

OPINION

Drugs, their targets and the nature and number of drug targets

Peter Imming, Christian Sinning and Achim Meyer

Abstract | What is a drug target? And how many such targets are there? Here, we consider the nature of drug targets, and by classifying known drug substances on the basis of the discussed principles we provide an estimation of the total number of current drug targets.

Estimations of the total number of drug targets are presently dominated by analyses of the human genome, which are limited for various reasons, including the inability to infer the existence of splice variants or interactions between the encoded proteins from gene sequences alone, and the fact that the function of most of the DNA in the genome remains unclear. In 1997, when 100,000 protein-coding sequences were hypothesized to exist in the human genome, Drews and Ryser estimated the number of molecular targets 'hit' by all marketed drug substances to be only 482 (REF. 1). In 2002, after the sequencing of the human genome, others arrived at ~8,000 targets of pharmacological interest, of which nearly 5,000 could be potentially hit by traditional drug substances, nearly 2,400 by antibodies and ~800 by protein pharmaceuticals². And on the basis of ligand-binding studies, 399 molecular targets were identified belonging to 130 protein families, and ~3,000 targets for small-molecule drugs were predicted to exist by extrapolations from the number of currently identified such targets in the human genome³.

In summary, current target counts are of the order of 10², whereas estimations of the number of potential drug targets are an order of magnitude higher. In this paper, we consider the nature of drug targets, and use a classification based on this consideration, and a list of approved drug substances (TABLES 1–8, BOX 1), to estimate the number of known drug targets, in the following categories:

- Enzymes (TABLE 1)
- Substrates, metabolites and proteins (TABLE 2)
- Receptors (TABLE 3)
- Ion channels (TABLE 4)
- Transport proteins (TABLE 5)
- DNA/RNA and the ribosome (TABLE 6)
- Targets of monoclonal antibodies (TABLE 7)
- Various physicochemical mechanisms (TABLE 8)
- Unknown mechanism of action (BOX 1)

The nature of drug targets

A prerequisite for counting the number of targets is defining what a target is. Indeed, this is the crucial, most difficult and also most arbitrary part of the present approach. For the purpose of this paper, we consider a target to be a molecular structure (chemically definable by at least a molecular mass) that will undergo a specific interaction with chemicals that we call drugs because they are administered to treat or diagnose a disease. The interaction has a connection with the clinical effect(s).

This definition implies several constraints. First, the medicinal goal excludes pharmacological and biochemical tools from the present approach. Second, a major constraint is a lack of technique. Life, including disease, is dynamic, but as we do not yet directly observe the interactions of drugs and targets, and only partly notice the subsequent biochemical 'ripples' they produce; we are generally limited to 'still life' (for example, X-ray crystal structures)

and to treating targets as static objects.

In the case of G-protein-coupled receptors (GPCRs), the pharmaceutically most useful class of receptors, a re-organization of the protein after drug binding was derived from biochemical data⁴, but such approaches are still in their infancy.

For most drugs, several if not many targets were identified. Consequently, we had to decide for every drug substance or drug class which target(s) to include in our list. For this, we relied on the existence of literature data that showed some connection between the interaction of the drug with the biochemical structure of the target and the clinical effect(s) (not side effects).

A chemical with a certain reactivity or binding property is used as a drug because of its clinical effects, but it should be stressed that it can be challenging to prove that a certain molecular interaction is indeed the one triggering the effect(s). In this respect, knockout mice are proving increasingly useful. For example, a lack of effect of a drug in mice lacking a particular target can provide strong support that the effects of the drug are mediated by that target (for a review on knockout mice in target validation, see REF. 5).

