changes of body-weight are given show gains as well as losses. We have compared trends in the body-weights of thirty-seven Aberdeen primiparae who lactated for at least 10 weeks and thirty-five who lactated for not more than 3 weeks. A majority in both groups lost weight, but more in the breast-feeding group, in which the average loss was also greater. The women who lost most weight suffered most from tiredness, backache and other symptoms of impaired health.

There seems to be no correlation between the amount of weight gained during pregnancy and the yield and composition of breast milk. Apparently the production of milk, like foetal growth, can if necessary proceed more or less normally at the expense of the ordinary maternal tissues. The provision of special stores during pregnancy thus seems to be more important from the point of view of the lactating mother's health and well-being than from that of milk production.

Conclusions

Obviously, more research is needed. It is certain that the balance sheet of storage and loss varies greatly. We need measurements of the amounts of materials stored or lost, and analyses of the clinical significance of different patterns. Only in this way can we decide what is 'normal' and physiological, and what indicates inefficiency or abnormality.

REFERENCES

Dieckmann, W. J., Turner, D. F. & Ruby, B. A. (1945). *Amer. J. Obstet. Gynec.* **50**, 701.
Harding, V. J. (1925). *Physiol. Rev.* **5**, 279.
Hoffström, K. A. (1910). *Skand. Arch. Physiol.* **23**, 326.
Hunscher, H. A., Donelson, E., Nims, B., Kenyon, F. & Macy, I. G. (1932–3). *J. biol. Chem.* **99**, 507.
Hunscher, H. A., Hummell, F. C., Erickson, B. N. & Macy, I. G. (1935). *J. Nutr.* **10**, 579.
Hytten, F. E. & Thomson, A. M. (1960). In *Milk : the Mammary Gland and its Secretion.* (S. K. Kon and A. T. Cowie, editors.) New York: Academic Press Inc. (In the Press.)
Macy, I. G. & Hunscher, H. A. (1934). *Amer. J. Obstet. Gynec.* **27**, 878.
Mellanby, E. (1933). *Lancet*, **245**, 1131.
Murlin, J. R. (1917). *Amer. J. Obstet. Dis. Wom.* **75**, 913.
Nehring, K. (1957). *Biochem. Z.* **328**, 549.
Thomson, A. M. (1959). *Brit. J. Nutr.* **13**, 509.
Thomson, A. M. & Billewicz, W. Z. (1957). *Brit. med. J.* i, 243.
Wilson, K. M. (1916). *Johns Hopk. Hosp. Bull.* **27**, 121.

The influence of nutrition on female fertility in some of the large domestic animals

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Little reliable information concerning the influence of nutrition on the fertility of larger animals exists. There have been few well-planned experiments, but many expressions of opinion based on field observations. The views put forward are often conflicting and irreconcilable with the existing circumstances, which tends to throw suspicion on those who attach importance to the influence of nutrition on the fertility of large animals.

In this contribution I propose to deal with cows and to compare ewes and sows with them. Although little work has been undertaken in this field, it will be impossible to refer to it all.

Energy

Blaxter (1957) suggested that impaired breeding efficiency can be due to a lack of energy in the diet, but Hignett (1959) expressed the opinion that, within wide limits, shortage of carbohydrate and protein in the diet is self-limiting in its effect on the breeding performance of female cattle. If the energy available is low anoestrus is likely to result. When the plane of nutrition is higher, i.e. sufficient to permit ovarian activity but insufficient to maintain growth or lactation, a mating will most probably be successful. Cattle receiving a still more generous ration show increased growth or milk yield or both, but we believe it is this last group that is most sensitive to a deficiency or excess of certain mineral elements in the diet so that fertility may be impaired.

In observations over a period of 3 years Foote, Pope, Chapman & Casida (1959) showed that the plane of nutrition seemed to have a variable influence on the reproductive efficiency of ewe lambs. One year the 'flushing' of ewes appeared to increase the number of ova shed, whereas another year a higher plane of nutrition was associated with an increase in early foetal deaths.

With the sow and the gilt Kvasnitsky (1956), Saunders (1958) and Gossett & Sorensen (1959) produced evidence that a high plane of nutrition is likely to reduce reproductive efficiency, although the first author also incriminates inadequate feeding as a cause of porcine infertility.

