Conclusion

The efficacy of the new drugs is not greater than that of the NSAIDs. However, if the current large outcome studies of celecoxib and rofecoxib confirm the reduced gastrointestinal toxicity then these drugs will increase the options for the treatment of arthritis.

REFERENCES

1. Vane JR. Inhibition of prostaglandin synthesis as a mechanism of action for aspirin-like drugs. Nature 1971;231:232-5.
2. Wolfe MM, Lichtenstein DR, Singh G. Gastrointestinal toxicity of nonsteroidal antiinflammatory drugs. N Engl J Med 1999;340:1888-99.
3. Masferrer JC, Seibert K, Zweifel B, Needleman P. Endogenous glucocorticoids regulate an inducible cyclooxygenase enzyme. Proc Natl Acad Sci USA 1992;89:3917-21.
4. Brooks P, Emery P, Evans JF, Fenner H, Hawkey CJ, Patrono C, et al. Interpreting the clinical significance of the differential inhibition of cyclooxygenase-1 and cyclooxygenase-2. Br J Rheumatol 1999;38:779-88.
5. Warner TD, Giuliano F, Vojnovic I, Bukasa A, Mitchell JA, Vane JR. Nonsteroid drug selectivities for cyclo-oxygenase-1 rather than cyclo-oxygenase-2 are associated with human gastrointestinal toxicity: a full in vitro analysis. Proc Natl Acad Sci USA 1999;96:7563-8.
6. Simon LS, Weaver AL, Graham DY, Kivitz AJ, Lipsky PE, Hubbard RC, et al. Anti-inflammatory and upper gastrointestinal effects of celecoxib in rheumatoid arthritis: A randomized controlled trial. JAMA 1999;282:1921-8.
7. Langman MJ, Jensen DM, Watson DJ, Harper SE, Zhao P-L, Quan H, et al. Adverse upper gastrointestinal effects of rofecoxib compared with NSAIDs. JAMA 1999;282:1929-33.
8. Dubois RN, Abramson SB, Crofford L, Gupta RA, Simon LS, Van De Putte LB, et al. Cyclooxygenase in biology and disease. FASEB J 1998;12:1063-73.
9. Giardiello FM, Hamilton SR, Krush AJ, Piantadosi S, Hylind LM, Celano P, et al. Treatment of colonic and rectal adenomas with sulindac in familial ademonatous polyposis. N Engl J Med 1993;328:1313-6.
10. Peterson WL, Cryer B. COX-1-sparing NSAIDs – Is the enthusiasm justified? JAMA 1999;282:1961-3.

FURTHER READING

Hawkey CJ. Cox-2 inhibitors. Lancet 1999;353:307-14

Professor Brooks has acted as a consultant to Searle and is on advisory boards for Merck Sharpe and Dohme.

(A summary of all clinical trials of the COX-2 inhibitors appears on the National Prescribing Service web site at www.nps.org.au under Topics)

Self-test questions

The following statements are either true or false (answers on page 47)

1. The efficacy of COX-2 inhibitors is greater than the efficacy of non-steroidal anti-inflammatory drugs.
2. It is currently unknown if an inhibitor with high selectivity for COX-2 will be safer than a less selective COX-2 inhibitor.

Your questions to the PBAC

Brand premiums

A number of years ago, benchmark pricing was introduced to the Pharmaceutical Benefits Schedule, whereby a drug company would be allowed to introduce a brand surcharge for their particular product. My understanding of the operation of this scheme was that it would follow the guidelines of the Australian Competition and Consumer Commission with respect to collusive pricing and price fixing. This would not appear to be the case, as many products today are obviously manufactured by the same company, their logo and name appearing on both the generic and premium-priced product (despite having a 'different' manufacturing code on the Pharmaceutical Benefits Schedule). An explanation of how brand price premiums are allowed, and calculated, would be appreciated.

Michael D. Rumpff
Pharmacist
Sale, Vic.

The Secretary of the Pharmaceutical Benefits Pricing Authority comments:

The Brand Premium Policy was introduced in December 1990 to reduce price controls where possible by allowing pharmaceutical suppliers to set their own price on multi-branded and therapeutically interchangeable brands listed on the Pharmaceutical Benefits Scheme, provided one brand was available at the subsidised price. This also encourages the development of the generic pharmaceutical industry in Australia.

Under the policy, suppliers of multi-branded items are able to set their own prices at a level they think the market will bear. At the same time, prescribers, pharmacists and patients can decide whether it is necessary to pay more for a particular brand when a cheaper equivalent and therapeutically interchangeable brand is available.

As the brand premium is not a government charge, it does not count towards a patient's safety net. The premium arises from the supplier's price setting and the majority of it goes to the supplier, with wholesalers and pharmacists receiving a percentage.

Under the competitive environment, it is up to the sponsor of the product to set the price at which it sells its brand. The government only sets the subsidised price. The pricing freedom that applies is similar to that of many other commodities such as food, clothing and cosmetics.

As of February 2000 there were 236 benefit items with a brand premium that could be therapeutically interchanged. The average brand premium was $1.45 and premiums ranged from $0.23 to $43.28.