Le calcul du rapport:

$$\frac{G^T - G^{DDT}}{G^T} \times 100$$

où G^T correspond au nombre moyen de gonocytes chez l'embryon témoin et G^{DDT} au nombre moyen chez l'embryon traité au DDT, donne le pourcentage de stérilité des embryons issus d'œufs traités au DDT. Celui-ci est de 60% à la concentration de $2^0/_{00}$, de 61% à $3^0/_{00}$, de 63% à $5^0/_{00}$ et de 76% à $10^0/_{00}$.

Il existe une grande disproportion entre le nombre de cellules germinales contenues dans la gonade gauche et celui de la gonade droite. Et ceci, aussi bien chez les témoins que chez les traités. Ainsi, le nombre moyen de gonocytes de la gonade droite témoin est d'environ 200 (pour 1200 dans la gonade gauche); après traitement au DDT, on ne dénombre plus que 10 à 50 cellules germinales dans la glande droite de la majorité des embryons.

L'examen histologique révèle la présence de gonocytes hypertrophiés, en dégénérescence. Ces cellules pycnotiques, en très faible nombre chez les embryons témoins (0,2% environ), sont plus fréquentes chez les embryons traités. Elles représentent 2,5% du nombre total des gonocytes à la concentration de $2^0/_{00}$, 3% à 3 et $5^0/_{00}$, 3,5% à $10^0/_{00}$.

Discussion et conclusions. Le DDT, employé sous l'une de ses formes commerciales, provoque une baisse importante de la population germinale des gonades des embryons de Poulet de 6 jours. Cette diminution est déjà forte aux faibles concentrations. Ainsi, le DDT à $2^0/_{00}$ est responsable d'une chute de 60% du nombre des cellules germinales. Aux concentrations de 3 et $5^0/_{00}$, le taux de stérilité des gonades s'élève encore par rapport au résultat précédent, mais dans de faibles proportions (60 et 63%). Ainsi, le DDT employé aux concentrations préconisées par le fabricant provoque une stérilisation importante quoique partielle des embryons de Poulet. La concentration nettement plus forte de $10^0/_{00}$ n'est que légèrement plus létale que les précédentes, mais elle accentue encore la chute de la population gonocytaire (76%).

Si l'on compare la taille des gonades des deux types d'embryons, on constate que dans la plupart des cas celle des embryons traités au DDT est inférieure. Parfois, les gonocytes ne sont pas tous localisés dans la gonade, ils forment de petits amas dispersés dans le mésentère, certains sont en dégénérescence. Dans d'autres cas, les gonades sont au contraire de taille sensiblement normale mais pauvres en cellules germinales.

Cette chute du nombre des gonocytes mise en évidence au 6e jour de l'incubation, suggère plusieurs hypothèses: 1. Le DDT provoquerait l'hypertrophie puis la pycnose progressive des gonocytes déjà en place dans les gonades. 2. Cette chute serait la conséquence d'une dégénérescence précoce du matériel germinal, antérieure au stade de sa migration. 3. Enfin, le DDT s'opposerait à une migration normale, en inhibant partiellement le pouvoir attractif des crêtes génitales, ou bien en atténuant la capacité amiboïde des cellules germinales primordiales.

Un comptage des gonocytes dans le croissant germinal et à des stades intermédiaires de leur migration, devrait nous permettre de résoudre ce problème.

Summary. A brief immersion of chick eggs in a DDT aqueous solution, before the incubation, provokes a strong reduction of the number of germ cells in the gonads, when the embryos are 6 days old. Some hypertrophied gonocytes are pycnotic. Hypotheses are proposed in order to explain the mechanism of DDT action.

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Inhibition of Post-Decapitation Convulsions by Reserpine

Decapitation of rats in the cervical region, but not at lower spinal segments, is regularly followed by generalized clonic convulsions. Previous treatment with high reserpine doses prevents almost completely the post-decapitation seizures.

Methods. 386 male Wistar rats (medium weight 200 g) were sacrificed by rapid transsection of the mid cervical spine with well sharpened scissors. Control group: untreated animals. Test groups: 1. a) Reserpine 5 mg/kg i.p., b) Reserpine 5 mg/kg on 2 sucessive days, i.p. Sacrifice in a) and b) 24 h after last i.p. injection, c) Reserpine 10 mg/kg i.p., 30 min before sacrifice. d) 2 days treatment with reserpine (like in b). 30 min before sacrifice, 0.5 mg/kg of noradrenaline i.p. e) I.p. injection of dibenzyline 10 mg/kg; propranolol 10 mg/kg, pentobarbital sodium 40 mg/kg, choralhydrate 300 mg/kg, respectively, 30 min before sacrifice. 2. Determination of the LD_{50} of convulsive drugs (Strychnine sulfate, Picrotoxin, Pentylenetetrazol in normal rats and after 2 days treatment with reserpine 5 mg/kg i.p.[1] 3. Neuromuscular transmission and direct muscle excitability was tested in the sciatic gastrocnemius preparation of 6 normal and 6 reserpinized rats (2 days treatment).

