The effect of sudden solar exposure on thermophysiological parameters and on plasma prolactin and cortisol concentrations in male Creole goats

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Summary. Rectal temperature (RT), respiratory rhythm (RR), plasma cortisol and prolactin (PRL) levels and haematocrit were measured at noon in male Creole goats during their habituation to shade, during sudden exposure to sunlight and then while they were kept outdoors. Data on the microclimatic environment, especially black-globe temperature (Tg), were recorded.

On the day (DO) the bucks were put in the sun, the increase of RT and RR, characteristic of circadian rhythm, was amplified (RT : 40.84 ± 0.23 °C vs 39.48 ± 0.19 °C P < 0.001 ; RR : 98 ± 22 vs 35 ± 3 respirations/min ; P < 0.01). Plasma cortisol also increased (17.7 ± 8.6 vs 5.57 ± 1.7 ng/ml ; P < 0.05). After 24 h (D1), plasma prolactin concentration increased dramatically (923 ± 653 vs 90 ± 22 ng/ml ; P < 0.05) and haematocrit decreased (28.6 ± 2.3 vs 33 ± 2.3 % ; P < 0.05).

To clarify the simultaneous effects of time (t) and Tg, we developed a model Log-normal on t and linear on Tg for RR and PRL. A model decreasing exponentially with t and linear with Tg was developed for RT ; the determination coefficients were R² = 0.96, 0.75 and 0.59, respectively.

About 3 weeks later, after the adaptation period, RT, RR and plasma PRL stabilized ; RT and RR returned to shade values while the PRL level remained higher than the shade PRL level (248 ± 109 vs130 ± 50 ng/ml ; P < 0.05).

The 24-hour time-lag in prolactin hypersecretion and the role of prolactin in thermoregulation are discussed.

Introduction.

Ruminants subjected to heat develop thermolytic mechanisms, such as panting and sweating (Bianca, 1965), and voluntarily restrict their food intake. However, these mechanisms are not fully efficient immediately and a period of adaptation to the new environment is necessary (Ladell, 1964).
The aim of the present experiment was to define the parameters involved in physiological response to heat constraint and to study the adaptation period using such simple thermophysiological criteria as rectal temperature, respiratory and cardiac rhythms and hormonal (prolactinemia and cortisolemia) and blood (haematocrit and serum electrolyte concentration) factors.

Shade-acclimatized male Creole goats were suddenly exposed to the sun in a natural tropical environment that also included nocturnal cooling (natural photoperiod: 12 to 13 h of light). This paper describes the physiological events occurring when the bucks were abruptly submitted to direct sunlight. We were particularly interested in the time required to develop a heat-loss mechanism and the time-lag in prolactin hypersecretion.

Material and methods.

The experiment was carried out at the I.N.R.A. farm « Gardel in Grande-Terre » on a dry plateau in Guadeloupe, French West Indies (16° N latitude and 61° W longitude).

**Animals and feeding.** — Four entire dwarf male goats with a mean age of 390 ± 9 days and a mean live weight of 16.75 ± 0.86 kg (mean ± SD) were kept tethered in cages similar to metabolism crates under a metal shelter from September 1982 to April 1983. On April 15, 1983 they were suddenly put outside in their cages and remained there until June 20, the dry season.

They were fed pangola (*Digitaria decumbens*) *ad libitum*; the pangola was cut at 33 days (mean age of regrowth) and included about 23% dry matter without concentrate supplement. Water was available *ad libitum*.

**Measurement frequency.** — Four days before solar exposure, bioclimatic and physiological measurements were taken every day between 11.00 and 12.30. The first day of exposure, they were taken every hour from 8.00 and every 20 min between 11.00 and 12.00 noon.

At 12.00 when the animals were still under the shelter, hormones, but not respiratory rhythm (RR), rectal temperature (RT) or black-globe temperature (Tg), were measured because of the lack of time; the goats were put in the sun between 12.00 noon and 12.10. These measurements were continued every 10 min until 13.00 and then every hour until 17.00. However, cardiac rhythm and haematocrit were not measured every time. Serum electrolytes were measured only during the hour the animals were actually in the sun.

Every day on the following days, the measurements were carried out between 11.00 and 12.00 and then at wider intervals. The experimental period extended from D-4 (four days before solar exposure) to D66 (last measurement).

