1. Phylogeny  
   COQ8B (also known as ADCK4) is an evolutionarily ancient mitochondrial protein that belongs to the UbiB family, a subgroup of atypical kinases now recognized as part of the broader ABC1/ADCK/UbiB superfamily. Orthologs of COQ8B are found in a wide array of species ranging from bacteria and yeast to plants and vertebrates, underscoring its deep evolutionary conservation (abumaziad2021theroleof pages 6-7). In mammals, COQ8B is paralogous to COQ8A (ADCK3); these two proteins are thought to have arisen from a gene duplication event in vertebrates, which gave rise to specialized paralogs with tissue-specific functions (lagiertourenne2008adck3anancestral pages 6-8, lagiertourenne2008adck3anancestral pages 8-10). Phylogenetic analyses based on conserved catalytic motifs place COQ8B in a distinct clade within the human kinome that is separate from canonical eukaryotic protein kinases; instead, it clusters with other atypical kinases that share a common ancestral origin, consistent with evolutionary studies extending from yeast to human (lundquist2012abc1katypicalkinases pages 2-4, lundquist2012abc1katypicalkinases pages 7-8). This evolutionary grouping reflects the specialized roles these kinases have acquired in the regulation of coenzyme Q biosynthesis—a function critical to mitochondrial energy metabolism.
2. Reaction Catalyzed  
   COQ8B catalyzes the phosphoryl transfer reaction using ATP as a phosphate donor, yielding ADP and a phosphorylated product. Although the precise nature of its substrate remains unclear, its activity is generally described by the fundamental kinase reaction:  
     ATP + substrate → ADP + phosphorylated substrate + H⁺  
   Early reports have suggested that COQ8B may mediate the phosphorylation of protein components involved in coenzyme Q (ubiquinone) biosynthesis such as COQ3, while alternative reports propose that it could function as a small molecule kinase that targets prenyl lipid intermediates within the ubiquinone biosynthetic pathway (abumaziad2021theroleof pages 7-8). This duality in potential substrate specificity is reflective of its classification as an atypical kinase, in which the catalytic reaction may be adapted to both protein and small molecule substrates.
3. Cofactor Requirements  
   The catalytic activity of COQ8B is dependent on divalent metal ions, with magnesium (Mg²⁺) being the primary cofactor required for ATP binding and phosphoryl transfer. This Mg²⁺ dependency is a characteristic feature shared by both canonical and atypical serine/threonine kinases and is critical for the proper orientation of ATP within the active site to facilitate efficient catalysis (abumaziad2021theroleof pages 6-7).
4. Substrate Specificity  
   The substrate specificity of COQ8B remains only partially defined. On one hand, COQ8B has been reported to function as a protein kinase that may phosphorylate COQ3, suggesting a role in modulating the activity of downstream enzymes in the coenzyme Q biosynthetic pathway (abumaziad2021theroleof pages 7-8). On the other hand, there are reports proposing that COQ8B acts as a small molecule kinase with potential lipid kinase activity, capable of phosphorylating prenyl lipid intermediates within the ubiquinone biosynthesis cascade (abumaziad2021theroleof pages 7-8). Analogous studies on its closely related paralog COQ8A have revealed substrate preferences for peptide motifs characterized by basic residues at approximately the −3 position and hydrophobic residues at the +1 site relative to the phosphorylation target (awad2020intragenicsuppressormutations pages 2-4), although these findings have yet to be definitively extended to COQ8B. Thus, while the precise consensus motif for COQ8B remains to be fully established, current evidence supports a model in which its substrate specificity may involve recognition of either protein segments, such as those present in COQ biosynthetic proteins, or lipid molecules that are intermediates in ubiquinone synthesis.
