so constant as it has been assumed to be, but that it is affected by a number of conditions, and that at least some of these may not affect length and spread in the same manner. Attention has been called to these facts because they have not been given adequate consideration in genetic research on the behavior of flower size in *Nicotiana* and other genera.

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[1] Setchell, Studies in *Nicotiana*, I, Univ. of Cal. Pub. Bot., **5**, 8, 1912.
[2] Ibid., p. 29.

RETENTION IN THE CIRCULATION OF DEXTROSE IN NORMAL AND DEPANCREATIZED ANIMALS, AND THE EFFECT OF AN INTRAVENOUS INJECTION OF AN EMULSION OF PANCREAS UPON THIS RETENTION

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The content of dextrose in the circulating blood of normal animals is almost constant, it amounts to about 0.1 percent. The carbohydrates of the foodstuffs form the main source of dextrose in the body. On their way from the digestive tract the carbohydrates are transformed into various forms of saccharides; but all are finally converted largely into glycogen, which is stored up mostly in the liver. The blood obtains its supply of dextrose from the glycogen of the liver, and distributes it among the tissues of the body according to their demand for it. In the normal animal none of the dextrose escapes through the kidneys. Accordingly the constancy of the amount of dextrose in the blood is regulated by a mechanism which controls either of the two factors; the *supply* of or the *demand* for it.

In diabetes the dextrose content of the blood is higher than normal, is variable in amount, and, when it is sufficiently high, dextrose escapes through the kidneys. The cause of the increase of the blood dextrose, or hyperglycaemia, may be found either in a decrease in the demand of the tissues for dextrose, that is, the tissues burn dextrose less readily than in normal conditions; or in an increase in the supply, that is, the liver supplies the blood with more dextrose than in normal conditions.

Both possibilities have groups of followers who have brought forward numerous experiments in support of either of the respective theories.

It was experimentally established years ago that the complete removal of the pancreas brings on a severe hyperglycaemia, leading to considerable glycosuria and to the death of the animal. Here we are confronted again with the question, whether the presence of the pancreas in the normal animal favors the burning of the dextrose by the tissues, or whether it restricts the supply of dextrose to the blood by the liver. But there also arose another question, namely, whether the effect of the removal of that gland was due to a removal of a controlling substance, or, whether it was due, in consequence of the operation, to an interference with the nervous mechanism which controls the supply of the dextrose. In case of the first alternative, namely, that the resulting hyperglycaemia is due to a removal of some substance, normally secreted by the pancreas, we have to assume that that substance belongs to that group of substances which are provided by glandular and other tissues for the proper maintenance of functions of the animal body, i.e., *internal secretions*, since a complete suppression of the external secretions of the pancreas never leads to hyperglycaemia or diabetes.

We wish to report briefly results of several series of experiments which are likely to shed some light upon these questions. Most of our experimental series have one feature in common, namely, that the blood was provided with dextrose directly and not by way of the liver. In one series of experiments large quantities of dextrose (20 cc. of a 20% solution of dextrose per kilo body weight) were injected intravenously into normal dogs. It was found by us, as it was found by some investigators before us, that the injected large surplus of dextrose disappeared quite rapidly from the circulation. *Ninety minutes after the infusion, the dextrose content of the blood reached nearly the same level which it had before the injection.* About one-half of the injected quantity of dextrose left the circulation through the kidneys; the other half went into the tissues. In nephrectomized animals also the dextrose returned to the previous level in the same length of time. *Here we have a striking example of the ability and readiness of the circulation of a normal animal to rid itself of a large surplus of dextrose.*

