



Duration of different parts of the sleep cycle in rats without (C) and with stimulation (ST) of the caudate nuclei. White columns: duration of telencephalic sleep phase in sec (measured from the end of one rhombencephalic sleep phase to the beginning of the following one); hatched columns: rhombencephalic sleep phase (measured with respect to the onset and termination of the regular theta activity in the hippocampi); black columns: arousal reaction following immediately after rhombencephalic sleep. Average values with S.E. of the mean; n: number of measurements in the group.

starts with TP, which is followed by RP. The gradual desactivation of the RAS during TP seems to be one of the necessary conditions for the onset of RP. The intrinsic process of reticular desactivation might be influenced to some extent by stimulation of the caudate nuclei. This stimulation can influence the excitability of the brain without visible changes in the sleeping EEG (the caudate spindles recorded at the cortex disappear at the beginning of stimulation).

Zusammenfassung. Die telencephalische Schlafphase der Ratte kann bei lang dauernder elektrischer Nucleus-caudatus-Reizung deutlich verkürzt werden.

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Variability Within and Between Strains for Mating Behaviour Parameters in *Drosophila pseudoobscura*

SPIESS and LANGER¹ demonstrated variable mating speeds for homokaryotypes in *D. pseudoobscura*, and KAUL and PARSONS² confirmed this for Standard (ST) and Chiricahua (CH) homokaryotypes. Both experiments showed ST homokaryotypes to have a faster mating speed than CH, in spite of differences in experimental technique. KAUL and PARSONS' results were based on single pair matings, which made it easy to obtain data for duration of copulation also. The strains used by KAUL and PARSONS were derived by intercrossing 3 ST and 3 CH strains collected at Mather, California by Prof. TH. DOBZHANSKY in 1959. In view of the evidence of EHRMAN³, who showed the development of sexual isolation between 6 population cages of *D. pseudoobscura* set up from the same initial population, after a period of 4 years and 5 months of isolation, it seems reasonable to enquire into the possibility of behavioural differences in the original 3 ST and 3 CH strains.

The experimental procedure consisted of 50 single pair matings for each of the 6 strains repeated for 3 trials, so giving a total of 150 observations per strain. In Table Ia, the number of matings out of 50 in 5 min with totals and mean mating speeds are given for each strain. It is clear that there is substantial variability between strains but little between trials, as is confirmed by an analysis of variance (Table Ib) on the proportions mated out of 50 in 5 min after applying the angular transformation^{2,4}. This means that observations published previously^{1,2} probably represent more or less the average situation, depending on how many strains were used to set up the experimental cultures. It is interesting, too, that although ST has been reported to have a greater mating speed than CH^{1,2}, one of the ST strains has the slowest mating speed of all.

Table Ia. Numbers mating in 5 min out of 50 for ST and CH strains, with mean mating speeds calculated as in KAUL and PARSONS²

Strain	ST-1	ST-2	ST-3	CH-2	CH-5	CH-6
Trial I	9	37	38	10	32	31
II	8	28	15	15	27	24
III	6	42	28	10	15	27
Total	23	107	81	35	74	82
Mean mating speed (min)	110	2.4	4.0	17	5.1	4.0

Table Ib. Analysis of variance for ST and CH strains

Karyotype	ST			CH	
	Degree of freedom	Mean square	Variance ratio	Mean square	Variance ratio
Strains	2	968.3	14.42 ^b	307.4	7.45 ^a
Trials	2	143.7	2.14	52.9	1.28
Error	4	67.2		41.3	

^a $P < 0.05$. ^b $P < 0.01$.

¹ E. B. SPIESS and B. LANGER, Proc. natn Acad. Sci., USA 51, 1015 (1964).

² D. KAUL and P. A. PARSONS, Heredity 20, 381 (1965).

³ L. EHRMAN, Genet. Res. 5, 150 (1964).

⁴ P. A. PARSONS, Genetica 35, 141 (1964).

Table IIa gives mean durations of copulation for ST based on 15 observations, since the minimum number of observations was 15 for given strain and trial. For CH the means are based on 34 observations. Compared with the mating speed there is far less variability between strains,

Table IIa. Mean durations of copulation (min) for the ST and CH strains

Strain	ST-1 ^a	ST-2 ^a	ST-3 ^a	CH-1 ^b	CH-5 ^b	CH-6 ^b
Trial I	5	4.9	6.5	3.7	4.4	4.2
II	6.7	4.5	5.3	3.7	3.0	4.0
III	5.4	5.3	6.0	2.9	3.4	3.5
	5.7	4.9	5.9	3.4	3.6	3.9

^a Means based on 15 observations. ^b Means based on 34 observations.

