implication of a genetic relation in the series of abnormalities affecting the hands and feet and the definite extension of the developmental disturbance well beyond the site of obvious deformity. The connection between the anatomical abnormalities and the functional disorders in these animals is uncertain. Further studies may clarify these several relationships.

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THE REDUCING POWERS OF PHYSIOLOGICALLY IMPORTANT CARBOHYDRATES

IN view of the increasing attention paid to the rôle of the physiologically important sugars in body economy, it seemed worth while to determine the relative and actual reducing values of these sugars, using the newer techniques devised for the determination of glucose.

The sugar methods employed were: Folin's modification of the Folin-Wu method[1]; Somogyi's modification of the Shaffer-Hartmann technique[2]; the revised Folin-Malmros micro-sugar method[3]; the Hagedorn-Jensen ferricyanide method[4]; and the new copper-iodometric method (reagents) of Shaffer and Somogyi.[5]

In this paper are presented a comparison of the reducing values of d-glucose, l-arabinose, d-fructose, d-galactose, lactose (hydrate) and maltose (hydrate).

The sugars employed were all of the highest purchasable purity (Pfanstiehl brand). All the sugar samples were dried in a vacuum desiccator to constant weight and the purity checked by means of the polariscope. All pipettes, sugar tubes and boiling tubes were calibrated. Stock sugar solutions were made by accurately weighing out 75 milligrams of sugar and diluting in retested 50 milliliter volumetric flasks with one-half saturated benzoic acid as a preservative. From these 0.15 per cent. solutions the proper dilutions for the techniques were made.

The determinations were carried out for each method exactly as described for glucose, the glucose reference standard being made up to contain the same weight of material as the solutions of the other sugars.

Since the sugar methods employed were designed primarily for the determination of glucose, the reducing powers of the other sugars are expressed in terms of this carbohydrate as unity.

In Table I is presented a comparison of the relative reducing values of equal weights of the carbohydrates studied, while in Table II are given the results on the basis of equimolecular solutions, as calculated from Table I.

TABLE I

A COMPARISON, TO GLUCOSE AS 1, OF THE RELATIVE REDUCING POWERS OF EQUAL WEIGHTS OF THE CARBOHYDRATES

	Method	Glucose	Arabinose	Fructose	Galactose	Lactose*	Maltose*
Weinbach and Calvin	New Folin-Wu	1	0.65	1.05	0.75	0.41	0.40
	Somogyi-Shaffer-Hartmann	1	0.80	1.03	0.70	0.40	0.38
	Folin-Malmros	1	0.96	0.98	0.82	0.47	0.39
	Hagedorn-Jensen	1	0.87	1.02	0.74	0.67	0.75
	Shaffer-Somogyi, Reagent 50, 1 gm KI	1	0.85	0.96	0.75	0.46	0.34
	Shaffer-Somogyi, Reagent 50, 5 gm KI	1	0.87	1.00	0.76	0.46	0.35

* One molecule water of hydration.

TABLE II

A COMPARISON, TO GLUCOSE AS 1, OF THE RELATIVE REDUCING POWERS OF EQUIMOLECULAR CARBOHYDRATE SOLUTIONS (AS CALCULATED)

	Method	Glucose	Arabinose	Fructose	Galactose	Lactose*	Maltose*
Weinbach and Calvin	New Folin-Wu	1	0.54	1.05	0.75	0.82	0.80
	Somogyi-Shaffer-Hartmann	1	0.67	1.03	0.70	0.80	0.76
	Folin-Malmros	1	0.80	0.98	0.82	0.94	0.78
	Hagedorn-Jensen	1	0.73	1.02	0.74	1.34	1.50
	Shaffer-Somogyi, Reagent 50, 1 gm KI	1	0.71	0.96	0.75	0.92	0.68
	Shaffer-Somogyi, Reagent 50, 5 gm KI	1	0.73	1.00	0.76	0.92	0.70

* One molecule water of hydration.

