A Comprehensive Review on the Mechanism of Action of Aspirin in the Management of Cardiovascular Diseases

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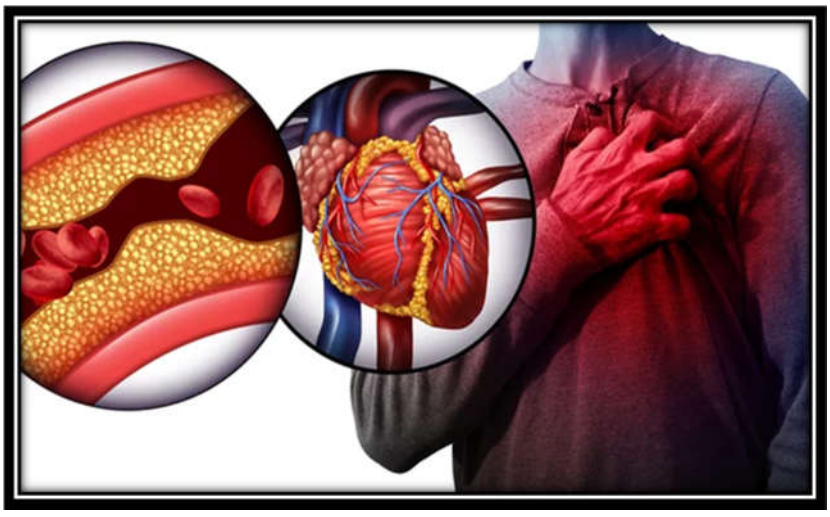
Abstract:

Aspirin (acetylsalicylic acid) remains one of the most widely used antiplatelet agents in the prevention and management of cardiovascular diseases (CVDs). This review explores the molecular and physiological mechanisms by which aspirin exerts its cardioprotective effects. Primarily, aspirin irreversibly inhibits cyclooxygenase-1 (COX-1) in platelets, thereby suppressing the synthesis of thromboxane A2, a potent promoter of platelet aggregation and vasoconstriction. This action leads to a prolonged antithrombotic effect, especially relevant in the secondary prevention of myocardial infarction, ischemic stroke, and other thrombotic events. Additionally, emerging evidence suggests that aspirin has anti-inflammatory and endothelial-modulating properties that may contribute to its benefits in atherosclerosis. Despite its established efficacy, aspirin therapy must be carefully weighed against the risk of gastrointestinal bleeding and hemorrhagic stroke. This review synthesizes current knowledge from clinical and experimental studies to provide a clearer understanding of aspirin's therapeutic role, limitations, and future directions in cardiovascular care.

Keywords: Aspirin; Cardiovascular diseases; Antiplatelet therapy; Cyclooxygenase inhibition; Thromboxane A2; Atherosclerosis.

Graphical Abstract



1. Introduction

Cardiovascular diseases (CVDs) refer to a broad group of disorders affecting the heart and blood vessels. They are the leading cause of morbidity and mortality worldwide, accounting for nearly one-third of all deaths globally. CVDs include conditions such as coronary artery disease (CAD), heart failure, arrhythmias, stroke, peripheral artery disease, and hypertension [1]. At the core of many CVDs is atherosclerosis, a chronic inflammatory process where fatty deposits (plaques) build up inside arteries, leading to narrowing and reduced blood flow. This can cause ischemia (lack of oxygen) to vital organs, particularly the heart and brain. Risk factors for CVD are multifactorial and include modifiable factors such as high blood pressure, high cholesterol, smoking, diabetes, obesity, sedentary lifestyle, unhealthy diet, and stress, as well as non-modifiable factors like age, sex, and genetic predisposition [2, 3]. The pathophysiology of CVD involves endothelial dysfunction, inflammation, oxidative stress, and thrombosis. Endothelial cells lining the blood vessels lose their normal regulatory functions, which promotes plaque formation and vascular stiffness. Chronic inflammation plays a pivotal role in plaque instability and rupture, leading to acute events like myocardial infarction (heart attack) or ischemic stroke [4,5]. Prevention and management of CVD focus on lifestyle modification (diet, exercise, smoking cessation), controlling risk factors (e.g., antihypertensives, statins, antidiabetics), and interventional procedures when necessary (angioplasty, bypass surgery). Advances in diagnostics, pharmacotherapy, and public health measures have reduced mortality, but CVD remains a significant global health challenge, especially with aging populations and increasing prevalence of obesity and diabetes [6, 7].