Fibre

Saunders (1958) recorded that, in a group of twenty gilts, a sudden change from succulent food to dry fibrous grazing resulted in severe constipation and eighteen of the animals produced partly stillborn litters.

Vitamins

Of the fat-soluble vitamins, vitamins A and D are important for reproduction in the larger animals. On the other hand, vitamin E deficiency has no influence on fertility in female cattle (Gullickson, Palmer, Boyd, Nelson, Olson, Calverly & Boyer, 1949).

Vitamin A. Vitamin A deficiency definitely leads to infertility in female cattle. Depending on the degree of the deficiency and the time at which it occurs, the signs associated with the reproductive mechanism include anoestrus, frequent return to service, abortions and the birth of dead and weakly calves at full term (Hart & Guilbert, 1933; Axelsson, 1947). Avitaminosis A is not uncommon among heifers yarded during the winter and receiving a ration consisting almost entirely of sugar-beet pulp and oat straw. In ewes Guilbert, Miller & Hughes (1937) state that shortage of vitamin A interferes with the establishment of pregnancy and leads to

the birth of weakly lambs. With gilts, according to Saunders (1958), vitamin A deficiency increases the incidence of stillborn piglets.

Vitamin D. Wallis (1938) has produced evidence which suggests that a deficiency of vitamin D can lead to a fall in the fertility of female cattle. This finding is supported by the observations of Hignett & Hignett (1953). They pointed out that if the phosphorus intake was rather low (less than 20 g P_2O_5 in excess of the 23 g for maintenance and 19 g/gal milk usually accepted) then a widening of the calcium : phosphorus ratio led to no depression of fertility of animals mated in October and November, when presumably the vitamin D status was high, but led to a marked fall in breeding performance of animals served the following February and March (Fig. 1). It is of

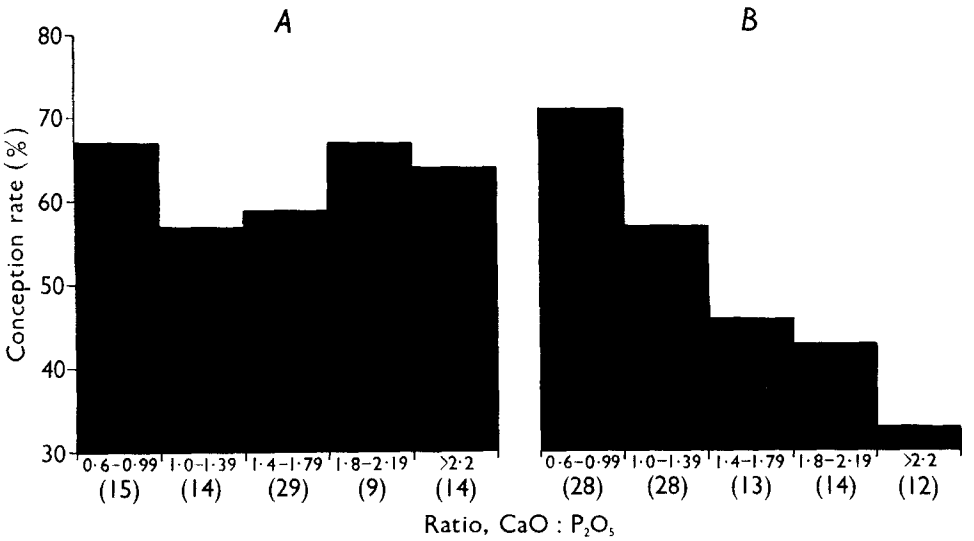

Fig. 1. Relationship between the ratio $CaO:P_2O_5$ in the diet and conception rate ((no. animals holding/no. mated) × 100) at first mating of cows receiving P_2O_5 less than 20 g in excess of the usually accepted amount of 23 g for maintenance and 19 g/gal milk and served during (*A*) October and November 1949 or (*B*) February and March 1950.

