centrifuged, washed, dried, weighed, and analyzed for radioacitvity. The dead cells of *Chlorella,* with no added salts and at all levels of potassium (i), were found to concentrate Cs^{137} by a factor varying from 35 to 68; the variations appeared to be random. In mixtures containing stable cesium, (ii) and (iii), the concentration factor varied around 1—that is, the algal cell bodies contained the same amount of Cs^{137} as did an equal amount of the medium.

These results suggest that structural components persist in dead *Chlorella* which adsorb cesium from very dilute solutions and that this adsorption is not affected by the concentration of potassium in the medium. From these data it may be inferred that, in killed cells of *Chlorella,* potassium and cesium behave independently, but that in live cells of *Chlorella* and *Euglena,* particularly at tracer levels of potassium, ions of potassium and cesium form a metabolic pool.

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23 September 1957

Bronchodilator Action of the Anticoagulant Warfarin Sodium

During the administration of warfarin (Coumadin) sodium, or 3-(α-acetonylbenzyl)-4-hydroxycoumarin sodium, to patients with coronary thrombosis and other forms of thromboembolic disease, Livesay (*1*) noticed improvement in the asthmatic condition of several patients who had bronchial asthma in addition to the thromboembolic involvement. This observation prompted us to look for a direct bronchodilator action of warfarin sodium. For this purpose we employed the isolated guinea pig tracheal chain (*2*), a method which has been used extensively in pharmacological studies and which has been found to correlate well with clinical bronchodilator activity.

The results are shown in Table 1. Aminophylline, a well-established clinical bronchodilator, was used as a reference standard for the kymographic recording of relaxation or dilatation of the uncontracted tracheal chain. The lever system was adjusted so that 5 mg of aminophylline in the 100-ml bath, or a bath concentration of 0.05 mg per milliliter, produced a fall of about 2 cm on the kymograph tracing. Warfarin sodium was indeed found to possess some tracheodilator activity, being about 50 percent as active as aminophylline. By comparison, two different commercial samples of heparin sodium were found to be very weak, only about 5 percent as active as aminophylline. Since warfarin sodium and heparin sodium are sometimes injected simultaneously to secure the immediate anticoagulant effect of heparin and to initiate the slower but more prolonged effect of warfarin, a combination of equal amounts of the two drugs was tested on the tracheal chain. The tracheal dilatation was again approximately 50 percent that of aminophylline, demonstrating that the dilator effect of warfarin sodium was not influenced by the simultaneous presence of the heparin.

Table 1. Tracheodilator potency of anticoagulants, as compared with that of aminophylline.

Source	Bath concn. for 2-cm lever fall (mg/ml)	Approximate tracheodilator potency
Aminophylline		
10-ml ampule	0.05	100
Warfarin sodium		
Powder	0.1	50
Warfarin sodium		
3-ml vial	0.1	50
Heparin sodium–A		
10-ml vial	1.0	5
Heparin sodium–U		
10-ml vial	1.0	5
Warfarin sodium plus heparin sodium–U		
Vials—equal wts.	0.1 plus 0.1	50

It has been pointed out (*3*) that some of the beneficial action of anticoagulants in myocardial infarction may be due to properties other than that affecting coagulation. The initial dose of warfarin sodium is usually 75 mg by intravenous, intramuscular, or oral administration, as compared with an aminophylline dosage of 250 to 500 mg intravenously or intramuscularly for emergency bronchodilatation and 100 to 250 mg orally for nonemergency use. If the dilator effect demonstrated on the tracheal chain is reflected in a corresponding bronchodilatation in man, it seems possible that the initial injection of warfarin sodium may produce some immediate bronchodilatation as well as initiating the slower anticoagulant action. Perhaps prolonged administration of small oral maintenance doses might also account for a bronchodilator effect by the same mechanism.

It is interesting to note, in this connection, that a coronary dilator action has been reported in dogs following intravenous injection of the disodium salt of bishydroxycoumarin (Dicumarol) (*4*) and of solubilized ethyl biscoumacetate (Tromexan) (*5*). Owren (*6*) has noted an improved effort tolerance from long-term anticoagulant therapy in patients with angina pectoris. The coronary dilator activity of warfarin sodium is yet to be investigated.

Studies will be extended to other anticoagulants to determine whether the observed warfarin sodium tracheodilatation or bronchodilatation is a general property of 4-hydroxycoumarin anticoagulants.

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5 September 1957

Action of Selected Redox Substances on Bacterial Bioluminescence

Bioluminescence in *Achromobacter fischeri* and other luminous bacteria (*1*) depends upon a series of electron transfer reactions. The demonstration by Strehler (*2*) of bioluminescence in cell-free extracts of *A. fischeri,* and subsequent studies on the properties of this system, reviewed by McElroy and Strehler (*3*), showed that its essential components are reduced flavin mononucleotide ($FMNH_2$), a higher fatty aldehyde, from C_6 to C_{16}, atmospheric oxygen, and an extract of bacterial enzymes. Substrate and phosphopyridine nucleotide-specific dehydrogenase, reduced di- or triphosphopyridine nucleo-