# COMPARATIVE STUDY OF ASPIRIN TABLET AS ANALGESIC AND ANTIPLATELET AGENT- A RIVIEW

**YAMINI SONEWANE*, VARSHA BHELAWE*, DIPIKA BOPCHE, TULSIDAS NIMBEKAR**

SHRI LAXMANRAO MANKAR INSTITUTE OF PHARMACY, AMGAON ,441902

## ABSTRACT

Aspirin (acetylsalicylic acid) naturally originates from a glycoside known as salicin and is essential regarded as the oldest and most widely used drug in the world. Aspirin is a non-steroidal anti-inflammatory drug that has potent antiplatelet action. Aspirin has antithrombotic effects due to the inhibition of Z cyclo-oxygenase activity in platelets, which reduces the extent of thromboxane A2 formation and consequently the aggregability of platelet. Analgesic drugs are those drug or agents which are used to reduce pain. Antiplatelet drugs are those drug or agents which are used to inhibit the clumping of platelets.

**Keywords:** Aspirin, Antiplatelet, COX inhibitor, Analgesic

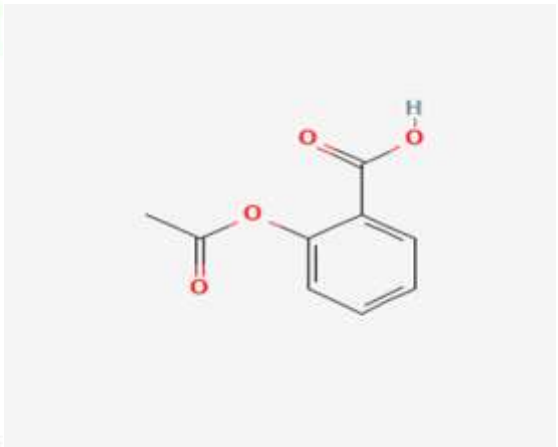
## INTRODUCTION

**Aspirin**: Aspirin is the inhibitor of platelet aggregation that display good antithrombotic activity. It is used in the prophylaxis of patient undergoing vascular grafting or percutaneous angioplasty, in the medical management of acute coronary syndromes, and in long term prevention of cardiovascular and cerebrovascular events.

Aspirin is non-steroidal anti-inflammatory drug that has potent antiplatelet actions. Aspirin was initially used as antipyretic and analgesic drug before its anti-inflammatory property was discovered , aspirin also has antithrombotic effects due to inhibition of cyclo-oxygenase activity in platelets.

Platelets play a key role in atherothrombosis, and the measurement of platelet activity is independently associated with cardiovascular events and long-term mortality.

**Platelet**: Platelets are a nucleated discoid blood elements formed by fragmentation of megakaryocytes cytoplasm. Although a nucleated platelets retain a limited biosynthesis machinery to provide for *denovo* protein synthesis.

## AIM AND OBJECTIVE

**Aim:** To compare the efficacy of aspirin as an analgesic and antiplatelet agent, exploring its therapeutic benefits and potential risks in clinical practice.

**Objectives**

1. Analgesic Efficacy: To assess the pain-relieving effects of aspirin in patients with acute or chronic pain conditions.

2. Antiplatelet Efficacy: To evaluate the antiplatelet activity of aspirin in preventing thrombotic events in patients with cardiovascular disease.

3. Comparative Analysis: To compare the therapeutic outcomes, safety profiles, and patient tolerability of aspirin as an analgesic versus antiplatelet agent.

