Antihistamine mechanism of action pdf

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$$C-O-(CH_2)-N(CH_3)$$
 $H$ 
Diphenylhydramin

 $C-C-(CH_2)-N(CH_3)$ 
 $H$ 

Chlorpheniramin

 $S-C-(CH_2)-N(CH_3)$ 
 $C-CH_2-CH-N(CH_3)$ 
 $C-CH_3$ 
Promethazine

## H1 antihistamine drugs. Alkylamines (2)



## Dexchlorpheniramine

Dexchlorpheniramine, d(+)-3-(p-chlorophenyl)-3-(2-pyridyl) propyldimethylamine, is synthesized by separating the racemate obtained from the synthesis of chlorpheniramine using D-phenylsuccinic acid.

Activity of this drug is approximately twice that of chlorpheniramine. Dexchlorpheniramine is also used for allergy symptoms, rhinitis, and dermatitis. A synonym of this drug is polaramin.

## Brompheniramine

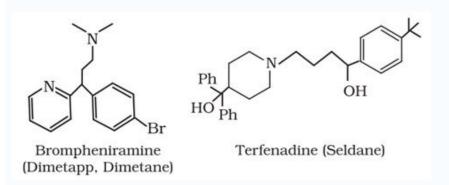
Brompheniramine, 3-(p-bromophenyl)-3-(2-pyridyl)propyldimethylamine, is an analog of chlorpheniramine. The only difference is that the chlorine atom in the benzene ring is replaced with a bromine atom. It is also synthesized in an analogous manner.

Brompheniramine is also used for allergy symptoms, rhinitis, and dermatitis. Its activity is approximately the same as that of chlorpheniramine. Synonyms of this drug are dimetane, brombey, spentan, veltane, and others.

## Antihistamines: Mechanism of Action

- The binding of H<sub>1</sub> blockers to the histamine receptors prevents the adverse consequences of histamine stimulation:
  - Vasodilation
  - Increased gastrointestinal and respiratory secretions
  - Increased capillary permeability





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The primary mechanism of antihistamine action in the treatment of allergic diseases is believed to be competitive antagonism of histamine binding to cellular receptors), which are present on nerve endings, smooth muscles, and glandular cells. This notion is supported by the fact that structurally unrelated drugs antagonize the H1-receptor and provide clinical benefit. However, H1-receptor antagonism may not be their sole mechanism of action in treating allergic rhinitis. On the basis of in vitro and animal experiments, drugs classified as H1-receptor antagonists have long been recognized to have additional pharmacological properties. Most first-generation H1-antihistamines have anticholinergic, sedative, local anaesthetic, and anti-5-HT effects, which might favourably affect the symptoms of the allergic response but also contribute to side-effects. These additional properties are not uniformly distributed among drugs classified as H1-receptor antagonists. Azatadine, for example, inhibits in vitro IgE-