Significance of linear regression: (*A*) > 0·90; (*B*) < 0·02. Significance of deviations from linearity: (*A*) >0·99; (*B*) 0·95. Figures in parentheses are the numbers of animals mated.

interest that these results were obtained during the winter of 1949–50, a winter following one of the best summers on record in the south of England. Present trends in crop husbandry and conservation—kale growing, silage making and grass drying—often result in carotene-rich feeding-stuffs being given to the almost complete exclusion of hay; and hay is the dairy cow's chief source of vitamin D during the winter months unless a vitamin D supplement is provided. Furthermore, it is accepted that some at least of these carotene-rich foods contain a factor antagonistic to vitamin D (Grant, 1951; Weits, 1952). Stockdale (1956, unpublished) obtained evidence that a low vitamin D status in pigs could have an adverse effect on reproductive efficiency.

Vitamin E. Although vitamin E is generally regarded as having little influence on the fertility of female domestic animals, Lunca, Bratescu, Otel, Maior & Siminel

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(1956) reported that injections of this vitamin into sows on the 1st day of oestrus and again 15–16 days later led to an appreciable increase in litter size.

Water-soluble vitamins. As far as fertility is concerned the most important is probably vitamin B_{12}, a shortage of which occurs chiefly in ruminants suffering from cobalt deficiency. In affected females anoestrus is common, usually resulting from impaired appetite and greatly reduced food consumption. Dunlop (1948) claimed that the provision of cobalt supplements increased the lamb crop and decreased the number of barren ewes on the majority of Scottish hill grazings included in his investigations. There is also evidence that a vitamin B_{12} deficiency had an adverse effect on the breeding capacity of pigs (Møllgaard (1952), reporting the work of Moustgaard & Olsen).

Minerals

Of the minor mineral elements, cobalt has already been mentioned.

Copper. Absolute and conditioned deficiencies of copper are encountered in cattle and sheep in many parts of the world, including Britain. There has been some controversy about the effect of hypocuprosis on bovine fertility. In gross deficiency inanition occurs and anoestrus is not uncommon, but in less severely affected animals ovarian activity is maintained. It would appear that in the majority of herds with low blood-copper values there is impaired female fertility. On the other hand, there are some herds which have low blood-copper levels but maintain a high conception rate. These findings suggest that copper deficiency, absolute or conditioned, has little effect on breeding performance, but with hypocupraemic cattle of low fertility the administration of copper leads, almost always, to a sudden and dramatic improvement in fertility. With the ewe, low blood copper does not seem to reduce the size of the lamb crop. Nevertheless, because of the associated swayback, it may result in a marked reduction in the number of viable and useful lambs born.

Iodine. Iodine is usually considered an essential for normal reproduction of all species, but it is difficult to find conclusive evidence that iodine deficiency, absolute or conditioned, leads to infertility in cattle. Even when fertility problems are associated with thyroid dysfunction the latter is not necessarily syonymous with iodine deficiency. There is some circumstantial evidence that under certain conditions an iodine deficiency can interfere with the establishment of pregnancy in cows (Hignett, 1957). It has been suggested that goitrogens in kale and other members of the brassica family may lead to a conditioned iodine deficiency resulting in abortion or the birth of stillborn calves (White, 1954). With sheep it has been established that when pregnant ewes are folded on kale, the plant goitrogens present can lead to a very high percentage of weakly lambs, many of which die within 2–6 days of birth (Shand, 1952). The role of iodine in porcine reproduction is not clearly defined, but work in North America (Smith, 1917) indicates that iodine deficiency increases the incidence of stillborn and weakly piglets. According to the report very few young produced by sows on iodine-deficient diets survive for more than 36 h.

Manganese. There is much speculation about the importance of manganese in bovine reproduction. Hignett (1941) suggested that manganese deficiency leads to

ovarian dysfunction in cattle. Bentley & Phillips (1951) produced more convincing evidence that adequate levels of manganese in the diet are essential for fertility of heifers. Seekles (1949) has also drawn attention to the importance of manganese. More recently, Hignett (1956, 1959) has produced evidence to support the view that the dietary level of manganese necessary for high female fertility depends on the relative amounts of certain major mineral elements and perhaps other nutritional factors present. Van Koetsfeld (1954, 1958) has shown that the manganese content of hair is related to conception rate. It is generally accepted that manganese is essential for breeding efficiency of sows and gilts.

