A SUSPECTED VIRUS INFECTION OF THE ORAL MUCOSA IN SWEDISH REINDEER (*RANGIFER TARANDUS* L)

Misstanke om virusinfektion i munnslemhinnan på svensk ren.

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Abstract: During the winter 1980 reindeer herds in the Tornedalen area, along the Finnish border, were hit by grazing difficulties. Thus minor parts of the reindeer herds in this area were given supplementary feed in pens. Some of the supplementary fed animals were taken ill and some deaths occurred. According to the owners sick animals showed loss of appetite and signs of fever. A total of 8 carcasses were necropsied at The National Veterinary Institute. In 5 of these cases oral lesions were observed. The histological investigation of the oral mucosa revealed intracytoplasmic inclusion bodies, inter- and intracellular oedema and vesicle formation. An electron-microscopical study of 2 of the cases confirmed the histological findings. At the bacteriological investigations *Coli, ß-haemolyzing streptococci, Conrynebacterium pyogenes* and *Fusobacterium necrophorum* could be indentified. The found bacteria were all considered secondary invaders.
Serological samples from four affected reindeer flocks were tested for antibodies against *BVD-, Pi3* and *IBR-virus* as well as *Chlamydia.* A few samples showed low positive titres for agents tested but for *BVDV.*
The result of the investigation indicates that a still unidentified virus could be the primary cause of this enzootically appearing disease of the oral mucosa in reindeer.

Key words: Reindeer, oral lesions, virus, necrobacillosis.

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Sammanfattning: Under vintern 1980 drabbades renhjordar i Tornedalsområdet av betessvårigheter. Av denna anledning fördes mindre flockar, ur hjordarna, till inhägnader och tillskottsutfodrades. En del av de tillskottsutfodrade djuren insjuknade och dödsfall inträffade. Enligt djurägarna förlorade de sjuka djuren aptiten och uppvisade tecken på feber. Av de döda renarna erhölls åtta för obduktion vid Statens Veterinärmedicinska anstalt. Hos fem av de obducerade renarna förelåg skador i munhålan. Vid histologisk undersökning av munslemhinnan påvisades intracytoplasmatiska inklusionskroppar, inter- och intracellulärt ödem och vesikelbildningar. Elektronmikroskopisk undersökning av två av fallen konfirmerade de histologiska undersökningarna.
Vid de bakteriologiska undersökningarna påvisades växt av *kolibakterier, ß-hemolyserande streptokocker, Corynebacterium pyogenes* och *Fusobacterium necrophorum.* I samtliga fall betraktades dessa som sekundärinfektioner. Serologiska undersökningar företogs, i fyra flockar där dödsfall förekommit, avseende förekomst av antikroppar emot BVD-virus, Pi3 och IBR-virus samt Chlamydia. Ett mindre antal prover uppvisade positiva titrar för de undersökta agens utom vad avser BVD. Resultaten av undersökningarna indikerar att ett ännu ej identifierat virus kan vara primärorsak till de enzootiskt upptrådande utbrotten av sjukliga förändringar i munhålen hos ren.

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INDTRODUCTION

During the winter 1980 the Tornedalen area along the Swedish/Finnish border was hit by grazing difficulties for the reindeer herds. Thus supplementary feeding was organized at several places from December through April. Mostly this turned out well but at some places animals were taken ill, mostly showing loss of appetite and signs of fever. Sudden deaths also occurred.

This paper deals with the pathological lesions found in animals sent in fo necropsy at the National Veterinary Institute, Uppsala, Sweden. In addition blood samples, for serological investigation, were obtained from flocks or reindeer on feeding sites where animals had succumbed.

MATERIAL AND METHODS

1. Animals

The eight reindeer autopsied in this investigation had all been subjects to artificial feeding in groups of different sizes and at different places. For most of the reindeer this feeding started up in mid-December. The feed usually consisted of a minor amount of commercial feed-mixture and hay *ad lib.*

For most of the reindeer the feeding period was spent in permanent pens but at some places the pens were regularly shifted to untouched land in order to provide the animals with fresh snow as water supply.

Nos 1 and 2 were sent in from a feeding place at Männikkö (Lat 67°15'N) where about 50 reindeer had been fed for some weeks. Several diseased reindeer had been treated with antibiotics by the local veterinarian.

Nos 3, 4 and 5 emanated from another feeding place, Övertorneå, about 130 km to the south. Here some twenty reindeer had been kept for a couple of months. Several animals became ill some of which had succumbed and some had recovered.

No 6 was sent in from a place located another 20 km to the south. This group numbered about 15 heads and had been fed for a couple of months. Several reindeer had fallen ill, showing mouth lesions.

Nos 7 and 8 belonged to a group of some 30 reindeer kept at a place located 50 km south of nos 1 and 2. The animals were kept in a rather large pen in which they obviously had spent most of the feeding period.