In another series of experiments similar injections of dextrose were made into animals, the pancreas of which was removed by an operation a day or two before the dextrose infusion. The sample of blood taken from the animal before the intravenous injection of dextrose was strongly hyperglycaemic. It was then found that in these experiments *the dextrose content of the sample of blood, which was taken ninety minutes after*

the end of the dextrose injection, was at least twice as high as that of the sample taken before the injection. Apparently the removal of the pancreas interfered with the power of the circulation to rid itself readily of a surplus of dextrose. The possible ability of the liver to provide the circulation with a greater supply of dextrose in the absence of the pancreas can certainly be no important factor in these results. Our experiments show, then, *that in the presence of the pancreas the circulation rids itself easily of intravenously introduced dextrose; but that it is unable to do it satisfactorily in the absence of the pancreas.* On the basis of these facts is it not plausible to assume further that the pancreas exerts exactly the same influence, when the dextrose in the circulation is being supplied in some other manner than by a burette into a vein, for instance, by the liver through its connections with the circulation? Or, in other words, do not our experiments make the assumption plausible that pancreatic hyperglycaemia is due to the fact that *in the absence of the pancreas the circulation is unable to dispose properly of the dextrose which it receives in normal amounts from the liver?*

In a third series, the experiments were again made by injecting dextrose intravenously in depancreatized dogs. But in these cases a strained pancreatic emulsion was added to the dextrose solution, and in some instances the infusion of the pancreatic emulsion was continued for some time after the infusion of the dextrose was finished. We present here merely a preliminary communication; we have not yet mastered all the details of this part of the investigation. But there seems to be no doubt about the nature and validity of the main result, which is this: In such experiments, *ninety minutes after the end of the dextrose infusion, the dextrose content of the blood is again at about the same level as it was before the infusion of the dextrose.* Apparently the presence of a pancreatic emulsion within the blood helps the circulation to get rid of the surplus of dextrose injected intravenously. In these experiments it is evident that the *effect of the pancreas can be only of a chemical and not of a nervous nature,* or, in other words, it is the internal secretion of the pancreas which helps the circulation to get rid of the surplus of dextrose.

To the above results we wish now to add the following preliminary statement derived from three experiments. *In depancreatized animals the blood of which has shown a marked hyperglycaemia, an intravenous infusion of a pancreas emulsion brought the dextrose content of the blood to 0.09% and even 0.08%.* The hyperglycaemia returned next day and could be reduced again to the normal level by an infusion of pancreas emulsion.

Conclusions.—In normal animals the circulation possesses the ability to get rid readily of a surplus of dextrose injected intravenously. In the absence of the pancreas this ability of the circulation is impaired. This ability can be temporarily restored by an intravenous injection of a pancreas emulsion.

Furthermore, an intravenous injection of a pancreas emulsion is capable of reducing the hyperglycaemia due only to depancreatization to a normal level of the dextrose content of the blood.

As to the nature of the factors which may constitute the ability or inability of the circulation to get rid of a certain degree of surplus of the dextrose content of the blood, we are not willing to discuss it at this stage of our investigation. We are rather bent upon seeing how many more facts we shall be enabled to bring to light on the basis of the hypothesis which set us to work on these problems.

PARTHENOCARPY AND PARTHENOGENESIS IN NICOTIANA

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The occurrence of parthenogenesis in the genus *Nicotiana* has, in general, been considered to be a negligible factor so far as the interpretation of breeding experiments with tobacco is concerned. The only outstanding instance in which castrated or mutilated tobacco flowers have yielded viable seed is to be found in a report describing the experiments of Mrs. R. H. Thomas.[1] Her experiments, apparently conducted with due regard to the various sources of error, indicate that for her cultures parthenogenesis in various species and hybrids of *Nicotiana* is of frequent occurrence. Conflicting evidence is furnished by the experiments of others. Thus, Howard,[2] following experiments which involved the emasculation of over 5000 flowers on many strains of Indian Tobacco, found but five capsules containing seed and in only two cases was it shown that this seed was viable. Hartley[3] obtained two capsules of seed 'by treating fully receptive stigmas with magnesium sulphate,' employing in his experiments flowers of 'Cuban Tobacco (*Nicotiana Tabacum*).' The seeds thus produced proved, however, to be nothing more than empty shells. Further, East[4] and Wellington[5] claim that hybrid seed, produced by crosses between certain species of *Nicotiana*, has given plants 'like the mother species and also true hybrids,' plants 'like the mother species and no true hybrids,' and that this seed gave 'no true hybrids on one occasion but did produce true hybrids on other occa-