New Developments in Potassium and Cell Physiology: 1940-50[1]

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POTASSIUM IS FOUND IN GREAT ABUNDANCE in plant and animal tissues, where it plays an important part in cellular function. Research on potassium has continued to be productive during the past decade, which began in 1940 with the Cold Spring Harbor Symposium on Permeability (*1*) and ended with the 1950 annual meeting of the Society of General Physiologists. At this meeting a special conference on electrolytes illustrated some of the gains made during this important period (*2*).

In 1940 the situation was summarized by Fenn (*3*). Potassium was known to be the most plentiful metallic cation in most animal cells and in many plant cells also. It was recognized to be essential for the growth and maintenance of man, the lower animals, and plants. Full recognition was given to the important role it plays in the physical biochemistry of proteins, in the physiology of excitable tissues, and in the electrochemistry of cells in general. Biologists were in agreement that to gain an increased understanding of the behavior of this element would be to contribute to many fields, including plant and animal physiology, cell physiology, endocrinology, and clinical medicine. The use of radioactive potassium as a tracer in biological studies had just begun, and pioneer experiments using this new tool were under way.

In discussing the contributions during the years that followed, a compromise must be made between the necessary conciseness required by the general reader and the detailed critical analysis desired by the specialist. Several recent reviews are recommended for those with more scholarly requirements. These include the discussions by Krogh (*4*) and Ussing (*5*), written from the point of view of general physiology, and the papers by Weller and Taylor (*6*) and Hoffman (*7*), which include some clinical considerations. The review by Overman (*8*) will meet some of the requirements of the endocrinologist, and the review by Steinbach (*9*) will answer many questions in muscle physiology. The more specific questions of permeability are discussed by Teorell (*10*), and neurophysiological considerations, by Lorente de Nó (*11*). For the nonspecialist, Fenn's recent popular review (*12*) is recommended.

Potassium and metabolism. In the earlier literature it was the fashion to regard potassium as a more or less passive metallic cation which responds in a biological system to the local concentration gradient and electrical field in its neighborhood. From this point of view its movement was thought to be controlled by the permeability or impermeability of membranes, and the element was believed to participate in biological phenomena primarily through its influence on the hydration of protoplasmic material. It is now becoming increasingly clear that potassium must be thought of in more complete terms as participating in enzymatic reactions, possibly as an essential element, and at the same time being accumulated into cells against ionic concentration gradients and electrical forces, deriving the necessary chemical free energy from metabolism. Many physiologists regard such processes as *active*, and the concept of active accumulation has been clearly presented in the review by Ussing (*5*).

It is now a familiar biochemical principle that various inorganic ions participate in the activation of particular enzyme systems. Potassium is required by the important enzyme enolphosphopyruvate–ADP transphosphorylase in muscle extracts (*13*). Without potassium the reaction has been shown to be irreversible (*14*, *15*). A possibly related fact is that the element plays a significant role in the aerobic metabolism of brain (*16*), and increases the synthesis of glycogen from glucose in rabbit liver slices (*17*). It has been reported to depress the activity of saccharase, β-glycosidases, and catalase in molds and yeasts grown on potassium-containing media (*18*).

The symptoms of adrenal cortical insufficiency are the most familiar example of a relation between potassium and endocrine function. The relation between tissue potassium and the adrenal hormones has been reviewed by Overman (*8*). Recently a method has been described for determining small amounts of desoxycorticosterone with adrenalectomized rats, using the radioactive potassium isotope (*19*). The relation between potassium accumulation in cells and carbohydrate metabolism is still not clear. It has been known for many years that intravenous injections of epinephrine cause a rapid but transient increase in plasma potassium (*20*), which is particularly evident in arterial blood (*21*). In cats, injection by vein of those sugars, such as glucose, that are selectively absorbed in the intestine causes a transient rise in plasma potassium. This effect is removed by adrenalectomy (*22*). It was found in the early days of insulin therapy that injection of insulin causes a de-

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pression in the plasma potassium level in animals (*23*) and in diabetic patients (*24*). This phenomenon continues to be useful in the clinical management of potassium disturbances.

