The lack of the signal for a primary hydroxyl carbon atom in the ^{13}C-NMR spectrum of the intact glycoside indicated a substitution at the HO-C(6) carbon of galactose and led to the cyclic structure **2** for the *E. luzonicus* major saponin, luzonicoside. We would note that in both sepositoside A(**1**) from *E. sepositus,* and luzonicoside (**2**), from *E. luzonicus,* the macrocyclic ring made up by the sugar moiety has the same size and conformation.

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9 HPLC-chromatograms of *E. sepositus* and *E. luzonicus* saponins are almost indistinguishable. Both chromatograms contained 3 peaks of which the central one corresponded to the major component. In the *E. luzonicus* it accounts for more than 90% of the total saponin content. The 2 minor saponins were obtained still in admixture with the major one and, in analogy with the minor saponins of *E. sepositus,* on acid hydrolysis, they gave 24-nor-22(R)-chloro-5α-cholesta-8,14-diene-3β,23(S)-diol (more polar saponin) and 22(R)-chloro-5α-cholesta-8,14-dien-3β,23(S)-diol along with minor amounts of 27-nor-24-methyl-22(R)-chloro-5α-cholesta-8,14-dien-3β,23(S)-diol (less polar saponin)[13]. The ^{13}C-NMR spectra of both mixtures confirmed the origin of these chlorohydrins from their corresponding epoxides (part-structures **5**–**7**).
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Absolute configuration of (+)-1,4-diphenyl-2,3-butanediol[1]

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Summary. The absolute configuration of the title compound, isolated earlier from bull testicular tissue, has been determined to be (2S,3S) by synthesis of the natural diol from L-(+)-tartaric acid.

In 1963 Neher reported the isolation of (+)-1,4-diphenyl-2,3-butanediol (**1**) from the testicular tissue of bulls and rats[2]. The occurrence of such a novel structure, apparently biogenetically unrelated to the usual steroidal androgens, was surprising and elicited interest in its possible physiological role. Neher reported that the diol was not found in the adrenals, liver, or ovaries, though it was later detected in the postmortem human liver[3]. The diol is secreted at a low rate in the spermatic venous blood of the gonadotrophic stimulated dog[4].

The diol is practically free of estrogenic and androgenic activity, although at high concentration it weakly inhibited testicular secretion of testosterone[2,4]. S.c. administration in gonadectomized, adult male rats did not depress serum LH or FSH levels,[5,6] but local implanation of the diol in the median eminence of the hypothalamus resulted in a significant elevation of serum FSH[6]. On the basis of these findings Iturriza et al.[6] postulated a physiological role for the diol in the control of FSH release.

Because of the possible importance of this substance in reproductive endocrinology and the dependence of biological activity on stereochemical configuration, we have determined the absolute configuration. The similarity of the chiral centers to those in tartaric acid prompted us to synthesize the diol from L-(+)-tartaric acid, using the reactions shown in the scheme.

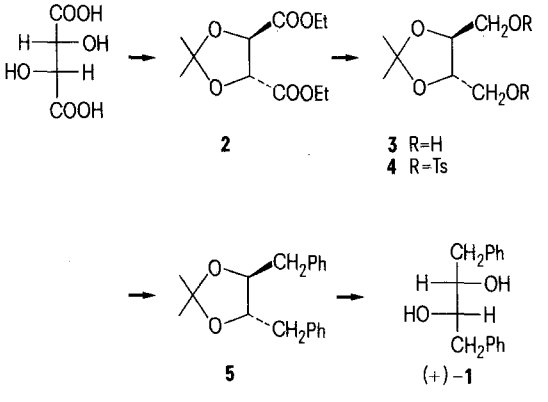
L-Tartaric acid was converted to the diethyl ester acetonide **2**. Following published procedures, **2** was reduced with lithium aluminum hydride to 2,3-O-isopropylidene-L-threitol (**3**) and then converted to ditosylate **4**. Displacement of the tosylate functions by phenyl was achieved with lithium diphenyl cuprate[7], and the resulting acetonide **5** was hydrolyzed by dilute acid to afford diol **1**. The synthetic product had the same melting point, IR-spectrum, and optical rotation as reported for the naturally occurring diol. Since the absolute configuration of (+)-tartaric acid has been defined by anomalous X-ray dispersion measurements[8] and the reactions used in the correlation sequence do not affect the asymmetric centers, the natural (+)-diol has the (2S,3S) configuration.

