These observations are broadly parallel with those made on plasma proteins[4].

It has now been conclusively proved that a major proportion of ferritin synthesis in the liver takes place mainly on free polysomes, whereas the synthesis of export proteins, like plasma proteins, occurs predominantly on ribosomes bound to endoplasmic reticulum[11-13]. Further, it has been indicated that proteins synthesized on bound polysomes may be more susceptible to inhibition by ethanol than those synthesized on free polysomes[14,15]. Our present and previous[4] data indicate that both the types of proteins are equally affected by ethanol.

Very little is known about the effects of chronic alcohol ingestion on absorption and storage of iron, and synthesis of liver ferritin in man. From one report it appears that alcohol may increase iron absorption in normal subjects[16]. In many alcoholics parenchymal iron overload is encountered[17]. The stimulation of ferritin synthesis by iron is a very well recognized phenomenon[11]. From all this one would expect that under the conditions of chronic alcoholism ferritin synthesis would be increased. However, our studies in experimental animals suggest that chronic alcohol ingestion may inhibit ferritin synthesis. Recently studies in guinea-pigs have hinted that an adequate tissue concentration of ascorbic acid may be necessary for the maintenance of a quantitative correlation between tissue iron stores and ferritin[18]. This may be an important point for humans, since vitamin C status has been shown to be affected in alcoholism in humans[19].

At present the mechanism of action of PTU is speculative[3,4]. It is known that the level of reduced glutathione (GSH) is decreased by alcohol[20,21]. One of the recently-suggested mechanisms of PTU action is that the GSH levels are significantly raised in PTU-treated rats[22]. Further, the rate of metabolism of ethanol has been shown to be reduced in alcoholic rats treated with PTU[23]. This has strengthened the concept that the thyroid hormones may play a direct or permissive role in the process leading to liver damage. Our observation on liver ferritin synthesis may be yet another manifestation of the beneficial role of PTU in alcoholism.

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Photoperiod-induced changes in temperature-metabolism curve, shivering threshold and body temperature in the pigeon

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Summary. A short photophase combined with cold-acclimation changed the shivering threshold of the pigeon from 21 to 14 °C. The change of critical temperature was also found in the temperature-metabolism curve. A long photophase both alone and combined with warm-acclimation induced a reduction of body temperature to a lower level (about 2 °C) at all ambient temperatures, ranging from 5 to 40 °C.

Seasonal thermoregulatory adaptation, involving insulation and basal metabolic rate (BMR), has been demonstrated in many bird species[1-4]. This adaptation is also indicated by a decrease in a bird's critical temperature in winter[5-7], the decrease being, for example, 10 °C in the willow ptarmigan[5]. It is assumed that seasonal variations in heat production may reflect mainly variations in the overall insulation of the bird[8]. Rautenberg's[9] results showing that the critical temperature of pigeons did not shift during cold- or warm-acclimation lasting for several months in laboratory conditions suggest that during acclimation the adaptation is not insulative. However, when ambient temperature (Ta) is decreased, the heat production capacity of fasted birds seems to be higher when they are cold-acclimated than when they are warm-acclimated (see Hart[10]).

Until now most acclimation studies have been performed under standard photoperiod conditions, thus eliminating the effect of the length of photophase on birds' metabolic adaptation. Our recent results show that the photoperiod may have a strong influence on birds' temperature regulation[11,12] and tissue monoamine and lipid metabolism[13]. In this paper we report experiments investigating the effect of acclimation combined with a natural length of photoperiod (i.e. short photophase and cold-acclimation, and long

photophase and warm-acclimation) on the temperature-metabolism curve and on the presence of shivering thermogenesis in the pigeon.