The fact that many cells selectively accumulate potassium remains an unsolved problem in physiology, but the mere existence of a large potassium concentration gradient need not prove active accumulation of potassium directly. Our ideas concerning the selectivity of potassium accumulation in muscle have been strongly influenced by the Boyle-Conway theory, which has been reviewed by Conway (*25, 26*). According to this theory, potassium is accumulated in the muscle fiber passively as a result of a double Donnan effect. This effect arises from the electrical potential gradient set up by nonpenetrating colloidal anions inside the fiber and positive sodium ions that are excluded from the fiber in some way. Many writers now assume that sodium is kept out by a process of active extrusion. The active accumulation of potassium is thus thought to be indirect in this case.

The active accumulation of potassium either directly or indirectly has been observed in such widely different tissues as the chorion membrane of the hen's egg (*27*) and the isolated frog's heart (*28*). Recent experiments have shown that increased glucose utilization by rat diaphragm in the presence of added insulin is accompanied by an uptake of potassium after correction for leakage, perhaps caused in part by trauma of the excised tissue (*29*). In the process of active transport of potassium in brain tissue and in ox retina, glutamic acid is required in the medium, in addition to glucose (*30*). This has given rise to some interesting speculations concerning potassium absorption in the kidney (*31*). Active accumulation of potassium in yeast, although originally thought to be similar to that in animal tissue, is now known to be accompanied by a high production of acid and seems to proceed by the exchange of potassium for hydrogen ions (*32, 33*). It is a familiar fact that salt accumulation in plant cells is an active process intimately related to cellular metabolism. A recent discussion of the quantitative relation between salt accumulation and "salt respiration" includes a critical analysis of the Lundegårdh theory (*34*).

The maintenance of the resting potential of nerve by metabolic processes provides circumstantial evidence for active maintenance of ionic concentration gradients (*35*). Neurophysiologists have usually considered that sodium plays the active role in resting nerve. Recently Hodgkin and Katz (*36*) have provided strong evidence that, during passage of a nerve impulse, the reversal of polarization is due to the momentary shift of the active role from sodium to potassium. Frog sciatic nerve preparations lose more potassium in the absence of oxygen. Evidence was obtained that, when oxygen is present, accumulation may actually occur against a concentration gradient (*37*). It is becoming increasingly evident that potassium is lost from nerve during activity (*38*).

Isotope studies. The conclusive evidence that ionic concentration gradients in cells are not the result of membrane impermeability came from experiments with isotopically labeled ions. Pioneer studies with radioactive potassium have been reviewed by Hevesy (*39*), including the important early work by the University of Rochester investigators. The principal isotope used in studying the physiology of potassium has been K^{42}, since it has the advantage of a highly penetrating β-ray that minimizes self-absorption difficulties in counting. Although the associated γ-ray is weak, the β-ray activities could in principle be determined in a thick-walled ionization chamber by the Bremsstrahlung method of Tompkins *et al.* (*40*). K^{42} has been employed successfully in macroscopic radioautography (*41*). The short half-life (12.4 hours) precludes its use in long-term studies and complicates its use at a distance from the source of production.

Recently it has been shown that, under certain conditions, the reactor-activated isotope can contain detectable quantities of long-lived contaminants in addition to K^{40}. These include Rb^{86} caused by the presence of small amounts of the parent element in the irradiated material (*42*). Such contaminants usually appear when the material has been exposed to slow neutrons for periods of a week or more, followed by an extended period of decay. The use of reactor-produced K^{42} (*43*) greatly lessens the problem of Na^{24} contamination; nevertheless, the formation of the latter isotope is still favored by a factor of 20, and the parent material must be practically sodium-free, unless a radiochemical purification is to be used.

Where the short-lived isotope is unacceptable, it becomes necessary to resort to the naturally occurring radioactive isotope K^{40} obtained by isotope separation methods. In this case the usual radiochemical precautions in the use of a long-lived istotope become necessary. According to Mullins and Zerahn (*44*), analyses of the normal K^{40} content of various animal, vegetable, and mineral sources showed no variation within 0.5 per cent.

