1. Phylogeny  
   MYO3A (Myosin‐IIIa) is a member of the class III unconventional myosin family and is evolutionarily conserved across vertebrate species. In invertebrates, a homolog of MYO3A is represented by Drosophila NINAC, and in vertebrates two paralogs exist – MYO3A and MYO3B – which diverged to assume overlapping yet distinct roles in sensory tissues (dose2003myo3aoneof pages 4-5, coluccio2008myosins pages 291-295, houdusse2021themanyroles pages 2-3). Within the kinome and the broader superfamily of myosins, MYO3A occupies a niche characterized by its combined actin‐motor and intrinsic kinase activities. Phylogenetic analyses based on sequence comparisons have confirmed its assignment as a class III myosin that arose before the radiation of vertebrates, with its evolutionary roots traceable to an ancestral gene that predated the diversification of nonmuscle motor proteins (coluccio2008myosins pages 297-300, nishio2015geneexpressionprofiles pages 30-32).
2. Reaction Catalyzed  
   The intrinsic serine/threonine kinase domain of MYO3A catalyzes the transfer of the γ‐phosphate from ATP to the hydroxyl group of specific serine or threonine residues. In this reaction, ATP and a substrate protein (which may be MYO3A itself via autophosphorylation or another effector protein) are converted into ADP, a phosphorylated protein, and a proton (an2014phosphorylationofthe pages 9-10, quintero2010intermolecularautophosphorylationregulates pages 5-7).
3. Cofactor Requirements  
   The kinase domain of MYO3A requires divalent magnesium ions (Mg²⁺) as an essential cofactor for efficient ATP binding and catalysis during phosphoryl transfer. In addition, the motor domain’s ATPase activity similarly depends on Mg²⁺, while the regulatory properties of its IQ motifs are modulated by Ca²⁺ via calmodulin binding (mcmenamin2018calciumregulationof pages 7-12, coluccio2008myosins pages 307-310).
4. Substrate Specificity  
   MYO3A phosphorylates serine and threonine residues predominantly through autophosphorylation of conserved sites within its kinase domain, notably at residues such as Thr184 and Thr188. Although the precise consensus substrate motif is not fully defined in the literature, MYO3A’s activity is directed toward serine/threonine contexts on itself and potentially on associated actin‐regulatory proteins, with its substrate specificity aligning broadly with that of many serine/threonine kinases (an2014phosphorylationofthe pages 1-2, grati2016myo3acauseshuman pages 1-2, cirilo2021functionalroleof pages 1-2).
5. Structure  
   MYO3A is organized into several structurally and functionally distinct domains. It possesses an N-terminal kinase domain that exhibits a conserved catalytic core with an activation loop harboring phosphorylation sites critical for autophosphorylation and regulation (an2014phosphorylationofthe pages 1-2, coluccio2008myosins pages 300-302). Attached to the kinase domain is a motor (head) domain that contains the classical actin-binding sites, an ATPase catalytic pocket with subdomains (including regions analogous to the upper and lower 50 kDa domains), and a converter element that facilitates conformational shifts during the ATP hydrolysis cycle (miyoshi2024pathophysiologyofhuman pages 4-5, raval2016mechanismofclass pages 91-99).  
   Following the motor domain is a neck region that features two IQ motifs—which serve as binding sites for calmodulin and calmodulin-like light chains—acting as a lever arm to amplify motor-generated force (cirilo2021functionalroleof pages 7-8, mcmenamin2018calciumregulationof pages 7-12). The C-terminal tail region of MYO3A is characterized by two conserved subdomains: tail homology domain I (THDI) and tail homology domain II (THDII). THDII, in particular, contains an actin-binding motif and is key to mediating interactions with actin bundles and with regulatory binding partners such as espin and MORN4 (cirilo2024thedynamicsof pages 9-10, coluccio2008myosins pages 310-312, liu2016myosiniiimediatedcrosslinking pages 14-15). Predicted three-dimensional models (e.g. from AlphaFold) show a well-folded kinase domain connected in series to a canonical myosin motor domain followed by an elongated neck and an unstructured yet functionally modular tail (coluccio2008myosins pages 307-310).
6. Regulation  
   MYO3A is regulated principally through post-translational modifications and calcium-dependent binding interactions. The kinase domain undergoes autophosphorylation on specific threonine residues—which includes sites such as Thr184 and Thr188—thereby reducing the motor domain’s affinity for actin and modulating its enzymatic activity (an2014phosphorylationofthe pages 9-10, quintero2010intermolecularautophosphorylationregulates pages 5-7). Additionally, regulatory control is mediated by calcium levels via calmodulin binding to the IQ motifs present in the neck region. Variations in intracellular Ca²⁺ concentrations influence calmodulin’s binding affinity and, consequently, the structural conformation and lever arm function of the motor domain (mcmenamin2018calciumregulationof pages 7-12, cirilo2021functionalroleof pages 5-7). These regulatory modifications combine to fine-tune MYO3A’s intracellular localization and actin-based motility.
7. Function  
   MYO3A functions as an actin‐dependent motor protein with an intrinsic kinase activity that is essential for the proper development and maintenance of cochlear hair bundles. It is required during early stages of hair bundle morphogenesis, where it influences both the number and the lengths of stereocilia and limits the growth of microvilli within the developing auditory hair bundles—thereby contributing to the precise staircase architecture observed in these sensory structures (grati2016myo3acauseshuman pages 1-2, jamis2015asilentsymphony pages 63-68). In addition, MYO3A facilitates the elongation of actin filaments at stereocilia tips by transporting the actin regulatory factor espin to the plus ends of actin bundles, supporting the dynamic remodeling necessary for auditory mechanotransduction (cirilo2021functionalroleof pages 1-2, cirilo2021functionalroleof pages 7-8, miyoshi2024pathophysiologyofhuman pages 2-4). MYO3A is expressed predominantly in the sensory hair cells of the inner ear and shows additional expression in retinal photoreceptor cells, suggesting a role in visual function as well (OpenTargets Search: -MYO3A, gunther2022deafnessmutationin pages 10-11).
8. Other Comments  
   Mutations in MYO3A have been implicated in nonsyndromic hereditary hearing loss (DFNB30), with alterations in either the motor or kinase domains disrupting the protein’s capacity to regulate stereocilia development and maintenance (grati2016myo3acauseshuman pages 1-2, jamis2015asilentsymphony pages 25-32, miyoshi2024pathophysiologyofhuman pages 9-11). Despite its critical role in hair bundle morphogenesis, no specific small molecule inhibitors targeting MYO3A have been described in the peer-reviewed literature. The dual functionality of MYO3A, incorporating both motor and kinase activities, continues to generate interest as a potential molecular target for therapeutic intervention in sensorineural hearing loss, although current efforts remain focused on its genetic and functional characterization (OpenTargets Search: -MYO3A, cirilo2021functionalroleof pages 1-2).
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