**SHAMBHUNATH INSTITUTE OF PHARMACY**

 **1st Sessional Examination (2019-20)**

 **B. Pharm. 3rd year (5th Sem.)**

 **SUBJECT- PHARMACOLOGY-II (BP-503 T)**

**Time: - 1.30 hrs. Max. Marks: 30**

**Paper Code: BP 503T Roll No………………………...**

 **SECTION-A**

**Note: Attempt all the questions: 10x1=10**

**1. Write triple response of Histamine**.

Red spot, Flare and wheal

**2. Give the name of histamine receptors**.

Musculotropic and Neurotropic

**3. Give the extra renal actions of Acetazolamide**.

Lowering IOT, Raised level of Co2 in brain and Lowering of PH.

**4. Write the two uses of Thiazide diuretics**.

Hypertension, Oedema and Diabetes Incipidus

**5. Give the ADR of Spironolactone**.

Hirsutism, Gynaecomastia and irregular menstrual cycle

**6. Give the uses of Mannitol**.

Increased Intracranial & Intraoccular tension

**7. Write the name of lipid derived autocoids**.

Prostaglandins, Leukotriens and Nitric Oxide

**8. Give the name of Precursor of Histamine**.

Histidine

**9. Bradykinin is the type of………. autocoid.**

Peptide

**10. Write the percentage of Iron present in Hemoglobin.**

66%

 **SECTION-B**

**2. Attempt any *two* of the following: (2X5=10)**

**1. Write about Absorption, Transportation, Utilization and Storage of iron.**

Iron is vital for living organisms because it is essential for multiple metabolic processes to include oxygen transport, DNA synthesis, and electron transport. However, iron must be bound to proteins to prevent tissue damage from free radical formation. Thus, its concentrations in body organs must be regulated carefully. Intestinal absorption is the primary mechanism regulating iron concentrations in the body.

Intestinal mucosal cell iron seems to exit the cell via a distinct apotransferrin receptor and a newly described protein named hephaestin. Unlike the absorptive surface of intestinal cells, most other cells possess transferrin receptors on their surfaces and the vast majority of iron entering these cells is transferrin associated. There seem to be 2 distinct pathways by which transferrin iron enters nonintestinal cells. In the classical clathrin-coated pitendosome pathway, iron accompanies transferrin into the cell to enter a vesicle, which releases the iron to the cytosol with acidification (high affinity, low capacity). Under physiological conditions, a second transferrin associated pathway (low affinity, high capacity) exists which has been named the transferrin receptor independent pathway (TRIP).

Cellular uptake of iron from iron salts probably occurs in iron overloading disorders and may be responsible for free radical damage when the iron binding capacity of plasma is exceeded. Radioiron entering the cell via the heme and transferrin associated pathways can be found in isolates of mobilferrin/paraferritin and hemoglobin. This interaction probably occurs to permit NADPH dependent ferri reduction so iron can be used for synthesis of heme proteins. Production of heme from iron delivered via these routes indicates functional specificity for the pathways.

 

 Iron absorption. Iron enters the stomach from the esophagus. Iron is oxidized to the Fe3+ state no matter its original form when taken in orally. Gastric acidity as well as solubilizing agents such as ascorbate prevent precipitation of the normally insoluble Fe3+. Intestinal mucosal cells in the duodenum and upper jejunum absorb the iron. The iron is coupled to transferrin (Tf) in the circulation which delivers it to the cells of the body. Phytates, tannins and antacids block iron absorption.

The absorption of dietary iron is a variable and dynamic process. The amount of iron absorbed compared to the amount ingested is typically low, but may range from 5% to as much as 35% depending on circumstances and type of iron. The efficiency with which iron is absorbed varies depending on the source.

 Like most mineral nutrients, the majority of the iron absorbed from digested food or supplements is absorbed in the [duodenum](https://en.wikipedia.org/wiki/Duodenum) by [enterocytes](https://en.wikipedia.org/wiki/Enterocyte%22%20%5Co%20%22Enterocyte) of the duodenal lining. These cells have special molecules that allow them to move iron into the body. To be absorbed, dietary iron can be absorbed as part of a protein such as heme protein or iron must be in its ferrous Fe2+ form.