mediated histamine and leukotriene (LT) release from mast cells and basophils. In human challenge models, terfenadine, azatadine, and loratadine reduce IgE-mediated histamine release. Cetirizine reduces eosinophilic infiltration at the site of antigen challenge in the skin, but not the nose. In a nasal antigen challenge model, cetirizine pretreatment did not affect the levels of histamine and prostaglandin D2 recovered in postchallenge lavages, whereas the levels of albumin, N-tosyl-L-arginine methyl ester (TAME) esterase activity, and LTs were reduced. Terfenadine, cetirizine, and lorated in postchallenge lavages, whereas the levels of albumin, N-tosyl-L-arginine methyl ester (TAME) esterase activity, and LTs were reduced. Terfenadine, cetirizine, and lorated in postchallenge lavages, whereas the levels of albumin, N-tosyl-L-arginine methyl ester (TAME) esterase activity, and LTs were reduced. Terfenadine, cetirizine, and lorated in postchallenge lavages, whereas the levels of albumin, N-tosyl-L-arginine methyl esterase activity, and LTs were reduced. Terfenadine, cetirizine, and lorated in postchallenge lavages, whereas the levels of albumin, N-tosyl-L-arginine methyl esterase activity, and LTs were reduced. pathophysiology of allergy, a number of H1 antagonists with additional properties are currently under development for allergic response. In animal models, mizolastine inhibits antigen-induced eosinophil infiltration into mouse skin and into the nasal cavity of guinea-pigs. Mizolastine also significantly inhibits antiquen-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. Mizolastine also significantly inhibits arachidonic acid-induced neutrophil infiltration into the bronchoalveolar lavage fluids of guinea-pigs. man, mizolastine inhibits early and late antigen-induced soluble intercellular adhesion molecule 1 (ICAM-1) levels in skin blisters. It also inhibits early and LTB4 release from mouse bone-marrow-derived mast cells, LTC4 release from rat intestinal mast cells, and 5-lipoxygenase activity of polymorphonuclear neutrophils of guinea-pig intestines and rat basophilic leukaemia cells. It is clear that a number of H1-antihistamines have multiple effects on the allergic inflammatory response. It is equally clear that these antiallergic effects on the allergic inflammatory response. It is equally clear that a number of these results and research regarding the parts of the molecules responsible for these activities are underway. Antihistamine receptors and H-2 receptors and H-2 receptors and H-2 receptors. Antihistamine drugs that bind to H-1 receptors are generally used to treat allergies and allergic rhinitis. Drugs that bind to H-2 receptors can treat upper gastrointestinal conditions, activity, adverse events, and other vital elements of antihistamine therapy in the clinical setting as relates to the essential points needed by members of an interprofessional team managing the care of patients with conditions that respond to histamine receptor blockers. Identify the approved and off-label indications for the different histamine receptor blockers. Describe the adverse event profile and contraindications of the members of the antihistamine class. Outline interprofessional team strategies for improving care coordination and communication to advance appropriate clinical outcomes with antihistamine therapy to treat indicated conditions, leading to optimal patient outcomes. Access free multiple choice questions on this topic. Antihistamines are a pharmaceutical class of drugs that act to treat allergies and allergic rhinitis. Drugs that bind to H-2 receptors treat upper gastrointestinal conditions that are caused by excessive stomach acid.[1]H-1 antihistamines are further classified according to first and second-generation H-1 antihistamines do not. The firstgeneration drugs will bind to both central and peripheral histamine-1 receptors, whereas second-generation drugs selectively bind to peripheral histamine-1 receptors; this leads to different therapeutic and side effect profiles.[2] FDA-approved Indications H-1 Antihistamines [3] Allergic rhinitis Allergic conjunctivitis Allergic dermatological reaction(s)SinusitisUrticariaAngioedemaAtopic dermatitisBronchitisMotion sicknessNauseaVomiting H-2 Antihistamines include insomnia, and for H-2, antihistamines include indigestion. Use of dual H-1 and H-2 antihistamines has been used for refractory urticaria that fails therapy with an H-1 antihistamine. There are two other classes of histamine receptors: H-3 and H-4. While compounds exist that bind them, there is no specific clinical benefit to clinicians using those compounds in humans. Example Drugs H-1 Antihistamines DiphenhydramineCetirizineChlorpheniramineCyclizineDimenhydrinate (incorporates diphenhydramine and a stimulant compound)DoxylamineHydroxyzineMeclizine H-2 Antihistamine CyclizineDimenhydrinate (incorporates diphenhydramineCyclizineDimenhydrinate)DoxylamineHydroxyzineMeclizineDimenhydrinate (incorporates diphenhydramineCyclizineDimenhydrinate)DoxylamineHydroxyzineMeclizine H-2 AntihistamineCyclizineDimenhydrinate (incorporates diphenhydramineCyclizineDimenhydrinate)DoxylamineHydroxyzineMeclizineDimenhydrinate (incorporates diphenhydramineCyclizineDimenhydrinate)DoxylamineHydroxyzineMeclizineDimenhydrinate (incorporates diphenhydrinate)DoxylamineHydroxyzineMeclizineDimenhydrinate (incorporates diphenhydrinates diphenh USA) Histamine (an endogenous chemical messenger) induces an increased level of vascular permeability, which leads to fluid moving from capillaries into the surrounding tissues. The overall outcome of this is increased swelling and dilation of vessels. Antihistamines stop this effect by acting as antagonists at the H-1 receptors. The clinical benefit is a reduction in allergy symptoms and any related symptoms. [5] First-generation antihistamines easily cross the blood-brain barrier into the central nervous system and antagonize H-1 receptors, leading to a different therapeutic and adverse effect profile in contrast to second-generation antihistamines selectively bind to peripheral histamine receptors. The duration of the pharmacological action of first-generation antihistamines is about 4 to 6 hours. In contrast, second-generation antihistamines work for 12 to 24 hours. They are both metabolized by the liver using the P450 cytochrome system. Parietal cells in the gastrointestinal tract secrete hydrochloric acid. They undergo regulation by acetylcholine, gastrin, and also histamine is released from enterochromaffin-like (ECL) cells. When histamine binds to the H-2 receptors on parietal cells, cyclic adenosine monophosphate (cAMP) increases, inducing protein kinase A. This action then leads to phosphorylation of the proteins that take part in the transport of hydrogen ions. Thus increased histamine leads to increased stomach acid, e.g., HCl secretion. [6]The use of antihistamines specific to the H-2 receptor blocks the entire process and reduces stomach acid secretion. Antihistamine medications are also possible, reserved chiefly for in-patient usage for the treatment of an allergic reaction or for treating a dystonic reaction of an antipsychotic medications. Antihistamine medications carry a broad range of adverse effects depending on the specific class of drugs utilized. H-1 receptor antihistamines will generally cause clinically noticeable adverse effects that are dose-dependent. These side effects are far more commonly seen in first-generation antihistamines do not easily cross the blood-brain barrier, and therefore their side effects profile is far more limited. In contrast to H-1 receptor antihistamines have anticholinergic properties, which are adverse effects except for cimetidine. H-1 receptor antihistamines have anticholinergic properties, which are adverse effects except for cimetidine. H-1 receptor antihistamines have anticholinergic properties, which are adverse effects except for cimetidine. may cause insomnia in some users. Due to their anticholinergic properties, dry mouth is a relatively common adverse effect. Some users experience dizziness and tinnitus. At increasing doses, euphoria and decreased coordination may also occur, and delirium is a potential adverse effect at even higher dose ranges.[7] Antihistamines may also be