Materials and methods. The study was carried out with 23 adult pigeons *(Columba livia)* of both sexes, weighing 0.28–0.37 kg. Prior to the experiments pigeons were housed in individual cages at 22 °C under a 12L:12D controlled photoperiod. Food and water were freely available. After 3 weeks of adaptation to laboratory conditions, the birds were divided into 4 groups which were acclimated for 21 days to the following experimental conditions:

Group 1 12L:12D/22 °C – control group (photophase 06.00–18.00 h).

Group 2 4L:20D/2 °C – simultaneous short photophase and cold-acclimation simulating winter conditions (photophase 10.00–14.00 h).

Group 3 20L:4D/22 °C – long photophase (scotophase 01.00–05.00 h).

Group 4 20L:4D/32 °C – simultaneous long photophase and warm-acclimation simulating summer conditions (scotophase 01.00–05.00 h).

Body weight and body temperature (Tb, from the wingpit) were recorded before and after acclimation to the various experimental conditions. The influence of photoperiod on temperature regulation was investigated under different Ta's ranging from 5 to 40 °C by recording the patterns of heat production as 1. oxygen consumption (measured using Beckman E2 and Taylor Servomex OA184 precision oxygen analyzers, and a flow rate of dried and CO_2-free air at 0.7 l/min), and as 2. an integrated EMG of the nonpostural pectoral muscle (measured using a frequency integrator FA-790 coupled to a Varioscript V822, Schwarzer). The patterns of Tb and foot temperature (Tf, naked tarsometatarsus) were recorded by an Ellab Z94-B and a Foster Cambridge Clearspan P250L recorder. Gross activity was eliminated by using a special frame constructed for pigeons. Experiments were conducted from 09.00 to 17.00 h, and all tests were run in the dark.

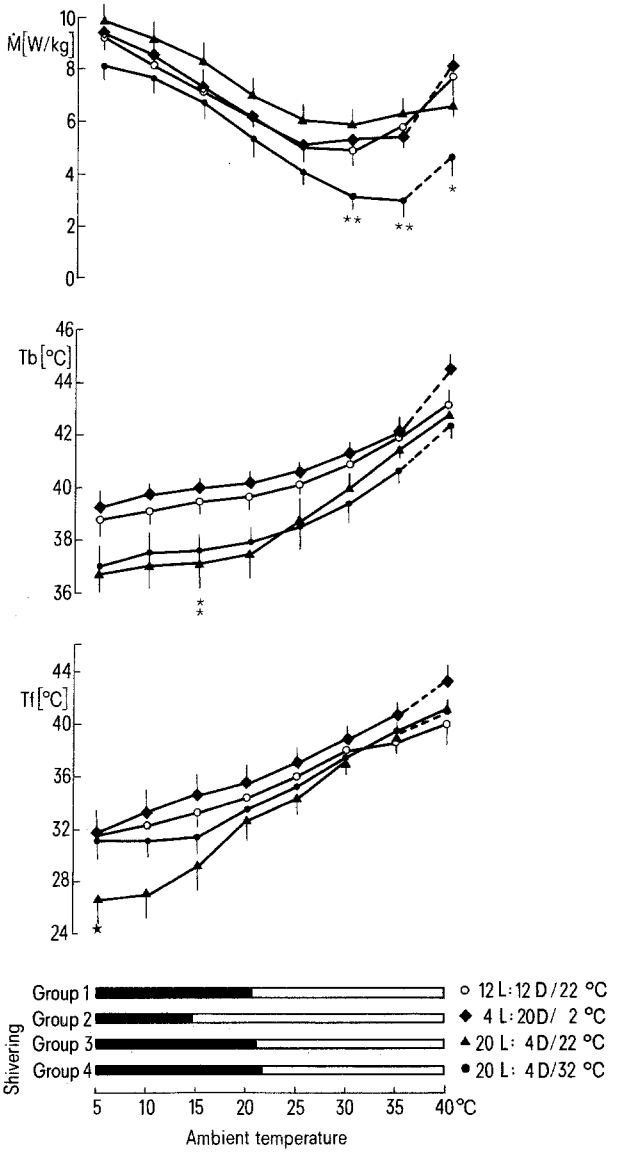
The direction of changing Ta was found to have no effect on results. Usually pigeons were exposed first to 5 °C. The recording of variables was not started before the equilibration. After getting 3 similar values for each variable, Ta was raised by steps of 5 °C. For each Ta the recording procedure was as above.

