THE CLINICAL HISTORY OF THE ADULT FEMALE SUMATRAN RHINOCEROS, CALLED "SUBUR", IN CAPTIVITY IN SUMATRA AND AT PORT LYMPNE ZOO, KENT, GREAT BRITAIN

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"SUBUR" IN SUMATRA

This animal, an adult female, was caught on 22nd June 1986 in a pit trap in a forest in Northern Sumatra. When caught she had a wire poacher's snare imbedded in the skin around her right forefoot at the level of the carpal joint.

Mr. Begg flew out to remove the snare and treat the injury. This was performed under anaethesia using 0.6ml Large Animal Immobilon (C. Vet Ltd.) delivered by projectile syringe. The wire was removed, the wound was cleaned and powdered and the animal received a course of oxtetracycline by injection.

During the procedure several ticks were removed from the face, behind the ears, and from the ventrum beside the udder. Two very large ticks were found near the teats.

While in captivity in a stockade built in the forest, she was fed fresh browse, cut twice a day, from the local vegetation. Approximately 95% of her diet was browse. The remainder was local fresh fruit. Attempts were made to wean her onto lucerne hay imported from Australia to facilitate feeding once in England. The hay had first to be dipped into crushed pineapple before Subur would eat it. She consumed only very small quantities.

Her body condition while in captivity in the forest was fair. No signs of clinical disease, apart from her foot injury were noted. The snare wound healed rapidly and without complications.

A dung sample taken shortly after capture contained no parasitic ova.

"SUBUR" AT PORT LYMPNE

She arrived on the evening of Tuesday 26th August 1986. She had been transported in a suitable crate, had not suffered undue stress during transport, and emerged from the crate into her stall in a satisfactory condition. There were no signs of high excitement or fear.

In the days following her arrival, two things became apparent. First, she was upset by people approaching her, particularly during the day, and second, she was eating only the bare minimum. She appeared suspicious of leafy twigs and branches, but ate bananas, figs and a little fresh lucerne, all dipped in honey. She drank large amounts of water in the week after arrival. As time passed, her consumption of fresh tropical and some British fruit increased, but she never browsed on the leaves and twigs offered to her in the same way that the male "Torgamba" did. She first ate some birch twigs on September 3rd.

After approximately five weeks her appetite for roughage did increase, and she began to consume oak, silver birch, and maple leaves, in addition to all the fruit. Progress was made and she may even have gained a little weight.

Then she suddenly ate nothing for three days beginning on 8th October. She was given a dose of multivitamins to boost her appetite on 10th October and stimulate her metabolism. After a further two days her appetite returned and a much greater quantity of food was

consumed over the following weeks, including apples, bananas, oranges, figs, carrots, parsnips, potatoes, bread as well as browse.

Her skin required spraying with arachis oil during her sojourn indoors, to prevent cracking. From 3rd October she was given access on a continual basis to the outdoor pen, and this made a big difference to her. She used the wallow to roll in and to defecate in every day. She continued to make excellent progress until the morning of Wednesday 29th October 1986, when she was found lying on her side, in some discomfort, and reluctant to rise to her feet.

CLINICAL MANAGEMENT

The onset of acute abdominal discomfort was observed first at approx 08.00 hours. The animal was constantly lying down and getting up. Respiration sounded forced and laboured on expiration, indicative of pain. She would allow people to touch her in contrast to her usual shyness.

At 09.00 hours the animal vomited some stomach contents, and appeared bloated. She was given 2mls Buscopan and 1.5mg Flumethasone intramuscularly. At 11.30 hours she had passed a little dung and appeared less bloated. She was given a further 20mls Buscopan i.m. and 10ml Tribrissen i.m. At 14.00 hours more dung was passed, accompanied by some flatus. She was given 5mg Flumethasone i.m.

