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, 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 previously[2].

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] $P < 0.001$.

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.

P. A. PARSONS and D. KAUL[6]

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 'Clopamide' (DT 327) on Insulin Release in vitro

Numerous reports have appeared indicating that thiazide diuretics may exert a diabetogenic effect in man[1–4]. 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 diuretic[3] and lowers the blood insulin concentration[5]. Evidence showed that the most likely explanation was an inhibition of insulin release[6], confirmed by the findings of a direct effect on the insulin release from an in vitro preparation of mammalian pancreas[7]. 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 postprandial 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 TAYLOR[7], 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. DOLLERFY, B. L. PENTECOST, and N. A. SAMAAN, 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. FRERICHS and W. CREUTZFELDT, Diabetologia *1*, 80 (1965).
[7] S. L. HOWELL and K. W. TAYLOR, Lancet *1*, 128 (1966).

possible hypoglycaemic effect observed in our preliminary trial[8]. Nevertheless, it may be that our choice of subjects was not critical, and that if diabetic patients or those with vascular disease and obesity are preferentially selected a more significant answer will be obtained as to the effect of this diuretic on already altered carbohydrate tolerance as suggested in a study[9] of thiazide diuretics[10].

Effects of insulin release of varying concentrations of diuretic at 2 glucose concentrations

Final concentration of DT 327	Media containing 60 mgm% glucose	Media with 60 mgm% glucose + DT	Media containing 300 mgm% glucose 130	Media with 300 mgm% glucose + DT
20 µg/ml	46	47	130	138
100 µg/ml	51	65	277	287
200 µg/ml	48	45	115	–

There is no significant effect in the rate of insulin secretion from rabbit pancreas at either high or low concentrations.

Résumé. Un diurétique sulphamyl Brinaldix (Clopamide DT327) fut expérimenté pour savoir si on pouvait reproduire in vitro une hypoglycémie observée après injection. L'expérience fut effectuée avec des cellules isolées d'ilots et un radio-immunoessai pour mesurer la libération d'insuline. Différentes concentrations de glucose et de diurétique furent utilisées et ni inhibition ni stimulation ne furent observées dans la libération d'insuline.

V. Parsons

Department of Medicine, King's College Hospital, London S.E.5 (England), August 3, 1966.

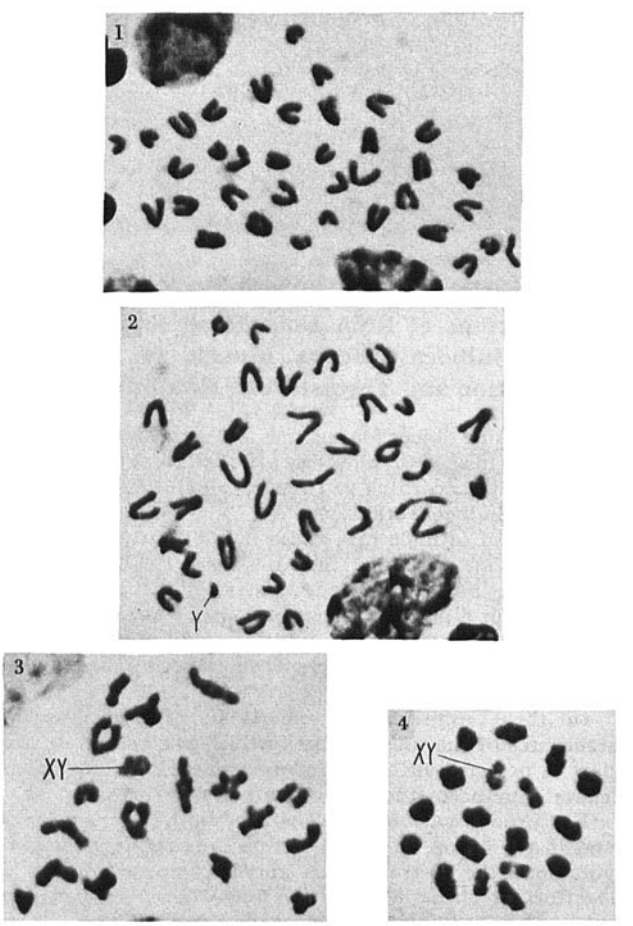
[8] V. Parsons and R. K. Price, Practitioner *195*, 648 (1965).
[9] Fr. W. Wolff, R. G. Langdon, B. H. Rueoner, Ch. Hollander, and R. D. Skoglund, Diabetes *12*, 355 (1963).
[10] Thanks are due to Mr. S. L. Howell for assistance in conducting the experiments, to Dr. K. W. Taylor for supervision and to Prof. John Anderson in whose department this investigation was carried out. Dr. D. Freestone of Sandoz Ltd. kindly supplied the Clopamide used in this study and I am indebted to the Research Funds of King's College Hospital for secretarial assistance carried out by Mrs. M. Banks.

Cytogénétique des *Leggada*: (1) La formule chromosomique de *Mus* (*Leggada*) *bufo* Th., (2) Nouvelles données sur la délétion portant sur le bras court d'un *X* chez *Mus* (*Leggada*) *triton* Th.

Le polymorphisme chromosomique des *Mus* africains du sous-genre *Leggada* confère à ce groupe de Souris un intérêt exceptionnel, en ce qui concerne les rapports existant entre mutations chromosomiques et processus de spéciation[1]. Grâce à une invitation du Dr. U. Rahm, Directeur de l'IRSAC (Lwiro, près Bukavu, République démocratique du Congo), j'ai pu, au cours d'un séjour d'un mois, récolter 35 *Leggada* et confectionner, pour chaque individu, des préparations de rate et de gonade.

(1) Parmi mes captures, figurent 5 *M. bufo*, espèce qui, selon Ellerman[2], doit être rattachée au groupe *bufo-triton*. La détermination sub-spécifique de mes sujets (4 ♂♂, 1 ♀) lesquels ont été examinés par le Dr. F. Petter (Musée national d'Histoire naturelle, Paris) et par le Dr. X. Misonne (Institut royal des Sciences naturelles de Belgique) demeure en suspens: ils différeraient quelque peu de la forme typique décrite du Ruwenzori, massif situé à environ 300 km au nord de Lwiro.

Le complément chromosomique de *M. bufo* est celui que j'ai qualifié de primitif et qui caractérise plusieurs espèces ou sous-espèces de *Leggada* (*minutoides ssp*$_1$, *tenellus*, *indutus*, *setulosus*): $2N = 36$, chromosomes sexuels *PR*, $N.F. = 36$. Autosomes et hétérochromosomes sont donc acrocentriques, l'*X* et l'*Y* étant semblables à ceux de la Souris domestique (Figures 1–4). Notons que les 5 exemplaires de *bufo* ont été capturés en compagnie de nombreuses *minutoides ssp*$_1$ présentant la même formule chromosomique, *bufo* étant beaucoup plus rare.



Figs. 1–4. *Mus bufo*. (1) Femelle: division diploïde dans la rate. (2) Mâle: division diploïde dans la rate. (3) Mâle: diacinèse. (4) Mâle: métaphase I. × 1800.

[1] R. Matthey, Experientia *20*, 657 (1964).
[2] J. R. Ellerman, *The Families and Genera of Living Rodents* (Trust. Brit. Mus., London 1940–1941).