In this way speeds from 13 to 64 miles per hour were produced.

"Observations in a room, with a brightly lighted white ceiling as background, showed that at 13 miles per hour (580 cm./sec.) the 'fly' was merely a blur—the shape could not be seen, but it could be recognized as a small object of about the correct size.

"At 26 m./hr. (1,150 cm./sec.) the fly was barely visible as a moving object. . . ."

He concludes that "a speed of 25 miles per hour is a reasonable one for the deer fly."

It may well be that energy considerations limit the speed of the deer fly to a mere 25 miles/hour, but I wish to give evidence indicating that the fly would be visible at much higher linear velocities. Neither the article in the *Illustrated London News* nor the original article by Dr. Townsend (*2*) states at what distance the fly was observed. Had Dr. Langmuir been able to produce similar rotations with a thread 94 instead of 3 feet long, it seems likely that his artificial "fly" would have been visible though traveling at over 100 miles/hour. The reason is that the acuteness of human vision is diminished not by the linear, but by the angular, velocity of the object viewed.

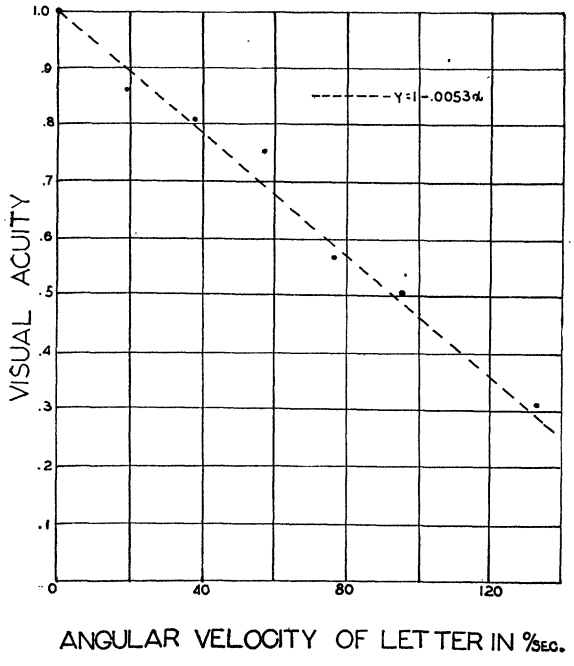

FIG. 1

The data presented in Fig. 1 show some results I obtained during a general experimental investigation of visual acuity while viewing a moving object. The data are for constant angular velocity in the horizontal plane. The test objects were Snellen letters.

The expression $y = 1 - .0053x$ is a fair empirical fit to the data. A stationary black disc on a white background can be seen when it subtends an angle of 25–30″ of arc at the eye, and the corresponding value for a stationary black line on a white background is 4–6″ of arc (*3*). The artificial fly presents a roughly rectangular cross section to the eye, and we may assume an intermediate value of 18″, or .005°, as the angle necessary for vision when the fly is stationary. The angle necessary for vision when acuity is reduced by moving the fly is, then, $\beta = \frac{.005°}{1-.0053\,\alpha}$, where α is the angular velocity in degrees per second. This velocity equals approximately $57.3\,\frac{v}{r}$, where v is the linear velocity in feet per second and r the distance to the object in feet. The angle, θ, subtended by the fly, is approximately $57.3\,\frac{s}{r}$ in degrees, s being the size of the object in feet.

For visibility, $\theta = \beta$, or $57.3\,\frac{s}{r} = \frac{.005°}{1 - .304\,\frac{v}{r}}$. From this, $v = 3.29\left(r - \frac{.005\,r^2}{57.3\,s}\right)$ and $\frac{\partial v}{\partial r} = 3.29\left(1 - \frac{.01\,r}{57.3\,s}\right)$. It appears that the highest velocity with good visibility will occur when $r = 5{,}730\,s$. Taking Dr. Langmuir's value of .5 cm., or .0164 feet, for s, we find that the optimum distance for observation of the fly is 94 feet. At a distance of 3 feet, the fly would begin to blur at about 7 miles/hour, a figure in substantial agreement with Dr. Langmuir's observations. However, at a distance of 94 feet and with contrast and other conditions of vision optimal, the deer fly might be seen while traveling at 105 miles/hour, if it can fly that fast.

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Production of Yellow Bean Mosaic in Beans by Virus From Mottled Gladiolus

F. P. MCWHORTER, LYTTON BOYLE, and B. F. DANA[1]

Oregon State College, Corvallis

The first observation conducive to this record of an improbable source of Bean Virus 2 was made by Carl Robertson, of the Eugene Fruit Growers Association, Eugene, Oregon. In 1939 Mr. Robertson asked local pathologists interested in vegetable diseases to observe a field of *Phaseolus vulgaris* L. var. Blue Lake, planted contiguous to a field of *Gladiolus* spp. He wished confirmation of his observation that a gradient infection of mosaic extended from the row nearest the gladioli to a distance of approximately 150 feet, and of his opinion that the gladioli were the probable source of the virus. While the correlation between high mosaic percentage in the bean planting and the nearness of gladiolus plants was amazing, we supposed the apparent correlation was nonsignificant, having found an occasional mosaic-diseased clover plant in

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the vicinity. Subsequent observations and tests have proved that Mr. Robertson's surmise was correct.

