

Transient Receptor Potential channels (TRP) in GtoPdb v.2025.4

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Abstract

The TRP superfamily of channels (**nomenclature as agreed by NC-IUPHAR [177, 1080]**), whose founder member is the *Drosophila* Trp channel, exists in mammals as six families; TRPC, TRPM, TRPV, TRPA, TRPP and TRPML based on amino acid homologies. TRP subunits contain six putative TM domains and assemble as homo- or hetero-tetramers to form cation selective channels with diverse modes of activation and varied permeation properties (reviewed by [734]). Established, or potential, physiological functions of the individual members of the TRP families are discussed in detail in the recommended reviews and in a number of books [404, 690, 1163, 258]. The established, or potential, involvement of TRP channels in disease [1134] is reviewed in [452, 689], [692] and [468], together with a special edition of *Biochemica et Biophysica Acta* on the subject [689]. Additional disease related reviews, for pain [637], stroke [1143], sensation and inflammation [994], itch [130], and airway disease [313, 1058], are available. The pharmacology of most TRP channels has been advanced in recent years. Broad spectrum agents are listed in the tables along with more selective, or recently recognised, ligands that are flagged by the inclusion of a primary reference. See Rubaiy (2019) for a review of pharmacological tools for TRPC1/C4/C5 channels [810]. Most TRP channels are regulated by phosphoinositides such as $\text{PtdIns}(4,5)\text{P}_2$ although the effects reported are often complex, occasionally contradictory, and likely to be dependent upon experimental conditions, such as intracellular ATP levels (reviewed by [1015, 693, 806]). Such regulation is generally not included in the tables. When thermosensitivity is mentioned, it refers specifically to a high Q10 of gating, often in the range of 10-30, but does not necessarily imply that the channel's function is to act as a 'hot' or 'cold' sensor. In general, the search for TRP activators has led to many claims for temperature sensing, mechanosensation, and lipid sensing. All proteins are of course sensitive to energies of binding, mechanical force, and temperature, but the issue is whether the proposed input is within a physiologically relevant range resulting in a response.

TRPA (ankyrin) family

TRPA1 is the sole mammalian member of this group (reviewed by [295]). TRPA1 activation of sensory neurons contribute to nociception [417, 895, 606]. Pungent chemicals such as mustard oil (AITC), **allicin**, and **cinnamaldehyde** activate TRPA1 by modification of free thiol groups of cysteine side chains, especially those located in its amino terminus [579, 60, 368, 581]. Alkenals with α , β -unsaturated bonds, such as propenal (**acrolein**), butenal (**crotylaldehyde**), and **2-pentenal** can react with free thiols via Michael addition and can activate TRPA1. However, potency appears to weaken as carbon chain length increases [26, 60]. Covalent modification leads to sustained activation of TRPA1. Chemicals including **carvacrol**, menthol, and local anesthetics reversibly activate TRPA1 by non-covalent binding [428, 515, 1089, 1088]. TRPA1 is not mechanosensitive under physiological conditions, but can be activated by cold temperatures [429, 213]. The electron cryo-EM structure of TRPA1 [745] indicates that it is a 6-TM homotetramer. Each subunit of the channel contains two short 'pore helices' pointing into the ion selectivity filter, which is big enough to allow permeation of partially hydrated Ca^{2+} ions.

TRPC (canonical) family

Members of the TRPC subfamily (reviewed by [286, 783, 18, 4, 94, 450, 744, 70]) fall into the subgroups outlined below. TRPC2 is a pseudogene in humans. It is generally accepted that all TRPC channels are activated downstream of $\text{G}_{q/11}$ -coupled receptors, or receptor tyrosine kinases (reviewed by [770, 959, 1080]). A comprehensive listing of G protein coupled receptors that activate TRPC channels is given in [4]. Hetero-oligomeric complexes of TRPC channels and their association with proteins to form signalling complexes are detailed in [18] and [451]. TRPC channels have frequently been proposed to act as store-operated channels (SOCs) (or components of multimeric complexes that form SOC), activated by depletion of intracellular calcium stores (reviewed by [746, 18, 775, 825, 1129, 157, 730, 64, 158]). However, the weight of the evidence is that they are not directly gated by conventional store-operated mechanisms, as established for STIM-gated Orai channels. TRPC channels are not mechanically gated in physiologically relevant ranges of force. All members of the TRPC family are blocked by **2-APB** and **SKF96365** [350, 349]. Activation of TRPC channels by lipids is discussed by [70]. Important progress has been recently made in TRPC pharmacology [810, 623, 440, 102, 856, 192, 293]. TRPC channels regulate a variety of physiological functions and are implicated in many human diseases [298, 71, 890, 1038, 1032, 154, 103, 565, 918, 412].