**Climatic measurements.** — Dry (Td) and wet (Tw) air temperatures were measured using an Assman psychrometer placed at the height of the animals. Air velocity (V) was recorded with two cup anemometers (one inside the shelter and the other outside) having a starting threshold of 0.8 m/sec.

Black-globe temperature (Tg) was measured with an empty copper sphere painted black and containing a bulb thermometer at its center. This black-globe
temperature is a good index of the heat load (temperature and radiation) actually felt by a non-sweating animal (Berbigier, 1982; Chemineau and Ravault, 1984).

**Thermophysiological measurements and blood sampling.** — The physiological parameters were measured simultaneously as follows:
- rectal temperature (RT) by a medical thermometer left for 1 min in the rectum;
- respiratory rhythm (RR) by counting the flank beats;
- cardiac rhythm (CR) by a stethoscope.

At the same time, blood samples were taken from the jugular vein with EDTA/Na vacutainers for plasma hormone assay; silicon tubes were used for serum electrolyte determination. Blood samples were immediately put at 4 °C, then centrifuged for 10 min at 5 000 rpm. The plasma was collected and deep-frozen (−15 °C) until assay.

A sample of the total blood was put into capillar tubes for haematocrit determination (centrifugation at 10 000 rpm for 4 min).

**Hormone assay and ionic concentration.** — Cortisol was quantified according to the competitive protein binding radioassay of Murphy (1967) modified by Bosc and Frevre (1977) using dog CBG. Intra-assay variability averaged 10 %. Prolactin (PRL) was measured according to the radioimmunoassay of Kann (1971); assay sensitivity was 0.3 ng/ml. The results are expressed in ng of PRL PS 7 NIH/ml of plasma. Intra and inter-assay variation coefficients were 5.3 and 8.4 %, respectively. Electrolyte (sodium and potassium) concentrations in the serum were determined using an emission spectrometer (Guéguen, Nutrition, Jouy-en-Josas).

**Statistical analysis.** — The mean was always expressed with the standard deviation (M ± SD). The paired t-test was used to compare mean sun values with mean shade values. The correlation coefficient (simple linear regression) was tested by the t-test.

**Mathematical models.** — Black globe temperature and time had the strongest effects on RR, PRL and RT; in order to better quantify these effects, mathematical models were used on the three variables. These models are only descriptive, not explicative.

- At a given time, RR, PRL and RT depended linearly on Tg.
- At a given Tg, RR and PRL reached a maximum then tended towards a limit value when t → ∞. Maximal RT was reached as soon as t = 0.

A model, Log-normal (i.e., involving a Gaussian function of the Neper-log of time) on time and linear on Tg, was developed to show the simultaneous effects of these parameters on RR and PRL. For RT, which almost reached its absolute maximum immediately upon solar exposure, we used a model that decreased exponentially with time and was linear with Tg. The model parameters were adjusted to the experimental data using the least-squares method (Bachacou et al., 1981).

- The general equation of the model for RR and PRL was:

\[(X - A) = b \left( Tg - (A + c)/b \right) \left( 1 + B \exp \left( - (\text{Lnt} - C)^2/D \right) \right)\]

where t is time in days and Tg the black-globe temperature in °C; A, B, C, D, b and c are parameters.
The zero on the time coordinate corresponds to the instant that solar exposure began. The time-dependent term of the right-hand side of the equation tends to 1 when $t$ tends towards the positive infinite. The $X$ value tends towards $X = b Tg - c$. To avoid adjusting too many parameters by the least-squares method, $b$ and $c$ were determined by the linear regression on $Tg$ of data observed after stabilization.

This model allowed us to estimate:
- the time at which the maximum of $X$, at a given $Tg$ ($t_{\text{max}} = \exp (C)$), was reached;
- the time of return to equilibrium (i.e. relaxation time, arbitrarily defined as the time after which $(X - X_\infty)/(X_{\text{max}} - X_\infty) \leq 0.05$). This condition in the log-normal model amounted to $\exp (- (\ln t - C)^2/D) \leq 0.05$ and in the exponential model to $\exp (- t/C) \leq 0.05$;

The RT model was:

$$(X - A) = b (Tg - (A + c)/b). (1 + B \exp(-t/C));$$

$b$ and $c$ were determined as above. In this model relaxation time was $\exp (- t/c) \leq 0.05$.