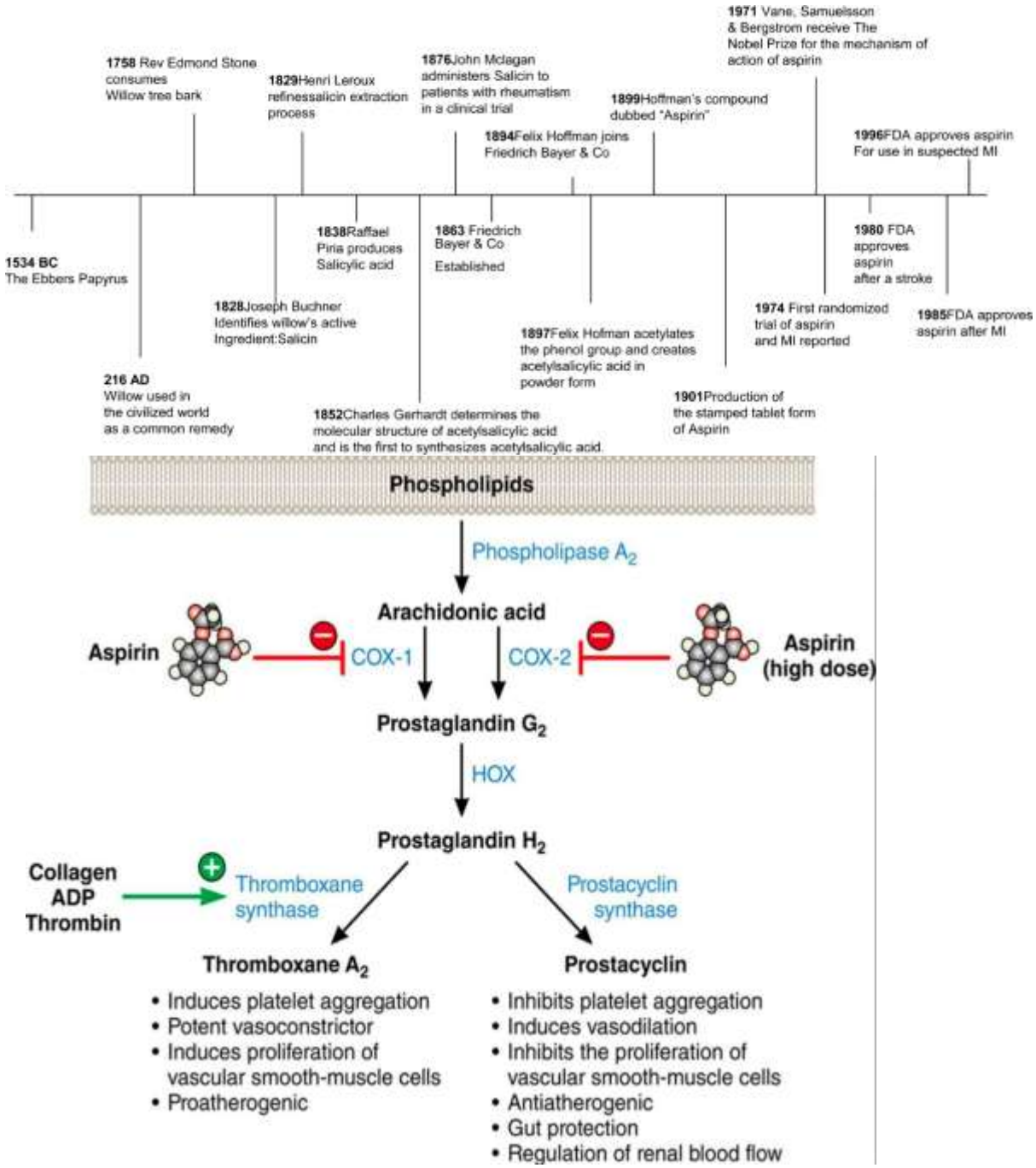
4. Clinical Implications: To identify the clinical implications of using aspirin as an analgesic and antiplatelet agent, including potential benefits, risks, and contraindications.

## HISTORICAL DEVLOPMENT

Acetylsalicylic acid was first synthesised in 1853 by Von Gerhardt, by mixing salicylic acid with acetic acid. In 1894, Felix Hoffmann, a pharmacist at the Bayer company in Elberfield , Germany, gave acetylsalicylic acid to his father to treat rheumatoid arthritis; formal clinical trials soon followed. In 1899, acetylsalicylic acid was patented by Bayer Co. under the trade name of Aspirin. ‘a’ stood for acetyl and ‘spirin’ stood for Spirsaure (a German word for salicylic acid). So overwhelming was the popularity of acetylsalicylic acid, that its original trade name eventually became the generic name.