Since the effectiveness of isotope experiments is greatly increased by simultaneous chemical determinations, recent improvements in the methods of alkali metal analysis are of interest. Flame spectroscopy is not a new technique, having been used in biology by Lundegårdh more than twenty years ago. The fact that the flame photometer has only recently been perfected as a routine commercial instrument (*45*) illustrates a continuing lack of direct contact between some fields of physical instrumentation and biochemistry. With this instrument, potassium determinations of acceptable accuracy are completed in hours, compared with days for the chemical methods.

An increasing number of papers describing tracer experiments with K^{42} are appearing in the literature. On the assumption that earlier work has been discussed adequately in existing reviews, only recent reports will be considered. These cover a variety of biological experiments. In the field of marine biology, echinoderm eggs take up a strikingly larger amount of radioactive potassium from artificial sea water

when they are fertilized than do the unfertilized controls (*46*). Studies of chicken embryo muscle by the tissue culture method show that the potassium does not move into and out of the cells as though they were a single, uniformly mixed compartment. The results indicate either that potassium is present in more than one chemical state or, more probably, that there are structural inhomogeneities in the cells (*47*). Cultures of *Escherichia coli* show a labile potassium fraction that exchanges completely with the potassium of the suspension medium in less than five minutes, and a tightly bound fraction that increases as metabolism progresses (*48*). Sections of squid nerve, when suspended in artificial sea water, rapidly exchange about 10 per cent of their potassium in one to two hours, the remainder seemingly being slowly exchangeable, if at all (*49*). One limitation of experiments with material that is structurally intact, or nearly so, is that, if a bound fraction exists, in principle it cannot be identified as being chemically rather than physically bound. The decision requires additional information.

The inhomogeneities that occur in cellular systems do not always present an insurmountable obstacle to quantitative experimentation. Harris and Burn (*50*) have considered experiments on the penetration of labeled sodium or potassium into excised muscle suspended in solutions containing Na^{24} or K^{42}, where one must consider both the diffusion of ions in the extracellular space and the rate of crossing cell boundaries. They present an approximate mathematical solution to the problem based on the now familiar analogy between the movement of radioactive tracers in a closed steady state system and the classical problem of the flow of heat. This analogy has been recently discussed by Sheppard and Householder (*51*).

The permeability of mammalian erythrocytes to cations was investigated soon after the first successful large-scale cyclotron production of isotopes. This problem has continued to receive attention, the most clear-cut results having been obtained for potassium in human erythrocytes. Here independent investigations in two different laboratories have achieved remarkably good agreement (*43, 52*). When freshly drawn heparinized human blood is equilibrated at 38° C under an atmosphere containing 5 per cent CO_2 in the presence of added sugar, the entire cellular potassium exchanges at a uniform rate, the specific activity changes of cells and plasma proceeding as though the erythrocyte potassium were in a single pool. The exchange rate under normal conditions is 1.6–1.8 per cent of the cellular potassium per hour. Between 44° C and 15° C the logarithm of the exchange rate is proportional to the reciprocal of the absolute temperature with a Q_{10} of 2.35. The kinetics are unaltered by oxygenation or reduction of the hemoglobin or exposure to 1,200 r of γ-rays. Below 15° C the familiar net leakage of potassium from the cells ensues; above 44° C the system rapidly deteriorates.

The observation of Raker *et al.* (*52*) that the rate of exchange is essentially unaltered by increasing the plasma potassium concentration has been confirmed more recently (*53*). Danish investigators (*54*) report that increasing the plasma potassium roughly threefold produces about an 8:5 increase in rate. Whatever the explanation of the disagreement, the relation between concentration and exchange rate is not that of a passive diffusion of potassium. In the potassium-depleted cells of hypopotassemia the uptake of the element is due to an increased influx rather than to an arrested outgo (*54*).