 These intestinal lining cells can then either store the iron as [ferritin](https://en.wikipedia.org/wiki/Ferritin%22%20%5Co%20%22Ferritin), which is accomplished by Fe3+ binding to apoferritin (in which case the iron will leave the body when the cell dies and is sloughed off into [feces](https://en.wikipedia.org/wiki/Feces)), or the cell can release it into the body via the only known iron exporter in mammals, [ferroportin](https://en.wikipedia.org/wiki/Ferroportin%22%20%5Co%20%22Ferroportin). [Hephaestin](https://en.wikipedia.org/wiki/Hephaestin%22%20%5Co%20%22Hephaestin), a [ferroxidase](https://en.wikipedia.org/wiki/Ferroxidase%22%20%5Co%20%22Ferroxidase) that can oxidize Fe2+ to Fe3+ and is found mainly in the small intestine, helps ferroportin transfer iron across the basolateral end of the intestine cells.

**Iron recycling and loss**

 Most of the iron in the body is hoarded and recycled by the reticuloendothelial system, which breaks down aged red blood cells. In contrast to iron uptake and recycling, there is no physiologic regulatory mechanism for [excreting](https://en.wikipedia.org/wiki/Excretion) iron. People lose a small but steady amount by gastrointestinal blood loss, sweating and by shedding cells of the skin and the [mucosal](https://en.wikipedia.org/wiki/Mucosa) lining of the [gastrointestinal tract](https://en.wikipedia.org/wiki/Gastrointestinal_tract).

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Iron export occurs in a variety of cell types, including neurons, erythrocytes, macrophages and enterocytes. The latter two are especially important since systemic iron levels depend upon them. There is only one known iron exporter, [ferroportin](https://en.wikipedia.org/wiki/Ferroportin%22%20%5Co%20%22Ferroportin). It transports ferrous iron out of the cell, generally aided by [ceruloplasmin](https://en.wikipedia.org/wiki/Ceruloplasmin%22%20%5Co%20%22Ceruloplasmin) and/or [hephaestin](https://en.wikipedia.org/wiki/Hephaestin%22%20%5Co%20%22Hephaestin) (mostly in enterocytes), which oxidize iron to its ferric state so it can bind ferritin in the extracellular medium[Hepcidin](https://en.wikipedia.org/wiki/Hepcidin) causes the internalization of ferroportin, decreasing iron export. Besides, hepcidin seems to downregulate both TFR1 and DMT1 through an unknown mechanism.

**2.** **Write about coagulation mechanism with name of clotting factors**.

**Coagulation**, also known as **clotting**, is the process by which [blood](https://en.wikipedia.org/wiki/Blood) changes from a liquid to a [gel](https://en.wikipedia.org/wiki/Gel), forming a [blood clot](https://en.wikipedia.org/wiki/Thrombus). It potentially results in [hemostasis](https://en.wikipedia.org/wiki/Hemostasis%22%20%5Co%20%22Hemostasis), the cessation of blood loss from a damaged vessel, followed by repair. The mechanism of coagulation involves activation, adhesion and aggregation of [platelets](https://en.wikipedia.org/wiki/Platelet), as well as deposition and maturation of [fibrin](https://en.wikipedia.org/wiki/Fibrin).