We therefore considered the construction of knockout animals that lack the target, with pertinent observation of effects, strong proof or disproof for a certain mechanism of action. In the case of receptors, we regarded the availability and testing of both agonists and antagonists (and/or inverse agonists) proof for a mechanism. In the case of enzyme inhibitors (for example, cyclooxygenase inhibitors), molecular interactions and effects of structurally unrelated substances that are largely identical were considered proof of the mechanism. In cases where a drug interaction on the biochemical level was found, but the biochemical pathway was not yet known to be connected with the observed drug effect, the target was not counted. For antipsychotic drugs in particular, a plethora of target receptors and receptor subtypes are known and discussed (see PDSP⁵, Database in Further information and BOX 2). However, extensive discussion of such issues is outside the scope of an article

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Table 1a | **Enzymes**

Type	Activity of drug	Drug examples
Oxidoreductases		
Aldehyde dehydrogenase	Inhibitor	Disulfiram ³⁹
Monoamine oxidases (MAOs)	MAO-A inhibitor	Tranylcypromine ⁴⁰ , moclobemide ⁴¹
	MAO-B inhibitor	Tranylcypromine ⁴⁰
Cyclooxygenases (COXs)	COX1 inhibitor	Acetylsalicylic acid, profens, acetaminophen and dipyron (as arachidonylamides) ^{42,43}
	COX2 inhibitor	Acetylsalicylic acid, profens, acetaminophen and dipyron (as arachidonylamides) ⁴⁴
Vitamin K epoxide reductase	Inhibitor	Warfarin, phenprocoumon ⁴⁵
Aromatase	Inhibitor	Exemestane ⁴⁶
Lanosterol demethylase (fungal)	Inhibitor	Azole antifungals ⁴⁷
Lipoxygenases	Inhibitor	Mesalazine ⁴⁸
	5-lipoxygenase inhibitor	Zileuton ⁴⁹
Thyroidal peroxidase	Inhibitor	Thiouracils ⁵⁰
Iodothyronine-5' deiodinase	Inhibitor	Propylthiouracil ⁵⁰
Inosine monophosphate dehydrogenase	Inhibitor	Mycophenolate mofetil ⁵¹
HMG-CoA reductase	Inhibitor	Statins ⁵²
5 α -Testosterone reductase	Inhibitor	Finasteride, dutasteride ⁵³
Dihydrofolate reductase (bacterial)	Inhibitor	Trimethoprim ⁵⁴
Dihydrofolate reductase (human)	Inhibitor	Methotrexate, pemetrexed ⁵⁵
Dihydrofolate reductase (parasitic)	Inhibitor	Proguanil ⁵⁶
Dihydroorotate reductase	Inhibitor	Leflunomide ⁵⁷
Enoyl reductase (mycobacterial)	Inhibitor	Isoniazid ⁵⁸
Squalene epoxidase (fungal)	Inhibitor	Terbinafin ⁵⁹
Δ 14 reductase (fungal)	Inhibitor	Amorolfin ⁶⁰
Xanthine oxidase	Inhibitor	Allopurinol ⁶¹
4-Hydroxyphenylpyruvate dioxygenase	Inhibitor	Nitisinone ⁶²
Ribonucleoside diphosphate reductase	Inhibitor	Hydroxycarbamide ⁶³
Transferases		
Protein kinase C	Inhibitor	Miltefosine ^{64,65}
Bacterial peptidyl transferase	Inhibitor	Chloramphenicol ⁶⁷
Catecholamine-O-methyltransferase	Inhibitor	Entacapone ⁶⁸
RNA polymerase (bacterial)	Inhibitor	Ansamycins ⁶⁹
Reverse transcriptases (viral)	Competitive inhibitors	Zidovudine ^{70,71}
	Allosteric inhibitors	Efavirenz ^{72,73}
DNA polymerases	Inhibitor	Acyclovir, suramin ^{74,75}
GABA transaminase	Inhibitor	Valproic acid ⁷⁶ , vigabatrin ⁷⁷
Tyrosine kinases	PDGFR/ABL/KIT inhibitor	Imatinib ⁷⁸
	EGFR inhibitor	Erlotinib ⁷⁹
	VEGFR2/PDGFR β /KIT/FLT3	Sunitinib ⁶⁶
	VEGFR2/PDGFR β /RAF	Sorafenib ¹⁰⁹
Glycinamide ribonucleotide formyl transferase	Inhibitor	Pemetrexed ⁵⁵
Phosphoenolpyruvate transferase (MurA, bacterial)	Inhibitor	Fosfomycin ^{80,81}
Human cytosolic branched-chain aminotransferase (hBCATc)	Inhibitor	Gabapentin ⁸²

EGFR, epidermal growth factor receptor; GABA, γ -amino butyric acid; HMG-CoA, 3-hydroxy-3-methyl-glutaryl coenzyme A; PDGFR, platelet-derived growth factor receptor; VEGFR, vascular endothelial growth factor receptor.

