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Air Pollution and Domestic Animals

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1. Introduction

The Neolithic revolution, which began some 12,000 years ago in Turkey and in other parts of the Fertile Crescent, caused man to adopt a sedentary lifestyle, which on its turn speeded up the process of domestication of animals. The dog was already domesticated before this revolution and had served man as help in hunting. During hunting, man probably figured out that a few of the hunted species could be tamed easily, so subsequently other species such as the chicken, the duck, the goose, the sheep, the goat, the cow, the pig and the camel were domesticated. This implicated for these animals a life closely to their masters, many of them in stalls or in corrals. In ancient farm types, man and animals shared the same airspace, especially during winter. Alternatively, in some regions herd animals guided by shepherds were still allowed to be in the fields in relative freedom, some of them, however, only for a part of the year.

Interestingly, the horse was not domesticated by the sedentary peoples in the Middle East or those around the Mediterranean Sea, but from the nomadic people of the Eurasian steppes. Recent excavations in Kazakhstan showed that horses were ridden 5,500 years ago by the Botai people (Outram et al., 2009). About 1000 to 1500 BC the horse then enters the Near, Middle and Far East, mainly as a war animal. In those days, the horse was already an expensive animal that had to be well cared for and therefore was kept in stables. Some of these were really large, such as for example the one Pharaoh Ramses II had built for 460 horses at Piramesse 3300 years ago. According to Xenophon horses had to be stabled always. With the current knowledge this was not really smart from a veterinary point of view.

Compared to horses, cat and dog share much more indoor atmosphere with man, whereby these species become more exposed to harmful events like man. Swine, poultry and to a lesser extend cattle are exposed to natural, man-made and self-made air pollution. Furthermore, they may share their environment with their care takers for a part of the day. Therefore, studying diseases of animals living close with humans, or even sharing the same rooms, could bring clues for better understanding risk factors for human health and the pathophysiology caused by poor air quality.

2. General aspects air pollution on animals

It should be considered that, in the history of the Earth, the composition of the atmosphere has not always been ideal at every moment, yet life has evolved as we know it

today. Several huge environmental disasters occurred during the development of the Earth and countless forms of life were lost. From those few species that survived, new species have evolved. About 10 million years after the great Cretaceous-Tertiary extinction, the era of the dinosaurs suddenly had ended, the mammals subsequently entered the scene and prosper so successful that they dominated the life forms of the Eocene, which is about 55-40 million years ago. In the development of modern mammals, from the veterinary point of view also a by-product called man was created. This species managed within a relative short time to disturb the environment by the by-products of those activities that are euphemistically called the cultural development.

It was the increasing global population that caused intensive livestock production practices. The counter trade of the huge production of meat, eggs and milk resulted in the generation, accumulation and disposal of large amounts of wastes around the world. Aerosolization of microbial pathogens, endotoxins, odours, and dust particles are inevitable consequences of the generation and handling of waste material of the food production chain, originating from animals. Next to effects of the outdoor environmental air pollution, animals kept in huge facilities are exposed to and often diseased due to self-made indoor air pollution.

The effects of poor air quality on domestic animals principally can be divided in health damage caused by the in-door environment and by out-door air pollution. Pollutants may enter the system by inhalation or ingestion. In air pollution, mostly inhalation triggers the health problems, but occasionally deposition of particles from industrial exhaust on pasture land may affect health directly. Eventually, this may result in toxic residues in meat, milk or eggs without obvious clinical symptoms displayed by the animals producing these products. Problems with high dioxin levels in milk of dairy cows or zinc-induced arthritis in growing foals are examples of pasture grass contamination by deposits of smoke from nearby industrial activities.

The dog, the cat and the horse are exposed to the same health hazards as their masters regarding air pollution. Reineroa et al., (2009) reviewed the comparative aspects of feline asthma and brought evidence that important similarities between human and feline response to inhaled allergens exist. The role environmental aeroallergens, however, was only shown in a few studies, but evidence suggests that some environmental allergens can cause disease in both cats and humans. Ranivand & Otto (2008) showed in their epidemiology study that the prevalence of asthma had increased over the last 20 years in cats in a large urban city. This seems to have happened in man as well.

Animals may be involuntarily acting as sentinels for detecting potential harmful effect on the organism of indoor air pollution. From the scope of comparative pathology, diseases of domestic animals associated with adverse environmental factors may give clues to the pathophysiology of the health disorders of man caused by air pollution.

3. Effects of air pollution on animals

3.1 Production animals

Pigs, poultry, cattle, goats and to a far lesser extend sheep are kept in indoor facilities for a variable part of their life, often for all of their life. For dairy cattle, goats and sheep these facilities are quite open and air quality is to a certain degree comparable with the outdoor air quality. The quality of this air is still much better than that of the closed facilities for swine and poultry (Wathes et al., 1998). These buildings are rather closed and the natural or mechanically ventilation is via small air inlets and outlets. Indoor temperature is regulated

to create optimal growing conditions, whereby heat loss via ventilation is kept to a level that is just on the boundary of what is still physiologically tolerable. The other reasons for closing these types of buildings as much as possible are the strict bio security procedures applied in order to avoid or reduce introduction of potential infectious material via air or fomites. The temperature in the facilities for optimal growth can be quite high. For instance, one day-old broiler chicks are kept at a room temperature of 34°C the first days of the raising period. Thereafter, ambient temperature will be lowered daily by 1 °C. The high temperatures facilitate growth of fungi and bacteria especially around the drinkers where water is spilled by the animals. The most common used litter for broilers is wood shavings. Sometimes alternatives such as shredded paper, chopped straw and pulverised bark or peat may be used. The bird's respiratory tracts are challenged by dust coming off the litter. Up to 40,000 broilers may be raised in a single house, on littered floors. A production cycle of broilers only takes 42 days on average. In this period the chicks will grow from about 60 grams to about 2000 grams. Thus, by the end of the raising period, the houses are well filled with animals and their activities increase dust levels in the air. In laying birds, although stocking density is lower, this beneficial effect on pollution, however, is offset by the longer housing period. The result is a larger accumulation of manure, usually in pits, which are only emptied infrequently (Harry, 1978). Hence, it is not surprising that especially in poultry houses high concentrations of ammonia, airborne dust, endotoxin and micro organisms can be measured (Wathes et al., 1998).