Coagulation begins almost instantly after an injury to the blood vessel has damaged the [endothelium](https://en.wikipedia.org/wiki/Endothelium) lining the blood vessel. Exposure of blood to the subendothelial space initiates two processes: changes in platelets, and the exposure of subendothelial [tissue factor](https://en.wikipedia.org/wiki/Tissue_factor) to plasma [factor VII](https://en.wikipedia.org/wiki/Factor_VII), which ultimately leads to cross-linked fibrin formation. Platelets immediately form a plug at the site of injury; this is called *primary hemostasis*. *Secondary hemostasis* occurs simultaneously: additional coagulation (clotting) factors beyond factor VII ([listed below](https://en.wikipedia.org/wiki/Coagulation#Coagulation_factors)) respond in a cascade to form [fibrin](https://en.wikipedia.org/wiki/Fibrin) strands, which strengthen the [platelet plug](https://en.wikipedia.org/wiki/Platelet_plug)

The coagulation cascade is a complex chemical process that uses as many as 10 different proteins (called blood clotting factors or coagulation factors) found in plasma in the blood. Put simply, the clotting process changes blood from a liquid to a solid at the site of an injury. Here’s how the process works:

1. **Injury**
A small tear in a blood vessel wall (for example, from a cut on the skin or an internal injury) causes bleeding.
2. **Vessel constriction**
To control blood loss the blood vessel narrows (called constriction), thus limiting blood flow through the vessel.
3. **Platelet plug**
In response to the injury, tiny cells in the blood called platelets are activated. The platelets stick to one another and to the wound site to form a plug. The protein von Willebrand factor (VWF) helps the platelets stick to each other and to the blood vessel wall.
4. **Fibrin clot**
Next, clotting factor proteins trigger production of fibrin, a strong, strand-like substance that forms a fibrin clot, a mesh-like net that keeps the plug firm and stable. Over the next several days to weeks, the clot strengthens and then dissolves as the wounded blood vessel wall heals.



|  |  |  |
| --- | --- | --- |
| I ([fibrinogen](https://en.wikipedia.org/wiki/Fibrinogen)) | Forms clot (fibrin) | [Congenital afibrinogenemia](https://en.wikipedia.org/wiki/Congenital_afibrinogenemia), [Familial renal amyloidosis](https://en.wikipedia.org/wiki/Familial_renal_amyloidosis) |
| II ([prothrombin](https://en.wikipedia.org/wiki/Prothrombin%22%20%5Co%20%22Prothrombin)) | Its active form (IIa) activates I, V, VII, VIII, XI, XIII, [protein C](https://en.wikipedia.org/wiki/Protein_C), [platelets](https://en.wikipedia.org/wiki/Platelets) | [Prothrombin G20210A](https://en.wikipedia.org/wiki/Prothrombin_G20210A), [Thrombophilia](https://en.wikipedia.org/wiki/Thrombophilia%22%20%5Co%20%22Thrombophilia) |
| III ([tissue factor](https://en.wikipedia.org/wiki/Tissue_factor) or tissue thromboplastin) | Co-factor of VIIa (formerly known as factor III) |  |
| IV ([calcium](https://en.wikipedia.org/wiki/Calcium)) | Required for coagulation factors to bind to phospholipid (formerly known as factor IV) |  |
| [V](https://en.wikipedia.org/wiki/Factor_V) (proaccelerin, labile factor) | Co-factor of X with which it forms the [prothrombinase](https://en.wikipedia.org/wiki/Prothrombinase%22%20%5Co%20%22Prothrombinase) complex | [Activated protein C resistance](https://en.wikipedia.org/wiki/Activated_protein_C_resistance) |
| VI | *Unassigned* – old name of Factor Va |  |
| [VII](https://en.wikipedia.org/wiki/Factor_VII) (stable factor, proconvertin) | Activates IX, X | congenital [factor VII deficiency](https://en.wikipedia.org/wiki/Factor_VII_deficiency) |
| [VIII](https://en.wikipedia.org/wiki/Factor_VIII) (Antihemophilic factor A) | Co-factor of IX with which it forms the [tenase](https://en.wikipedia.org/wiki/Tenase%22%20%5Co%20%22Tenase) complex | [Haemophilia A](https://en.wikipedia.org/wiki/Haemophilia_A) |
| [IX](https://en.wikipedia.org/wiki/Factor_IX) (Antihemophilic factor B or Christmas factor) | Activates X: forms [tenase](https://en.wikipedia.org/wiki/Tenase%22%20%5Co%20%22Tenase) complex with factor VIII | [Haemophilia B](https://en.wikipedia.org/wiki/Haemophilia_B) |
| [X](https://en.wikipedia.org/wiki/Factor_X) (Stuart-Prower factor) | Activates II: forms [prothrombinase](https://en.wikipedia.org/wiki/Prothrombinase%22%20%5Co%20%22Prothrombinase) complex with factor V | Congenital Factor X deficiency |
| [XI](https://en.wikipedia.org/wiki/Factor_XI) (plasma thromboplastin antecedent) | Activates IX | [Haemophilia C](https://en.wikipedia.org/wiki/Haemophilia_C) |
| [XII](https://en.wikipedia.org/wiki/Factor_XII) (Hageman factor) | Activates factor XI, VII and prekallikrein | [Hereditary angioedema](https://en.wikipedia.org/wiki/Hereditary_angioedema) type III |
| [XIII](https://en.wikipedia.org/wiki/Factor_XIII) (fibrin-stabilizing factor) |  |  |

