The Concise Guide to PHARMACOLOGY 2015/16

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THE CONCISE GUIDE TO PHARMACOLOGY 2015/16: Overview

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Abstract

The Concise Guide to PHARMACOLOGY 2015/16 provides concise overviews of the key properties of over 1750 human drug targets with their pharmacology, plus links to an open access knowledgebase of drug targets and their ligands (www.guidetopharmacology.org), which provides more detailed views of target and ligand properties. The full contents can be found at http://onlinelibrary.wiley.com/doi/10.1111/bph.13347/full. This compilation of the major pharmacological targets is divided into eight areas of focus: G protein-coupled receptors, ligand-gated ion channels, voltage-gated ion channels, other ion channels, nuclear hormone receptors, catalytic receptors, enzymes and transporters. These are presented with nomenclature guidance and summary information on the best available pharmacological tools, alongside key references and suggestions for further reading. The Concise Guide is published in landscape format in order to facilitate comparison of related targets. It is a condensed version of material contemporary to late 2015, which is presented in greater detail and constantly updated on the website www.guidetopharmacology.org, superseding data presented in the previous Guides to Receptors & Channels and the Concise Guide to PHARMACOLOGY 2013/14. It is produced in conjunction with NC-IUPHAR and provides the official IUPHAR classification and nomenclature for human drug targets, where appropriate. It consolidates information previously curated and displayed separately in IUPHAR-DB and GRAC and provides a permanent, citable, point-in-time record that will survive database updates.

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**Searchable database:** [http://www.guidetopharmacology.org/index.jsp](http://www.guidetopharmacology.org/index.jsp)


Introduction

In order to allow clarity and consistency in pharmacology, there is a need for a comprehensive organisation and presentation of the targets of drugs. This is the philosophy of the IUPHAR/BPS Guide to PHARMACOLOGY presented on the online free access database (http://www.guidetopharmacology.org/). This database is supported by the British Pharmacological Society (BPS), the International Union of Basic and Clinical Pharmacology (IUPHAR), the Wellcome Trust and the University of Edinburgh. Data included in the Guide to PHARMACOLOGY are derived in large part from interactions with the subcommittees of the Nomenclature Committee of the International Union of Basic and Clinical Pharmacology (NC-IUPHAR). The Editors of the Concise Guide have compiled the individual records, in concert with the team of Curators, drawing on the expert knowledge of these latter subcommittees. The tables allow an indication of the status of the nomenclature for the group of targets listed, usually previously published in Pharmacological Reviews. In the absence of an established subcommittee, advice from several prominent, independent experts has generally been obtained to produce an authoritative consensus on nomenclature, which attempts to fit in within the general guidelines from NC-IUPHAR. This current edition, the Concise Guide to PHARMACOLOGY 2015/16, is the latest snapshot of the database in print form, following on from the Concise Guide to PHARMACOLOGY 2013/14. It contains data drawn from the online database as a rapid overview of the major pharmacological targets. Thus, there are fewer targets presented in the Concise Guide (1761) compared to the online database (2761, as of August 2015). The priority for inclusion in the Concise Guide is the presence of quantitative pharmacological data. This means that often orphan family members are not presented in the Con-