5. Structure  
   Structurally, COQ8B is predicted to contain a central kinase-like domain that is atypical in its organization compared to classical eukaryotic protein kinases. Homology modeling based on the known structure of its paralog COQ8A (using PDB ID: 4PED) via I-TASSER has yielded a model with a C-score of 0.09 and TM-score of 0.73, indicating a reliable fold that supports a functional kinase-like domain (abumaziad2021theroleof pages 6-7). The overall domain organization of COQ8B includes an N-terminal region that likely contains a mitochondrial targeting sequence (MTS), which directs the protein to mitochondria where it is subsequently integrated into the inner mitochondrial membrane. This is further supported by its classification as a single transmembrane-spanning kinase-like protein, with a predicted transmembrane helix anchoring it to the mitochondrial membrane (abumaziad2021theroleof pages 6-7). Within its kinase domain, COQ8B exhibits several unique structural features: an alanine‐rich loop that replaces the classical glycine‐rich loop found in typical serine/threonine kinases and a distinctive N-terminal extension that includes a conserved KxGQ motif. This KxGQ motif is thought to serve as an autoinhibitory element that occludes the substrate-binding cleft under basal conditions, potentially requiring displacement or structural reorganization for activation (lagiertourenne2008adck3anancestral pages 8-10, lundquist2012abc1katypicalkinases pages 5-7). The overall tertiary structure, as derived from computational models, suggests a bilobal architecture with an N-terminal lobe primarily involved in ATP binding and a larger C-terminal lobe that may facilitate substrate recognition and binding. In addition, although direct crystal structures of COQ8B are not yet available, the modeled structure supports conserved core kinase motifs such as motifs responsible for ATP binding and catalysis, even if several canonical motifs are substituted with atypical features.
6. Regulation  
   The regulation of COQ8B’s catalytic activity appears to involve multiple layers of control. At the structural level, the presence of the N-terminal autoinhibitory KxGQ motif suggests that COQ8B is maintained in an inactive or low-activity state under basal conditions (abumaziad2021theroleof pages 6-7). This motif is thought to block substrate access to the active site, thereby requiring a regulatory event—possibly a conformational change triggered by post-translational modifications such as phosphorylation or by protein–protein interactions—to relieve this autoinhibition and activate the kinase. Experimental mutation studies in related ADCK family members indicate that alterations in conserved residues within this regulatory motif can significantly impact the kinase’s catalytic activity and its ability to mediate coenzyme Q biosynthesis (awad2020intragenicsuppressormutations pages 21-22). Furthermore, although the specific upstream kinases or phosphatases that might modulate COQ8B through direct phosphorylation have not been definitively identified, changes in the phosphorylation status of either COQ8B itself or associated components of the coenzyme Q biosynthetic complex may serve as a critical regulatory mechanism. In addition to direct post-translational modifications, the spatial organization of COQ8B within mitochondrial membranes—such as its proximity to other CoQ biosynthetic enzymes at specialized membrane contact sites—may further contribute to its regulation by facilitating substrate channeling or by enabling allosteric interactions with partner proteins (lagiertourenne2008adck3anancestral pages 8-10, awad2020intragenicsuppressormutations pages 21-22). Overall, the regulatory properties of COQ8B are consistent with its role as an atypical kinase whose activity must be tightly controlled to ensure proper mitochondrial function and coenzyme Q biosynthesis.
7. Function  
   COQ8B is fundamentally involved in the biosynthesis of coenzyme Q (ubiquinone), a lipid-soluble electron carrier that plays an essential role in the mitochondrial electron transport chain and aerobic cellular respiration. As an atypical kinase, COQ8B is believed to execute regulatory phosphorylation events that modulate the activity and stability of enzymes within the coenzyme Q biosynthetic complex. For instance, there is evidence suggesting that COQ8B may phosphorylate COQ3—a key enzyme in the ubiquinone biosynthesis pathway (abumaziad2021theroleof pages 7-8). This phosphorylation event is critical for maintaining efficient coenzyme Q production, which is necessary for sustaining optimal electron flux through the respiratory chain. COQ8B’s function is further underscored by its tissue-specific expression pattern; it is prominently expressed in podocytes, specialized cells in the kidney glomerulus. The unique expression profile in podocytes is clinically significant, as mutations in COQ8B are causally linked to steroid-resistant nephrotic syndrome and glomerulopathies such as focal segmental glomerulosclerosis (abumaziad2021theroleof pages 6-7, zhai2020earlyonsetcoq8b(adck4) pages 8-9). These renal manifestations are thought to result from impaired coenzyme Q biosynthesis, leading to mitochondrial dysfunction and compromised podocyte migration—a process critical for maintaining the integrity of the glomerular filtration barrier. In addition to its role in kidney function, by influencing the formation and/or stability of the coenzyme Q biosynthetic complex, COQ8B indirectly supports mitochondrial ATP production and overall cellular energy metabolism. The functional importance of COQ8B is thus evident at both the biochemical level, where its kinase activity contributes to the regulation of ubiquinone biosynthesis, and at the physiological level, where its deficiency manifests as a significant mitochondrial defect with clinical consequences in the kidney (lagiertourenne2008adck3anancestral pages 6-8, lundquist2012abc1katypicalkinases pages 7-8, zhai2020earlyonsetcoq8b(adck4) pages 8-9).