**Results.**

1) *Mean microclimatic conditions.* — During the experiment the mean values of maximal and minimal temperatures, maximal and minimal relative humidity, mean daily global radiation and air velocity (measured at a meteorological station near the experimental site) were $29.05 \pm 0.77 \, ^\circ\text{C}$, $24.39 \pm 1.39 \, ^\circ\text{C}$, $100 \%$, $78.74 \pm 5.78 \, \%$, $18.89 \pm 4.99 \, \text{MJ/m}^2/\text{day}$ and $3.06 \pm 0.71 \, \text{m/sec}$, respectively. Mean rainfall during this period was $3.86 \pm 7.3 \, \text{mm/day}$. However, black-globe temperature was the main bioclimatic reference.

2) *Physiological parameters in shade.* — From D-4 up to and including the morning of DO, the mean values of the thermophysiological and endocrine parameters measured between 11.00 and 12.00 were: $RR: 35 \pm 3 \, \text{inspirations/min}$; $RT: 39 \pm 0.27 \, ^\circ\text{C}$; $CR: 81 \pm 3 \, \text{heartbeats/min}$; haematocrit: $33 \pm 2.3 \, \%$; plasma cortisol: $4.5 \pm 0.83 \, \text{ng/ml}$; plasma prolactin: $130 \pm 50 \, \text{ng/ml}$.

3) *Immediate physiological reactions.* — Figure 1 (DO) shows the thermophysiological and hormonal data in relation to $Tg$; in passing from shade to direct sunlight, the animals had a heat overload ($Tg$ increase of $7.8 \, ^\circ\text{C}$, 29.57 to 37.37) inducing an increase in the RT of $1.32 \, ^\circ\text{C}/\text{h}^{-1}$ between 11.40 and 12.40.

A simultaneous increase in RT ($40.84 \pm 0.23$ at 13.00 vs $39.48 \pm 0.19 \, ^\circ\text{C}$ at 11.40; $P < 0.001$), $RR$ ($98 \pm 22$ at 13.00 vs $35 \pm 3 \, \text{insp/min}$ at 11.40; $P < 0.01$) and cortisol ($17.7 \pm 8.61$ at 12.10 vs $5.57 \pm 1.67 \, \text{ng/ml}$ at 12.00; $P < 0.05$) was observed.

Before solar exposure, RT and RR began to increase due to circadian rhythm; this point will be discussed later.
There was no significant increase in PRL, and CR increased non-significantly from 85 ± 9 to 120 ± 20 heartbeats/min. Serum potassium rate tended to decrease one hour after insolation (256 ± 6 vs 223 ± 13 mg/I ; P < 0.02), whereas sodium rate (3.13 ± 0.07 g/I) remained steady ; between 1 and 5 hours after insolation, the haematocrit did not change significantly.

4) Mean and long-term physiological reactions. — Figure 2 shows the variation of the thermophysiological (RR and RT) and endocrine (prolactin) parameters compared to that of Tg from D-4 to D49. Generally speaking, PRL an RR varied according to Tg. On D1, PRL increase was significant (923 ± 653 vs 90 ± 22 ng/ml ; P < 0.05) (the first large standard deviation, due to high variability between animals, ranged from 369 to 1 832 ng/ml). From D1 to D45, the correlation coefficient between RT and PRL was r = 0.64 (P < 0.01) when globe temperature was higher than the threshold of 32 °C.

An absolute PRL maximum (1 035 ± 279 ng/ml) was obtained at D9 which then decreased to a steady level after about 20 days. At this time (i.e. from D20), the circulating PRL rate was significantly higher in the sun than in the shade (248 ± 109 vs 130 ± 50 ng/ml ; P < 0.05). This confirms our previous results which showed a higher prolactinemia in bucks kept outdoors permanently in the sun and fed a concentrate-poor diet than in those kept in a shelter and fed the same diet (Sergent et al., 1984). The PRL increase between D45 and D49 was not significant.

An absolute RR maximum was observed at D3 (266 ± 38 inspirations/min) which then decreased and stabilized at D15 (42 ± 4 not significantly different from 35 ± 3 inspirations/min for shaded animals), while RT returned to equilibrium at D24 (39.51 ± 0.32 not significantly different from shaded RT : 39.48 ± 0.19 °C).
Cortisol decreased up to D8 and recovered the level recorded in shaded animals (4.06 ± 0.51 and 4.63 ± 0.79 ng/ml).