Initially, aspirin was used as an analgesic and an antipyretic. It was thought that aspirin affected the thalamus, thereby increasing the pain threshold. The anti-inflammatory properties of aspirin were subsequently recognised in 1971. The mechanism of aspirin -the inhibition of prostaglandin synthesis – was elucidated by sir john Vane, who was awarded the Nobel prize in 1982 for this important contribution to medicine.

## GENRAL MECHANISM OF ACTIO N OF ASPIRIN




## ASPIRIN AS ANALGEGESIC AGENT

Mechanism of Action

Aspirin's analgesic effect is primarily due to its inhibition of cyclooxygenase (COX) enzymes, specifically COX-1 and COX-2. These enzymes are responsible for converting arachidonic acid into prostaglandins, which are pro-inflammatory mediators that sensitize nerve endings to painful stimuli.

Effects on Pain Pathways

Aspirin's analgesic effect involves the following mechanisms:

1. Inhibition of prostaglandin synthesis: Aspirin's inhibition of COX enzymes reduces the production of prostaglandins, which in turn reduces the sensitization of nerve endings to painful stimuli.

2. Blockade of pain transmission: Aspirin may also block the transmission of pain signals to the brain by inhibiting the release of pain-producing neurotransmitters.

3. Anti-inflammatory effects: Aspirin's anti-inflammatory effects may also contribute to its analgesic effect by reducing inflammation and swelling at the site of injury.

Clinical Uses

Aspirin is commonly used to relieve mild to moderate pain associated with:

1. Headaches: Tension headaches, migraines, and cluster headaches.

2. Musculoskeletal pain: Pain associated with arthritis, fibromyalgia, and muscle strains.

3. Menstrual cramps: Pain associated with menstrual cramps (dysmenorrhea).

4. Post-operative pain: Pain associated with surgical procedures.

Dosing and Administration

The recommended dose of aspirin for analgesic purposes varies depending on the specific condition being treated:

1. Adults: 325-1000 mg every 4-6 hours as needed.

2. Children: 10-15 mg/kg every 4-6 hours as needed.

Side Effects and Contraindications

Aspirin can cause several side effects, including:

1. Gastrointestinal upset: Nausea, vomiting, diarrhoea, and abdominal pain.

2. Bleeding: Increased risk of bleeding due to aspirin's antiplatelet effects.

3. Allergic reactions: Rare but potentially life-threatening allergic reactions.

Aspirin is contraindicated in patients with:

1. Bleeding disorders: Haemophilia, von Willebrand disease, and other bleeding disorders.

2. Gastrointestinal ulcers: Active peptic ulcers or a history of gastrointestinal bleeding.

3. Allergies: Known allergy to aspirin or other NSAIDs.

## ASPIRIN AS ANTIPLATELET AGENT

Mechanism of Action

Aspirin's antiplatelet action is primarily due to its inhibition of cyclooxygenase-1 (COX-1) enzyme in platelets. This inhibition:

1. Blocks thromboxane A2 (TXA2) synthesis: TXA2 is a potent platelet activator and aggregator. Aspirin's inhibition of COX-1 reduces TXA2 production, thereby preventing platelet activation and aggregation.

2. Inhibits platelet aggregation: Aspirin's inhibition of COX-1 also reduces the production of other platelet activators, such as prostaglandin H2 (PGH2) and prostaglandin E2 (PGE2).

Effects on Platelet Function

Aspirin's antiplatelet action affects platelet function in several ways:

1. Inhibits platelet aggregation: Aspirin reduces the ability of platelets to aggregate and form blood clots.

2. Reduces platelet adhesion: Aspirin decreases the ability of platelets to adhere to the vascular endothelium.

3. Inhibits platelet activation: Aspirin reduces the activation of platelets, which is necessary for platelet aggregation and blood clot formation.

Clinical Uses

Aspirin's antiplatelet action is used to prevent and treat various cardiovascular conditions, including:

1. Myocardial infarction (MI): Aspirin is used to prevent recurrent MI and reduce mortality.

2. Stroke: Aspirin is used to prevent ischemic stroke and reduce the risk of recurrent stroke.

3. Peripheral artery disease (PAD): Aspirin is used to prevent PAD and reduce the risk of cardiovascular events.

4. Coronary artery bypass grafting (CABG): Aspirin is used to prevent graft occlusion and reduce the risk of cardiovascular events.

