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Adenylate kinase 7 is a differentiation marker of kinocilia-bearing cells

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Ependymal cells form a mostly single-layered epithelium which covers the surfaces of the cerebral ventricles. Considerable evidence has accumulated for the ependyma to be involved in the pathogenesis of hydrocephalus, a dilation of the ventricles at the expense of brain parenchyma. The underlying mechanism may include gene defects in ependymal cells, which prevent their proper differentiation and render them unable to fulfil their physiological functions. The most prominent marker of ependymal differentiation is the presence of kinocilia at the apical cell surface. In order to identify molecular players involved in the differentiation process, a subtractive cDNA library of ependyma minus brain was screened for ependyma-specific transcripts. One of the resulting candidates is the message for putative adenylate kinase 7 (pAK7), which is exclusively produced in rat tissues known for the presence of kinocilia, namely testis, lung and ependyma, as determined by real-time RT-PCR.

After generation of peptide antibodies against the C-terminus of the pAK7 protein, this expression pattern was further verified by Western blot and immunochemical stainings. The molecular mass of rat pAK7, 82 kDa as deduced from its primary structure, is significantly higher than the molecular masses of the other identified rat andenylate kinases, which remain below 30 kDa. Since a fusion protein of pAK7 and green fluorescent protein exhibited no measurable adenylate kinase activity when expressed in HEK 293T cells, the 82 kDa pAK7 protein probably is not really an adenylate kinase, but must have other functions. The time course of pAK7 mRNA and protein levels during development of rat testis and ependymal primary cultures parallels the expression of kinocilia. The protein may therefore be considered as an ependymal differentiation marker involved in ciliogenesis. A yeast two-hybrid screen was employed to gain leads on possible functions of pAK7 not related to adenylate kinase activity. Putative, not yet verified interaction partners include a protein involved in Bardet-Biedl syndrome (BBS), a pleiotropic disease complex with symptoms comprising hydrocephalus. Specifically, the particular BBS protein under investigation has been implicated in the generation or maintenance of cilia, organelles with a proven role in hydrocephalic disease.