Table 1b | Enzymes

Type	Activity of drug	Drug examples
Hydrolases (proteases)		
Aspartyl proteases (viral)	HIV protease inhibitor	Saquinavir, indinavir ⁹⁴
Hydrolases (serine proteases)		
Unspecific	Unspecific inhibitors	Aprotinine ⁹⁵
Bacterial serine protease	Direct inhibitor	β -lactams ⁹⁶
Bacterial serine protease	Indirect inhibitor	Glycopeptides ⁹⁷
Bacterial lactamases	Direct inhibitor	Sulbactam ⁹⁸
Human antithrombin	Activator	Heparins ⁹⁹⁻¹⁰¹
Human plasminogen	Activator	Streptokinase ^{102,103}
Human coagulation factor	Activator	Factor IX complex, Factor VIII ¹⁰⁴
Human factor Xa	Inhibitor	Fondaparinux ¹⁰⁵
Hydrolases (metalloproteases)		
Human ACE	Inhibitor	Captopril ¹⁰⁶
Human HRD	Inhibitor	Cilastatin ¹⁰⁷
Human carboxypeptidase A (Zn)	Inhibitor	Penicillamine ¹⁰⁸
Human enkephalinase	Inhibitor	Racecadotril ¹¹⁰
Hydrolases (other)		
26S proteasome	Inhibitor	Bortezomib ⁸³
Esterases	AChE inhibitor	Physostigmine ⁸⁴
	AChE reactivators	Obidoxime ⁸⁵
	PDE inhibitor	Caffeine ⁸⁶
	PDE3 inhibitor	Amrinon, milrinone ⁸⁷
	PDE4 inhibitor	Papaverine ⁸⁸
	PDE5 inhibitor	Sildenafil ⁸⁹
	HDAC inhibitor	Valproic acid ⁷⁶
	HDAC3/HDAC7 inhibitor	Carbamezepine ⁹⁰
Glycosidases (viral)	α -glycosidase inhibitor	Zanamivir, oseltamivir ⁹¹
Glycosidases (human)	α -glycosidase inhibitor	Acarbose ⁹²
Lipases	Gastrointestinal lipases inhibitor	Orlistat ⁹³
Phosphatases	Calcineurin inhibitor	Cyclosporin ¹¹¹
	Inositol polyphosphate phosphatase inhibitor	Lithium ions ^{112,113}
GTPases	Rac1 inhibitor	6-Thio-GTP (azathioprine metabolite) ¹¹⁴
Phosphorylases	Bacterial C55-lipid phosphate dephosphorylase inhibitor	Bacitracin ¹¹⁵
Lyases		
DOPA decarboxylase	Inhibitor	Carbidopa ¹¹⁶
Carbonic anhydrase	Inhibitor	Acetazolamide ¹¹⁷
Histidine decarboxylase	Inhibitor	Tritoqualine ¹¹⁸
Ornithine decarboxylase	Inhibitor	Eflornithine ¹¹⁹
Soluble guanylyl cyclase	Activator	Nitric acid esters, molsidomine ¹²⁰⁻¹²³
Isomerases		
Alanine racemase	Inhibitor	D-Cycloserine ¹²⁴
DNA gyases (bacterial)	Inhibitor	Fluoroquinolones ¹²⁵
Topoisomerases	Topoisomerase I inhibitor	Irinotecan ¹²⁶
	Topoisomerase II inhibitor	Etoposide ¹²⁷
$\Delta 8,7$ isomerase (fungal)	Inhibitor	Amorolfin ¹²⁸
Ligases (also known as synthases)		
Dihydropteroate synthase	Inhibitor	Sulphonamides ¹²⁹
Thymidylate synthase (fungal and human)	Inhibitor	Fluorouracil ¹³⁰
Thymidylate synthase (human)	Inhibitor	Methotrexate, pemetrexed ^{55,131}
Phosphofructokinase	Inhibitor	Antimony compounds ¹³²
mTOR	Inhibitor	Rapamycin ¹³³
Haem polymerase (<i>Plasmodium</i>)	Inhibitor	Quinoline antimalarials ¹³⁴
1,3- β -D-glucansynthase (fungi)	Inhibitor	Caspofungin ¹³⁵
Glucosylceramide synthase	Inhibitor	Miglustat ¹³⁶