Although potassium does exchange in the erythrocytes of other species, the quantitative description of the process is less satisfactory. Human cells show a stability *in vitro* that is often lacking in other red cells, particularly those of the dog. Often it is not possible to continue an experiment long enough to test the completion of exchange. As in the chicken embryo muscle experiments, inhomogeneities in the exchange rates are often found. In canine blood one such cause of multiplicity is the exchange in the cells of the buffy coat (*53*). Isotope experiments on red cells *in vivo* are complicated by the exchange of potassium in other body tissues, although semiquantitative kinetic studies are possible.

Isotope experiments *in vivo* have the advantage that they produce minimal disturbance of the tissue under investigation. When potassium containing a radioactive label is injected into the circulation of an animal, it will appear in various tissues at different rates. Early studies in the rat (*55*) showed a wide variation in the appearance rate, the liver and kidney being among the most rapid, muscle tissue being intermediate, and erythrocytes and brain being slow. Such investigations are now being pursued more thoroughly in the rabbit (*56*). The most striking phenomenon is the extremely precipitous fall in the circulating level of intravenously injected radioactive potassium. In order to observe the decline quantitatively, samples must be obtained seconds apart following injection. Since these times are small compared to the circulation time it must be accepted that the rate of removal of the isotope by exchange of potassium with that in the tissues is now limited by the circulation rate, which controls the speed of delivery of the isotope to the site of removal. It has been repeatedly observed that the specific activity of the liver exceeds that of the plasma for a considerable period shortly after injection, showing that in this organ the mixing of injected potassium with that of the organ does not proceed by simple passive diffusion. The penetration of potassium into normal and atrophied rabbit muscle has been observed by Fischer *et al.* (*57*), who noted a tendency for the muscle specific activity to overshoot that of the plasma.

It is well to inject a word of caution concerning the interpretation of experiments on the kinetics of disappearance of injected substances from the circulation. Observations of the circulating radioactivity alone are, in principle, not sufficient uniquely to determine exchange rates between the circulation and other body compartments.

Experiments with isotopes, such as K^{42}, which are rapidly removed from the circulation when injected, have recently called attention to the mechanics of the circulatory mixing process. An ingenious method has been developed by Morel (*58*) whereby the activity of the blood may be continuously recorded following injection. Working with Na^{24}, it was shown that the first twenty seconds of the disappearance curve of this isotope are complicated by the presence of a wave of activity during the process of mixing. These considerations apply to potassium experiments as well.

Potassium accumulation in erythrocytes. The potassium metabolism of human erythrocytes has been of considerable practical interest in connection with studies of blood preservation both here and abroad (*59–62*). The behavior of potassium is of considerable fundamental interest, however, since it is not unlikely that processes that occur in a relatively simple cellular system, such as the erythrocyte, may resemble processes in other tissues. These processes of cation control are intimately related to the maintenance of osmotic stability of cells and, in excitable tissues, to the production of bioelectric phenomena. For the period prior to 1942 most of the literature on the transport of potassium in cellular systems, including erythrocytes, is reviewed in the monograph by Davson and Danielli (*63*), where a number of now familiar principles are discussed and documented. One point that may be recalled is the large species variation in the selective accumulation of potassium by mammalian red cells, ranging from practically no accumulation in the cells of the dog to a nearly twenty-fold concentration ratio between cells and plasma in the case of human blood. Familiar also is the tendency for high-potassium cells to lose potassium when suspended in nonelectrolyte solutions or in media containing a wide variety of mildly injurious substances, such as glycolytic inhibitors, lytic agents, heavy metals, and rose bengal activated by light.

It has recently been shown by Ponder (*64*) that in some of these disturbances the progressive potassium loss is accompanied by an almost equivalent penetration of sodium. Overman (*65*) has reported the exchange of sodium for potassium in nonparasitized erythrocytes of the malarious monkey. Reciprocal potassium leakage and sodium penetration have also been noted in human red cells when exposed to relatively high doses of x-rays (*66*). The exchange is not confined to red cells alone. Reciprocal exchange of sodium for potassium has also been observed in anoxic nerve (*67*), and good evidence exists that it also occurs in rabbit leucocytes under varying sugar concentrations (*68*). Recent studies with liver slices suggest that the exchange occurs in hepatic tissue (*69*). It is thus apparent that a specific failure of potassium selectivity must be included among the injurious effects that can occur in erythrocytes and other tissues.

