Evidence for an Active Process and a Cochlear Amplifier in Nonmammals

GEOFFREY A. MANLEY
Lehrstuhl für Zoologie, Technische Universität München, 85747 Garching, Germany

Received 26 January 2001; accepted in final form 20 March 2001

Manley, Geoffrey A. Evidence for an active process and a cochlear amplifier in nonmammals. J Neurophysiol 86: 541–549, 2001. The last two decades have produced a great deal of evidence that in the mammalian organ of Corti outer hair cells undergo active shape changes that are part of a “cochlear amplifier” mechanism that increases sensitivity and frequency selectivity of the hearing epithelium. However, many signs of active processes have also been found in nonmammals, raising the question as to the ancestry and commonality of these mechanisms. Active movements would be advantageous in all kinds of sensory hair cells because they help signal detection at levels near those of thermal noise and also help to overcome fluid viscosity. Such active mechanisms therefore presumably arose in the earliest kinds of hair cells that were part of the lateral line system of fish. These cells were embedded in a firm epithelium and responded to relative motion between the hair bundle and the hair cell, making it highly likely that the first active motor mechanism was localized in the hair-cell bundle. In terrestrial nonmammals, there are many auditory phenomena that are best explained by the presence of a cochlear amplifier, indicating that in this respect the mammalian ear is not unique. The latest evidence supports siting the active process in nonmammals in the hair-cell bundle and in intimate association with the transduction process.

INTRODUCTION

The great sensitivity of sensory systems has often been a source of amazement to neuroscientists. Over the years, evidence has accumulated indicating that in many cases, sensitivity has essentially reached its “theoretical” maximum (e.g., Hudspeth 1997). In the case of the vertebrate auditory inner ear, sensitivity has been increased by a number of mechanisms, including using the external ear—or its equivalent, e.g., in the facial disk of owls—to funnel sounds to the eardrum. By this and other means, behavioral sensitivities down to −20 dB SPL (2 μPa) have been achieved (Fay 1988). Further increases in sensitivity would not seem to be useful for three reasons—because of much higher levels of external noises that mask signals, because of thermal noise that becomes very significant at these levels, and because of the drag exerted by the viscosity of the fluids surrounding the hearing organ (Hudspeth 1997).

Exactly how the mammalian ear—including the healthy human ear—achieved its high sensitivity was a mystery for a long time. The earliest measurements of the motion of inner-ear components were made at unphysiologically high sound pressure levels (von Bekesy 1960). Extrapolating from these measurements, von Bekesy concluded that at threshold, inner-ear sensory cells responded to motions equivalent to the diameter of hydrogen atoms. These early results added fuel to discussions of mechanisms of achieving such sensitivity in the face of molecular noise sources and the viscosity of inner-ear fluids. In 1948, Gold (1948, cited in Kemp 1978) had already concluded that using only passive responses to sound, the inner ear simply could not be as sensitive as it is. His conclusion was that in some way, the inner ear must contain an amplifying mechanism that released coherent mechanical energy into the cellular system and thus boosted sensitivity at low sound levels.

Gold’s speculations were untestable at the time of their publication since contemporary measurement equipment was too insensitive and too slow. In 1978, Kemp discovered what he termed “echos” being emitted with a short delay by the ear when stimulated with brief sound stimuli. Kemp was soon able to show that these signals were in fact not echos since they sometimes contained more energy than was present in the stimulus itself. These were sounds actively emitted from the inner ear in a way that could have been expected from Gold’s predictions if the energy produced by the inner-ear amplifier was not being perfectly absorbed by the organ of Corti.

This discovery of what over the years came to be known as otoacoustic emissions was mildly revolutionary for research in peripheral hearing mechanisms. At one and the same time, it provided evidence that there was, in fact, an active process in the inner ear, and it also made it possible to investigate exactly those properties of the hearing organ noninvasively. For the first time, a technique became available to carry out the same objective and nondestructive tests on human and animal cochleae, in this case by measuring the characteristics of sounds emitted by the ear. The assumption of cochlear amplification also had powerful explanatory value for earlier findings on, for example, the deleterious effects of hypoxia on the sensitivity and frequency selectivity of hearing, and on the physiological consequences of damage to hair cells on cochlear frequency selectivity. Davis (1983) suggested that such an active process underlies all sensitive hearing and coined the term “cochlear amplifier” as a global designation for as then undescribed mechanisms that feed mechanical energy into hair-cell motions with a phase appropriate, at some frequencies at least, to strongly increase the cells’ sensitivity.
It is not my purpose in this paper to review the evidence that indicates that the mechanical energy really derives from the sensory hair cells themselves or to describe the many pieces of work that have attempted to localize and dissect the motor mechanism at the level of single cells. For this, the reader is referred to previous reviews (e.g., Ashmore 1992; Ashmore and Kolston 1994; Dallos et al. 1993; Hudspeth 1989, 1997; Pickles 1993). Suffice it to say that in mammals, there is evidence that the specialized outer hair cells (OHC) have an amplification motor consisting of densely packed protein complexes located in the lateral cell membrane. Activation of conformational changes in these proteins causes cell contraction and elongation that in turn increase the amplitude of the vibrational patterns of the organ of Corti.

