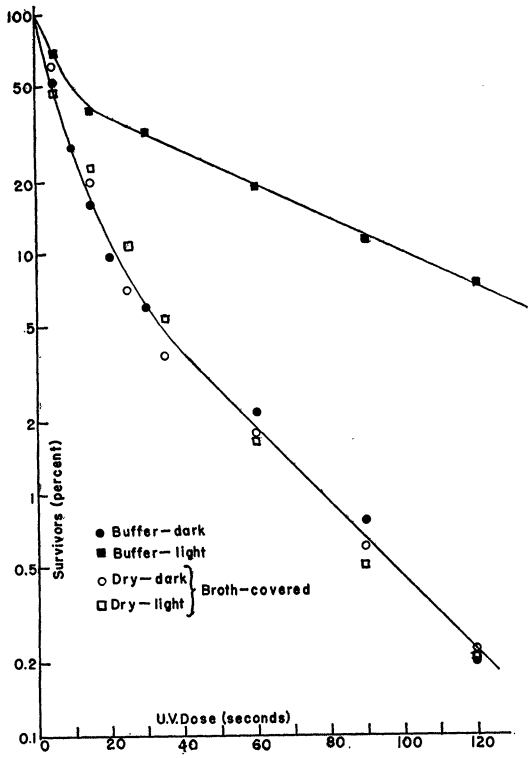

FIG. 1. UV inactivation (dark) and photoreactivation (light) of T1 irradiated in buffer solution and in dry preparations made by spraying a broth suspension.

also. There seems to be no reason to assume that the survival in the dry state should be compared with the acetate suspension rather than the buffer suspension, since drying leads not only to evaporation of water, but also to sublimation of ammonium acetate. It has not as yet been determined whether the lowered sensitivity in acetate is due to UV absorption in this medium or to some protective effect.

The experiments were carried out at room temperature. The effect also persists, however, at lower temperatures. This was shown by experiments in which the cover glass preparation was supported inside a watertight cell supplied with a quartz ceiling. The cell was filled with Drierite and kept in a water bath at 5° C for ½ hr before irradiation, in order to ensure temperature equilibrium. No photoreactivation was detected either for the phage irradiated at 5° or at 25°. The effects of temperatures above 25° C are under study.

Since the UV survival curve is unchanged in the dry state, it seems likely that inactivation proceeds by the same mechanism. It would appear, however, that photoreactivation of some of this damage requires not only adsorption to bacteria and application of light but is also dependent on the state of the phage at the time of UV exposure.

TABLE 1*

EFFECT OF DRYING ON PHOTOREACTIVATION OF PHAGE

Treatment	Assay	Ratio (UV): (UV → PR)
A	1.0×10^{10}	
A → UV	5.0×10^{8}	1: 6.2
A → UV → PR	3.1×10^{9}	
A → D	1.3×10^{9}	
A → D → UV	2.8×10^{7}	1: 1.4
A → D → UV → PR	3.9×10^{7}	
A → UV → D	8.8×10^{7}	1: 5.6
A → UV → D → PR	4.9×10^{8}	
A → D → A → UV	8.3×10^{7}	1: 6.3
A → D → A → UV → PR	5.2×10^{8}	

* *A* refers to suspension (also resuspension) of T1 in 2% ammonium acetate, *D* to drying, *UV* to a 60-sec exposure to the germicidal lamp, and *PR* to exposure to the H-5 lamp of sufficient duration to obtain maximal reactivation. All data are normalized to an initial titer of 1.0×10^{10}.

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Greater Resistance of the Female to Experimental Burns Following Starvation

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In the course of experimentation in the chemoprophylaxis of burns (*1, 2*), certain trends in the survival rates of intact rats subjected to thermal injury were observed that could not, under the conditions described, be unequivocally attributed to sex factors. In order to investigate sex differences under more rigorous conditions, starvation was selected as an additional stressor prior to burning. Since it has long been known that liver lipids increase during fasting (*3*), and, more recently, that under fasting conditions the lipid accumulation is higher in the liver of the female than in that of the male (*4*), it was hoped that the marked lipotropism seen in the starved female would be a factor in increasing the female survival rate over that of the male after burns. Of the lipotropic substances, only methionine has to our knowledge been tested in burns and found to be protective (*5*). Accordingly, each of three groups of albino rats consisting of nearly equal numbers of males and females of similar body weights was starved for 50, 75, or 100 hr, anesthetized with pentobarbital sodium, immersed up to the neck in water at 75° C for 20 sec, according to the method of Hazán and Treadwell (*5*), and returned to a standard diet. Water was available

