

Supporting Materials

Aquaporin Graphene Interface: Relevance to Point of Care Device for Renal Cell Carcinoma and Desalination

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Table S1. Critical residues of aquaporins (other than AQP1) affecting their permeability and selectivity.

Name	Mutation	Functional significance	Ref
AQP0 (human)	E134G, T138R	Loss of membrane water channel activity due to impaired trafficking. In <i>Xenopus laevis</i> oocyte swelling assay E134G and T138R mutants showed water permeabilities which were less than half of the wild-type level.	[1-3]
AQP0 (selection from PDB)	Y23F, Y149T	Y23F substitution causes a moderate increase of the positive potential at the periplasmic side, between the NPA and the ar/R constriction sites. Y149T substitution significantly decreases the negative potential in the periplasmic region of the channel, thus restoring an AQP1-like electrostatic profile.	[4]
AQP2 (human)	R187C	Mutation disrupts the normal folding of the narrow constriction in the aqueous pore leading to impaired trafficking to the plasma membrane. Inability to facilitate water transport.	[5-7]
AQP2 (human)	E258K	Altered intracellular transport of AQP2-E258K causes dominant nephrogenic diabetes insipidus. E258K mutant was retained in the Golgi apparatus.	[5]
AQP2 (human)	L22V, T126M, A147T, C181W, R187C	These mutations cause non-X-linked nephrogenic diabetes insipidus (NDI). In <i>Xenopus</i> oocytes expressing AQP2 cRNAs, single-channel water permeabilities of mutants L22V, T126M, and A147T were similar to that of wt AQP2, whereas C181W and R187C were nonfunctional.	[7]
AQP2 (human)	G64A, S216P	Mutants less stable than wt AQP2. Impaired trafficking to the plasma membrane. Inability to facilitate water transport in <i>Xenopus</i> oocytes.	[6]
AQP2 (human)	L22V, R254L	Mutation associated with nephrogenic diabetes insipidus. Partial loss of function – indicating that these AQP variants can be disease-associated even if they do not block the protein function completely.	[8]

AQP4 (human)	D184E	Mutation causes loss of the ionic interaction between the R182 and the mutated D184E. This mutation affects the mobility of the D loop, which acquires a higher propensity to equilibrate in a “closed conformation”. The increase in the conformational freedom forces loop D to obstruct the pore at the cytoplasmic side, hampering water crossing inside the channel.	[8]
AQP4 (human)	D69E, D69H	D69 is crucial for the spatial control of loop A, the particular molecular conformation of which enables the assembly of NMO-IgG epitopes. The interaction with NMO-IgG induces multiple sclerosis-like immunopathology called Neuromyelitis Optica (NMO) and several other disorders resulting from imbalanced water homeostasis.	[9,10]
AQP4 (human)	I128T, I205L, M224T	Reduced water permeability.	[8]
AQP4 (human)	M278T	Increased normalized water permeability.	[8,11]
AQP4 (human)	Δ61-64, S180D	Increased tendency of the mutant to aggregate and form dimers.	[8,9]
AQP5 (human)	N185D	AQP5 mutant targeting asparagine–proline–alanine (NPA) loop motif, was distributed throughout the whole cytoplasm, not to the apical membrane.	[12,13]
AQP5 (human)	S156E	Mutation S156E increased constitutive membrane expression of AQP5 and its surface abundance. S156E mutation does not cause any significant structural changes to the protein.	[13]
PfAQP (<i>Plasmodium falciparum</i>)	E125S, L192M	Mutation causes changes in protein electrostatics which leads to 10-fold reduction in water permeability.	[14]
PfAQP	E28A	Reverse the PfAQP swelling rates in favor of glycerol, changes in protein electrostatics.	[14]
PfAQP	L192V	High permeability of the L192V mutant for both water and glycerol. L192V mutant significantly decreased the permeability for xylitol and D-arabitol and raised 3-fold that of ribitol.	[14]
PfAQP	E28A, E125S	Changes in protein electrostatics.	[4,14]
α-TIP*	S7A, S23A, S99A, S131A	Mutation of residues participating in α-TIP activation by phosphorylation. Mutation reduced the apparent water transport activity of α-TIP in oocytes.	[15]
PM28A (spinach)	S6A, S36A, S115A, S188A, S192A, S274A	Mutations of potential phosphorylation sites at the cytoplasmic side of PM28A - lowered osmotic water permeability. Mutant S115A showed only 50% of the wild-type activity in water transport. Mutant S274A was sensitive to protein kinase inhibitor K252a. Double mutant S115A/S274A was not affected by K252a and okadaic acid.	[16]
AqpZ (E.coli)	T183C, L170C	Mutation T183C made the channel sensitive to inhibition by Hg ²⁺ . Mutant L170C was 20 times more sensitive to mercury than wt AqpZ and 4 times more sensitive than T183C	[17]

* Tonoplast Intrinsic Protein (seeds expressed in *Xenopus* oocytes)

Table S2. Performance of FO membrane for seawater desalination (draw solution is NaCl).

Feed solution	NaCl Draw solution	Water flux L/m ² h	Membrane orientation	Membrane type	Ref
DI water	0.5 M	18.6	FO	CTA HTI membrane	[18]
DI water	1.0 M	16.8	FO	CTA HTI membrane	[19]
50 mM NaCl	4 M	37.8	PRO	CTA HTI membrane	[20]
50 mM NaCl	4 M	27.0	FO	CTA HTI membrane	[20]
Tap water	0.5 M	10.3	FO	CTA HTI membrane	[21]
DI water	0.5 M	32.2	PRO	TFC membrane	[22]
DI water	1.5 M	18	FO	TFC membrane	[23]
DI water	2 M	47.5	PRO	TFC membrane	[23]
0.5 M NaCl	2 M	11.8	FO	TFC membrane	[23]
3.5 M NaCl	2 M	22	PRO	TFC membrane	[24]
DI water	2 M	33	PRO	TFC membrane	[25]
3.5% NaCl	2 M	15	PRO	TFC membrane	[25]
DI water	4 M	55	PRO	TFC membrane	[26]
DI water	1 M	31	PRO	TFC membrane	[27]
DI water	1 M	48	PRO	PVDF membrane	[27]
DI water	1.5 M	41	PRO	TFC membrane	[27]
DI water	1.5 M	62	PRO	PVDF membrane	[27]
DI water	1 M	22	FO	TFC membrane	[27]
DI water	1 M	28	FO	PVDF membrane	[27]
DI water	1.5 M	30	FO	TFC membrane	[27]
DI water	1.5 M	35	FO	PVDF membrane	[27]
DI water	117 g/L	24	PRO	CTA HTI membrane	[28]
50 g/L NaCl	155 g/L	14.6	PRO	CTA HTI membrane	[28]
100 g/L NaCl	194 g/L	8.5	PRO	CTA HTI membrane	[28]

DI - deionized water

FO - forward osmosis

PRO - pressure retarded osmosis

CTA HTI - cellulose tri-acetate (produced by Hydration Technology Innovations)

TFC - thin-film composite

PVDF - polyvinylidene difluoride

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