The focus of this paper is on the question as to whether this phenomenon, which has played such an important role in research on the mammalian cochlea for the past 18 yr, is unique to mammals. Certainly most of the literature on the subject of active processes in the inner ear has derived from work on mammals, and it has become an unspoken expectation—in the long and harmful tradition of anthropocentric views on science—that in this regard the mammalian—and thus human—hearing organ is very special. In the words of cladistic phylogeny, the active process is often regarded as an apomorphy of mammals—a feature newly derived during the early evolution of the mammals. Is this so or are active mechanisms much older (Manley and Köppl 1998)?

To avoid terminological confusion, it is useful here to clarify the meaning I attach to some terms that will appear repeatedly in this paper. The term “active mechanism” will be used for any cellular-biochemical or -biophysical process that is capable of generating a force that produces motions of hair-cell parts at frequencies in the audio range. The term “cochlear amplifier” is used for the summed effects of the active process as seen in phenomena measurable in the organ itself, in the responses of the auditory nerve afferent fibers, or as otoacoustic emissions. Thus cochlear amplification is the result of an active process at the hair-cell level. Since the term “cochlear amplifier” was coined by Davis (1983) before the underlying cellular mechanisms were known, it cannot be claimed that this term should be restricted to describing what happens in mammals.

In recent years, it has become customary to refer to a “cochlea” in lizards, birds, and even amphibia, animals in which the hearing organ is enclosed in bony recesses that are differently shaped and not coiled as in mammals. Thus the term cochlear amplifier can—if active processes are found—also be extended to cover the phenomena seen in all terrestrial vertebrates. Finally, I shall use the term stereovillar bundle rather than stereocilia bundle of the hair cells since the structure concerned is essentially not made up of cilia but of large microvilli.

HISTORICAL PERSPECTIVE

Since the early 1980s, many cochlear models had already been based on the assumption of the existence of some kind of cochlear amplifier, but the authors had used other terms to describe the phenomenon, such as feedback motor, negative damping, active feedback, reverse transduction, etc. The basic idea in all cases was that hair cells can react in a frequency-selective fashion to faint sounds with a response that in some way favorably affects the local motion of the hearing organ. Initially, the molecular basis of active processes was not considered to be important for creating models, it was simply assumed that they were fast enough to enhance cycle-by-cycle motion at auditory frequencies. Nonetheless, the elucidation of the molecular basis of the active process quickly became a focus of physiological studies, and nonmammalian data have played an important role in this undertaking.

From an evolutionary perspective, dissipative losses in hair-cell motion due to the drag caused by fluid viscosity is a problem that confronts hair cells of all systems (lateral line, vestibular, auditory). It would thus not be surprising if an active process that reduces these losses originated early in the evolution of hair-cell systems and thus early in vertebrate evolution. Ancestral systems had no hair-cell specializations such as seen in the different kinds of hair cells of the cochlea of mammals and birds, and they also had no freely moving basilar membrane supporting the hair cells and that could have been driven as part of a micromechanical resonance system. The hair cells of the lateral line and vestibular organs were and are firmly embedded in the tissue complex of the sensory organ. The ancestral amplifier motor mechanism must thus have been sited in the hair-cell bundle (Manley and Köppl 1998) as it was the only freely moving component of the system. Could such hair-cell bundles perform useful work? The amount of work done by hair cells to produce the energy in otoacoustic emissions is very small, and is well within the capabilities of several different potential motor mechanisms driving the bundle (Hudspeth 1997; Manley and Gallo 1997).

The first cochlear models to assume an active process (e.g., Kim et al. 1980) incorporated negative damping into their equations and were quite successful at modeling the then-known behavior of the cochlear partition. As those authors stated: “It is unknown what kind of mechanism may underlie the hypothesized internal energy source in the cochlear partition.” Our conjecture is that energy available through cellular metabolism in the cochlear partition may be somehow transduced into mechanical energy.” In a later, more complex model, the source was later assumed to be in the outer hair cells (Neely and Kim 1983).