TABLE 1

SEX DIFFERENCE IN BODY WEIGHT LOSS

Duration of starvation	MF	50 hr	75 hr	100 hr
No. animals	M	18	18	19
	F	19	20	18
Mean initial body weight and range (g)	M	165 (122–205)	166 (125–200)	164 (124–200)
	F	162 (115–195)	163 (119–208)	162 (130–198)
Body weight loss (% of initial)	M	22	27	31
	F	18	21	24

at all times. A series of animals of similar body weights and equally divided as to sex, but well fed, was included and burned under the same conditions as the starved animals.

The percentage body weight lost by each of the starving groups for the period between the beginning of starvation and the time of burn is tabulated with related data in Table 1. It may be noted that (*a*) within any one level of starvation males appear to suffer a greater loss of body weight than females, and that (*b*) the percentage weight loss of males at 50 and 75 hr of starvation either equals or exceeds that of females at 75 and 100 hr, respectively.

When survival rates of fasting burned males are compared to those of similarly treated females (Table 2), it is quite evident that at the stated levels of starvation the female survival rates at 12, 24, or 48 hr post-burn are higher than that of males at the same time intervals. Although the number comprising each group is small, nevertheless it is of interest that if the level of significance is set at a probability of 1% or less for the absolute number of animals in each group surviving at 12 or 24 hr, a statistically significant sex difference is apparent in animals starved for 75 or 100 hr but not in those fasting for 50 hr, though here, too, the males suffered a higher mortality. When the number of survivors at all levels of starvation is grouped by sex, a statistically significant sex difference is evident again at the same 12th or 24th hr post-burn.

In the administration of a lethal burn the survival times, as well as the survival rates, are of interest. It is noteworthy that the last surviving female outlived by 48 hr the last starved male survivor. When the survival rates of nonfasted burned males are compared to those of nonfasted burned females, differences are not easily apparent. We have, however, insufficient grounds for rejecting the hypothesis that there are no sex differences in the survival rates of fed rats.

The observation that the male, after having lost in 50 hr as much weight as the female in 75 hr of starvation, still evidenced a higher mortality does not negate the possibility that the factor of the sex-related differential weight loss may be causal in determining survival rates, but is indicative at least of other *modi operandi*. In evaluating possible mechanisms of action one must cite, besides anatomical sex differences, the differential processing of male and female gonadal hormones by the liver (*6*). This process is accentuated in starvation, resulting in an increased estrogen/androgen ratio. Estrogens or progesterone may thus offer the female greater protection against burns either directly or through lipotropic actions, or through the intermediary of other indirect metabolic pathways. The work of Cowie (*7*) points to a sex difference in the survival time of adrenalectomized rats in favor of females. These findings lend further support to the inference that sex-related endocrine factors may be

TABLE 2

SEX DIFFERENCE IN MORTALITY

Groups	Sex	No. survivors (hours post-burn)					
		0	3	12	24	48	72
Unfasted	M	13	12	7	5	4	2
50-hr fast		18	17	6	5	0	0
75-hr "		18	17	4	0	0	0
100-hr "		19	16	3	0	0	0
Unfasted	F	13	9	6	5	3	3
50-hr fast		19	18	11	9	1	1
75-hr "		20	15	14	11	1	0
100-hr "		18	15	12	8	1	1
Fasting groups (totals and % surviving)	M	55 (100%)	50 (91%)	13* (24%)	5* (9%)	0 (0%)	0 (0%)
	F	57 (100%)	48 (84%)	37* (65%)	28* (49%)	3 (5%)	2 (4%)

* $p = < 0.01$ when comparison is made by chi-square (Yates' correction) between absolute number of fasting male and female survivors at the same time interval after burn.

operative in the greater resistance of starved female rats to burns.