2. Pathological investigations

All animals were sent to the National Veterinary Institute with the history of a sudden death. In addition they were all, by the owners, considered to be of a moderate nutritional state. Age was estimated on the basis of dental wear. For histological studies tissues were fixed in 10% formol saline, embedded in paraffin and stained with haematoxyline-eosine, periodic-acid-Schiff, Grocotte and Gram stains. From each case material was taken fro routine bacteriological investigations.

3. Electron microscopical investigations

Selected areas were cut from paraffin blocks, put in xylol for 24 hours, at room temperature, hydrated in graded ethanol series 90% - 70% - 50% - 30% for 10 min. each at 4°C, post fixed in 2% glutaraldehyde (0.1 M cacodylate buffer) for 60-120 min. at 4°C and in 1% osmiumtetroxide in 0.2 M cacodylate buffer for 60 min. at 4°C, dehydrated in ethanol series 30% - 50% - 70% - 90% for 10 min. each at 4°C, put in propylan oxide/Epon 1/1 during 60 min. at room temperature and embedded in Epon. After embedding in Epon 1µ thick sections were cut and stained with toludine blue for light microscopy and thin sections were prepared on a LKB ultrotome, picked up on uncoated copper grids, stained with uranyl acetate and examined in a Philips electron microscope 401.

4. Serological investigations

Bloodsamples were drawn from av jugular vein of 26 animals, at selected feeding sites where sudden deaths had occurred, using 10 ml vacutainer tubes (Becton - Dickinson) without anticoagulant (Table 1). From three of these herds dead animals were brought in for necropsy.

From Hedenäset no 6, Övertorneå I no 3, 4 and 5, Männikkö no 1 and 2 (Table 1).

Cell cultures. Secondary calf kidney cells or bovine turbinate cells were grown in Eagle's Minimum Medium (EMM) containing 10 percent fetal calf serum, 100 units/ml penicillin, 10 µg/ml streptomycin and 1µg/ml Fungizone. The maintenance medium was EMM containing two percent of horse or fetal calf serum and antibiotics.

Table 1. Results of serological investigation.
Tabell 1. Resultater av serologiska undersökningar.

Herd	Animal no.	Sex	Age	BVD		Pi 3		IBR		Chlamydia	
				3/3	17/4	3/3	17/4	3/3	17/4	3/3	17/4
Hjord	*Djur no.*	*Kön*	*Ålder*								
Hedenäset	1	♀	ad	<5	<5	<8	<8	0	0	0	0
	2	♀	ad	<5	<5	<8	<8	0	0	4	0
	3	♂	ad	<5	<5	8	<8	0	0	8	0
	4	♀	ad	<5	<5	<8	<8	0	0	0	2
	5	♂	ad	<5	<5	<8	<8	0	0	0	2
	6	♂	ad	<5	<5	8	16	8	16	0	0
	7	♀	22 m	<5	<5	8	16	0	0	16	2
	8	♀	10 m	<5	<5	<5	<5	0	0	4	0
	9	♀	10 m	<5	<5	16	16	0	0	4	0
Övertorneå I	10x	♀	ad	<5	<5	<8	<8	0	0	0	2
	11x	♀	22 m	<5	<5	16	16	0	0	0	0
	12x	♀	ad	<5	<5	8	8	0	0	0	0
Männikkö	13	♀	ad	<5	<5	16	16	0	0	2	0
	14	♀	10 m	<5	<5	8	8	0	0	0	0
	15	♂	10 m	<5	-	<8	-	0	-	0	-
	16	♀	ad	<5	<5	8	8	0	0	2	2
	17	♀	ad	<5	<5	<8	<8	0	0	0	8
	18	♀	ad	<5	<5	16	16	0	0	0	0
	19	♀	10 m	<5	<5	<5	<5	0	0	0	0
	20	♀	ad	<5	<5	<5	<5	4	4	8	0
	21	♀	ad	<5	<5	8	16	0	0	0	0
	22	♀	ad	<5	<5	<8	<8	0	0	0	4
Övertorneå II	23x	♀	ad	<5	<5	0	<8	0	0	0	0
	24	♀	ad	<5	<5	8	8	0	0	0	0
	25	♀	ad	<5	<5	8	8	4	8	2	0
	26x	♀	ad	<5	<5	16	16	0	0	0	0

ad=adult *(vuxna)* m=months *(månader)*
x=has shown cilinical signs of disease *(Har visat kliniska sjukdomstecken)*

Viruses. Following antigens were used: The Ug-59 strain of bovine virus diarrhoea virus (BVDV), the U-23 strain of parainfluenza-3 virus (Pi_3), and the Colorado strain of infectious bovine rhinotracheitis virus (IBR).