An active process in the hair-cell stereovillar bundle

The first model that proposed a specific cellular location for the active process implicated the stereovillar bundles of the hair cells. Weiss (1982) assumed that hair-cell bundles would move under the influence of electrical potential changes of the cell membrane. Since such changes in electrical potential could be the consequence of the transduction of sound stimuli, the resulting motion of the hair-cell bundle—that is also responsible for stimulus transduction—would set up a feedback mechanism. This feedback, if occurring with appropriate phase relationships to the original signal, could amplify the stimulus. Following the discovery of whole cell motion of outer hair cells (see following text), Kim (1986) incorporated into his model two active processes: the hair-cell bundle mechanism as the basis of a fast cellular motor and a cell-membrane mechanism driving whole cell motion and underlying a slow motor.

Between 1985 and 1990 several research groups, using in vitro preparations, showed that in some hair-cell systems at least, the bundles are indeed capable of active motions. Fol-
following Crawford and Fettiplace’s (1985) description of such bundle properties in the turtle auditory organ, Howard and Hudspeth (1987) demonstrated the presence of an adaptational motor system in the stereovillar bundle of saccular hair cells from the frog. Also using vestibular hair cells of the frog sacculus, Denk and Webb (1989) found a correlation between intracellular voltage noise and the bundle position that suggested a reverse-transduction process just as Weiss (1982) had proposed. Recent measurements on lateral-line organs revealed similar nonlinearities to those associated with low-level amplification and thus support the idea that an active process evolved very early in the evolution of vertebrate hair cells (van Netten 1997).

Similarly Rüsch and Thurm (1990) described movements of both the kinocilium and the stereovilli of ampullary hair cells of the vestibular system, movements that were both spontaneous and could be electrically induced. In the same year and using saccular hair cells, Jaramillo et al. (1990) demonstrated that hair bundles could execute more rapid movements than those previously observed during adaptation (e.g., Assad et al. 1989) and that these movements were strongly influenced by the concentration of calcium ions. In a similar vein, Benser et al. (1996) demonstrated rapid bundle movements whose frequency components lay in the same range as those necessary for normal saccular responses. More recently, Hudspeth’s group has demonstrated that hair-cell bundles are really capable of doing work by providing clear confirmation that the bundle actually amplifies the response to mechanical stimuli (Martin and Hudspeth 1999). Ricci et al. (2000) provided further evidence for a calcium-sensitive force generator linked to the gating of the transducer channels in turtle auditory hair cells. Finally, Manley et al. (2001) described evidence from in vivo experiments using ac electrical current injection into the cochlea of the bobtail lizard that can only be explained on the assumption that an active process is present in the hair-cell bundle.

To close this historical section, two points should be made. At present, the motor mechanism driving the hair-cell bundle is not fully understood and there is no evidence for or against such a mechanism in mammals, although an active process in the bundle in mammals is held by some authors to be at least as appropriate as the oft-discussed active process that is sited in the cell membrane (Yates and Kirk 1998).

**An active process in the hair-cell membrane**

In 1985, the same year as Crawford and Fettiplace were providing evidence to support the then-current models of feedback systems in the hair-cell bundle, Brownell et al. (1985) reported that isolated outer hair cells of mammals move in response to electrical fields by shortening or lengthening the entire cell. This finding unleashed efforts in many labs to describe the phenomenon in detail and quantify its capabilities (e.g., Zener et al. 1985) and, of course, to incorporate the idea into cochlear models (e.g., Neely and Kim 1986a,b). These data have been the subject of a number of reviews (e.g., Ashmore 1992; Dallos and Evans 1995; Hudspeth 1989) and will not be repeated here. Today, the majority of papers describe the active process in mammalian auditory outer hair cells as resulting from very fast configurational changes in densely packed protein complexes of the basolateral hair-cell membrane. Studies of possible movements of hair-cell bundles in mammals are few.

Since there is no evidence from nonmammals for hair-cell motion based on such cell-membrane components, it has been tacitly assumed that cochlear amplification is a process unique to mammals. In fact, the available evidence clearly shows that the physiological phenomena in mammals that are usually attributed to a cochlear amplifier are universally present in tetrapods, suggesting that active processes in hair cells are an ancient—synplesiomorphic—feature of tetrapod vertebrates. An active mechanism may even have been inherited from the fishes.

