ly, RP triggers ATP secretion by the sensory neuron located at the center of the hair bundle (Watson, Venable, Hudson, and Repass, 1999). The reversible inhibition of vibration dependent discharge observed at relatively high concentrations of ATP in animals preloaded with W7 or nifedipine is interesting in that it suggests that ATP may be affecting vibration sensitivity by at least two mechanisms, only one of which depends on CAM. Whereas the CAM dependent inhibition requires a 6 hr recovery, the CAM independent inhibition is rapidly reversible.

Taken together, it seems reasonable to speculate that components of the repair protein mixture regulate purinoceptors to enhance the repair process while also preventing the destructive potential of activated purinoceptors. The apparent involvement of purinoceptors in repair places at least two classes of molecules at the extracellular surface of damaged hair bundles: repair proteins, several different protein complexes each having an estimated mass of 2000 kD; and ATP. The ATP may serve both as an energy source for ATPase domains residing in at least some of the RP protein complexes (Watson et al., 1999), and also as a ligand for purinoceptors which initiate calcium transients associated with the repair process. Preliminary results from ongoing experiments suggest that elevated cytoplasmic calcium activates calcium calmodulin (CAM) to reorganize the actin cytoskeleton of stereocilia.

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