Experiments on the exchange of potassium for sodium in red cells deal primarily with the movement of sodium and potassium from regions of high to regions of low concentration. The reverse process requires a source of chemical free energy doubtless derived from metabolic processes. It was first reported by Danowski in freshly drawn defibrinated human blood (*70*) and by Harris in human cells which were depleted of their potassium by low temperature storage (*71*). These studies both showed that, at 38° C in the presence of sugar, potassium enters the cells, the current being reversed as the sugar concentration approaches zero. Addition of sugar prolongs the period before potassium leakage sets in. Upon addition of fluoride an irreversible potassium leakage from the cells occurs. Danowski showed that the normal movement of potassium was not accompanied by large water transfers, and Harris showed that this was due to the reciprocal movement of sodium. More recently the phenomenon was re-examined by Maizels (*72*), who demonstrated that the accumulation process has a pronounced pH optimum at about 7.4. The view was favored that the process was essentially one of active sodium exclusion. That sodium transport in red cells is indeed related in some way to an active process is suggested by the pH and temperature sensitivity observed in the studies of Davson (*73*) and Davson and Reiner (*74*) on the loss of sodium from cat erythrocytes suspended in isotonic NaCl; nevertheless, sodium exclusion alone cannot be accepted in the erythrocyte without modification. The recent investigation by Ponder (*75*) demonstrates that accumulation of potassium occurs almost as well when sodium in the external environment is replaced by lithium or cesium. Ponder found that the optimal glucose concentration lies in the region 50–200 mg per cent and that the Q_{10} of the process is 2.4. Maizels searched for a relationship between potassium accumulation and the breakdown of organic phosphorous compounds without obtaining clear-cut results. It is of interest to recall that in the earlier literature Kerr (*76*) noted a correlation between the intracellular potassium content in the erythrocytes of several species and the organic acid soluble phosphorus. An interesting aspect of these experiments is that all the potassium does not exchange by the same amount. This has been discussed recently by Ponder, who stresses the effect of nonuniformity among the cell population (*77*).

Perhaps the most interesting recent contribution to the problem of potassium accumulation in erythrocytes has been the discovery by Greig and Holland (*78*) that the specific cholinesterase inhibitor physostigmine disturbs the accumulation process. In experiments by the isotope technique Taylor and Weller claim that cholinesterase inhibitors depress the rate of penetration of potassium, whereas inhibitors known to affect choline acetylase increase the rate of loss from cells to plasma (*79*). Washed cells which are provided with acetyl choline remain intact *in vitro* for longer periods of time than those to which substrate is denied (*80*). Recent observations by Russian workers also indicate a correlation between the activity of cholinesterase in erythrocytes and their permeability (*81*). The assignment of a role for the

cholinesterase associated with the red cell envelope (*82*) thus establishes an unexpected similarity between ionic effects in erythrocytes and in excitable tissues.

Mechanism of selective accumulation. Following presentation of the conclusive evidence that potassium is accumulated directly or indirectly by active processes in most, if not all, biological systems, it is pertinent to inquire into the mechanism by which the element is concentrated in cells against the concentration gradient. One suggestion that is occasionally offered is that potassium in the cell exists in some chemically bound form. Indeed, in the case of *E. coli* the evidence favors some potassium binding. In animal tissues the concept of binding of alkali cations remains in an equivocal state. Nondialyzable potassium fractions have been reported in extracts of rat brain and muscle (*83*). That these observations may be due to an artifact is suggested by the lack of a comparable effect in frog muscle homogenates, where the sedimentable fraction contains, if anything, a slight excess of sodium (*84*). Little is known about the tendency of alkali metals to form undissociated chemical compounds; however, Christensen and Hastings describe such a compound between sodium or potassium and cephalin (*85*).