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cise Guide, although structural information is available on the online database. An expansion in the current version of the Concise Guide is the increased inclusion of approved drugs, which reflects the aim of the online database to reflect the clinical exploitation of human molecular targets. Although many of these agents are much less selective than the tool compounds listed to define individual targets or groups of targets, we have included them for the significant interest associated with their use and mechanisms of action. The emphasis on approved drugs means that the online database has been expanded to include 8024 ligands (as of August 2015), meaning that additional records now appear in the Concise Guide, primarily in the enzymes section. The organisation of the data is tabular (where appropriate) with a standardised format, where possible on a single page, intended to aid understanding of and comparison within a particular target group. The Concise Guide is intended as an initial resource, with links to additional reviews and resources for greater depth and information. Pharmacological and structural data focus primarily on human gene products, wherever possible, with links to HGNC gene nomenclature and UniProt IDs. In a few cases, where data from human proteins are limited, data from other species are indicated. Pharmacological tools listed are prioritised on the basis of selectivity and availability. That is, agents (agonists, antagonists, inhibitors, activators, etc.) are included where they are both available (by donation or from commercial sources, now or in the near future) AND the most selective. This edition of the Concise Guide is divided into nine sections, which comprise pharmacological targets of similar structure/function. These are G protein-coupled receptors, ligand-gated ion channels, voltage-gated ion channels, other ion channels, catalytic receptors, nuclear hormone receptors, enzymes, transporters and other protein targets. A new aspect of the Concise Guide 2015/16 is that each of these sections contains a complete listing of the families available for inspection on the online database, identifying those families reported in the Concise Guide by their page numbers. We hope that the Concise Guide will provide for researchers, teachers and students a state-of-the-art source of accurate, curated information on the background to their work that they will use in the Introductions to their Research Papers or Reviews, or in supporting their teaching and studies. We recommend that any citations to information in the Concise Guide are presented in the following format: Alexander SPH et al. (2015). The Concise Guide to PHARMACOLOGY 2015/16: Overview. Br J Pharmacol XXX.

A dedication

This Edition of the Concise Guide to PHARMACOLOGY is dedicated to Tony Harmar (1951-2014). Tony was a friend and colleague, who was involved with IUPHAR for over 15 years and worked on the IUPHAR database for over a decade at Edinburgh, working hard to establish the curators as a team of highly informed and informative individuals imbued with Tony’s passion and dogged determination to focus on high-quality data input, ensuring high-quality data output. With time and the resources of the BPS and Wellcome Trust, combined with the expertise of the NC-IUPHAR committee members mentioned above, Tony established the online database at http://www.guidetopharmacology.org/ as the exceptional resource it is today.

Acknowledgements

We are extremely grateful for the financial contributions from the British Pharmacological Society, the International Union of Basic and Clinical Pharmacology, the Wellcome Trust (099156/Z/12/Z), which support the website and the University of Edinburgh, who host the guidetopharmacology.org website. We are also tremendously grateful to the long list of collaborators from NC-IUPHAR subcommittees and beyond, who have assisted in the construction of the Concise Guide to PHARMACOLOGY 2015/16 and the online database www.GuideToPHARMACOLOGY.org

Conflict of interest

The authors state that there are no conflicts of interest to disclose.

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Other Protein Targets

Family structure

- Adiponectin receptors
- B-cell lymphoma 2 (Bcl-2) protein family
- Bromodomain-containing proteins
- Non-enzymatic BRD containing proteins
- Carrier proteins
- CD molecules
- Chromatin-interacting transcriptional repressors
- Methyllysine reader proteins
- Circadian clock proteins
- Cytokines and growth factors
- EF-hand domain containing
- Fatty acid-binding proteins
- Heat shock proteins
- Inhibitors of apoptosis (IAP) protein family
- Kelch-like proteins
- Kinesins
- Mitochondrial-associated proteins
- Notch receptors
- Pentaxins
- Serum pentaxins
- Regulators of G protein signaling (RGS) proteins
- RZ family
- R4 family
- R7 family
- R12 family
- Reticulons
- Ribosomal factors
- Sigma receptors
- Tubulins
- Tumour-associated proteins
- WD repeat-containing proteins

Adiponectin receptors

Other protein targets → Adiponectin receptors

Overview: Adiponectin receptors (provisional nomenclature, ENSM0000000270960) respond to the 30 kDa complement-related protein hormone adiponectin (also known as ADIPOQ: adipocyte, C1q and collagen domain-containing protein; ACRP30, adipose most abundant gene transcript 1; apM-1; gelatin-binding protein: Q15848) originally cloned from adipocytes [49]. Although sequence data suggest 7TM domains, immunological evidence indicates that, contrary to typical 7TM topology, the carboxyl terminus is extracellular, while the amino terminus is intracellular [86]. Signalling through these receptors appears to avoid G proteins. Adiponectin receptors appear rather to stimulate protein phosphorylation via AMP-activated protein kinase and MAP kinase pathways [86], possibly through the protein partner APPL1 (adaptor protein, phosphotyrosine interaction, PH domain and leucine zipper containing 1, Q9UKG1 [52]). The adiponectin receptors are a class of proteins (along with membrane progesterin receptors), which contain seven sequences of aliphatic amino acids reminiscent of GPCRs, but which are structurally and functionally distinct from that class of receptor.

