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The Endolymphatic Sinus is a Possible Detector of Endolymph Volume Status

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In the paper, in question (Salt and Rask-Andersen, 2004), we presented direct measurements of hydrostatic pressure and potassium concentration from the luminal space of the endolymphatic sac during pressure manipulations of the perilymph. Negative pressures (i.e. suction) applied to the perilymphatic space induced ionic, hydrostatic pressure and resting potential changes in the sac while positive pressures typically did not. Thus, our evidence for a one-way valve between the vestibule and the endolymphatic sac, which is now questioned by Wit and Hofman, was based on experimental measurements. As anatomic data in the same study showed the membrane of the endolymphatic sinus was in close proximity to the opening of the endolymphatic duct into the vestibule, we proposed that the membrane of the endolymphatic sinus was the anatomic structure acting as the valve. Wit and Hofman provide no new data to question the experimental findings of our paper yet they pose the question “Does the endolymphatic sinus function as a one way valve?” Since they have no experimental data conflicting with our original findings and suggest no alternative anatomic structure to act as the valve that was demonstrated experimentally, we can find no rational basis to doubt our original conclusions that the sinus membrane can act as a valve between the endolymphatic spaces of the vestibule and the endolymphatic duct.

Wit and Hofman go on to question the concept that the endolymphatic sinus could be involved in endolymph volume regulation. On the basis of their calculations, they conclude that “perilymph pressure has no influence [on endolymph flow]” and that “the model for the regulation of endolymph flow through the sinus, as proposed by Salt and Rask-Andersen (2004); Salt and Rask-Andersen (2005) has to be rejected”. We strongly disagree with both of these statements. The flow explanation described in their Figs. 2 and 3 that their subsequent arguments appear to disprove, appears to be of Wit and Hofman’s own making and is completely different from the interpretation we presented in our papers (Salt and Rask-Andersen, 2004; Salt, 2005). We have never suggested that perilymph pressure changes could induce a flow between the sacculus and the endolymphatic sinus in the manner presented in their Fig. 3. This is simply a “straw man argument” in which the concept they disprove is of their own making and has no bearing on the interpretation we presented in our paper.

In order to clarify the situation, we present in Fig. 1 a more complete explanation of the pressures and flows involved in our original interpretation. Fig. 1a diagrams the membranous endolymphatic structures (thin lines) within the non-compliant bony otic capsule (heavy lines). Since neither endolymph or perilymph are flowing at a high rate, it is reasonable to assume that perilymph pressure (P_p) and endolymph pressure (P_e) are uniform throughout each fluid space of the system. To our knowledge, no pressure differences between different parts of the endolymphatic system (P_1 - P_2 in Wit and Hofman’s Fig. 2; P_1 in Wit and Hofman’s Fig. 3) have ever been demonstrated. If the membranes exert a small pressure on the endolymph then

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$P_e = P_p + P_m$, as Wit and Hofman suggest, but as endolymph and perilymph pressures have been measured to be equal in the normal ear (Takeuchi et al., 1991; Wit et al., 2000), P_m appears to be very close to zero unless endolymphatic hydrops has been induced. With communication present through the cochlear aqueduct, both P_p and P_e will be close to P_{CSF} . Middle ear pressure (P_{me}) is assumed to be equal to atmospheric pressure (P_o). Communication with compartments outside the labyrinth can occur through four major routes (with their reference pressures shown in parentheses): via the stapes (P_{me}), the round window membrane (P_{me}), the cochlear aqueduct (P_{CSF}) and the endolymphatic duct (P_{CSF}). In Fig. 1b we demonstrate the consequences of a pressure increase in perilymph. Perilymph pressure increases were induced in our fluid-injection experiments and also occur during tensor tympani muscle contractions that occur during swallowing. Tensor tympani contractions drive the stapes into the labyrinth for a period of approximately 0.5 s (Salt and DeMott, 1999). The pressure increase in the labyrinth (P_s) causes the round window membrane to be visibly displaced towards the middle ear. As perilymph pressure ($P_p + P_s$) will now exceed P_{CSF} , perilymph will be driven through the cochlear aqueduct. Since the bounding membranes of endolymph are compliant, endolymph pressure ($P_e + P_s$) will similarly exceed P_{CSF} and endolymph will also be driven from the sinus through the endolymphatic duct. Thus, contrary to Wit and Hofman's argument, in this situation perilymph pressure change does induce an endolymph flow. A small loss of endolymph from the sinus, possibly combined with Bernoulli forces of the flow passing the sinus membrane near the duct opening, would cause the membrane to collapse towards the bone. When the membrane contacts the bony wall and obstructs further flow into the duct, this would account for the "valve-like" properties we observed experimentally. We further suggested (Salt, 2005) that the degree of distension of the endolymphatic sinus could play an important part in detecting endolymph volume changes, as more endolymph would be available to enter the duct if the endolymphatic sinus had previously been distended by an endolymph volume increase. To demonstrate the validity of this assertion, we have constructed a simple physical model in which the principle is demonstrated. A brief movie showing operation of this model can be viewed at <http://oto.wustl.edu/cochlea/sinus>.

In trying to reconcile the calculations of Hoffman and Wit, we can accept the mathematical argument given by Wit and Hofman (their Fig. 3) that flow only occurs when $P_5 > P_4$ (when the membrane pressure of the saccule is greater than that of the sinus), and that an increase in perilymph pressure (P_{peri}) has "no influence [on endolymph flow]", but only for the situation where there is no outlet for endolymph from their chamber. In the presence of the outlet shown (at the right side of their Fig. 3) and with P_2 remaining constant, then an increase in P_{peri} must increase endolymph pressure in the sinus (no symbol given), causing a pressure gradient between the sinus endolymph and P_2 , thereby inducing flow out of the chamber. This is exactly what our original interpretation proposed.

Finally, we agree there was an error in our paper that Wit and Hofman pointed out. Calibration bars in Figs. 7 and 8 were stated to be 25 l when the correct figure should have been 250 l. We apologize for this scaling error.

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Fig. 1.

(a) Schematic of the inner ear with the compliant membranous boundaries of endolymph (thin lines) and the bony otic capsule (heavy lines). Abbreviations for structures are ST: scala tympani, SV: scala vestibuli, RWM: round window membrane. Pressure symbols are explained in the text. (b) Pressure changes and volume flows (heavy arrows) resulting from contraction of the tensor tympani muscle. Due to the pressure gradients, perilymph will be driven out through the cochlear aqueduct and endolymph will be driven out through the endolymphatic duct.