ACE, angiotensin-converting enzyme; AChE, acetylcholinesterase; HDAC, histone deacetylase; HRD, human renal dehydropeptidase; mTOR, mammalian target of rapamycin; PDE, phosphodiesterase.

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Table 2 | **Substrates, metabolites and proteins**

Substrate	Drug substance
Asparagine	Asparaginase ¹³⁷
Urate	Rasburicase (a urate oxidase) ¹³⁸
VAMP–synaptobrevin, SNAP25, Syntaxin	Light chain of the botulinum neurotoxin (Zn-endopeptidase) ¹³⁹

SNAP, synaptosomal-associated protein; VAMP, vesicle-associated membrane protein.

that tries to cover 'all' drug substances. For the present purpose, we chose to limit our analysis to published consensus data on one to three of the main biochemical targets of drug substances. If there was no consensus or proof of target and/or target–effect connection, we included the respective substances in a part of our list called 'Unknown mechanism of action'.

The dynamics of drug effects. It would ultimately be desirable to move away from static target definition, but this is hindered mainly by our inability to gauge the interaction of the aforementioned 'ripples' — in other words, the actual pharmacodynamics of drugs. All drugs somehow interfere with signal transduction, receptor signalling and biochemical equilibria. For many drugs we know, and for most we suspect, that they interact with more than one target. So, there will be simultaneous changes in several biochemical signals, and there will be feedback reactions of the pathways disturbed. In most cases, the net result will not be linearly deducible from single effects. For drug combinations, this is even more complicated. A mechanism-based simulation of pharmacodynamic drug–drug interactions was published recently⁶, highlighting the complexity of interaction analyses for biological systems. Awareness is also increasing of the nonlinear correlation of molecular interactions and clinical effects. For example, the importance of receptor–receptor interactions (receptor mosaics) was recently summarized for GPCRs, resulting in the hypothesis that cooperativity is important for the decoding of signals, including drug signals⁷. Another paper reported dopamine fluctuations after

administration of cocaine, followed by a gradual increase in steady-state dopamine concentration⁸. Indeed, the dynamics of the response are what really matters, but are difficult to assess experimentally. Further examples of dynamic (process) mechanisms of drug action include non-covalent modifications of the active centre (for example, acetylation of bacterial transpeptidases by β -lactam antibiotics); allosteric modulation (for example, benzodiazepine modulation of GABA (γ -amino butyric acid) receptors); drugs that require the receptor to be in a certain state for binding and inhibition (for example, 'trapping' of K⁺ channels by methanesulphoanilide anti-arrhythmic agents⁹); drugs that exert their effect indirectly and require a functional background (for example, the catechol-*O*-methyl transferase inhibitor entacapone, the effect of which is due to the accumulation of non-metabolized dopamine); anti-infectives that require the target organism to be in an active, growing state (for example β -lactams); molecules requiring activation (prodrugs, such as paracetamol); and cases of modifications of a substrate or cofactor (for example, asparaginase, which depletes tumour cells of asparagine; isoniazide, which is activated by mycobacteria leading to an inactive covalently modified NADH; and vancomycin, which binds to the building block bacteria use for constructing their cell wall).

The macro- and micro-world of targets.

So, for estimations of the total number of targets, a clinically relevant 'target' might consist not of a single biochemical entity, but the simultaneous interference of a number of receptors (pathways, enzymes and so on).

Only this will give a net clinical effect that might be considered beneficial. As yet, we are unable to count 'targets' in this sense ('macro-targets'), and it is only by chance that most of the current *in vitro* screening techniques will identify drugs that work through such targets.