Calcium and phosphorus. Of the major minerals, most attention has been paid to calcium and phosphorus. It has long been accepted that a gross deficiency of phosphorus, leading to clinical aphosphorosis, results in lowered breeding performance of cattle (Du Toit & Bisschop, 1929). In such animals there is a high incidence of anoestrus. Cows will come on heat after calving and, if served then, do not return to service, but on examination are found not to be in calf. Hignett & Hignett (1951) produced evidence suggesting that the recommended levels of dietary phosphorus for dairy cows, to which reference has already been made, were not always adequate for normal fertility of cows and heifers. Furthermore, with such levels of phosphorus, a widening of the calcium : phosphorus ratio of the food could depress fertility still further. As previously mentioned, planned feeding experiments demonstrated that the heifer's requirement for manganese for high fertility is largely determined by dietary calcium and phosphorus (Hignett, 1956, 1959). The dietary calcium : phosphorus status (x_1) was assessed by the use of the formula $\frac{P^2}{Ca}$ (where P is the oral consumption in g of elementary phosphorus for each 100 lb body-weight and Ca the corresponding measure of calcium). When manganese consumption was low (less than 40 mg elementary manganese/100 lb body-weight) fertility could be high when calcium and phosphorus were balanced ($x_1 = 1{\cdot}0$–$2{\cdot}0$). On the other hand, if dietary manganese levels were high (more than 100 mg/100 lb body-weight) fertility was high when calcium was excessive relative to phosphorus or vice versa ($x_1 < 0{\cdot}5$ or $> 3{\cdot}75$). This finding is well illustrated in Fig. 2. Further experimental evidence has shown that, even with a high consumption of manganese, it is possible to get calcium and phosphorus so very much out of balance that fertility of heifers is impaired. Analysis of feeding-stuffs used at the three Bull Progeny Testing Stations in the United Kingdom indicates that during a series of first matings of first-calf heifers at those centres, during the years 1955, 1956 and 1957, the dietary calcium, phosphorus and manganese levels placed all animals in the top right-hand corner, i.e. in areas *B*, *C*, *E* or *F*, of the field shown in Fig. 2, at the time of first mating. It is probable that, in the average herd, animals fall into this part of the field at the time of service. The whole story of the calcium–phosphorus–manganese complex and its influence on bovine fertility suggests that major minerals affect the utilization of the manganese required for one or more enzyme systems essential for the establishment and maintenance of pregnancy. In fact, one is left wondering if there is not an

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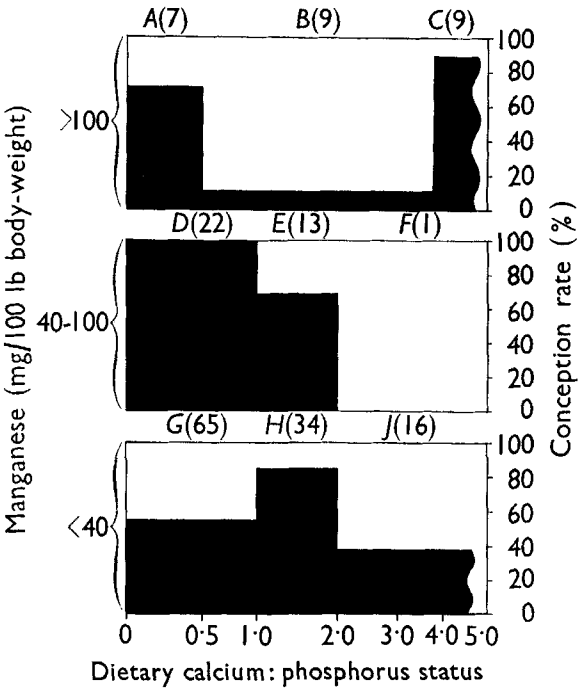

Fig. 2. Relationship between dietary calcium, phosphorus and manganese and conception rate ((no. animals holding/no. mated) × 100) at first mating of heifers. The dietary calcium : phosphorus status, x_1, is determined by the formula $\frac{P^2}{Ca}$ where P is the intake of elementary phosphorus in g/100 lb body-weight and Ca is the intake of elementary calcium in g/100 lb body-weight. Figures in parentheses are the numbers of animals mated.

important connexion here with the most interesting and valuable observations of Lutwak-Mann and her colleagues on the biochemistry of the foetus and its immediate environment (Lutwak-Mann, 1955–6; Adams & Lutwak-Mann, 1955–6; Jacobson & Lutwak-Mann, 1956–7).

