200-foot interval, making it possible to choose the most significant 100-foot contour in any specific area. Approximately a third of the work of this map has been done.

For part of the Ozark area and part of the North Central portion of the state no sheets are available and airplane photographs are to be used to aid in plotting the physiography. Field work may be necessary in order to check some parts of the map. At present the project has been interrupted, but there is no doubt that an accurate physiography wall map of Missouri will be needed as much after the war as now.

SUMMARY

In addition to the two wall maps and the accompanying isopleth map of population distribution the project calls for a detailed analysis of the distribution of the population of the state in terms of the physiography. The two maps on the same scale may be published as twin wall maps in order to portray the geographic distribution of population. In a state with such variety in land forms it is hoped that a close relationship between population distribution and physiography can be brought out with great emphasis. It is hoped that this brief report will record the good intentions of the author to provide new maps useful in the field of geographic teaching and research.

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SPECIAL ARTICLES

A FURTHER INTERFERENCE IN EXPERIMENTAL POLIOMYELITIS

WE have long sought a method of demonstrating and studying the "sparing effect" or "interference phenomenon" in poliomyelitis[1,2] in cheaper animals than the monkey. Mouse experiments have failed to provide a substitute, but an equivalent has been demonstrated in hamsters using strains of virus recovered from the Battle Hill cases.[3]

Young hamsters (40–45 gms) are almost invariably paralyzed within 5 days following the intraperitoneal injection of 0.2 cc of a 10 per cent. suspension of mouse brain collected from animals infected with M-hamster virus. This strain of rodent-paralyzing virus was recovered from a fatal human case by hamster passage.[3] Susceptible animals develop flaccid paralyses of 1 or more extremities between the second and fifth days. Older hamsters are often refractory and can not be used.

If the animals have been injected intracerebrally with certain other rodent paralyzing viruses, they remain free of symptoms. The first experiments were made with McG virus, a weak strain of rodent paralyzing virus isolated in this laboratory. The intracerebral injection of 0.05 cc of a 10 per cent. suspension of mouse brain harvested from animals infected with McG virus rarely paralyzes hamsters but fully protects them against subsequent inoculation with the M-hamster strain. The protection is well developed within 6 days and persists for from 6 to 8 weeks. Armstrong's Lansing strain of mouse poliomyelitis[4] and Jungeblut and Sander's murine SK strain[5] are equally effective. Lymphocytic choriomeningitis also confers protection, as we originally demonstrated in monkeys.[1] Since the other effective viruses may all be related, the action of choriomeningitis and the time relationships are important in showing that the phenomenon is an interference rather than cross immunity.

Suspensions prepared from the brains of young, normal mice afford no protection, but the brains of older mice occasionally have done so. This is believed to imply that latent mouse encephalomyelitis virus is not infrequently present in the brains of old mice.

A suspension of pooled monkey cords, collected 4

EFFECT OF VARIOUS INTRACEREBRAL INJECTIONS ON THE RESISTANCE OF HAMSTERS TO M-HAMSTER VIRUS

Preliminary inoculum	Response	Interval (days)	Number of animals	Response to M-hamster virus	
				None	Paralysis
McG strain	none	1	2		2
" "	"	4	2		2
" "	"	7	2	2	
None			2		2
McG strain	none	6	2	2	
" "	"	26	4	4	
" "	"	34	2	2	
" "	"	47	3	2	1
None			6		6
Lansing (Armstrong)	1 paralyzed	26	4	4	
SK (murine)	All paralyzed	33	3	3	
MV monkey cord	none	21	3	2	1
None			3		3
Feces "Di"	none	7	3	3	
" (heated)	"	7	3		3
Feces "McG"	"	7	3	2	1
" (heated)	"	7	3		3
Feces "Pa"	"	7	3	1	2
" (heated)	"	7	3		3
Feces of newborn	"	7	3		3
None			6	1	5

[1] G. Dalldorf, M. Douglass and H. E. Robinson, SCIENCE, 85: 184, 1937. G. Dalldorf, *Jour. Exp. Med.*, 70: 19, 1939.

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[3] C. W. Jungeblut and G. Dalldorf, *Am. Jour. Pub. Health*, 33: 169, 1943.

[4] C. Armstrong, *Pub. Health Rep.*, 54: 1719, 1939.