8. Other Comments  
   At present, there are no well-established small molecule inhibitors that selectively target COQ8B; however, clinical management of diseases associated with COQ8B mutations has included high-dose coenzyme Q10 supplementation, which in some cases ameliorates renal dysfunction and improves mitochondrial bioenergetics (zhai2020earlyonsetcoq8b(adck4) pages 8-9). Notable disease associations for COQ8B include steroid-resistant nephrotic syndrome and focal segmental glomerulosclerosis, conditions that are directly linked to impaired coenzyme Q biosynthesis and resultant mitochondrial dysfunction in podocytes (abumaziad2021theroleof pages 6-7, abumaziad2021theroleof pages 8-8). Furthermore, mutations in COQ8B that disrupt conserved regulatory motifs or compromise its kinase activity have been shown to lead to renal-specific phenotypes without causing the neurological manifestations seen in COQ8A (ADCK3) mutations. Such genotype–phenotype correlations underscore the tissue‐specific functional specialization that has arisen following gene duplication in vertebrates (lagiertourenne2008adck3anancestral pages 6-8, lundquist2012abc1katypicalkinases pages 7-8). In addition, as part of the atypical kinase family, COQ8B shares some mechanistic and structural features with other members of the ABC1/ADCK/UbiB family, including the utilization of autoinhibitory regulatory domains and divergent ATP-binding loops. These unique characteristics suggest that while conventional kinase inhibitors may not be applicable, future therapeutic strategies might focus on modulating the autoinhibitory interactions or stabilizing the active conformation of COQ8B, thereby restoring its proper function in coenzyme Q biosynthesis. Finally, although some reports have debated whether COQ8B functions primarily as a protein kinase or a small molecule (lipid) kinase, the consensus from multiple studies indicates that its activity is indispensable for maintaining mitochondrial respiratory function, particularly in the renal cell context (abumaziad2021theroleof pages 7-8, awad2020intragenicsuppressormutations pages 22-23).
9. References
10. AbuMaziad, A. S., Thaker, T. M., Tomasiak, T. M., Chong, C. C., Galindo, M. K., & Hoyme, H. E. “The role of novel coq8b mutations in glomerulopathy and related kidney defects.” American Journal of Medical Genetics Part A, 185:60-67, Oct 2021. (abumaziad2021theroleof pages 6-7, abumaziad2021theroleof pages 7-8, abumaziad2021theroleof pages 8-8)
11. Lagier-Tourenne, C., Tazir, M., Lopez, L., Quinzii, C., Assoum, M., Drouot, N., Busso, C., Makri, S., Alipacha, L., Benhassine, T., Anheim, M., Lynch, D., Thibault, C., Plewniak, F., Bianchetti, L., Tranchant, C., Poch, O., Dimauro, S., Mandel, J., Barros, M. H., Hirano, M., & Koenig, M. “Adck3, an ancestral kinase, is mutated in a form of recessive ataxia associated with coenzyme q10 deficiency.” American Journal of Human Genetics, 82(3):661-72, Mar 2008. (lagiertourenne2008adck3anancestral pages 6-8, lagiertourenne2008adck3anancestral pages 8-10)
12. Lundquist, P., Davis, J. I., & van Wijk, K. V. “Abc1k atypical kinases in plants: filling the organellar kinase void.” Trends in Plant Science, 17(9):546-55, Sep 2012. (lundquist2012abc1katypicalkinases pages 2-4, lundquist2012abc1katypicalkinases pages 4-5, lundquist2012abc1katypicalkinases pages 5-7, lundquist2012abc1katypicalkinases pages 7-8)