Dosing and Administration

The recommended dose of aspirin for antiplatelet purposes varies depending on the specific condition being treated:

1. Low-dose aspirin: 75-81 mg/day for long-term prevention of cardiovascular events.

2. High-dose aspirin: 325-650 mg/day for acute treatment of cardiovascular events.

Side Effects and Contraindications

Aspirin's antiplatelet action can increase the risk of bleeding, particularly when used in combination with other anticoagulants or antiplatelet agents. Other side effects include:

1. Gastrointestinal upset: Nausea, vomiting, diarrhoea, and abdominal pain.

2. Allergic reactions: Rare but potentially life-threatening allergic reactions.

Aspirin is contraindicated in patients with:

1. Bleeding disorders: Haemophilia, von Willebrand disease, and other bleeding disorders.

2. Gastrointestinal ulcers: Active peptic ulcers or a history of gastrointestinal bleeding.

3. Allergies: Known allergy to aspirin or other NSAIDs.

# COMPARETIVE STUDY OF ASPIRIN AS ANALGESIC AND ANTIPLATELET AGENT

| | ANTIPLATELET ACTIVITY | ANALGESIC ACTIVITY |
|---|---|---|
| **1.Mechanism:** | Inhibition of cyclooxygenase-1 (COX-1) enzyme in platelets, reducing thromboxane A2 (TXA2) production. | Inhibition of cyclooxygenase (COX) enzymes, reducing prostaglandin production, which sensitizes nerve endings to painful stimuli. |
| **2. Effect:** | Prevents platelet aggregation and blood clot formation. | Relieves pain, reduces inflammation, and lowers fever. |
| **3. Dose:** | Low dose (75-81 mg/day) is effective for antiplatelet activity. | Higher dose (325-1000 mg) is typically required for analgesic activity.4. Duration: Reversible inhibition of COX enzymes, with effects lasting for 4-6 hours.5. Clinical use: Relief of mild to moderate pain, reduction of inflammation, and treatment of fever. |
| **4. Duration:** | Irreversible inhibition of platelet COX-1, lasting for the lifespan of the platelet (7-10 days). | Reversible inhibition of COX enzymes, with effects lasting for 4-6 hours. |
| **5. Clinical use:** | Prevention of myocardial infarction, stroke, and other cardiovascular events. | Relief of mild to moderate pain, reduction of inflammation, and treatment of fever. |

## PROPERTIES OF ASPIRIN/ACETYLSALICYLIC ACID – $C_9H_8O_4$

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|---|---|
| $C_9H_8O_4$ | Acetylsalicylic acid |
| Molecular Weight/ Molar Mass | 180.159 g/mol |
| Density | 1.40 g/cm³ |
| Boiling Point | 140°C |
| Melting Point | 136°C |

**Uses of Aspirin/Acetylsalicylic acid-($C_9H_8O_4$)**

- Acetylsalicylic acid acts as an inhibitor of cyclooxygenase.
- It is used to prevent venous and arterial thrombosis.
- It is used in the treatment of different types of headaches.
- It is used as an anti-inflammatory agent for long-term as well as acute inflammation.
- It is thought to reduce the overall risk of getting cancer, and dying from cancer.
- Aspirin is an important part of the treatment of those who have had a heart attack.
- It is a first-line treatment for the fever and joint pain symptoms of acute rheumatic fever.

**CONCLUSION**

- Dose: Antiplatelet activity requires a lower dose than analgesic activity.
- Duration: Antiplatelet activity is irreversible and lasts for the lifespan of the platelet, while analgesic activity is reversible and lasts for 4-6 hours. -
- Mechanism: Antiplatelet activity involves inhibition of COX-1 in platelets, while analgesic activity involves inhibition of COX enzymes in peripheral tissues.
- Clinical use: Antiplatelet activity is used to prevent cardiovascular events, while analgesic activity is used to relieve pain, reduce inflammation, and treat fever.

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