During later years we have observed this same Bean Virus 2 correlation whenever snap beans were exposed to commercial plantings of gladioli. Two 1945 occurrences are illustrative. A two-acre commercial test of varieties of *P. vulgaris* for canning was planted in a portion of a large, well-tilled field near Salem, Oregon. There were no other legume crops in the area nor any apparent usual sources of Bean Virus 2. One end of the bean field was exposed to a five-acre planting of gladioli growing weed free in exceptionally good culture. The rows of beans ended about 15 feet from the gladiolus plants. An extremely severe infection of yellow bean mosaic began early in the season at the end of the planting next to the gladioli and developed into a gradient infection extending away from them to a distance of about 200 feet. All the bean plants within 50 feet of the gladioli were ruined by the mosaic. The second case occurred in a field near Portland. Two rows of snap beans, each approximately 150 yards long, were planted parallel to, and 20 feet from, rows of gladioli in a commercial field. At the end of the bean rows, and equally exposed to the gladiolus plants, was a planting of Lima beans, *P. lunatus* L. All of the snap beans and none of the Lima beans were dwarfed by necrotic and mottle strains of yellow mosaic. The significance of this field evidence is enhanced by the fact that we have been unable to prove seed transmission for any strain of Bean Virus 2.

During the war years a serious virus disease of snap beans appeared in Oregon. This disease is characterized by various necrotic symptoms which, in extreme cases, lead to the death of young plants. An investigation of this disease (*1*) and of yellow mosaic commonly associated with it has shown that the necrotic symptoms and death of plants are caused by strains of Bean Virus 2. Yellow mosaic appears to be due to a complex of Bean Virus 2 strains. Some of these have modified properties, but all form specific cytological evidence (*2*) in the foliar cells of *Vicia Faba* L. During the investigation we tested commercial gladiolus plants as sources of the yellow mosaic complex. Preliminary inoculations to *P. vulgaris* L. var. Blue Lake by the carborundum method (*3*) were not conclusive. Later trials, using plants of *V. Faba* L. as inoculates, gave transfers of 1/8, 1/9, 0/12, 0/12, 0/12, 0/20, 2/44, 0/9, 1/10, thus giving positive transmission in four out of nine trials. These cross-inoculations were made with precautions and controls that make these low percentages of transfer significant. The isolations from gladiolus included not only the typical yellow bean mosaic but also some of the necrotic types. It is very probable that the numerous plantings of gladioli in western Oregon, where beans are grown for canning, may have played some definite role in the recent epiphytotic of virulent strains of Bean Virus 2. The details of this investigation will be reported elsewhere.

All the gladiolus foliage used for preparing inocula had the usual mottle characteristic of gladiolus mosaic in commercial plantings. No virus-free gladiolus plants were available to determine whether gladiolus mosaic can be induced by inoculating gladioli with Bean Virus 2 from beans.

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Production and Treatment of Hepatitis With Focal Necrosis of the Liver in White Mice

Paul Engel

Laboratorios Hormona, Bogotá, Colombia

Different series of white mice were treated during various weeks with coal tar, an area of about 1½ cc.[2] being painted every second day. Some of the animals died in the first days; most of them, after 2–6 weeks. In the livers of these animals were found small, well-determined yellow spots.

Histological examination[1] revealed hepatitis and acute congestion of the organ, with zones of focal necrosis, some of them small and others extending over different lobes. In the portal areas there appeared a leucocyte reaction, with cloudy swelling and nuclear alterations, especially cariorrhexis. No other organ was affected. The percentage of animals with liver damage was rather high, as indicated in Table 1.

TABLE 1

Series No.	No. of animals	Sex	Treatment	No. of animals	
				With liver damage	Without liver damage
1	40	male	Coal tar	22	18
2	15	female	"	8	7
3	24	male	"	14	10

Sato and, later on, Forbes and Neale (*1, 2, 4*) have described antitoxic substances in the liver. In this laboratory we have studied the influence of liver extracts on the coal-tar-produced liver damage, using a concentrated, sour, aqueous liver extract, which had been made protein free by alcohol precipitation, and 0.1 cc. of which corresponded to 3 grams of fresh beef liver. The animals were painted three times a week with coal tar and also received injections of the liver extracts three times a week. As controls, various series of mice were treated: two series only with coal tar, and two others with

TABLE 2

Series No.	No. of animals	Sex	Treatment	No. of animals	
				With liver damage	Without liver damage
4	23	male	Coal tar	0	23
5	20	"	Liver extract "	2	18
6	20	female	"	2	20
7	31	male	"	0	31
8	10	female	Coal tar	4	6
9	14	male	"	9	5
10	6	"	Coal tar Histamine	5	1
11	10	female	Coal tar Vitamin B	2	8
12	16	male	"	9	7

[1] The author is indebted to Dr. Augusto Gast Galvis, of the Instituto de Estudios Especiales in Bogotá, for the histological studies of the livers.