cardiotoxic in some users as they have QTc-prolonging effects. [8] H-2 receptor antihistamines are generally well tolerated by users but do carry the risk of uncommon side effects. Gastrointestinal changes can be seen, including both diarrhea and constipation. Reports exist of fatigue, dizziness, and confusion. One specific drug in this category that may cause a range of adverse effects is cimetidine. Its antiandrogenic effects correlate with the possible occurrence of gynecomastia in men. In women, it can cause galactorrhea. Other H-2 receptor antihistamines do not exhibit the same properties as cimetidine. [9] Ranitidine was previously removed from the market in the United States due to concerns of potential contamination with a carcinogen. H-2 receptor antihistamines can cause inhibition of the cytochrome system, especially cimetidine, thereby leading to drug toxicity and interactions with other medications. Patients who present with hemodynamic alterations, increased intraocular pressure or increased urinary retention should use antihistamines with caution as these conditions can become exacerbated. Given the potential cardiotoxic effects of certain antihistamines, they are relatively contraindicated in any patient with QTc prolongation. Patients using other QTc-prolonging drugs require careful monitoring for further prolongation of the QTc interval due to the risk of potentially fatal cardiac arrhythmias.[8] Usage in pregnant women is a relative contraindication. Additionally, women who are lactating should use antihistamines with caution. Hypertension, cardiovascular disease, urinary retention, increased ocular pressure are relative contraindications to the use of antihistamines. Doses of the antihistamines may be monitored on an electrocardiogram (ECG) to assess prolongation of the QTc interval. There is no specific antihistamine are a class of medications that can be subdivided into H-1 and H-2 categories. H-1 antihistamines, which can be further divided into first and second generations, are primarily used to treat allergic symptoms and illnesses mediated through similar mechanisms. H-2 antihistamines can lower excessive stomach acid and thereby treat acid reflux, gastritis, and gastrointestinal ulcers. Pharmacists serve as dispensers and educators of these medications, given the availability of antihistamines at pharmacies for off-the-shelf purchase. They have a crucial role in advising the patient to utilize the correct dose and be cautious of any contraindications and adverse effects. Nurses should be prepared to answer questions regarding these medications, as well as offer counsel on adverse effects and note their therapeutic effectiveness, and report any findings to the clinician. Providers that recommend antihistamines, such as nurse practitioners, physician assistants, and physician assistants, and physician assistants, and report any findings to the clinician. pharmacist can review the patient's medication profile to determine if there are any clinically significant drug interactions, especially in elderly patients. [Level 5]Review Questions1.Monczor F, Fernandez N. Current Knowledge and Perspectives on Histamine H1 and H2 Receptor Pharmacology: Functional Selectivity, Receptor Crosstalk, and Repositioning of Classic Histaminergic Ligands. Mol Pharmacol. 2016 Nov;90(5):640-648. [PubMed: 27625037]2. Schaefer TS, Zito PM. StatPearls Publishing; Treasure Island (FL): May 8, 2022. Antiemetic Histamine H1 Receptor Blockers. [PubMed: 30422595]3. Curto-Barredo L, Giménez-Arnau AM. Treatment of chronic spontaneous urticaria with an inadequate response to H1-antihistamine. G Ital Dermatol Venereol. 2019 Aug;154(4):444-456. [PubMed: 30717573]4.Kuna L, Jakab J, Smolic R, Raguz-Lucic N, Vcev A, Smolic M. Peptic Ulcer Disease: A Brief Review of Conventional Therapy and Herbal Treatment Options. J Clin Med. 2019 Feb 03;8(2) [PMC free article: PMC6406303] [PubMed: 30717467]5.Pirahanchi Y, Sharma S. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jul 26, 2021. Physiology, Bradykinin. [PubMed: 30725872]6.Heda R, Toro F, Tombazzi CR. StatPearls Publishing; Treasure Island (FL): May 8, 2022. Physiology, Pepsin. [PubMed: 30725690]7.Boley SP, Olives TD, Bangh SA, Fahrner S, Cole JB. Physostigmine is superior to non-antidote therapy in the management of antimuscarinic delirium: a prospective study from a regional poison center. Clin Toxicol (Phila). 2019 Jan;57(1):50-55. [PubMed: 29956570]8. Farzam K, Tivakaran VS. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): May 15, 2022. QT Prolonging Drugs. [PubMed: 30521285]9.Bowman JD, Kim H, Bustamante JJ. Drug-induced gynecomastia. Pharmacotherapy. 2012 Dec;32(12):1123-40. [PubMed: 23165798]

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