Table IIb. Analysis of variance for ST and CH strains

Karyotype	ST			CH		
	Degree of freedom	Mean square	Variance ratio	Degree of freedom	Mean square	Variance ratio
Strains	2	12.80	3.98 ^a	2	6.02	5.60 ^a
Trials	2	0.10	0.03	2	18.64	17.34 ^c
Trials						
× strains	4	9.93	3.08	4	6.07	5.65 ^b
Error	126	3.22		297	1.08	

^a $P < 0.05$. ^b $P < 0.01$. ^c $P < 0.001$.

and in fact there is more variability between trials than between strains for CH (Table IIb gives the analyses of variance). Furthermore, all the CH durations are less than for ST, as reported previously².

Thus for a given karyotype, strains set up from the same locality may show extreme variability for one component of mating behaviour, namely mating speed, but less for another, the duration of copulation. For a given karyotype, therefore, there seems to be more genetic variance controlling mating speed than duration of copulation, as has been shown in biometric experiments in *D. melanogaster*^{4,5}. This is perhaps reasonable, since in the transmission of genes from one generation to the next, the time taken in courtship will clearly be far more significant than the actual time spent copulating. The time taken in courtship will need to be extremely flexible to adapt rapidly to new situations as they arise. Thus differences found between karyotypes for mating speed must be interpreted with more caution than differences for duration of copulation.

Résumé. Chez un caryotype donné de *Drosophila pseudoobscura* on constate dans quelques lignées plus de variabilité dans la durée du comportement sexuel précédant l'accouplement que dans la durée de celui-ci.

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Department of Genetics, University of Melbourne, Victoria (Australia), June 13, 1966.

⁵ I. T. MACBEAN and P. A. PARSONS, *Experientia* 22, 101 (1966).

⁶ Supported by the Commonwealth of Australia Scholarship and Fellowship Plan.

The Effects of a Substituted Sulphamoyl Diuretic 'Cloпамide' (DT 327) on Insulin Release in vitro

Numerous reports have appeared indicating that thiazide diuretics may exert a diabetogenic effect in man¹⁻⁴. Particular attention has been directed towards the benzothiadiazine derivative diazoxide, which having no diuretic activity is diabetogenic in man when given together with small doses of benzothiadiazine diuretic³ and lowers the blood insulin concentration⁵. Evidence showed that the most likely explanation was an inhibition of insulin release⁶, confirmed by the findings of a direct effect on the insulin release from an in vitro preparation of mammalian pancreas⁷. These findings do not preclude other actions either on insulin metabolism once it is released or other alterations in carbohydrate metabolism.

Preliminary clinical trials of a non-thiazide substituted sulphamoyl diuretic 4-chloro-N-(2,6-dimethyl piperidino)-3-sulphamoyl benzamide suggested that rather than a hyperglycaemic effect, after several days on this compound, there was a progressive lowering of the 2 h post-prandial blood glucose concentration in a series of patients with oedema of cardiac and hepatic origin. It was thought worthwhile to examine the effects of this drug on the in

vitro release of insulin from mammalian pancreas using different concentrations of glucose and diuretic.

Method. Slices of rabbit pancreas were prepared and incubated in varying concentrations of glucose and diuretic using the same method as that described by HOWELL and TAYLOR⁷, insulin release being estimated by immunoassay.

The Table shows no significant effect on the rate of insulin secretion at 3 different diuretic concentrations with 2 different glucose concentrations.

Discussion. No clear hyper- or hypoglycaemic effect has been demonstrated in these in vitro studies. Further clinical trials of this effective diuretic with estimation of serum insulin concentrations are needed to explain the

¹ B. CURCHOD, *Diabète* 8, 201 (1960).

² J. M. FERGUSON, *Am. J. Cardiol.* 7, 568 (1961).

³ C. T. DOLLERY, B. L. PENTECOST, and N. A. SAMAN, *Lancet* 2, 735 (1962).

⁴ W. R. WILSON and R. OKUM, *Clin. Res.* 10, 184 (1962).

⁵ H. S. SELTZER and E. W. ALLEN, *Diabetes* 14, 439 (1965).

⁶ H. FRERICH and W. CREUTZFELDT, *Diabetologia* 1, 80 (1965).

⁷ S. L. HOWELL and K. W. TAYLOR, *Lancet* 7, 128 (1966).