Metabolic rate (Ṁ) was calculated by using the flow rate into the chamber multiplied by the percentage of oxygen consumed and correcting for CO_2-free air. The Beckman analyzer was calibrated against a known standard of O_2-gas, the Servomex against N_2-gas and both analyzers also against atmospheric O_2. Statistical analyses were performed according to Student's t-test.

Results and discussion. Whereas long photophase-warm acclimation (20L:4D/32 °C) induced a significant decrease (11.4%, $p < 0.001$) in the body weight of pigeons, a result which is in agreement with our previous findings[11], pigeons in the other 3 groups maintained their body weight during the experimental acclimation period.

As can be seen from the curves in the figure, Ṁ of group 4 (='summer'-acclimation) remained at the lowest level in all Ta's with the greatest difference occurring in warm Ta's. However, 'winter'-acclimation (group 2) produced the same shape of curve as the control group (group 1). The curve of group 3 was above all these 3 curves. This result resembles the one we reported previously at 6 and 26 °C[11], and in addition, it has been found that birds maintained in continuous light have an elevated BMR[14]. The slopes of the curves did not differ in cold Ta's, but M of groups 1 and 2 increased faster between 35 and 40 °C compared with the curve of group 3, perhaps because of the poor adapation to high Ta's. The minima of M were at 30 °C in groups 1 and 3 (Tb = 41.1 and 40.1 °C, respectively), while pigeons in group 4 reached the minimum at 35 °C (Tb = 40.8 °C) and 'winter'-pigeons (group 2) at 25 °C (Tb = 40.8 °C). When Tb is calculated as a moving average, Tb of pigeons in group 3 did not differ from the controls at the minimum of Ṁ, although long photophase-warm acclimation (20L:4D/32 °C = group 4) seems to induce a decrease of 1.1 °C and 4L:20D/2 °C (group 2) an increase of 0.5 °C in Tb's.

In trying to find the critical temperature according to the presence/absence of shivering, we observed that the critical point for control pigeons was 21 °C (see fig.), the same that Rautenberg[9] also observed for cold- and warm-acclimated birds. The inhibition of shivering by increasing Ta was noted as starting in group 3 at 22 °C and in group 4 at 23 °C. On the other hand, shivering disappeared in group 2 at a temperature as low as 14 °C. This critical temperature is the same that Steen[15] determined by means of Ṁ for cold-acclimated pigeons. However, Rautenberg[9] did not find any shift in the shivering threshold during temperature acclima-



Relation of metabolic rate (Ṁ), body temperature (Tb), foot temperature (Tf) and shivering to ambient temperature. Each point is a mean ± SE of 5–8 values, except in ◆ at 40 °C n = 3 and in ● at 40 °C n = 2 expressed as dotted lines.

tion of pigeons, and Hart[2] did not report any seasonal difference in the shivering of pigeons. Although a progressive decrease was found in shivering as Ta approached the critical point, so conflicting with the results of West[16], we did not find a linear correlation between the intensity of shivering and M. Although shivering disappeared, M remained elevated.

The relatively high critical temperature of the pigeon can be understood, when it is pointed out that unlike mammals, shivering is perhaps the only mechanism by which birds produce extra heat when Ta decreases below thermoneutrality. Thus shivering is elicited just at birds' critical temperature. Furthermore, the shivering threshold is correlated to bird's size and insulation, being highest in small tropical birds and lowest in 'insulative specialists', like arctic gallinaceous birds.