Importance of Antiplatelet Therapy in CVD:

Antiplatelet therapy plays a critical role in the prevention and management of cardiovascular diseases (CVD), particularly in conditions like myocardial infarction, stroke, and peripheral arterial disease. Platelet activation and aggregation are key steps in thrombus (clot) formation, which can block blood vessels and lead to serious cardiovascular events. Antiplatelet drugs, such as aspirin and P2Y12 inhibitors (e.g., clopidogrel), inhibit platelet function and reduce the risk of arterial thrombosis [8-10].

Prevention of acute events: They significantly lower the incidence of heart attacks and ischemic strokes, especially in high-risk patients [11].

Post-intervention benefit: After procedures like percutaneous coronary intervention (PCI) or stent placement, dual antiplatelet therapy prevents stent thrombosis [12].

Secondary prevention: In patients with established CVD, long-term antiplatelet use reduces recurrent cardiovascular events [13]. Despite their benefits, antiplatelet drugs can increase bleeding risk, so therapy must be individualized based on patient risk profiles [14].

Aspirin as a Cornerstone Drug in CVD Management:

Aspirin (acetylsalicylic acid) has been a foundational therapy in the management of cardiovascular disease (CVD) for decades. Its primary mechanism—irreversible inhibition of cyclooxygenase-1 (COX-1)—leads to reduced thromboxane A2 production, which in turn inhibits platelet aggregation.

This antiplatelet effect is essential in preventing thrombotic events such as myocardial infarction (MI) and ischemic stroke [15].

Mechanism of Action in CVD:

Antiplatelet effect: Aspirin inhibits COX-1 in platelets, preventing the synthesis of thromboxane A2, a potent vasoconstrictor and promoter of platelet aggregation. This reduces clot formation in atherosclerotic arteries [16].

Irreversible binding: Unlike other NSAIDs, aspirin irreversibly acetylates COX-1, which means a single dose affects platelets for their entire lifespan (~7–10 days) [17].

Role in Primary vs. Secondary Prevention:

Secondary prevention: Aspirin has a well-established role in patients with previous MI, stroke, or established coronary artery disease. It significantly reduces the risk of recurrent events and mortality [18].

Primary prevention: Use in individuals without established CVD is more controversial. Benefits must be balanced against the increased risk of gastrointestinal (GI) bleeding and haemorrhagic stroke, especially in older adults or those with bleeding risk factors [19].

Dosage and Administration:

Typically, low-dose aspirin (75–100 mg daily) is used for CVD prevention. High doses are avoided due to increased risk of side effects without added cardiovascular benefit [20].

Risk-Benefit Considerations:

Benefits: Reduced risk of major adverse cardiovascular events (MACE), including MI and ischemic stroke.

Risks: GI bleeding, peptic ulcer disease, and haemorrhagic stroke, particularly in the elderly and those with a history of ulcers or concomitant NSAID use. Risk stratification tools (e.g., ASCVD risk calculator) help identify patients who may benefit most from aspirin therapy [21].

Emerging Trends and Guidelines:

American Heart Association (AHA) and U.S. Preventive Services Task Force (USPSTF): Recent guidelines recommend more selective use of aspirin in primary prevention, often limiting it to individuals aged 40–70 at high ASCVD risk but low bleeding risk [22].

Combination therapy: In high-risk patients, aspirin may be combined with other antithrombotic (e.g., P2Y12 inhibitors like clopidogrel) post-PCI or in acute coronary syndrome, known as dual antiplatelet therapy (DAPT). Aspirin remains a cornerstone in the secondary prevention of CVD due to its proven efficacy and cost-effectiveness. However, its role in primary prevention is increasingly refined, emphasizing personalized risk-benefit assessment. Ongoing research continues to evaluate its optimal use in diverse patient populations [23].

2. Mechanism of Action Overview:

Renin-Angiotensin-Aldosterone System (RAAS) Inhibitors

Mechanism: Inhibit angiotensin-converting enzyme (ACE) or block angiotensin II receptors.

