The effect of heat treatment on the nutritive value of milk for the young calf

7*. The effect of the addition of selenium

By K. W. G. SHILLAM† and J. H. B. ROY
National Institute for Research in Dairying, Shinfield, Reading

(*Received* 11 *May* 1962—*Revised* 4 *January* 1963)

Earlier experiments in this series have shown that the growth and well-being of the young calf may be profoundly affected by the heat treatment to which the milk fraction of its diet is submitted during some of the common methods of processing (Shillam, Dawson & Roy, 1960; Shillam, Roy & Ingram, 1962*a–c*; Shillam & Roy, 1963*a*, *b*). Heating causes changes in the properties and availability of many constituents of milk, some of which appear to be associated with this adverse effect on the calf, but a causal relationship has not yet been clearly established.

In the experiment now reported, the effect on performance of the calf of the addition of selenium to a diet containing the 'severely' preheated spray-dried skim milk was studied. This investigation was prompted largely by the extensive studies of Fink and his co-workers who have reported that many skim-milk powders cause a fatal liver necrosis in young rats (Fink, 1955, 1956; Fink & Schlie, 1955*a*, *b*; Fink & Brenner, 1957; Fink, Schlie & Ruge, 1957, 1958; Fink, Schlie, Herold, Brenner & Fink, 1957; Fink, Schlie, Ruge, Brenner & Herold, 1958). Summarizing their studies covering the years 1949–57, Fink, Schlie & Ruge (1958, 1959) calculated that, in a total of 855 rats, the incidence of fatal liver necrosis was 76 % with forty-nine samples of roller-dried skim milk, 40 % with eleven samples of spray-dried skim milk and < 1 % with 123 samples of fresh skim milk, from which they concluded that the drying process clearly affected the nutritive value of milk.

These workers considered originally that their findings could be attributed to protein damage (Fink & Schlie, 1955*a*; Fink, 1956), since Schwarz (1951*a*, *b*) had found that L-cystine was one of three factors effective against liver necrosis; cystine is the only amino acid in milk protein which is in a markedly higher concentration in the heat-labile albumin fraction than in the casein. However, Schwarz & Foltz (1957) later identified Se as an integral component of Factor 3, the third, and hitherto unidentified, dietary agent that prevents necrotic liver degeneration in rats, and Fink (1959) subsequently showed that a daily supplement of 0·4–4 μg of sodium selenite prevented the occurrence of fatal liver necrosis in his rats. It is now known that in this respect cystine owes its potency largely to a minute contamination with a trace of Se (Schwarz, Stesney & Foltz, 1959; Schwarz, 1960).

It is well known that when milk is heated at a minimum temperature of 75°, or at

* Paper no. 6: *Brit. J. Nutr.* (1963), **17**, 183.
† Present address: Huntingdon Research Centre, Huntingdon.

somewhat lower temperatures when holding periods are employed, sulphydryl compounds and volatile sulphides are liberated, mainly from the sulphur-containing amino acids of the β-lactoglobulin fraction (Gould & Sommer, 1939; Josephson & Doan, 1939; Townley & Gould, 1943; Larson & Jenness, 1950; Hutton & Patton, 1952; Zweig & Block, 1953). As Se is closely related to sulphur, the two often occurring together (Sharman, 1960; Schwarz, 1960), it becomes apparent that when milk is heated there may well be a loss of available Se. Difficulty has been encountered in devising a satisfactory analytical method for determining the trace amounts of Se in milk, and it is only recently that the Se content of normal milk has been determined: Hadjimarkos & Bonhorst (1961), using the method of Bonhorst & Mattice (1959), found that the average Se content of milk from a number of farms in three counties in non-seleniferous areas of the state of Oregon in the USA was 0·005, 0·049 and 0·067 p.p.m. By other techniques, milk from cows grazing in seleniferous areas has been found to contain from 0·02 to 2·0 p.p.m. (Dudley & Byers, 1935), from 0·16 to 1·27 p.p.m. (Smith, 1941) and from 0·3 to 1·2 p.p.m. (Williams, Lakin & Byers, 1940).

The daily intake of Se by the calves on the supplemented diet in the experiment now reported was similar to that found to be effective in preventing muscular dystrophy in calves (Sharman, Blaxter & Wilson, 1959), i.e. about 0·55 mg anhydrous sodium selenite, equivalent to 0·25 mg Se/calf daily.

METHODS

Plan of experiment

The experiment was made during the winter months of 1959–60 under conditions of moderate 'infection' in the calfhouse (see Roy, Palmer, Shillam, Ingram & Wood, 1955; Ingram, Shillam, Hawkins & Roy, 1958). Two treatments in each of ten blocks were:

Treatment no.	Colostrum	Milk	Selenium
39	6 pints whole colostrum	Spray-dried skim milk ('severe' heat treatment, containing 2 % fat)	None
40			0·55 mg sodium selenite/10 lb milk

Calves

Ayrshire and Shorthorn calves that had not suckled were used; management was as described earlier (Shillam *et al.* 1962*c*).