By mid afternoon at 16.30 hours the development of bloat was quite apparent. At 17.00 hours another 20ml dose of Buscopan was given i.m. At 19.00 hours due to the continual discomfort of the animal 10mls Pethidine (500mg) was given i.m. Between 18.00 hours and 20.30 hours she regurgitated strong smelling stomach fluids and gas, but no solids. Her condition worsened as the evening progressed and it became obvious that unless the build up of gases in the abdomen was released, the rhino would die rapidly. She became recumbent.

Accordingly she was immobilised with 0.5ml Large Animal Immobilon and examined. She became tractable within five minutes. A rectal examination was performed and all the dung in the rectum manually extracted. An enema (5L) of hot water and liquid paraffin was given, followed by another 10L of warm water. She was given 50ml Clamoxyl LA and a further 30mls Buscopan i.m. Her respiration during immobilisation was laboured and 40mls Myllophyline and a whole bottle of Dopram were administered i.m. to alleviate this. The antidote to Immobilon, Revivon, was given via an ear vein to revive the animal, but her breathing became rapidly weaker and stopped altogether. Attempts were made to stimulate the animal by stamping on the chest but these were unsuccessful. Death ensued within approximately ten minutes of injection.

POST-MORTEM FINDINGS

An autopsy was performed on the carcass within two hours of death.

The gross appearance of the animal reflected poor body condition. The skin was normal and displayed a large (15cms) scar over the left gluteal region, and a second horizontal scar was recorded at the left hock joint. Both were completely healed. Hair covering the body was sparse and coloured dark brown. The mouth, nostrils, eyes, anus and vulva were normal. The left ear was badly torn in an injury sustained prior to capture. The feet appeared healthy. The total body weight was estimated at 500kgs, this estimate being based on her weight on arrival (which was known) minus the approximate weight lost over the nine weeks at Port Lympne. The carcass of the female which died at Basel Zoo in 1963 of pernicious anaemia weighed 350kgs (Groves and Kurt, 1972).

The dentition was examined and showed severe attritional wear of the upper incisors, uniformly from right to left, leaving the biting surfaces level with the gum margin. The

upper and lower cheek teeth on both sides of the mouth also showed evidence of considerable wear, being flat and smooth. A black, hardened deposit covered all check teeth. No teeth were missing. The third lower molars had erupted and appeared well worn, indicating an age well in excess of nine years. The dental formula of this species is I 1/0, C 0/1, P 3/3, M 3/3 total 28. (Groves and Kurt, 1972).

The respiratory tract showed some minor pathological and age related changes. The trachea was normal. Both lungs, coloured a uniform light maroon, showed slight loss of natural elasticity throughout the parenchyma. One thread of fibrous adhesion (a few mm thick) was observed between the right diaphragmatic lobe and the chest wall. This adhesion was not associated with any current disease process either in the lung or in the chest wall, and was therefore deemed of chronic origin and irrelevant to the immediate clinical history. No other lesions were present.

Histopathology revealed slight intrabronchial mucus and collapse of some alveoli with mild infiltration of polymorphs.

The heart, very elongated in shape, showed no lesions at all except for a 1-2cms sized area of focal congestion and myocardial haemorrhage at the apex of the heart, in the left ventricle. No endocardial discoloration was observed. The great vessels were of normal appearance when sliced open.

Under histological examination, this area of congestion in the left ventricle showed necrosis of cells with polymorph infiltration. A sample of myocardium from the right ventricle revealed no lesions.

The major pathology of the carcass was confined to the alimentary tract. The oesophasgus appeared grossly normal, showing a light brown mucosal surface thrown into several longitudinal folds. However, under microscopic examination loss of epithelium was noted, accompanied by marked inflammation of the inner surface with polymorphs and mononuclear cells. The surface layers contained well-recognisable colonies of bacteria.

The stomach contained many kgs of normal food material, consisting of a thick, flowing, green, slightly mucous gruel of fibre and fruit. There was mild gastric distension by gas when the abdominal cavity of the carcass was opened, but no tympany.