Fattening pigs are kept in grid floored pens and thus are exposed to fumes of their own faeces and urine for their entire existence, which is of not more than 6-7 months. Also in many piggeries high levels ammonia, airborne dust, endotoxin and micro organisms can be found (Wathes et al., 1998).

The indoor atmosphere in swine and poultry confinement buildings thus contains toxic gases, dusts and endotoxin in much higher concentrations than those in outdoor environments. Apart from minimal ventilation, poor stable design leading to poor homogeneity of ventilation causes locally stagnant air pockets. According to Dunham (1991), recommended maximal concentrations of gases or contaminants in piggeries are: 2.4 mg dust /m³; 7 ppm ammonia, 0.08 mg endotoxin/m³, 10⁵ colony-forming units (cfu) of total microbes/m³; and 1,540 ppm carbon dioxide. Concentrations of bacteria up to 1.1 x10⁶ cfu/m³, inhalable dust content of 0.26 mg/m³ and ammonia concentration of 27 ppm have been reported to occur in facilities during winter, while at summer lower concentrations were measured (Scherer & Unshelm, 1995). Less difference between in- and outdoor temperature in summer allows better ventilation of the buildings.

A fraction of the smallest and most respirable particles are manure particles containing enteric bacteria and endotoxin (Pickrell, 1991). The concentration of these airborne bacteria and endotoxin, of course, is related to the level of pen cleanliness. Regarding generated toxic gasses, ammonia concentrations in the air are primarily affected by level of pen hygiene, but also by volume of the building, pig density and pig flow management (Scherer & Unshelm, 1995). Furthermore, season plays a role as well as was shown by Scherer & Unshelm (1995). Similar factors on ammonia levels are known to play a role in farrowing units and poultry houses (Harry, 1978). Ammonia is considered as one of the most important inhaled toxicant in agriculture. Dodd & Gross (1980) reported that 1000 ppm for less than 24 hours caused mucosal damage, impaired ciliary activity, and secondary infections in laboratory animals. Since this level is nearly never achieved, it is rather the long-term, low level exposure to

ammonia that seems to be related to its ability to cause mucosal dysfunction with subsequent disrupting of innate immunity to inhaled pathogenic micro organisms (Davis & Foster, 2002). Generally, the toxic effects of chronic ammonia exposure do not extend into the lower respiratory tract (Davis & Foster, 2002).

In pigs this combined effects of ammonia and endotoxin predispose the animals to infections with viruses and bacteria, both primary pathogenic and opportunistic species. Although food producing animals appear to be capable of maintaining a high level of efficient growth in spite of marked degrees of respiratory disease (Wilson et al., 1986), at a certain level of respiratory insufficiency rapid growth can no longer be attained. In that case the production results will be uneconomically. Ventilation is often at a just acceptable level. In their overview, Brockmeier et al., (2002) summarized the facts on porcine respiratory diseases. They are the most important health problem for the industrial pork production today. Data collected from 1990 to 1994 revealed a 58% prevalence of pneumonia at slaughter in pigs kept in high-health herds. These animals originate from better farms and thus incidence of pneumonia in less well managed farms is higher. Respiratory disease in swine is mostly the result of a combination of primary and opportunistic infectious agents, whereby adverse environmental and management conditions are the triggers. Primary respiratory infectious agents can cause serious disease on their own, however, often uncomplicated infections are observed. More serious respiratory disease will occur if these primary infections become complicated with opportunistic bacteria. Common agents are porcine reproductive and respiratory syndrome virus (PRRSV), swine influenza virus (SIV), pseudorabies virus (PRV), possibly porcine respiratory coronavirus (PRCV) and porcine circovirus type 2 (PCV2) and *Mycoplasma hyopneumoniae*, *Bordetella bronchiseptica*, and *Actinobacillus pleuropneumoniae*. *Pasteurella multocida*, is the most common opportunistic bacteria, other common opportunists are *Haemophilus parasuis*, *Streptococcus suis*, *Actinobacillus suis*, and *Arcanobacterium pyogenes*.

Workers in pig or poultry facilities are exposed to the same increased levels of carbon monoxide, ammonia, hydrogen sulphide, or the dust particles from feed and manure as the animals (Pickrell, 1991). As a result, workers in swine production tend to have higher rates of asthma and respiratory symptoms than any other occupational group. Mc Donnell et al. (2008) studied Irish swine farm workers in concentrated animal feeding operations and measured their occupational exposure to various respiratory hazards. It appeared that swine workers were exposed to high concentrations of inhalable (0.25–7.6 mg/m³) and respirable (0.01–3.4 mg/m³) swine dust and airborne endotoxin (166,660 EU/m³). Furthermore, the 8 hour time weighted average ammonia and peak carbon dioxide exposures ranged from 0.01–3 ppm and 430–4780 ppm, respectively.

Lesions caused by air pollution in production animals mainly include inflammatory processes. Neoplastic diseases are rather uncommon. This holds true for animals such as swine that are mainly kept indoors, as well as for cattle and sheep that are kept a variable part of their lives outdoors. This was shown in an abattoir survey some 5 decades ago performed in 100 abattoirs throughout Great Britain during one year (Anderson et al., 1969). All tumours found in a total of 1.3 million cattle, 4.5 million sheep and 3.7 million pigs were recorded and histologically typed. Just 302 neoplasias were found in cattle, 107 in sheep and 133 in pigs. Lymphosarcoma was the commonest malignancy in all three species. Lymphosarcoma was considered as entirely sporadic, since herds with multiple cases were not found in the UK. The other form, a lentivirus infection that causes outbreaks of enzootic

bovine leukaemia was not present in the UK at those days. The 25 primary lung carcinomas in cattle were well-differentiated adenocarcinomas of acinar and papillary structure, squamous and oat-cell forms and several anaplastic carcinomas of polygonal-cell and pleomorphic types. They represented only 8.3 % of all neoplasms, occurring at a rate of 19 per million cattle slaughtered. No primary lung cancers were encountered in sheep or pigs.

Outdoor air pollution could affect farm animals kept at pastures in urban and peri-urban areas. In the past (1952), a severe smog disaster in London was reported to have caused respiratory distress of prize cattle that were housed in the city for a cattle exhibition (Catcott, 1961). It was likely the high level of sulphur dioxide that was responsible for acute bronchiolitis and the accompanying emphysema and right-sided heart failure. Since some of the city farms are located rather in the periphery of cities than in the centre, the inhaled concentrations of pollutants by production animals is likely less than the concentrations inhaled by pet animals living in the city centres or close to industrial estates.