TRPC1/C4/C5 subgroup

TRPC1 alone may not form a functional ion channel [230]. The structures of the apo and antagonist-bound states of TRPC1/TRPC4 heteromeric channels have been resolved by cryo-EM [1070]. TRPC4/C5 may be distinguished from other TRP channels by their potentiation by micromolar concentrations of La^{3+} . TRPC2 is a pseudogene in humans, but in other mammals appears to be an ion channel localized to microvilli of the vomeronasal organ. It is required for normal sexual behavior in response to pheromones in mice. It may also function in the main olfactory epithelia in mice [1122, 727, 728, 1123, 543, 1176, 1117].

TRPC3/C6/C7 subgroup

All members are activated by diacylglycerol independent of protein kinase C stimulation [350].

TRPM (melastatin) family

Members of the TRPM subfamily (reviewed by [277, 349, 746, 1159]) fall into the five subgroups outlined below.

TRPM1/M3 subgroup

In darkness, glutamate released by the photoreceptors and ON-bipolar cells binds to the metabotropic glutamate receptor 6, leading to activation of Go . This results in the closure of TRPM1. When the photoreceptors are stimulated by light, glutamate release is reduced, and TRPM1 channels are more active, resulting in cell membrane depolarization. Human TRPM1 mutations are associated with congenital stationary night blindness (CSNB), whose patients lack rod function. TRPM1 is also found melanocytes. Isoforms of TRPM1 may present in melanocytes, melanoma, brain, and retina. In melanoma cells, TRPM1 is prevalent in highly dynamic intracellular vesicular structures [401, 712]. TRPM3 (reviewed by [718]) exists as multiple splice variants which differ significantly in their biophysical properties. TRPM3 is expressed in somatosensory neurons and may be important in

development of heat hyperalgesia during inflammation (see review [947]). TRPM3 is frequently coexpressed with TRPA1 and TRPV1 in these neurons. TRPM3 is expressed in pancreatic beta cells as well as brain, pituitary gland, eye, kidney, and adipose tissue [717, 946]. TRPM3 may contribute to the detection of noxious heat [1024].

TRPM2

TRPM2 is activated under conditions of oxidative stress (respiratory burst of phagocytic cells). The direct activators are calcium, adenosine diphosphate ribose (ADPR) [976] and cyclic ADPR (cADPR) [1126]. As for many ion channels, PI(4,5)P2 must also be present [1117]. Numerous splice variants of *TRPM2* exist which differ in their activation mechanisms [240]. Recent studies have reported structures of human (hs) TRPM2, which demonstrate two ADPR binding sites in hsTRPM2, one in the N-terminal MHR1/2 domain and the other in the C-terminal NUDT9-H domain. In addition, one Ca²⁺ binding site in the intracellular S2-S3 loop is revealed and proposed to mediate Ca²⁺ binding that induces conformational changes leading the ADPR-bound closed channel to open [390, 1034]. Meanwhile, a quadruple-residue motif (979FGQI982) was identified as the ion selectivity filter and a gate to control ion permeation in hsTRPM2 [1128]. TRPM2 is involved in warmth sensation [853], and contributes to several diseases [76]. TRPM2 interacts with extra synaptic NMDA receptors (NMDAR) and enhances NMDAR activity in ischemic stroke [1172]. Activation of TRPM2 in macrophages promotes atherosclerosis [1173, 1155]. Moreover, silica nanoparticles induce lung inflammation in mice via ROS/PARP/TRPM2 signaling-mediated lysosome impairment and autophagy dysfunction [1035]. Recent studies have designed various compounds for their potential to selectively inhibit the TRPM2 channel, including ACA derivatives A23, and 2,3-dihydroquinazolin-4(1H)-one derivatives [1145, 1147].