The BVDV was propagated in bovine turbinate cells. The Pi_3 and IBR viruses in secondary calf kidney cells.

The Chlamydia antigen was brought from Wellcome Laboratories.

Serological tests. For the serosurvey conventional methods in a microtiter system or in cell culture tubes were used, i.e. the heamagglutination-inhibition (HI) test for Pi_3-virus, the complement-fixation (CF) test for Chlamydia and the serum neutralization (SN) test for BVDV and IBR. Appropriate pre-treatment of the sera was made for different tests.

RESULTS

Pathological investigations

Due to the variations between cases, the findings at each necropsy are described.

No. 1. Received Januar 21. Female, 8 months.
The animal was emaciated and in a state of mixed dentition. Fodder impactions were present between the teeth and the gingiva was partly necrotic showing intense inflammatory changes. An acute focal pneumonia and a mild purulent pleuritis was present in the lungs and also a moderate amount of *Dictycaulus viviparus* eggs and larvae and larvae of *Elaphostrongylus rangiferi*. A necrotizing thrombotic process was found in one lung.

Bacteriological investigation: A rich growth of ß-haemolyzing streptococci was found in the necrotic areas of the gingiva and in the lungs growth of coli.

No. 2. Received Januar 21. Male, 8 months.
The animal was emaciated. Present in the hoof of the right hindleg was a stabwound leading into a sequestered hoofbone. In addition a rumen indigestion was found and a focal subacute myocard-degeneration.
Bacteriological investigation: A pure rich growth of *Corynebacterium pyogenes* was found in the sequestered bone.

No. 3. Received February 20. Female, 6 years.
The animal was pregnant and emaciated. In the mouth focal areas of ulcerative, purulent stomatitis were present. Histologically the mucous membranes of the mouth revealed focal areas of marked inter- and intracellular oedema and vesiculation mainly located in *Stratum Malpighii* (Plate 1 & 2). Numerous cells appeared to contain pycnotic nuclei compressed by cytoplasmic and eosinophilic inclusion bodies (Plate 3 & 4). Sloughing of affected areas of the epithelium was a common feature. In focal areas the epithelium was totally necrotized and lost with a marked infiltration of polymorphonuclear cells. In such areas bacterial colonies and plant particles could be observed. In areas with still remaining epithelium the infiltration of leucocytes in the submucosa was of a mild character but a moderate oedema and hyalinization of muscle fibres was observed. Minor haemorrhagic erosions were found on the abomasal leaves. Lungs and liver showed circulatory disturbances, such as hyperemia, oedema and stasis.

Bacteriological investigations: From necrotic areas in the mouth was found a moderate growth of coli bacteria while lungs and spleen reavealed a rich gowth of coli bacteria.

No. 4. Received February 20. Female, 6 years.
The animal was in a moderate state of nutrition. Present om the right lateral part of the tongue was a 2 cm deep and 8 cm long wound. A 1x4 cm large shallow ulceration covered with pus was found on the hard palate, along the molars of the left side.

Histologically all mucous membranes of the oral cavity revealed focal vesicle formations in the epithelium, inter- and intracellular oedema, degenerative changes of the nuclei and cytoplasmic, eosinophilic inclusion bodies. Areas with sloughed necrotic epithelium and ulcer formation were also present in different parts of the oral cavity. The ulcers were frequently covered with colonies of fungi and bacteria and similar colonies were found in necrotic foci of the *epithelium lamina propria* and the submucosa at varying depths. Plant particles were also found in these necrotic areas. Filamentous bacterial colonies typical for *Fusobacterium necrophorum* were present in som ulcerated areas.

The submucosa, with no inflammatory response towards bacteria or fungi, presented areas with marked oedema but almost no infiltration of leucocytes.

An acute sero-fibrinous pericarditis and an acute fibrinous-necrotizing pneumonia were also present. In the necrotized parts of the lungs areas were found with filamentous bacterial colonies, typical for *F. necrophorum* but fungi were present as well.

Bacteriological investigation: It was not possible to prove infection with *F. necrophorum* but growth of non-haemolyzing streptococci was obtained from lung and spleen.

No. 5. Received Februar 20. Male, 9 months.
The carcass was cachectic. No other lesions were found than those connected with inanition.

No. 6. Received February 26. Female, 5-6 years.
The animal was pregnant and in a poor nutritional status. Present on the dorsolateral aspect of the tongue was a large (8x3 cm) sharply demarcated ulceration covered by a yellowish exudate.

The histological investigation showed an acute purulent, necrotizing glossitis. In additon the epithelium revealed vesicle formations, inter- and intracellular oedema, degenerative changes of the nuclei and cytoplasmic eosinophilic inclusion bodies. The lungs presented a purulent necrotizing pleuro-pneumonia with a sequestration 4-5 cm in diameter. There were also an acute embolic nephritis, acute splenitis and lymphadenitis.