Experimental section. 1,4-Diphenyl-2,3-O-isopropylidene-L-threitol (**5**). To a solution of 3.0 g of cuprous iodide in 10 ml of dry ether, stirred at 0 °C under argon was added dropwise 20 ml of a 20% solution (2.1 M) of phenyl lithium in 75% benzene /25% hexane (Foote Chemical Co). A solution of 1.93 g of 2,3-O-isopropylidene-L-threitol ditosylate (**4**)[9], mp 90–92 °C, in 12 ml of ether and 3 ml of THF was added dropwise to the resulting green solution and the mixture was stirred at 25 °C for 2 h. Saturated aqueous ammonium chloride was added and the volatile solvents were removed at reduced pressure. The aqueous residue was extracted with several portions of ether, and the

extracts were washed with saturated brine solution, dried, and concentrated. The yellow oily residue was chromatographed on 20 g of silica gel, eluting first with hexane to remove biphenyl, then with hexane-ethyl acetate (3:1) to elute the acetonide. Distillation at 140 °C (0.1 mm) yielded 650 mg (47%) of colorless product. NMR ($CDCl_3$) δ1.4 (s, 6H, CH_3), 2.8 (m, 4H, CH_2), 4.0 (m, 2H, O-CH), 7.25 (s, 10H, aromatic). IR (neat) 3080, 3060, 3010, 2940, 2880, 1620, 1500, 1460, 1380, 1370, 1240, 1215, 1160, 1075, 1050, 750, 695 cm^{-1}.

1,4-Diphenyl-2,3-butanediol (**1**). The acetonide **5** (250 mg) was mixed with 4 ml of 1 N HCl and just enough ethanol to make the mixture homogeneous, then stirred at room temperature overnight. The solid which had filled the flask was filtered and recrystallized from acetone-petroleum ether to give 138.9 mg (81%) of colorless diol, m.p. 146–147 °C (lit.[2] m.p. 146–147 °C); $[\alpha]^{23}D+4.7\pm0.5°$ ($CHCl_3$, c 11); lit. $[\alpha]^{26}D+4.6\pm0.9°$ ($CHCl_3$, c 1.10). IR 3500–3150, 3030, 2960, 2910, 1450 cm^{-1}; NMR ($CDCl_3$) δ2.9 (m, 4H, CH_2), 3.72 (m, 2H, O-CH), 7.3 (s, 10H, aromatic).

Analysis. Calculated for $C_{16}H_{18}O_2$: C, 79.31; H, 7.49. Found: C, 79.09; H, 7.52.



Synthesis of (+)-1,4-diphenyl-2,3-butanediol from L-(+)-tartaric acid.

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The activity of monoamine oxidases A and B in gamma-irradiated rabbit brains[1]

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Summary. The activities of monoamine oxidases A and B towards 5-hydroxytryptamine and β-phenethylamine, respectively, were compared in the left and right caudatus, hippocampus, parietal cortex, cerebellum and frontal cortex 6 months after gamma-irradiation (single dose of 23 Gy) of either the right hemisphere or of the whole rabbit brain (in which case, a dose of 16 Gy). No difference in monoamine oxidase A or B activities were found in any of the brain regions.

In the rabbit brain, the activity of monoamine oxidase (MAO, monoamine O_2:oxidoreductase, EC 1.4.3.4) appears to exist as 2 forms, termed MAO-A and MAO-B, where the A form of the enzyme is responsible for the deamination of 5-hydroxytryptamine (5-HT), and is inhibited by low concentrations of clorgyline, and the B form, responsible for the deamination of β-phenethylamine (PEA), is inhibited by low concentrations of deprenil[2–5]. Although the exact functions of the 2 forms of MAO are not as yet known, the activity of MAO-A appears to be confined, in the main, to the neuronal tissue of the brain, whereas the activity of MAO-B is more non-neuronal in nature[6–8].

In 1973, Pausescu et al.[9] reported that exposure of rabbits to a low dose of gamma-irradiation (4 Gy) produced an increased activity of brain monoamine oxidase. A similar result was found for rat brain MAO after gamma-irradiation[10], whereas neutron-irradiation produced a decrease in the activity of MAO. In contrast, however, a single dose of 100 Gy gamma-irradiation was found to be without effect on the activity of rat brain MAO-A activity, the rats being sacrificed 24 h after the irradiation[11], which may suggest that the increases found by other authors might have been due to the effects of stress rather than the effect of gamma-irradiation, since stress is known to produce short-term increases in MAO activity[12–14].

Of long-term effects of irradiation upon MAO, it has been reported that the catalytic properties of MAO are 'transformed' to properties resembling those of diamine oxidase upon radiation injury (7 Gy) of experimental animals[15,16], due to a build-up in the concentration of lipid-peroxides[15–17]. In consequence, it was felt to be of importance to see whether long-term changes in the activity of MAO-A and -B of rabbit brain could be brought about by a single dose of gamma-irradiation.

Materials and methods. Rabbits (2.0 ± 0.1 kg) were irradiated over either the right hemisphere alone (radiation dose 23 Gy) or over both hemispheres (radiation dose 16 Gy).