13. Vazquez-Fonseca, L., Schäfer, J., Navas-Enamorado, I., Santos-Ocaña, C., Hernández-Camacho, J. D., Guerra, I., Cascajo, M. V., Sánchez-Cuesta, A., Horvath, Z., Siendones, E., Jou, C., Casado, M., Gutiérrez-Ríos, P., Brea-Calvo, G., López-Lluch, G., Fernández-Ayala, D., Cortés-Rodríguez, A., Rodríguez-Aguilera, J., Matté, C., Ribes, A., Prieto-Soler, S. Y., Domínguez‐del‐Toro, E., Di Francesco, A., Aon, M., Bernier, M., Salviati, L., Artuch, R., de Cabo, R., & Navas, P. “Adck2 haploinsufficiency reduces mitochondrial lipid oxidation and causes myopathy associated with coq deficiency.” Journal of Clinical Medicine, Aug 2019. (vazquezfonseca2019adck2haploinsufficiencyreduces pages 18-20, vazquezfonseca2019adck2haploinsufficiencyreduces pages 8-10)
14. Awad, A. M., Nag, A., Pham, N. V. B., Bradley, M., Jabassini, N., Nathaniel, J., & Clarke, C. “Intragenic suppressor mutations of the coq8 protein kinase homolog restore coenzyme q biosynthesis and function in saccharomyces cerevisiae.” PLoS ONE, Jun 2020. (awad2020intragenicsuppressormutations pages 1-2, awad2020intragenicsuppressormutations pages 21-22, awad2020intragenicsuppressormutations pages 22-23, awad2020intragenicsuppressormutations pages 15-17)
15. Cullen, J. K., Abdul Murad, N., Yeo, A., McKenzie, M., Ward, M., Chong, K. L., Schieber, N., Parton, R., Lim, Y., Wolvetang, E., Maghzal, G., Stocker, R., & Lavin, M. “Aarf domain containing kinase 3 (adck3) mutant cells display signs of oxidative stress, defects in mitochondrial homeostasis and lysosomal accumulation.” PLoS ONE, Feb 2016. (cullen2016aarfdomaincontaining pages 2-4, cullen2016aarfdomaincontaining pages 24-25, cullen2016aarfdomaincontaining pages 25-26, cullen2016aarfdomaincontaining pages 10-12, cullen2016aarfdomaincontaining pages 25-26)
16. Zhai, S., Zhang, L., Sun, B.-C., Zhang, Y., & Ma, Q.-S. “Early-onset coq8b (adck4) glomerulopathy in a child with isolated proteinuria: a case report and literature review.” BMC Nephrology, Sep 2020. (zhai2020earlyonsetcoq8b(adck4) pages 8-9)

References

1. (abumaziad2021theroleof pages 6-7): Asmaa S. AbuMaziad, Tarjani M. Thaker, Thomas M. Tomasiak, Chyi Chyi Chong, Maureen K. Galindo, and H. Eugene Hoyme. The role of novel coq8b mutations in glomerulopathy and related kidney defects. American Journal of Medical Genetics Part A, 185:60-67, Oct 2021. URL: https://doi.org/10.1002/ajmg.a.61909, doi:10.1002/ajmg.a.61909. This article has 6 citations.
2. (lagiertourenne2008adck3anancestral pages 6-8): C. Lagier-Tourenne, M. Tazir, L. Lopez, C. Quinzii, M. Assoum, N. Drouot, C. Busso, S. Makri, L. Alipacha, T. Benhassine, M. Anheim, D. Lynch, C. Thibault, F. Plewniak, Laurent Bianchetti, C. Tranchant, O. Poch, S. Dimauro, J. Mandel, M. H. Barros, M. Hirano, and M. Koenig. Adck3, an ancestral kinase, is mutated in a form of recessive ataxia associated with coenzyme q10 deficiency. American journal of human genetics, 82 3:661-72, Mar 2008. URL: https://doi.org/10.1016/j.ajhg.2007.12.024, doi:10.1016/j.ajhg.2007.12.024. This article has 361 citations and is from a highest quality peer-reviewed journal.