**HOW DO WE RECOGNIZE THE EXISTENCE OF AN ACTIVE PROCESS?**

This question can—since almost all work to date has been carried out on mammals—initially be reduced to the questions: What is accepted as evidence for the existence of a cochlear amplifier in mammals? How does the underlying active process—OHC producing motion that influences the micromechanical environment and thus the movement of the whole organ—manifest itself?

In mammals, a cochlear amplifier is taken to underlie many phenomena, such as the high sensitivity to sound, the narrow frequency selectivity (tuning) of hair cells or their afferent nerve fibers, the typical patterns in rate-level functions of primary auditory neurons, the sensitivity of both threshold and frequency selectivity of auditory afferent nerve fibers to hypoxia and other insults, and the presence and characteristics of otoacoustic emissions. Is similar evidence available from nonmammals? We can consider the preceding points in turn.

Trivial comparisons show that some nonmammals have hearing systems that are as sensitive as those of the most sensitive mammals. If we take, for example, the two most sensitive representatives of the endothermic vertebrates, the cat for the mammals and the barn owl for the birds, it is clear that there is no difference between their best sensitivities (Fig. 1). It is true that the cat—like many other mammals—has an

![FIG. 1. Behavioral audiograms for the domestic cat (Fay 1988) and the barn owl (Konishi 1973). These hunters are among the most sensitive mammals and birds: their absolute sensitivity is remarkably similar.](http://jn.physiology.org/)

*J Neurophysiol* • VOL 86 • AUGUST 2001 • www.jn.org
upper frequency limit much higher than that of any bird, but there is as yet no evidence that this is related to peculiarities of cochlear amplifiers. It is more likely related to the evolutionary accident that the three-ossicle middle ear of mammals transfers higher frequencies better than do the columellar middle-ear systems of nonmammals (Manley 1990). Thus if a high absolute sensitivity in mammals is a manifestation of a cochlear amplifier, then at least some birds must also have one.

The frequency selectivity of single primary auditory nerve fibers can be very high. Once it became possible to measure the motion of the organ of Corti in uncompromised preparations (e.g., Sellick et al. 1982), it became clear that in mammals, a high-frequency selectivity is intrinsic to the entire organ and is produced by coupled interactions between the hair cells and the micromechanics of structures of the entire local organ. Thus the frequency selectivity, or tuning, of afferent nerve fibers strongly reflects the workings of an active process. Interestingly, frequency selectivity in mammals is usually less selective that that seen in nonmammals at the same frequencies (Fig. 2). In the cases shown, frequency selectivity measured as the Q10dB coefficient (the center frequency divided by the frequency bandwidth at 10 dB above the best sensitivity) is highest in primary afferents from a bird, the emu. In a lizard, the Tokay gecko, tuning is not as high as in the emu but still substantially higher than in the guinea pig, an animal that has been extensively studied with regard to tuning of the entire organ of Corti. Of course, examples could be chosen where the differences are not as great as illustrated here, but this does not detract from the fact that in general, the frequency selectivity in mammals over the frequency range where nonmammals can also hear is not especially great. If an active process is necessary for establishing frequency tuning in mammals, then it is can equally well be considered necessary for the more selective tuning in nonmammals. The possibility exists, of course, that some of the selectivity in nonmammals is due to electrical tuning of the hair cells, which would not be dependent on a cochlear amplifier process. However, the evidence for electrical tuning at higher frequencies is weak (e.g., in birds, Gleich and Manley 2000). In lizards, there is no evidence for electrical tuning in cells with best frequencies higher than 1kHz (refs in Manley 1990, 2000).

For a number of years, it was difficult to explain the different shapes of the transfer functions (spike rate vs. sound level) of primary auditory nerve fibers of mammals. These rate-level functions describe the increase in the rate of spike discharge to increasing sound levels, generally at the most sensitive (characteristic) frequency (Sachs and Abbas 1974). However, Sachs and Abbas (1974) and Yates (1990) and colleagues (Yates et al. 1990) were able to show that these different forms of
rate-level functions were intimately related to the nonlinear response of the organ of Corti to sound pressure. This nonlinearity is directly due to the active process, and disappears in a damaged organ, in parallel to the loss of sensitivity and selectivity of the neural elements. Thus the shapes of transfer functions can also be taken to be an indicator of the presence of an active process that enables interactions between hair-cell populations. Interestingly, all three shapes of transfer function (flat-saturating, sloping-saturating, and straight) have also been described from two avian species (Fig. 3) (Köppl and Yates 1999; Yates et al. 2000). Their relationship to the absolute sensitivity of the neurons studied is different to that described for the guinea pig, but Köppl and Yates suggested that in birds the active process acts more locally rather than globally as in the mammalian organ of Corti (Yates et al. 1990). In lizards, only one kind of transfer function is found for the low-frequency region and another for the high-frequency region (Eatock et al. 1991). Since interactions between populations do not affect lizard transfer functions, they have been omitted from Fig. 3.

