

Table 2. Oxide composition of Gabbro 1 (of Table 1) recalculated for a plagioclase composition of An 85.

Oxide (% by weight)	Gabbro 1	Lunar surface
SiO <sub>2</sub>	42.3	46.4
TiO <sub>2</sub>	6.9	7.6
Al <sub>2</sub> O <sub>3</sub>	14.4	14.4
FeO	15.5	12.1
MnO	0.2	
MgO	6.4	4.4
CaO	12.5	14.5
Na <sub>2</sub> O	0.6	0.6
K <sub>2</sub> O	.5	
P <sub>2</sub> O <sub>5</sub>	.2	
CO <sub>2</sub>	.3	
	99.8	100.0

date for the rock analyzed in Mare Tranquillitatis is a gabbro or gabbroic anorthosite possibly associated with highly anorthitic anorthosite masses. If this is true, the conclusions of Turkevich *et al.* still hold. Anorthosite is not a common terrestrial rock, and it involves some special geochemical (or petrological) processes, as witness the past controversies involved in accounting for the major anorthosite masses around the world.

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Mitochondrial Genetics:

A Conjecture

The genetics and phenogenetics of mitochondria have been reviewed (1). There may be a major implication for mitochondrial genetics in higher organisms based upon the differences in number of mitochondria contributed by the sperm and the ovum. In the mature egg, the number of mitochondria is very great compared to that in the sperm. It has been estimated that the number of mitochondria in the sea urchin ovum varies from 14,000 to 150,000 depending upon the species (2). In *Priapulius* oogenesis, the number of mitochondria increases from 5 to 8 in the oogonia to 40,000 in the mature oocyte (3). On the other hand, during maturation of the sperm the number of mitochondria decreases. In the sea urchin a single mitochondria ring is present at the base of the sperm head (4).

Thus, the number of mitochondria contributed by the sperm is much less than that contributed by the ovum. In instances where the midpiece does not enter the egg, presumably all the mitochondria would be contributed by the ovum.

The large number of mitochondria in the ovum of a sea urchin is reflected in the quantity of extractable DNA having a density different from that of DNA derived from the nucleus. The amount of this DNA corresponds to the number of mitochondria in the egg, if one assumes that the DNA content is the same as in most other mitochondria derived from somatic tissues in other species (5).

The above facts would imply that the dosage of strictly mitochondrial genes would be much greater from the mother than from the father. Thus, those proteins directly under mitochondrial control (at transcription) should show a strong maternal characteristic. One assumes that there are indeed such genes in the mitochondria (see 1, for example). If new mitochondria arise randomly from the existing mitochondria, then there would be no preferential increase in the relative number of mitochondria originally contributed by the sperm. This would imply that the number of such mitochondria would never catch up during development to the number of mitochondria derived from the ovum.

From these facts and assumptions, one can conclude that there should be some properties of the mitochondria which have striking maternal inheritance. If there were a clinically manifested lesion A in the F<sub>1</sub> generation from a mother with A and a normal father, all progeny would have such a lesion. The F<sub>1</sub> daughters would transmit the disease to all progeny of the F<sub>2</sub> generation, but the F<sub>1</sub> son would have all normal progeny provided his mate were normal. Because of the lack of information concerning the role of mitochondria in development, the lack of knowledge of the gene loci of the mitochondrial DNA and its relationship to the nuclear gene loci, and the lack of knowledge of the interplay among individual mitochondria, the foregoing comments remain only a conjecture.

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Uptake of Actinomycin by  
Sea Urchin Eggs and Embryos

In experiments with <sup>14</sup>C-actinomycin D, uptake and binding were measured in sea urchin eggs and embryos before and after hatching (1). Embryos at various stages were exposed to the drug at a concentration of 20 µg per milliliter of seawater for 45 minutes. Biochemical and autoradiographic data showed significant uptake after hatching and little or none before hatching. These findings were discussed in relation to the fact that actinomycin D inhibits protein synthesis in sea urchin embryos beginning 6 to 8 hours after fertilization, but not earlier in development.

We have discussed these experiments and their interpretation, and we agree that the following comment ought to be made: The hypothesis that eggs of sea urchins (and of other forms) contain untranslated ("maternal") messenger RNA depends in part upon experiments with actinomycin (2). This proposal is not invalidated by the uptake results (1). It is supported by data from experiments done without inhibitors of any kind (3). Actinomycin, administered in doses of 20 to 50 µg per milliliter of seawater for appropriate intervals to prehatching embryos, produces characteristic effects upon the polyribosomes (4), and it inhibits RNA synthesis under these conditions (5). Whatever the penetration rates, therefore, actinomycin exerts the metabolic effects expected of it.

