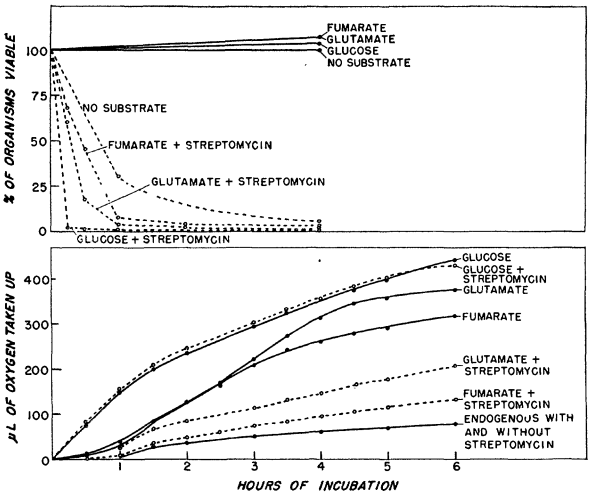

FIG. 1. Effect of streptomycin on respiration and viability of *E. coli*. The oxygen uptake studies are shown in the lower portion of the figure; conventional Warburg techniques were used. Fifty-milliliter Erlenmeyer flasks were used for the viability tests, shown in the upper part of the figure. The same suspension of cells was used in both instances and the ratios of cells to substrate and to streptomycin were kept the same, as follows: cells, 0.5 mg/ml (dry weight) containing approximately 2×10^9 viable cells; substrates, 5 μmoles; streptomycin, (dihydrosulfate) 100 μg/ml; phosphate buffer pH 7, 0.0036 *M*. The experiments were conducted at 37° C, with agitation to insure adequate oxygenation. Aliquots were removed at intervals for pour plate counting in heart infusion agar (Difco) with 1% glucose.

Streptomycin is noted to have no effect on endogenous respiration or on respiration in the presence of glucose, whereas there is a marked inhibition of oxygen uptake with fumarate and glutamate as substrates. Noticeable inhibitory effects become apparent after 1 to 2 hr of incubation. In the absence of streptomycin there is a lag period with glutamate and fumarate before the maximum rate of oxygen uptake occurs, whereas there is no lag period in respiration with glucose as substrate.

Killing of the cells by streptomycin is most rapid in the presence of glucose as substrate, with over 99% of the cells being killed in the first 15 min. Despite this fact, however, oxygen uptake with glucose as substrate continues in an unaffected fashion. The rates of killing are less rapid with glutamate and fumarate as substrates and least rapid in the absence of added substrate. The killed cells could not be revived by repeated washing in an attempt to remove the streptomycin. Similar experiments using streptomycin-resistant and streptomycin-dependent variants of this strain of *E. coli* showed no killing or respiratory inhibition by streptomycin.

These results indicate that streptomycin can kill susceptible coliform cells in the resting state in the absence as well as in the presence of inhibitory effects on respiration. The inhibition of oxygen uptake by streptomycin seems to bear no particular relation to the killing effect of this drug but rather to the substrate being metabolized. In view of the lag phase demonstrated in Fig. 1 in the oxygen uptake curves with fumarate and glutamate as substrates, respiratory inhibition with these two substances by streptomycin may simply represent an interference with adaptive enzyme formation, as earlier suggested by other workers (*5*).

It is not possible to say categorically that the cells have already been killed before plating because viability can be determined with certainty only by subculture. The streptomycin may merely be "fixed" in the bacterial cells during the period of exposure and "death" may occur following the initiation of metabolic activities concerned with growth when the cells are transferred to a suitable culture medium. Perhaps streptomycin renders the cells "sterile," that is, they are not dead but cannot reproduce themselves.

In any case it is fair to say that the fixing of streptomycin to the susceptible site in the cell, with subsequent death of the cell, occurs most rapidly in the presence of metabolizable substrates and least rapidly with endogenous metabolism. The rate of fixing of the drug to the cells appears to be directly related to the rate or degree of metabolism of the cells.