The bacteriological investigation proved the presence of a rich growth of *C. pyogenes* in the material from the tongue while a rich growth of coli bacteria was obtained in material from lung, liver, and spleen.

No. 7. Received April 15. Female, 11 months.
Tha animal was in a poor nutritional state. Wounds in the oral cavity were not found. The major finding was a necrotizing pleuro-pneumonia and pericarditis. Histologically and bacteriologically the presence of *F. necrophorum* could be verified in the lungs.

No. 8. Received April 15. Male, 2 years.
The animal was cachectic. In the oral cavity was found fodder impactions between the teeth but also focal areas of necrotizing glossitis and somatitis histologically. The epithelium revealed vesicle formations, inter- and intracellular oedema, degenerative changes of nuclei and cytoplasmic, eosinophilic inclusion bodies and purulent necrotizing ulcers in which were found bacterial colonies typical for *F. necrophorum.*

Two, well demarcated necrotic areas with a diameter of about 2 cm were found in the rumen. In connection with these areas was a fibrinous peritonitis. The histological investigation of the rumen wall revealed necrotic changes and bacterial colonies typical for *F. necrophorum.*

The bacteriological investigation confirmed the presence of *F. necrophorum.*

Electron microscopical investigations

The investigations were affected by the fact that the material obtained showed varying degrees of post mortal changes. The presence of intracytoplasmic inclusion bodies was confirmed. Some cells contained more then one inclusion body. They appeared round or oval with a single membrane and with a granular protein-like content. Numerous inclusions, however, were only partly filled or almost empty. Inclusions were regularly located close to and compressing the nuclei into a crescent shape. The compressed nuclei showed a marked condensation of the chromatin but with areas of less electrone density in which varying patterns of granular and fibrillar components were present. In these areas were also regularly observed oval or elongated structures with an average diameter of 40-50 nm. Cells either revealed dispersed cytoplasmic organelles or a condensation of the cytoplasm with an increased electron density. A constant feature was clumping of tonofilaments and inter- and intracellular oedema.

Serological investigations

The results are shown in Table 1.

DISCUSSION

In reindeer mouth lesions, not seldom infected with *Fusobacterium necrophorum*, have been observed several times in connection with supplementary feeding (Rehbinder and Nordkvist, 1983), while outbreaks of foot rot, which earlier were not uncommon (Horne, 1897; Nordkvist, 1966; Skjenneberg and Slagsvold, 1968), today are rare (Rehbinder and Nordkvist, 1983).

The necropsied cases were, by the owners, all considered to have died from loss of apetite and fever. The majority of the cases (five out of eight), at autopsy, revealed mouth lesions (glossitis, gingivitis and stomatitis) and of these, two were infected with *F. necrophorum* (Table 2).

Table 2. Pathological findings and bacterial infections.
Tabell 2. Patologiska fynd och bakteriella infektioner.

No. *Nr.*	Sex *Kön*	Age *Ålder*	Diagnosis *Dagnos*	Bacterial infection *Bakteriell infektion*
1	♀	8 months	Necrotizing gingivitis Alveolar cell pneumonia Purulent pleuritis	ß-haemolyzing streptococci (gingiva) Coli (lungs)
2	♂	8 months	Sequestration of hoofbone Sepsis	*Corynebacterium pyogenes* (hoof) Coli
3	♀	6 years	Inanition. Ulcerative stomatitis. Vesicle formation, intracytoplasmic inclusion bodies.	Coli (whole case)
4	♀	6 years	Ulcerative necrotizing glossitis and gingivitis. Vesicle formation, intracytoplasmic inclusion bodies.	*Fusobacterium necrophorum* (gingiva, tongue) (by histology) Non-haemolyzing streptococci (lung, spleen)
5	♂	8 months	Inanition.	-----
6	♀	5-6 years	Purulent necrotizing glossitis. Vesicle formation, intracytoplasmic inclusion bodies. Purulent necrotizing pleuropneumonia.	*Corynebacterium pyogenes* (tongue) Coli (lung, liver, spleen)
7	♀	10 months	Necrotizing pleuropneumonia and pericarditis	*Fusobacterium necrophorum* (lung) (by histology and bacteriology)
8		2 years	Necrotizing stomatitis and glossitis Vesicle formation, intracytoplasmic inclusion bodies Necrotizing ruminitis	*Fusobacterium necrophorum* (mouth and rumen) (by histology and bacteriology)

One of the remaining animals died from necrotizing pleuro-pneumonia and pericarditis caused by *F. necrophorum*. The entrance port of the infection could not be determined. Of the additional two animals one died from sepsis emanating from a hoof lesion infected by *C. pyogenes* while the other died from inanition most problably due to indigestion. Thus the main finding in the autopsied animals, is the epithelial lesions in the oral cavity in five of the cases. The histological and electron microscopical investigations may be indicative of a virus infection but the micrographs did not, however, reveal any virus particles.