3.2 Companion animals

Bukowski & Wartenberg (1997) described clearly the importance of pathological findings in domestic animals with respect to analysis of the effects of indoor air pollution in a review. Radon and tobacco smoke are believed to be the most important respiratory indoor carcinogens. Already 42 years ago Ragland & Gorham (1967) reported that dogs in Philadelphia had an eight times higher risk developing tonsillar carcinoma than dogs from rural areas. Bladder cancer (Hayes et al., 1981), mesothelioma (Harbison & Godleski, 1993), lung and nasal cancer (Reif et al., 1992, 1993) in dogs are strongly associated with carcinogens released by human in-door activities. In cats, passive smoking increased the incidence of malignant lymphoma (Bertone et al., 2002). By measuring urinary cotinine, passive smoking of the cats can be quantified. However, the late Catherine Vondráková (unpublished results) observed that there was no direct association with the amount of cigarettes that were smoked in a household and the level of cotinine in the urine of the family cat. Nevertheless, there was evidence that exposed cats showed reduced lung function. Measurement of lung function in small animals and in cats particularly, is difficult and usually only possible with whole body plethysmography (Hirt et al., 2007). For this purpose the cat is placed in a Perspex plethysmography box. Whether this method has sufficient accuracy is still to be proven (van den Hoven, 2007).

The effect of outdoor air pollution on companion animals, so far, has not been studied extensively. Catcott (1961) however described that in the smog incident of 1954 in Donora, Pennsylvania about 15% of the cities dogs were reported to have experienced illness. A few died. Diseased dog were mostly less than 1 year old. Symptoms were mostly mild respiratory problems lasting for of 3-4 days. Also some cats had been reported ill. Further indirect evidence exists provided by observations made during the smog disaster of 1950 in Poza Rica Mexico. Many pets were reported ill or died. Especially canary birds appeared sensitive, since 100% of the population died (Catcott, 1961). The cause of mortality in the dogs and cats, however, was not professionally established; the information was merely that what the owners had reported, when asked on the incident.

Recently, Manzo et al. (2010) reported that dogs with chronic bronchitis and cats with airways inflammatory disease are at increased risk of exacerbating their conditions if exposed to prolonged urban air pollutants. In this respect they respond similar to man. The

authors advise to suppress ongoing inflammatory processes by medical therapy and avoid exercising pets outdoors in urban areas during peak pollutant periods.

3.3 Horses

The reason for the domestication of the horse must be attributed to its athletic ability. The quieter donkey and the ox had been domesticated earlier as draft animals. The horse is one of the mammals with the highest relative oxygen uptake and therefore capable of covering long distances at high speed. The tidal volume of a 500 kg horse at rest is 6-7 L and at racing gallops 12-15 L. At rest a horse breathes 60-70 L of air per minute, which corresponds to about 100,000 L/day. During a race, the ventilation rate increases up to 1800 L /min. With this huge amount of air moving in and out the respiratory track, large quantities of dust particles are inhaled and may sediment in the airways. This on its term could have adverse consequences for lung function. Any decrease of lung function could affect the horse's performance over any distance that is longer than 400 meters. Respiratory problems have a direct impact on the racing career of racehorses, if not successfully treated. Horses that are submitted to less intensive exercise, however, can perform up to expectation for quite a long time, if they are only affected by a small decrease of lung function. This can easily be understood if one considers the huge capacity of the equine cardiopulmonary system. An overview of the physiological aspects of the sport horse is given by van den Hoven (2006).

Horses are not exposed to the negative effects of tobacco smoke or radiation, because stables and the living rooms of man mostly do not share common air spaces. Yet, this does not automatically imply that there is a healthy atmosphere in a horse stable. In those countries where horses are kept in stalls, subacute and chronic respiratory diseases are serious and common problems. In countries like New Zealand, where horses live almost exclusively outdoors, these diseases are less well known.

Many equestrian enterprises are situated in the periphery of urbanized areas. Thus urban air pollution must be considered next to the health challenge by poor indoor air quality. In the suburban and urban enterprises mostly adult animals are employed. Riding schools, racehorse training yards and fiacre horse enterprises are examples of yards that may be located in or near city parks or urban green zones. Horses on these yards are either housed in barns or in individual open-fronted loose boxes. The latter have top doors that are mostly left open (Jones et al., 1987) in order to optimize air circulation. Nevertheless, in many of these boxes due to their small doors, the minimal air change rate of 4/ hour is hardly attained (Jones et al., 1987).

The younger animals mainly are kept in rural areas, mostly at stud farms. Here they are kept out-doors partly or continuously. In winter and prior to horse auctions the youngsters will be stabled for longer periods, just to the moment that many of them will be shipped to suburban or urban enterprises. Other young animals will remain in the countryside. A special category of animals are the breeding animals. After having served in sporting events for short or longer periods in the (sub)urban environment, these animals return to the countryside. Mares are bred to stallions and are mostly kept at pasture for all day, or at least a part of the day. If housed, stables are not necessarily well designed and are as traditional as those of racehorses. Thus, exposure to poor air quality is not uncommon in broodmares. Breeding stallions, have only limited freedom, and yet remain large parts of the day in the barn. Stallion barns are mostly better designed than those for mares; often the more valuable stallions have open-front boxes.

Principally almost all horses will be exposed during a variable period of their life to air of poor quality. The sports and working horses stabled and exercised in (sub)urban regions are exposed to the air pollution caused by traffic and industrial activities too (Fig.1.). Indoor and outdoor air pollution must have an impact on the lung health of our horses. Therefore it is not unexpected that respiratory disease is a major problem for horse industries worldwide (Bailey et al., 1999).



Fig. 1. Fiacre horses of Vienna waiting for tourist. Horses are daily exposed for at least 12 hours to air of the inner city. Most horses are also housed in the city (Foto by R. van den Hoven)

Traditional stable design for horses is based on non-empirical recommendations extrapolated from studies of other agricultural species (Clarke, 1987), ignoring fundamental differences in requirements of the equine athlete. Even now in 2010, only a fraction of the horses are housed in modern well designed stables. But even in the traditional stables, with a median floor space of about 12 m² (Jones et al., 1987) stocking density is much less than with production animals. Moreover, many horses have their individual living area, but often still share a common airspace with poor air quality.