Haematocrit decreased significantly (28.62 ± 0.51 vs 32.99 ± 2.29 %; P < 0.05) on D1, i.e. after a time-lag of 24 h, then increased gradually between D11 and D18, reaching a value of 32.12 ± 2.39 % on D66.

5) Models

Respiratory rhythm (fig. 3a). — The Tg-linear asymptotic equation calculated from the data obtained from D18 after solar exposure was RR (inspirations/min) = 7.14 Tg − 192 (n = 7; R² = 0.66).

The complete model was:

$$RR = 28.5 + 7.14 (Tg - 30.8) (1 + 8.67 \exp\left(-\left(Lnt - 1.22\right)^2 / 0.73\right)).$$

This accounts for $R^2 = 96$% of the total variance. The values of $T_{\text{max}}$ (corresponding to a maximal RR at a given Tg) and relaxation time (time for which $(RR - RR_{\infty})/(RR_{\text{max}} - RR_{\infty}) \leq 0.05$) occurred 3.4 and 15 days, respectively, after insolation. Figure 3a shows that the model and experimental data are in good agreement.

Prolactinemia. — Due to a longer relaxation time, the asymptotic equation, calculated with only 5 points (from D26), was:

$$PRL_{\infty} \text{ (ng/ml)} = 40.7 \ Tg - 1 \ 185 \ (n = 5; \ R^2 = 0.67).$$

The complete model was:

$$PRL \text{ (ng/ml)} = 1.4 + 40.7 \ (Tg - 29.2) \ (1 + 2.75 \exp\left(-\left(Lnt - 1.03\right)^2 / 2.10\right)).$$
The adjustment, though less satisfactory than for RR, was fairly good ($R^2 = 0.75$). $R^2$ decrease might be due either to the higher variability of prolactinemia or to less precise model simulation of the actual facts (see discussion).

As defined above, the values of $T_{\text{max}}$ and relaxation time were 2.8 and 35 days, respectively. Theoretically, the asymptotic value of PRL should be calculated on only 3 points (after 35 days). However, on D25, the value of $X - X_\infty$ represented only 10% of the maximum and calculation on 5 points did not lead to a large error.

Rectal temperature (fig. 3b). — The asymptotic equation was:

$$RT_\infty = 0.09032 \ T_g + 36.45 \ (n = 5 ; R^2 = 0.88).$$

The global model was:

$$RT = 38.98 + 0.09032 (T_g - 28.01) (1 + 1.007 \ exp (- t/8.048)).$$
This model accounted for only 59% of the variance; figure 3b shows that the adjustment was not very good. Maximal rectal temperature was reached as soon as the animals were exposed to the sun. The relaxation time calculated as previously was 24 days.

Discussion.

Sudden and prolonged exposure to solar radiation was found to have a marked effect on the respiratory rhythm, rectal temperature and prolactinemia of male Creole goats (fig. 2); these criteria increased together with Td, Tw, and mainly Tg.

Hyperprolactinemia has already been reported in cattle (Wetteman and Tucker, 1974, 1976; Hooley et al., 1979; Schams et al., 1980; Roman-Ponce et al., 1981; Tucker and Petitclerc, 1982; Berbigier et al., 1984), sheep (Schillo et al., 1978); Bocquier (personal communication) and goats (Chemineau and Ravault, 1984; Sergent et al., 1984; Sergent, 1985).

The hypothalamo-pituitary axis of bucks strictly habituated to shade (7 months of habituation) did not seem to be immediately activated by the stimulus of solar radiation since hyperprolactinemia was only detected after a time-lag of 24 h, even though in goats permanently habituated outside plasma prolactin immediately follows variations in insolation (Chemineau and Ravault, 1984; Sergent et al., 1984). Moreover, in our experimental conditions (fig. 1), the hourly pattern of circulating prolactin on DO was not disturbed by solar exposure and was not different from that observed at the same season in the similar bucks kept permanently in the shade (Sergent et al., 1985, in preparation).