Nomenclature

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<th>Adipo2 receptor</th>
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<td>HGNC, UniProt</td>
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<td>ADIPOR2, Q86V24</td>
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Rank order of potency

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<th>globular adiponectin (ADIPOQ, Q15848)</th>
<th>adiponectin (ADIPOQ, Q15848)</th>
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Comments: T-Cadherin (CDH13, P55290) has also been suggested to be a receptor for (hexameric) adiponectin [35].

Searchable database: http://www.guidetopharmacology.org/index.jsp
Further Reading


Blood coagulation components

Other protein targets → Blood coagulation components

Overview: Coagulation as a patho/physiological process is interpreted as a mechanism for reducing excessive blood loss through the generation of a gel-like clot local to the site of injury. The process involves the activation, adhesion (see Integrins), degranulation and aggregation of platelets, as well as proteins circulating in the plasma. The coagulation cascade involves multiple proteins being converted to more active forms from less active precursors, typically through proteolysis (see Proteases). Listed here are the components of the coagulation cascade targeted by agents in current clinical usage.

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<th>coagulation factor VIII, procoagulant component</th>
<th>serpin peptidase inhibitor, clade C (antithrombin), member 1</th>
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<td>–</td>
<td>–</td>
<td>heparin (pKd 7.8) [25], fondaparinux (pKd 7.5) [65], dalteparin [34], danaparoid [15, 58], enoxaparin [17], tinzaparin [19]</td>
</tr>
<tr>
<td>Selective antagonists</td>
<td>drotrecogin alfa (Inhibition) [40, 41]</td>
<td>drotrecogin alfa (Inhibition) [40, 41]</td>
<td></td>
</tr>
</tbody>
</table>

Further Reading


Non-enzymatic BRD containing proteins

Other protein targets → Bromodomain-containing proteins → Non-enzymatic BRD containing proteins

Overview: Bromodomains bind proteins with acetylated lysine residues, such as histones, to regulate gene transcription. Listed herein are examples of bromodomain-containing proteins for which sufficient pharmacology exists.
Carrier proteins

Other protein targets → Carrier proteins

Overview: TTR is a homo-tetrameric protein which transports thyroxine in the plasma and cerebrospinal fluid and retinol (vitamin A) in the plasma. Many disease causing mutations in the protein have been reported, many of which cause complex dissociation and protein mis-assembly and deposition of toxic aggregates amyloid fibril formation [66]. These amyloidogenic mutants are linked to the development of pathological amyloidoses, including familial amyloid polyneuropathy (FAP) [1, 13], familial amyloid cardiomyopathy (FAC) [37], amyloidotic vitreous opacities, carpal tunnel syndrome [57] and others. In old age, non-mutated TTR can also form pathological amyloid fibrils [85]. Pharmacological intervention to reduce or prevent TTR dissociation is being pursued as a therapeutic strategy. To date one small molecule kinetic stabilising molecule (tafamidis) has been approved for FAP, and is being evaluated in clinical trials for other TTR amyloidoses.

Nomenclature

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>transthyretin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common abbreviation</td>
<td>TTR</td>
</tr>
<tr>
<td>HGNC, UniProt</td>
<td>TTR, P02766</td>
</tr>
</tbody>
</table>
CD molecules
Other protein targets → CD molecules

Overview: Cluster of differentiation refers to an attempt to catalogue systematically a series of over 300 cell-surface proteins associated with immunotyping. Many members of the group have identified functions as enzymes (for example, see CD73 ecto-5’-nucleotidase) or receptors (for example, see CD41 integrin, alpha 2b subunit). Many CDs are targeted for therapeutic gain using antibodies for the treatment of proliferative disorders. A full listing of all the Clusters of Differentiation is not possible in the Guide to PHARMACOLOGY; listed herein are selected members of the family targeted for therapeutic gain.