Organic dust in the common or individual air space, released by moving of bedding and hay is the main pollutant in horse stables (Ghio et al., 2006). Sometimes dust levels in stalls are less than 3 mg/m³, but during mucking out, the amount increased to 10-15 mg/m³, of which 20 - 60% is of respirable particles. Measured at the level of the breathing zone, during eating of hay, dust levels may be 20-fold higher than those measured in the stable corridor (Woods et al., 1993). Dust concentrations of 10 mg/m³ are known to be associated with a high prevalence of bronchitis in humans. Apart from hay and bedding,

cereal food may contain considerable levels of dust. It has been shown that dry rolled grains may contain 30 – 60-fold more respirable dust than whole grains or grains mixed with molasses (Vandenput et al., 1997). Respirable dust is defined as particles smaller than 7 μm (McGorum et al., 1998). Respirable particles are capable of reaching the alveolar membrane (Clarke, 1987) and interact with alveolar cells and Clara cells. In this respect current findings by Snyder et al., (2011) in chemical and genetic mouse models of Clara cell and Clara cell secretory protein (CCSP) deficiency coupled with *Pseudomonas aeruginosa* LPS elicited inflammation provide new understanding on the pathophysiology of chronic lung damage. In this study, the authors reported evidence for anti-inflammatory roles of the airway epithelium and elucidated a mechanism whereby Clara cells likely regulate this process. Injured airway epithelium and mice deficient in expression of CCSP respond more robustly to inhaled LPS, leading to increased recruitment of PMNs.

Kaup et al. (1990b) mention that their ultrastructural study suggests that Clara cells are the main target for antigens and various mediators of inflammation during bronchial changes that occur in horses with recurrent airway obstruction (RAO).

The main constituents of stable dust are mould spores (Clarke, 1987) and it may contain at least 70 known species of fungi and Actinomycetes. Most of these micro organisms are not considered as primary pathogens. Occasionally infection of the guttural pouch with *Aspergillus fumigatus* may occur (Church et al., 1986). The guttural pouch is a 300 mL diverticulum of the Eustachian tube (Fig 2).

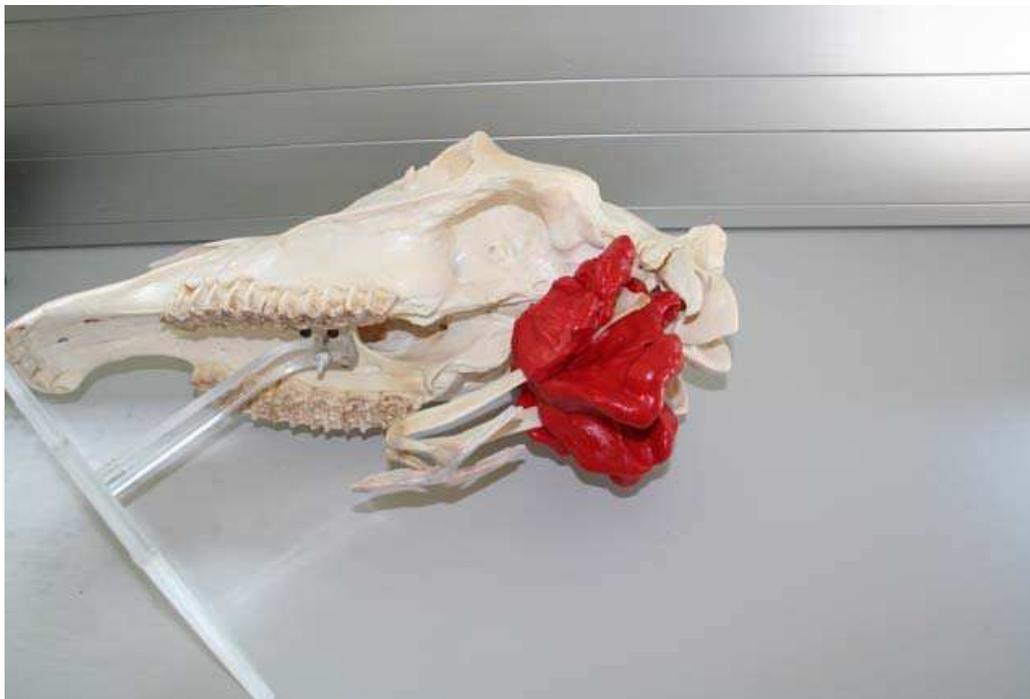


Fig. 2. Horse skull with plastinated guttural pouches. (Preparation kindly provided by Univ-Prof. Dr. Horst König, Section Anatomy, Vetmeduni, Vienna, Austria)

The walls of the guttural pouches are in contact with the base of the skull, some cranial nerves and the internal carotic artery. In case of a fungal infection of the air sac, the fungal plaque is commonly located at the dorsal roof, but may occupy the other walls as well

(Fig.3). The fungus may invade and erode the wall of the adjacent artery. The resulting haemorrhage is not easily controlled and the horse may die due to blood loss.

