

# Antiaggregation

*Antiplatelet* drugs are a group of drugs that inhibit **primary hemostasis** and thus prevent the formation of a **primary platelet thrombus** (antiplatelet drugs). Antiaggregants are mainly used in the prophylaxis of arterial thrombus formation.

The most common indications:

- secondary prevention of *myocardial infarction* ,
- secondary prevention of ischemic stroke and transient ischemic attacks.

## Mechanism of action

According to the mechanism of action, antiaggregants can be divided into:

- irreversible cyclooxygenase (COX) inhibitors of platelets and endothelium,
- platelet ADP receptor inhibitors,
- inhibitors of receptors for fibrinogen (gpIIb-IIIa),
- phosphodiesterase inhibitors.

### Platelet and Endothelial Irreversible COX Inhibitors [ [edit](#) | [edit source](#) ]

*Acetylsalicylic acid* is the most commonly used antiplatelet agent. In the body, ASA blocks platelet cyclooxygenase, thereby preventing the formation of prothrombotic thromboxane A<sub>2</sub>, but at the same time blocks the synthesis of PGI<sub>2</sub>, which has a vasodilating and antiaggregatory effect, in endothelial cells. The goal of the therapy is to block only the aggregation-acting substance TXA<sub>2</sub> while maintaining the secretion of PGI<sub>2</sub>. We achieve this by administering low doses of ASA (100-300mg/day). At these doses, platelet cyclooxygenase is irreversibly blocked and the effect lasts for the entire lifetime of the platelet, roughly 7-8 days.

### Inhibitors of platelet receptors for ADP [ [edit](#) | [edit source](#) ]

This group does not affect platelet cyclooxygenase, but blocks receptors for ADP (Adenosine diphosphate) and thus prevents platelet activation. These substances are used in patients who cannot tolerate ASA administration for various reasons (e.g. peptic ulcer) and in patients who need a dual antiplatelet agent. Representatives of this group are *clopidogrel* , *ticlopidine* and the new preparations *ticagrelor* and *prasugrel* . Ticlopidine was withdrawn from use because of its potential hematotoxicity. Ticagrelor and prasugrel in combination with acetylsalicylic acid are used for antiplatelet therapy in interventions on arteries (mainly before and some time after percutaneous interventions on coronary arteries).

### Inhibitors of receptors for fibrinogen (gpIIb-IIIa) [ [edit](#) | [edit source](#) ]

GpIIb-IIIa serve as receptors for fibrinogen on the platelet surface. By blocking these receptors, we inhibit the final step of platelet activation. Substances that act in this way include *abciximab* , *tirofiban* and *eptifibatide* . *Abciximab* is a fragment of a monoclonal chimeric antibody, which is indicated for patients unresponsive to conventional therapy and for the prevention of cardiac complications during percutaneous interventions. *Tirofiban* is a non-peptide antagonist that is indicated for unstable *angina pectoris* . The cyclic heptapeptide *eptifibatide* has similar indications . The importance of gpIIb-IIIa inhibitors decreased after the introduction of ticagrelor and prasugrel into clinical practice.

### Phosphodiesterase inhibitors [ [edit](#) | [edit source](#) ]

A representative of this group is *dipyridamole* . It is a coronary vasodilator that does not show antiplatelet effects *in vitro* . It increases the level of cAMP in platelets. It reduces the adhesiveness of platelets to the damaged endothelium. In therapy, it is most often used in combination with another antiplatelet agent.

## Links

### Related Articles

- Hemostasis
- Acetylsalicylic acid
- Exercise: Antiaggregants

### References

- LINCOVA, Dagmar, et al. *Basic and applied pharmacology*. 1st edition. GALÉN, 2002. 601 pp. ISBN 80-7262-168-8 .
- HYNIA, Sixtus. *Pharmacology in a nutshell*. 2nd edition. Prague: Triton, 2001. 520 pp. ISBN 80-7254-181-1 .

- WIVIOTT, Stephen D and Philippe Gabriel STEG. Clinical evidence for oral antiplatelet therapy in acute coronary syndromes. *The Lancet* [online] . 2015, vol. 386, no. 9990, pp. 292-302, also available from < <https://www.ncbi.nlm.nih.gov/pubmed/25777663> >. ISSN 0140-6736 (print), 1474-547X.