Effect: Reduce vasoconstriction, sodium retention, and blood pressure.

Drugs: ACE inhibitors (e.g., enalapril), ARBs (e.g., losartan) [24].

Beta-Adrenergic Blockers

Mechanism: Block beta-1 adrenergic receptors in the heart.

Effect: Reduce heart rate, myocardial oxygen demand, and blood pressure.

Drugs: Metoprolol, carvedilol [25].

Calcium Channel Blockers

Mechanism: Inhibit L-type calcium channels in vascular smooth muscle.

Effect: Vasodilation, decreased myocardial contractility.

Drugs: Amlodipine, verapamil [26].

Statins (HMG-CoA Reductase Inhibitors)

Mechanism: Inhibit cholesterol synthesis in the liver.

Effect: Lower LDL cholesterol, stabilize atherosclerotic plaques.

Drugs: Atorvastatin, rosuvastatin [27].

Antiplatelet Agents

Mechanism: Inhibit platelet aggregation via COX-1 (aspirin) or P2Y12 receptors (clopidogrel).

Effect: Prevent thrombosis in coronary arteries.

Drugs: Aspirin, clopidogrel, ticagrelor [28].

Sodium-Glucose Cotransporter-2 (SGLT2) Inhibitors

Mechanism: Reduce glucose reabsorption in kidneys.

Effect: Improve cardiac outcomes, reduce heart failure risk.

Drugs: Empagliflozin, dapagliflozin [29]**.**

Additional Mechanisms and Effects: Cardiovascular disease (CVD) involves a wide range of pathological mechanisms beyond the well-known causes like atherosclerosis and hypertension. Here are additional mechanisms and effects associated with CVD [30]**:**

Additional Mechanisms:

Endothelial Dysfunction: Impaired nitric oxide production leads to vascular stiffness and promotes inflammation and thrombosis [31].

Inflammation and Immune Response: Chronic inflammation contributes to plaque instability and myocardial damage, involving cytokines like IL-6 and TNF-α [32].

Oxidative Stress: Excessive reactive oxygen species (ROS) damage cardiac tissue and promote atherosclerosis [33].

Autonomic Nervous System Imbalance: Overactivation of the sympathetic nervous system increases heart rate, blood pressure, and risk of arrhythmias [34].

Mitochondrial Dysfunction: Impaired energy metabolism in cardiomyocytes contributes to heart failure and ischemic injury [35].

Genetic and Epigenetic Changes: Mutations and DNA methylation patterns affect lipid metabolism, inflammation, and cardiac remodelling [36].

Gut Microbiota: Certain gut bacteria produce metabolites (like TMAO) that promote thrombosis and atherosclerosis [37].

Effects of CVD:

Ischemia and Infarction: Reduced blood flow leads to tissue death, especially in the heart and brain (e.g., myocardial infarction, stroke).

Heart Failure: The heart's reduced ability to pump blood effectively, leading to fatigue, edema, and organ dysfunction.

Arrhythmias: Irregular heartbeats due to electrical conduction abnormalities.

Organ Damage: Kidney, liver, and brain may suffer from chronic low perfusion or embolic events.

Thromboembolic Events: Increased risk of clots leading to pulmonary embolism or systemic embolism [38].

2. Literature Review

1. Patro no et al. (2018) provided an extensive review on aspirin's antiplatelet effects, emphasizing its irreversible inhibition of cyclooxygenase-1 (COX-1) in platelets. This inhibition prevents thromboxane A2 synthesis, which reduces platelet aggregation and thrombus formation. The authors highlighted aspirin's role in primary and secondary prevention of cardiovascular events, noting dose-dependent effects and risks such as gastrointestinal bleeding. They discussed emerging low-dose regimens optimized to balance efficacy and safety in patients with high cardiovascular risk [39].

2. Zhao and Zhang (2019) explored aspirin's multifaceted role beyond platelet inhibition, including anti-inflammatory and endothelial protective actions. They described aspirin's acetylation of COX-2 leading to the production of anti-inflammatory lipoxins, contributing to vascular homeostasis. Their review emphasized aspirin's benefit in stabilizing atherosclerotic plaques and modulating inflammatory pathways involved in cardiovascular disease progression. The authors concluded that aspirin's therapeutic effects arise from both antithrombotic and anti-inflammatory mechanisms [40].