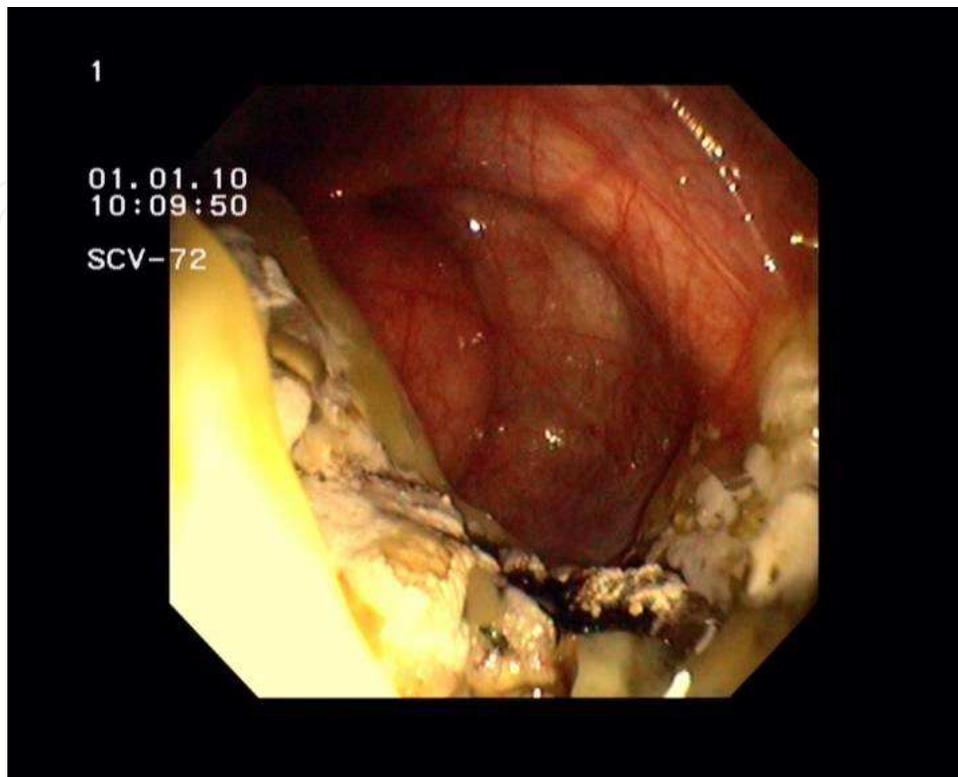


Fig. 3. Endoscopic view of the guttural pouch with a mycotic plaque

A special infection associated with inhalation of bacteria present in the dust generated by dried faeces is the pneumonia caused by *Rhodococcus equi* of young foals (Hillidge, 1986). *R. equi* is a conditional pathogen causing disease in immunologically immature or immunodeficient horses. It can even cause disease in immunocompromised man. The key to the pathogenesis of *R. equi* pneumonia is the ability of the organism to survive and replicate within alveolar macrophages by inhibiting phagosome-lysosome fusion after phagocytosis. Only the virulent strains of *R. equi* having virulence-associated plasmid-encoded 15–17 kDa proteins (VapA) cause the disease in foals (Byrne et al., 2001; Wada et al, 1997). This large plasmid is required for intracellular survival within macrophages. Next to VapA an antigenically related 20-kDa protein, VapB is known. These two proteins however are not expressed by the same *R. equi* isolate. Additional genes carrying virulence plasmids e.g. VapC, -D and -E are known. These are co-ordinately regulated by temperature with VapA (Byrne et al., 2001). Expression of the first occurs when *R. equi* is cultured at 37 °C, but not at 30° C. Thus it is plausible that the majority of cases of *R. equi* pneumonia are seen during the summer months. The prevalence of *R. equi* pneumonia is further associated with the airborne burden of virulent *R. equi*, but unexpectedly it seems not directly to be associated with the burden of virulent *R. equi* in the soil (Muscatello et al., 2006). Only under special conditions of the soil, the virulent organisms may be a threat to foals. Dry soil and little grass and holding pens and lanes which are sandy, dry, and lack sufficient grass cover are associated with elevated airborne concentrations of virulent *R. equi*. Hence, Muscatello et al. (2006) consider that environmental management strategies aiming to reduce the level of

exposure of susceptible foals to airborne virulent *R. equi* likely will reduce the impact of *R. equi* pneumonia on endemically affected farms.

If contaminated dust is inhaled by foals of less than 5 month, pulmonary abscesses will develop (Fig. 4). Faecal contamination of pasture and stalls are a prerequisite for the bacteria to establish. Other dust-born bacterial infections are not known in the horse. The non viable components of dust appear to play a major role in the airway diseases of mature horses.

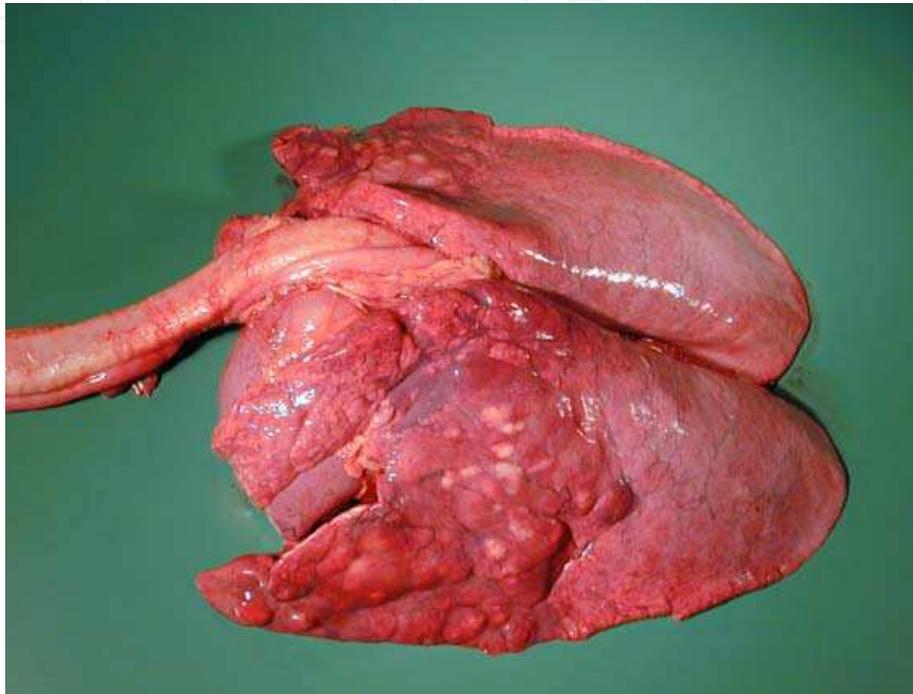


Fig. 4. Pulmonary abscesses

Any threshold limiting value (TLV) for exposure to mould spores or dust are yet not known in horses (Whittaker et al., 2009). In man working for 40 h/week in a dusty environment, the TLV is 10 mg/m³ (Anonymous, 1972). However, chronic exposure of 5 mg/m³ caused serious loss of pulmonary function in operators of grain elevators (Enarson et al., 1985). Also Khan & Nachal, 2007 showed that long-term exposure to dust or endotoxin is important for the development of occupational pulmonary diseases in man. In this respect long periods of stabling causing a cumulative exposure effect of dust and endotoxins could result in the development of pulmonary disease in both horses that are susceptible to respiratory disorders and horses that are otherwise healthy (Whittaker et al., 2009).

