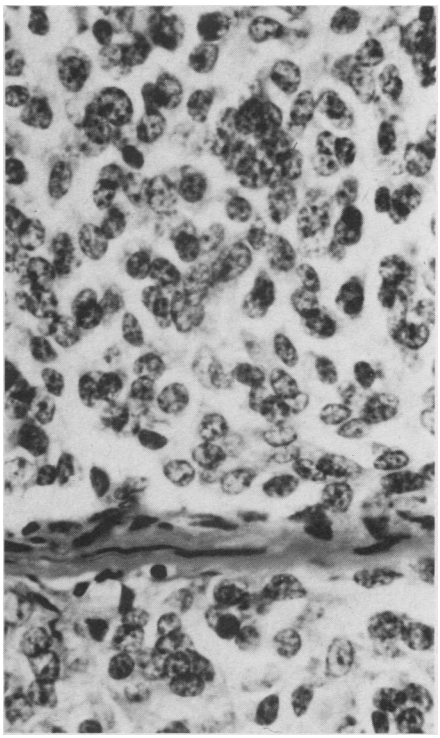

FIGURE 4. Cellular detail, showing spherical to oval nuclei and scant cytoplasm. H & E. X480.

Most veterinary pathology textbooks omit cattle from lists of species in which aortic body chemoreceptor tumors occur, or they state that such neoplasms occur only in dogs and humans. It would be of interest to know whether this neoplasm is seen more frequently than published reports indicate.

We are grateful for the expert assistance of G.B. Tiffin, I.G. Shirley and G. Appl.

Yours truly,

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Hemophilus Pneumonia in B.C. Swine

DEAR SIR:

British Columbia has had no known isolates of *Hemophilus pleuropneumoniae* (*parahemolyticus*) in swine until November of 1979. On September 22, 1979 two complete herds of swine were imported from Ontario and established in new facilities on two farms which had never raised swine before. The pigs were purchased from one owner but had been raised on two separate farms four miles (6.4 km) apart in Ontario. The total shipment was mixed and delivered in two semi-trailers to B.C. where the pigs raised on one farm were delivered to farm A and those raised on the other farm were delivered to farm B.

Farm A consisted of 305 gilts and 17 boars with a weight range of 75 to 200 lb (34 to 90.9 kg) and an average weight of 150 lb (68 kg). There were two dead pigs on the truck when it arrived and two pigs died on September 24. None of these pigs were examined at the laboratory. No pigs died between September 25 and November 6, but between November 7 and November 8 eight pigs died. One of the eight dead pigs was presented to the B.C. Veterinary Diagnostic Laboratory the same day and a diagnosis of fibrinous pneumonia was made with *H. pleuropneumoniae* being recovered. On November 13 two more pigs died and both were positive for *H. pleuropneumoniae*.

Farm B consisted of 61 sows and four boars having a weight range and average weight similar to those on Farm A. Many of these pigs were coughing when delivered and continued to cough during the time they were on the farm. No deaths occurred.

Between January 30 and July 17, 1980 *H. pleuropneumoniae* was isolated from a further four herds in B.C.

Farm C was a large breeding farm. The organism was identified from an area of acute inflammation in a lung being also affected with pleurisy and pleural adhesions.

Farm D reared pigs originating from farm C and the organism was cultured along with *Pasteurella multocida* from a pneumonic area. The lungs of this pig also exhibited adhesions and some fibrin was present on the surface.

Farm E was a large feeding operation in which the pigs were derived from a variety of sources. *Hemophilus pleuropneumoniae* along with *P. multocida* was isolated from the lungs of a dead animal which showed a severe purulent bronchopneumonia histologically and consolidation grossly.

Farm F was a medium sized farrow-to-finish operation from which one pig, of four which died suddenly, was submitted for necropsy. It showed a very severe acute necrotizing bronchopneumonia from which *H. pleuropneumoniae* was isolated. The gross appearance of this lung was one of extensive consolidation of the ventral lobes. Several other pigs from this farm died from pneumonia during the next 45 days. *Hemophilus pleuropneumoniae* and *P. multocida* were both isolated from the lungs of these pigs.

Following the diagnosis of *H. pleuropneumoniae* on November 9, the owners of herd A and B voluntarily consented to quarantine their premises. A British Columbia Order-in-Council, providing for quarantine and slaughter, if necessary, was approved on November 26, 1980.

On November 28, herd A and B were slaughtered at an approved slaughterhouse. The lungs of eight pigs in Herd A exhibited consolidation, six of these lungs yielded *H. pleuropneumoniae* on culture. Over 50% of the lungs of pigs from Herd B exhibited anterior ventral lung consolidation. There were no *Hemophilus* isolations from herd B, but the gross and microscopic examination of these lungs was indicative of enzootic pneumonia.