**3. Give a note on Vitamin K.**

The body needs [vitamin](https://www.medicalnewstoday.com/articles/195878.php) K to produce prothrombin, a protein and clotting factor that is important in blood clotting and bone metabolism. People who use blood-thinning medications, such as warfarin, or Coumadin, should not start consuming additional vitamin K without first asking a doctor.

Deficiency is rare, but, in severe cases, it [can increase](https://ods.od.nih.gov/factsheets/VitaminK-HealthProfessional/) clotting time, leading to hemorrhage and excessive bleeding.

Vitamin K1, or phylloquinone, comes from plants. It is the [main type](https://ods.od.nih.gov/factsheets/VitaminK-HealthProfessional/%20) of dietary vitamin K. A lesser source is vitamin K2, or menaquinone, which occurs in some animal-based and fermented foods.

Phylloquinone, also known as vitamin K1, is found in plants. When people eat it, bacteria in the large intestine convert it to its storage form, vitamin K2. It is absorbed in the small intestine and stored in fatty tissue and the liver.

Without vitamin K, the body cannot produce prothrombin, a clotting factor that is necessary for blood clotting and bone metabolism.

Most Americans are [not at risk](https://ods.od.nih.gov/factsheets/VitaminK-HealthProfessional/%20) of a vitamin-K deficiency. It is most likely to affect newborns and those with a malapsorption problem, due, for example, to short-bowel syndrome, [cystic fibrosis](https://www.medicalnewstoday.com/articles/147960.php), [celiac disease](https://www.medicalnewstoday.com/articles/38085.php), or ulcerative colitis.

Newborns normally [receive](http://lpi.oregonstate.edu/mic/vitamins/vitamin-K%20) a vitamin K injection to protect them from bleeding in the skull, which could be fatal.

The recommended adequate intake for vitamin K depends on age and gender. Women aged 19 years and over should consume [90 micrograms](https://ods.od.nih.gov/factsheets/VitaminK-HealthProfessional/%20) (mcg) a day, and men should have 120 mcg.

Vitamin K deficiency is only considered clinically relevant when prothrombin time increases significantly due to a decrease in the prothrombin activity of blood. Thus, bleeding and hemorrhage are the classic signs of vitamin K deficiency, although these effects occur only in severe cases. Because vitamin K is required for the carboxylation of osteocalcin in bone, vitamin K deficiency could also reduce bone mineralization and contribute to osteoporosis.

Vitamin K deficiency can occur during the first few weeks of infancy due to low placental transfer of phylloquinone, low clotting factor levels, and low vitamin K content of breast milk. Clinically significant vitamin K deficiency in adults is very rare and is usually limited to people with malabsorption disorders or those taking drugs that interfere with vitamin K metabolism. In healthy people consuming a varied diet, achieving a vitamin K intake low enough to alter standard clinical measures of blood coagulation is almost impossible

 **SECTION-C**

**3. Attempt any *one* of the following: (1X10=10)**

1. Write about Anticoagulants in detail with description of Heparin.

Anticoagulants are medicines that increase the time it takes for blood to clot. They are commonly called blood thinners.

