Table 1. Percentages of cotton seedlings with red hypocotyls produced from seed obtained at three pickings made at biweekly intervals.

| Variety | Picking | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| *Upland cotton* | | | |
| Empire | 75 | 77 | 62 |
| Acala 4-42 | 33 | 40 | 33 |
| *American-Egyptian cotton* | | | |
| Amsak $F_{18}$ | 88 | 77 | 85 |
| Pima S-1 | 75 | 78 | 66 |

the extent of bud shedding and the consequent delay of flowering, in the duration and regularity of pollen suppression, and in the promptness of the male-sterility response. The presence of pollen, even when the anther opening appears normal, has been found to be an uncertain criterion of pollen viability. Anthers of treated plants sometimes remain unopened in the forenoon but shed pollen in the afternoon. One of the compounds suppressed the pollen of one of the varieties into the ninth week but that of another only for a short period. A measure of response has been obtained by applying several of the materials to the soil. Wetting agents have been tried in some tests, but they appear to hasten absorption and increase burning.

In searching for means of reducing toxicity and extending the male-sterile reaction, the idea is now prominent that a first and second spraying with lower dosages would be effective. A second spraying could be carried out if the rows were spaced further apart than is practiced in commercial cotton production. Where there is a substantial difference in the threshold concentrations of the desired parents, several field-wide sprayings become possible if the most reactive variety is used as the female parent and the less reactive variety as the male parent.

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5. The initial greenhouse tests reported in this paper were made by me as part of cooperative investigations between the Field Crops Research Branch, Agricultural Research Service, U.S. Department of Agriculture, and the Texas Agricultural Experiment Station, College Station, Tex.
6. The sodium α,β-dichloroisobutyrate was received from Rohm and Haas Company in 1955.

9 September 1957

## Alkaline Denaturation of Hemoglobin of Postlarval and Adult Scorpaenichthys marmoratus

The hemoglobin of the bullfrog tadpole is different from that of the adult (*1*). Since the teleost fish *Scorpaenichthys marmoratus* undergoes an extensive postlarval metamorphosis (*2*), an investigation of the alkaline denaturation of the hemoglobin of three adult and nine postlarval specimens of this fish has been undertaken in order to find out whether an ontogenetic change in the hemoglobin is associated with the metamorphosis (*3*). The rate at which oxyhemoglobin is converted to alkaline methemochromogen through denaturation at *p*H 11 to 13 has been a standard technique in the differentiation of adult and fetal mammalian hemoglobins (*4, 5*).

Adults and postlarvae were bled from the heart into heparinized Ringer's solution (*6*); the erythrocytes were washed three times in an excess of nonheparinized Ringer's solution. Centrifugally packed erythrocytes were lysed in distilled water (9 volumes of water to 1 volume of cells). Stromata were removed by prolonged centrifugation. The preparation of the hemoglobin was done at 0° to 1°C. No bloods were pooled. The hemoglobin solutions were used immediately for alkaline denaturation in sodium phosphate buffer (*p*H 11.0 to 12.0; Γ/2, 3.0), the reaction being followed spectrophotometrically (*7*).

Some of the results obtained at *p*H 11.0 are shown in Fig. 1. Similarly to the hemoglobin in several mammals—but not in man (*4, 5*)—*Scorpaenichthys* postlarval hemoglobin has an initial alkaline labile component that denatures faster than that of the adult hemoglobin. In addition, the diphasic nature of the alkaline denaturation curve is more obvious in the case of postlarval hemoglobin. The relative proportions of fast- and slow-denaturing components vary in postlarval hemoglobin much more so than in adult hemoglobin; similar variation has been described by others for human adult hemoglobin (*5, 8*). The difference in alkaline denaturation between adult and postlarval *Scorpaenichthys* hemoglobins was consistently observed at both *p*H 11.0 and 12.0. Hence, there is a change in the biochemical nature of the hemoglobin in the development of *Scorpaenichthys* as in mammals (*5, 9*), the chicken (*10*), the terrapin (*11*), and the bullfrog (*1*).

In general, the oxygen tensions to which adult fishes and their pelagic young are subjected are approximately the same (150 mm-Hg)—a situation in contrast to that observed for most other vertebrate embryos and fetuses. Therefore, at present, a particular physiologically significant role cannot be assigned to the equivalent of a fetal hemoglobin in *Scorpaenichthys*. The occurrence of a distinct postlarval hemoglobin in *Scorpaenichthys* may represent the chance evolutionary development of a biochemical feature of little selective value. In fact, some larval and postlarval fishes—for example, the leptocephalus of the eel (*12*)—lack hemoglobin. Preliminary experiments on alkaline denaturation indicate the presence of a fetal hemoglobin in the live-bearing surf-perch *Embiotoca*

