where $^{230}Th_0$ = extrapolated surface activity of excess ^{230}Th (d.p.m. g^{-1}); ρ = bulk density (g cm^{-3}); D = depth in crust; $\lambda = 0.693/D^{\frac{1}{2}}$ ($D^{\frac{1}{2}}$ = depth at which activity of excess ^{230}Th equals $\frac{1}{2}$ that of surface value) and ^{230}Th flux = 2.35 d.p.m. cm^{-2} per 10^3 yr per km seawater. Depth of crust = 3,340 m.

For 1,052: $\Sigma^{230}Th_{ex} = 145$ d.p.m. cm^{-2}. Therefore, 'exposure' age = 18,500 yr.

Based on the correspondence between the ages derived from hydration-rind and ^{230}Th flux techniques, and the fact that the ^{232}Th distribution in the ferromanganese crust precludes radioactive decay being the only process affecting the ^{230}Th gradient, the lower age estimate for this sample would seem to be the more accurate estimate. If the age of the volcanic glass is actually about 19,000 yr, the minimum rate of accretion of the Fe–Mn oxides would be about 800 mm per 10^6 yr. In fact, it must have been even faster, since we have calculated that more than 18,000 yr of exposure to seawater would be necessary for accumulation of the excess ^{230}Th that is present in the crust today. Volcanism could have supplied some of the ionium, but this is unlikely since other hydrothermal deposits sampled from oceanic ridges have been shown to contain no excess ^{230}Th (refs 2 and 6).

Conclusions

Our results show that in our sample from the Hess Deep, first, a ferromanganese crust has been deposited without severe fractionation between Fe and Mn. Second, the trace metals Ni, Co and Cu are low compared with typical manganese nodules but the content of REE is about the same. Third, the REE pattern, normalised to shale, is very similar to the seawater pattern. Fourth, the major assumptions in applying U-series dating techniques for assessing rates of growth seem to fail in this case and we conclude that the deposit must have accreted considerably faster than that indicated by the excess ^{230}Th method.

On the basis of these results, we suggest that Fe and Mn as well as some of the trace metals in this crust were deposited from hydrothermal fluids while REE were scavenged from seawater.

This study was supported by the NSF–IDOE Nazca Plate Program through a grant to the University of Hawaii. We were visiting scientists on board the USSR Research Ship Dmitry Mendeleev during her 14th cruise in the eastern Pacific; we thank our Soviet colleagues for cooperation. We thank Oliver Schaeffer, Bill Moore and Yousif Kharaka for comments on the original manuscript, and Floyd Hodges and Maury Morgenstein for technical assistance.

Received September 23; accepted December 9, 1976.

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Electrophoresis along cell membranes

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Bioelectric fields may segregate charged components floating in the plasma membranes of cells by a process of electrophoresis along the membrane. Molecules in cell membranes may be sorted to different portions of the cell surface by such electrical gradients. We present here a theory to support this hypothesis.

MANY molecules and particles are freer to move along the outer membrane of cells than out of this membrane[1,2]. Many of the externally protruding components bear a high negative charge density[3–6]; whereas lecithin (and sphingomyelin) have a substantial positive charge at the calcium ion concentrations typical of extracellular fluids[7,8], and neutral lipids such as cholesterol and certain glycolipids are also common in the outer leaflets of cell membranes[9,10]. If lateral mobility and charge heterogeneity among these membrane components are sufficient, application of a steady field to a cell should substantially redistribute them within the plane of the plasma membrane (Fig. 1). This process may be called surface, lateral, or perhaps perimembrane electrophoresis. I present here a study of the basis and implications of this idea.

Equilibrium electrophoresis along the membrane of a spherical cell

A complete theory of lateral electrophoresis is analogous to the theory of sedimentation. Both the equilibrium with back diffusion and the approach to this equilibrium must be considered. For the present purpose, only the simpler and apparently more useful equilibrium theory will be presented.