Generally, horses that are exposed to excess organic dust will develop mild, often subclinical lower airway inflammation. This may contribute to poor performance (see IAD). The symptoms initially seem to share common aspects with the organic dust toxic syndrome in man (van den Hoven, 2006). Some horses could show severe hyperreactivity to organic dust and will display asthma-like attacks after exposure (see RAO). Especially the feeding of mouldy hay is a well-known risk factor for this (McPherson et al., 1979). Commonly incriminated allergens for such sensitive horses are the spores of *Aspergillus fumigatus* and endotoxins. The specific role of β -glucans is still in discussion.

The origin of the moulds may be found in the feedstuff offered to horses. Buckley et al. (2007) analysed Canadian and Irish forage, oats and commercially available equine

concentrate feed and found pathogenic fungi and mycotoxins. The most notable fungal species were *Aspergillus* and *Fusarium*. Fifty per cent of Irish hay, 37% of haylage and 13% of Canadian hay contained pathogenic fungi. Apart from problems by inhalation, these fungi may produce mycotoxins that are rather ingested with the feed than inhaled. T2 and zearalenone appeared to be the most prominent. Twenty-one per cent of Irish hay and 16% of pelleted feed contained zearalenone, while 45% of oats and 54% of pelleted feed contained T2 toxins.

Next to fungal antigens, inhaled endotoxins induce a dose dependent airway inflammatory response in horses (Pirie et al., 2001) and even a systemic response on blood leucocytes can be observed (Pirie et al., 2001; van den Hoven et al., 2006). Inhaled endotoxins in horses suffering RAO are likely not the only determinants of disease severity, but do contribute to the induction of airway inflammation and dysfunction (Pirie et al., 2003).

Whittaker et al. (2009) measured total dust and endotoxin concentrations in the breathing zone of horses in stables. Dust was collected for six hours with an IOM MultiDust Personal Sampler (SKC) positioned within the breathing zone of the horse and linked to a Sidekick sampling pump. The study confirmed earlier studies that forage has a greater effect on the total and respirable dust and endotoxin concentrations in the breathing zone of horses than the type of bedding.

Due to absence of slurry pits under their living area and the low stocking density, noxious gases generated indoors generally play a less important role in development of equine airway disease. Nevertheless, with poor stable hygiene, ammonia released from the urine by urease producing faecal bacteria may contribute to airway disease too.

The effect of air pollution on horses working in the open air has not been extensively studied, but the few studies performed on ozone showed that horses appear less susceptible to the acute effects of ozone compared to humans or laboratory animals (Tyler et al., 1991; Mills et al., 1996). Marlin et al. 2001 found that the anti-oxidant activity of glutathione in the pulmonary lining fluid is likely a highly efficient protective mechanism in the horse. Although it is not likely that ozone is a significant risk factor for the development of respiratory disease in horses, the ability of ozone to act in an either additive or synergistic way with other agents or with already existing disease can not be neglected. Foster (1999) described that this occurs in humans. Diseases associated with poor air quality are follicular pharyngitis, equine inflammatory airway disease and recurrent airway obstruction.

In man exposed to air pollution in large cities, respirable particles and toxic gas levels appear to be associated with acute and subacute cardiopulmonary mortality (Neuberger et al., 2007). Such effects have not been noticed in horse exposed to urban air pollution.

3.3.1 Follicular pharyngitis

Follicular pharyngitis in horses causes narrowing of the pharyngeal diameter and increased upper respiratory airway resistance with impairment of ventilation at high speeds. The symptoms are a snoring noise at in- and expiration during high-speed exercise. The disease is easily detected by endoscopy (Fig. 5.). The disease was previously attributed to a variety of viral infections, but according to Clarke et al. (1987) it must be considered as a multi factor disease. The disease is mostly self limiting within a variable time interval.

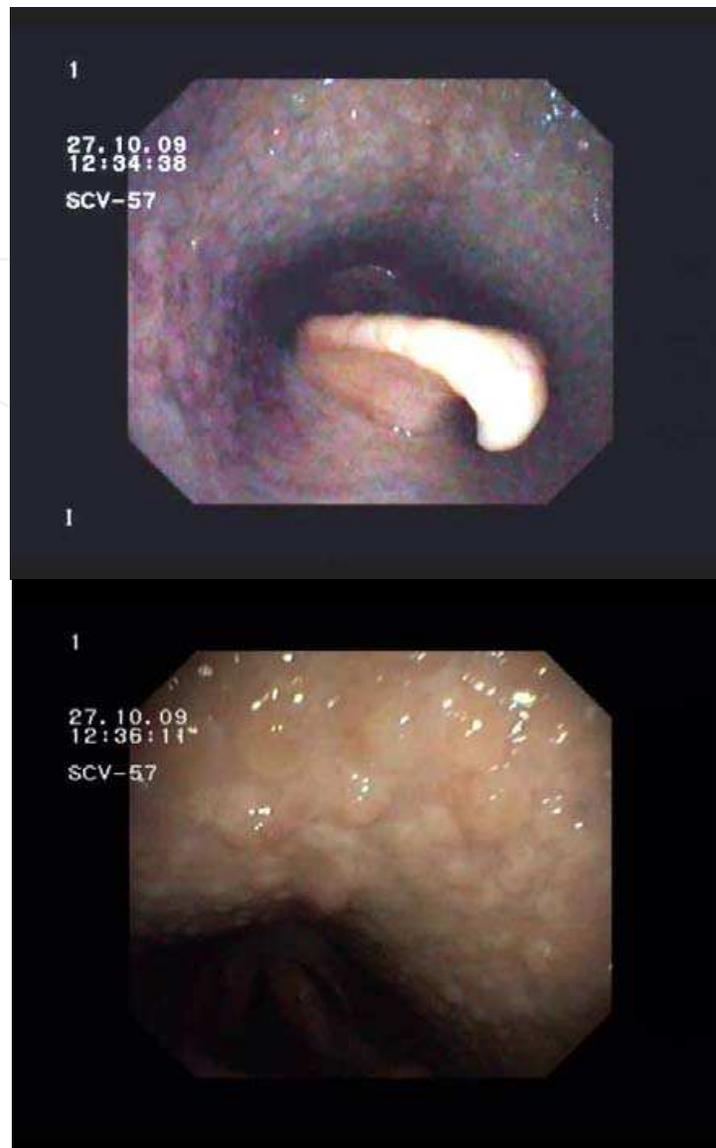


Fig. 5. Endoscopic view of follicular pharyngitis. Upper panel: swollen pharynx and an oblique view on the side and apex of the epiglottis. Lower panel: a closer view of the large lymphoid follicle of the dorsal pharyngeal wall