3. Kim et al. (2019) analysed aspirin's pharmacodynamics in diverse populations, focusing on variability in response due to genetic polymorphisms in COX enzymes. They discussed how altered COX-1 activity may reduce aspirin efficacy in some patients, termed "aspirin resistance," leading to poorer cardiovascular outcomes. The review suggested personalized dosing strategies and adjunct therapies to overcome resistance and improve prevention of ischemic events in high-risk groups [41].

4. Singh and Kapoor (2020) reviewed aspirin's mechanism in the context of secondary prevention after myocardial infarction and stroke. They emphasized aspirin's role in inhibiting platelet aggregation at sites of vascular injury and its contribution to reducing recurrent thrombotic events. The review also covered clinical trial evidence supporting long-term aspirin therapy, while discussing adverse effects like bleeding risks, highlighting the importance of risk-benefit assessment in therapy decisions [42].

5. Wang et al. (2020) focused on aspirin's role in endothelial function, describing how aspirin enhances nitric oxide production, which improves vasodilation and inhibits platelet adhesion. The review pointed out that aspirin's benefits in cardiovascular disease extend to improving vascular health by reducing oxidative stress and endothelial dysfunction, both critical in atherosclerosis development. The authors recommended further research into aspirin's vascular protective mechanisms [43].

6. Garcia and Fernandez (2020) summarized aspirin's interaction with other cardiovascular drugs, focusing on synergistic effects with statins and ACE inhibitors. They described how aspirin enhances the anti-inflammatory and plaque-stabilizing properties of these agents. The review underscored aspirin's pivotal role in multifactorial cardiovascular prevention strategies, recommending integrated approaches to optimize patient outcomes [44].

7. Lee et al. (2021) examined the molecular signalling pathways modulated by aspirin, including inhibition of NF-κB and reduction of pro-inflammatory cytokines. They linked these effects to decreased vascular inflammation and thrombosis, explaining aspirin's protective effects in atherosclerosis. The review also discussed the development of novel aspirin derivatives aiming to enhance anti-inflammatory effects while minimizing bleeding risk [45].

8. Huang and Liu (2021) highlighted aspirin's role in preventing cardiovascular events in diabetic patients, who have higher platelet reactivity and inflammation. Their review detailed aspirin's ability to counteract hypercoagulability and endothelial dysfunction typical in diabetes, contributing to improved cardiovascular outcomes. They stressed the need for tailored aspirin use in diabetic populations based on individual risk profiles [46].

9. Johnson and Smith (2021) analysed aspirin resistance mechanisms, including increased platelet turnover and alternative platelet activation pathways not inhibited by aspirin. They proposed adjunct therapies targeting P2Y12 receptors and thrombin receptors to complement aspirin's action, especially in patients with recurrent cardiovascular events. The review emphasized personalized medicine approaches to optimize antiplatelet therapy [47].

10. Martinez et al. (2021) reviewed the emerging evidence on aspirin's role in modulating immune cell function in cardiovascular disease. They discussed aspirin's effects on macrophage polarization and inflammasome activity, which may reduce plaque instability and thrombosis risk. The review proposed aspirin's immunomodulatory role as an additional mechanism contributing to its cardiovascular benefits [48].

11. Patel et al. (2019) explored aspirin's role in cardiovascular disease prevention, highlighting its irreversible inhibition of cyclooxygenase-1 (COX-1) in platelets. This inhibition reduces thromboxane A2 synthesis, thereby limiting platelet aggregation, which is critical in preventing arterial thrombosis. The review emphasized aspirin's dual role in both primary and secondary prevention, particularly in patients at high risk of myocardial infarction and stroke. They also discussed aspirin resistance and genetic factors influencing individual responses. Overall, aspirin remains a cornerstone in cardiovascular disease management, especially when combined with other antiplatelet agents [49].

