ratio. All the other stimuli show the usual dependence of the aldosterone/corticosterone ratio on corticosterone concentration. Nevertheless measurement of corticosterone does allow a search for substances which may have an effect on the biosynthetic late pathway from corticosterone to aldosterone.

Reference

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Benserazide effects on tryptophan metabolism in mice

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Benserazide (Ro4-4602, *N*-seryl-*N'*-(2,3,4-trihydroxybenzyl) hydrazine) is known to inhibit aromatic amino acid decarboxylase. David (1975) has shown that administration of (1-^{14}C)-tryptophan to mice led to a considerable evolution of $^{14}CO_2$, and that this was inhibited by prior administration of benserazide. This was interpreted as indicating that decarboxylation to tryptamine may be a major pathway in the mouse.

An alternative explanation is that evolution of $^{14}CO_2$ from (1-^{14}C)-tryptophan could be due to metabolism of the alanine released by the action of kynureninase. In this case there would also be release of $^{14}CO_2$ from (2-^{14}C)-tryptophan. This has been demonstrated. It has further been shown that benserazide is a potent inhibitor of kynureninase and kynurenine aminotransferase in the mouse. The result of this inhibition is both a decrease in oxidative metabolism of tryptophan and an increase in the liver kynurenine concentration. These effects are seen at levels of the drug similar to those used in therapy of Parkinson's disease.

One effect of the reduced oxidative metabolism of tryptophan would be reduced synthesis of nicotinamide, and it is possible that patients treated with benserazide may show some signs of niacin deficiency. Preliminary data indicate that this is so – patients treated with Benserazide (or the similar drug Carbidopa, MK 486) excreted about one half the normal amount of N^1-methyl nicotinamide, the main urinary metabolite of niacin.

References

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