THE CONCISE GUIDE TO PHARMACOLOGY 2013/14: OVERVIEW

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Abstract

The Concise Guide to PHARMACOLOGY 2013/14 provides concise overviews of the key properties of over 2000 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 from the IUPHAR database. The full contents can be found at http://onlinelibrary.wiley.com/doi/10.1111/bph.12444/full.

This compilation of the major pharmacological targets is divided into seven areas of focus: G protein-coupled receptors, ligand-gated ion channels, ion channels, catalytic receptors, nuclear hormone receptors, transporters and enzymes. These are presented with nomenclature guidance and summary information on the best available pharmacological tools, alongside key references and suggestions for further reading. A new landscape format has easy to use tables comparing related targets.

It is a condensed version of material contemporary to late 2013, which is presented in greater detail and constantly updated on the website www.guidetopharmacology.org, superseding data presented in previous Guides to Receptors & Channels. 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.

Table of contents

1449  OVERVIEW
1454  Adiponectin receptors
1455  Fatty acid binding proteins
1457  Sigma receptors
1459  G PROTEIN-COUPLED RECEPTORS
1462  Orphan GPCRs
1471  5-Hydroxytryptamine receptors
1474  Acetylcholine receptors (muscarinic)
1476  Adenosine receptors
1478  Adhesion Class GPCRs
1480  Adrenoceptors
1484  Angiotensin receptors
1485  Apelin receptor
1486  Bile acid receptor
1487  Bombsin receptors
1488  Bradykinin receptors
1489  Calcitonin receptors
1491  Calcium-sensing receptors
1492  Cannabinoid receptors
1494  Chemerin receptor
1495  Chemokine receptors
1500  Cholecystokinin receptors
1501  Complement peptide receptors
1502  Corticotropin-releasing factor receptors
1503  Dopamine receptors
1505  Endothelin receptors
1506  Estrogen (G protein-coupled) receptor
1507  Formylpeptide receptors
1508  Free fatty acid receptors
1510  Frizzled Class GPCRs
1511  GABA receptors
1513  Galanin receptors
1514  Ghrelin receptor
1515  Glucagon receptor family
1517  Glycoprotein hormone receptors
1518  Gonadotrophin-releasing hormone receptors
1519  GPR18, GPR55 and GPR119
1520  Histamine receptors
1521 Hydroxycarboxylic acid receptors
1522 Kisspeptin receptors
1523 Leukotriene, lipoxin and oxoeicosanoid receptors
1525 Lysophospholipid (LPA) receptors
1526 Lysophosphatidylcholine (PLC) receptors
1527 Melanin-concentrating hormone receptors
1528 Melatonin receptors
1530 Metabotropic glutamate receptors
1532 Motilin receptor
1533 Neuromedin U receptors
1534 Neuropeptide FF/neuropeptide F receptors
1535 Neuropeptide S receptor
1536 Neuropeptide W/neuropeptide B receptors
1537 Neureptide Y receptors
1538 Neurotensin receptors
1539 Opioid receptors
1541 Orexin receptors
1542 Oxoglutarate receptor
1543 P2Y receptors
1545 Parathyroid hormone receptors
1546 Peptide PS18 receptor
1547 Platelet-activating factor receptor
1548 Prokineticin receptors
1549 Prolactin-releasing peptide receptor
1550 Prostanoid receptors
1552 Proteinase-activated receptors
1553 Relaxin family peptide receptors
1555 Somatostatin receptors
1556 Succinate receptor
1557 Tachykinin receptors
1558 Thyrotropin-releasing hormone receptors
1559 Trace amine receptor
1560 Urotensin receptor
1561 Vasopressin and oxytocin receptors
1562 VIP and PACAP receptors

1582 LIGAND-GATED ION CHANNELS
1584 S-HT3 receptors
1586 GABA, receptors
1590 Glycine receptors
1592 Iontromotropic glutamate receptors
1597 Nicotinic acetylcholine receptors
1601 P2X receptors
1603 ZAC

1607 ION CHANNELS
1609 Acid-sensing (proton-gated) ion channels (ASICs)
1611 Aquaporins

1612 CatSper and Two-Pore channels
1613 Chloride channels
1620 Connexins and Panxexins
1621 Cyclic nucleotide-regulated channels
1623 Epithelial sodium channels (ENaC)
1625 IP, receptor
1626 Potassium channels
1630 Bynadine receptor
1632 Sodium leak channel, non-selective
1633 Transient receptor potential channels
1643 Voltage-gated calcium channels
1645 Voltage-gated proton channel
1646 Voltage-gated sodium channels