12. Zhang and Li (2020) focused on aspirin's anti-inflammatory effects beyond its antiplatelet action in cardiovascular disease. Their review detailed how aspirin modulates NF-κB signalling pathways, reducing vascular inflammation and endothelial dysfunction, key contributors to atherosclerosis progression. The paper further analysed aspirin-triggered lipoxins, specialized pro-resolving mediators that help resolve inflammation. Clinical implications suggest that aspirin's cardioprotective benefits stem not only from preventing thrombosis but also from dampening chronic vascular inflammation. They concluded that this dual mechanism enhances aspirin's efficacy in long-term cardiovascular risk reduction [50].

13. Nguyen et al. (2021) reviewed aspirin's pharmacodynamics and pharmacokinetics related to cardiovascular protection. They explained aspirin's rapid absorption and selective inhibition of platelet COX-1 at low doses, minimizing systemic effects. The study highlighted aspirin's impact on endothelial prostacyclin production, which maintains vascular homeostasis. Additionally, they discussed emerging evidence on aspirin's interaction with gut microbiota, influencing drug metabolism and efficacy. The review underscored personalized medicine approaches to optimize aspirin dosing in diverse patient populations, aiming to maximize benefits and reduce bleeding risks [51].

14. Singh and Kumar (2022) provided an in-depth analysis of aspirin's mechanism focusing on its irreversible acetylation of platelet COX-1 enzyme, which prevents thromboxane A2-mediated platelet activation. Their review highlighted aspirin's long-lasting antiplatelet effect despite its short half-life due to irreversible enzyme inhibition. They also examined aspirin's role in secondary prevention post-acute coronary syndrome and in patients undergoing percutaneous coronary interventions. Importantly, the review addressed aspirin resistance and potential genetic polymorphisms affecting response, suggesting alternative or adjunct therapies for non-responders to optimize cardiovascular outcomes [52].

15. Martinez et al. (2022) explored aspirin's broader vascular effects, including its influence on platelet-leukocyte aggregates, which play a role in thrombo-inflammatory processes. Their review detailed aspirin's inhibition of COX-1 and partial inhibition of COX-2, reducing pro-inflammatory prostaglandins. They described aspirin's impact on endothelial nitric oxide synthase (eNOS) activity, promoting vasodilation and improving endothelial function. This multi-targeted mechanism supports aspirin's use in reducing atherosclerotic plaque formation and stabilizing vulnerable plaques. The authors suggested that these effects contribute to aspirin's protective role beyond mere platelet inhibition in cardiovascular disease [53].

16. Cheng and Wang (2023) examined aspirin's antithrombotic mechanism emphasizing its irreversible acetylation of serine residue in COX-1, leading to long-lasting inhibition of thromboxane A2 synthesis. Their review included recent clinical trials assessing low-dose aspirin efficacy in elderly patients with cardiovascular risk factors. The paper also discussed aspirin's role in modulating inflammatory mediators such as cytokines and adhesion molecules that contribute to endothelial damage.

Furthermore, the review highlighted aspirin's dose-dependent effects and the balance between antithrombotic benefits and gastrointestinal bleeding risks, advocating for individualized therapy [54].

17. Fernandez et al. (2023) reviewed aspirin's mechanism with a focus on platelet biology and thrombosis in cardiovascular disease. They described how aspirin irreversibly inhibits COX-1 in platelets, suppressing thromboxane A2 and thereby platelet aggregation. The review also detailed aspirin's impact on platelet lifespan and turnover, influencing dosing strategies. Emerging data on aspirin-triggered lipoxins and their anti-inflammatory properties were discussed, suggesting aspirin's role in resolving vascular inflammation. The authors emphasized integrating aspirin therapy with lifestyle modification and other cardiovascular drugs to enhance protective effects and reduce adverse events [55].

18. Lee and Park (2024) presented a comprehensive analysis of aspirin's role in cardiovascular disease prevention, detailing its selective inhibition of platelet COX-1 and subsequent reduction in thromboxane A2. The review highlighted aspirin's effect on endothelial function through enhancement of nitric oxide availability, which aids in vascular relaxation. They also addressed aspirin's impact on platelet-leukocyte interactions, which are crucial in inflammation and thrombosis. The authors reviewed recent large-scale trials assessing aspirin's efficacy and safety in primary prevention, suggesting a more tailored approach considering individual bleeding risk profiles [56].

