International Journal of Pharmacy and Pharmaceutical Research (IJPPR)
Volume 31, Issue 5, May 2025 ijppr.humanjournals.com ISSN: 2349-7203

A Comprehensive Review on Aspirin

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Received: 2025-04-28 Revised: 2025-05-15 Accepted: 2025-05-20

ABSTRACT

Aspirin can prevent cerebro-cardiovascular diseases, but it also has adverse effects, especially on the kidneys and gastrointestinal tract. Specifically, a recent advancement in endoscopy has revealed that aspirin-induced damage to the mucosa of the small intestine is much greater than previously believed. However, the mechanism behind this phenomenon is still unknown. Moreover, there isn't a reliable preventative. First, we examined the cytotoxic effects of high dosages of aspirin on a rat intestinal epithelial cell line. We found that aspirin promoted the death of epithelial cells, raised the formation of reactive oxygen species, and drastically decreased the expression of heat shock protein 70. These events were halted by the upregulation of heat shock protein 70 expression. Next, we investigated the impact of epithelial cell permeability on a reduced aspirin content. Researchers discovered that aspirin dramatically raised epithelial permeability, reduced tight junction protein expression, and raised reactive oxygen species generation. An antioxidant suppressed these phenomena. In conclusion, we used an in vivo model to examine the impact of intestinal mucus on aspirin-induced mucosal damage and discovered that mucus inhibited a high concentration of aspirin-induced mucosal damage. Mucus-increasing therapy may be helpful in reducing aspirin-induced small intestine mucosal damage, according to research on long-term aspirin users.

Keywords: small intestine, permeability, apoptosis, mucus introduction, and acetyl salicylic acid.

INTRODUCTION

Aspirin has long been thought to be helpful in lowering the risks of cancer and heart disease when taken in the right dosage over an extended period of time. However, some people experience unfavorable side effects including bleeding and stomach pain after using aspirin.

Therefore, the potential harm from side goods must be weighed against the implicit advantages of protection. Each person may have a distinct equilibrium. When aspirin is used as primary prevention, that is, when people who are not yet suffering from cardiovascular disease (CVD) or cancer use it, it is especially critical to be aware of the risk of side effects. The purpose of this report is to locate the most recent scientific evidence supporting this and to Summarize this literature by examining the situation of adverse effects from the preventive use of aspirin in individuals who are free of cancer and cardiovascular disease in systematic reviews, meta-analyses, and randomized controlled trials (RCTs).

Over the past 25 years, research on the use of precautionary aspirin for the primary prevention of CVD has been conducted. Since the first RCT on this topic was published in 1988, eight more have been published, the most recent being in 2010. This problem is still being addressed by continuing trials. At the moment, focus has also been on the potential role that preventative aspirin may have in the primary prevention of cancer. To illustrate the implicit impact of successful primary prevention strategies, we first provide a brief overview of the incidence of cancer and CVD in the UK. Additionally, we outline the potential mechanisms of action through which aspirin may exert its natural benefits. Finally, we highlight a few of the challenges faced by researchers attempting to examine the advantages of aspirin in primary forestallment.

An explanation of the health issue (mostly the prevention of cancer and cardiovascular disease)

In terms of morbidity, mortality, and expense, cancer and cardiovascular disease place a significant burden on the people of the United Kingdom. These burdens are significantly impacted implicitly by primary forestallment methods. According to some

recommendations and research, taking aspirin on a regular basis may help with this. However, some people who use aspirin have unwanted side effects that can occasionally be fatal. The purpose of this brief report is to assess and analyze the relevant evidence.

The National Institute for Health and Care Excellence's (NICE) definition of main forestallment for CVD is interpreted as follows in this report. "... interventions aimed at assisting cardiovascular (CV) events in individuals who do not have clinical evidence of CVD." One Primary prevention of cancer may be described similarly; in the sentence below, we utilize an equivalent description by replacing "CVD" with "cancer."

Continuing Education Activity

Since the early 1900s, salicylates have been accessible. This effort describes salicylic acid's recommendations, mode of action, administration methods, significant side effects, contraindications, and monitoring so that healthcare professionals can guide case remedies in treating designated conditions as part of the interprofessional team. Items determine the salicylic acid's mode of action. Describe salicylic acid's implicit negative effects. Go over the many different conditions for which salicylic acid is prescribed. Describe the importance of improving interprofessional platoon care collaboration in order to ensure the safe administration of salicylic acid. Get free multiple-choice questions related to this topic.

Recommendations

Salicylates have been inferred from the dinghy of the willow tree. It has long been known that the Sumerians employed willow tree-derived pain relief techniques. as if 4000 times had passed. Hippocrates used it to treat fever and discomfort. In fact, he used the tea he made from it to help with the discomfort during parturition.

Reverend Edward Stone conducted the first clinical experiment of its sort in 1763 to examine the effectiveness of willow dinghy greasepaint in curing fever. The products of the willow dinghy greasepaint were examined for acute rheumatism around a century later.

Professor Johann Buchner used the Latin name for willow, salicin, in 1828. In 1829, Henri Leroux separated it in a crystalline form and employed it to cure rheumatism. The Heyden Chemical Company was the first to commercially produce salicylic acid in large quantities in the 1800s. A modified interpretation known as acetylsalicylic acid was not registered until 1899 and sold by Bayer under the Aspirin trade name.

In fact, its true mode of action was unknown until the late 1970s, despite the fact that it had been available since the early 1900s.

