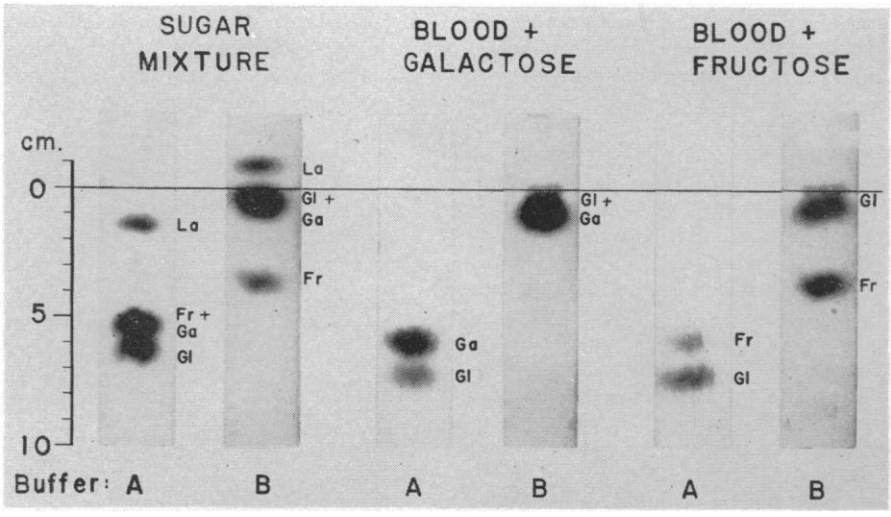

Fig. 1. Typical strips illustrating the separation of glucose (Gl), galactose (Ga), fructose (Fr), and lactose (La) in a standard sugar mixture and the separation of added galactose or fructose from the glucose of normal blood.

percent (weight by volume) aqueous solutions of reagent-grade glucose, galactose, fructose, and lactose, respectively, and of a mixture containing all four sugars, are used as controls. On the standard strips, 2 μl are used per sample. The aniline hydrogen phthalate bath used consists of 930 mg of redistilled aniline and 1.66 g of phthalic acid, dissolved in a solution of 5 ml of water in 95 ml of acetone. This solution, used as a bath, proved far superior to sprays based upon various alcohols.

Method. Heparinized blood (0.1 ml per determination) is pipetted into 2 ml of ice-cold ethanol (95 percent) and chilled in ice water for 15 minutes to precipitate the proteins. The mixture is then centrifuged (at 2000 rev/min) for 5 minutes. The supernatant liquid is decanted into a 2-in. petri dish and evaporated to dryness in a vacuum. The residue is taken up in 50 μl of water in a micropipette (*4*) and is applied to the mid-point of a paper strip (3 by 30 cm) and air-dried. The strip is mounted on the apparatus with the applied spot at the apex and is wetted carefully with buffer on each side, just below the spot, in accordance with Durrum's technique (*5*). After 1 hour is allowed for equilibration, an electromotive force of 210 v is applied for 4 hours. The strips are then dried, dipped in the aniline hydrogen phthalate bath, and suspended in an oven at 125°C for 10 minutes. Compact, stable, brown spots indicate the presence of the sugars.

Under these conditions, when buffers A and B are used in turn, the sugars migrate, as indicated in Fig. 1. Comparison with control strips indicates the presence, or absence, of glucose, galactose, fructose, or lactose in the blood sample. Heavy streaking of the spots indicates either insufficient equilibration time or too concentrated a sample.

The method has been applied to a large number of blood samples, and no interfering substances have been encountered in either normal or galactosemic blood. To date, neither fructose nor lactose has been found naturally in any sample (*6*).