As the material, concerning all cases, was obtained a considerable time after the death of the animal no conclusive alterations but for intracyto---plasmic inclusions, vesicle formations and epithelial ulcers could be established.

The prevalence of antibodies against BVD-virus, in reindeer has earlier been demonstrated (Elazhary *et al.* 1981; Dieterich, 1981). In this investigation none of the tested animals had antibodies against BVD. In addition the histological picture of the lesions found in the necropsied animals did not indicate infection with BVD-virus.

Some animals had low antibody titres against Pi_3-virus (Table 1) but the oral lesions found in the necropsied animals can not be connected with this disease.

Antibodies against *Chlamydia* has been reported from Finnish reindeer (Neuvonen, 1976). Its significance for the health of reindeer has not been established. Of the tested animals eight were

considered positive. The morphology of the oral lesions did not, however, indicate any *Chlamydia* infection.

In Finland Ek-Kommonen *et al.* (1982) found a high frequency of antobodies against IBR and Dietrich (1981) reports on similar results from Alaska. According to Ek-Kommonen *et al.* (1982) the presence of a closely related cross-reacting herpes-virus can not be excluded.

It seems apparent that none of the serologically investigated agents are responsible for the epithelial lesions found in the oral cavity of five out of eight necropsied animals.

The histopathological and electron microscopical investigations rather indicates similarities with pox-virus infections (Fenner, 1979; Crandell and Grosser 1974, Pospischil and Bachman, 1979).

Already in 1897 Horne described the entity of «foot rot» in reindeer, as often producing two different diseases at the same time, one affecting the hoof, the other affecting the oral cavity. Also Skjenneberg and Slagsvold (1968) reports on a mouth disease which can be quite reminiscent of calf diphteria. Nikolaevskii (1961) reports on a little known disease of the skin of the extremities and the lips of reindeer with vesicle formation and rapid developement.

Principally any lesion of the oral mucosa wether caused by trauma, foreign bodies, parasites or an infection may give different bacteria the possibility to penetrate into the submucosa and underlying tissues. Thus the varying pattern of the mouth lesions may depend on the type of the secondarily invading bacteria.

The possibility of a primary virus infection, as indicated in this investigation, may explain the rapid spread of mouth lesions in herds of reindeer as reported by Horne (1897), Nikolaevskii (1961), Nordkvist (1966) and Skjenneberg and Slagsvold (1968) and observed in this investigation.

Attempts to isolate a virus have, however, not yet been performed.

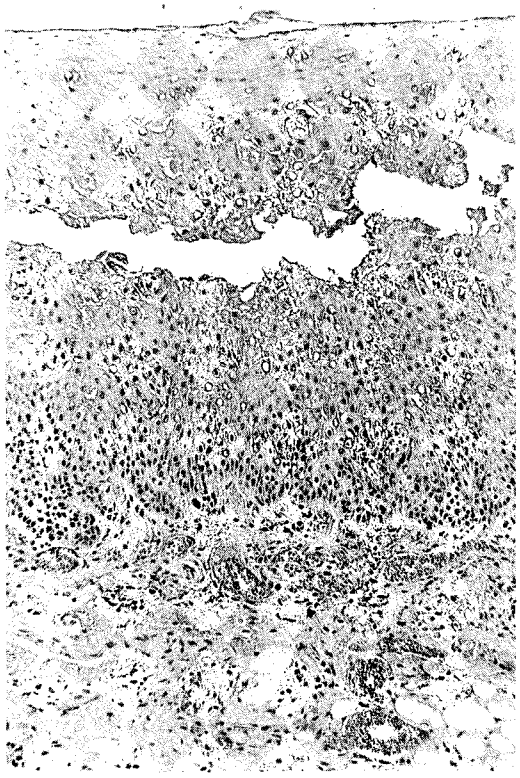

Plate 1. Lip of reindeer no. 3. Note vesicle. HE x 36.

Bild 1. Läpp från ren nr. 3. Lägg märke till vesikelbildningen. HE x 36.