Here are some recommendations about the use of aspirin:

Pectoris angina

The prevention of angina pectoris

Spondylitis with ankylosing

lowering of cardiovascular risks

Colorectal cancer

A fever

Stroke ischemic

Avoiding ischemic stroke

Myocardial ischemia

Prevention of myocardial infarction

Osteoarthritis Pain

Revascularization techniques Preventive measures

It is rheumatoid arthritis.

Lupus erythematosus systemic

Method :

Procedure for Preparation Acetyl Salicylic Acid (C9H8O4) :

1. Put three grams of salicylic acid in an Erlenmeyer beaker.

2. Fill the beaker with 6 mL of acetic anhydride and 5 to 8 drops of 85 phosphoric acid.

3. After mixing the results, leave the beaker in warm water for fifteen seconds.

4. Drop by drop, add 20 drops of cold water to the warm result. (This eliminates the unnecessary acetic anhydride.)

5. To speed up crystallization and cool the admixture, keep the beaker in an ice bath.

6. Use a Buckner channel to pour the admixture when the crystallization process is finished.

7. To reduce product loss, wash the chargers with ice-cold water.

8. The product can be purified by recrystallization.

Add 10 milliliters of ethanol to the chargers.

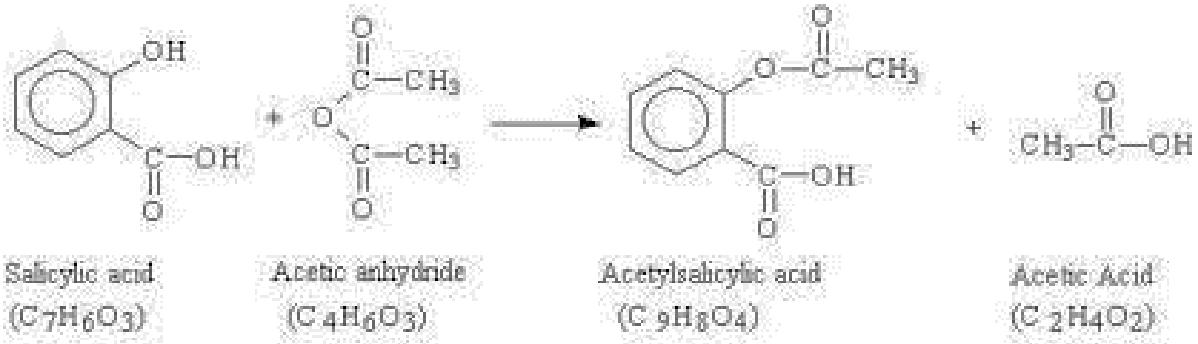
9. To dissolve the charges, stir the mixture.

10. As the result cools, chargers are formed by pouring 25 ml of warm alcohol into it and covering it.

11. Keep the teacup in an ice bath for recrystallization after the crystallization process starts.
After the teacup's contents are drained out, suction filtering is used.

12. The chargers are kept on dry paper to eliminate any excess water.

13. Verify that the melting point of acetylsalicylic acid is 135 °C.



Medium of Action :

A cyclooxygenase-1 (COX-1) asset is aspirin. It modifies the cyclooxygenase-2 (COX-2) enzyme activity. Aspirin list is irreversible, in contrast to other NSAIDs (ibuprofen/naproxen), which bind to this enzyme reversibly. (5) It also prevents platelet aggregation by irreversibly blocking thromboxane A2 on platelets.

Researchers speculate that the arachidonic acids are transferred into the lipoxygenase route as a result of the COX pathway being blocked. When prostaglandin-endoperoxide synthase (PTGS2), also known as COX-2, is modified, lipoxins are produced, the majority of which have anti-inflammatory properties to them. Aspirin-touched off lipoxins, aspirin-touched off resolving, and aspirin-touched off maresins are the names of these composites.

Administration :

There are three ways to deliver aspirin:

1. orally, rectal, and intravenously (IV).

2. It comes in a variety of boluses;

3. the smallest, known as a baby aspirin, is 81 milligrams. 325 mg and 500 mg tablets.

4. Tablet with delayed release Chewable: 81 mg, 325 mg, 500 mg, 650 mg.

5. intravenous: 250 mg, 500 mg; suppository: 60 mg, 120 mg, 200 mg, 300 mg, 600 mg;

Pharmacokinetics :

The expression status determines whether aspirin is absorbed from the gastrointestinal (GI) tract. Unlike tablets, liquid medications are quickly absorbed when taken. Salicylic acid is produced when it is hydrolyzed. Salicylic acid's window for remediation is limited. However, if kept within that certain range, it has the appropriate anti-inflammatory action.

Because of the small intestine's location, aspirin immersion is sensitive to pH. For the same pH range, immersion moves more quickly through the small intestine than the stomach. Aspirin's intestinal immersion is lower than the emulsion's gastric immersion at pH 3.5 or 6.5. At pH 6.5, aspirin is not absorbed by the stomach.

Two mechanisms lead to the removal of salicylate: the production of salicylic phenolic glucuronide and salicyluric acid. Renal clearance of salicylic acid can be raised by increasing the pH of the urine. Antacids, for example, enhance the pH of the urine, which can increase renal concurrence. It is able to pass through the placental-blood hedge. Bone milk also expresses it.

Pharmacodynamics :

160–325 mg of aspirin can be used to achieve almost 90 percent COX inhibition. These products typically have a shelf life of 7–10 days, which is comparable to a platelet's lifespan. It is possible to suppress prostacyclin by using sophisticated boluses. The blood artery endothelial cells are where this inhibition takes place.

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How to cite this article:
Mr. Rachit Shukla et al. Ijppr.Human, 2025; Vol. 31 (5): 332-336.
Conflict of Interest Statement: All authors have nothing else to disclose.