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>CD2</th>
<th>CD3e molecule, epsilon (CD3-TCR complex)</th>
<th>CD20 (membrane-spanning 4-domains, subfamily A, member 1)</th>
<th>CD33</th>
<th>CD52</th>
<th>CD80</th>
<th>CD86</th>
<th>cytotoxic T-lymphocyte-associated protein 4 (CD152)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common abreviation</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>CTLa-4</td>
</tr>
<tr>
<td>HGNC, UniProt</td>
<td>CD2, P06729</td>
<td>CD3E, P07766</td>
<td>MS4A1, P11836</td>
<td>CD33, P20138</td>
<td>CD52, P31358</td>
<td>CD80, P33681</td>
<td>CD86, P42081</td>
<td>CTL4, P16410</td>
</tr>
<tr>
<td>Selective inhibitors</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Selective antagonists</td>
<td>alefacept (Inhibition) [56, 89]</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Antibodies</td>
<td>–</td>
<td>catumaxomab (Binding) [46], muromonab-CD3 (Binding) [24], otezolizumab (Binding) [7]</td>
<td>ofatumumab (Binding) (pKd 9.9) [47], rituximab (Binding) (pKd 8.5) [78], ibritumomab tiuxetan (Binding), obinutuzumab (Binding) [2, 68], tositumomab (Binding)</td>
<td>lintuzumab (Binding) (pKd ~10) [8], gemtuzumab ozogamicin (Binding) [6]</td>
<td>alemtuzumab (Binding) [22]</td>
<td>–</td>
<td>–</td>
<td>ipilimumab (Binding) (pKd ~9) [28], tremelimumab (Binding) (pKd 8.9) [30]</td>
</tr>
</tbody>
</table>

Searchable database: [http://www.guidetopharmacology.org/index.jsp](http://www.guidetopharmacology.org/index.jsp)
Nomenclature: programmed cell death 1 (CD279)  
Common abbreviation: PD-1  
HGNC, UniProt: PDCD1, Q15116  
Antibodies: pembrolizumab (Binding) (pK_d 10) [9], nivolumab (Binding) (pK_d 9.1) [29, 42, 43]  
Comments: The endogenous ligands for human PD-1 are programmed cell death 1 ligand 1 (PD-L1 aka CD274, Q9NZQ7) and programmed cell death 1 ligand 2 (PD-L2; PDCD1LG2). These ligands are cell surface peptides, normally involved in immune system regulation. Many types of cancer cells evolve mechanisms to evade control and elimination by the immune system. Such mechanisms can include inhibition of so-called ‘immune checkpoints’, which would normally be involved in the maintenance of immune homeostasis. An increasingly important area of clinical oncology research is the development of new agents which impede these evasion techniques, thereby switching immune vigilance back on, and effecting immune destruction of cancer cells. Three molecular targets of checkpoint inhibitors which are being extensively pursued are cytotoxic T-lymphocyte antigen 4 (CTLA4), programmed cell death 1 (PD-1), and programmed cell death ligand 1 (PD-L1). Using antibody-based therapies targeting these pathways, clinical responses have been reported in various tumour types, including melanoma, renal cell carcinoma [64] and non-small cell lung cancer [39, 51]. pembrolizumab is the first-in-class, anti-PD-1 antibody to be approved by the US FDA, with ongoing clinical trials for nivolumab (e.g. NCT01673867, NCT01721746) and pidilizumab (NCT02077959, NCT01952769).

Methyllysine reader proteins

Overview: Methyllysine reader proteins bind to methylated proteins, such as histones, allowing regulation of gene expression.

Further Reading

Cytokines and growth factors

Other protein targets → Cytokines and growth factors

**Overview**: cytokines and growth factors are a group of small proteins released from cells, which act upon the same cell or neighbouring cells, often with a role in immune regulation and/or proliferation. Listed herein are examples of cytokines and growth factors targeted for therapeutic benefit.

