Rare Chemicals of Armour Research Foundation, Chicago, are sodium alpha naphthalene sulfonchloramide, 3-p′-tolueneazo-p-cresol, 3-m′-tolueneazo-p-cresol, 2,3,4- or 2,3,5-trimethyl pyrrole, thorium iodide, o- or p-tolylhydrazine hydrochloride, 2,2,3,3-tetramethyl butane, triphenyl aluminum or antimony, triethyl antimony, tin or thallium, tetraethyl or tetrabutyl tin, d-arabo ascorbic acid, arachidonic acid, allyl cyanamide, 1-allyl piperidine, boroethane, 1-benzyl piperidine or morpholine, bilobol, coenzyme II, 5,6-cyclopenteno-1,2-benzanthracene, and masurium or its compounds.

DISCUSSION

PIGMENT AND BIOCHROME

MY colleague, Professor Fox, has dragged into the open a feud which has been smoldering between us for many years.[1] While the mores of our species, both as to the use of words, and the use of pigments themselves, are little influenced by arguments, it has seemed to me worth while to put some of my contentions in this field on record again.

I have long been irked by some of the prevailing uses, in biology, of the word "pigment." Dr. Fox admits the force of these objections, in part, though he insists upon retaining this word as a convenient vernacular (?) term. For more accurate scientific usage, he has adopted the suggestion of a professor of Greek, and substituted a new word "biochrome."

Whether it is reasonable to set up, as a special class, all the colored substances which can be extracted from animals or plants, in contrast with those substances which do not happen to be colored, is decidedly debatable. However, if we grant the desirability of such a term, "biochrome" would seem to meet the requirements pretty well. That "pigment" does not do so, I have already argued.[2] For this word has a definitely functional connotation, which we can hardly escape. In the inorganic world, colored substances are "pigments" only in so far as they are used as such. Thus lead chromate is (or may be) a pigment; copper sulfate never is. Save for an unfortunate precedent, I can see no excuse for applying any different criterion in the organic world. Why every colored substance, derivable in any way from a living organism, should forthwith become a "pigment" is difficult to understand.

Needless to say, I am offering no general protest against the use of the word "pigment" in biology. I have used the word freely, in connection with my own studies and shall continue to do so. But I believe that the word, when transferred to the organic world, should retain its functional significance. There is no propriety in calling a substance a "pigment" except in so far as it is used by the organism to influence its color scheme. I trust that it is no longer necessary to insist that the *appearance* of an animal is at times one of its important biological assets. Thus, melanin, the carotenoids, guanine and some other substances, occasionally even hemoglobin, may play the functional role of pigments. In so saying, let us repeat, we are not thereby assigning these substances to a definite physical and chemical category.

My chief protest against current usage in this field relates to the expression "respiratory pigment." We are here combining words belonging to two utterly different vocabularies. It is like talking about a "locomotor enzyme" or an "invertebrate catalyzer"! Dr. Fox, in his recent communication to SCIENCE, has given some theoretical reasons for believing that the same features of molecular organization which give to certain substances their color may likewise render them available as oxygen carriers. But even if this association between these attributes should prove to be true, we should hardly be justified in such a bit of semantic miscegenation as we have in "respiratory pigment"! Nor would "respiratory biochrome" be much better.

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1 SCIENCE, November 24, 1944.
2 *Scientific Monthly*, April, 1937.

LATENT VIRUSES IN STONE FRUITS[1]

RESEARCH workers in the field of stone-fruit viruses have become aware of the presence of latent or hidden viruses or virus complexes in cherry trees. These viruses have been demonstrated by placing apparently healthy sweet or sour cherry buds in various peach varieties, in which case the inoculated peach tree becomes dwarfed, rosetted, with split or cracked bark, ring spots in the foliage or various combinations of the above symptoms. Some trees recover after the initial shock, while others die, apparently depending on the strain or variety of virus present. The more severe strains are easily detected on peach, but the mild strains may cause only a slight dwarfing, with rapid recovery, or may show only an occasional leaf with ring spotting. The Kwanzan and Shirofugen

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varieties of *Prunus serrulata* are proving to be much more reliable test plants for these viruses. When sweet or sour cherry buds having the latent virus complex as it occurs in Oregon are budded into these flowering cherries, a severe reaction occurs.