Consider a spherical cell which bears a population of one species of negatively charged molecules or particles on its surface, and which is placed in a uniform field (Fig. 1). Neglect any interactions (other than hydrodynamic drag) between various surface components. Allow them to come to equilibrium between electrophoresis which tends to concentrate them at the positive pole and back diffusion. It will be shown that the equilibrium surface concentration, C, is given by

$$C = \overline{C}[\varepsilon \cdot csch(\varepsilon)]e^{\varepsilon \cos \theta}, \qquad (1)$$

where $\overline{C}$ = the particles' average concentration, $\varepsilon = (m/D) \cdot (\overline{V}/2)$, m = electrophoretic mobility, D = diffusion constant,

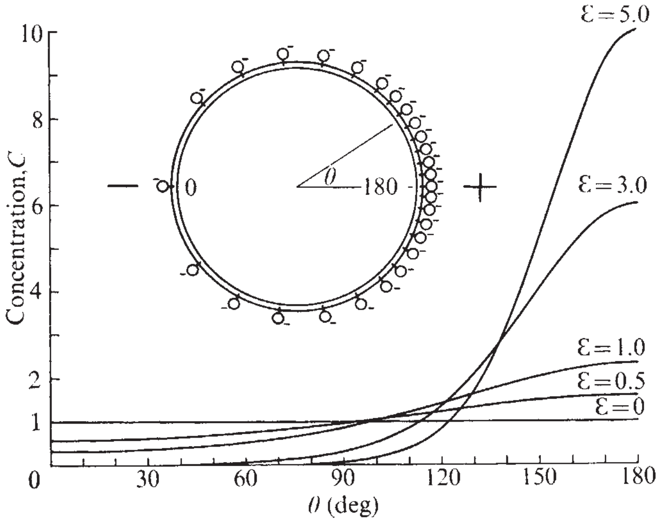


Fig. 1 Distributions of mobile charged particles on the surface of a sphere at electrophoretic equilibrium. Curves plot equation (1).

and $\bar{V}$ = voltage difference established across the sphere by the field. This is plotted in Fig. 1.

The particles are at a true equilibrium between several forces produced by the external field and Brownian movement, so it might be supposed that a Boltzmann distribution could be applied. Their counter ions are not, however, in equilibrium but in a steady state, so this most direct method cannot be used. Instead one writes the equation for a balance between electrophoretic and diffusional transport

$$mC(\mathrm{d}V/\mathrm{d}S) = D(\mathrm{d}C/\mathrm{d}S) \tag{2}$$

A sphere is known to distort an otherwise uniform field so that the potential just outside its surface, V, remains proportional to cosθ (ref. 11). Hence

$$V = (\bar{V}/2)\cos\theta \tag{3}$$

Substituting equation (3) in (2) and integrating yields equation (1).

Exactly the same derivation holds for the distribution of particles attached to the inner surface of a cell's membrane and concentrated by a uniform internal field, except that in this internal case $\bar{V}$ would refer to the transcytoplasmic potential difference. A uniform internal field might be set up by either a uniform external one[11] or by a cosine varying current source in the cell's own plasma membrane[12].

For comparison with cells' galvanotropic responses (or perhaps other polar responses) it is helpful to characterise the degree of polarisation of each of these equilibrium distributions[1] by some parameter, φ. The tropistic responses of cell populations are commonly characterised by the weighted cosine of the distribution of outgrowth angles (13)

$$V_1 = \Sigma n \cos\theta / \Sigma n \tag{4}$$

where n is the number of outgrowths at an angle, θ.

Hence we define φ in an analogous way

$$\varphi = \int C \cos\theta \, \mathrm{d}s / \int C \, \mathrm{d}S \tag{5}$$

Note that if the outgrowth angles or surface particles were all concentrated at one pole or the other, V_1 and φ would be +1 or −1 whereas if they were evenly distributed, these parameters would have a value of zero.

