this assignment. In phenol strong absorption occurs at 1.43 μ. This corresponds to the first O-H overtone. Since uracil and its derivatives do not absorb in this region but do absorb in the region characteristic of the first N-H overtone, it is inferred that these compounds exist predominantly in the keto form when dissolved in antimony trichloride.

The absorption at 1.65 μ shown by uracil, chlorouracil, and thymine is attributed to the first C-H overtone characteristic of ring compounds. The absorption is quite strong for phenol, which has five such hydrogen atoms, and quite weak for chlorouracil, which has only one C-H bond per molecule. The broadening of the C-H band toward the infrared in the case of thymine can be attributed to the aliphatic C-H overtone of the methyl group. The second dip in this broad band is centered at 1.75 μ, which coincides nicely with the methyl type C-H band of acetamide.

Uracil, chlorouracil, thymine, and acetamide show absorption at 1.99–2 00 μ; this band is absent in phenol. It is probably due to the second carbonyl overtone vibration which is known to lie in this region (*1*). All of the compounds except acetamide absorb at 2.13 μ. This band may be due to a combination of the fundamental valence and deformation vibrations of a ring C-H group. Beyond 2.1 μ the results are uncertain because the flint glass prism begins to absorb strongly and reduces the light intensity. Complete details of the spectra will be published elsewhere.

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Experimental Mixed Infection of Mice with Lansing Poliomyelitis Virus and Western Equine Encephalomyelitis Virus

Fred W. Gallagher

Department of Bacteriology and Clinical Pathology, College of Medicine, University of Vermont, Burlington

The phenomenon of interference between virus agents has been claiming increasing attention during the last decade. Interference has been demonstrated between plant viruses, between bacterial viruses (bacteriophages), and between animal viruses. In some cases when interference occurs the growth of one virus is inhibited, as evidenced by failure subsequently to demonstrate its presence (*1, 6*), while in other cases the growth of both viruses is inhibited (*3*). On the other hand, it has been shown that two viruses may grow together without apparent interference (*4, 5*).

In an attempt by the author to demonstrate interference between the viruses of equine encephalomyelitis and poliomyelitis in mice, it became apparent that such interference reported in monkeys (*2*) did not occur in mice. It was evident that both viruses grew in mice and that either or both could produce symptoms and death. If any antagonistic effect at all was manifested it consisted solely of a prolongation of life in some of the mice.

Lansing poliomyelitis and western equine encephalomyelitis viruses were chosen because their incubation periods and symptomatology made it easy to distinguish between them in mice. Because the Lansing virus has an incubation period averaging 7–9 days and the W.E.E. virus kills regularly in 48 hr, the W.E.E. virus was inoculated later so that both viruses could reach effective concentrations at about the same time. Accordingly, several preliminary experiments were run to work out the mutual relationships of dosage and time interval between administration of the viruses.

In one of these experiments using 30 mice it became apparent that LD_{50} doses of the two viruses would not give significant data without the use of extremely large groups of animals. It seemed desirable that all animals in the two control groups (Lansing virus alone and W.E.E. virus alone) should die in a reasonable period of time. Titration of the W.E.E. virus showed that 0.03 ml of a 1: 1000 dilution inoculated intracerebrally would kill all mice injected in 2–3 days. The Lansing virus, however, presented a more complex problem. A single intracerebral inoculation of the supernatant fluid (allowing suspension to stand for 10–15 min for settling of coarser tissue fragments) of a 10% suspension of mouse brain and cord never killed all of the mice injected, and even among those that did die the incubation period of some was prolonged to 2 or 3 weeks. However, killing of all mice could be achieved by two inoculations of virus spaced 48 hr apart. Further, this served to shorten the incubation period somewhat. The preliminary experiments showed also that a time interval of 3 days between the second inoculation of Lansing virus and the administration of W.E.E. virus would probably be optimal.

A group of 62 10-g white mice were used for the experiment here reported. Fifty-two of these mice received two intracerebral inoculations of Lansing virus spaced 48 hr apart. One mouse was killed by the inoculation procedure. On the morning of the third day following the second injection, 14 of these mice were either dead, moribund, or showed typical poliomyelitis paralysis. These were arbitrarily set aside to constitute a poliomyelitis virus control group. The remaining 37 Lansing mice, together with the ten uninoculated mice, were injected with W.E.E. virus. The ten W.E.E. mice all died with typical symptoms in 48 hr. Of the 37 mice receiving both viruses, 17 died in 2–17 days with typical poliomyelitis symptoms, 11 died in 2–3 days with typical W.E.E. symptoms, and eight died in 1–9 days with what were judged to be mixed symptoms. One mouse did not die.

