direct effect of the X-rays on the malignant lymphocytes and may be explained by assuming that some malignant lymphocytes are destroyed by irradiation, the length of life varying inversely with the dose. In most of our experiments irradiation of mice after inoculation of stock susceptible to lymphomatosis did not decrease the number of successful inoculations. In a small number of experiments a decrease of successful inoculations occurred and since the number of successful inoculations is proportional to the quantity of injected cells, it may be assumed that X-rays have destroyed some malignant lymphocytes.

The results of these experiments indicate that X-rays lower the resistance of mice to malignant lymphocytes and suggest that X-rays diminish the number of inoculated leucemic cells but seldom if ever destroy all the cells that have been introduced.

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A Secondary Bio-Electric Effect of Potassium.

L. R. BLINKS.

From the Laboratories, Rockefeller Institute for Medical Research.

The usually large bio-electric effect of potassium salts is often ascribed to the high mobility of K^+ ion in the protoplasmic surface It sometimes appears to be more complex, however, involving the mobility effect as an initial stage, with a secondary effect due to the deeper penetration of potassium. The two steps are often fused into an almost continuous curve in *Nitella,* but are more clearly distinguished in the closely related *Chara coronata.* In the cells of this plant the initial effects of KCl and NaCl are almost identical. The P. D. across[1] the protoplasm (100 to 150 mv. outside positive) is quickly reduced 40 or 50 mv. when tap water is replaced by 0.01 M NaCl or KCl, and then remains at this level for some time. During this time the polarization response remains about as quick and as large as in tap water, so that the effective resistance is high (*e. g.,* 250,000 ohms) to small direct currents passing in either direction across the protoplasm. If an outward current be increased beyond the threshold of stimulation, or if an action current occurs spontaneously, the P. D. quickly falls to about zero. The polariza-

[1] For technique see earlier papers in the *J. Gen. Physiol.*

tion response also disappears, so that the resistance is entirely ohmic and very low (20,000 ohms).

Up to this point the effects of NaCl and KCl are almost identical. In the presence of NaCl recovery begins within a second or 2 and is completed in 5 to 30 seconds, with restoration of the normal P. D. and polarizability. But in the presence of KCl no such recovery occurs: the P. D. remains at about zero and polarization is absent even for rather large currents.

The secondary potassium effect is therefore the inhibition of recovery after an action current. There is increasing evidence that in another plant, *Halicystis,* electrical effects analogous to stimulation occur when the inner surface of the protoplasm is exposed to solutions made alkaline beyond a sharply critical point, recovery occurring when acidity is restored. Outward currents apparently cause this increased alkalinity, inward ones increased acidity. The penetration of ammonia and other bases can cause effects analogous to stimulation and can inhibit recovery.

It is therefore suggested that in *Chara* and *Nitella* the secondary effect of potassium is also to prevent the increase of acidity during recovery from an action current. Its specific prevention of the production of acid (as in iodo-acetic poisoning of muscle) seems unlikely, since potassium is already present in large concentration in the sap. It may simply act by its entrance as a base, neutralizing acids of the cell. This would be expected whether it entered in molecular form (as KOH), or by exchange of K^+ ions for H^+ ions. But the latter seems unlikely since in *Chara,* Na^+ has about the same mobility (initial effect) as K^+ and should therefore exchange as rapidly with H^+. Yet NaCl does not inhibit recovery.

If potassium penetrated very rapidly as base it should itself produce an action current (without recovery); this seems to be usually the case in *Nitella.* In *Chara* (1) it does not enter so fast; (2) the protoplasm may be buffered with more acid; or (3) the stimulation threshold may be at a more alkaline point. The secondary potassium effect is therefore delayed. It may be assisted by an outward current which increases alkalinity; and conversely the potassium inhibition may be counteracted by extremely large currents (*e. g.,* under tension of 0.5 to 1.0 volt) passing inward. The P. D. then rises to high positive values as long as such currents flow, which may be attributed to an increased acidity.

Recovery from the secondary potassium effect occurs slowly when NaCl or tap water is restored. This may be due to the exit of potassium, to the production of more acid by the cell, or to both.