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>Antagonists</th>
<th>Selective antagonists</th>
<th>Antibodies</th>
</tr>
</thead>
<tbody>
<tr>
<td>interleukin 1, beta (IL1β)</td>
<td>TNF, P01375</td>
<td></td>
<td>gevokizumab (Binding) (pKd 12.5)</td>
</tr>
<tr>
<td>HGNC, UniProt</td>
<td>−</td>
<td>−</td>
<td>(IL1B) [36, 53, 71]</td>
</tr>
<tr>
<td>Antagonists</td>
<td>−</td>
<td>−</td>
<td>etanercept (Inhibition) [18, 23]</td>
</tr>
<tr>
<td>Selective antagonists</td>
<td>−</td>
<td>−</td>
<td>ranibizumab (Inhibition) (pKd ~9.8) [3]</td>
</tr>
<tr>
<td>Selective antagonists</td>
<td>−</td>
<td>−</td>
<td>pegaptanib (Inhibition) [26, 61]</td>
</tr>
<tr>
<td>Antibodies</td>
<td>−</td>
<td>−</td>
<td>infliximab (Inhibition) (pKd 8.7) [44]</td>
</tr>
<tr>
<td>(Binding) (pKd 10.5) [27]</td>
<td></td>
<td></td>
<td>adalimumab (Inhibition) (pKd &gt;8)  [75]</td>
</tr>
<tr>
<td>(Binding) [32, 55]</td>
<td></td>
<td></td>
<td>certolizumab pegol (Inhibition) [60]</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatty acid-binding proteins</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Overview**: Fatty acid-binding proteins are low molecular weight (100-130 aa) chaperones for long chain fatty acids, fatty acyl CoA esters, eicosanoids, retinols, retinoic acids and related metabolites and are usually regarded as being responsible for allowing the otherwise hydrophobic ligands to be mobile in aqueous media. These binding proteins may perform functions extracellularly (e.g. in plasma) or transport these agents; to the nucleus to interact with nuclear receptors (principally PPARs and retinoic acid receptors [76]) or for interaction with metabolic enzymes. Although sequence homology is limited, crystallographic studies suggest conserved 3D structures across the group of binding proteins.
### Nomenclature

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>fatty acid binding protein 6, ileal</th>
<th>fatty acid binding protein 7, brain</th>
<th>peripheral myelin protein 2</th>
<th>fatty acid binding protein 9, tests</th>
<th>fatty acid binding protein 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGNC, UniProt</td>
<td>FABP6, P51161</td>
<td>FABP7, O15540</td>
<td>PMP2, P02689</td>
<td>FABP9, Q0Z758</td>
<td>FABP12, A6NFH5</td>
</tr>
<tr>
<td>Comments</td>
<td>Able to transport bile acids [88].</td>
<td>Crystal structure of the human FABP7 [4].</td>
<td>In silico modelling suggests that FABP8 can bind both fatty acids and cholesterol [50].</td>
<td>– -</td>
<td>– -</td>
</tr>
</tbody>
</table>

### Nomenclature

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>retinol binding protein 1, cellular</th>
<th>retinol binding protein 2, cellular</th>
<th>retinol binding protein 3, interstitial</th>
<th>retinol binding protein 4, plasma</th>
<th>retinol binding protein 5, cellular</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGNC, UniProt</td>
<td>RBP1, P09455</td>
<td>RBP2, P05120</td>
<td>RBP3, P10745</td>
<td>RBP4, P02753</td>
<td>RBP5, P82980</td>
</tr>
<tr>
<td>Rank order of potency</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

### Nomenclature

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>retinol binding protein 7, cellular</th>
<th>retinaldehyde binding protein 1</th>
<th>cellular retinoic acid binding protein 1</th>
<th>cellular retinoic acid binding protein 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGNC, UniProt</td>
<td>RBP7, Q96R05</td>
<td>RLBP1, P12271</td>
<td>CRABP1, P29762</td>
<td>CRABP2, P29373</td>
</tr>
<tr>
<td>Rank order of potency</td>
<td>–</td>
<td>11- cis-retinal, 11-cis-retinol -&gt; 9-cis-retinal, 13-cis-retinal, 13-cis-retinol, all-trans-retinal, retinol [14]</td>
<td>tretinoin -&gt; alitretinoin stearic acid -&gt; palmitic acid, oleic acid, linoleic acid, α-linolenic acid, arachidonic acid [70]</td>
<td>–</td>
</tr>
</tbody>
</table>

### Comments

Although not tested at all FABPs, BMS309403 exhibits high affinity for FABP4 (pIC50 8.8) compared to FABP3 or FABP5 (pIC50 – 6.6) [20, 81]. HTS01037 is reported to interfere with FABP4 action [31]. Multiple pseudogenes for the FABPs have been identified in the human genome.