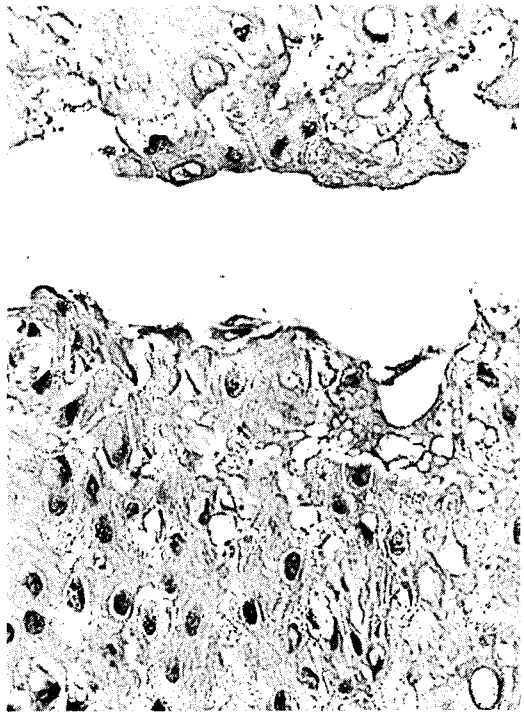

Plate 2. Lip of reindeer no. 3. Note vesicle and vesicle formation, inter- and intracellular oedema and necrotized epithelial cells. HE x 280.

Bild 2. Läpp från ren nr. 3. Lägg märke till vesikel och vesikelbildning, inter- og intracellulärt ödem och nekrotiserande epitelceller. HE x 280.

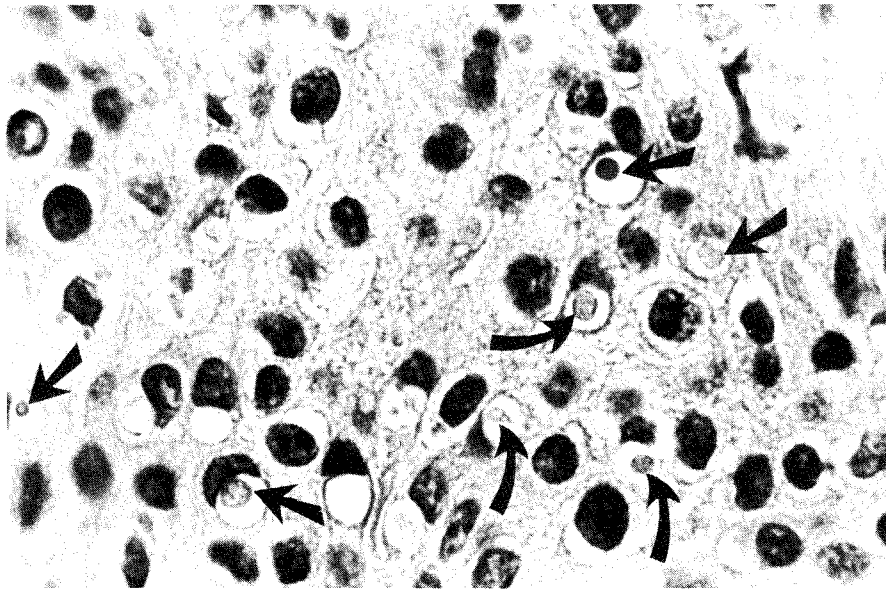

Plate 3. Lip of reindeer no 3. Note numerous intracytoplasmic inclusion bodies (arrows). Some of them compressing the nucleus into a crescent shape. HE x 1000.

Bild 3. Läpp från ren nr. 3. Lägg märke till de rikligt förekommande intracytoplasmatiska inklusionskropparna (pilar). En del av dem ihoppressande kärnan till halvmånform. HE x 1000.

