

tities enclosed within cells. The bursting of these cells results in the large numbers of extracellular organisms.

This method of vaccine production should be undertaken only by immunes, since it is next to impossible to avoid eventual infection.

We do not believe that it will be possible to obtain the results described unless the Mexican strain is used. But since the Mexican vaccine gives a definite though imperfect immunity to the European virus, a sufficient cross immunization can be expected to justify the testing of this method against the European disease.

We have not been able to obtain results in guinea pigs analogous to those obtained in rats. Experiments with rabbits are being made, but the proper exposure to X-ray has not yet been ascertained. Rabbits may die within 24 hours of an exposure to X-ray which rats survive for a week.

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Influence of the Concentration of Iodoacetic Acid Upon Excitability and Chemical Changes in Muscle.

PAUL W. SMITH AND MAURICE B. VISSCHER.

From the Department of Physiology, University of Illinois.

The report of Lundsgaard that muscle from animals treated with iodoacetic acid gives large numbers of relatively normal contractions without perceptible lactate accumulation stimulated us to study the phenomenon in order to rule out several possible sources of error. It seemed possible that iodoacetic acid might accelerate the removal of lactate rather than prevent its formation, and that the poison served to diminish the irritability and the number of contractions rather than influence the lactic acid formation. In studying this problem an important relation between concentration of iodoacetic acid and the rate of lactic acid formation has appeared.

Iodoacetic acid was dissolved in Ringer's fluid, and injected, unneutralized, into the ventral lymph sac, in doses ranging from 1 part in 5,000, on the basis of body weight of the frog, to 1 part in 30,000. Similar concentrations were used in experiments in which iodoacetic acid was added to normal intact muscle or to muscle brei.

Numerous experiments upon the effect of changing the concentration of iodoacetic acid were made. At 1:5,000 lactic acid production is completely inhibited. At 1 to 30,000 there is a consider-

able production. The muscles poisoned at less concentration went into rigor early and were obviously not normal although there was lactic acid production. In order to analyze the effect of differing poison dosage, the fate of lactic acid in the post-contraction period was studied. After stimulation to fatigue by single shocks spaced one second apart normal muscle shows no "recovery" lactate production. The fact is well known. But we found that iodoacetic-poisoned muscle shows a very large "recovery" lactate accumulation at the low dosage, and none at high.

In doses larger than 1/30,000, no lactate accumulation occurred, regardless of the interval allowed. These observations show that small doses of iodoacetic acid, which do not inhibit completely the accumulation of lactate, decrease the velocity of its formation. Extending this line of reasoning, the effect of high dosage is to slow the process to the extent of a practical stoppage.

In view of the fact that iodoacetic acid eventually renders muscles non-irritable it was conceivable that the lack of lactic acid production in them might be related to their inability to respond to excitation. A crucial test of this possibility lies in the study of a condition such as chloroform rigor, which does not depend on natural irritability. Iodoacetic acid poisoned muscles are found to go into strong chloroform rigor without demonstrable production of lactate. It seems inescapable that non-irritability is not responsible for the failure of lactate production.

We were also interested in knowing whether or not iodoacetic acid might accelerate the removal of lactic acid rather than inhibit its formation.

TABLE I.
Influence of Concentration of Iodoacetic Acid upon Lactate Production.

Experiment		Iodoacetic Injected	Min. after Injection	Procedure	Lactic Acid Found %
27	A	1/5,000	37	resting	.019
	B			fatigued	.013
29	A	1/15,000	35	resting	.015
	B			fatigued	.019
34	A	1/20,000	30	resting	.028
	B			fatigued	.033
31	A	1/30,000	30	resting	.091
	B			fatigued	.195

TABLE II.
Influence of Iodoacetic Acid upon Rate of Lactate Production of Fatigued Muscle.

Experiment	Iodoacetic Injected	Min. after Injection	Lactic Acid Found %	
15	A	1/30,000	.054	Frozen immediately.
	B		.128	In N ₂ 20 min. after stim.
17	A	1/6,000	.018	Frozen immediately.
	B		.018	In N ₂ 20 min.
20	A	Control	.242	Frozen immediately.
	B		.256	In N ₂ 20 min.
21	A	Control	.210	Frozen immediately.
	B		.215	In N ₂ 20 min.

Muscle brei of thigh muscle taken from injected frogs uniformly showed complete inhibition of lactic acid formation after doses of 1/5,000, 1/6,000, or 1/15,000, but incomplete inhibition after doses of 1/30,000. The rate of lactic acid production in brei of muscle is slowed in proportion to the concentration of iodoacetic acid.

Summary. 1. There is conclusive evidence that iodoacetic acid slows the rate of lactic acid production in the anaerobic recovery period, and the slowing is in proportion to the concentration of the poison. 2. Iodoacetic acid does not accelerate removal of lactate under anaerobic conditions.

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Sexual Reactions of Certain Anurans after Anterior Lobe Implants.

HALCYON W. BARDEEN. (Introduced by M. F. Guyer.)

From the Department of Zoology, University of Wisconsin.

It is well known that homoplastic implants of the anterior lobe of the pituitary induce sexual precocity in many animals. The present investigation continues the work of Wolf,¹ on frogs. The same technique of injection is used. Since, by giving daily implants of anterior lobe of the pituitary to mature frogs that had ovulated normally in April, it was possible to induce a second ovulation as early as October, it seemed of interest to determine how soon a sec-

¹ Wolf, O. M., *Proc. Soc. Exp. Biol. and Med.*, 1929, **26**, 692.