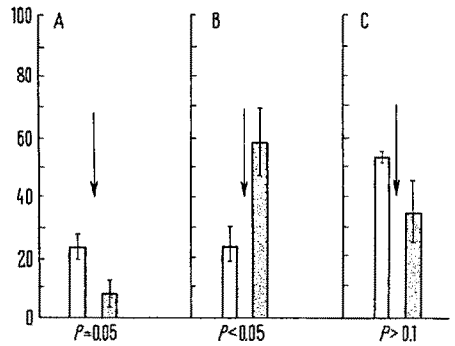
with control mean value (23.5 ± 6.6%) was dominant in all animals. Highly expressed desynchronization followed each injection of adrenaline and desynchronizing periods were present through the 6 h recuperative recording period. Their intensity and duration varied, although in 4 out of 6 animals they were most vividly expressed and longest at the beginning of the recording immediately after adrenaline injection. The other animals showed long waking periods in the middle and in the second half of the 6 h recording time. Slow wave sleep was also affected by the action of adrenaline, but not significantly (53.5 ± 1.6% in control experiments and 34.0 ± 9.4% after adrenaline). Periods of the slow sleep were present during the whole time of the recording, except immediately after intraventricular injection of adrenaline when desynchronization was dominant.



The action of adrenaline on different states of sleep in cats. Ordinate: columns in (A), paradoxical sleep in % of total time; columns in (B), wakefulness in % of total time; columns in (C), slow wave sleep in % of total time. Unfilled columns represent the mean values of 6 control experiments with its S.E.M. Columns with dots represent the mean values of 6 experiments with its S.E.M. after intraventricular injections of adrenaline. The arrow in (A), (B) and (C) denotes 10 days rest.

The dissociation of adrenaline activity exerted upon the duration of slow wave and paradoxical sleep contributes to the research findings which have shown that the sleep is not a homogenous state of behaviour. This dissociation would speak in favour of the dissociation of the origin of the 2 states of sleep where the drug target action site would be the brain stem structures underlying the mechanisms of vigilance and paradoxical sleep[5].

Résumé. On a étudié l'action de l'adrénaline sur les différents états de sommeil chez le chat. Pendant les premières 6 h de récupération après la privation de phase paradoxal du sommeil, l'injection intraventriculaire d'adrenaline (0.2–2.0 mg) diminue la phase paradoxale du sommeil et prolonge l'état de veille.

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2,4-Dinitrophenol Inhibition of P^{32} Release from Human Red Cells

We have previously reported[1] that the release of radioactive sulfate from labeled human erythrocytes can be inhibited by certain agents which uncouple oxidative phosphorylation in liver mitochondria, such as 2,4-dinitrophenol (DNP). This observation interests us because further investigation may reveal the nature of metabolic events at the membrane that may affect transport or permeability to various solutes. This report will show that $5 \times 10^{-4} M$ DNP also inhibits the release of radioactive phosphate (P^{32}) from human red cells and that the effect is more clearly observed when medium P_i concentration is elevated.

Fresh blood was collected in the usual mixture of acid, citrate, and dextrose (used in blood storage) and centrifuged at 800 *g* and 5 °C for 5 min. After removal of the plasma and buffy coat, the cells were washed 4 times with a modified Ringer-Locke's medium. This salt solution contained no calcium and was buffered with *Tris*(hydroxymethyl)aminomethane adjusted to pH 7.4 at 37 °C. The washed cells were suspended in an equal volume of this medium and labeled by incubating the suspension for 2 h at 37 °C in a Dubnoff shaking incubator, in the presence of 0.5 μC of P_i^{32}/ml of medium and 1 mM P_i. The labeled cells were washed twice and resuspended with sufficient non-radioactive medium to yield a hematocrit of 20%.

For the study of P^{32} release, 3.5 ml samples of the labeled blood suspension were placed in 25 ml Erlenmeyer flasks. These samples were removed after 10 and 130 min of incubation and aliquots of the suspension and of the medium were plated on concentrically-ringed aluminum planchets and air-dried. The radioactivity was counted with an end-window Geiger-Mueller counter and the results were expressed as % P^{32} released in 2 h. There was no significant change noted in the pH and hematocrit. When medium P_i levels were varied, appropriate amounts of NaCl were withheld to maintain iso-osmolality.