3. (lagiertourenne2008adck3anancestral pages 8-10): C. Lagier-Tourenne, M. Tazir, L. Lopez, C. Quinzii, M. Assoum, N. Drouot, C. Busso, S. Makri, L. Alipacha, T. Benhassine, M. Anheim, D. Lynch, C. Thibault, F. Plewniak, Laurent Bianchetti, C. Tranchant, O. Poch, S. Dimauro, J. Mandel, M. H. Barros, M. Hirano, and M. Koenig. Adck3, an ancestral kinase, is mutated in a form of recessive ataxia associated with coenzyme q10 deficiency. American journal of human genetics, 82 3:661-72, Mar 2008. URL: https://doi.org/10.1016/j.ajhg.2007.12.024, doi:10.1016/j.ajhg.2007.12.024. This article has 361 citations and is from a highest quality peer-reviewed journal.
4. (lundquist2012abc1katypicalkinases pages 2-4): P. Lundquist, Jerrold I. Davis, and K. V. van Wijk. Abc1k atypical kinases in plants: filling the organellar kinase void. Trends in plant science, 17 9:546-55, Sep 2012. URL: https://doi.org/10.1016/j.tplants.2012.05.010, doi:10.1016/j.tplants.2012.05.010. This article has 76 citations and is from a domain leading peer-reviewed journal.
5. (lundquist2012abc1katypicalkinases pages 4-5): P. Lundquist, Jerrold I. Davis, and K. V. van Wijk. Abc1k atypical kinases in plants: filling the organellar kinase void. Trends in plant science, 17 9:546-55, Sep 2012. URL: https://doi.org/10.1016/j.tplants.2012.05.010, doi:10.1016/j.tplants.2012.05.010. This article has 76 citations and is from a domain leading peer-reviewed journal.
6. (lundquist2012abc1katypicalkinases pages 5-7): P. Lundquist, Jerrold I. Davis, and K. V. van Wijk. Abc1k atypical kinases in plants: filling the organellar kinase void. Trends in plant science, 17 9:546-55, Sep 2012. URL: https://doi.org/10.1016/j.tplants.2012.05.010, doi:10.1016/j.tplants.2012.05.010. This article has 76 citations and is from a domain leading peer-reviewed journal.
7. (vazquezfonseca2019adck2haploinsufficiencyreduces pages 18-20): L. Vázquez-Fonseca, J. Schäfer, I. Navas-Enamorado, C. Santos-Ocaña, J. D. Hernández-Camacho, I. Guerra, María V Cascajo, Ana Sánchez-Cuesta, Z. Horvath, E. Siendones, C. Jou, M. Casado, Purificación Gutiérrez-Ríos, G. Brea-Calvo, G. López-Lluch, D. Fernández-Ayala, A. Cortés-Rodríguez, J. Rodríguez-Aguilera, C. Matté, A. Ribes, Sandra Y. Prieto-Soler, E. Domínguez‐del‐Toro, A. Di Francesco, M. Aon, M. Bernier, L. Salviati, R. Artuch, R. de Cabo, S. Jackson, and P. Navas. Adck2 haploinsufficiency reduces mitochondrial lipid oxidation and causes myopathy associated with coq deficiency. Journal of Clinical Medicine, Aug 2019. URL: https://doi.org/10.3390/jcm8091374, doi:10.3390/jcm8091374. This article has 41 citations and is from a peer-reviewed journal.
8. (abumaziad2021theroleof pages 7-8): Asmaa S. AbuMaziad, Tarjani M. Thaker, Thomas M. Tomasiak, Chyi Chyi Chong, Maureen K. Galindo, and H. Eugene Hoyme. The role of novel coq8b mutations in glomerulopathy and related kidney defects. American Journal of Medical Genetics Part A, 185:60-67, Oct 2021. URL: https://doi.org/10.1002/ajmg.a.61909, doi:10.1002/ajmg.a.61909. This article has 6 citations.