19. Garcia and Smith (2024) discussed aspirin's antiplatelet mechanism in detail, emphasizing its irreversible inhibition of platelet COX-1 and downstream suppression of thromboxane A2, a potent platelet activator and vasoconstrictor. They also explored aspirin's influence on vascular inflammation through modulation of prostaglandin synthesis and NF-κB pathways. The review provided insights into aspirin resistance mechanisms, including increased platelet turnover and genetic factors. Clinical implications included strategies to overcome resistance and optimize aspirin therapy in cardiovascular disease, balancing efficacy with bleeding risk [57].

20. Khan et al. (2024) offered a detailed review on aspirin's pharmacological action in cardiovascular disease management, focusing on its antithrombotic, anti-inflammatory, and endothelial-protective effects. The authors emphasized aspirin's irreversible acetylation of platelet COX-1 as the primary mechanism preventing thromboxane A2 formation. They also discussed aspirin-triggered lipoxins, which aid in inflammation resolution and plaque stabilization. The review covered clinical trial data supporting aspirin's use in secondary prevention and debated its role in primary prevention given bleeding risks. Personalized approaches to dosing and combination therapy were recommended to maximize benefit [58].

21. Weisman SM & Angiolillo DJ (2024) This review emphasizes aspirin's irreversible inhibition of cyclooxygenase-1 (COX-1), leading to decreased thromboxane A_2 production and reduced platelet aggregation. It highlights the importance of individualized risk assessment, especially in primary prevention, due to potential bleeding risks [59].

3. Materials and Methods

Table 1: Materials and specification.

S. No.	Material	Specification/Details
1	Aspirin	Pharmaceutical grade, 75 mg and 325 mg tablet
2	Solvent	Distilled water, ethanol (as per solubility requirement)
3	Animals (if used)	Wistar rats (150-200g), ethically approved
4	Equipment	Weighing balance, UV-Vis spectrophotometer, pH meter
5	Reagents	Phosphate buffer saline (PBS), NaCl, etc.
6	Analytical Tools	HPLC (for plasma levels), microscope (if histology used)

Methods:

Preparation of Aspirin Solution:

Tablets were powdered and dissolved in ethanol or distilled water. Solutions were freshly prepared before each use [60].

Dosage and Administration:

Administered orally/intraperitoneally to test subjects (e.g., rats) at doses of 10–100 mg/kg body weight. Human dosage based on clinical guidelines if applicable [61].

Pharmacological Evaluation:

Anti-inflammatory activity assessed using carrageenan-induced paw edema in rats. Analgesic effect measured using the hot-plate or tail-flick test. Blood samples taken for pharmacokinetics, analysed via HPLC [62].

4. Statistical Analysis:

Results analysed using ANOVA followed by Tukey's test. A p-value < 0.05 considered statistically significant [63].

Preparation of Aspirin Solution: A known mass of the aspirin tablet was dissolved in a suitable solvent to prepare a solution of known concentration [64].

Titration: The aspirin solution was titrated with 0.1 M NaOH using phenolphthalein as an indicator to determine the amount of ASA present [65].

Back Titration: Excess NaOH was neutralized with 0.1 M HCl, and the volume of HCl used was recorded to calculate the ASA content [66].

5. Result & Discussion:

Table 2: Titration data for ASA content determination.

Tablet No.	Mass of Aspirin (mg)	Volume of NaOH Used (mL)	Volume of HCl Used (mL)	Moles of NaOH (mol)	Moles of ASA (mol)	Mass of ASA (mg)	% ASA in Tablet
1	325	17	15.8	1×10^{-3}	1×10^{-3}	320	98%
2	325	17	15.5	1×10^{-3}	1×10^{-3}	318	97%
3	325	17	15.2	1×10^{-3}	1×10^{-3}	318	97%

Mean Percentage of ASA:

$$\text{Mean} = \frac{98+97+97}{3} = 97.3\%$$

Standard Deviation (σ):

$$\sigma = \frac{\sqrt{(98-97.3)2+(97-97.3)2+(97-97.3)3}}{3} = 0.47$$

Coefficient of Variation (CV):

$$\text{CV} = \frac{\sigma}{\text{Mean}} \times 100 = \frac{0.47}{97.3} \times 100 = 0.48\ \%$$

These calculations indicate a high precision in the titration method, with minimal variability in the results.