It may be noted in the Figure that the influence of DNP seems to be virtually absent when the experiment is conducted in a P_i-free medium. At a medium P_i concentration which corresponds to the usual plasma level (1 mM), a clear difference is noted with $10^{-3} M$ DNP but the change with $5 \times 10^{-4} M$ DNP is relatively small. At higher external P_i concentrations, there seems to be no doubt that both DNP levels inhibit P^{32} release. On the other hand, $10^{-4} M$ DNP has no noticeable effect at any medium P_i concentration.

We have noted before that augmentation of the medium P_i level results in an increase in P^{32} release[2]. This effect appears to be best explained by an internal metabolic

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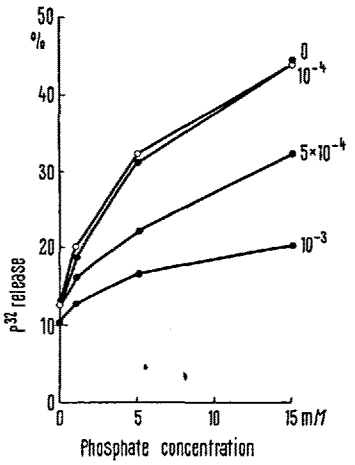
interaction. That is, as cellular P_i is elevated, the P^{32} that is being released continuously from organic phosphate is not re-esterified as rapidly because of the increased probability that non-radioactive P_i would enter into the esterification reactions. This leads to greater P^{32} availability for release. Cellular P^{32} has been found to be increased as a function of cellular P_i under these conditions[3]. Moreover, there appeared to be no change in anion permeability in these studies[3].



DNP inhibition of P^{32} release from human red cells as a function of medium phosphate concentration.

The results of this study indicate that DNP does inhibit P^{32} release and that this effect can be greatly magnified by the simple expedient of suspending the labeled cells in high P_i media. Both sulfate[1] and phosphate release from human red cells thus appear to be inhibited by the same concentration of DNP, indicating that the mechanism involved may be the same for the 2 anions. Current thought[4,5] appears to favor a passive mechanism such as simple diffusion or an equilibrating carrier mechanism with a high K_m[6].

Zusammenfassung. Inkubiert man P^{32}-vorbeladene menschliche Erythrozyten unter Zusatz von $5 \times 10^{-4} M$ Dinitrophenol, so lässt sich eine Reduktion der Phosphatabgabe feststellen. Mit Erhöhung des extrazellulären Orthophosphatgehalts kann diese Wirkung besser gesehen werden.

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Superior Cervical Ganglionectomy in the Japanese Quail

The ability of light to act as a 'Zeitgeber' (synchronizer) for the circadian rhythm of ovulation-oviposition in birds is well known[1-3] and also its role as a stimulator for the growth and development of avian gonadal tissue[4-6]. In recent years, a great deal of attention has been focused on the function of the pineal gland as a possible participant in light-induced neural mechanisms. The mass[7,8], morphology[9], serotonin content[10], melotonin content[11,12], and HIOMT[13-15] can be altered by varying the amount of light to which the animal is exposed. In the rat, constant exposure to light decreases the weight of the pineal gland, reduces the size of the pinealocyte and its nucleoli, and the level of cytoplasmic basophilia. The content of serotonin is nine times greater during the light phase than during the dark phase, and HIOMT is inhibited by light.

In chickens diurnal or constant light causes an increase in the size of the pineal gland as well as in the HIOMT activity of the pineal[15]. Pinealectomy in the chicken caused testicular atrophy in the coung cockerel[16] but testicular hypertrophy in older cockerels[17]. Pinealectomy in the Japanese quail permitted rapid oviducal growth in the female exposed to diurnal photoperiods but was without effect in quail kept under non-stimulatory photoperiods. Melatonin implantation in low concentrations inhibited gonadal growth, whereas higher concentrations changed the time of lay but did not interfere with ovarian activity[18].

Since it had been shown in the rat that light influenced the pineal by way of the superior cervical ganglion[19], it

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