A note on the evolution of the transducer mechanism of the vertebrate retinal rod

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Summary. The Ca^{2+}-gating mechanism that is the key component of membrane excitability in Paramecium has recently been localized on the cilia of this animal. Such a finding shows how the change in Ca^{2+} permeability (which is the probable consequence of the photoisomerization of rhodopsin) of the discs of the outer segments of the retinal rods could have evolved.

Recent studies on the control of the ciliary beat in Paramecium reveal how the transducer mechanism of sense organs, in particular the vertebrate retinal rods, might well have evolved. A mechanical stimulus at the anterior end of Paramecium causes an increase in calcium permeability, with a consequent depolarization which further activates the Ca^{2+}-channels in the membrane. Depolarization, therefore, develops regeneratively towards the equilibrium potential for Ca^{2+}. The resultant rise in $[Ca^{2+}]_i$ causes a reversal of the ciliary beat together with an increase in frequency[1,2]. Depolarization produced by raising extracellular Na^+ or K^+ also causes ciliary reversal, and recent measurements with $^{45}Ca^{2+}$ have confirmed an associated rise in Ca^{2+}-influx[3]. A stimulus at the posterior end of Paramecium, however, activates K^+-channels in the membrane, causing hyperpolarization and, again, an increase in the frequency of ciliary beat[1,2]. Mutants of P. aurelia with modified locomotor behaviour (named 'Pawn') have been isolated[4] and these animals fail to reverse their ciliary beat when depolarized. Such mutants show no regenerative Ca^{2+} response[5] and no stimulated $^{45}Ca^{2+}$-influx, and it is evident that the Ca^{2+}-gating mechanism is a major component of membrane excitability in Paramecium[3].

When Paramecium is deciliated by treatment with chloral hydrate, the animals retain their hyperpolarizing response to posterior stimulation and the normal resting membrane potential is unaffected. However, deciliated Paramecium do not show a depolarizing response to anterior stimulation and a stronger stimulus now causes hyperpolarization, even if applied at the anterior end. The cilia regenerate in the absence of chloral hydrate in 6–8 h and the reciliated Paramecium once more respond with depolarization to anterior stimulation[6]. These experiments confirm studies by Dunlap and Eckert[7,8] that deciliation eliminates the 'calcium response' to depolarizing current whilst regrowth restores the normal response. Such findings strongly suggest that the Ca^{2+}-channels are localized on the ciliary membrane, whereas the K^+-channels are not[6].

It has been emphasized previously how many sensory transducers are based structurally on cilia[9,10], as for example retinal rods, olfactory cells and the kinocilia of the organ of Corti. The vertebrate retinal rod is of particular importance in this respect; the modification of its outer segment from a cilium has been traced during morphogenesis and the remnants of the microtubular structure, together with the centrioles, persist in the stalk of the mature cell. The retinal discs of the outer segment form by infolding of the plasma membrane of the cilium[11]. Photoisomerization of the rhodopsin molecule initiates the sequence of events that culminate in the hyperpolarization of the cell which is produced by a reduction in Na^+-permeability[12]. It has been suggested that a molecular reorientation of the visual pigment causes a change in the Ca^{2+}-permeability of the discs, so allowing the release of Ca^{2+} stored therein which acts as an intracellular messenger, amplifying and coupling the photochemical signal from the disc to the bounding plasma membrane[13,14]. This hypothesis has received support from experiments in which a) Ca^{2+} has been shown to be concentrated in dark-adapted discs[15]; b) Ca^{2+} release has been demonstrated on bleaching[16–19]; c) a Ca^{2+}-ATPase, presumably associated with the reuptake of Ca^{2+}, has been localized in receptor cell outer segments[20]; d) measurements with aequorin showed that $[Ca^{2+}]_i$ rises in Limulus photoreceptors during illumination[21]; e) treatment of rod outer segments with Ca^{2+} causes their hyperpolarization[22].

Thus, the Ca^{2+}-gating mechanism of the transduction process of the mechanosensory response of Paramecium cilia provides circumstantial evidence for the existence of Ca^{2+}-permeability changes in the response of vertebrate photoreceptors that are developed from modified cilia. More important, the studies on the sensory response in Paramecium reveal how the transducer mechanism of retinal rods could have evolved. Early photoreceptors could have developed by linking the Ca^{2+}-gates on cilia to a photopigment whose photoisomerization caused a modification of Ca^{2+}-permeability. The subsequent formation of the characteristic discs by the infolding of the plasma membrane means that these were equipped with the appropriate Ca^{2+}-gating mechanism together with the vectorially organized Ca^{2+}-transport ATPase which translocates Ca^{2+} into these storage sites rather than to the external medium.

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