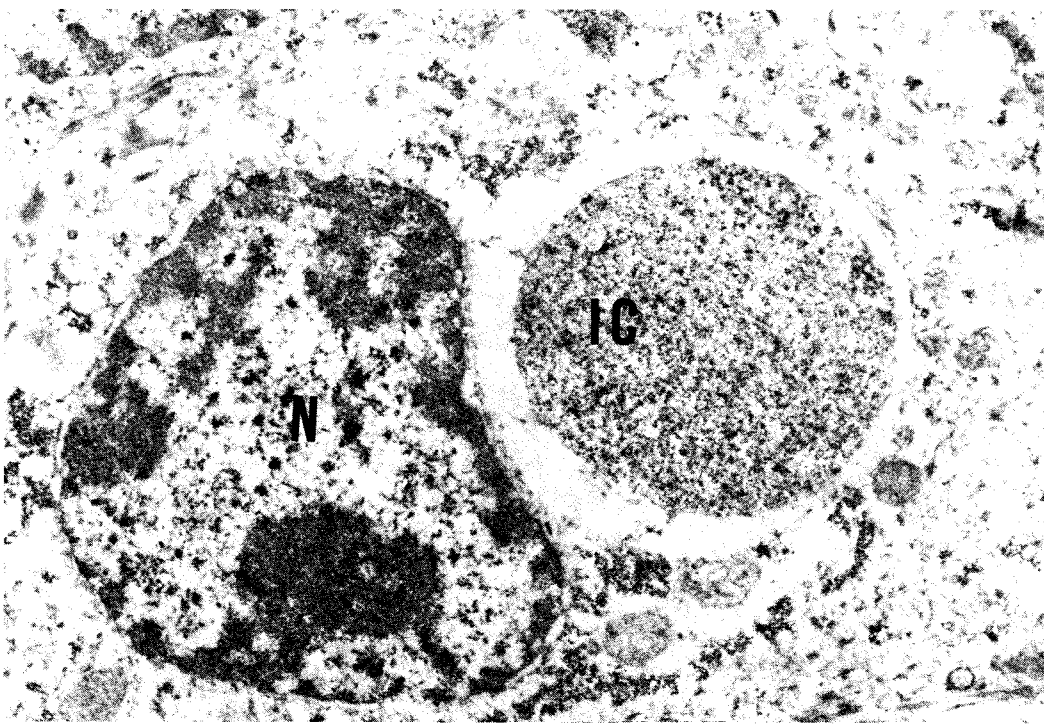

Plate 4. Tongue of reindeer no. 4. Cell with intracytoplasmic inclusion body (IC) partly compressing nucleus (N). x 10.300.

Bild 4. Tunga från ren nr. 4. Cell med intracytoplasmatisk inklusionskropp (IC) delvis sammanpressande kärnan (N). x 10.300.

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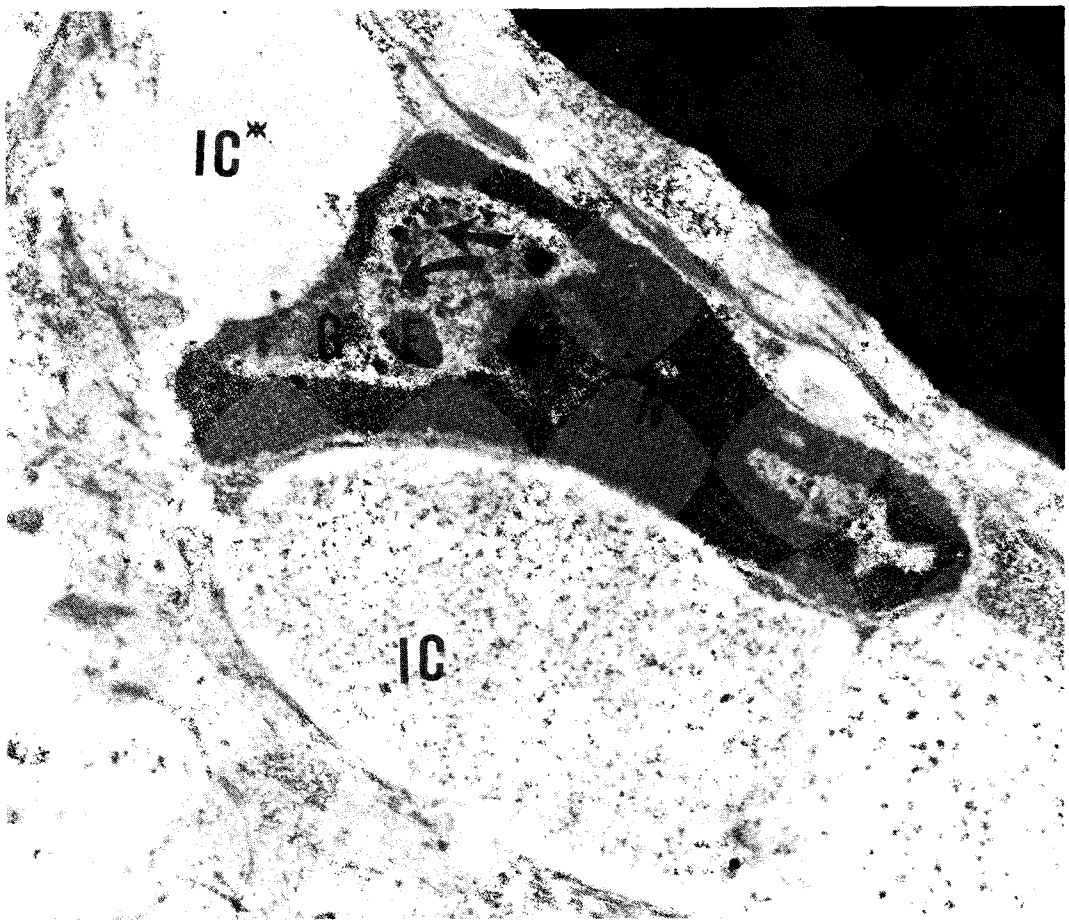

Plate 5. Tongue of reindeer 3. Cell with two inclusion bodies (IC). One is partly filled with a granular protein-like content (IC) while the other (IC*) is almost empty. Note the compressed nucleus (N) with condensated chromatin but also areas with less electron density and contining varying patterns of granular (G) and fibrillar components (F). Note also electron dense structures (arrows). x 6000.