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On the Nature and Color of the Moon's Surface

The interstellar dust particles presumably responsible for the reddening of distant stars may consist largely of random aggregates of unsaturated and free-radical molecular species rich in carbon, nitrogen, and oxygen (*1, 2*). By theoretical and empirical analogy with similar terrestrial species it has also been shown that such particles would need to have diameters only of the order of 20 A or less in order to absorb strongly in the visible region of the spectrum (*3*), since with such dimensions the average gap for allowed transitions between randomly filled Fermi levels is of the order of 3 ev. The random geometric and magnetic anisotropy of such particles also appears adequate to account for the polarization associated with interstellar reddening.

Particles of such properties and sizes differ considerably from the chemically stable or closed-shell dielectric and ferroelectric particles about 0.5 μ in diameter which are classically postulated for the interstellar dust. To give the same optical effects, the free-radical particles can be of the order of 200 times smaller in linear dimensions and 10^7 times smaller in total bulk than the dust particles required by approaches based only on classical metallic and dielectric scattering theory. Even if chemically stable dielectric or metallic particles should be formed, laboratory experience suggests that, under high-energy collisions and ultraviolet light, the particles in interstellar space would soon come to resemble radiation-damaged material, with randomized structures highly absorbing in the visible region.

These arguments should apply not only to particles but also to large airless surfaces in space if they are poor in hydrogen—perhaps, for example, the surface of the moon or of the planet Mercury. Whatever the original composition of the underlying material or its overlying interplanetary dust-fall, the surfaces we see may be extensively damaged by radiation and highly absorbing in the visible region. Even if there is progressive healing of the damage with ionic recombination and formation of renewed stable bonds during the hot lunar day, laboratory experience suggests that it may not be complete, especially when ultraviolet radiation is also present during the heating.

This prediction of surface disorder and opacity, possibly accompanied by porosity in layers of recombined dust, would help account for two perennially striking properties of the moon's surface: (i) the generally low albedo of the moon (and Mercury), about 7 percent, which has previously been taken as evidence that the surface resembles our blackest terrestrial rocks, slates, and basalts; (ii) the low thermal conductivity even on high lunar surfaces that might be expected to be free of loose dust. [Gold (*4*) has mentioned "radiation-induced coloration" but seems to suppose that it is negligible "in the denuded (eroded) highlands."]

Surfaces of such a chemically unstable character could also be highly reactive, and extensive exothermic reactions might be easily triggered, as in the Donn-Urey explanation of cometary outbursts (*2*). The first man who plants a rubber boot on a lunar surface may be in for an unpleasant surprise. This suggests a number of possible experiments as well as a num-

ber of precautions for early moon missiles. The gettering action of such a porous reactive surface may also be important in the final disappearance of any original lunar atmosphere.

Meanwhile, there are several laboratory experiments that would throw light on these questions. It would be worthwhile to study the changes in the reflectivities and other surface properties of common rocks and of carbon-, nitrogen-, and oxygen-containing compounds when exposed to high-energy particle radiation, ultraviolet radiation, and heating cycles. Such studies would have to be made under the most stringent vacuum conditions to avoid disturbances from oxygen-recombination effects. A comparison of the results with various lunar reflectivities might then make possible the closer identification of lunar rock species (*5–7*).

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6 May 1958

Functional and Structural Observations on Chronically Reserpinized Monkeys

The effects of reserpine on the behavior of monkeys and chimpanzees were noted as reversible phenomena, resembling those encountered in human Parkinsonism—namely, hypokinesia, lethargy, mask-like face, muscular rigidity with "cogwheel" quality, sialorrhea, and tremors at rest (*1*). Even though experimental animals recovered completely from any ill effects shortly after the interruption of reserpine medication, and although no anatomical changes have hitherto been reported, it would be surprising if the profound neurological aberrations that occur in the course of prolonged medication had no anatomical counterparts in the central nervous tissue. The clinicoanatomical study described in this report was carried out on two adolescent female African green monkeys kept under reserpine (*2*) medication for 18 months and one male monkey given reserpine for 2 days and kept for 2 years without any drugs.