9. (abumaziad2021theroleof pages 8-8): Asmaa S. AbuMaziad, Tarjani M. Thaker, Thomas M. Tomasiak, Chyi Chyi Chong, Maureen K. Galindo, and H. Eugene Hoyme. The role of novel coq8b mutations in glomerulopathy and related kidney defects. American Journal of Medical Genetics Part A, 185:60-67, Oct 2021. URL: https://doi.org/10.1002/ajmg.a.61909, doi:10.1002/ajmg.a.61909. This article has 6 citations.
10. (awad2020intragenicsuppressormutations pages 1-2): Agape M. Awad, Anish Nag, Nguyen V. B. Pham, M. Bradley, N. Jabassini, Juan Nathaniel, and C. Clarke. Intragenic suppressor mutations of the coq8 protein kinase homolog restore coenzyme q biosynthesis and function in saccharomyces cerevisiae. PLoS ONE, Jun 2020. URL: https://doi.org/10.1371/journal.pone.0234192, doi:10.1371/journal.pone.0234192. This article has 5 citations and is from a peer-reviewed journal.
11. (awad2020intragenicsuppressormutations pages 2-4): Agape M. Awad, Anish Nag, Nguyen V. B. Pham, M. Bradley, N. Jabassini, Juan Nathaniel, and C. Clarke. Intragenic suppressor mutations of the coq8 protein kinase homolog restore coenzyme q biosynthesis and function in saccharomyces cerevisiae. PLoS ONE, Jun 2020. URL: https://doi.org/10.1371/journal.pone.0234192, doi:10.1371/journal.pone.0234192. This article has 5 citations and is from a peer-reviewed journal.
12. (awad2020intragenicsuppressormutations pages 21-22): Agape M. Awad, Anish Nag, Nguyen V. B. Pham, M. Bradley, N. Jabassini, Juan Nathaniel, and C. Clarke. Intragenic suppressor mutations of the coq8 protein kinase homolog restore coenzyme q biosynthesis and function in saccharomyces cerevisiae. PLoS ONE, Jun 2020. URL: https://doi.org/10.1371/journal.pone.0234192, doi:10.1371/journal.pone.0234192. This article has 5 citations and is from a peer-reviewed journal.
13. (awad2020intragenicsuppressormutations pages 22-23): Agape M. Awad, Anish Nag, Nguyen V. B. Pham, M. Bradley, N. Jabassini, Juan Nathaniel, and C. Clarke. Intragenic suppressor mutations of the coq8 protein kinase homolog restore coenzyme q biosynthesis and function in saccharomyces cerevisiae. PLoS ONE, Jun 2020. URL: https://doi.org/10.1371/journal.pone.0234192, doi:10.1371/journal.pone.0234192. This article has 5 citations and is from a peer-reviewed journal.
14. (cullen2016aarfdomaincontaining pages 2-4): Jason K. Cullen, Norazian Abdul Murad, A. Yeo, M. McKenzie, M. Ward, K. L. Chong, N. Schieber, R. Parton, Y. Lim, E. Wolvetang, G. Maghzal, R. Stocker, and M. Lavin. Aarf domain containing kinase 3 (adck3) mutant cells display signs of oxidative stress, defects in mitochondrial homeostasis and lysosomal accumulation. PLoS ONE, Feb 2016. URL: https://doi.org/10.1371/journal.pone.0148213, doi:10.1371/journal.pone.0148213. This article has 63 citations and is from a peer-reviewed journal.
15. (cullen2016aarfdomaincontaining pages 24-25): Jason K. Cullen, Norazian Abdul Murad, A. Yeo, M. McKenzie, M. Ward, K. L. Chong, N. Schieber, R. Parton, Y. Lim, E. Wolvetang, G. Maghzal, R. Stocker, and M. Lavin. Aarf domain containing kinase 3 (adck3) mutant cells display signs of oxidative stress, defects in mitochondrial homeostasis and lysosomal accumulation. PLoS ONE, Feb 2016. URL: https://doi.org/10.1371/journal.pone.0148213, doi:10.1371/journal.pone.0148213. This article has 63 citations and is from a peer-reviewed journal.
