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Affixes are essential tools when teaching and studying pharmacological drug classes

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Abstract

Introduction: The study of pharmacology involves considerable memorisation of drug classes, individual drug names, and mechanisms of action, as well as other characteristics including side effects, drug interactions, and therapeutic uses. The voluminous memorisation involved in basic and clinical pharmacology represents a challenging task, especially considering the continuous rate at which new drugs enter the market, and given that generic and trade names often are used interchangeably.

Description of Educational Tool (Pharmacology Guide): Our comprehensive table consists of common affixes and roots of generic drugs that we have found beneficial to the study and retention of pharmacology. Our table includes important aspects of over fifty different drug classes including prototypes, mechanisms of action, and pertinent references.

Evaluation: Qualitative feedback indicates that students readily adopt this organised approach of using common affixes and roots when studying pharmacological drug classes.

Future Plans: Pharmacology educators and students at various institutions worldwide should benefit from this organised approach for teaching and learning pharmacological information. As well, it is anticipated that new editions of this table can be readily adapted to parallel the continuous emergence of new drugs and drug classes.

Keywords: *Affixes, health professions students, pharmacology study guide, pharmacy educators*

Introduction

Studying pharmacology inevitably results in substantial memorisation of drug classes or families, individual drug names, mechanisms of action, and other important pharmacological identifiers, such as side effects and therapeutic uses. Memorisation of drug names alone represents a daunting task, given that generic and trade names often are used interchangeably within drug classes, and given that the mnemonic and visual associations of generic and trade names are enigmatic. The sources of information when learning pharmacology include college or continuing education courses, common pharmacology texts used in pharmacy, medical, and nursing programmes (*e.g.* Brunton *et al*., 2011; Katzung *et al*., 2012), and guides for preparing for qualifying, board, or licensing exams. Using generic name affixes or roots as a study tool appears to be an effective means of reducing the amount of tedious memorisation when studying pharmacology. Furthermore, as students transition into health care professionals, the rapid and continuous advent of new drugs entering the market can create a challenge with remaining up-to-date on current drugs and drug classes. Our aim was to develop a comprehensive table to be used as a learning tool in pharmacology courses by both educators and students at pharmacy, medical, and other health sciences programmes.

Description of Educational Tool (Pharmacology Guide)

Herein, we present a comprehensive table of common affixes and roots found in generic drug names that should enhance the delivery and study of pharmacology (Table I). The impetus for development of this table derives from numerous pharmacology courses taught to a variety of different audiences, including undergraduate and graduate pharmacy and nursing students, where the use of affixes and roots has been a consistently successful strategy when studying pharmacology. An extensive search of the literature revealed that there is no published information on our strategy, and although very few internet websites contained partial lists of affixes or roots, none represented a comprehensive yet simplistic approach that could be utilised as an effective study guide to a wide variety of students and healthcare professionals; we believe that our table has the potential to become an important study tool and reference guide when studying pharmacology both clinically and in the classroom. Within the table (Table I), columns convey information in the following left-to-right order: 1) common affix or root; 2) drug class; 3) drug class prototype; 4) notable drug exceptions to the affix or root applied to each drug class; 5) a succinct but detailed mechanism of action of the drug class; and 6) a recent and relevant reference on the drug class and mechanism.

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In some cases, several affixes could have been applied to a particular drug class (*e.g.* "olol" vs. "lol" as suffixes for the beta blockers); we chose affixes that we conventionally use as study tools in the pharmacology classroom. Prototypes represent the typical and most commonly referred example of a particular drug class. Knowledge of a prototype facilitates comparisons and contrasts to new drug class members as they enter the market. There are some cases where our prototype selection is purposefully limited to simplify the table. For example, there are over twenty different monoclonal antibody drugs that are identified by the suffix "mab" on the market, many with unique mechanisms of action. To remain consistent with our concise yet detailed format, we chose abciximab and adalimumab as our generic drug representatives.