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## Evaporation Retardation by Monolayers

MacRitchie (1) presented data for the evaporation of water from a liquid surface and also studied the retardation of evaporation by monolayers of hexadecanol. Using laminar boundary layer theory, he analyzed both systems. We reexamined MacRitchie's data and found that the mass of water vapor transferred in the absence of hexadecanol is proportional to  $U_0^{0.8}$  (where  $U_0$  is the air velocity), which would indicate a turbulent boundary layer (2). MacRitchie justified his application of laminar boundary theory, which would yield a  $U_0^{0.5}$  dependence (2), by noting that the maximum value of the Reynolds number ( $Re = LU_0/\nu$ , where  $L$  is length and  $\nu$ , kinematic viscosity) is only 28,800. However, the use of a fan in these experiments may introduce a complicated rotational motion, with vortices starting from the ends of the blades, unless shrouds, straighteners, and appropriate entry sections are provided; there is no mention of these. It is therefore not surprising that one

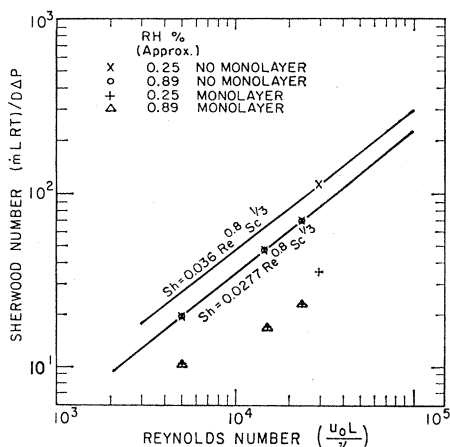


Fig. 1. Variation in Sherwood number with Reynolds number.

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obtains experimental data for this system which is consistent with results obtained for turbulent flows.

Data from previous experiments on turbulent flow have been correlated by the relation

$$Sh = C Re^{0.8} Sc^{1/3} \quad (1)$$

where  $Sh$ , the Sherwood number, is defined by  $mRTL/D\Delta P$ , with  $m$  being the mass transferred;  $R$ , the universal gas constant;  $T$ , temperature;  $D$ , diffusivity; and  $\Delta P$ , partial pressure difference (2); the Schmidt number  $Sc$  is defined by  $\nu/D$ ;  $C$  is equal to 0.036 although there is significant scatter in the data. The experimental results for the velocities and relative humidities reported (1) are in good agreement with Eq. 1 with  $C$  equal to 0.0277 (Fig. 1).

MacRitchie's experiments with hexadecanol monolayers showed that the "evaporation retardation ratio"  $\dot{m}_{II}/\dot{m}_I$  is independent of the relative humidity at a given Reynolds number where  $\dot{m}_{II}$  and  $\dot{m}_I$  are the evaporation rates with and without hexadecanol, respectively. From this result he concluded that the hexadecanol produces no barrier to the vaporization step (the migration of water molecules from the liquid water phase into the vapor phase) but exerts its sole effect by altering the hydrodynamic boundary layer. However, the presence of a vaporization step with hexadecanol does yield results which are consistent with the above experimental observation. In detail, the presence of two resistances in series, that is, both in the vaporization step and in the hydrodynamic boundary layer, yields the relation

$$\frac{\dot{m}_{II}(x)}{\dot{m}_I(x)} = \frac{h_{V,II} h_{H,II}}{h_{V,II} + h_{H,II}} \cdot \frac{1}{h_{H,I}} \quad (2)$$

which is also independent of the relative humidity, where  $h_{V,II}$  and  $h_{H,II}$  are the vaporization and hydrodynamic conductances, respectively, with hexadecanol present, and  $h_{H,I}$  is the hydrodynamic conductance without hexadecanol. Equation 2 is a local relation, so that the (total) retardation ratio is obtained by integrating it over the length of the plate. The integrated result would also be independent of the relative humidity at a given Reynolds number.

To obtain the total retardation ratio we assume that the hydrodynamic conductances  $h_{H,I}$  and  $h_{H,II}$  are equal and given by the turbulent boundary layer results  $Ax^{-0.2}$  where  $A = D(C/0.8)(U_0/\nu)^{0.8}/Sc^{1/3}$  (2) with  $C = 0.0277$ .

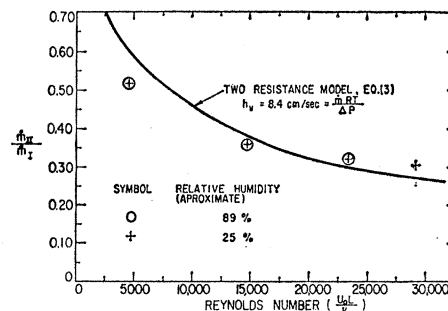


Fig. 2. Evaporation retardation ratio as a function of Reynolds number.

This relation is consistent with Eq. 1. We also take the vaporization step conductance

$$h_v = \dot{m}RT/\Delta P$$

to be constant; upon integrating Eq. 2, we obtain

$$\frac{\dot{m}_{II}}{\dot{m}_I} = \int_0^1 \frac{d(x/L)}{1 + (A/h_v L^{0.2})(x/L)^{-0.2}} \quad (3)$$

Based on the experimental results of MacRitchie (1) a value of  $h_v$  equal to 8.4 cm/sec has been chosen and the results are shown in Fig. 2.

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## References and Notes

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## Ecological Succession

E. P. Odum (1) asserts the need of an understanding of ecological succession. He offers three parameters of succession, one being that, "It results from modification of the physical environment by the community; that is, succession is community-controlled even though the physical environment determines the pattern, the rate of change, and often sets limits as to how far development can go." Succession is certainly one of the key principles of ecology and requires examination, but the parameter quoted needs clarification. Odum asserts that succession "results from modification of the physical environment by the community" and "refers to changes which are brought