Greater knowledge of how drugs interact with the body (mechanisms of action, drug–target interactions) has led to a reduction of established drug doses and inspired the development of newer, highly specific drug substances with a known mechanism of action. However, a preoccupation with the molecular details has sometimes resulted in a tendency to focus only on this one aspect of the drug effects. For example, cumulative evidence now suggests that the proven influence of certain psychopharmaceuticals on neurotransmitter metabolism has little to do with the treatment of schizophrenia or the effectiveness of the drug for this indication¹⁰. Here, we touch on a very basic and important point that cannot be expanded in the context of this paper but which deserves to be stressed: with all our efforts to understand the molecular basis of drug action, we must not fall into the trap of reductionism. As Roald Hoffmann aptly said in his speech at the Nobel Banquet:

"Chemistry reduced to its simplest terms, is not physics. Medicine is not chemistry knowledge of the specific physiological and eventually molecular sequence of events does not help us understand what [a] poet has to say to us."

With diseases such as type 1 diabetes, for example, the molecule insulin is indeed all that is needed to produce a cure, although we cannot imitate its regulated secretion. With diseases such as psychoses, for example, antipsychotic drugs might not correct nor even interfere with the aspect of the human constitution that is actually deranged, and with such drugs molecular determinism might be counterproductive to the use and development of therapeutic approaches. It is thought that rather than chemically providing a 'cure', these drugs make the patient more responsive to a therapy that acts at a different level. Reflections on molecular targets are very important because drugs are molecules, but it is important not to be too simplistic.

Returning to the key question, what do we count as a target? In the search for molecular reaction partners of drug substances, we will have to be content with losing sight

Box 1 | **Drugs with unknown mechanism of action**

4-Aminosalicylic acid | Alendronate | Ambroxol | Arsenic trioxide | Becaplermin | Bexarotene | Chloral hydrate | Clofazimine | Dactinomycin (RNA synthesis inhibitor) | Dapsone (folic acid synthesis inhibitor) | Diethyl carbamazepine | Diethyl ether | Diloxanide | Dinitric oxide | Ethambutol | Gentian violet | Ginkgolides | Griseofulvin | Halofantrine | Halothane | Hydrazinophthalazine | Limefantrine (antimalarial; prevents haem polymerization) | Levetiracetam | Mebendazole | Methyl-(5-amino-4-oxopentanoate) | Niclosamide | Pentamidine | Podophyllotoxin | Procarbazine | Selenium sulphide

Box 2 | One drug — many targets

Over the past 20 years, drug approval authorities and many pharmacologists have moved away from combination therapies and asked for rational, single-drug, single-target therapies. This is understandable, as it rapidly becomes challenging to analyse the contributions of multiple drugs or those that hit multiple targets to the observed effects, both desirable and undesirable. The principle that blocking a single pharmacological target with high potency is desirable because it minimizes the side effects that come with non-specific drugs has become well-established, almost dogma, in drug development circles. However, a few examples will suffice to show that it is an oversimplification. First, despite the appeal of a single-drug-target strategy for drug development, the most effective anti-arrhythmic compound, amiodarone, is the 'dirtiest' of all anti-arrhythmics³³. Second, the problems with highly selective cyclooxygenase-2-inhibitors are considered to be due to their very selectivity, which seems to tip the balance of pro- and anti-thrombotic mediators in an unfavourable way³⁴. Third, propranolol is the first and classic β -sympatholytic agent, but it has neither an absolute selectivity for an adrenoceptor subtype nor does it address receptors exclusively; for example, it also inhibits phosphatidic acid phosphorylase. It is not clear whether the latter activity contributes to the net clinical effects (hypotension and so on)³⁵. Fourth, oestrogens not only have an intracellular nuclear receptor, but also activate a membrane-bound one as well (GPR30)³⁶. The effects of oestrogen result from the interplay of the two mechanisms. Fifth, for papaverine, a smooth-muscle relaxant agent, the following activities were recorded, and all seem to be important for the net effect: cyclic nucleotide phosphodiesterase inhibition, Ca^{2+} -channel blockade and α -adrenoreceptor antagonism³⁷. And last, the anticancer drug imatinib was originally moved into clinical development on the basis of its capacity to inhibit a single target: the BCR-ABL kinase. It has since become clear that its success could be linked to interaction with at least two other targets; indeed, two anticancer drugs, sorafenib and sunitinib, that were developed to inhibit multiple kinases have recently been approved. As with antipsychotics³⁸, such 'dirty' or 'promiscuous' anticancer drugs might be increasingly sought in the near future³⁸.