In our table, notable exceptions are drugs within a particular drug class that do not have the given affix or root in their generic name. Here again, we chose selected examples to be concise in our presentation. We recognise that, for some of the drug classes, there are other examples of drugs not listed that could be considered notable exceptions for various reasons. On occasion, there are generic drugs that contain the given affix or root of a particular drug class, yet nonetheless are not considered members. For example, the chemotherapy drug bleomycin would be considered a drug that contains the commonly used suffix "mycin" that identifies aminoglycosides and macrolides; however, bleomycin is not a member of either antibiotic drug class. As another example, the dopamine agonist fenoldopam would be considered a drug that contains the commonly used suffix "pam" that identifies benzodiazepines; however, fenoldopam clearly is not a member of this sedative/hypnotic drug class.

Table I: Rupprecht & Rhodes (R&R) Pharmacology Guide – 2014 Edition

Affix or Root	Drug Class	Prototype	Notable Exception(s)	Mechanism of Action	Reference
-aban	Oral Direct Factor Xa Inhibitors	Rivaroxaban		Inhibits factor Xa (↓ conversion of prothrombin to thrombin)	(Perzborn et al., 2010)
-afil	Phosphodiesterase Type 5 Inhibitors	Sildenafil		Inhibits phosphodiesterase type 5 activity which increases [cGMP] in the corpus cavernosum	(Rosen and Kostis, 2003)
-al	Barbiturates	Phenobarbital	Primidone	Increases the duration of Cl^- channel opening at the $GABA_A$ receptor; enhances membrane hyperpolarisation	(Loscher and Rogawski, 2012)
-am; -pam	Benzodiazepines	Diazepam	Chlordiazepoxide; Clorazepate	Increases the frequency of Cl^- channel opening at the $GABA_A$ receptor; enhances membrane hyperpolarisation	(Campo-Soria et al., 2006)
-ane	Volatile Liquids	Halothane	Nitrous Oxide	Possible expansion of lipid membrane and interactions with various ligand-gated ion channels	(Franks, 2008)
-flurane		Isoflurane			
-ase	Fibrinolytics	Alteplase		Enhances conversion of plasminogen to plasmin (degrades fibrin clots)	(Weitz et al., 1999)
-caine	Local Anaesthetics	Lidocaine (Amide)		Blockade of axonal voltage-gated Na^+ channels	(Butterworth and Strichartz, 1990)
		Procaine (Ester)			
cef-	Cephalosporins	Cefazolin (IV)		Inhibits bacterial mucopeptide layer formation (cell wall); enhances murein hydrolase activity (cell wall)	(Biek et al., 2010)
ceph-		Cephalexin (oral)			
-chol; -choline	Choline Esters (Direct-Acting Cholinergics)	Acetylcholine	Cevimeline; Pilocarpine	Muscarinic receptor agonist	(Wess et al., 2007)
-cillin	Penicillins	Penicillin G		Inhibits bacterial mucopeptide layer formation (cell wall); enhances murein hydrolase activity (cell wall)	(Yocum et al., 1980)
-curium	Nondepolarising Neuromuscular Blockers (Isoquinoline)	Atracurium	Metocurine; Tubocurarine	Muscle-type nicotinic acetylcholine receptor (N_m) antagonist at the neuromuscular junction	(Appiah-Ankam and Hunter, 2004)
-curonium	Nondepolarising Neuromuscular Blockers (Steroid)	Pancuronium		N_m antagonist at the neuromuscular junction	(Appiah-Ankam and Hunter, 2004)
-cycline	Tetracyclines	Tetracycline		Inhibition of bacterial protein synthesis (30S ribosomal subunit)	(Chopra and Roberts, 2001)