The interior of the stomach has been described as having three regions - the cardiac cuticular, the mucosal and pyloric secretory (Cave and Aumonier, 1963). However, on this occasion, only two were instantly differentiated. The washed inner surface of the stomach revealed no lesions in the hardened crusty, cornified epithelium of the cardiac cuticular region. This was abrasive, and pale yellow in colour.

The fundus or glandular part of the stomach showed mild development of petechiae along the glandular - cornified junction. Erosion or ulceration had not developed. In the centre of the fundus one patch (approx. 6cms wide) of mild subepithelial inflammation, dark maroon in colour was recorded. Acute inflammation was absent.

Under histological examination, the glandular stomach showed corrugated mucosal surface. The cut surface of a section of stomach wall taken from the glandular - cornified junction showed dark discoloration beneath the glandular mucosa and dark focal areas beneath the cornified epithelium. Evidence of a metazoan parasite (nematode) was found at the junction embedded in the mucosa. The glandular epithelium appeared cystic, with submucosal lymphocytic infiltration. A sample of normal glandular stomach wall showed minimal loss of the surface mucosa accompanied by submucosal oedema.

When the carcass was opened severe distension and discoloration of both large and small intestines was apparent. The peritoneum and associated structures, such as mesentery, were not involved in the pathology.

The intestines were unravelled and laid out for detailed examination. Associated lymph nodes were not enlarged. The duodenum we normal, showing a pale mucosa with thick circular ridges. It was empty. The jejunum contained a dirty reddish-brown slightly frothy fluid, probably a mixture of water, mucus and bile. No solids were noted. The mucosa became inflamed at the proximal jejunum, and this inflammation became progressively more severe towards and throughout the ileum. Ileal contents matched those of the jejunum. The mucosal folds were almost black in the ileum, but actual haemorrhage into the lumen had not developed. The general picture was of an acute inflammation becoming more severe as the small intestine reached the ileo-caeco-colic junction. The serosal surface of the jejunum and ileum reflected the internal inflammation by a deep red discoloration. However, no petechial or fibrinous reactions had developed.

Tissue sections of the small intestine confirmed the visible pathology. The duodenum showed some erosion of the surface epithelium. The jejunum demonstrated congestion and mucosal haemorrhage, with some villous atrophy. The ileum, the most badly affected portion of the small intestine, showed severe inflammation, accompanied by extensive villous atrophy and erosion of the epithelium itself. All three portions of the small intestine showed hypocellularity of the lamina propria.

The small intestine of this species has *valvulea conniventes*, but lacks Peyer's patches seen in the genus *Rhinoceros* (Cave and Aumonier, 1963).

The caecum was tympanic with gas when the abdominal cavity was opened. Its serosal surface appeared largely normal. The organ is pyriform and resembles an equine caecum.

Interiorly, the mucosa was unrecognisable. Caecal contents were minimal, consisting of perhaps half to one kilo of food material (green forage and recognisable pieces of fruit).

A large amount of gas was present, but this gas was not putrid or foul-smelling. The mucosa and the caecal wall exhibited an extremely severe oedematous reaction. Caecal wall thickness was approximately 2-3cms and on cut surface showed massive oedema of the inner layers of the wall. The epithelium was thrown into gelatinous "globules" the size of grapes with a slippery rubbery texture and consistency. The caecal surface was uniformly coloured a dark wine-red. There was no evidence of petechial formation or frank haemorrhage.

Under histological examination, the congestion of the mucosa and submucosa was confirmed. Some inactive Peyer's patches were noted. Interstitial oedema of the submucosa was observed.

Following examination of the caecum, the colon was inspected. There appeared to be two segments to this tract. The first, here designated the 'large colon', lay alongside the caecum and was attached to it for the greater part of the latter's length. The diameter was wide. The second portion, here designated the 'small colon' was independent of the caecum, had a small diameter and lay in one or two coils before entering the pelvic cavity.

The large colon contained a large amount (several kilos) of green food material, of a gruel like consistency. Other portions of semi-digested fruit were recognisable. The large colon was severely distended with gas when removed from the carcass.