of some of the net biochemical and especially clinical effects of the drug's action. A target definition derived from the net effect rather than the direct chemical interaction will require input from systems biology, a nascent research field that promises to significantly affect the drug discovery process¹¹. At the other end of the scale of precision, we can define some targets very precisely on the molecular level: for example, we can say that dihydropyridines block the $\text{Ca}_v1.2$ splicing variant in heart muscle cells of L-type high-voltage activated calcium channels. This is an example of a 'micro-target'. It does make sense to define it because a subtype or even splicing variant selectivity could alter the effectiveness of calcium channel blockers. We could further differentiate between genetic, transcriptional, post-transcriptional or age differences between individuals, and again this will make sense in some cases. But for a target count, a line needs to be drawn somewhere, otherwise the number of individual patients that receive a drug could be counted and equated with the number of known targets. In summary, we will count neither macro- nor micro-targets, but something in between — admittedly a somewhat arbitrary distinction.

Classification of current drugs

There are a number of possible ways to classify drug substances (active pharmaceutical ingredients). From the end of the

nine-teenth century until the 1970s, drug substances were classified in the same way as other chemical entities: by the nature of their primary elements, functional moieties or organic substance class. Recently, the idea of classifying drug substances strictly according to their chemical constitution or structure has been revived. Numerous databases now attempt to gather and organize information on existing or potential drug substances according to their chemical structure and diversity. The objective is to create substance 'libraries' that contain pertinent information about possible ligands for new targets (for example, an enzyme or receptor) of clinical interest^{12,13} and, more importantly, to understand the systematics of molecular recognition^{14,15} (ligand-receptor).

“ In situations in which the dynamic actions of the drug substance stimulate, or inhibit, a biological process, it is necessary to move away from the descriptions of single proteins, receptors and so on and to view the entire signal chain as the target. ”

At present, the most commonly used classification system for drug substances is the ATC system¹⁶ (see WHO Collaborating Centre for Drug Statistics Methodology, Further information). It categorizes drug substances at different levels: anatomy, therapeutic properties and chemical properties. We recently proposed an alternative classification system¹⁷, although we did not follow it fully in the arrangement of entries in TABLES 1–8, BOX 1, as explained below.

Classification of drug substances according to targets. In TABLES 1–8, we arranged drug substances according to their mechanism of action. Although the term 'mechanism of action' itself implies a classification according to the dynamics of drug substance effects at the molecular level, the dynamics of these interactions are only speculative models at present, and so mechanism of action can currently only be used to describe static (micro)targets, as discussed above.

The actual depth of detail used to define the target is primarily dependent on the amount of knowledge available about the target and its interactions with a drug. If the target structure has already been determined, it could still be that the molecular effect of the drug cannot be fully described by the interactions with one target protein alone. For example, anti-bacterial oxazolidinones interact with 23S-rRNA, tRNA and two polypeptides, ultimately leading to inhibition of protein synthesis. In this case, a description of the mechanism of action that only includes interactions with the 23S-rRNA target would be too narrowly defined. In particular, in situations in which the dynamic actions of the drug substance stimulate, or inhibit, a biological process, it is necessary to move away from the descriptions of single proteins, receptors and so on and to view the entire signal chain as the target. Indeed, it has been pointed out by Swinney in an article on this topic that "two components are important to the mechanism of action ... The first component is the initial mass-action-dependent interaction ... The second component requires a coupled biochemical event to create a transition away from mass-action equilibrium" and "drug mechanisms that create transitions to a non-equilibrium state will be more efficient"¹⁸. This consideration again stresses that dynamics are essential for effective drug action and, as discussed above, indicates that an effective drug target comprises a biochemical system rather than a single molecule.

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