Nephrogenic diabetes insipidus(NDI) is a disorder caused by the inability of cells in the kidney to respond to antidiuretic hormone(ADH) (Seibold et al. 1993). Lack of response to ADH leads to excessive dilute urine production and chronic thirst. One gene implicated in causing NDI is aquaporin-2 (*AQP2*), which is used for concentrating urine in the collecting duct of the kidney prior to urine moving to the bladder (Deen et al. 1994, Vanlieburg et al. 1994). Under normal physiological conditions, ADH binding to the VPR receptor in the collecting duct leads to increased transport of vesicles contacting AQP2 to the cell surface and increased transcription of the *AQP2* gene (Seibold et al. 1993). Prior studies have found *AQP2* alleles of NDI patients with altered phosphorylation sites that prevent movement of AQP2 between compartments of the endomembrane system (Deen et al. 1995, Arnspang et al. 2016). However, it is not currently understood what interactions needed to form vesicles are impacted by changes in phosphorylation state of AQP2. Identification of interactions that are trapping AQP2 within the endomembrane system may facilitate the development of therapeutics for patients with *AQP2* related NDI.

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