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[5] C. W. Jungeblut and M. Sanders, *Jour. Exp. Med.*, 72: 407, 1940.

years ago from animals dying of MV poliomyelitis, also protected hamsters. This led to the testing of Seitz and Berkfeld "N" filtrates of 10 per cent. suspensions of feces collected from human cases of poliomyelitis. Duplicate portions of the filtrates were heated for 1 hour at 60° C. and the feces of newborn infants and presumably uninfected children were also tested. The results of representative experiments have been summarized in the accompanying table.

The procedure seems to afford a satisfactory means of investigating the interference phenomenon in poliomyelitis. The results are sufficiently definite to permit the use of small groups of inexpensive animals and the test period is relatively brief. Whether the procedure is useful in demonstrating the presence of poliomyelitis virus, as in feces where other interfering viruses probably do not occur, requires investigation.

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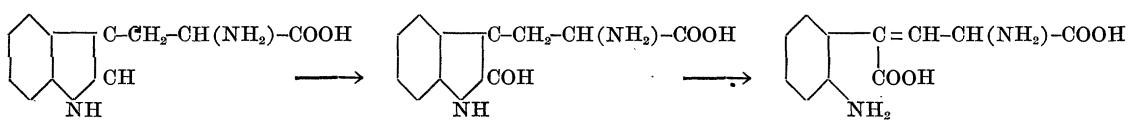
THE TRYPTOPHANE CONTENT OF a^+a^+ and aa EPHESTIA KÜHNIELLA Z.[1]

IT has been shown that in Ephestia homozygous for the gene *a* and in Drosophila homozygous for the gene *v* the lack of pigmentation of the eyes and other organs is due to lack of a specific diffusible substance necessary for the formation of these pigments.[2, 3] This substance has turned out to be kynurenin, a derivative of tryptophane.[4] The oxidation of tryptophane to kynurenin proceeds according to Kotake in the accompanying formula.



tryptophane α-hydroxytryptophane kynurenin

From the observation that α-hydroxytryptophane has a similar though slighter effect than kynurenin itself, it has been concluded that the genes *a* and *v* in homozygous condition inhibit the oxidation of tryptophane in the α position.[5] This is, however, not the only possible explanation for lack of kynurenin. Any mechanism breaking down tryptophane in a different way or removing kynurenin in a way so that it can not be used for the formation of pigment would have the same effects. In order to obtain evidence bearing on this question, tryptophane determinations in Ephestia homozygous for *a* and for the corresponding wild type allele a^+ have been undertaken.

Ephestia imagoes were crushed in a mortar and dried in a desiccator over concentrated H_2SO_4. After three days, the dry material was pulverized in a mortar and kept in a dessiccator in the dark until used. In some samples, males and females were kept separately, whereas in other samples specimens of both sexes were used together. Samples of these powders were extracted over night (14 to 16 hours) in 10 per cent. NaOH in a water bath at $56 \pm 2°$ C. The fluid was decanted, the solid washed once with 10 per cent. NaOH, and the washing fluid added to the first solution. The amount of tryptophane in the solution was determined with glyoxylic acid.[6] The developing color was read with an electric colorimeter. In a few instances, tryptophane was determined with p-dimethylaminobenzaldehyde after acid hydrolysis at 37° C.[7] In these cases, the color was determined with a Dubosq colorimeter. The results obtained are indicated in Table I.

In the a^+ a^+ animals, the males contain significantly more tryptophane than the females ($t = 4.08$, $n = 20$, $P < .01$). This is different from the findings of Demyanovskii[8] in *Bombyx mori*, where the females had consistently higher amounts of tryptophane than the males. The difference in tryptophane content between the two sexes in the *aa* race is in the same direction, but insignificant ($t = 0.58$, $n = 14$, P between .5 and .6). In all cases, the *aa* animals contain more tryptophane than the a^+ a^+ animals. This difference is highly significant in the samples derived from both sexes ($t = 6.07$, $n = 26$, $P < .01$) and from the females ($t = 3.59$, $n = 18$, $P < .01$), only barely significant in the males ($t = 2.58$, $n = 18$, P slightly larger than .02). The results with p-dimethylaminobenzaldehyde are less reliable than those obtained with glyoxylic acid, since the visual color determination was interfered with by the appearance of a yellow color in the a^+ a^+ material, which might perhaps be due to kynurenin.[9] It agrees,

[1] This work was aided by a grant-in-aid of the American Association for the Advancement of Science. The author wishes to acknowledge valuable advice given by Dr. J. H. Wilson and Dr. F. O. Zillesen of Easton.

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