Results. The inhibition of post-decapitation convulsions by reserpine was complete after a 2 days' treatment and only partial after a 1 day treatment with 5 mg/kg a day. Refilling the reserpine depleted catecholamine stores with noradrenaline (0.5 mg/kg i.p.), 30 min before sacrifice did not abolish the anticonvulsive effect of reserpine (2 days treatment). The i.p. injection of high doses of reserpine or of the sympathetic blockers dibenzyline or propranolol, when given 30 min before sacrifice, did not reduce either intensity or duration of postdecapitation seizures. Pentobarbital sodium and chloralhydrate weakened but did not totally abolish agonal convulsions in the deeply anesthetized animals. (Table I). Drug-induced convulsions, by strychnine sulfate, picrotoxin, pentylenetetrazol, were not inhibited by previous reserpinization (2 days treatment). The LD_{50} of picrotoxin dropped to half its value, after reserpinization, but remained unchanged with strychnine and pentylenetetrazol. Only in the case of pentylenetetrazol did reserpine treatment prolong the duration of convulsions and postpone time of death from 7.0 ± 1.6 min to 30.3 ± 5.6 min. In addition, there was also a change in the type of convulsions: the incidence of clonic convulsions was greatly diminished and more strychnine-like extension seizures used to appear. The increase of pulmonary weight, produced by all these convulsant drugs, was normalized by reserpine treatment (Table II).

Discussion. Decapitation of rats in the cervical medulla triggers generalized convulsions lasting up to 30 sec.

[1] W. S. CARROL, Biometrics *8*, 249 (1952).

Table I. Incidence of post-decapitation convulsions in a) untreated rats; after treatment with: b) reserpine, c) noradrenaline, d) reserpine and noradrenaline

Treatment	No. of animals	Time of decapitation after last injection	Animals with convulsions (%)	Grade of convulsion	Pulmonary index
a) None (control)	22	–	100	+	0.845 ± 0.068
b) Reserpine (5 mg/kg i.p.)	12	24 h	33.3	±	0.927 ± 0.073
Reserpine (2 × 5 mg/kg i.p.) on 2 following days	22	24 h	0	0	0.826 ± 0.077
Reserpine (10 mg/kg i.p.)	12	30 min	100	+	0.896 ± 0.089
c) Noradrenaline (0.5 mg/kg i.p.)	5	30 min	100	+	0.811 ± 0.057
d) Reserpine (2 × 5 mg/kg i.p.) on 2 following days	5	24 h after last reserpine	0	0	1.403 ± 0.121
Noradrenaline (0.5 mg/kg i.p.)		30 min after noradrenaline injection			

+, full strength; ±, isolated, weak; 0, no convulsions.

Table II. LD_{50} and pulmonary index after i.p. injection of convulsant drugs (strychnine, picrotoxin, pentylene-tetrazol): a) normal rats; b) rats, after 2 days' treatment with 2 × 5 mg/kg of reserpine i.p.

Drug	No. of animals	LD_{50} (mg/kg)	*P*-value[a]	Pulmonary index[b]	*P*-value[a]
a) Strychnine sulfate	48	3.386 ± 0.036		1.218 ± 0.02	
b) Reserpine 2 × 5 mg/kg and Strychnine sulfate	48	4.878 ± 1.034	not significant	0.942 ± 0.01	< 0.01
a) Picrotoxin	48	6.317 ± 0.471		1.611 ± 0.07	
b) Reserpine 2 × 5 mg/kg and Picrotoxin	48	3.693 ± 0.785	< 0.01	0.870 ± 0.05	< 0.01
a) Pentylenetetrazol	48	52.100 ± 0.990		2.240 ± 0.07	
b) Reserpine 2 × 5 mg/kg and Pentylenetetrazol	48	51.100 ± 0.790	not significant	1.035 ± 0.05	< 0.01

[a] Comparing 'b' with the control 'a'; [b] in animals dying from drug poisoning. Pulmonary index = lung weight × 100 : body weight.

Transsection of the spine at lower levels produces an immediate flaccid paralysis. This proves that in the rat there must exist a 'convulsion centre' in the higher spinal cord which might be stimulated mechanically (decapitation) or chemically by the progressive anoxia and/or a secondary adrenaline release from the suprarenals. This 'convulsion centre' must be localized at a lower medullary segment than Landois's[3] 'Krampfzentrum' situated between medulla oblongata and pons.

Our experiments have further shown that the post-decapitation convulsions are not related to any adrenergic mechanism. Though these seizures are prevented by high reserpine doses, neither α- nor β-sympatholytics show any anticonvulsive activity. The inhibiting effect of reserpine is not reversed either by refilling the depleted catecholamine stores with noradrenaline. We therefore conclude that the anticonvulsive effect of reserpine must be due to a particular pharmacodynamic activity unrelated with its antiadrenergic properties. These findings are in contraposition to the observed lowering of electrical seizure threshhold by reserpine[2], and its facilatory effect on synaptic transmission in the spinal cord[4]. It also results from our experiments that the mechanisms responsible for the decapitation seizures must be different from those operative in drug-induced convulsions which are not prevented by pretreatment with reserpine.

Zusammenfassung. Vorbehandlung von Ratten mit Reserpin unterdrückt die nach Dekapitation eintretenden Konvulsionen. Der Mechanismus dieser Konvulsionen scheint von den durch Krampfgifte ausgelösten verschieden und von den antiadrenergenen Eigenschaften von Reserpin unabhängig zu sein.

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