nucleus and cytoplasm. We think that the simplest interpretation of the data is that free K^+ is, indeed, uniformly distributed across the nuclear membrane.

Similar results were obtained by Starodubov and Kurella (*5*) for the salivary glands of *Drosophila*. However, the significance of that study was limited by two technical difficulties. First, the K^+ electrode they used (*6*) was an order of magnitude less sensitive for K^+ over Na^+ than the electrodes used in our study. Second, they used two rather than three electrodes, so that the nuclear membrane resistance was not measured. Thus, the intranuclear position of their electrodes was not rigorously established, and possible damage to the nuclear membrane on impalement was not excluded.

Kroeger *et al.* (*7*) attempted to measure K^+ chemically in samples of nucleoplasm and cytoplasm extracted from *Chironomus thummi* salivary glands with tungsten needles. They stated that the ionic concentrations changed during development, but that the nuclear and cytoplasmic concentrations closely tracked one another, which is consistent with our data.

In other tissues, estimated ratios of nuclear to cytoplasmic K^+ concentrations have ranged from 0.84 to 2.4 (*8*). These values depend not only on the tissue studied and the technique utilized, but also on whether the K^+ concentrations were calculated as millimoles per kilogram of water or millimoles per kilogram dry weight.

Our study differs from those cited above in that ionic activity, rather than concentration, was measured. The two parameters need not be related in a simple manner, insofar as the amount of ion binding and the activity coefficient for K^+ may be different in nucleus and cytoplasm.

The data reported here bear on the possible specific physiological roles that have been suggested for intranuclear ions. Kroeger and Lezzi (*9*) proposed that gene activity may be regulated in part by the nuclear Na^+/K^+ ratio. Allfrey *et al.* (*10*) found that entry of amino acids into the isolated nucleus was facilitated by a high Na^+ concentration in the medium and suggested that the Na^+ content of the nucleus in vivo must be high, possibly because of direct ionic pathways between the nucleus and the extracellular fluid. Siebert *et al.* (*11*) proposed a similar model based on studies of the flux of radioactive ions into rat liver nuclei and cytoplasm.

The nuclear membrane might at least partially control nuclear ionic activities. This concept was supported by the finding of Ito and Loewenstein (*12*) that ecdysone, the insect moulting hormone, induced changes in the permeability of the nuclear envelope in *Chironomus* salivary glands.

In the case of K^+, however, our data demonstrate an ionic equilibrium across the nuclear membrane. This suggests that nuclear K^+ activity is controlled not by the nuclear membrane, but by the plasma membrane, or alternatively, by the putative nuclear-extracellular pathway.

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BCG Inhibition of Melanoma: Specific?

Faraci and Schour studied the effect of pretreatment with bacillus Calmette-Guérin (BCG) on tumor growth (*1*): "Three different malignant tumors—a melanoma (S-91), a mammary carcinoma, and a methylcholanthrene-induced sarcoma—were used in syngeneic BALB/c mice." Pretreatment of BALB/c mice with BCG inhibited growth of the S-91 melanoma, but did not inhibit growth of the mammary carcinoma or sarcoma. In vitro studies were extended in another article (*2*). The results obtained were interpreted as evidence ". . . of a specific BCG-induced protection against malignant melanoma in an inbred strain of laboratory mice."

The S-91 melanoma arose in a DBA/1J mouse (*3, 4*). The DBA/1J and BALB/c strains differ at the major murine histocompatibility locus ($H\text{-}2^q$ and $H\text{-}2^d$, respectively), as well as at minor histocompatibility loci (*3*). Although S-91 is transplantable in allogeneic BALB/c mice, it would be expected to express some of the histocompatibility characteristics of the DBA/1J strain. Specificity is a relative term. From the data reported, it is not possible to determine whether the resistance observed by Faraci and Schour should be interpreted as melanoma specific, tumor-line specific, or strain specific.

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