The concept of bound potassium fails to give a satisfactory explanation of accumulation in erythrocytes. In human cells the isotope results show that all the intracellular potassium exchanges as though it were in a single pool. It would thus be necessary to postulate that all the intracellular cation (mostly potassium) was bound but slowly exchangeable. Although the erythrocyte is not a perfect osmometer, the binding of all the intracellular cation would certainly be reflected in its osmotic behavior. It would also seem to be no accident that the intracellular concentration of total cations in species with widely different intracellular potassium is quite constant and not far different from that of the plasma, where the alkali cations are essentially entirely free (*86*).

It may be argued that the forces between cations and the intracellular material are as yet incompletely understood. Such forces might be selective for potassium, and such a hypothetical effect coupled with an internal resistance to water transfer might fit the experimental facts and yet permit potassium accumulation in erythrocytes by a binding process. The osmotic deficit would be compensated for by the binding of water, so that the cation is bound in an isosmotic solution. Certainly the space occupied by hemoglobin in the cell is great, and the ions are at all times close to protein molecules. The argument thus rests on how radically this proximity alters the thermodynamic activity of the ions and of the water molecules. Some evidence on this question was recently obtained by Stratmann and Wright (*87*), who found that hemoglobin solutions, when dialyzed against semipermeable membranes, could accumulate potassium. The less than threefold increases in concentration they obtained were much less than those occurring in high potassium erythrocytes. A similar conclusion can be drawn for the accumulation of potassium by myosin (*88*). Although such accumulations are not impressive, their results cannot be lightly discarded without further investigation.

The Donnan theory provides a ready explanation for the relatively small static concentration gradient which classical thermodynamics predicts for ions when a semipermeable membrane constrains a solution of impermeable electrically charged molecules. It has been shown that such a gradient will also arise as a result of active transport of ions (*89, 90*). The question whether this mechanism alone could account for selective potassium accumulation was discussed by Spiegelman and Reiner (*91*), who concluded that the unmodified classical theory was unable to account for potassium selectivity. Denying that differences in the mobility of sodium and potassium ions can account for it, they concluded that a specific chemical force of some type acting on sodium or potassium, or both, was required to explain the observed facts. Such a force has been postulated for sodium alone in muscle by the Boyle-Conway theory (*25, 26*). The thesis that potassium is passive rests on such experiments as those of Wilde, who has shown that, when the plasma potassium is elevated in nephrectomized rats by feeding potassium, there is an increase in the muscle fiber potassium, presumably as a passive result of the concentration increase (*92*). That sodium is actively excluded (*5*) is based on the ability of animals with elevated muscle sodium produced by a low potassium diet to extrude this sodium when the dietary potassium is increased (*93*).

In the erythrocyte the modified Donnan theory presents a difficulty, if it be postulated that potassium is accumulated passively as a consequence of active sodium exclusion. If potassium is regarded as a passive ion, then according to the classical picture it will be distributed across the cell membrane in conformance with the Donnan ratio. This would require a variable ratio for the red cells of different species ranging from nearly one for canine cells to nearly twenty for the cells of man. Correspondingly, the chloride ratios and intracellular pH would vary between these wide limits. Although ratios of two are observed, a tenfold greater ratio will not fit the experimental facts. The same argument holds for passive control of sodium as well.

One promising explanation for the selectivity which has received considerable attention assumes that a process occurs at the cell interface in which the rate of transport of the cation inward is made to exceed the rate outward. A particular version of this point of view has been maintained for some time by Osterhout (*94*) and recently summarized. He postulates that potassium is carried into the cell by some carrier molecule, from which it dissociates on the intracellular side of the interface. The Lundegårdh theory for ion transport in plant cells (*34*) has been cited freely during the past decade. This postulated the existence of a potential difference across the plant wall as a

result of a redox reaction involving cytochrome. A more general theory that retains cytochrome as the redox system has recently been advanced by Conway (*95*) and Conway, Brady, and Carton, for the exchange of potassium for hydrogen ions in yeast and other acid- or alkali-forming tissues (*96*).