As might be expected, the active process in mammals—at least in the whole organ in vivo—is highly dependent on a normal physiological state. Any kind of damage, such as brief hypoxia, leads to a rapid loss of both sensitivity and frequency selectivity of primary afferent fibers. This loss is reversible on restoration of the normal oxygen supply (e.g., Robertson and Manley 1974). However, hearing sensitivity and tuning in nonmammals is similarly compromised by hypoxia. This process is rapid in birds, which are highly dependent on a constant supply of oxygen, but is in principle exactly the same in lizards, whose oxygen requirements are lower and who thus respond more slowly to this insult (Fig. 4).

Finally, as in mammals, there are numerous reports of OAE in nonmammals (see e.g., Köppl 1995; Manley and Taschenberger 2000). The spontaneous otoacoustic emissions (SOAE), which can be measured in the absence of any sound stimulus and can be traced to spontaneous movements of hair cells, are regarded as the clearest and even the most dramatic evidence that hair cells contain an active process. Both spontaneous and evoked emissions have been described in amphibians, lizards, and birds (Fig. 5). More than this, there are clear and sometimes startling similarities between mammalian and nonmammalian OAE. For example, in all species, each emitting ear shows an individual pattern of SOAE frequencies (Fig. 6).
SOAE are also physiologically vulnerable, vanishing reversibly under brief hypoxia or even (in nonmammals) when the anesthetic level is too deep. The statistical properties of the amplitude distributions of SOAE in frogs, lizards, and birds show that they originate, as in mammals, from hair cells acting as sine-wave oscillators (van Dijk et al. 1996). Also, the center frequency of each individual SOAE spectral peak in mammals and nonmammals is sensitive to the temperature of the inner ear (e.g., Manley 1997; Ohyama et al. 1992) (Fig. 7).

In all species, also, SOAE can be influenced by tones added via a loudspeaker, and the reactions of the emission peaks are very similar whether measured in a mammal or in a nonmammal. Depending on the frequency and level of the tone and its nearness in frequency to an emission peak, SOAE can react to tones in the ear canal with frequency-specific suppression (i.e., a fall in amplitude of the emission), facilitation (an increase in amplitude) and/or shifts in the center-frequency of the SOAE peak. Using tones of different frequencies and levels, a threshold curve can be plotted that describes the sound-pressure level at each stimulus frequency that is necessary to suppress the SOAE amplitude by a criterion amount. This is known as the suppression tuning curve (STC) of the individual SOAE (e.g., Köppl and Manley 1994). These STC strongly resemble primary-neural tuning curves (Fig. 8), and this is one of the strongest pieces of evidence that the SOAE arise from groups of hair cells that at the same time form the tuned units giving rise to auditory-nerve fiber excitation.

Some comparisons between mammalian and nonmammalian otoacoustic emission properties have yielded results that are remarkably similar. In both human and lizard OAE, for example, the frequency distances between SOAE peaks are on average essentially identical in spite of the fact that the mammalian cochlea has a length of more than 30 mm, the length in the lizards so far used in measurements varies from 0.2 to 2 mm. Even the frequency distances over which interactions occur between SOAE peaks (as manifest in correlated amplitude fluctuations) are almost the same in both animal groups. In both cases, such influences are only found up to a between-

**FIG. 6.** Spontaneous otoacoustic emission spectra from 4 different species of lizards representing 4 different families, as follows: *Callopistes maculatus*, a teiid; *Varanus exanthematicus*, a varanid; *Gekko gecko*, a gekkonid; and *Anolis sagrei*, an iguanid lizard. In all cases, the sound spectrum in a closed acoustic system around the eardrum was averaged 200 times. The spectral patterns seen were stable in all except the iguanid over long time periods (weeks, months). Note the extended frequency axis for *Anolis*. 