The mucosa of this structure showed extremely severe, acute haemorrhagic inflammation with marked development of petechiae and small ecchymoses throughout its entire length. The mucosa therefore appeared an angry darkish red in colour. However no erosions or ulcerations were found. Oedema of the wall of the large colon had not developed at all, in sharp contrast to that of the caecum.

Tissue sections from the large colon examined histologically again confirmed mucosal congestion and haemorrhage. There was oedema and congestion of the submucosa accompanied by hypocellularity of the lamina propria.

The small colon showed only mild inflammation proximally, but was normal distally. There were no contents, the rectum having been already emptied manually while the animal was alive and immobilised. The enema had also helped to empty this portion of the gut. The mucosa was unremarkable. A tissue section from the distal portion of the small colon showed only submucosal oedema histologically.

The gross picture of the alimentary tract was one of a very severe acute inflammation and oedematous reaction on a massive scale localised primarily in the caecum. The jejunum, ileum and large colon were also severely affected, where inflammation was haemorrhagic.

A cluster of grape-sized lymph nodes was located at the ileo-caeco-colic junction. These showed no sign of acute activity either whole or in cross section. Of other lymph nodes elsewhere in the body, a prescapular node showed thick trabeculea containing foci of macrophages. Some atrophy was observed with scattered necrosis and evidence of granulomas. Using special stains crystalline material was noted in the macrophages. A few Gram-positive cocci were seen and Ziehl-Neelsen positive material was noted in some macrophages. The significance of these findings is not clear.

A glandular mass, "probably secreting watery mucus" has been recorded in the caecum near the ileocaecal valve in this species (Groves and Kurt, 1972). This mass was not observed during this examination, probably masked by the extensive pathology.

Other organs and systems in the body were also examined, but any lesions or features found were not related to the cause of death.

The spleen appeared normal, without evidence of previous pathology. It was slightly firm in consistency. Histologically there was minimal lymphoid tissue. Macrophage pigmentation was marked.

The liver resembled a pig's liver externally in that thick white interstitial tissue separated the lobules. There were four lobes. No major lesions were recorded. There were a few patches of white discoloration affecting the capsule only. Along the tip of the Spigelian lobe was a deposit of fat within the capsule. When this lobe was cut, a black coloured solid body approximately 2 cms long was found in one of the larger bile ducts. It was a direct cast of the duct. A similar, though smaller, spherical mass was discovered in another duct. There was no gall bladder. Hepatic parenchyma was macroscopically normal.

Histologically the hepatocytes exhibited some pigmentation and minimal vacuolation. Some pigmentation of the sinusoidal macrophages was also evident. This pigment was identified as lipofuscin by using special stains. Very small amount of haemosiderin were noted in the Kupffer cells. The small masses found in the bile ducts were laminated in structure with a few epithelial cells on the periphery. These masses were bile positive in the centres surrounded by non-bile positive material suggesting that acretion had ceased.

Both kidneys were flat ovoid bodies presenting a multilobulate architecture when sliced open longitudinally. The pelvis was wide and extensive. Both kidneys showed subcapsular haemorrhagic lesions, 1-3cms in diameter, which transpired to be small blood filled cysts. These lesions were diffusely distributed over the subcapsular tissue, with a density of approximately one lesion per $4cm^2$. They did not appear to be degenerative or inflammatory in nature. Both kidneys consisted of normal renal tissue. The left kidney held some white, flocculent urine in the pelvis.

Under histological examination tissue from the left kidney showed small subcapsular fluid filled cysts with minimal surrounding fibrosis. The glomeruli appeared healthy. The

proximal tubules showed a very thick dense basement membrane without casts or evidence of inflammation. The right kidney demonstrated identical features, but also held some pigment in tubular and glomerular cells. This pigment was identified by staining as lipofuscin.

The uterus was markedly bicornuate with a short body and compact cervix. There was no evidence at all of previous pregnancy visible when examining the blood vessels of the uterus and broad ligaments. Nor was one side of the organ larger than the other.