16. (cullen2016aarfdomaincontaining pages 25-26): Jason K. Cullen, Norazian Abdul Murad, A. Yeo, M. McKenzie, M. Ward, K. L. Chong, N. Schieber, R. Parton, Y. Lim, E. Wolvetang, G. Maghzal, R. Stocker, and M. Lavin. Aarf domain containing kinase 3 (adck3) mutant cells display signs of oxidative stress, defects in mitochondrial homeostasis and lysosomal accumulation. PLoS ONE, Feb 2016. URL: https://doi.org/10.1371/journal.pone.0148213, doi:10.1371/journal.pone.0148213. This article has 63 citations and is from a peer-reviewed journal.
17. (lundquist2012abc1katypicalkinases pages 7-8): P. Lundquist, Jerrold I. Davis, and K. V. van Wijk. Abc1k atypical kinases in plants: filling the organellar kinase void. Trends in plant science, 17 9:546-55, Sep 2012. URL: https://doi.org/10.1016/j.tplants.2012.05.010, doi:10.1016/j.tplants.2012.05.010. This article has 76 citations and is from a domain leading peer-reviewed journal.
18. (zhai2020earlyonsetcoq8b(adck4) pages 8-9): Shusen Zhai, Li Zhang, Bai-chao Sun, Yan Zhang, and Qing-shan Ma. Early-onset coq8b (adck4) glomerulopathy in a child with isolated proteinuria: a case report and literature review. BMC Nephrology, Sep 2020. URL: https://doi.org/10.1186/s12882-020-02038-7, doi:10.1186/s12882-020-02038-7. This article has 16 citations and is from a peer-reviewed journal.
19. (awad2020intragenicsuppressormutations pages 15-17): Agape M. Awad, Anish Nag, Nguyen V. B. Pham, M. Bradley, N. Jabassini, Juan Nathaniel, and C. Clarke. Intragenic suppressor mutations of the coq8 protein kinase homolog restore coenzyme q biosynthesis and function in saccharomyces cerevisiae. PLoS ONE, Jun 2020. URL: https://doi.org/10.1371/journal.pone.0234192, doi:10.1371/journal.pone.0234192. This article has 5 citations and is from a peer-reviewed journal.
20. (cullen2016aarfdomaincontaining pages 10-12): Jason K. Cullen, Norazian Abdul Murad, A. Yeo, M. McKenzie, M. Ward, K. L. Chong, N. Schieber, R. Parton, Y. Lim, E. Wolvetang, G. Maghzal, R. Stocker, and M. Lavin. Aarf domain containing kinase 3 (adck3) mutant cells display signs of oxidative stress, defects in mitochondrial homeostasis and lysosomal accumulation. PLoS ONE, Feb 2016. URL: https://doi.org/10.1371/journal.pone.0148213, doi:10.1371/journal.pone.0148213. This article has 63 citations and is from a peer-reviewed journal.
21. (vazquezfonseca2019adck2haploinsufficiencyreduces pages 8-10): L. Vázquez-Fonseca, J. Schäfer, I. Navas-Enamorado, C. Santos-Ocaña, J. D. Hernández-Camacho, I. Guerra, María V Cascajo, Ana Sánchez-Cuesta, Z. Horvath, E. Siendones, C. Jou, M. Casado, Purificación Gutiérrez-Ríos, G. Brea-Calvo, G. López-Lluch, D. Fernández-Ayala, A. Cortés-Rodríguez, J. Rodríguez-Aguilera, C. Matté, A. Ribes, Sandra Y. Prieto-Soler, E. Domínguez‐del‐Toro, A. Di Francesco, M. Aon, M. Bernier, L. Salviati, R. Artuch, R. de Cabo, S. Jackson, and P. Navas. Adck2 haploinsufficiency reduces mitochondrial lipid oxidation and causes myopathy associated with coq deficiency. Journal of Clinical Medicine, Aug 2019. URL: https://doi.org/10.3390/jcm8091374, doi:10.3390/jcm8091374. This article has 41 citations and is from a peer-reviewed journal.