1652 NUCLEAR HORMONE RECEPTORS
1654 1A. Thyroid Hormone Receptors
1655 1B. Retinoic acid receptors
1656 1C. Peroxisome proliferator-activated receptors
1657 1D. Rev-Erb receptors
1658 1F. Retinoic acid-related orphans
1659 1H. Liver X receptor-like receptors
1660 1I. Vitamin D receptor-like receptors
1661 2A. Hepatocyte nuclear factor-4 receptors
1662 2B. Retinoid X receptors
1663 2C. Testicular receptors
1664 2E. Tailess-like receptors
1665 2F. COUP-TF-like receptors
1666 3B. Estrogen-related receptors
1667 4A. Nerve growth factor IB-like receptors
1668 5A. Fushi tarazu FL-like receptors
1669 6A. Germ cell nuclear factor receptors
1670 8B. DAX-like receptors
1671 Steroid hormone receptors

1676 CATALYTIC RECEPTORS
1678 Cytokine receptor family
1684 GDNF receptor family
1685 Integrins
1688 Natriuretic peptide receptor family
1689 Pattern Recognition receptors
1692 Receptor serine/threonine kinase (RSTK) family
1695 Receptor tyrosine kinases
1702 Receptor tyrosine phosphatases (RTP)
1703 Tumour necrosis factor (TNF) receptor family

1706 TRANSPORTERS
1708 ATP-binding cassette transporter family
1712 F-type and V-type ATPases
1714 P-type ATPases

1717 SLC1 family of amino acid transporters
1719 SLC2 family of hexose and sugar alcohol transporters
1721 SLC3 and SLC7 families of heteromeric amino acid transporters (HATs)
1723 SLC4 family of bicarbonate transporters
1724 SLC5 family of sodium-dependent glucose transporters
1728 SLC6 neurotransmitter transporter family
1732 SLC8 family of sodium/calcium exchangers
1733 SLC9 family of sodium/hydrogen exchangers
1734 SLC10 family of sodium-bile acid co-transporters
1736 SLC11 family of proton-coupled metal ion transporters
1737 SLC12 family of cation-coupled chloride transporters
1739 SLC13 family of sodium-dependent sulphate/carboxylate transporters
1740 SLC14 family of facilitative urea transporters
1741 SLC15 family of peptide transporters
1742 SLC16 family of monocarboxylate transporters
1744 SLC17 phosphate and organic anion transporter family
1746 SLC18 family of vesicular amine transporters
1748 SLC19 family of vitamin transporters
1749 SLC20 family of sodium-dependent phosphate transporters
1750 SLC22 family of organic cation and anion transporters
1753 SLC23 family of ascorbic acid transporters
1754 SLC24 family of sodium/potassium/calcium exchangers
1755 SLC25 family of mitochondrial transporters
1760 SLC26 family of anion exchangers
1762 SLC27 family of fatty acid transporters
1763 SLC28 and SLC29 families of nucleoside transporters
1765 SLC30 zinc transporter family
1766 SLC31 family of copper transporters
1767 SLC32 vesicular inhibitory amino acid transporter
1768 SLC33 acetylCoA transporter
1769 SLC34 family of sodium phosphate co-transporters
1770 SLC35 family of nucleotide sugar transporters
1772 SLC36 family of proton-coupled amino acid transporters
1773 SLC37 family of phosphosugar/phosphate exchangers
1774 SLC38 family of sodium-dependent neutral amino acid transporters
1776 SLC39 family of metal ion transporters
1777 SLC40 iron transporter
1778 SLC41 family of divalent cation transporters
1779 SLC42 family of Rhesus glycoprotein ammonium transporters
1780 SLC43 family of large neutral amino acid transporters
1781 SLC44 choline transporter-like family
1782 SLC45 family of putative sugar transporters
1783 SLC46 family of folate transporters
The great proliferation of drug targets in recent years has driven the need to provide a logically-organised synopsis of the nomenclature and pharmacology of these targets. This is the underlying reason for this Guide to PHARMACOLOGY 2013/14, distributed with the British Journal of Pharmacology, and produced in association with NC-IUPHAR, the Nomenclature Committees of the International Union of Basic and Clinical Pharmacology. Our intent is to produce an authoritative but user-friendly publication, which allows a rapid overview of the key properties of a wide range of established or potential pharmacological targets. The aim is to provide information succinctly, so that a newcomer to a particular target group can identify the main elements ‘at a glance’. It is not our goal to produce all-inclusive reviews of the targets presented; references to these are included in the Further Reading sections of the entries or, for many targets, the website www.guidetopharmacology.org provides access to more extensive information. The Guide to PHARMACOLOGY 2013/14 presents each entry, typically a circumscribed target class family on, wherever possible, a single page, so as to allow easy access and rapid oversight.