There are several different types of anticoagulant. Each type works at a different level on the blood coagulation pathway. Some can be given by mouth; others can only be given by injection.

Anticoagulants may be used to treat blood clots, or in conditions where the risk of blood clots is increased to reduce the risk. Examples of conditions where anticoagulants may be used include:

* [Atrial fibrillation](https://www.drugs.com/cg/a-fib-atrial-fibrillation.html)
* [Deep vein thrombosis](https://www.drugs.com/cg/deep-vein-thrombosis.html) ([DVT](https://www.drugs.com/cg/deep-vein-thrombosis.html))
* Hip or knee replacement surgery
* Ischemic [stroke](https://www.drugs.com/health-guide/stroke.html)
* Myocardial infarction ([heart attack](https://www.drugs.com/cg/heart-attack.html))
* [Pulmonary embolism](https://www.drugs.com/cg/pulmonary-embolism.html)
* [Unstable angina](https://www.drugs.com/cg/angina.html).

### Heparins

The [heparins](https://www.drugs.com/drug-class/heparins.html) are a group of anticoagulants that consist of unfractionated heparin, low molecular weight heparins, and heparinoids.

Unfractionated heparin (usually just called heparin) needs to be given directly into the blood by intravenous (IV) injection, and inhibits thrombin and factor Xa, factors necessary in the final stages of the blood clotting cascade. Heparin may also be called high molecular weight heparin. Daily monitoring is required with heparin to check the aPTT. The aPTT is the speed at which clotting occurs.

Low molecular weight heparins (LMWH) also work on thrombin and factor Xa; however, they preferentially inactivate factor Xa. Because their anticoagulant response is more predictable, they do not need daily blood monitoring. LMWHs last much longer in the body than heparin and are injected under the skin (subcutaneously). Some people can learn to inject LMWHs at home by themselves.

Heparinoids have a similar action to heparin and are extracted from specific animal and plant tissues or made synthetically. They are usually applied topically and are easily absorbed into the skin where they can reduce small blood clots, reduce inflammation and associated pain and discomfort. Chitin and chondroitin sulfate are also heparinoids.

| Generic name | Brand name examples |
| --- | --- |
| **Unfractionated heparins** |
| [heparin](https://www.drugs.com/monograph/heparin-sodium.html) | [Hep-Lock](https://www.drugs.com/pro/hep-lock.html) |
| **LMWHs** |
| [dalteparin](https://www.drugs.com/mtm/dalteparin.html) | [Fragmin](https://www.drugs.com/fragmin.html) |
| [enoxaparin](https://www.drugs.com/mtm/enoxaparin.html) | [Lovenox](https://www.drugs.com/lovenox.html) |
| tinzaparin | Discontinued |
| **Heparinoids** |
| heparinoid | Hirudoid (not available in the U.S.) |

### Direct thrombin inhibitors

Direct thrombin inhibitors bind directly to thrombin, inhibiting its action. Direct thrombin inhibitors that need to be given by injection include [desirudin](https://www.drugs.com/mtm/desirudin.html) which binds to both the active enzymatic site and to exosite 1, and [argatroban](https://www.drugs.com/mtm/argatroban.html) which binds to the active enzymatic site only. [Dabigatran](https://www.drugs.com/mtm/dabigatran.html) is an oral direct thrombin inhibitor which binds reversibly to the active enzymatic site.

| Generic name | Brand name examples |
| --- | --- |
| [argatroban](https://www.drugs.com/mtm/argatroban.html) | [Acova](https://www.drugs.com/mtm/acova.html) |
| [bivalirudin](https://www.drugs.com/mtm/bivalirudin.html) | [Angiomax](https://www.drugs.com/mtm/angiomax.html) |
| [dabigatran](https://www.drugs.com/mtm/dabigatran.html) | [Pradaxa](https://www.drugs.com/pradaxa.html) |
| [desirudin](https://www.drugs.com/mtm/desirudin.html) | [Iprivask](https://www.drugs.com/mtm/iprivask.html) |
| lepirudin | Discontinued in 2012 |