3.3.2 (Sub)chronic bronchitis

Cough and nasal discharge, caused by increased mucous production in the tracheo-bronchial tree, are common problems in equine medicine. It should be noticed that horses generally have a high threshold for coughing and thus cough is a strong indication for a respiratory disorder. In fact, coughing as clinical sign has an 80% sensitivity for diagnosing tracheo-bronchial disorder. Today, endoscopy is the common technique to diagnose respiratory diseases. For this purpose, 3 meter long human colonoscopes are inserted via the nasal passages and the rima glottis into the trachea. The scope is further advanced into the larger bronchi. Via the endoscope samples can be taken. Commonly, a tracheo-bronchial aspirate or a broncho-alveolar lavage (BAL) is performed. Occasionally cytobrush samples or small biopsies are collected. The endoscopic image in relation to the cytological and bacteriological findings of the samples mostly leads to the diagnosis. The use of lung

function tests in horses is only limited to those techniques that require little cooperation. Most commonly the intrapleural pressure in relation to airflow parameters is measured (Fig 6.).



Fig. 6. Intrapleural pressure and air flow measurement in a horse. Intrapleural pressure is measured via an oesophageal balloon connected to a plastic tube. Flow is measured with a Fleisch type pneumotachograph connected to an airtight facemask

The two most important and frequent forms of bronchitis in the horse are Inflammatory Airway Disease (IAD) and Recurrent Airway Obstruction (RAO). In both conditions, a variable degree of airway hyperreactivity to inhaled dust particles plays a role (Ghio et al., 2006). In the case of RAO, next to bronchiolar pathology, secondary changes in the larger airways and in the alveoli will develop.

3.3.2.1 Inflammatory Airway Disease (IAD)

IAD is a respiratory syndrome, commonly observed in young performance horses (Burrell 1985; Sweeney et al., 1992; Burrell et al. 1996; Chapman et al. 2000; Wood, et al. 1999; Christley et al. 2001; MacNamara et al. 1990; Rush Moore et al. 1995), but it is not exclusively a disease of the younger horse. Gerber et al. (2003a) showed that many asymptomatic well-performing show-jumpers and dressage horse have signs of IAD. These horses are generally 7-14 years, which is older than the age of affected flat race horses that mostly is between 2 to 5 years.

Although a universally accepted definition of IAD does not exist, a working definition was proposed by the International Workshop on Equine Chronic Airway Disease. IAD is defined as a non-septic airway disease in younger, athletic horses that does not have a clearly defined aetiology (Anonymous, 2003). This approach was reconfirmed in the ACVIM Consensus Statement (Couëtil, 2007).

The incidence of IAD in thoroughbred and standardbred racehorses is estimated between 11.3 and 50% (Burrell 1985; Sweeney et al., 1992; Burrell et al. 1996; Chapman et al. 2000; Wood, et al., 1999; MacNamara et al., 1990; Rush Moore et al., 1995).

The clinical symptoms are often so subtle, that they may go unnoticed. In that case, disappointing racing performance may be the only indication for the presence of IAD. Endoscopic examination is the major help in diagnosing IAD. Mucous accumulation in the airways is commonly observed. The result of cytology of collected BAL fluid (BALF) samples is an important parameter for diagnosing the disease. Various inflammatory cells can be seen in cytopspins of BALF samples (Fig. 7.). In contrast to RAO, slightly increased numbers of eosinophil granulocytes may be observed.

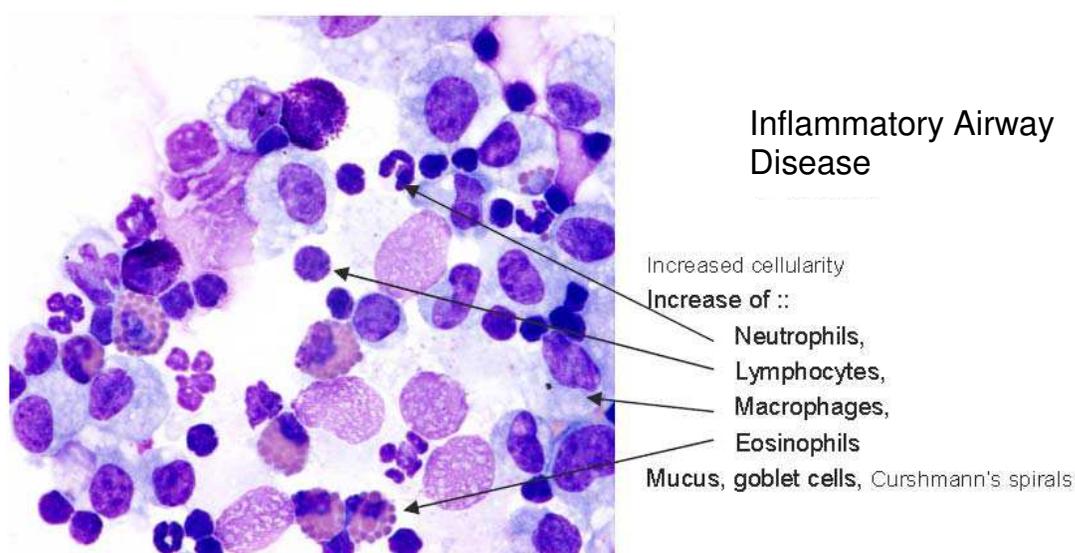


Fig. 7. Cytospin of BALF of a horse with IAD. Romanowsky stain (Foto kindly provided by Dr. C.Tumel, Veterinary School of Toulouse)

There is consensus that the clinical symptoms (Anonymous, 2003; Couëttil, 2007) should include airway inflammation and lung dysfunction. However clinical signs are rather obscure and lung function test may only show very mild changes in respiratory resistance. At endoscopy the horses may have accumulated secretions in the trachea without necessarily displaying cough. Therefore, in contrast to other respiratory disorders, cough is an insensitive indicator of IAD in racehorses. IAD in racehorses seems to diminish with the time being in a training environment (Christley et al., 2001).

Respiratory virus infections do not appear to play a direct role in the syndrome (Anonymous, 2003), but there is still no consensus on their indirect role in the development of IAD. Bacterial colonisation of the respiratory mucosa is regularly detected (Wood et al., 2005). This could be associated with decreased mucociliary clearance. Poor mucosal clearance on its term could be the result of ciliar damage by dust or toxic gases such as ammonia. Common isolates include *Streptococcus zooepidemicus*, *S. pneumoniae*, members of the Pasteurellaceae (including *Actinobacillus* spp), and *Bordetella bronchiseptica*. Some studies have demonstrated a role for infections with Mycoplasma, particularly with *M. felis* and *M. equirhinis* (Wood et al., 1997; Hoffman et al., 1992).