The list of targets present is, in many cases, a comprehensive reflection of the known targets within the particular group. Our philosophy has been to present data on human proteins wherever possible, both in terms of structural information and pharmacology. To this end, the HGNC gene nomenclature and UniProt unique ID are indicated to allow rapid access through free online databases for further information. In a few cases, where structural or pharmacological information is not available for human targets, we have used data from other species, as indicated. A priority in constructing these tables was to present agents which represent the most selective and which are available by donation or from commercial sources, now or in the near future.

The Guide is divided into seven further sections, which comprise pharmacological targets of similar structure/function. These are G protein-coupled receptors, ligand-gated ion channels, ion channels, catalytic receptors, nuclear hormone receptors, transporters and enzymes. In this overview are listed protein targets of pharmacological interest, which are not G protein-coupled receptors, ligand-gated ion channels, ion channels, nuclear hormone receptors, catalytic receptors, transporters or enzymes. In comparison with the Fifth Edition of the Guide to Receptors & Channels [1], we have added a number of new records, expanding the total to include over 2000 protein targets, primarily from increasing the content on transporters and enzymes.

The Editors of the Guide have compiled the individual records, taking advice from many Collaborators (listed on page 1452). Where appropriate, an indication is given of the status of the nomenclature, as proposed by NC-IUPHAR, published in Pharmacological Reviews. Where this guidance is lacking, advice from several prominent, independent experts has generally been obtained to produce an authoritative consensus, which attempts to fit in within the general guidelines from NC-IUPHAR [2]. Tabulated data provide ready comparison of selective agents and probes (radioligands and PET ligands, where available) within a family of targets and additional commentary highlights whether species differences or ligand metabolism are potential confounding factors. We recommend that any citations to information in the Concise Guide are presented in the following format:


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ID KERR, Nottingham, UK
AA KHAN, Chicago, USA
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JC MCGRATH, Glasgow, UK
MC MICHEL, Amsterdam, The Netherlands
NS MILLAR, London, UK
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Conflict of interest

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

List of records presented

1454  Adiponectin receptors
1455  Fatty acid binding proteins
1457  Sigma receptors
Overview: Adiponectin receptors (provisional nomenclature, ENSFM005000000270960) 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 [4]. 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 [6]. 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 [6], possibly through the protein partner APPL1 (adaptor protein, phosphotyrosine interaction, PH domain and leucine zipper containing 1, Q9UKG1 [5]). The adiponectin receptors are a class of proteins (along with membrane progestin receptors), which contain seven sequences of aliphatic amino acids reminiscent of GPCRs, but which are structurally and functionally distinct from that class of receptor.

<table>
<thead>
<tr>
<th>Nomenclature</th>
<th>Adipo1 receptor</th>
<th>Adipo2 receptor</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGNC, UniProt</td>
<td>ADIPOR1, Q96A54</td>
<td>ADIPOR2, Q86V24</td>
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<tr>
<td>Rank order of potency</td>
<td>globular adiponectin &gt; adiponectin</td>
<td>globular adiponectin = adiponectin</td>
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</table>

Comments: T-Cadherin (CDH13, P55290) has also been suggested to be a receptor for (hexameric) adiponectin [3].

Further reading


Fatty acid binding proteins

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 [16]) or for interaction with metabolic enzymes. Although sequence homology is limited, crystallographic studies suggest conserved 3D structures across the group of binding proteins.