Anticoagulants are considered safe when administered exactly as intended for the recommended duration of time. However, they have been associated with some serious side effects such as:

* Major or fatal bleeding and haemorrhage: Because of the way they work to prolong bleeding time, there is always a risk of severe bleeding with anticoagulants, particularly in people with risk factors such as active ulceration, bleeding disorders, [**hemorrhagic stroke**](https://www.drugs.com/cg/intracerebral-hemorrhage.html), following certain types of surgery, with kidney disease, or in people taking medicines that also increase the risk of bleeding. Any bleeding that does not stop or other signs such as persistent nosebleeds, blood in the urine or stools, [**heavy menstrual bleeding**](https://www.drugs.com/cg/menorrhagia.html), or coughing up blood should be investigated further.
* Spinal/epidural hematomas: Risk is higher with LMWHs when administered to people undergoing neuraxial (spinal or epidural) anesthesia or spinal puncture. These hematomas may result in permanent paralysis
* [**Thrombocytopenia**](https://www.drugs.com/cg/thrombocytopenia.html) (a deficiency of platelets in the blood)
* Necrosis and/or gangrene of the skin: rare, but has been associated with **[warfarin](https://www.drugs.com/warfarin.html)** use
* An increased risk of thrombotic events on premature discontinuation of **[dabigatran](https://www.drugs.com/mtm/dabigatran.html)** (before completion of a course of therapy).

Warfarin can also interact with certain foods and many commonly used medicines. Regular blood monitoring (international normalized ratio-INR) is necessary because there is a fine line between an effective dose and a toxic one.

For a complete list of severe side effects, please refer to the individual drug monographs.

##  Side effects of anticoagulants:

* Bleeding
* Gastrointestinal effects such as [**diarrhea**](https://www.drugs.com/cg/acute-diarrhea.html), [**heartburn**](https://www.drugs.com/cg/gastroesophageal-reflux-disease.html), [**nausea**](https://www.drugs.com/health-guide/nausea.html), and loss of appetite
* Irritation and pain around the site of injection (injectable anticoagulants only)
* Elevations in liver enzymes
* Shortness of breath.

**2. Write about Antihistamines in detail with their classifications.**

Antihistamines are a class of agents that block histamine release from histamine-1 receptors and are mostly used to treat [**allergies**](https://www.drugs.com/cg/allergies.html) or cold and flu symptoms, although some first-generation antihistamines may also be used for other conditions.

Histamine-1 receptors are located in the airways, blood vessels and gastrointestinal tract (stomach and esophagus). Stimulation of these receptors can lead to conditions such as a [**skin rash**](https://www.drugs.com/cg/acute-rash.html) or inflammation, a narrowing of the airways (bronchoconstriction), [**hay fever**](https://www.drugs.com/cg/allergic-rhinitis.html), or [**motion sickness**](https://www.drugs.com/cg/motion-sickness.html). Histamine-1 receptors are also found in the brain and spinal cord, and stimulation of these receptors makes you more awake and alert. Sedating antihistamines oppose the effects of histamine on H1 receptors in your brain, which is why they cause sedation and drowsiness.

Chronic allergies increase the risk of health problems which antihistamines might not treat, including [asthma](https://en.wikipedia.org/wiki/Asthma), [sinusitis](https://en.wikipedia.org/wiki/Sinusitis), and [lower respiratory tract infection](https://en.wikipedia.org/wiki/Lower_respiratory_tract_infection).[[1]](https://en.wikipedia.org/wiki/Antihistamine#cite_note-Consumer_Reports_2013-1) Consultation of a medical professional is recommended for those who intend to take antihistamines for longer-term use.