Diets

Colostrum. Each calf was given 6 pints of whole colostrum within the first 36 h of birth, consisting of 1 pint from each of six different batches.

Milk. The spray-dried skim milk was prepared by the process described earlier (Shillam *et al.* 1962*a*); it was heated at a temperature of 74° for about 30 min before being spray-dried.

Selenium. A solution containing 55 mg anhydrous sodium selenite/l. was prepared.

Of this solution, 10 ml were added to each 10 lb of diet for calves on treatment 40; for calves on treatment 39, 10 ml water were added to each 10 lb of diet. Thus the supplemented diet contained about 0·12 p.p.m. of sodium selenite.

RESULTS

The results are summarized in Table 1. One calf given the diet containing Se and three of the control calves died; autopsies, kindly done by Dr P. L. Ingram, Royal Veterinary College, showed that the deaths were associated with an *Escherichia coli* localized intestinal infection.

In agreement with our earlier findings, the mean growth rate of surviving calves given the 'severely' heated spray-dried milk was low. Se, at the level given, had no effect on performance. A multiple covariance analysis showed that, of the variables likely to have an effect on weight gain, only birth weight had a significant effect. Treatment means were therefore adjusted by use of the regression coefficient of live-weight gain/day on birth weight given in Table 1. The adjustment was small, and after adjustment the mean weight gains of calves on the two treatments were still similar.

Table 1. *Comparison of the performance (mean values with their standard errors or ranges) of calves given a 'severely' preheated spray-dried skim milk either with or without a supplement of selenium*

	Treatment no. and details	
	39. No supplement	40. About 0·25 mg Se daily
Calves		
No. used	10	10
No. died	3	1
Live-weight gain/day of surviving calves (lb)	0·13 ± 0·06	0·11 ± 0·05
No. of days on which surviving calves scoured	4 (range 0–9)	4 (range 0–8)
No. of days on which surviving calves had a high rectal temperature (> 102·8° F)	1 (range 0–3)	2 (range 0–7)
Birth weight of surviving calves (lb)	81·8 ± 3·3	85·9 ± 2·9
Adjusted live-weight gain/day (lb)†	0·11 ± 0·05	0·13 ± 0·04
Regression coefficient of live-weight gain/day on birth weight	0·0114 ± 0·0037**	

** Significant at $0{\cdot}001 < P < 0{\cdot}01$.
† Adjusted for differences between treatment means in birth weight.

DISCUSSION

Certain conditions, including muscular dystrophy in the calf, appear to be prevented by supplementation of the diet either with vitamin E (Factor 1) or with Se (see review by Sharman, 1960). As the fat used in our diets contained about 12 mg total tocopherols/100 g, the calves received 8–12 mg tocopherols daily, which should be adequate for normal growth. At first sight, therefore, it does not seem surprising that there was no response to the Se supplement. However, in a recent review, Schwarz (1960) has clarified the relationship between vitamin E and Se in the prevention of a wide range of deficiency diseases, and has pointed out that, whereas acute damage

to the tissues in diseases such as liver necrosis and muscular dystrophy is essentially the result of a simultaneous deficiency of both factors, a lack of either factor alone often produces mild chronic syndromes. Schwarz states also that Se neither spares vitamin E nor substitutes for it and that there are other conditions, which include growth depression in the rat and chick and also probably in the lamb (Muth, Oldfield, Schubert & Remmert, 1959; Oldfield, Muth & Schubert, 1960; McLean, Thomson & Claxton, 1959; Drake, Grant & Hartley, 1960), that are caused purely by Se deficiency and are not affected by vitamin E.

It is of interest to record that Dr P. L. Ingram (personal communication) has observed centrilobular necrosis in the livers of some calves used in an earlier experiment whose deaths were associated with an *E. coli* localized intestinal infection. An association between heat treatment of the milk diet and the occurrence of liver necrosis is unlikely, however, since necrotic areas were present in the livers of calves that had been given fresh separated milk as well as in those of calves given the 'severely' preheated spray-dried milk.

SUMMARY

1. Twenty newborn calves were used in an experiment to find if selenium supplementation would improve the poor performance of calves given a diet containing a spray-dried skim-milk powder subjected to a severe preheating treatment.

2. A daily supplement of about 0·25 mg Se (as sodium selenite) did not improve the subnormal growth rate of the calves. Three calves given the unsupplemented diet and one calf given Se died; their deaths were associated with an *Escherichia coli* localized intestinal infection.

Footnote added 4 January 1963. Since this paper was submitted for publication, our colleague J. Toothill (personal communication) has determined the Se contents of the 'severely' preheated (at 74° for about 30 min) spray-dried skim-milk powder used in this experiment and the 'mildly' preheated (at 77° for 15 sec) powder used in earlier experiments (Shillam *et al.* 1962*b*, *c*; Shillam & Roy, 1963*a*, *b*) and found them to be similar.

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Printed in Great Britain