred sometimes. This circadian pattern of the sweating rate measured locally from the anterior of the left thigh is substantially similar to that of the total dry heat loss³ and insensible perspiration¹². In figure 2, the sweating rate and rectal temperature in a male subject are represented simultaneously as a typical example. At about 23.00 h rectal temperature began to decline rapidly and at about 24.30 h became a steady state which continued until 5.00 h. Between the period 23.00-24.00 h, when rapid decline of rectal temperature occurred (the rates of decline of rectal temperatures in 3 cases were 0.54, 0.70 and 0.69 °C, respectively), the sweating rate remained high, but decreased sharply during the period 2.00-5.00 h. This reduction of sweating rate was accompanied by a minimum level of rectal temperature (figure 2). The average values of the sweating rate during the period 23.00-24.00 h when it remained high, were 0.13 ± 0.03 mg/min/cm², significantly higher than those (0.07 ± 0.05 mg/min/cm²) during the period 2.00-5.00 h ($p < 0.001$). On the other hand, there is a circadian rhythm of heat production^{2,3}: maximum values lie between

the period 20.00-23.00 h and after that time heat production falls abruptly, showing minimum values from 2.00 to 3.00 h. With these in mind, a high level of sweating rate and rapid falling of heat production^{2,3} at about 23.00 h might be responsible for the sudden and abrupt decline of rectal temperature under these comparatively high ambient temperatures, where dry heat loss plays a role to a lesser degree.

- 1 Acknowledgment. The authors thank sincerely the late Prof. S. Mizunashi for her constant encouragement throughout the work. This research was aided by a grant from Japan Chemical Fiber Association.
- 2 J. Aschoff and A. Heise, in: *Advances in Climatic Physiology*, p.334. Ed. S. Itoh, K. Ogata and H. Yoshimura. Igaku Shoin, Tokyo 1972.
- 3 J. Aschoff, H. Biebach, A. Heise and T. Schmidt, in: *Heat Loss from Animals and Man*, p.147. Ed. J.L. Monteith and L.E. Mount. Butterworth, London 1974.
- 4 E.H. Geschickter, P.A. Anders and R.W. Bullard, *J. appl. Physiol.* 21, 623 (1966).
- 5 K. Takagi, in: *Physiological and Behavioral Temperature Regulation*, p.669. Ed. J.D. Hardy, A.P. Gagge and J.A.P. Stolwijk. Thomas, Springfield 1970.
- 6 R. Henane, A. Buguet, B. Roussel and J. Bittel, *J. appl. Physiol., respir. envir. Exercise Physiol.* 42, 50 (1977).
- 7 C.B. Wenger, M.F. Roberts, J.A.J. Stolwijk and E.R. Nadel, *J. appl. Physiol.* 41, 15 (1976).
- 8 J. Timbal, J. Colin and C. Boutelier, *J. appl. Physiol.* 39, 226 (1975).
- 9 T. Nakayama and K. Takagi, *Jap. J. Physiol.* 9, 359 (1959).
- 10 J. Aschoff, in: *Physiological and Behavioral Temperature Regulation*, p.905. Ed. J.D. Hardy, A.P. Gagge and J.A.J. Stolwijk. Thomas, Springfield 1970.
- 11 J. Aschoff, *Klin. Wschr.* 56, 425 (1978).
- 12 H. Tokura, M. Shimomoto, T. Tsurutani and T. Ohta, *Int. J. Biomet.* 22, 271 (1978).