### Further Reading


Schroeder F et al. (2008) Role of fatty acid binding proteins and long chain fatty acids in modulating nuclear receptors and gene transcription. Lipids 43: 1-17 [PMID:17882463]


Sigma receptors

Other protein targets → Sigma receptors

Overview: Although termed ‘receptors’, the evidence for coupling through conventional signalling pathways is lacking. Initially described as a subtype of opioid receptors, there is only a modest pharmacological overlap and no structural convergence with the G protein-coupled receptors. A wide range of compounds, ranging from psychoactive agents to antihistamines, have been observed to bind to these sites, which appear to be intracellular.

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>sigma non-opioid intracellular receptor 1</th>
<th>α2</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGNC, UniProt</td>
<td>S1GMAR1, Q99720</td>
<td>–</td>
</tr>
<tr>
<td>Agonists</td>
<td>–</td>
<td>PB-28 (pKᵢ 8.3) [5], 1,3-ditolylguanidine (pKᵢ 7.4) [45] – Guinea pig</td>
</tr>
<tr>
<td>(Sub)family-selective agonists</td>
<td>(RS)-PPCC (pKᵢ 8.8) [67]</td>
<td>–</td>
</tr>
<tr>
<td>Selective agonists</td>
<td>PRE-084 (pIC₅₀ 7.4) [80], (+)-SK&amp;F10047</td>
<td>–</td>
</tr>
<tr>
<td>Antagonists</td>
<td>(-)-pentazocine</td>
<td>SM 21 (pIC₅₀ 7.2) [48]</td>
</tr>
<tr>
<td>Selective antagonists</td>
<td>NE-100 (pIC₅₀ 8.4) [62], BD-1047 (pIC₅₀ 7.4) [54]</td>
<td>–</td>
</tr>
<tr>
<td>Labelled ligands</td>
<td>[³H]pentazocine (Agonist)</td>
<td>[³H]-di-o-tolyguanidine (Agonist)</td>
</tr>
<tr>
<td>Comments</td>
<td>–</td>
<td>There is no molecular correlate of the α2 receptor.</td>
</tr>
</tbody>
</table>

Comments: (-)-pentazocine also shows activity at opioid receptors.

Further Reading


Searchable database: http://www.guidetopharmacology.org/index.jsp
**Tubulins**

**Other protein targets → Tubulins**

**Overview**: Tubulins are a family of intracellular proteins most commonly associated with microtubules, part of the cytoskeleton. They are exploited for therapeutic gain in cancer chemotherapy as targets for agents derived from a variety of natural products: taxanes, colchicine and vinca alkaloids. These are thought to act primarily through β-tubulin, thereby interfering with the normal processes of tubulin polymer formation and disassembly.

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>tubulin, alpha 1a</th>
<th>tubulin, alpha 4a</th>
<th>tubulin, beta class I</th>
<th>tubulin, beta 3 class III</th>
<th>tubulin, beta 4B class IVb</th>
<th>tubulin, beta 8 class VIII</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGNC, UniProt</td>
<td>TUBA1A, Q71U36</td>
<td>TUBA4A, P68366</td>
<td>TUBB, P07437</td>
<td>TUBB3, Q13509</td>
<td>TUBB4B, P68371</td>
<td>TUBB8, Q3ZCM7</td>
</tr>
<tr>
<td>Inhibitors</td>
<td>-</td>
<td>-</td>
<td>vinblastine (pIC_50 9), vincristine</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>(Sub)family-selective inhibitors</td>
<td>-</td>
<td>-</td>
<td>eribulin (pIC_50 8.2) [59], paclitaxel (Mitotic cell cycle arrest in A431 cells) (pEC_50 8.1) [63], colchicine (pIC_50 8) [12], cabazitaxel, docetaxel, ixabepilone</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

**Further Reading**


References

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