Kwanzan budded in August with sweet cherry buds containing virus, instead of producing normal growth the following spring, develops small curled leaves with split necrotic veins, which form tight rosettes with very little or no stem elongation. Small nursery trees may be killed, or may produce new growth only below the point of bud insertion. Often the sweet-cherry bud grows and develops in a normal manner and may be the only living branch on the trunk.

Buds from the same source placed into Shirofugen produce an entirely different reaction. If the buds are inserted in August, the area immediately around the bud becomes necrotic and the bud and budding rubber become embedded in a mass of black gum by fall. By spring the stem is completely girdled for 5 or 6 inches, but the foliage and new growth develop in the normal manner. About the time the first leaves have expanded to full size, entire spurs of foliage near the canker suddenly turn brown and die with the leaves still attached. This may gradually spread up one side of the branch or may move on all sides of the branch. As soon as the weather becomes warmer and drier, the entire branch beyond the point of bud insertion suddenly dies. Many of the branches now break over at the point of bud insertion, because this area has become much constricted. The necrosis spreads slowly down from the point of bud insertion and out into the laterals, causing cracking, constrictions and gumming, especially on the new or current-season lateral branches.

When Shirofugen is budded after growth starts in the spring, a similar reaction takes place. Necrosis occurs about the bud insertion and eventually girdles the stem. By fall the canker extends 2 or 3 inches each way from the point of bud insertion, but no symptoms appear on the foliage, until suddenly the entire branch dies. The stem is constricted below the canker, and gum is forced out in tendrils over the necrotic area.

Buds have been taken from several different trees of Bing, Lambert, Royal Ann, Black Republican, Black Tartarian and Montmorency sour cherry and placed on Kwanzan and Shirofugen. Of all the trees tested, only one tree of Bing and one tree of Black Tartarian have failed to give a positive test on these two flowering cherries. Preliminary tests with these two sweet cherry trees have also given negative tests for virus on Elberta peach and Mahaleb seedling. A much more extensive test is now under way to determine if these two trees are free from all known virus.

It is hoped that by testing enough trees one virus-free tree of each of the standard commercial varieties may be found that will serve as a foundation for future nursery stock.

There is some indication that some peach varieties may also carry a somewhat different latent virus that produces a local canker effect on Shirofugen.

A more complete paper on the above subject is being prepared.

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PENICILLIN SODIUM TREATMENT OF EXPERIMENTAL TRYPANOSOMIASIS OF MICE

THE following preliminary report offers the results of the experiences carried out in order to test sodium penicillin[1] against *Trypanosoma cruzi.*

Two groups of six mice each, weighing about 25 grams each one, were inoculated 33 and 16 days prior to the treatment with the same strain of *Trypanosoma cruzi* kept in our institute by successive passages through dogs.

The total individual dose administered was of 250,000 and 500,000 Oxford units per kilogram to 4 mice of each group. The infection in the 2 untreated mice served as control. The calculated individual dose of sodium salt of penicillin in 0.1 or 0.2 cc of saline solution was given intramuscularly five times daily at 3-hour intervals and twice at night with a 6-hour interval. The entire therapy covered a period of 84 hours.

Parasite observations were made 24 hours after the initial dose and every two days afterwards, for 10 days. The results of the therapy were negatives; both the treated and the untreated mice showed practically the same amounts of trypanosomes in the blood during the treatment and thereafter.

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A NEW QUARRY FOR JURASSIC DINOSAURS

A LARGE deposit of well-preserved dinosaur bones, heretofore undescribed, occurs in the Morrison formation, about 8 miles east of Cleveland, Emery County, Utah. The date and circumstances of original discovery are unknown, but the first systematic investigations were carried out by parties of University of Utah students, who obtained much excellent material. In 1938 the writer brought this deposit to the attention of Dr. G. L. Jepsen, professor of vertebrate

[1] The sodium penicillin was kindly supplied by Winthrop Products, Inc., through the courtesy of Laboratorios Winthrop Ltda., Chile.