Oestrogens

Since Bennetts, Underwood & Shier (1946) demonstrated that oestrogens in subterranean clover could lead to reproductive disorders in sheep in Western Australia, much thought has been given to the possible effect of plant oestrogens on the fertility of grazing animals. The true position with regard to cattle has not yet been assessed, although Rowson (1959) has cast new light on the possible role of these oestrogens in bovine infertility. It may be that plant oestrogens and minerals have a joint effect. There is circumstantial evidence that these oestrogens can depress porcine fertility (Saunders, 1958).

Conclusions

In this review little attempt has been made to suggest the manner in which deficiencies or excesses of dietary constituents influence reproductive efficiency. Gross deficiencies depress ovulatory activity and often result in anoestrus. On the

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other hand, when ovulation continues it is not always clear whether there is a failure of fertilization or an increase in prenatal mortality. There is evidence that plant oestrogens interfere with fertilization of ewes (Sanger & Bell, 1959) and also affect the time taken for cattle and sheep ova to reach the uterus (Rowson, 1959). A high plane of nutrition sometimes appears to increase ovulation rate in sheep (Foote *et al.* 1959); it also increases ovulation rate in gilts (Haines, Warnick & Wallace, 1959). The latter workers, together with Gossett & Sorensen (1959) have demonstrated that on such a ration there is, in addition, an increase in the percentage of prenatal mortality. With avitaminosis A it is thought by some that changes in the endometrium can prevent implantation. Deficiency of the vitamin during the later stages of pregnancy results in foetal death.

Finally, the importance of stress associated with growth rate and lactation cannot be overemphasized. Time after time it has been noted that the animals which first show the effect of deficiencies or excesses of dietary constituents are those receiving an otherwise desirable and liberal ration and hence showing a high growth rate or milk yield or both. This feature of nutritional disturbance is frequently overlooked and much confusion thereby arises because animals on a quantitatively poor ration and consequently showing little growth rate or giving poor milk yields have a high level of fertility provided their energy consumption is sufficient to maintain ovarian activity.

REFERENCES

Adams, C. E. & Lutwak-Mann, C. (1955–6). *J. Endocrinol.* **13**, xix.
Axelsson, J. (1947). *Landtmannen, Uppsala*, **31**, 37.
Bennetts, H. W., Underwood, E. J. & Shier, F. L. (1946). *Aust. vet. J.* **22**, 2.
Bentley, C. G. & Phillips, P. H. (1951). *J. Dairy Sci.* **34**, 396.
Blaxter, K. L. (1957). *Proc. Nutr. Soc.* **16**, 52.
Dunlop, G. (1948). *Congr. int. Physiol. Réprod. anim.* I. *Milan*, p. 4.
Du Toit, P. J. & Bisschop, J. H. R. (1929). *Rep. vet. Res. S. Afr.* **15**, 1059.
Foote, W. C., Pope, A. L., Chapman, A. B. & Casida, L. E. (1959). *J. Anim. Sci.* **18**, 453.
Gossett, J. W. & Sorensen, A. M. (1959). *J. Anim. Sci.* **18**, 40.
Grant, A. B. (1951). *Nature, Lond.*, **168**, 789.
Guilbert, H. R., Miller, R. F. & Hughes, E. H. (1937). *J. Nutr.* **13**, 543.
Gullickson, T. W., Palmer, L. S., Boyd, W. L., Nelson, J. W., Olson, F. C., Calverly, C. E. & Boyer, P. D. (1949). *J. Dairy Sci.* **32**, 495.
Haines, C. E., Warnick, A. C. & Wallace, H. D. (1959). *J. Anim. Sci.* **18**, 347.
Hart, C. H. & Guilbert, H. R. (1933). *Bull. Calif. agric. Exp. Sta.* no. 560.
Hignett, S. L. (1941). *Vet. Rec.* **53**, 21.
Hignett, S. L. (1956). *Int. Congr. Anim. Reprod.* III. *Cambridge. Plenary Pap.* p. 116.
Hignett, S. L. (1957). *Vet. Rec.* **69**, 210.
Hignett, S. L. (1959). *Vet. Rec.* **71**, 247.
Hignett, S. L. & Hignett, P. G. (1951). *Vet. Rec.* **63**, 603.
Hignett, S. L. & Hignett, P. G. (1953). *Vet. Rec.* **65**, 21.
Jacobson, W. & Lutwak-Mann, C. (1956–7). *J. Endocrinol.* **14**, xix.
Kvasnitsky, A. (1956). *Int. Congr. Anim. Reprod.* III. *Cambridge, Plenary Pap.* p. 59.
Lunca, N., Bratescu, I., Otel, V., Maior, I. & Siminel, N. (1956). *Int. Congr. Anim. Reprod.* III. *Cambridge, Plenary Pap.* p. 134.
Lutwak-Mann, C. (1955–6). *J. Endocrinol.* **13**, 26.
Møllgaard, H. (1952). *Int. Congr. Anim. Husb.* VII. *Copenhagen*, **2**, 7.
Rowson, L. E. (1959). *Int. vet. Congr.* XVI. *Madrid*, **2**, 919.
Sanger, V. L. & Bell, D. S. (1959). *J. Amer. vet. med. Ass.* **134**, 237.
Saunders, C. N. (1958). *Vet. Rec.* **70**, 965.
Seekles, L. (1949). *Int. vet. Congr.* XIV. *London*, **3**, 9.