Integration of equation (5) yields

$$\varphi = ctnh\,(\varepsilon) - \varepsilon^{-1} \tag{6}$$

Use of Pierce's series[14] number 793 yields

$$\varphi = \varepsilon/3 - 0.133\,\varepsilon^3 + 0.127\,\varepsilon^5 \dots . \tag{6a}$$

This suggests that for $\varepsilon < 1$, φ is usefully approximated by

$$\varphi \simeq \varepsilon/3 = (m/D)(\bar{V}/6). \tag{6b}$$

Equations (6) and (6b) are plotted in Fig. 2.

Expected polarisability of real membrane particle distributions

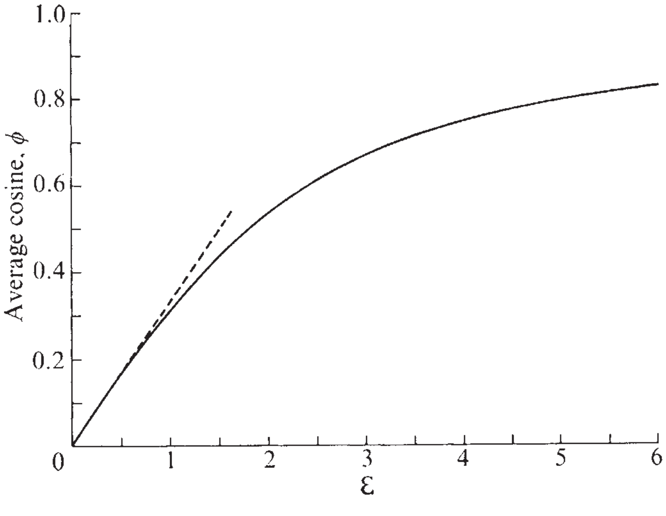
Let us characterise the polarisability of a particle distribution on a cell by the voltage $\bar{V}_{0.1}$ required to induce a polarisation of 0.1. From equation (6b) one gets

$$V_{0.1} = 0.6\,(D/m) \tag{6c}$$

To estimate this voltage, one must estimate the diffusion to mobility ratio, D/m of the particles being redistributed. We will show below that the larger and more highly charged particles found in cell membranes are likely to have D/m values of about a millivolt. So we predict that long-lasting fields, whether endogenous or imposed, are likely to bring about significant electrophoresis along cell membranes when the voltage drop per cell is only one to a few millivolts.

To estimate D/m values, we will first show that D/m is independent of the drag, f, which they encounter. This drag is an estimated 100–1,000 times greater than such particles would meet if they were only hindered by the extracellular medium[2]. If this greater drag were exerted by the viscosity of the medium which fills and surrounds their electrical double layer, then D and m are both well known to vary with f^{-1}. Hence D/m would be independent of f. It is likely, however, that this extra drag is exerted on a sort of molecular clog, that is, a part or extension of each particle that lies within the lipid bilayer and is not surrounded by a field-sensitive double layer; whereas the electrical pull is exerted on each particle's 'sail', that is, its part that extends outside of the bilayer in the external aqueous phase (Fig. 1). Then m would equal F, the net electrophoretic force per field (which acts on the sail), divided by the drag which acts on the clog. Henry's analysis[15], directly confirmed by Sumner and Henry's experiment[16], shows F to be unchanged whether the particle is free or tethered. Hence m is inversely proportional to the drag wherever it originates. Since this also holds for D (ref. 17), D/m is again independent of the drag.

Fig. 2 Average cosines of the distributions shown in Fig. 1. The curve plots equation (6); the dashed line, equation (6b).



Since D/m values are independent of the drag, we can use the values known for molecules or particles immersed in the usual 1-cP aqueous media. In media with this viscosity (and an ionic strength of 0.05 M) most biological molecules or particles have electrophoretic mobilities in the range from 1 to 3, or occasionally up to 5×10^{-4} cm s^{-1} (V cm^{-1})$^{-1}$ (refs 5,18). Electrophoretic mobilities are independent of the component's radius, but diffusion constants are inversely proportional to it. So to estimate how small a voltage drop per cell may be expected to bring about significant redistribution, let us consider relatively large membrane particles. Let us consider spherical particles with 10-nm diameters since the larger particles seen in freeze-fracture electron micrographs of plasma membranes are of about that size and shape[19–23]. In 1-cP media, such 10-nm spheres have a diffusion constant of 4×10^{-7} cm^2 s^{-1} (ref. 24). Then considering particles with a mobility value of 3×10^{-4} units, which is towards the upper end of those usually found, one obtains a D/m value of 1.3 mV and applying equation (6) we infer that the distribution of such particles would be one tenth to one half polarised by a steady voltage drop of only 0.8–4.0 mV.