One hypothesis is that the 24-hour time-lag might correspond to the time required for the prolactin secretory cells to become sensitive. The second hypothesis (which does not exclude the first one) is that a high plasma cortisol level at the moment of solar exposure (between 12.00 and 13.00) would prevent PRL response to solar stimulation (see Yelvington et al., 1984).

Rectal temperature increased immediately after insolation. This is in agreement with the rapid RT increase observed in animals subjected to heat stress in a climatic chamber (e.g. Halipre, 1973; Bouvier et al., 1974), i.e. in the beginning, the organism responds poorly to the sun since rectal temperature (heat storage) and respiratory rate (heat dissipation) rise simultaneously.

However, RT and RR began to increase before solar exposure (fig. 1, large arrow) due to the endogenous rhythm particular to homeotherms (inner temperature increase from 6.00 to 16.00). The maximal levels of these parameters are comparable with the ones reached using an experimental sun-shade design in similar animals. In that experiment, we observed circadian variations in RT and RR in each « climatic » treatment, i.e. in shade between 11.00 and 12.00, the RT rose $+ 0.11 \degree C$ (39.35 to 39.46), RR remained at $\approx 70$ insp/min for $Ta = 30.35 \degree C$ and $Tg = 32.6 \degree C$. In sunlight, RT increased $+ 0.12 \degree C$ (39.39 to 39.51) and RR $+ 15$ insp/min (131 to 146) for a $Ta$ rise of $+ 0.30 \degree C$ (31.15 to 31.45) and a $Tg$ rise of $+ 0.15 \degree C$ (36.85 to 37) (Sergent et al., 1985, in preparation).
In the present experiment, RT rose $+1.28 \, ^\circ C$ (39.12 at 11.00 in shade to 40.40 at 12.10 in the sun) and RR increased $+31 \, \text{insp/min}$ (33 to 64).

Therefore, the values of RT, RR (fig. 1) and PRL (fig. 2), obtained when the bucks were suddenly exposed to the sun, were several orders of magnitude greater than the values obtained during a normal circadian rhythm; these events are actually induced by sudden solar exposure.

The intensified respiratory rate (or panting) was probably a thermolytic emergency response to acute solar exposure; however, it was not fully developed at the beginning of heat stress.

The variation models of RR, PRL and RT, allowing us to better quantify the former observations, gave an order of magnitude of the combined effect of black globe temperature and time on these 3 variables by means of determination coefficients ($R^2 = 0.96$, 0.75 and 0.59, respectively; $T_g$ and $t$ accounted for 96% of the variance in respiratory rhythm). These models showed where the maxima of RR and PRL were located in time after insolation (3.4 and 2.8 days, respectively, corrected to 3-days considering measurement frequency) and permitted quantification of the times of return to equilibrium of the variables (15, 35 and 24 days for RR, PRL and RT, respectively); adaptation to the new environment was complete after a maximum of one month. However, these models were chosen only on the basis of the curve shape of the 3 variables, according to black globe temperature and time, and because of some simple, easily verifiable hypotheses. Some results are apparently not valid: the model used for prolactinemia supposes an increase with $T_g$ from $D_0$; in fact, prolactinemia did not react to $T_g$ on the first day of sun exposure (see fig. 1), or at least it was doubtful. Taking into account the scattered RT values, it is not possible to say that the exponential model is better than any other curvilinear one. However, these models seem efficient, as shown by the high values of the determination coefficients.

During the first 3 days after solar exposure, the major part of the extra heat seemed to be eliminated by respiratory evaporation. Then, as this thermolytic route declined, sweating (skin transpiration) became the main thermolytic reaction, but we did not measure it. This observation may be related to the fact that serum potassium rate already tended to decrease 1 h after insolation. It has been well established that potassium is the main electrolyte eliminated in cattle during sweating (Macfarlane, 1976; El Nouty et al., 1980).

The last thermolytic mechanism might be a reduction of metabolism (decrease in heat production) accompanied by a slowing-down of the glucocorticoid and thyroid functions. This could not be affirmed by our experiment on the basis of only the cortisolemia since cortisolemia recovered its initial level (shade) after 8 days of insolation. Moreover, the average daily body gain after heat exposure was not significantly different from that recorded during the last two months in shade (15.05 ± 10 vs 7.71 ± 4.5 g). Cortisol, generally considered as a catabolic hormone (Thomas and Rodway, 1983) and negatively correlated with growth in cattle (Purchas et al., 1971), did not seem to be involved here. This is in agreement with our previous results, i.e. the plasma...
cortisol level was not significantly different whether the animals were kept permanently under a shelter or outside (Sergent et al., 1984).