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The Effects of Wind-Drift of Weed-Killer on Some Puerto Rican Trees

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For several years one of the junior writers has been noticing the toxic effects of 2-4-D and other weed-killers on susceptible species of cultivated plants such as Sea Island cotton, *Gossypium barbadense*, and papaya, *Carica papaya*. The damage to these plants is particularly noticeable when they grow near fields of sugarcane in Puerto Rico, where weed-killers are used to control weeds.

Last year at the Isabela Substation an entire papaya grove of about 600 plants was seriously affected by 2-4-D sprayed 2000 yd or more to the east. The fine spray of the weed-killer carried by the trade winds from the northeast was powerful enough to affect all the papaya plants.

Since January 1952, the writers have been making field observations and taking numerous photographs of the effects of weed-killers on our flora. Detailed observations will be published elsewhere in the near future. However, it is of interest to note that two common species of trees in Puerto Rico serve as an

index to indicate the susceptibility of plants to weed-killers, particularly 2-4-D.

On the south coast of Puerto Rico, the "Santa María" or "emajagüilla," *Thespesia populnea,* a malvaceous tree commonly grown along the roadsides, serves as an index of susceptibility in this area of Puerto Rico. The distorted leaves of this tree (Fig. 1) show symptoms of 2-4-D toxicity at all times of year, but most noticeably during the spring, when "primavera" cane as well as ratoon cane are being sprayed with weed-killers. Even more conspicuous is the injury to another common roadside tree which grows more generally in the northern part of the Island: the "almendro," *Terminalia catappa* (Fig. 2). In the Naguabo-Humacao area many mature trees, 30 to 50 ft in height, have been observed that were killed by the effects of 2-4-D used in adjacent cane fields (Fig. 3).

FIG. 1. Foliage of *Thespesia,* showing effect of 2-4-D toxicity.

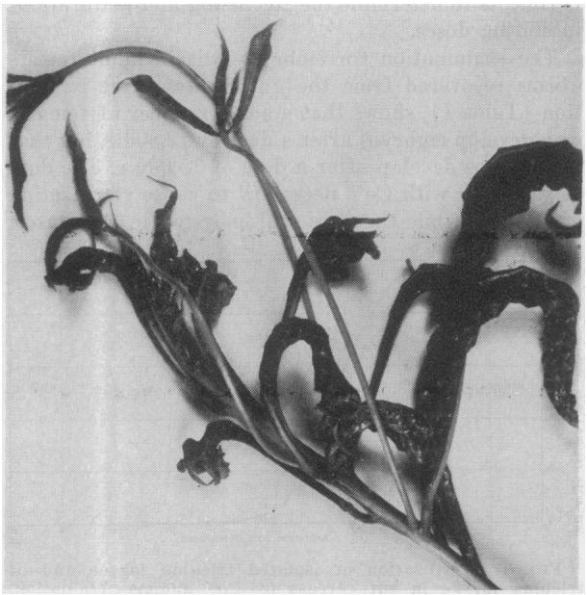

FIG. 2. Terminal twig of "almendro," *Terminalia,* showing effect of weed-killer.

FIG. 3. Dead and dying roadside trees of "almendro," *Terminalia catappa,* on the Naguabo-Humacao road, Puerto Rico. (Photos courtesy of the Photographic Department, Agricultural Experiment Station, Río Piedras, Puerto Rico.)

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Effect of Irradiation with Cobalt-60 on Trichina Larvae

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A previous study of the effect of 200 kv x-rays on trichina larvae (*1*) showed that a dose of 750,000 r kills trichina larvae *in vitro,* whereas a dose of approximately 5000 r inhibits maturation of larvae to adult forms, and a dose of approximately 3500 r produces sexual sterility of the adult forms that mature from irradiated larvae. The present study was undertaken to determine the radiation dose from a cobalt-60 source that would produce the same effects.

Alicata and Burr (*2*) found that exposure of trichinous meat to 12,000 r of cobalt irradiation produced sterility in 60–100% of the adult female worms recovered from the intestinal tract of experimental rats 6 days after the latter were fed the irradiated larvae.

Trichinous rat muscle and isolated trichina larvae obtained by the digestion of trichinous rat muscle