Isolates from herd A, made up of pigs from Ontario, along with those from the four B.C. herds were sero-

typed. Herd A was found to be infected with serotype 1. The isolates from the other herds autoagglutinated on first examination but, following subculture, a satisfactory test was carried out and the strains were identified as serotype 3.

Following the isolation from individual animals of serotype 3 which exhibited lung pathology, other than a fibrinous pleuropneumonia, it was decided to modify our method of handling these herds. This decision was aided considerably by the fact that we were able to do our own serological typing within 24 hours of identifying the organism. Initially, upon a gross or bacteriological diagnosis of *Hemophilus* infection, the premises were immediately placed under quarantine. Upon the identification of serotype, the quarantine was lifted.

To date, only three serotypes of *H. pleuropneumoniae* have been identified in Canada (2), serotypes 1 and 5 with certainty, and serotype 2 presumptively. Serotype 3, which has been identified in the U.S.A. was described by Nicolet (1) as being isolated from subcutaneous periarticular and pulmonary abscesses, and not from pleuropneumonia. These observations were considered to be justification for a completely different approach to the four B.C. herds.

There has been no further evidence of serotype 1 *H. pleuropneumoniae* in B.C. since the slaughter of the two herds.

We acknowledge the assistance of T.K. Sebunya of the Department of Veterinary Microbiology, Western College of Veterinary Medicine, Saskatoon, Saskatchewan who carried out the serological tests of the isolates from herds A,C,D and E and Drs. A.D. Osborne and J.R. Saunders for advice regarding the significance of the results. The methodology of Nicolet has been adapted for rapid serological identification of the various serotypes by Dr. J.V. Greenfield.

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Monensin Toxicity in Broiler Chickens

DEAR SIR:

Monensin toxicity has been reported recently in several species of domestic animals (1, 3) and poultry (1, 2) in western Canada. The occurrence of signs of monensin toxicity in a small farm flock of broiler chickens, plus the characteristic position of many of the dead birds, the notable lesions and the importance of the proper sampling of feed, prompted this report.

The affected flock consisted of 150 ten week old broiler chickens that had been fed a 16% commercial broiler-grower plus barley screenings. In mid-July, the owner had purchased several new bags of nonmedicated 16% broiler grower. Within two days after the introduction of the new feed, most of the flock began to stagger, to walk reluctantly and to lie in sternal recumbency. Generally, affected birds appeared alert and ate and drank when feed and water were placed nearby. Approximately half of the 26 birds that died were found in sternal recumbency with neck and hind limbs outstretched, a position reported to be characteristic of birds dying of monensin toxicity (1, 2).

One dead and nine live, affected birds were submitted to our laboratory for examination. Postmortem examination did not reveal significant gross lesions in any tissues except in one live bird, the heart of which had a pale, parboiled appearance. Histologically, the myocardium of this and other birds examined was hypercellular, pale, vacuolated and granular, especially in perinuclear areas. Several skeletal muscles including the major pectoral, supracoracoideus, cranial iliotibial, medial crural flexor, emoral adductor and gastrocnemius had an extensive myopathy. Acute degeneration was characterized by loss of cross-striations, sarcoplasmic granularity, fragmentation and mineralization. More advanced changes were seen as intense infiltrations of macrophages and occasional heterophils, and proliferation of sarcolemmal nuclei. Although skeletal muscular lesions of a minor nature have been reported in monensin toxicity in chickens (1, 2), the contrasting extensive myopathy seen on histological examinations in the present case was remarkable.

To investigate the possibility of a feed-related problem, we initiated a limited feeding trial, using unopened bags of the suspect test feed and, as a control, a similar 16% broiler-grower produced by another company. Twenty-six six week old broiler chickens obtained from a commercial broiler operation were divided at random into two groups of 13 birds. One group was given the test feed and the other group, the control feed, *ad libitum*. After four days of feeding, the birds on the test feed were noted to move somewhat stiffly and reluctantly and to be less active than the control birds. Compared with the control group, the test group ate less feed in the same period of time. Overt feed refusal however, was not observed, as all feed placed before the test group was eaten eventually by these birds. During this feeding trial, there was no significant difference in total weight between the two groups of birds. Two birds of the test group died of a staphylococcal septicemia but natural mortalities did not occur in the control group. At necropsy gross lesions were not found in birds from either group. Subtle histological lesions seen in skeletal muscle as a mild granular degeneration similar to that described in previous reports of monensin toxicity in chickens (1, 2), were seen only in birds of the test group.

Samples of test feed from the unopened bags used subsequently in the feeding trial were collected by an inspector of the Plant Products Div-