Variations in plasma prolactin levels are associated with temperature variations, especially under direct natural insolation. Prolactin reactivity does not seem to be a general (aspecific) stress response but is specific to solar radiation, at least above a $T_g$ threshold of 32 °C. In addition, significant correlations between RT and PRL show that PRL secretion could be involved in thermoregulation.

These results and the thermoregulation failure observed in animals treated with an inhibitor of prolactin secretion at the hottest hours of the day (Berbigier, Sergent, and Ravault, 1985) support the putative role of prolactin in caprine thermoregulation. However, other experiments must be carried out to demonstrate a perfect causal relationship between prolactin and thermoregulation.

Conclusion.

When male Creole goats, previously acclimatized to shade, were suddenly exposed to the sun over a period of time in natural conditions, the following observations were made:

1) plasma cortisol level rose immediately but returned to shade values after one week;
2) rectal temperature rose immediately then decreased and stabilized at its initial level about 3 weeks later;
3) blood potassium rate tended to decrease 1 h after insolation, but the blood sodium rate remained steady;
4) haematocrit decreased on D1 (24 h after solar exposure began), but returned to initial level after 3 weeks;
5) respiratory rhythm increased gradually but returned to shade equilibrium about 2 weeks later;
6) there was no specific solar-exposure increase in PRL on D0; PRL rose dramatically on D1 and stabilized after about 3 weeks at a significantly higher level than the shade level;
7) RR and PRL decreased Log-normally on time (t) and linearly on black-globe temperature ($T_g$), while RT decreased exponentially with t and linearly with $T_g$;
8) body weight gain was not affected.

It is concluded that habituation to direct solar radiation produces high plasma prolactin levels and that, even in tropical genotypes, thermolytic mechanisms are not immediately available, i.e. an adaptation period of about 3 weeks is required.

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Résumé. Effet d'une insolation brutale sur les paramètres thermophysiologiques, la cortisolémie et la prolactinémie de boucs Créoles.

La température rectale (TR), le rythme respiratoire (RR), les concentrations plasmatiques de cortisol et de prolactine (PRL) et l'hématocrite sont mesurés à midi chez des boucs lors de leur accoutumance à l'ombre, au cours de leur exposition soudaine au soleil, puis lors de leur maintien en plein air. Simultanément sont relevées les données microclimatiques notamment la température de la boule noire (Tb).

Le jour de la mise au soleil (Jo), l'augmentation de TR et de RR caractéristique du rythme circadien est amplifiée sitôt l'exposition (TR : 40,84 ± 0,23 °C vs 39,48 ± 0,19 °C, P < 0,001 ; RR : 98 ± 22 vs 35 ± 3 resp/mn, P < 0,01). Le cortisol plasmatique augmente aussi (17,7 ± 8,6 vs 6,57 ± 1,7 ng/ml, P < 0,05). Au bout de 24 h (J1), la concentration plasmatique de prolactine augmente démesurément (923 ± 653 vs 90 ± 22 ng/ml, P < 0,05) et l'hématocrite décroît (28,6 ± 2,3 vs 33 ± 2,3 %, P < 0,05).

Pour rendre compte des effets simultanés du temps (t) et de Tb, sont développés un modèle Log-normal en fonction du temps et linéaire en fonction de Tb pour RR et PRL, ainsi qu'un modèle exponentiel décroissant en fonction de t et linéaire en fonction de Tb pour TR : les coefficients de détermination sont respectivement $R^2 = 0.96$, 0.75 et 0.59.

Environ 3 semaines plus tard (« période d'adaptation ») TR, RR et la PRL plasmatique se stabilisent : TR et RR retrouvent leurs valeurs à l'ombre, tandis que la PRL se maintient à un niveau supérieur à celui qu'elle avait à l'ombre (248 ± 109 vs 130 ± 50 ng/ml, P < 0,05).

Le délai de 24 h dans le déclenchement de l'hypersécrétion prolactinique et le rôle de la prolactine dans la thermorégulation sont discutés.

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