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_2	42.3	46.4
TiO_2	6.9	7.6
Al_2O_3	14.4	14.4
FeO	15.5	12.1
MnO	0.2	
MgO	6.4	4.4
CaO	12.5	14.5
Na_2O	0.6	0.6
K_2O	.5	
P_2O_5	.2	
CO_2	.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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11 August 1969

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 *Priapulus* 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_1 generation from a mother with A and a normal father, all progeny would have such a lesion. The F_1 daughters would transmit the disease to all progency of the F_2 generation, but the F_1 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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12 May 1969

Uptake of Actinomycin by Sea Urchin Eggs and Embryos

In experiments with ^{14}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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