The first female received 0.5 mg of reserpine per kilogram of body weight intramuscularly daily for 238 days. Spontaneous activities lessened (*3*). Tremors at rest were frequently observed about 4 hours after each daily dose, lasting for several hours. Increasing the dosage to 0.75 mg/kg led to the appearance of some muscular rigidity. Reserpine administration was discontinued for 14 days. During the first 4 days of this interval the monkey still exhibited tremors at rest; during this entire period spontaneous activity remained less than it had been prior to the experiment. Administration of the drug was then resumed, at dosages of 0.2 to 0.4 mg/kg daily for 160 days. Tremors reappeared on the second day, becoming almost continuous and quite violent toward the end of this period. Treatment was again interrupted for 5 days, during which time the tremors disappeared but the activity level remained low. Treatment with reserpine was resumed at a dosage of 0.4 mg/kg; the dosage was gradually increased to 0.8 mg/kg and continued at this level until the experiment was terminated, after a total of 615 days. During this last period of treatment the animal developed what amounted to "status tremoris."

The second female was given reserpine daily for 512 days. Doses larger than 0.6 mg/kg led to some rigidity and hypokinesia (*3*). Doses as low as 0.3 mg/kg maintained the animal in a state of reduced activity and almost continuous tremor at rest. The drug was withheld for 5 days and then the animal appeared normal, with no tremors. Treatment was resumed at a dosage of 0.6 mg/kg; dosage was increased to 0.7 mg/kg and continued at this level until the experiment was terminated, after a total of 552 days.

The three animals were killed by the perfusion fixation method (*4*). Postmortem examinations revealed no gross abnormalities of visceral organs, brains, or spinal cords.

Microscopic examination revealed normal histological and cellular structures in the central nervous system of the monkey that was given no reserpine for 2 years.

The brains and spinal cords of the two chronically reserpinized monkeys showed no signs of hemorrhages, infarcts, softening, demyelination, glial reaction such as gliosis, or activation of histiocytes with phagocytosis. The cerebellum was notably free of any indication of change, all elements being well preserved and well stained.

Neuronal differences between the two chronically treated animals and the one which had had no drug for 2 years were clearly evident in some parts of the brain, especially in the cerebral cortex, basal ganglia, and brain stem. Conspicuous cytological changes involved the neuronal nucleus and nucleolus. A considerable number of the nuclei were markedly enlarged and pale. The pallor of the nuclei was caused by what appeared as a hole in the karyoplasm. These holes varied in size, and each had a scalloped periphery, as though some substance had been removed in the histological preparation. The nucleolus was located in the karyoplasm and stained lightly with gallocyanin at *p*H 1.7. The chromaphil bodies adherent to the nucleolus were slightly smaller in the two reserpinized monkeys than in the animal that had been given no drug for 2 years. Considerable variation from cell to cell and in different regions of the brain was observed.

The fact that neither severe changes nor loss of neurons was observed suggests reversibility of the process. However, the significance of these observations is as yet not known. In man, somewhat similar nuclear changes have been reported in insulin shock (*5*), anoxia (*6*), and so on. These have been ascribed to a process of vacuolization of the nucleolus (*7*). In the study discussed in this report, no vacuolization of the nucleolus was demonstrated. In keeping with the current concepts that nuclear-nucleolar activities are involved in protein metabolism of neurons (*8*), the results given here indicate that during chronic reserpine medication, cell metabolism is influenced at all levels of the nervous system. Furthermore, this cellular response is not specific to the monkey; we have identified similar nuclear changes throughout the brains of cats subjected to powerful reserpine treatment. The ubiquitous effect on the nervous system is consistent with the wide range of symptoms which these animals exhibit, but we would hesitate to correlate this morphological alteration with any specific symptom. Although many of the cerebral neurons were involved, we observed no marked changes in temperament and "personality." Whether the changes represent a primary effect of reserpine on neurons or a secondary effect through other systems cannot be settled on the basis of the present morphological observations. It is noteworthy that the neuronal changes did not appear predominantly in the brain stem and basal ganglia, in contrast to the pathological changes in *paralysis agitans* and postencephalitic Parkinsonism.

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