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Nephrite (jade) Occurrence in the Great Serpentine Belt of New South Wales, Australia

THIS communication is to report the presence in the Great Serpentine Belt of New South Wales, Australia[1–3] of a nephrite (jade) occurrence. Within the Great Serpentine Belt that outcrops discontinuously for more than 350 km in northern New South Wales[4] (Fig. 1) several economic min-

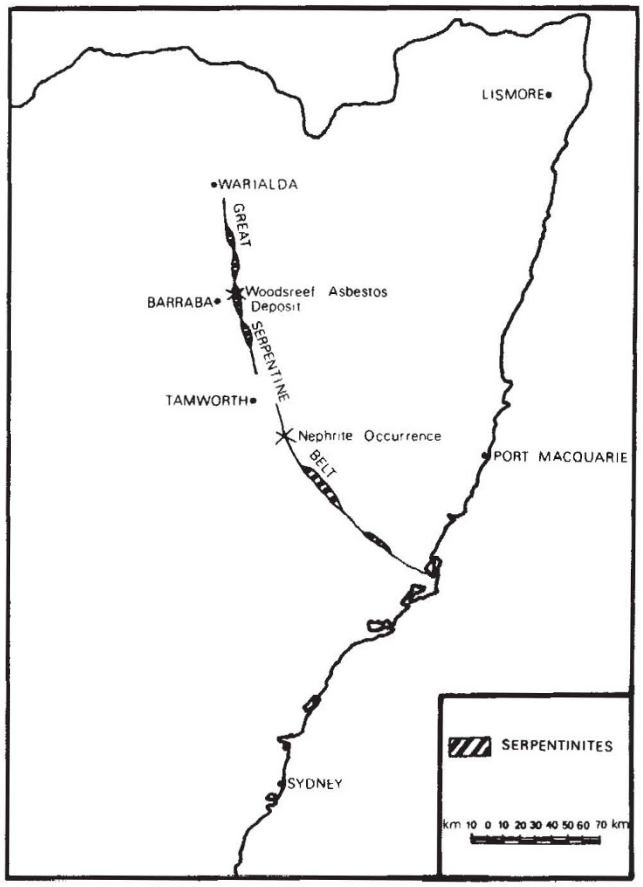

FIG. 1 Location of the Great Serpentine Belt in nothern New South Wales, Australia.

eral occurrences have been discovered and developed in recent years. The Woodsreef asbestos deposit, near Barraba, the largest deposit of its type in the Southern Hemisphere, is by far the best known of the recent discoveries.

The lensoidally shaped nephrite occurrence 24 km southeast of Tamworth may prove to be Australia's first economic deposit as high quality gemstones have been cut from the occurrence. Geological investigations are currently in progress to determine the size, overall grade and economic potential of this very interesting mineral occurrence.

J. J. HOCKLEY

"Meadvale",
Werris Creek, 2341
New South Wales, Australia

Received December 12, 1973.

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BIOLOGICAL SCIENCES

Transamination is a Major Pathway of L-Dopa Metabolism following Peripheral Decarboxylase Inhibition

WHEN L-3,4-dihydroxyphenylalanine (L-dopa) is administered to human subjects[1], the greater proportion is metabolised by decarboxylation, a small amount by O-methylation and traces only by transamination[2]. It seems likely that the benefit resulting from L-dopa therapy in Parkinsonian patients derives largely from the generation of one of its metabolites, dopamine, by decarboxylation within the central nervous system[3]. Less than 5% of administered L-dopa becomes available for this purpose: most of the dose is decarboxylated peripherally[4]. There is good reason to believe that the dopamine so formed is unable to penetrate the blood-brain barrier to any great extent[5], so that little of the original dose is available to contribute to the therapeutic response. By blocking peripheral decarboxylase, however, certain decarboxylase inhibitors[6,7], which are themselves unable to cross the blood-brain barrier to any significant extent[8], bring about an accumulation in the plasma of administered L-dopa, thus providing a higher concentration gradient to enter the central nervous system and allowing lower oral doses of L-dopa to be employed therapeutically[9].

It has so far been assumed that, apart from some increase in the formation of 3-O-methyldopa[10], peripheral degradation of L-dopa is largely prevented by the concomitant use of a decarboxylase blocking drug. We now present evidence that the combination of drugs results in a major diversion of L-dopa metabolism to a pathway where transamination substitutes for decarboxylation.

Thirteen patients with idiopathic Parkinsonism, taking part in a double blind comparison of L-dopa plus L-α-methyldopahydrazine (MK 486) against L-dopa alone[11], were investigated. Urine samples (24 h), preserved at −15° C with 6 N HCl (25 ml), were collected during the following treatment regimens: (1) before treatment (controls); (2) whilst receiving an optimal oral dose of L-dopa (1.0–5.75 g per 24 h; mean=3.5 g per 24 h) ('normal' dose experiment); and (3) whilst receiving a reduced dose of L-dopa (0.1–2.0 g per 24 h; mean=0.65 g per 24 h) plus oral MK 486 (300 mg per 24 h) ('low' dose L-dopa plus MK 486 experiment). Urine samples were also collected from (4) three of these patients during treatment with their own particular reduced dose of L-dopa (0.3–1.1 g per 24 h; mean 0.62 g per 24 h) employed during regime (3) but in the absence of MK 486 ('low' dose L-dopa experiment). Urinary *m*-hydroxyphenylacetic acid (*m*-HPAA), *p*-hydroxyphenylacetic acid (*p*-HPAA), homovanillic acid (HVA) and dihydroxyphenylacetic acid (DOPAC) were quantified as described previously[12,13], except that silylation was performed at 65° C for 1 h, using N,O-*bis* (trimethylsilyl)-acetamide (BSA, 0.2 ml) and dioxane (0.1 ml) containing *n*-eicosane (8 μg ml^{-1}) as internal standard. Urinary 4-hydroxy-3-methoxyphenyllactic acid (VLA), 4-hydroxy-3-methoxyphenylglycol (HMPG) and 4-hydroxy-3-