Shand, A. (1952). *B.V.A. Publ.* no. 23, p. 58.
Smith, G. E. (1917). *J. biol. Chem.* **29**, 215.
Van Koetsfeld, E. E. (1954). *Tijdschr. Diergeneesk.* **79**, 405.
Van Koetsfeld, E. E. (1958). *Tijdschr. Diergeneesk.* **83**, 229.
Wallis, G. C. (1938). *J. Dairy Sci.* **21**, 315.
Weits, J. (1952). *Nature, Lond.*, **170**, 891.
White, J. B. (1954). *B.V.A. Publ.* no. 24, p. 43.

Effect of nutrition on androgenic activity and spermatogenesis in mammals

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To define the nutrient requirements essential to the male organism for its normal reproductive function is by no means a simple matter. It is a problem of many aspects because the processes involved are subject to fluctuations even within a single species, according to climate, environmental conditions, food habits, and the age of sexual maturation. A further complication arises from the effects of so-called 'nervous', 'stress' or 'conditioning' factors which can alter the normal requirements of an individual for a specific nutrient by influencing the absorption of food, storage and utilization of metabolites in the tissues, and their excretion to such an extent that the result is 'malnutrition' in spite of adequate food composition (Ershoff, 1948). It must also be kept in mind that failures of reproduction brought about by 'malnutrition' may be aggravated by congenital anatomical as well as genetic factors. However, by far the greatest difficulty encountered in the appraisal of reproductive disorders due to 'malnutrition' is to distinguish between primary effects due to the lack of a specific dietary component such as a vitamin, a trace element, or an essential amino acid, and secondary effects arising from diminished appetite, inadequate food intake and, finally, inanition, all of which are sequelae of deficient diets. This is one of the reasons why pair-feeding is such an essential and important prerequisite for all experimental investigations on the relationship between nutrition and reproduction.

The literature on the subject of nutritionally conditioned reproductive failures in the male is vast and abounds in observations on testicular atrophy, gynaecomastia, diminished libido and impotence, associated with such conditions as diabetes, hepatic cirrhosis, atrophic glossitis, gingivitis, seborrhea, keratosis of the eyelids and others; in this category belongs also the important study of Evans & Burr (1927) on degenerative and atrophic changes in the testes and male sterility, associated with deficiency of vitamins A and E. Together, these and similar studies represent an imposing total of valuable data which have been comprehensively reviewed from time to time, in particular by Biskind (1946), Hertz (1946), Samuels (1948), Russell (1948), Mason (1949), Ferrando (1953), Meites (1953), Roche (1957), and more recently by Lutwak-Mann (1958).