TRPM4/5 subgroup

TRPM4 and TRPM5 have the distinction within all TRP channels of being impermeable to Ca²⁺ [1080]. A splice variant of TRPM4 (*i.e.* TRPM4b) and TRPM5 are molecular candidates for endogenous calcium-activated cation (CAN) channels [330]. TRPM4 is active in the late phase of repolarization of the cardiac ventricular action potential. TRPM4 deletion or knockout enhances beta adrenergic-mediated inotropy [597]. Mutations are associated with conduction defects [407, 597, 884]. TRPM4 has been shown to be an important regulator of Ca²⁺ entry in to mast cells [999] and dendritic cell migration [52]. TRPM5 in taste receptor cells of the tongue appears essential for the transduction of sweet, amino acid and bitter stimuli [541]. TRPM5 contributes to the slow afterdepolarization of layer 5 neurons in mouse prefrontal cortex [517]. Both TRPM4 and TRPM5 are required transduction of taste stimuli [247].

TRPM6/7 subgroup

TRPM6 and 7 combine channel and enzymatic activities ('chanzymes') [173]. These channels have the unusual property of permeation by divalent (Ca²⁺, Mg²⁺, Zn²⁺) and monovalent cations, high single channel conductances, but overall extremely small inward conductance when expressed to the plasma membrane. They are inhibited by internal Mg²⁺ at ~0.6 mM, around the free level of Mg²⁺ in cells. Whether they contribute to Mg²⁺ homeostasis is a contentious issue. PIP2 is required for TRPM6 and TRPM7 activation [815, 1085]. When either gene is deleted in mice, the result is embryonic lethality [416, 1073]. The C-terminal kinase region of TRPM6 and TRPM7 is cleaved under unknown stimuli, and the kinase phosphorylates nuclear histones [483, 484]. TRPM7 is responsible for oxidant- induced Zn²⁺ release from intracellular vesicles [3] and contributes to intestinal mineral absorption essential for postnatal survival [626]. The putative metal transporter proteins *CNNM1-4* interact with TRPM7 and regulate TRPM7 channel activity [40, 471].

TRPM8

Is a channel activated by cooling and pharmacological agents evoking a 'cool' sensation and participates in the thermosensation of cold temperatures [63, 179, 225] reviewed by [1017, 566, 461, 653]. Direct chemical agonists include menthol and *icilin* [1094]. Besides, *linalool* can promote ERK phosphorylation in human dermal microvascular endothelial cells, down-regulate intracellular ATP levels, and activate TRPM8 [68]. Recent studies have found that TRPM8 has typical S4-S5 connectomes with clear selective filters and exowell rings [516], and have identified cryo-electron microscopy structures of mouse TRPM8 in closed, intermediate, and open states along the ligand- and PIP₂-dependent gated pathways [1119]. Moreover, the last 36 amino acids at the carboxyl terminal of TRPM8 are key protein sequences for TRPM8's temperature-sensitive function [195]. TRPM8 deficiency reduced the expression of S100A9 and increased the expression of HNF4α in the liver of mice, which reduced inflammation and fibrosis progression in mice with liver fibrosis, and helped to alleviate the symptoms of bile duct disease [560]. Channel deficiency also shortens the time of hypersensitivity reactions in migraine mouse models by promoting the recovery of normal sensitivity [12]. A cyclic peptide DeC-1.2 was designed to inhibit ligand activation of TRPM8 but not cold activation, which can eliminate the side effects of cold dysalgesia in oxaliplatin-treated mice without changing body temperature [9]. Analysis of clinical data shows that TRPM8-specific blockers WS12 can reduce tumor growth in colorectal cancer xenografted mice by reducing transcription and activation of Wnt signaling regulators and β-catenin and its target oncogenes, such as C-Myc and Cyclin D1 [736].