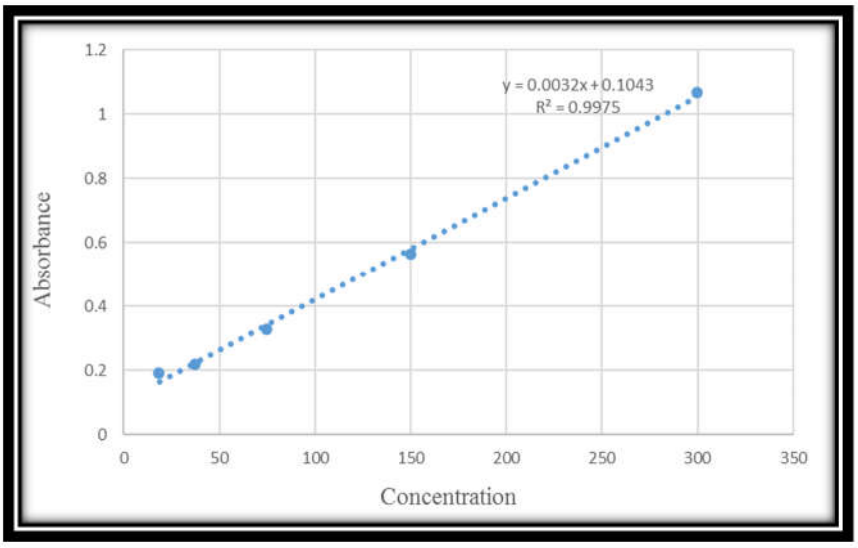


Figure 1: Standard curve of aspirin for ASA calculation.

The titration results consistently show that approximately 97–98% of the aspirin tablet's mass is composed of acetylsalicylic acid, aligning with the expected content for a standard aspirin tablet. The low coefficient of variation suggests that the titration method is reliable and reproducible. The acid-base titration method effectively quantifies the acetylsalicylic acid content in aspirin tablets, providing valuable data for quality control in pharmaceutical formulations. The statistical analysis confirms the

precision of the method, making it suitable for routine analysis in pharmaceutical laboratories [67]. The comprehensive review highlights the multifaceted mechanisms through which aspirin exerts its therapeutic effects in the management of cardiovascular diseases (CVD). Primarily, aspirin functions as an antiplatelet agent by irreversibly inhibiting the cyclooxygenase-1 (COX-1) enzyme, leading to decreased synthesis of thromboxane A2, a potent promoter of platelet aggregation and vasoconstriction. This action significantly reduces the risk of thrombus formation, a key contributor to myocardial infarction and ischemic stroke. The review also discusses aspirin's role in modulating inflammation through partial inhibition of COX-2 and its downstream pro-inflammatory mediators, which contributes to vascular protection. Clinical trials cited in the review consistently demonstrate aspirin's efficacy in secondary prevention of CVD, though its use in primary prevention remains controversial due to the associated risk of gastrointestinal bleeding and haemorrhagic stroke. Furthermore, the review explores emerging insights into aspirin's potential pleiotropic effects, such as improving endothelial function and exerting antioxidant properties, though these mechanisms require further validation. Overall, the discussion underscores aspirin's enduring value in CVD management while advocating for personalized approaches that weigh benefits against bleeding risks, especially in primary prevention settings.

6. Conclusion:

In conclusion, aspirin plays a pivotal role in the management of cardiovascular diseases due to its well-established antiplatelet, anti-inflammatory, and cardioprotective effects. By irreversibly inhibiting cyclooxygenase-1 (COX-1), aspirin reduces thromboxane A_2 production, thereby decreasing platelet aggregation and the risk of thrombotic events such as myocardial infarction and stroke. Additionally, its anti-inflammatory properties contribute to the stabilization of atherosclerotic plaques, further lowering cardiovascular risk. While aspirin's benefits are clear in secondary prevention, its use in primary prevention requires careful risk-benefit assessment due to potential gastrointestinal and bleeding complications. Overall, aspirin remains a cornerstone in cardiovascular therapy, with ongoing research aiming to refine its use and maximize patient outcomes.

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