In the central part of one horn was a 3cm wide spherical multilobular tumour, identified as a leiomyoma. This did not communicate with the lumen of the uterus. Leiomyomas have been previously identified in the uterus of an Indian rhino (Jones, 1979). Each horn measured 30cms in length and the ovaries 5 x 2 x 1 cms. The latter were elongated and flattened. The uterus from another female measured 42 cms along the length of each horn (Garrod, 1873).

Histology of the ovary on the tumour side of the organ revealed a dense fibrous coat and a large degenerating corpus luteum (Corpus albicans) with a fibrous centre containing lymphocytes and pigment-containing macrophages. The pigment was birefringent and probably lipofuscin. No follicles were noted. The remainder of the section showed active cellular stroma with vessels with hyalinised walls.

The other ovary was composed of whorling stromal tissue containing vessels with thick but non-hyalinised walls.

Both ovaries were apparently non-cyclical. The uterus showed mild degenerative changes. The vessels in the muscularis failed to show the characteristic changes seen in multiparous mares. It is remotely possible the rhino had one calf at some time in life, but not more.

Of the body cavities the pleural cavity was empty of excess fluid but the peritoneal cavity contained approximately 1L of clear aqueous fluid.

Histologically the skeletal muscle section, taken from the quadriceps, showed a chronic low grade myopathy. The diaphragmatic muscle fibres showed a variation in size and affinity for the stain. Samples of adipose tissue were normal. Some dense fibrous tissue showed perivascular lymphocytic infiltration. The skin, of which a sample was removed from the inner left thigh, showed a very thick keratinised epidermis. The non-keratinised epidermis was approximately five cell layers thick with long rete. The papillary dermis was very vascular. The remainder of the section was composed of very dense collagenous tissue. A few perivascular lymphocytes were noted below the papillary dermis. In this region hair follicles were very sparse, but when observed they extended deeply into the dermis.

Sweat glands were absent. There were a few sebaceous glands near the skin surface.

Of the endocrine glands, the thyroid was examined. At a cellular level there was a variation in follicular size, the majority being very small. Lining epithelium was cuboidal.

The ulceration of the oesophagus was probably significant and could have been the portal of entry for bacteria. The crucial point is the age of the lesion. The inflammation was more acute than chronic, showing vessels still full of polymorphs. Macrophages and fibroblasts were few in number and appeared only at the base of the lesion in the tissue section. These would normally take 48 hours to appear.

No fractures were detected in the appendicular or axial skeleton, though due to the muscle masses present, immediate examination of bones was not performed. The carcass was later skinned and fleshed out and the bones showed no visible abnormalities. The vertebral

formula for this species is - 7 cervical, 19 thoracic, 3 lumbar, 4 sacral and 26 caudal, (Groves and Kurt, 1972). This formula reflects the huge extensive rib-cage of all rhinos. There was general hypocellularity of the lamina propria of the intestine and the lymphoid tissue was hyporeactive. These features are frequently found in zoo equids. The inflammation noted in the stomach was probably related to the nematodes present. The lesion in the left ventricle of the heart could have been due to cardiac over-exertion during the course of clinical illness. The skeletal muscle changes were mild and chronic but these types of changes are frequently observed in zoo equids.

MICROBIOLOGY

During the post-mortem procedure several samples of gut contents were taken for microbiological testing.

Examination for nematode, cestode or trematode ova in jejunal and ileal contents failed to show any such ova. Similarly no protozoa were found in these contents either.

A test using live mice, for clostridial toxin in the ileal contents proved negative. Cultures for *Salmonella* and *Campylobacter* were also negative. No virus particles were observed during electron microscopy of ileal contents.

However, the bacteriology proved informative. Ileal contents contained *Klebsiella*, *Streptococcus faecalis*, *E. Coli*, *Clostridia* and a non-haemolytic, coagulase-negative, DNase-negative *Staphylococcus albus*. Duodenal contents yielded the same organisms.