Although people typically use the word “antihistamine” to describe drugs for treating allergies, doctors and scientists use the term to describe a class of drug that opposes the activity of [histamine receptors](https://en.wikipedia.org/wiki/Histamine_receptor) in the body. In this sense of the word, antihistamines are subclassified according to the [histamine](https://en.wikipedia.org/wiki/Histamine) receptor that they act upon. The two largest classes of antihistamines are [H1-antihistamines](https://en.wikipedia.org/wiki/H1_antagonist) and [H2-antihistamines](https://en.wikipedia.org/wiki/H2_antagonist).

H1-antihistamines work by binding to [histamine H1 receptors](https://en.wikipedia.org/wiki/HRH1) in [mast cells](https://en.wikipedia.org/wiki/Mast_cells), [smooth muscle](https://en.wikipedia.org/wiki/Smooth_muscle), and [endothelium](https://en.wikipedia.org/wiki/Endothelium) in the body as well as in the [tuberomammillary nucleus](https://en.wikipedia.org/wiki/Tuberomammillary_nucleus%22%20%5Co%20%22Tuberomammillary%20nucleus) in the brain. Antihistamines that target the [histamine H1-receptor](https://en.wikipedia.org/wiki/HRH1) are used to treat [allergic reactions in the nose](https://en.wikipedia.org/wiki/Allergic_rhinitis) (e.g., itching, runny nose, and sneezing). In addition, they may be used to treat [insomnia](https://en.wikipedia.org/wiki/Insomnia), motion sickness, or [vertigo](https://en.wikipedia.org/wiki/Vertigo) caused by problems with the [inner ear](https://en.wikipedia.org/wiki/Inner_ear). H2-antihistamines bind to [histamine H2 receptors](https://en.wikipedia.org/wiki/HRH2) in the upper [gastrointestinal tract](https://en.wikipedia.org/wiki/Human_gastrointestinal_tract), primarily in the [stomach](https://en.wikipedia.org/wiki/Stomach). Antihistamines that target the [histamine H2-receptor](https://en.wikipedia.org/wiki/HRH2) are used to treat [gastric acid](https://en.wikipedia.org/wiki/Gastric_acid) conditions (e.g., [peptic ulcers](https://en.wikipedia.org/wiki/Peptic_ulcers) and [acid reflux](https://en.wikipedia.org/wiki/Acid_reflux)).

**First generation antihistamines include:**

* brompheniramine
* chlorpheniramine (Chlor-Trimeton)
* [diphenhydramine](https://www.medicinenet.com/diphenhydramine/article.htm) ([Benadryl](https://www.medicinenet.com/diphenhydramine/article.htm))
* doxylamine (found in many OTC [sleep](https://www.medicinenet.com/sleep_quiz/quiz.htm) aids including Unisom)
* carbinoxamine (Karbinal ER)

Second generation antihistamines include

* [fexofenadine](https://www.medicinenet.com/fexofenadine/article.htm) ([Allegra](https://www.medicinenet.com/fexofenadine/article.htm))
* [loratadine](https://www.medicinenet.com/loratadine/article.htm) ([Claritin](https://www.medicinenet.com/loratadine/article.htm))
* loratadine ODT ([Alavert](https://www.medicinenet.com/loratadine/article.htm), [Tavist](https://www.medicinenet.com/clemastine-oral/article.htm) ND)
* [desloratadine](https://www.medicinenet.com/desloratadine/article.htm) ([Clarinex](https://www.medicinenet.com/desloratadine/article.htm))
* certirizine (Zyrtec)

## Side effects of antihistamines

Like all medicines, antihistamines can cause side effects.

Side effects of older types of antihistamines can include:

* sleepiness (drowsiness) and reduced co-ordination, reaction speed and judgement – don't drive or use machinery after taking these antihistamines because of this risk
* [dry mouth](https://www.nhs.uk/conditions/dry-mouth/)
* blurred vision
* difficulty emptying your bladder

Side effects of non-drowsy antihistamines can include:

* [headache](https://www.nhs.uk/conditions/headaches/)
* dry mouth
* feeling sick
* drowsiness – this is less common than with older types of antihistamines