-dazole	Nitroimidazoles	Metronidazole		The nitro group is chemically reduced in anaerobic bacteria and protozoans; these reactive and unstable reduction products disrupt the electron transport chain	(Edwards, 1993)
-dipine	Dihydropyrimidines (Calcium Channel Blockers)	Nifedipine		L-type Ca^{2+} channel (α_1 subunit) antagonist in arteriolar vascular beds	(Opie, 1997)
-done / **-pine**	Atypical Antipsychotics	Risperidone / Clozapine	Aripiprazole	Varying degrees of primarily dopamine (D_2) and serotonin (5-HT) receptor antagonism	(Seeman, 2002)
-dronate	Bisphosphonates	Alendronate	Zolendronic Acid	Reduces bone degradation by inhibiting osteoclast activity and may increase bone density by stimulating osteoblasts	(Drake et al., 2008)
-fenacin	Muscarinic (M_3) Antagonists	Solifenacin	Oxybutynin; Tolterodine	Muscarinic (M_3) acetylcholine receptor antagonist in the urothelium	(Iijima et al., 2007)
fent	Potent Synthetic Opioids	Fentanyl		μ, δ, and κ receptor agonist (to varying degrees)	(Bovill, 1987)
-floxacin	Quinolones and Fluoroquinolones	Ciprofloxacin	Nalidixic Acid	DNA gyrase inhibitor (inhibits bacterial DNA synthesis)	(Collin et al., 2011)
-glitazone	Thiazolidinediones (Glitazones)	Pioglitazone		Increases insulin sensitivity by acting on adipose tissue, skeletal muscle, and hepatocytes to increase glucose utilisation and decrease glucose production via peroxisome proliferator-activated receptors (PPARs) – PPAR-γ and/or PPAR-α.	(Smith, 2001)
-thiazide	Thiazide Diuretics	Chlorothiazide	Chlorthalidone; Indapamide; Metolazone	Blockade of Na^+/Cl^- and $Na^+/K^+/2Cl^-$ cotransporters in the distal convoluted tubule	(van Zwieten, 1992)
-irudin	Direct Thrombin Inhibitors	Hirudin	Argatroban; Dabigatran	Thrombin active site antagonist and fibrinogen binding site (on thrombin molecule) antagonist (Argatroban and Dabigatran only bind to thrombin active site); nitric oxide release (Argatroban only)	(Fareed et al., 2006)
-lol	Beta Antagonists (Beta Blockers)	Propranolol	Butoxamine	β_1 and/or β_2 receptor antagonist	(Mason et al., 2009)
-mab	Monoclonal Antibodies	Abciximab		Binds to the integrin GPIIb/IIIa receptor (activated platelets) and inhibits fibrinogen and von Willebrand factor from binding to activated platelets	(Nurden et al., 2004)
		Adalimumab		Binds to and inhibits tumor necrosis factor-alpha (TNF-α) to ↓ inflammation	(Vena and Cassano, 2007)
-micin; -mycin	Aminoglycosides and Macrolides	Gentamicin (Aminoglycoside) / Erythromycin (Macrolide)		Inhibition of bacterial protein synthesis: 30S ribosomal subunit (Aminoglycosides) or 50S ribosomal subunit (Macrolides)	(Davis, 1987); (Kanoh and Rubin, 2010)
-mustine	Nitrosoureas (Alkylating Antineoplastic Agents)	Carmustine	Streptozocin	Forms DNA cross-links which results in the inhibition of DNA synthesis and function	(Schallreuter et al., 1990)
nal	Opioid Antagonists	Naloxone	Alvimopan	μ, δ, and κ receptor antagonist (to varying degrees)	(Bowdle, 1998)
-nazole	Azole Antifungals	Ketoconazole	Clotrimazole	Reduction of ergosterol synthesis by inhibiting fungal cytochrome P450 enzymes	(Ghannoum and Rice, 1999)
-parin	Indirect Thrombin Inhibitors	Heparin	Danaparoid	Increases the activity of antithrombin and decreases the production of thrombin (↓ fibrin production)	(Hirsh et al., 2001)
-phylline	Methylxanthines	Theophylline	Caffeine; Theobromine; Xanthine	Nonselective phosphodiesterase inhibition; adenosine receptor antagonist; possible histone deacetylation enhancement	(Ito et al., 2002)