The order of reducing power in general, for all

[1] O. Folin, *Jour. Biol. Chem.*, 82: 83, 1929.
[2] M. Somogyi, *Jour. Biol. Chem.*, 86: 655, 1930; 70: 599, 1926.
[3] O. Folin and H. Malmros, *Jour. Biol. Chem.*, 83: 115, 1929.
[4] H. C. Hagedorn and B. N. Jensen, *Biochem. Zeitschr.*, 135: 46, 1923.
[5] P. A. Shaffer and M. Somogyi, *Jour. Biol. Chem.*, 100: 695, 1933.

the methods, per equal weight of sugar is *fructose* or *glucose* > *arabinose* > *galactose* > *lactose* > *maltose*. The order of reducing power per molecule is *fructose* or *glucose* > *lactose* > *maltose* or *galactose* > *arabinose*.

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A NEW CANKER DISEASE OF RED PINE, CAUSED BY TYMPANIS PINASTRI[1]

RED or Norway pine (*Pinus resinosa* Sol.) is to-day one of the most important of our native coniferous trees for reforestation purposes. Its silvicultural importance has been largely responsible for its wide use, but its freedom from serious disease has been at least contributory. Therefore, any new disease is of immediate interest and needs thorough investigation.

During the winter of 1932–33 a small area was observed in the Eli Whitney Forest (the watershed property of the New Haven Water Company, New Haven, Conn.) where the red pines were dead and dying. Subsequent studies have shown that the causal organism is *Tympanis pinastri* Tul. A preliminary report of these studies and a brief summary of the more important results are given in this article.

On red pine the disease is characterized by the formation of axially elongated stem cankers with or without definite margins and with depressed centers which become roughened and open after two or three years. The absence of any marked resinosis in or adjacent to the cankered tissue is noticeable. Each canker is centered at a node and always has one or more central branch stubs, indicating that the organism enters the stem at the bases of lateral branches. Because of the absence of cankers on the centrally located branches it appears that the fungus exists there primarily as a saprophyte and grows into the stem and produces cankers only when the host is weakened by some environmental factor. Infection has been found only in southern Connecticut in plantations established from 1916 to 1919.

The same fungus is associated with cankers on northern white pine (*P. strobus* L.), but on this host infection is limited to trees which are greatly weakened through shade suppression, root competition, poor soil or some other similar cause. Occasional cankers on white pine have been observed throughout New England and in New York and Maryland.

The fructifications of *T. pinastri* are glistening black cartilaginous bodies. They occur on practically all cankers on both hosts, but because of their small size—up to 1 mm in height and breadth—they may not be noticed unless one is particularly and closely searching for them. They are of two kinds—ovate or spherical pycnidia on a stromoid base and disk-shaped stalked apothecia. The presence of either is sufficient to identify the organism.

The parasitism of *T. pinastri* on red pine has been definitely established through artificial inoculation experiments. Pure cultures were secured from the fructifications and from inner bark mycelium from both red and white pines. Two hundred and twenty-one inoculations and 35 checks were made on 56 thrifty red pines in May, 1934. Examination of the inoculations in late September showed that small but typical cankers were present in a few cases and that fructifications were present in nearly all cases. At the same time the checks were sterile. The fungus has been reisolated in pure culture from the artificially induced cankers. No attempt has been made yet to inoculate white pines.

Studies now in progress indicate that the disease on red pine is present only in plantations; that it is much more prevalent in pure stands than in mixtures with white pine; that it is not limited to the poorer sites but may occur on the upper crown classes more on poor sites than it does on good sites; that on all sites the lower crown classes are much more susceptible than the upper ones; and that its incidence seems to be definitely correlated with the severe drought of 1930 in southern New England. It is to be expected that another period of infection need not be anticipated until another serious drought occurs.

Further studies of this disease are now under way. The writer would appreciate any information concerning diseased red pine trees or stands. Collections of *T. pinastri* or of closely related fungi on coniferous hosts are also requested.

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