Pigeons acclimated to long photophase (groups 3 and 4) regulated their Tb's and Tf's on a lower level (about 2 °C) than the controls or 'winter' pigeons, especially below their critical temperature (see fig.). The result is in agreement with previous observations that winter birds have a higher Tb and a greater difference between skin and Ta compared with summer birds[6]. The increased skin temperature of the body and extremities has also been noticed in cold-acclimated pigeons[9].

In conclusion it can be stated that photoperiod is important in birds' thermoregulatory adaptation; it provokes changes in insulation, in the lower critical temperature, in the shivering threshold and in the relation between skin temperature and Ta. These photoperiod-induced changes in pigeons' heat production, Tb and Tf may result from the influence of the photoperiod on catecholamine and lipid metabolism (as found just recently[13]), and they show that the pineal gland has an important role to play in the temperature regulation of birds. It is thought that the thermoregulatory influence may be mediated through an inhibitory action of pineal melatonin on the thyroid gland (discussed previously by Ralph et al.[17] and Saarela[18]).

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Enhancement of boll weevil *Anthonomus grandis* Boh. (Coleoptera: Curculionidae) pheromone biosynthesis with JH III[1]

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Summary. The juvenile hormone JH III, when incorporated at 1.0 ppm in the diet of adult male boll weevils (*Anthonomus grandis* Boh.), increased the biosynthesis of its 4 pheromone compounds by 3 times. The biosynthesis at lower and higher levels of JH III was less. JH I was not active at any of the concentrations tested.

The 4 monoterpene compounds that comprise the male boll weevil (*Anthonomus grandis* Boh.) pheromone [I, (+)-cis-2-isopropenyl-1-methylcyclobutaneethanol; II, (Z)-3,3-dimethyl-$\Delta^{1\beta}$-cyclohexaneethanol; III, (Z)-3,3-dimethyl-$\Delta^{1\alpha}$-cyclohexaneacetaldehyde; and IV, (E)-3,3-dimethyl-$\Delta^{1\alpha}$-cyclohexaneacetaldehyde] were identified and synthesized in 1969[2]. The biosynthesis of these compounds has since been reported to be a function of a number of factors. It increases with age[3], it is higher in the summer than in winter with insects reared in the laboratory[4], it decreases when males are fed a laboratory diet rather than cotton (*Gossypium hirsutum* L.) buds[5], it further decreases if chemosterilants are incorporated in the diet[5], and it decreases in insects with a high gut bacterial load[6]. The pheromones may be synthesized in the fat bodies of the abdomen shell or in the gut, because the greatest concentrations are found in these tissues[4]. If synthesized in the fat bodies, they may be transported to the gut via the malpighian tubules for excretion with fecal pellets. The most concentrated source of the pheromones is the fecal pellets[5], and the release is rhythmic with the peak from 07.00 to 13.00 h[7]. The peak periods for feeding and fecal release also occur during these hours.

For physiological studies and field release programs, it is very important that laboratory reared insects possess high vigor, and that males be sexually competitive with wild males. Improved pheromone biosynthesis would contribute to these objectives. Amerasinghe[8] reported that the pheromone biosynthesis of allatectomized *Schistocerca gregaria* males injected with juvenile hormone JH I (3, 11-dimethyl-10 epoxy-7-ethyl-trans, trans-2, 6-tridecadienoic acid, methyl ester) and JH III (10-epoxy-3,7,11-trimethyl-2,6-trans, trans-dodecadienoic acid, methyl ester) was restored. Therefore, this study was initiated to determine whether JH I or III would promote pheromone biosynthesis in the male boll weevil. Because administration of these compounds by topical application would be of little practical significance

Table 1. Pheromone biosynthesis of male boll weevils fed diets containing JH I, µg/♂/day

Days	JH I, ppm				Cotton buds
	0.0	0.1	1.0	10.0	
1–4	0.15	0.18	0.15	0.14	0.16
5–8	0.20	0.16	0.13	0.21	0.30
9–12	0.26	0.25	0.20	0.26	0.48