The effect of diet on blood urea levels in the beagle

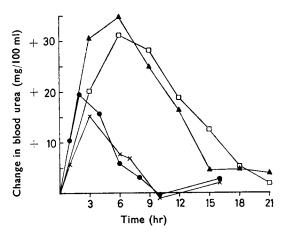
SIR,—Chance conversation between the authors revealed that, on certain occasions in the course of routine sampling of blood from beagles before or during long-term toxicity trials conducted independently at two centres (Huntingdon and Sandwich), marked variations in blood urea values had been noted. Amongst possible causes, that of dietary intake was suggested, and was investigated independently at the two centres. Our results indicate that, in the beagle, the blood urea concentration is significantly raised for a number of hours after a meal of one of various currently-used dog diets. This is particularly so if the animal receives only one main meal a day and therefore consumes virtually all its daily protein intake within 1 or 2 hr; its blood urea may then rise to levels usually considered indicative of a pathological abnormality.

Pedigree beagles of both sexes were used at both centres, those at Huntingdon (H) being 3-6 months old, whereas those at Sandwich (S) ranged from 1-5 years of age. Blood urea was determined throughout by the diacetyl-monoxime method, using a Technicon Autoanalyser (Marsh, Fingerhut & Kirsch, 1957).

TABLE 1. COMPOSITION OF BEAGLE DIETS

| Diet | | | Amount consumed (g) | Protein consumed (g) | n |
|------|-----|---------------------------------------------------------------------|---------------------------|----------------------------|----|
| I | (H) | Spiller P62 | 80-120 (mean 107) | 19-28-5 (mean 25-5) | 12 |
| 2 | (H) | Purina chow + tinned meat preparation (Lassie) + water (100:120:80) | 300 | 37.0 | 12 |
| 3 | (S) | Wet meat/biscuit diet (Reinert & Smith, 1963) | 500 | 85-0 | 37 |
| 4 | (S) | Spur | 350 | 87.5 | 26 |

(The amounts of diets 1 and 2 were normally fed twice daily; diets 3 and 4 were normally fed only once daily.)



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Blood urea levels in dogs used averaged (H) 31.2 mg/100 ml (n = 42) and (S) 29.9 mg/100 m (n = 102), when samples were taken after a fasting period of 18-24 hr. The mean net changes from fasting level noted in the dogs fed a single meal of one of four different diets (Table 1) were calculated (Fig. 1). Rises of up to 35 mg/100 ml (diet 3) were observed, roughly proportional to the amount of protein consumed. The peak responses were reached at 2 or 6 hr after feeding, and the elevations in blood urea lasted about 9 or 21 hr, in the animals fed the smaller (diets 1 and 2) or the larger (diets 3 and 4) amounts of diet respectively.

Our findings do not agree with the published opinions of many authorities who consider that, in the dog as in man, the level of the blood urea in the normal subject reflects the general state of protein nourishment rather than the direct influence of the subject's last meal (MacKay & MacKay, 1927; Robin, 1948; Coffin, 1953; Hoe & O'Shea, 1965; Coles, 1967). McKelvie, Powers & McKim (1966) reported an increase of only 17% in the urea nitrogen values in blood taken from beagles 1-2 hr after a meal, compared with 24-hr fasting values. In a recent paper, however, Vogin, Skeggs & others (1967) have reported increases in urea nitrogen in beagles after feeding which compared closely with our results.

In view of the popularity of the blood urea determination as a guide to possible renal damage, it would appear essential that, to obtain reproducible results in dogs used for example in long-term toxicity trials, blood samples for urea determination should be taken only after a sufficient fasting period (Bloom, 1960).

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References

Bloom, F. (1960). The blood chemistry of the dog and cat, p. 13. New York: Gamma Publics.

Coffin, D. L. (1953). Manual of veterinary clinical pathology, 3rd edn, p. 95. Cornell

Coles, E. H. (1967). Veterinary clinical pathology, p. 137. London: W. B. Saunders.

Coles, E. H. (1967). Veterinary cunical pathology, p. 137. London: W. B. Saunders. Hoe, C. M. & O'Shea, J. D. (1965). Vet. Rec., 77, 210-218.

MacKay, E. M. & MacKay, L. L. (1927). J. clin. Invest., 4, 295-306.

McKelvie, D. H., Powers, S. & McKim, F. (1966). Am. J. vet. Res., 120, 1405-1412.

Marsh, W. H., Fingerhut, B. & Kirsch, E. (1957). Am. J. clin. Path., 28, 681-688.

Reinert, H. & Smith, G. K. A. (1963). J. Anim. Techns. Ass., 14, 73-81.

Robin, V. (1948). Revue Path. comp. Hyg. gein., 48, 149-160.

Vogin, E. E., Skeggs, H. R., Bokelman, D. L. & Mattis, P. A. (1967). Toxic. appl. Pharmac., 10, 577–585.