The brains and spinal cords of five of the mice receiving both viruses and dying with typical poliomyelitis

symptoms were removed. The cord and brain stem were removed from each brain and the cords and brains pooled separately. These were ground, diluted to 1: 100 and 1: 1000 and injected into white mice, all of which died in 48 hr with typical W.E.E. symptoms. Similar results were obtained with brain and cord material removed from mice dying with W.E.E. symptoms and from mice dying with symptoms judged to be a mixture of the two diseases.

It is apparent that mice dying with typical poliomyelitis symptoms had W.E.E. virus in high concentration in the central nervous system, and it seems reasonable to infer that there was a concomitant growth of these viruses. It seems reasonable further to assume that whether a mouse died with poliomyelitis or encephalitis was a fortuitous circumstance depending on which virus happened to gain the ascendancy. Unfortunately, the long incubation period of poliomyelitis virus and the short incubation period of W.E.E. virus precluded the possibility of demonstrating the presence of poliomyelitis virus in mice dying with W.E.E. symptoms.

The question why certain viruses interfere with each other while other viruses may grow together in the same host organ or tissue remains unanswered. The observations here reported, that equine encephalomyelitis virus and poliomyelitis virus may grow together in the brains of mice, whereas these viruses interfere with each other in monkeys, do not clarify the situation but nevertheless provide additional information. An elucidation of this fundamental problem will undoubtedly add greatly to our knowledge of the basic mechanisms of virus growth.

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Thyroid Destruction by I^{131}, and Replacement Therapy[1]

C. F. Winchester,[2] C. L. Comar,[3] and George K. Davis

Department of Animal Industry, University of Florida, Gainesville

The early work of Graham (*1*) on milk cows, and of Winchester (*5*) on chickens demonstrated a very definite relationship between thyroid function and milk and egg production. Winchester (*6*) also found a parallelism between thyrotropic hormone content of the pituitary, basal metabolism, and egg production of hens. Subsequently, numerous reports have been made concerning the use of various drugs that in one way or another influence metabolism, and eventually the production of farm animals (*3, 4*). The literature is characterized by a certain amount of disagreement as to the effects of the drugs, and emphasizes the need for further research on the fundamental aspects of thyroid function.

This report presents a method by which thyroids of young chickens have been eliminated by I^{131} irradiation, apparently without damage to the animal other than some possible destruction of parathyroid tissue; a procedure similar to that of Gorbman (*2*), who has reported 100% destruction of thyroids of 2–5-month-old mice by I^{131} injected in doses ranging from 18 to 55 mc of activity per kg of body weight. The work reported here, however, deals with thyroid destruction by I^{131}, or radio-thyroidecrexis,[4] during the first few days of life rather than after the animal has reached full size. Further, by means of thyroxin therapy, birds lacking thyroid glands were brought to full size and into egg production, while similar birds that did not receive thyroxin survived for limited periods only, and grew very slowly.

TABLE 1

IODINE 131 DOSAGE AND PERCENTAGE THYROID DESTRUCTION (RESULTS OF FIVE TRIALS)

Dose I^{131}	No. of chicks	Exposure period	Estimated thyroid destruction
mc/100 g		days	%
1–1.9	2	5	0
	1	over 24	10
2–2.9	1	5	10
	1	5	50
	1	over 24	75
3–3.9	1	over 24	90
	4	over 24	100
4–4.9	1	5	nearly 100
	1	over 24	90
	4	over 24	100
5–5.9	1	5	nearly 100
	4	over 24	100
6–6.9	2	over 24	100
9.2	2	over 24	100

The birds used in these experiments were New Hampshire chicks, obtained from a commercial hatchery, and weighed 40–66 g at the time of I^{131} injection.

[1] Published with the permission of the Director of the Florida Agricultural Experiment Station.

[2] Present address: Animal Husbandry Division, U.S.D.A. Agric. Res. Center, Beltsville, Maryland.

[3] Present address: University of Tennessee, Knoxville, Tennessee.

[4] Since the literature apparently includes no term indicative of thyroid elimination by other than surgical means, the authors have deemed it necessary to introduce a suitable term in order to avoid undue repetition and use of descriptive material. After due consideration, the term "radio-thyroidecrexis" (pronounced *e-krek'-sis*) has been selected to indicate total destruction of the thyroid gland in the live animal by means of a radioactive substance such as I^{131}. Grateful acknowledgement is due Dr. Joseph Brunet, professor of ancient languages, University of Florida, for his cooperation in offering a number of possible terms from which a final selection was made by the authors.