



### Anti-Inflammatory Salicylates: Their Properties and Actions

The group of chemicals that has salicylic acid as the parent compound is often referred to generically as “salicylates”. Sodium salicylate and acetylsalicylic acid (aspirin) are two salicylates that are part of a larger group of anti-inflammatory agents known as non-steroid anti-inflammatory drugs (NSAIDs). Most NSAIDs act to relieve inflammation by reducing local fluid accumulation or edema, heat, and pain in injured or infected outlying tissues. They also act on the body’s central regulatory systems to reduce pain and fever (Lees and May, 1992 p. 847).

Sodium salicylate and aspirin have comparable therapeutic properties and potencies (Osol and Pratt, 1973 p. 1075). The United States Pharmacopeal Convention (USP) treats sodium salicylates and aspirin as one in its description of the therapeutic effects and proper uses of the drugs, while specifically noting where minor distinctions occur (USP, 1990 p. 1257). *The US Dispensatory* also recognizes that the anti-inflammatory properties of sodium salicylate and aspirin are the same (Osol and Pratt, 1973 p. 160), with some slight exceptions to be discussed later. Sodium salicylate and aspirin are both converted to the active metabolite salicylic acid when exposed to the hydrolysis of the gastrointestinal tract (May and Lees, 1996 p. 231; and Lees and May, 1992 p. 851-852).

Sodium salicylate and aspirin both work to relieve the inflammation that results from a cascade of events that occur because of injury or infection. The body produces prostaglandins that are responsible for the pain, fever and edema associated with inflammation. Prostaglandin synthesis depends on prostaglandin synthetase (more recently known as cyclo-oxygenase) enzymes. Live animal studies have been used to demonstrate that sodium salicylate, aspirin, and other NSAIDs reduce inflammation by inactivating prostaglandin synthetase and preventing the development of prostaglandins (Higgs, et al., 1976 p. 105; and Lees and May 1992 p. 846). Tests using tissue extracts in test tubes give different results because a test tube system does not allow the sodium salicylate to be converted into the active metabolite as occurs in live systems (Higgs, et al., 1976 p. 108).

A few noteworthy differences between sodium salicylate and acetylsalicylic acid have been reported in the literature. First, sodium salicylate does not prolong bleeding time as aspirin does (Osol and Pratt, 1973 p. 162). This is due to the acetyl group of aspirin irreversibly acetylating and inactivating the prostaglandin synthetase of platelets. New

platelets must be formed after aspirin use for full clotting to be restored (May and Lees, 1996 p. 231; and Plumb, 1995 p. 55). Sodium salicylate does not contain acetyl groups, and consequently its effect on platelets is not irreversible. Second, aspirin is subject to degradation when exposed to moisture (Plumb, 1955 p. 55; and Budavari et al., 1989 p. 134), where sodium salicylate has been demonstrated to be stable for periods in excess of one year in aqueous conditions (Izard, unpublished data). Finally, the solubility of sodium salicylate in water is very high, with one gram dissolving in slightly less than 1 ml (Budavari et al., 1989 p. 1367). Aspirin is as much as 300 times less soluble, with one gram of aspirin dissolving in 300 ml of water (Budavari et al., 1989 p. 134).

Budavari, S., M.J. O'Neil, A. Smith, and P.E. Heckelman, 1989. The Merck Index, 11<sup>th</sup> ed. Merck & Co. Rahway, NJ.

Higgs, G.A., E.A. Harvey, S.H. Ferreira, and J.R. Vane, 1976. The effects of anti-inflammatory drugs on the production of prostaglandins *in vivo*. Pages 105-109 *in*: Advances in Prostaglandin and Thromboxane Research, Vol. 1. B. Samuelsson and R. Paoletti, ed. Raven Press, NY.

Lees, P. and S.A. May, 1992. Inflammation and anti-inflammatory drugs. Pages 843-863 *in*: Bovine Medicine. A.H. Andrews, R.W. Blowey, H. Boyd, and R.G. Eddy, ed. Oxford Blackwell Scientific Publications, Cambridge, MA.

May, S.A. and P. Lees, 1996. Nonsteroidal anti-inflammatory drugs. Pages 223-237 *in*: Joint Disease in the Horse. C.W. McIlwraith and G.W. Trotter, ed. W.B. Saunders Co. Philadelphia, PA.

Osol, A., R. Pratt, and A.R. Gennaro, 1973. The United States Dispensatory, 27<sup>th</sup> ed. J.B. Lippincott Co. Philadelphia, PA.

Plumb, D.C., 1995. Veterinary Drug Handbook, 2<sup>nd</sup> ed. Pages 55-59. Iowa State University Press. Ames, IA.

United States Pharmacopeia, Drug Information for the Consumer, 1990. Consumers Union. Mount Vernon, NY.