The large colon also yielded *E. coli*, *Streptococcus faecalis*, *Clostridia* and *Klebsiella* organisms. A swab from the pelvis of the left kidney yielded no bacteria. A swab from the bile duct in the liver yielded *Klebsiella* and *Streptococcus faecalis*. It is possible the liver swab was contaminated with intestinal contents.

Klebsiella spp. dominated the aerobic cultures, especially from the ileal contents and the liver. This was identified as *Klebsiella pneumoniae*, sensitive to streptomycin, tetracycline, neomycin, furazolidone, trivetrin, synulox and apramycin. It was resistant to ampicillin and compound sulphonamides.

It appeared that the rhino was carrying a huge burden of *Klebsiella* organisms in the intestinal tract at death. We examined the environment to discover the actual level of *Klebsiella* activity which the animal was exposed.

The outdoor exercise area for the Sumatran rhinos is covered with a thick layer of mixed wood chippings and peat. This is far more comfortable for the animals to walk over than the underlying concrete. The area is approximately 15 x 25 yards. Along one side lies a strip of earth which includes a mud wallow. During Subur's life at Port Lympne, both she and the male used separate entrances from their indoor quarters into the exercise yard.

Samples of the wood chip bedding were taken for analysis. At Subur's entrance the bedding contained 10^4 *Klebsiella pneumoniae* organisms per gram. A sample of bedding from the deep layers near the concrete floor yielded 10^5 *Klebsiella pneumoniae* organisms per gram, accompanied by *Klebsiella oxytoca* and other bacteria. The mud wallow yielded 10^3 *E. coli* organisms per gram. and the bedding from Torgamba's entrance yielded 10^3 *Klebsiella oxytoca* organisms per gram. A sample from the 'peated walkway', a strip of bedding nearest the building, used by both animals yielded 10 *E. coli* organisms per gram.

Swabs were taken from the walls, floor, drains, doors and heated swimming pools of all indoor facilities. Subur's quarters had been disinfected using a powerful bacteriocidal disinfectant immediately after the carcass was removed from the premises following post-mortem. These swabs were taken about five days after death.

A total of 36 swabs were taken. *Klebsiella pneumoniae* was isolated from the door of Subur's entrance to the outdoor pen, the peat on the floor of Torgamba's quarters, the steps to Torgamba's swimming pool, the floor near Torgamba's entrance to the outdoor pen, and the rubber bucket used for feeding fruit to Torgamba. *Klebsiella oxytoca* was found on the concrete floor of Torgamba's swimming pool, which was empty at the time of testing.

All other swabs from the walls, drains etc.], of both animals quarters did not yield *Klebsiella pneumoniae*, nor did a sample of fruit or fruit knives used in food preparation.

The *Klebsiella pneumoniae* from the rhino quarters was identified as the same organism as was found in Subur's intestine, being serotype 5215773. The *Klebsiella oxytoca* was typed as serotype 5254773.

A dung sample and nasal swabs from the male rhino failed to yield cultures of *Klebsiella* organisms.

SUMMARY AND CONCLUSIONS

The history of this animal must be placed into context with regard to the history of Sumatran rhinos as a whole. Up to 1959, a total of 55 Sumatran rhinos had been kept in captivity (Groves and Kurt, 1972) and more have been caught since then, of course, in the present catching operation. They have been kept in both Far Eastern zoos and European zoos. The former have the distinct advantage of being able to provide a natural or near-natural diet. The longevity of these rhinos in captivity ranges from a few days to 32 years and 8 months, for "Begum" at Regents Park Zoo, London.

Only scattered histories and clinical observations of Sumatran rhinos formerly in captivity remain. One female "Mary" procured by the Austrian consul in Singapore and sent to Vienna lost all her body hair. This was attributed to the lack of a water pool. Ullrich (1955) states the first "Subur" at Copenhagen Zoo wrenched off her horn once. The Basel Zoo female died of anaemia.

An Indian rhino, another Asian rhino species, presented to Regents Park Zoo in 1924 by the King of Nepal, died of tuberculosis (Reynolds, 1960).