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The Effect of Applied Sugars, Light Intensity, and Temperature upon the Chemical Defoliation of Cotton[1]

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In experiments (*1–3*) conducted during the past two years it was noted that the addition of sucrose to defoliant sprays generally resulted in increased defoliation of the cotton plant. This response was more consistent in greenhouse-grown plants than in field plants (*1*). These results appear to be contrary to the effects of sucrose upon abscission obtained by other investigators (*4–6*) working with various species. Went and Carter (*6*) reported less abscission of flowers and increased fruit set in tomato following the application of sucrose under conditions of high temperatures and low light intensity. Livingston (*5*), working with citrus explants, and Brown and Addicott (*4*), using bean explants, obtained a retardation of the abscission process when sucrose was applied daily.

Most workers (*7–10*) seem to agree that hormone phytocides, particularly 2, 4-D, are absorbed into the leaf in the dark better than, or as well as, in the light, but there is probably no translocation of 2, 4-D from the leaf in complete darkness or from one that has been made starch-free. Moreover, other workers (*8–12*) have reported that the addition of sugars to leaves devoid of translocatable carbohydrates resulted in transport of 2, 4-D, as well as other synthetic auxins from leaves kept in the dark.

Because of the apparent importance of sugar in the translocation of materials from the foliage and the opposite effect noted upon abscission (seemingly depending upon whether sucrose was applied alone or in combination with defoliants), it appeared worth while to study the phenomenon in greater detail. To accomplish this an experiment was undertaken whereby chemically induced defoliation could be followed in cotton plants maintained under varying conditions of light intensity, temperature, and sugar supply.

On June 19, 1951, young, preflowering Stoneville 2B cotton plants, grown outside in fertile soil in 3-gal jars, were selected for uniformity and divided into eight lots of 16 plants each, 4 plants per jar. Duplicate lots were placed under the following conditions of light and temperature: *A*, Lots 1 and 2, outside in high light intensity (8000–10,000 ft-c at noon), high temperature (96° F av max, 72° F av min); *B*, Lots 3 and 4, moderate light intensity (3000–5000 ft-c at noon), moderate temperature (88° F av max, 74° F av min); *C*, Lots 5 and 6, darkness and high temperature (mean 98° F); and *D*, Lots 7 and 8, darkness and moderate temperature (mean 83° F).

On June 23, when starch could no longer be detected in the leaves of plants placed in the dark (Lots 5–8, inclusive) by the I_2KI test, aqueous solutions of 3% Shed-A-Leaf (sodium chlorate–pentaborate) and 2% Endothal (disodium 3, 6-endoxohexahydrophthalate + ammonium sulfate) were prepared, as well as other solutions of the defoliants, plus 2.5% of the various sugars as indicated in Table 1. The plants were then treated as indicated (Table 1) by rolling the leaf slightly to facilitate immersing it into a beaker of test solution, the excess allowed to drain, and the procedure repeated for all leaves in a given treatment. On the average each treatment included 32–36

TABLE 1

THE EFFECTS OF SUGARS, LIGHT INTENSITY, AND TEMPERATURE UPON THE CHEMICAL DEFOLIATION OF YOUNG COTTON PLANTS

Defoliated with Endothal		Defoliated with Shed-A-Leaf	
Sugar Additive	% Abscission	Sugar Additive	% Abscission
Lots 1 and 2			
Endothal Control	50	Shed-A-Leaf Control	60
Dextrose	80	Dextrose	84
Levulose	70	Levulose	88
Sucrose	92	Sucrose	84
Lots 3 and 4			
Endothal Control	40	Shed-A-Leaf Control	60
Dextrose	60	Dextrose	85
Levulose	100	Levulose	93
Sucrose	100	Sucrose	90
Lots 5 and 6			
Endothal Control	10	Shed-A-Leaf Control	6
Dextrose	45	Dextrose	95
Levulose	70	Levulose	92
Sucrose	70	Sucrose	80
Lots 7 and 8			
Endothal Control	Lethal	Shed-A-Leaf Control	5
Dextrose	"	Dextrose	90
Levulose	25	Levulose	100
Sucrose	Lethal	Sucrose	100

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