-pramine **-triptyline**	Tricyclic Antidepressants	Imipramine Nortriptyline	Amoxapine; Doxepin	Inhibition of norepinephrine (primarily) and serotonin reuptake	(Feighner, 1999)
-prazole	Proton Pump Inhibitors	Omeprazole		Blockade of proton pumps (H^+/K^+ ATPase) in parietal cells	(Der, 2003)
pred **-sone**	Corticosteroids	Prednisone Cortisone		Phospholipase A_2 inhibitor	(Lee et al., 1998)
-pril	Angiotensin-Converting Enzyme (ACE) Inhibitors	Captopril		Inhibits angiotensin-converting enzyme (↓ Angiotensin II)	(Brown and Vaughan, 1998)
-profen	Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)	Ibuprofen		Inhibits cyclooxygenase-1 (COX-1) and COX-2 leading to decreased levels of prostaglandins, thromboxanes, and other inflammatory mediators	(Burian and Geisslinger, 2005)
-rubicin	Anthracycline Antibiotics	Doxorubicin	Mitoxantrone	Forms oxygen free radicals that bind to DNA causing single- and double-strand DNA breaks; inhibits topoisomerase II; intercalates into DNA	(Gewirtz, 1999)
-sartan	Angiotensin II Antagonists	Losartan		Angiotensin II receptor (AT_1) antagonist	(Burnier, 2001)
-setron	Serotonin (5-HT_3) Antagonists	Ondansetron		Serotonin (5-HT_3) receptor antagonist in the chemoreceptor trigger zone, vomiting centre, and gastrointestinal tract	(Gan, 2005)
-sin	Alpha Antagonists (Alpha Blockers)	Prazosin	Phenoxybenzamine; Phentolamine; Yohimbine	α_1 and/or α_2 receptor antagonist	(Nash, 1990)
-statin	HMG-CoA (3-hydroxy-3-methylglutaryl–coenzyme A) Reductase Inhibitors (Statins)	Atorvastatin	Advicor and Vytorin (combination preparations)	Inhibits HMG-CoA reductase (↓ cholesterol synthesis)	(Istvan and Deisenhofer, 2001)
-stigmine	Acetylcholinesterase Inhibitors (Indirect-Acting Cholinergics)	Physostigmine	Donepezil; Edrophonium; Galantamine; Tacrine	Inhibits acetylcholinesterase	(Wilkinson et al., 2004)
sulfa-	Sulfonamides	Sulfamethoxazole		Inhibits bacterial DNA synthesis by inhibiting folate production (dihydropteroate synthase inhibitor)	(Skold, 2000)
-taxel **vin-**	Mitotic Spindle Poisons	Paclitaxel (Taxane) Vincristine (Vinca Alkaloid)		Disrupts microtubule formation/ polymerisation during mitosis	(Weaver and Cleveland, 2005)
-terol	Selective Beta$_2$ Agonists	Albuterol	Metaproterenol; Terbutaline	Stimulates β_2 receptors in bronchial smooth muscle	(Shore and Drazen, 2003)
-tidine	Histamine (H_2) Antagonists	Cimetidine		Histamine (H_2) receptor antagonist in parietal cells	(Giraldo et al., 1992)
-tinib	Tyrosine Kinase Inhibitors	Imatinib	Pazopanib; Sorafenib; Vandetanib	Binds to and interferes with protein kinase signaling pathways implicated in gene transcription, DNA synthesis, and cellular growth; inhibits proliferation and induces apoptosis	(Gotink and Verheul, 2010)
-triptan	Triptans	Sumatriptan		Stimulates serotonin (5-$HT_{1D/1B}$) receptors (presynaptic trigeminal nerve endings) which may inhibit the release of vasodilators and may cause vasoconstriction (decreasing activation of pain receptors)	(Tepper et al., 2002)
trop	Cholinergic Antagonists (Anticholinergics)	Atropine	Glycopyrrolate; Scopolamine	Competitive antagonist at acetylcholine receptors (muscarinic subtypes)	(Nair and Hunter, 2004)
-vir	Antivirals	Acyclovir	Foscarnet; Zidovudine	Inhibits one or more of the following: viral attachment and entry; viral penetration; viral uncoating; viral nucleic acid synthesis; viral protein synthesis and processing; viral release	(De Clercq, 2004)

