significant) loss of weight which had been regained after 7 hours. After 72 h guinea-pigs in the M group were as heavy as the group receiving the Vitamin C supplement alone. Administration of Vitamin C to guinea-pigs in the M group completely inhibited weight loss.

Vitamin C had increased food intake to 140% of that in the saline treated group 2 h after its administration. Fenfluramine inhibited food intake for 5 h in the same fashion as mazindol and diethylpropion. After 48 h food intake was 85% of that in the saline treated group. Supplementary Vitamin C caused guinea-pigs in the D group to increase their food intake 1 h sooner than the F group. In the M group Vitamin C restored food intake to 60% of that in the saline group within 2 h of administration. Administration of Vitamin C can differentiate between the anti-obesity and anorectic actions of weight-reducing drugs. It inhibits the anti-obesity action, but has little effect on the anorectic action, of fenfluramine and diethylpropion. Vitamin C inhibits the anti-obesity action of mazindol and reduces its anorectic effect.

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Rat liver tryptophan pyrrolase activity in iron deficiency anaemia

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Liver tryptophan pyrrolase (TP) is one of the peripheral factors affecting 5-hydroxytryptamine (5-HT) synthesis (Badawy & Evans, 1974) and is inversely-related to brain (5-HT) concentration (Curzon, 1969). The haem activator of TP contains Fe, and we therefore examined the effect of iron deficiency on TP activity and on three of its regulatory mechanisms.

Male Wistar rats (Tucks, Rayleigh, Essex) were made mildly- or severely-Fe-deficient by the diet of McCall, Newman, O'Brien, Valberg & Witts (1962). Control rats received the same diet supplemented with Fe. The deficiency was verified by haematological tests. The determination of TP activity in the absence (holoenzyme) or the presence (total enzyme) of added haematin, and the doses of injected compounds have previously been described (Badawy & Evans, 1973). The haem-free apoenzyme was calculated by difference. The results in mildly- and severely-Fe-deficient rats, as well as in their respective controls, were similar and were therefore pooled.

Control rats ('A' groups) gave typical results

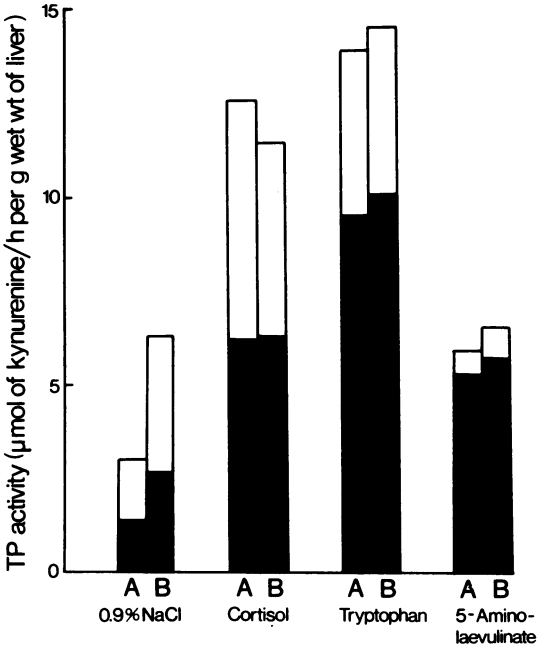

Figure 1 Comparison of tryptophan pyrrolase activities in iron-deficient and control rats.
The enzyme activity was determined at 4 h after intraperitoneal administration of various compounds. Closed columns represent holoenzyme whereas open ones apo-enzyme activity. The sum of the two columns gives the total activity. A, Control rats; B, iron-deficient rats.

(Figure 1). The basal (0.9% NaCl) holoenzyme was less than half the total activity. The induction of TP by cortisol (and stressful agents that release corticosteroids) involves increased apo-TP synthesis (Schimke, 1969). Here, the increase in the total TP activity is matched by a proportionate rise in that of the holoenzyme. A relatively larger rise in the holoenzyme is produced by tryptophan, which acts mainly by activating pre-existing apoenzyme. 5-Aminolaevulinate, acting via haem (Badawy & Evans, 1973), causes a strong saturation of apo-TP; the majority of activity being as holoenzyme.

Fe-deficient rats lost weight. The basal holoenzyme and total enzyme activities (after 0.9% NaCl, 'B' groups in Figure 1) rose in Fe deficiency by 93-110% ($P = 0.02$-0.001). Similar rises have been reported (Badawy & Evans, 1973) in starved rats which also exhibited weight loss. The stress condition of Fe-deficient rats, and the qualitative resemblance of the rise in their TP activities to that by cortisol suggest that corticosteroids may be involved in the effect of Fe deficiency. Cortisol, tryptophan and 5-amino-laevulinate were equally effective in increasing TP activities in Fe-deficient ('B' groups) and control ('A' groups) rats. This suggests that TP induction by hormones (cortisol), and its activation by substrate (tryptophan) or cofactor (haem) are not impaired in Fe deficiency.

The present and other results (Symes, Missala & Sourkes, 1971; Callender, Grahame-Smith, Woods & Youdim, 1974; Bailey-Wood, Blayney, Muir & Jacobs, 1975) suggest that enzymes requiring haem- or non-haem-Fe may be increased, unaltered or decreased in Fe deficiency. The activities of liver TP, brain tryptophan hydroxylase and monoamine oxidase(s) may all control the level of brain 5-HT in Fe-deficient rats. Work on these aspects is planned.

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The measurement of serum morphine levels by radioimmunoassay following oral administration of diamorphine or morphine

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The development of radioimmunoassay for morphine and related substances has resulted in greater opportunities for studying such compounds (Spector & Parker, 1970; Catlin, Cleeland & Grunberg, 1973; Morris, Robinson, Piall, Aherne & Marks, 1974). We report a method currently in use at the Department of Biochemistry, University of Surrey, and present preliminary data relating to the measurement of serum morphine in patients with advanced malignant disease receiving either diamorphine or morphine by mouth.

Antiserum was raised in a goat to a conjugate of 6-succinylmorphine-bovine serum albumin. Because the antiserum cross reacts almost equally with diamorphine, monoacetylmorphine, morphine and codeine, results are expressed as 'morphine equivalents'. Tritiated dihydromorphine was used as the radioactive label in the assay which has been described elsewhere (Aherne, Piall, Robinson, Morris & Marks, 1974). There is no cross reactivity for normorphine and only some 10% for morphine-3-glucuronide. Other drugs being taken by the patients did not cross react.