Although many ingenious attempts have been made during the past decade, the achievements in the potassium field do not include the establishment of a satisfactory theory of the maintenance of ionic concentration gradients in cells. However, some of the basic considerations that must be included in such a theory have been discussed. Definite progress has been made in recognizing the existence of active accumulation processes, in the realization of their widespread nature, and in their close connection with cell metabolism. The use of isotopically labeled substances has demonstrated in an unequivocal fashion the movement of cations into and out of cells which were postulated by earlier workers to be cation-impermeable. The first flush of ambition among isotope workers has been replaced with a more mature caution, and there is increased consciousness of what is easy and what is difficult in this field. Cell physiologists have obtained a larger body of information relating the physical aspects of cation movement to cellular biochemistry. The biochemist alone has been unable to explain all the factors controlling ionic movement. Nevertheless, he can say in certain instances how the necessary energy for the process is mobilized in the cell, and he can cite one possible use for the element after it is accumulated. It is left as a task for the future to establish how the cation interacts at the cell interface, how the energy is expended in the movement of the ion into the cell, and how the ion later leaves the cell in exchange for another.

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Technical Papers

Natural Black Uranium Powder

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A soft, black, uranium-bearing powder has been found in several localities during the exploration of uranium deposits in the western United States. The nature of this powder, its significance from the standpoint of origin, and its distribution constitute matters of scientific interest, as well as of practical importance. Among the localities in which the powder has been particularly noted, specimens were collected for study from Marysvale and White Canyon, Utah, and the Caribou and Bellvue-Rochester mines in Colorado. Material of similar appearance has also been noted in a collection of specimens from the Belgian Congo.

The soft pulverulent character of the material and the color suggest that it might be a form of the organic uranian mineral thucholite, recognized by Ellsworth (*1*) as a black uranian hydrocarbon in rocks of the Canadian shield. However, analyses for carbon (Table 1) made by the New Brunswick laboratory of the AEC yield amounts so low that thucholite could hardly be present in any significant amount.

The material in question is not thucholite, and laboratory studies indicate that it is largely uraninite. The various pulverulent materials collected yield substantial amounts of uranium on the basis of x-ray fluorescence analysis, with the use of a technique similar to that described by Birks and Brooks (*2*). Moreover, diffraction patterns yield interplanar spacings that establish the prevailing crystalline uranian constituent of the powder as uraninite. The material is commonly mixed with fine pyrite, and at some localities other metallic sulfides are associated.

[1] In the conduct of these studies the writer has had the benefit of cooperation by C. J. Rodden, chief, Microchemical Branch, New Brunswick Laboratory, AEC, and Harold Wright, who is studying the mineral relationships at the Caribou mine.

TABLE 1

CARBON CONTENT OF SOOTY URANINITE SAMPLES

	A (%)	B (%)
Bellvue-Rochester mine, Colo.	0.12	
White Canyon, Utah	0.15	0.09
Marysvale, Utah	0.18	0.13

It has been observed that certain samples of uraninite from the Shinkolobwe mine in the Belgian Congo are also sooty, and one specimen with slickensides even appears graphitic. On analysis these samples are likewise shown to be noncarbon-bearing. Such specimens yield x-ray diffraction patterns corresponding to uraninite but with low lattice constants, in contrast to the lattice constants of hard cubic crystals of uraninite from the same locality (Table 2).

A number of observations indicate that the sooty uraninite may be a later form high in UO_3 that has originated at the expense of earlier hard uraninite, high in UO_2. Ellsworth (*3*) studied the successive zones in a large, progressively altered uraninite crystal from Villeneuve, Quebec, and pointed out that UO_2 and total U decreased from the center outward but that, at the same time, UO_3 notably increased until accountable for the entire uranium content at the most highly altered surface. Kidd and Haycock (*4*), in their study of the ores of Great Bear Lake, noted a later type of uraninite, less lustrous and softer than original hard uraninite, and formed at the expense of the earlier mineral. In the earlier uraninite the ratio $UO_2 : UO_3$ was 10 : 2.2, but in the later uraninite the ratio was 1 : 10. Analyses of both sooty and graphitic

TABLE 2

LATTICE CONSTANTS, SHINKOLOBWE URANINITE

Well-formed cubic crystal	5.453 A.U.
Graphitic type	5.438
Sooty type	5.411