<table>
<thead>
<tr>
<th>Preferred abbreviation</th>
<th>Nomenclature</th>
<th>HGNC, UniProt</th>
<th>Rank order of potency</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>FABP1</td>
<td>fatty acid binding protein 1, liver</td>
<td>FABP1, P07148</td>
<td>stearic acid, oleic acid &gt; palmitic acid, linoleic acid &gt; arachidonic acid, α-linolenic acid [13]</td>
<td>A broader substrate specificity than other FABPs, binding two fatty acids per protein [18]</td>
</tr>
<tr>
<td>FABP2</td>
<td>fatty acid binding protein 2, intestinal</td>
<td>FABP2, P12104</td>
<td>stearic acid &gt; palmitic acid, oleic acid &gt; linoleic acid &gt; arachidonic acid, α-linolenic acid [13]</td>
<td>Crystal structure of the rat FABP2 [15]</td>
</tr>
<tr>
<td>FABP3</td>
<td>fatty acid binding protein 3, muscle and heart (mammary-derived growth inhibitor)</td>
<td>FABP3, P05413</td>
<td>stearic acid, oleic acid, palmitic acid &gt; linoleic acid, α-linolenic acid, arachidonic acid [13]</td>
<td>Crystal structure of the human FABP3 [19]</td>
</tr>
<tr>
<td>FABP4</td>
<td>fatty acid binding protein 4, adipocyte</td>
<td>FABP4, P15090</td>
<td>oleic acid, palmitic acid, stearic acid, linoleic acid &gt; α-linolenic acid, arachidonic acid [13]</td>
<td>Crystal structure of the human FABP5 [11]</td>
</tr>
<tr>
<td>FABP5</td>
<td>fatty acid binding protein 5 (psoriasis-associated)</td>
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<td></td>
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<tr>
<td>FABP6</td>
<td>fatty acid binding protein 6, ileal</td>
<td>FABP6, P51161</td>
<td></td>
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<tr>
<td>FABP7</td>
<td>fatty acid binding protein 7, brain</td>
<td>FABP7, O15540</td>
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<tr>
<td>FABP8</td>
<td>peripheral myelin protein 2</td>
<td>FABP8, P02689</td>
<td></td>
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<tr>
<td>FABP9</td>
<td>fatty acid binding protein 9, tests</td>
<td>FABP9, Q0Z7S8</td>
<td></td>
<td>In silico modelling suggests that FABP8 can bind both fatty acids and cholesterol [12]</td>
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<tr>
<td>FABP12</td>
<td>fatty acid binding protein 12</td>
<td>FABP12, A6NFH5</td>
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<td>RBP1</td>
<td>retinol binding protein 1, cellular</td>
<td>RBP1, P09455</td>
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<tr>
<td>RBP2</td>
<td>retinol binding protein 2, cellular</td>
<td>RBP2, P50120</td>
<td>stearic acid &gt; palmitic acid, oleic acid, linoleic acid, α-linolenic acid, arachidonic acid [14]</td>
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<tr>
<td>RBP3</td>
<td>retinol binding protein 3, interstitial</td>
<td>RBP3, P10745</td>
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<td>RBP4</td>
<td>retinol binding protein 4, plasma</td>
<td>RBP4, P02753</td>
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<td>RBP5</td>
<td>retinol binding protein 5, cellular</td>
<td>RBP5, P82980</td>
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Searchable database: http://www.guidetopharmacology.org/index.jsp
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<thead>
<tr>
<th>Preferred abbreviation</th>
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<th>RLBP1</th>
<th>CRABP1</th>
<th>CRABP2</th>
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<tr>
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<td>retinol binding protein 7, cellular</td>
<td>retinaldehyde binding protein 1</td>
<td>cellular retinoic acid binding protein 1</td>
<td>cellular retinoic acid binding protein 2</td>
</tr>
<tr>
<td>HGNC, UniProt</td>
<td>RBP7, Q96R05</td>
<td>RLBP1, P12271</td>
<td>CRABP1, P29762</td>
<td>CRABP2, P29373</td>
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</table>

Comments: Although not tested at all FABPs, BMS309403 exhibits high affinity for FABP4 (pIC_{50} ∼ 8.8) compared to FABP3 or FABP5 (pIC_{50} < 6.6, [9,17]). HTS01037 is reported to interfere with FABP4 action [10]. Multiple pseudogenes for the FABPs have been identified in the human genome.

Further reading

**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>σ1 (sigma non-opioid intracellular receptor 1)</th>
<th>σ2</th>
</tr>
</thead>
<tbody>
<tr>
<td>HGNC, UniProt</td>
<td>SIGMAR1, Q99720</td>
<td></td>
</tr>
<tr>
<td>Selective agonists</td>
<td>(+)-SK&amp;F10047, (RS)-PPCC (pK 8.8) [25], PRE-084 (pIC50 7.4) [26]</td>
<td>PB-28 (pK 8.3) [21]</td>
</tr>
<tr>
<td>Selective antagonists</td>
<td>NE-100 (pIC50 8.4) [24], BD-1047 (pIC50 7.4) [23]</td>
<td>(RS)-SM21 (pIC50 7.2) [22]</td>
</tr>
<tr>
<td>Radioligands (Kd)</td>
<td>[3H]-pentazocine (Agonist)</td>
<td>[3H]-di-o-tolyguanidine (Agonist)</td>
</tr>
</tbody>
</table>

**Comments:** (-)-pentazocine also shows activity at opioid receptors. There is no molecular correlate of the sigma2 receptor.

**Further reading**


References