From an inspection of the data in Table I it appears that:

1. The "Rough" strain is, and the "Smooth" strain is not spontaneously agglutinable in distilled water.
2. Both strains showed marked acid agglutination.
3. Only the "Rough" showed alkali agglutination.
4. In the acidulated solution there is correlation between the effects of acidulation upon agglutination and upon reduction of the negative P. D. to approximately zero (isoelectric) or to slightly positive values.
5. There is no definite correlation between P. D. and agglutination in alkaline solutions.
6. The "critical" potentials are different for the "Rough" and "Smooth" strains.

[1] *Cf.* Sharp, *Bot. Gaz.* (in press). Link and Sharp, *ibid.*

[2] *Cf.* Falk, Gussin and Jacobson, *J. Inf. Dis.*, 1925, xxxvii, 481-494.

3471

Production of Toxic Substances in Vitro by Fusarium Lycopersici.

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The purpose of this research was to determine how the pathological effects characteristic of wilt of the tomato *(Lycopersicon esculentum)* are produced. This disease, which is caused by the filamentous fungus, *Fulsarium lycopersici*, is characterized by gradual or sudden loss of turgor in the leaves and stems, and generally by eventual dessication and death. Internally, the most characteristic symptom is a decided vascular discoloration (necrosis). Precedent to, or accompanying loss of turgor, local or general chlorosis of the leaves often occurs. In fact, at times, when environmental factors, particularly temperature, are not optimal for the pathogen chlorosis, and resultant retarded or arrested growth of the plant are the outstanding or only external symptoms, so that the disease resembles a show blight.

The pathogen lives primarily in the xylem elements of the host. Following the early work of Smith,[1] who established that wilting in bacterial wilt of cucurbits is essentially due to a clogging of the vessels, it has been assumed quite generally that in such vascular mycoses as tomato wilt and cabbage yellows, the parasite affects the

host by clogging the water conducting elements and thus mechanically interfering with the water supply of the transpiring top of the plant. A considerable body of observational and experimental data, especially by Clayton,[2] suggests that this hypothesis alone is not adequate. A different, or an additional possibility is that the fungus produces or induces the production of substances which exert a slow and progressive, or a sudden and rapid toxic effect on the cells of the host.

In these experiments we were especially concerned in testing the validity of the second hyphothesis. Pure cultures of the fungus were grown for various lengths of time in various volumes of a synthetic medium which consisted of 1,500 cc. of H_2O, 50 grams of $C_{12}H_{22}O_{11}$, 10 grams of KNO, 5 grams of KH_2PO_4, 2.5 grams of $MgSO_4$, and a trace of Fe_2Cl_3. The fungus was filtered off and the filtrate passed through Berkefeld filters. Next, vigorous tomato plants were cut off just above the soil and placed in the culture filtrate and in aqueous dilutions (1-1, to 1-16) of the culture filtrate. Forty plants were thus set up, each in a separate test tube. The controls consisted of tops placed in (1) the nutrient medium, (2) distilled water, and (3) tap water. Fifty plants were used as controls. Each of the control plants in the nutrient medium remained unchanged and some of those in distilled and in tap water even developed adventitious roots. All of the plants placed in the culture filtrate developed typical symptoms of tomato wilt within varying lengths of time depending upon the concentration of the culture filtrate and the age of the culture from which the filtrate was derived. In general, with a given volume of culture medium, the older the culture the more rapid the yellowing and wilting. Furthermore, no wilting was obtained when the culture filtrate was diluted with more than 4 volumes of distilled water.

Next a series of experiments was run to determine whether plants of wilt-resistant varieties would be affected by the toxic culture filtrate. Plants of both wilt susceptible (John Baer, Bonnie Best) and wilt-resistant varieties (Marbanna, Marbellosa, and Norton) were placed in the filtrates. Forty plants of the former varieties were used and 30 of the latter. The resistant varieties wilted as readily as the susceptible ones.

To determine the effect of the toxic element upon plants with unbroken root systems the filtrate was applied to the soil of plants grown in garden soil and in pure quart sand. Walter culture plants also were arranged so that the root systems were immersed in the culture filtrate. In the controls, nutrient medium was substituted

for the culture filtrate. After 48 hours the potted plants, to which the culture filtrate had been added, had wilted. The controls were healthy even on the fifth day. The seedlings grown in water cultures wilted in 48 hours after transfer to the culture filtrate while those transferred to the nutrient medium remained healthy.

To determine whether the killing effect of the culture filtrate is specific for the tomato plant, cuttings of the following plants were placed in the culture filtrate: Selaginella sp., cotton, sunflower, garden bean, and mustard. All of these wilted in 48 hours.

The toxic element is not destroyed by prolonged boiling of the culture filtrate. It is not carried over in steam sterilization. A water extract of the residue from complete evaporation of the filtrate on a water bath, is still toxic.

It is apparent from these experiments that *Fusarium lycopersici* produces a thermo-stabile toxic element *in vitro* which, when introduced into tomato plants, produces typical wilt symptoms. Studies of the nature of the toxic element, of the mechanism of its effects on the host, and of other related problems are in progress.

[1] Smith, E. F., Bacteria in Relation to Plant Diseases, Vol. II, 1911.

[2] Clayton, E. E., *Am. J. Bot.*, 1923, x, 71-88.

3472

Organic Chlorides of Tissues and Possible Relation to Gastric Hydrochloric Acid Formation.

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Among the current theories for the chemistry of the gastric hydrochloric acid mechanism, there are none which allow for the production of the acid without a coincident *local* formation of an equivalent amount of alkali. Since there is no evidence that gastric tissue becomes alkaline during acid secretion, and since it would require an enormous concentration of chemical energy to separate HCl from an alkaline component, it seemed reasonable to develop a theory of gastric acidity production which would not labor under this assumption of local alkali formation. A chemical reaction which seems particularly probable in this connection is the hydrolysis of an alkyl halide, with the production of hydrochloric acid and the corresponding alkyl alcohol. $RCl+HOH \rightarrow HCl+ROH$. The