TRPML (mucolipin) family

The TRPML family [787, 1140, 780, 1092, 191] consists of three mammalian members (TRPML1-3). TRPML channels are probably restricted to intracellular vesicles and mutations in the gene (*MCOLN1*) encoding TRPML1 (mucolipin-1) cause the neurodegenerative disorder mucopolipidosis type IV (MLIV) in man. TRPML1 is a cation selective ion channel that is important for sorting/transport of endosomes in the late endocytotic pathway and specifically, fission from late endosome-lysosome hybrid vesicles and lysosomal exocytosis [827]. TRPML2 and TRPML3 show increased channel activity in low luminal sodium and/or increased luminal pH, and are activated by similar small molecules [322, 147, 882]. A naturally occurring gain of function mutation in TRPML3 (*i.e.* A419P) results in the varitint waddler (*Va*) mouse phenotype (reviewed by [787, 694]).

TRPP (polycystin) family

The TRPP family (reviewed by [217, 215, 303, 1068, 377]) or PKD2 family is comprised of PKD2 (PC2), PKD2L1 (PC2L1), PKD2L2 (PC2L2), which have been renamed TRPP1, TRPP2 and TRPP3, respectively [1080]. It should also be noted that the nomenclature of PC2 was TRPP2 in old literature. However, PC2 has been uniformed to be called TRPP2 [348]. PKD2 family channels are clearly distinct from the PKD1 family, whose function is unknown. PKD1 and PKD2 form a hetero-oligomeric complex with a 1:3 ratio. [910]. Although still being sorted out, TRPP family members appear to be 6TM spanning nonselective cation channels.

TRPV (vanilloid) family

Members of the TRPV family (reviewed by [1001]) can broadly be divided into the non-selective cation channels, TRPV1-4 and the more calcium selective channels TRPV5 and TRPV6.

TRPV1-V4 subfamily

TRPV1 is involved in the development of thermal hyperalgesia following inflammation and may contribute to the detection of noxious heat (reviewed by [767, 887, 927]). Numerous splice variants of TRPV1 have been described, some of which modulate the activity of TRPV1, or act in a dominant-negative manner when co-expressed with TRPV1 [849]. The pharmacology of TRPV1 channels is discussed in detail in [332] and [1022]. TRPV2 is probably not a thermosensor in man [740], but has recently been implicated in innate immunity [551]. Functional TRPV2 expression is described in placental trophoblast cells of mouse [205]. TRPV3 and TRPV4 are both thermosensitive. There are claims that TRPV4 is also mechanosensitive, but this has not been established to be within a physiological range in a native environment [127, 534].

TRPV5/V6 subfamily

TRPV5 and TRPV6 are highly expressed in placenta, bone, and kidney. Under physiological conditions, TRPV5 and TRPV6 are calcium selective channels involved in the absorption and reabsorption of calcium across intestinal and kidney tubule epithelia (reviewed by [1064, 206, 655, 272]). TRPV6 is reported to play a key role in calcium transport in the mouse placenta [1063].

Contents

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Database links

Transient Receptor Potential channels (TRP)

<https://www.guidetopharmacology.org/GRAC/FamilyDisplayForward?familyId=78>

Introduction to Transient Receptor Potential channels (TRP)

<https://www.guidetopharmacology.org/GRAC/FamilyIntroductionForward?familyId=78>

Channels and Subunits

TRPA1

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=485>

TRPC1

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=486>

TRPC2

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=487>

TRPC3

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=488>

TRPC4

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=489>

TRPC5

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=490>

TRPC6

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=491>

TRPC7

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=492>

TRPM1

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=493>

TRPM2

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=494>

TRPM3

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=495>

TRPM4

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=496>

TRPM5

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=497>

TRPM6

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=498>

TRPM7

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=499>

TRPM8

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=500>

TRPML1

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=501>

TRPML2

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=502>

TRPML3

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=503>

TRPP1

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=504>

TRPP2

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=505>

TRPP3

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=506>

TRPV1

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=507>

TRPV2

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=508>

TRPV3

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=509>

TRPV4

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=510>

TRPV5

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=511>

TRPV6

<https://www.guidetopharmacology.org/GRAC/ObjectDisplayForward?objectId=512>

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