Generic substitution in allergic rhinitis

To the Editor: We would like to clarify the paragraph on generic substitution (paragraph 9) in the guideline on allergic rhinitis published as a supplement to the *SAMJ* of December 2006.[1]

The authors of the guideline state that generic substitution of drugs should not be done, unless trials showing clinical equivalence have been undertaken. From a regulatory perspective, antihistamines and intranasal corticosteroids are not included in the Medicines Control Council (MCC)'s list of non-substitutable medicines[2] and this recommendation would therefore be incorrect.

We know of no randomised controlled clinical trials comparing innovator and therapeutically equivalent generic products in the treatment of allergic rhinitis. The statement in the guideline is therefore not evidence-based or substantiated. In the absence of evidence the Allergy Society of South Africa is unable to make a recommendation either way, and it will therefore be up to health care professionals, health care funders and patients themselves to decide which product is best suited to the individual. We believe that generic products that are therapeutically equivalent have enabled greater availability of more cost-effective therapy to patients with allergic rhinitis.

We would also like to point out that reference 191 should read 'The Medicines and Related Substances Act, Act 101 of 1965, as amended, Section 22F'.

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1. Potter PC, Carte G, Davis G, *et al*. Clinical Management of Allergic Rhinitis – the Allergy Society of South Africa Consensus Update. *S Afr Med J* 2006; 96(12, part 2): 1269-1272.
2. Medicines Control Council. Generic substitution December 2003 version 1. www.mccza.com (accessed 19 March 2007).

Plea for privacy

To the Editor: It is surprising that the South African Medical Association (SAMA) has 'called on the medical profession, media, political parties and the public to respect the privacy of the Minister of Health, and to stop speculating about her health and the cause of her medical condition' (*Medigram* vol 15, No. 4, 16 March 2007).

Compassion is, of course, due to all who have serious illness, and the seriousness of the minister's illness makes this particularly the case. However, requests for privacy from enquiry into the cause and sequelae of her illness raise a different aspect.

Dr Manto Tshabalala-Msimang elected to go into public office and *ipso facto* expose herself to public scrutiny. Further, as a Minister of Government she became a public servant, and the public has a right to know whether their employee is capable of fulfilling the demands of office.

Unfortunately the Minister has shown herself to be less than forthright regarding her illness, and the impression is that she sought to cover up its cause and effects. In these circumstances her employers (the electorate) have valid cause to speculate about her illness. Speculation will be inevitable and legitimate until she clarifies her incapacity.

If Dr Manto Tshabalala-Msimang had wished to preserve her privacy, the direction open to her was to resign early in her illness, and allow an unimpaired replacement to take office. This would have allowed her to control her privacy, as well as enhancing the esteem in which one assumes she would want to be held.

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Collapsing focal segmental glomerulosclerosis as a possible complication of valproic acid

To the Editor: Long-term use of valproic acid (VPA) leads to multiple organ damage, including tubulointerstitial nephritis.[1] Collapsing focal segmental glomerulosclerosis (CFSGS) associated with VPA has never been reported. Here we report on a case in which such an association appears highly probable.

In October 1995, a 35-year-old man was admitted to the nephrology unit (Lapeyronie Hospital, Montpellier, France) for oedema of the lower limbs. His history was of epilepsy since 1989, treated with VPA, and chronic renal failure (glomerular filtration rate (GFR) 34.14 ml/min in April 1995). On admission, clinical examination showed blood pressure 160/110 mmHg, weight 68.4 kg and oedema of the lower limbs. Acute abdominal pain occurred during hospitalisation. Laboratory examination showed the following findings: GFR 20.22 ml/min, albumin 22 g/l, and proteinuria 4.85 g/d. Other tests were normal, including VPA blood level and serology. Kidney biopsy (light microscopy) showed collapse of the glomeruli with severe vacuolisation of the podocytes (Fig. 1). Immunofluorescence study showed no immune deposits. Abdominal computed tomography (CT) scan revealed pancreatitis. As VPA toxicity was suspected, carbamazepine was substituted for VPA. Haemodialysis was started in April 1996.

CFSGS related to VPA toxicity has never been reported. CFSGS has been related to HIV.[2] In the present case chronic renal lesions might have evolved to CFSGS, but such a possibility has never been reported before.[3] The causal role of VPA appears