Of more dubious quality were statements regarding body weight. Skafte (1961) quoted a weight of 800kgs for this species (age and sex unstated). An adult female was said to have weighed 2000kgs by Ullrich (1955).

In the wild Sumatran rhinos have been noted to consume fruit, leaves, twigs and bark, and wild mangoes and figs are especially appreciated (Evans, 1904: Hubback, 1939). Bamboo was also a delicacy (Evans 1905: Thom, 1935). Figs were also mentioned by Metcalfe (1961), who also recorded the consumption of five species of fruits and leaves of nineteen species of plants. Strickland (1967) noted that the favoured food plants of all species found in secondary forest growth, including some plant species toxic to man (for example the nettle (*Laportea microstigma*). Sumatran rhinos are known to visit salt licks (Strickland). From this information, accompanied by observations of our staff in the present catching project, it is likely the Sumatran rhino is primarily a browser and an opportunist frugivore. Certainly Mr. Begg's own observation that, while in the stockade in the forest in Sumatra, Subur's diet was 95% browse, supports this statement.

The previous Sumatran rhino called "Subur" was kept at Copenhagen Zoo, Denmark. Until she arrived in Copenhagen she had been fed on boiled rice balls once the leaves of her natural diet were no longer available (presumably when she left Sumatra - she was transported by sea). She frequently became constipated on this diet. After three weeks at Copenhagen she began to eat lucerene hay, and this formed the bulk of her diet from then onwards (Anderson, 1961). To this was added ten kilos of a mixture of potatoes, raw carrots and apples daily.

Our Subur was fed on her natural diet in captivity in Sumatra and was introduced to lucerne hay imported specially from Australia to wean her onto a manageable diet in Europe. However, the hay was unpopular. She was taken to Singapore and flown to Britain, where she arrived on 26th August, a period of eight weeks after capture. In comparison, the male rhino here "Torgamba" was kept in Sumatra for seventeen weeks before transfer to Britain. He arrived in the spring just as plenty of browse was becoming available. Subur arrived in mid-winter, on 4th December, when no fresh browse was available whatsoever, but she had already been weaned onto a replacement diet of rice, albeit a nutritionally incomplete one. Both our Subur and Torgamba were very fond of pineapple and fruit: hay dipped in pineapple was used as a method of introducing bulk and browse to both animals after arrival in Britain. However, Subur never took much of it until her appetite increased just before her death.

The vast proportion of Subur's diet during her life at Port Lympne was fruit. This imbalance, deficient in fibre, may have contributed to an unnatural bacterial flora in the gut. The huge abundance of *Klebsiella pneumoniae* in her gut at death may be related to this.

The pathology and microbiology results obtained from the post-mortem examination point to a diagnosis of acute bacterial toxaemia, caused by *Klebsiella pneumoniae*, in an aged animal in poor condition living in an environment subsequently found to be heavily contaminated with this organism. The major pathology seen in the small and large intestines must be of great significance.

The picture is complicated by the oesophageal ulcer, a lesion usually associated with obstruction or reflux oesophagitis. The regurgitation of stomach contents on the day she died could have led to inhalation pneumonia, though there was no evidence of this in the bronchial tree.

It is questionable to what extent the mild chronic myopathy influenced the course of events. This condition can either be acute or chronic and when chronic may last for many weeks. It is closely associated with the stress of unaccustomed extreme exercise, such as being chased before capture. Subur was caught in a pit trap and thus no stressful exercise was involved at all.

It is likely rhinos have died in Africa from capture myopathy. For example, four out of sixteen Black rhinos caught by chasing died at or shortly after capture (McCulloch and Achard, 1969). However, none of the ten Sumatran rhinos caught in the 1959 Dutch catching project died at or shortly after capture. We believed Subur was an aged animal, and the pathology supports this. The worn incisors, inactive ovaries, uterine tumour, plentiful lipofuscin in tissues, and thickening of the kidney basement membranes are consistent with increasing age. It is almost certain she never calved and it is therefore likely she would have contributed nothing towards the breeding of this species in captivity.

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