Application of this equilibrium theory is restricted to the action of sufficiently long-lasting fields. The time needed is of the order of L^2/D where L is the cell's pertinent diameter and D the segregated components diffusion constant. The larger mobile components of cell membranes tend to have diffusion constants in the vicinity of 10^{-9} cm^2 s^{-1} (ref. 2), so for a 30-μm (that is, 3×10^{-3}-cm) cell, the equilibrium approach time might be about 10^4 s, or 3 h. When one considers that the bioelectric fields associated with growth and development generally last for hours to days or even weeks (that is, 10^4 to 10^6 s), one concludes that an equilibrium theory may be widely relevant.

The accompanying paper, by Poo and Robinson[25] seems to directly demonstrate the predicted phenomenon of lateral electrophoresis along the surface of explanted, embryonic muscle cells exposed to an artificially imposed field. Moreover, a number of galvanotropic responses of isolated plant cells to artificial fields occur in response to such small voltage drops as to suggest mediation by lateral electrophoresis[26–28].

Natural fields along cell membranes

Perhaps the most obvious place where large endogenous voltage drops occur along cell membranes is along the outside of the lateral membranes of cells in various metazoan epithelia. Various mature epithelia are well known to maintain voltage drops of 30–100 mV or more across themselves[29]. Furthermore, there are several reports of 3–10 mV potential drops across embryonic cell layers, for example, the mammalian blastocyst wall[30], a fish gastrula wall[31], and the chick chorioallantoic membrane[32]. A second place where good evidence for significant steady potentials drops along cell membranes can be cited is along the outer plasma membranes of epidermal cells in the coleoptiles of oat seedlings. A voltage drop of at least 40 mV (refs 33, 34) is measured along the axis of these organs and hence across about 30 cells in series[35]; whereas up to 80 or 90 mV appears across the axis in response to a gravitational stimulus[36,37] and thus across about 100 surface cells in series[35]. Thus, in both directions, about 1 mV per cell is present.

There are, moreover, at least three instances in which there is evidence that cells generate large enough cytoplasmic fields within themselves electrophoretically to segregate components of the inner leaflet of the plasma membrane. Woodruff and Telfer have quite directly measured a voltage drop of about 10 mV across the cytoplasmic bridge joining a developing insect oocyte and its nurse cell[38]; Hagins has measured a steady dark current outside of rat visual rod cells and infers an internal current sufficient to maintain a steady difference of about 1.6 mV along the interior of the cell[39]; while Robinson and Jaffe have measured a calcium current through developing fucoid eggs which might maintain the order of a millivolt difference across the interior of the cell[40,41].

I conclude that endogenous voltage gradients sufficient to bring about electrophoresis along cell membranes are very widespread in living organisms.

Received August 19; accepted December 21, 1976.

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Electrophoresis of concanavalin A receptors along embryonic muscle cell membrane

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Fluorescent concanavalin A (con A)-labelling showed that an electric field of 4 V cm^{-1} grossly redistributed con A receptors along the plasma membranes of living muscle cells within 4 h. This field produced a voltage drop of 12 mV across these 30 μm-wide cells. The movement of receptors was independent of cell metabolism and seemed to be electrophoretic in nature.

STUDIES of the cell membrane have demonstrated the dynamic nature of membrane constituents[1–5]. Both proteins and lipids may undergo rotational and translational diffusion in the plane of the membrane[6]. In the accompanying paper[7], Jaffe demonstrates on theoretical grounds that small voltage

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