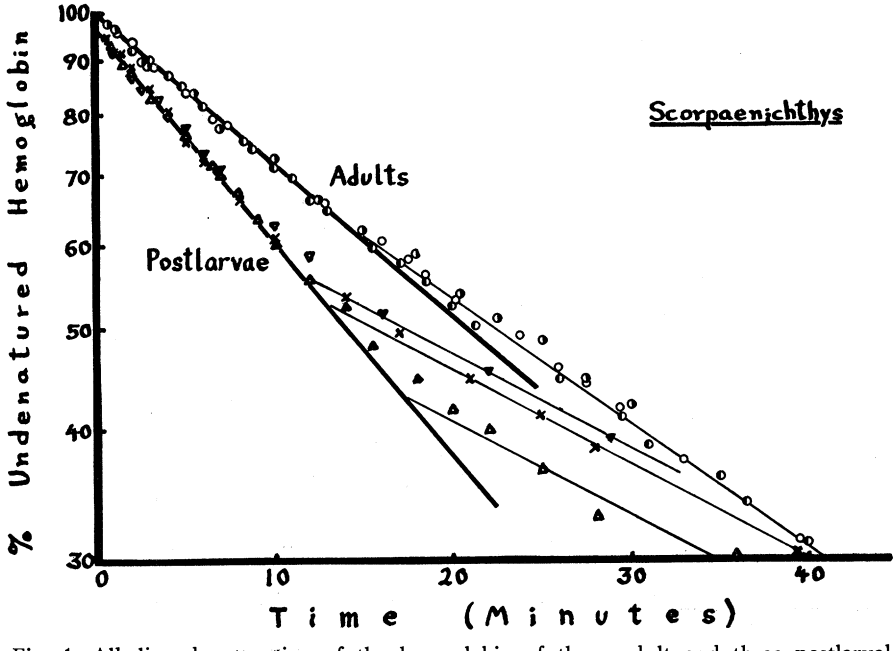


Fig. 1. Alkaline denaturation of the hemoglobin of three adult and three postlarval *Scorpaenichthys marmoratus* (*p*H 11.0; Γ/2, 3.0; 24°C).

*lateralis*; certainly in this viviparous fish it would be reasonable to assume that the function of a fetal hemoglobin would be the same as that accepted for the fetal hemoglobins of mammals (*9*).

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## Effect of Digestion on Distribution of Blood Flow in the Rat

The belief that splanchnic blood flow increases at the expense of flow in other organs during digestion was challenged by Herrick *et al.* (*1*) in 1934. By use of thermostromuhrs, dogs were found to show similar increases in carotid, femoral, and superior mesenteric arterial blood flow postprandially. Abramson and Fierst (*2*) found that the blood flow to the human hand, forearm, and leg tended to increase after eating. A meal increases the splanchnic blood flow of a human subject (*3*). However, the cardiac output also increases (*4*). When the experimental values are adjusted for surface area, it is found that the absolute postprandial increase in splanchnic blood flow in a 1.73 $m^2$ man is about 710 ml/min. A 24 percent increase in the cardiac output of such a man (*5*) represents 1300 to 1400 ml/min. From this it may be concluded that the nonsplanchnic blood flow is increased by digestion.

The subject has been reinvestigated (*6*) with the aid of a newly developed method (*7*). The method is based on the observation that all organs other than the brain have, during the first minute after a single intravenous injection of $K^{42}Cl$, substantially the same extraction ratios for $K^{42}$. The fractional distribution of $K^{42}$ among the organs during the first minute therefore corresponds to the fractional distribution of the cardiac output. The anomalous behavior of the brain has been shown to be of minor consequence in the measurements of the blood flow to other organs. Values obtained by this method describe the fractions of the cardiac output directed to each organ. A knowledge of the cardiac output permits the calculation of the blood flow to each organ.

One hundred and seventeen rats were used. Control animals were starved for 24 to 72 hours but were permitted to drink water *ad libitum*. "Fed" animals were allowed to eat and drink *ad libitum* up to the time of the experiment. The gastrointestinal tract of the "fed" animals always contained 10 to 15 g of food at autopsy. The animals were anesthetized with Nembutal (40 mg/kg intraperitoneally). The cardiac output was determined by dye dilution, with Evans blue as the indicator; the blood was sampled at a rate of 90 collections per minute (*8*). Other similarly treated animals of the same stock were used for the fractional distribution studies with $K^{42}$; the details of the method have been described previously (*7*).

The cardiac output of 17 control animals averaged $172 \pm 38$ ml/kg min. Eleven fed animals had a cardiac output of $223 \pm 59$ ml/kg min. Determinations of fractional distribution were made on 49 control and 40 fed animals. The fractions found for each organ were multiplied by the cardiac output value in animals of the same group (adjusted for body weight) to give blood flow values to the various organs.

Table 1 shows the blood flow values obtained in the organs of the two groups. For simplicity all values have been adjusted for the body weight and are presented as the blood flow to the organs of a 250-g rat.

It is clear from these results that, during digestion, there is a uniform increase in the blood flow to all organs of the rat. The splanchnic organs do not gain their increased blood supply at the expense of the blood supply to other organs; on the contrary, all organs benefit from the increased cardiac output associated with digestion.

These results, though obtained in anesthetized rats, are similar to those reported in conscious dogs and men; they do not conflict with any reported findings. In the absence of contrary evidence, it is suggested that the prevailing concept that digestion results in diversion of blood flow from other organs to the digestive tract be critically re-examined.

Table 1. Blood flow values in fasting and fed rats (all values have been adjusted to 250-g rats; blood flow is given in milliliters per minute).

| Organ | Blood flow | |
|---|---|---|
| | Fasted | Fed |
| Liver (arterial) | 3.2 | 4.3 |
| Gut and spleen | 7.1 | 9.4 |
| Myocardium | 1.1 | 1.3 |
| Skin | 3.2 | 4.2 |
| Kidneys | 6.6 | 8.7 |
| Carcass | 21.8 | 27.9 |

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## Activation of Enzymatic Hydrolysis of Benzoylcholine by Tryptamine

During an investigation of the anticholinesterase activity of indole derivatives (*1*) it was found that tryptamine accelerates the enzymatic hydrolysis of benzoylcholine by plasma cholinesterase (*2*). It has also been reported that analgesics (*3*) and other compounds (*4*) activate plasma cholinesterase, and in certain cases, red cell cholinesterase (*5*). Some authors attributed this activation to an interference wth the partial inhibition of the enzyme (E) by the excess