Evaluation

As mentioned previously, the development of this formalised and comprehensive table derives from over eighteen years of the informal and consistent use of affixes and roots as a successful strategy for teaching and learning pharmacology in a variety of courses (*e.g*. Basic Pharmacology, General Pharmacology, and Advanced Pharmacology), in a variety of formats (*e.g.* lecture-based instruction and online instruction), and to a variety of audiences (*e.g.* undergraduate and graduate pharmacy, medical, and nursing students.)

During the first few weeks of pharmacology courses, after basic principles have been established, students were often surprised by the numerous examples of common affixes/roots in generic names within drug classes. For the remaining weeks of the course, students often would actively seek the common affixes/roots within drug classes with the hope of making recall and learning much easier during exam preparation. Many students adopted this strategy as a way of simplifying the long list of drugs that would inevitably result from studying topics in pharmacology, thus easing their memorisation burden, and increasing their confidence when approaching examinations.

Qualitative feedback from end-of-semester course evaluations from the last five years, as well as verbal and written communications during and after the courses, overwhelmingly indicates that students readily adopt this organised approach of using common affixes and roots when studying pharmacological drug classes. Comments such as "material in this pharmacology course was presented in a structural way that made it easier to understand and comprehend, and I really liked using common prefixes and suffixes to make the task of studying less intimidating," and "the use of roots and affixes made it easier to compartmentalise the drugs to be learned, making preparation for exams more efficient and less daunting," are common assessments following course offerings. While no negative comments regarding our approach have been overtly stated by students in the course assessments, a few students have indicated that they were more comfortable using other methods of learning pharmacological information, such as via concept mapping and studying with flashcards. A recent communication from a former student summarises the potential value of our approach of using affixes and roots when studying pharmacology: "I was able to develop long-term retention of valuable information and the ability to easily recall pharmacological material from the use of affixes and roots to learn generic drug names. As a novice in pharmacology, this method allowed me to feel more comfortable and confident with the inordinate number of commonly used drugs within the medical community. I believe that the affix/root method can systematically introduce beginners to various drug classes and even solidify the working knowledge of more clinically experienced students. Overall, this technique provided me with a strong pharmacological foundation that I can carry with me throughout my educational and professional career."

Future Plans

To our knowledge, this is the first time that a comprehensive collection of drug class affixes and roots has been combined with other pertinent pharmacological information regarding the given drug class. We envision that pharmacy students, as well as undergraduate and graduate medical and nursing students, will find utility in this table. Furthermore, we believe that pharmacology educators from around the world will find this table to be a useful and beneficial teaching resource and reference guide. The organisation of affixes and roots of the various drug classes in table form also should facilitate the development of new editions of this table, to parallel the continuous emergence of new drugs and drug classes. With a formalised and comprehensive table now developed, future qualitative and quantitative assessments for developing this strategy in pharmacology courses should be more feasible.

We believe that affixes and roots are important tools when studying pharmacology, and we believe that our table represents a unique strategy that can be employed when learning pharmacology in various contexts, whether in the classroom, via the internet, in preparation for national board exams, qualifying or licensing exams, or continuing education experiences.

Declaration of interest

The authors report no conflict of interest. The authors alone are responsible for the content and development of this article.

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