

Prostaglandins and the Cardiovascular System

Emanuela Ricciotti¹, Garret A. FitzGerald²

*Do not seek to follow in the footsteps of the wise;
seek what they sought.*

—Basho

Prostaglandins (PGs) are low-abundance oxylipins that act as autocrine and paracrine signaling molecules with pleiotropic effects in the human body. PGs and thromboxane A₂ (TxA₂), collectively known as prostanoids, are metabolites derived from the enzymatic catabolism of arachidonic acid (AA). PLA₂ (phospholipase A₂) releases AA from membrane phospholipids, and free AA can then be metabolized by the COX (cyclooxygenase) enzymes: COX-1, which is commonly constitutively expressed, and COX-2, which is more commonly inducible. This reaction generates PGH₂, an unstable intermediate that is further metabolized by tissue-specific synthases and isomerases to PGD₂, PGE₂, PGF_{2α}, and prostacyclin (PGI₂). PGs play an important role in driving the inflammatory response. In inflamed tissues, their biosynthesis is markedly increased, contributing to the hallmark signs of acute inflammation, while their role in the resolution of inflammation remains less established.¹ The recent COVID-19 pandemic has reemphasized the immunomodulatory role of secretory PLA₂-driven PGs and related oxylipins in the inflammatory and thrombotic response to infection.²

NSAIDs, which reduce PG formation by inhibiting the activity of COX enzymes, are effective in alleviating pain, inflammation, and fever and are one of the most widely used classes of drugs worldwide.

There has been substantial progress in understanding the biology, regulation, and pathophysiological effects of PGs since their discovery in the early 1930s, including the elucidation of their biochemical pathways, the identification of their receptors, their role in health and disease, and the development of drugs that disrupt or

augment their formation or action. This article provides an overview of the discovery of PGs, discusses current drugs used to target this pathway, summarizes seminal pathophysiological findings in cardiovascular biology, and previews upcoming challenges and opportunities in PG research.

DISCOVERY OF PGs

In the early 1930s, Kurzrock and Lieb observed that human seminal fluid caused either stimulation or relaxation of isolated human uterine strips. In 1935, Ulf von Euler and Maurice Goldblatt independently isolated a lipid-soluble acid from seminal fluid and the reproductive glands of dogs, which von Euler named PG, believing that it was produced by the prostate gland.

By the 1940s and 1950s, researchers discovered PGs could be produced by tissues other than the prostate and had broader biological effects, including regulating gastrointestinal function and uterine contractions. In the 1950s, the molecular structure of PGs was identified, and it was found that they are derived from AA. In 1969, Corey synthesized PGE₂ and PGF_{2α}. In the early 1970s, Samuelsson uncovered how AA is metabolized into PGs, and Bergström helped isolate and characterize them using mass spectrometry. In the 1970s, Vane used superfusion bioassays to discover that aspirin inhibits COX activity and PG formation and together with Bunting and Moncada, discovered that PGI₂ causes vasodilation and inhibits platelet aggregation. Vane's research was pivotal to understanding the role of PGs in inflammation. The discovery of the mechanism of action of aspirin led to the development of NSAIDs.³ In the 1980s, Nugteren and his collaborators isolated and characterized the PG endoperoxidases, which are key intermediates in PG biosynthesis.⁴ Also in the 80s, Vanhoutte and his colleagues

Key Words: arachidonic acid ■ cyclooxygenase 2 ■ prostacyclin ■ prostaglandins ■ thromboxane A₂

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configure ipv4 and security settings 25 points part 4 power cycle all devices 5 points