A

![SOAE spectrum for *Callopistes*](image)

B

![SOAE spectrum for *Varanus*](image)

C

![SOAE spectrum for *Gekko*](image)

D

![SOAE spectrum for *Anolis*](image)
peaks frequency ratio of 1.6, despite the difference in the absolute size of the hearing organs (and thus the absolute distance between the same two frequencies in each ear) (van Dijk et al. 1998). In humans, the hearing organ is 35 mm long, but in lizards it is only between 0.2 and 2 mm in length (Manley 1990).

More recently, electrically evoked emissions (EEOAE), that in mammals are thought to derive from outer hair cell movements driven by the injected current, have also been demonstrated in lizards (Manley et al. 2001).

Is the motor mechanism of mammalian and nonmammalian OAE the same?

Considering all the reported data on OAE in mammals and nonmammals, it can be said that there are only minor differences between the phenomena in these groups. Thus these data—and indeed all the phenomena attributed to the active process—provide no a priori reason to suppose that mammals have evolved a special mechanism for driving the cochlear amplifier. Nonetheless, the literature, especially on single cell, in vitro studies, does contain evidence for two possible sources of the active mechanical input—one in the hair-cell bundle and one in the cell membrane wall of outer hair cells. The former has only been described in nonmammal hair-cell systems and the latter only from mammalian outer hair cells. Are we to conclude that, although all terrestrial vertebrates show strong evidence for an active process in their hearing organs, and the diverse phenomena described are so remarkably similar between the groups, that the cochlear amplifier is based on two totally different mechanisms? Is there an evolutionarily older motor mechanism based in the hair-cell bundle that arose in lateral-line hair cells and was “inherited” by the vestibular and auditory systems? And is there a new, membrane-based mechanism that arose much later and is only found in the mammalian cochlea?

At present, this question cannot be answered with certainty. In nonmammals, all the available evidence points to a motor mechanism in the hair-cell bundle and away from a motor in the hair-cell lateral membrane. 1) An attempt to induce rapid hair-cell body motion in avian hair cells via current injection was unsuccessful (Brix and Manley 1994). 2) The hair-cell membranes of nonmammals do not show the dense macromolecular complexes thought to represent the membrane motors of outer hair cells (A. Forge, C. Köppl, and G. A. Manley, unpublished data). 3) In birds, the hair-cell population that is not afferently innervated (Fischer 1994), and thus most likely to be carrying the motor mechanism, consists of cells that are as short as 3 μm with virtually no lateral cell membrane.
active movements observed were similar to the fastest time served in turtle auditory hair cells. The time courses of the saccular hair cells (e.g., Benser et al. 1996) can also be observed in hair cells of mammals, which have only a sparse afferent innervation (Manley et al. 1989), these short hair cells of birds thus resemble the outer hair cells of mammals, which have only a sparse afferent innervation. It is hardly conceivable that these tiny cells could produce motion using their membrane, but basal hair cells do have remarkably robust stereovillar bundles containing up to 300 stereovilli (Fischer et al. 2000; Gleich and Manley 2000; Tilney and Saunders 1983). These stereovillar bundles could move the tectorial membrane (Manley 1995). 4 The patterns caused by low-frequency sound modulation of EEOAE in lizards provide clear support for a bundle motor (Manley et al. 2001). In lizard auditory papillae, the hair-cell bundle orientation patterns permit an unequivocal test as to whether the active-process motor lies in the hair-cell membrane or in the bundle (Fig. 9). 5 Ricci et al. (2000) have shown that many of the fast bundle movement phenomena already reported for saccular hair cells (e.g., Benser et al. 1996) can also be observed in turtle auditory hair cells. The time courses of the active movements observed were similar to the fastest time constants observed in hair-cell adaptation and were related to the activation time constants of the transduction channels.

On the other hand, there appear to have been no serious attempts to study whether in mammals, the hair-cell bundle is capable of motility. Some experimental data are easier to explain if the motility is in fact in the bundle (e.g., Yates and Kirk 1998). It is, of course, conceivable that even if mammals have developed a new kind of lateral-membrane motor mechanism, they have still retained the older bundle mechanism, raising the question as to how the hair cells might match the input phases of these two systems.

Summary

The various types of evidence accepted as indicating the presence of an active process driving a cochlear amplifier in mammalian hearing organs clearly also exist in nonmammals. Evolutionary considerations suggest that a bundle-based mechanism originated first: this mechanism certainly persists in nonmammals and is correlated with an auditory performance that is often equivalent to or better than that of mammals. In mammals, the active process appears to be at least partly driven by a membrane-based motor. Further work is necessary to examine the possible persistence of an additional, bundle-based motor.

REFERENCES