Bild 5. Tunga från ren nr. 3. Cell med två inklusionskroppar (IC). En är delvis fylld med ett granulårt proteinliknande innehåll (IC) medan den andra (IC) är nästan tom. Lägg märke till den samanpressade kärnan (N) med kromatinkondensation med därtill också områden med mindre elektrontäthet innehållande varierande strukturer av granulära (G) och fibrillära (F) komponenter. Lägg också märke till elektrontäta strukturer (pilar). x 6000.*

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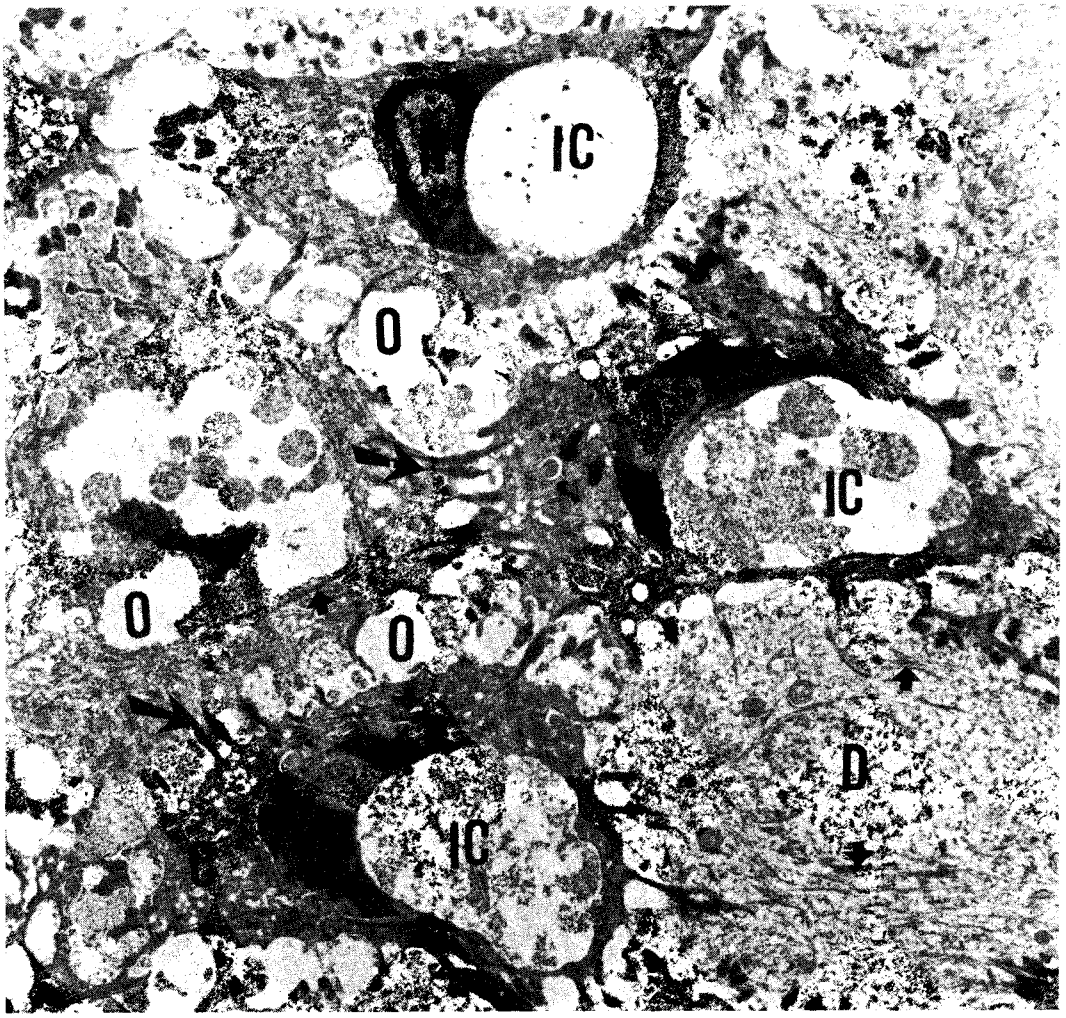

Plate 6. Lip of reindeer 3. Note inter- and intracellular oedema (O), inclusion bodies (IC), clumping of tonofilaments (small arrows), dispersed organelles (D) or a condensation of the cytoplasm (C), desmosomes (large arrows) and crescent shaped nuclei (N). x 4000.

Bild 6. Läpp från ren nr. 3. Lägg märke till det inter- och intracellulära ödemet (O), inklusionskroppar (IC), sammanklumping av tonofilament (små pilar), utspridda organeller (D) eller kondensation av cytoplasman (C), desmosomer (stora pilar) samt en halvmånformad kärna. x 4000.