It is estimated, however, that 35% to 58% of IAD cases are not caused by infections at all. Fine dust particles are assumed to be the trigger of these cases (Ghio et al 2006). Once IAD has established, long-term stay in conventional stables does not seem to worsen the

IAD symptoms (Gerber et al., 2003a). Christley et al. (2001) reported that intense exercise, such as racing, may increase the risk of developing lower airway inflammation. Inhalation of dust particles from the track surface or of floating infectious agents may enter deep into the lower respiratory tract during hard exercise and cause impairment of pulmonary macrophage function together with altered peripheral lymphocyte function (Moore, 1996). In theory, intense exercise in cold weather may allow unconditioned air to gain access to the lower airways and cause airway damage (Davis & Foster, 2002), but studies in Scandinavia showed unequivocal results.

Many authors (Sweeney et al., 1992; Hoffman, 1995; Christley et al., 2001; Holcombe et al., 2001) consider the barn or stable environment the important risk factor for development of respiratory disease in young horses. Interestingly, a study in Australia by Christley et al. (2001) reported that the risk of development of IAD decreased with the length of time horses were in training and thus stabled. An explanation for this finding is the development of tolerance to airborne irritants, a phenomenon that has been demonstrated in employees working in environments with high grain dust levels (Schwartz et al., 1994). IAD of the horse partly fit within the clinical picture of the human organic dust toxic syndrome (ODTS). Some evidence for this idea was presented by van den Hoven et al. (2004) et al., who could show inflammation of airways caused by nebulisation of *Salmonella* endotoxin.

3.3.2.2 Recurrent airway obstruction

Recurrent airway obstruction (RAO) is a common disease in horses. In the past, it used to be known as COPD, but as the pathophysiological mechanisms are more similar to human asthma than to human COPD, the disease is called RAO since 2001 (Robinson, 2001). The disease is not always clinically present, but after environmental challenge, horses show moderate to severe expiratory dyspnoea, next to nasal discharge and cough (Robinson, 2001). Exacerbation of disease is caused by inhalation of environmental allergens, especially hay dust, that cause severe bronchospasm and in addition hypersecretion too. The mucosa becomes swollen while accumulated mucous secretions further contribute to airway narrowing (Robinson, 2001). During remission, clinical symptoms may subside completely, but a residual inflammation of the airways and a hyperreactivity of the bronchi to nebulized histamine still remain present. A low degree of alveolar emphysema may develop as well, caused by frequent episodes of air trapping. In the past, severe end-stage emphysema was often diagnosed, but today this is rather uncommon and only sporadically occurs in old horses after many years of illness. The commonly accepted allergens that cause or provoke an exacerbation of RAO are especially spores of *Aspergillus fumigatus* and *Fusarium* spp.

Although the RAO share many similarities with human asthma, an accumulation of eosinophils in the BALF at exacerbation has never been reported. An asthma attack in humans is characterized by an early-phase response of bronchoconstriction, occurring within minutes of exposure to inhaled allergens. This phase is followed by a late asthmatic response with the continuation of airway obstruction and the development of airway inflammation. Mastcells play an important role in this early asthmatic response (D'Amato et al., 2004; Van der Kleij et al., 2004). The activation of mast cells after inhaling allergen results in the release of mastcell mediators, including histamine, tryptase, chymase, cysteinyl-leukotrienes, and prostaglandin D₂. These mediators induce airway smooth muscle contraction, clinically referred to as early-phase asthmatic response. Mastcells also release proinflammatory cytokines that, together with other mastcell mediators, have the potential to induce the influx of neutrophil and eosinophil granulocytes and the bronchoconstriction

that are involved in the late-phase asthmatic response. Activation of other type of mastcell receptors can also induce mastcell degranulation or amplify the Fc-RI mediated mastcell activation (Deaton et al., 2006).

In horses suffering RAO, such an early-phase response seems not to appear, whereas in healthy horses the early phase response does appear (Deaton et al., 2006). This early-phase response may be a protective mechanism to decrease the dose of organic dust reaching the peripheral airways (Deaton et al., 2006). Apparently in the horse with RAO, this protective mechanism has been lost and only the late-phase response will develop. The time of exposure to dust plays a determining role, as was shown by studies with exposure to hay and straw for 5 hours. This challenge caused an increase of histamine concentrations in BALF of RAO-affected horses, but not in control horses. In contrast, exposure of only 30 minutes to hay and straw did not result in a significant increase in BALF histamine concentration of RAO horses (McGorum et al., 1993b). A study of McPherson et al., 1979 showed that exposure to hay dust of at least 1 hour is needed to provoke signs. Also Giguère et al. (2002) and others (Schmallenbach et al., 1998) provided evidence that the duration of exposure to organic dust must be longer than 1 hour. They are the opinion that the necessary exposure to provoke clinical signs of airway obstruction varies from hours to days in RAO affected horses.

The role of IgE-mediated events in RAO is still puzzling. Serum IgE levels against fungal spores in RAO horses were significantly higher than in healthy horses, but counts of IgE receptor-bearing cell in BALF were not significantly different between healthy and RAO affected horses (Kunzle et al., 2007). Lavoie et al. (2001) and Kim et al. (2003) held a T-helper cell response of type 2 responsible for the clinical signs, similar to human allergic asthma. However, their results are in contradiction with results of other research groups who could not find differences in lymphocyte cytokine expression patterns in cases with exacerbation of RAO compared to a control group (Kleiber et al., 2005).

The diagnosis of RAO is made if at least 2 of the following criteria are met: expiratory dyspnoea resulting in a maximal intra pleural pressure difference (Δp_{Plmax}) > 10 mm H₂O before provocation or > 15 mm H₂O after provocation with dust or by bad housing conditions. Any differential granulocyte count of $> 10\%$ in BALF is an indication for RAO. If symptoms can be ameliorated with bronchodilator treatment, the diagnosis is totally established (Robinson, 2001). In some severe cases the arterial PaO₂ may be below 82 mmHg. After provocation with hay dust, RAO patients may reach equally low arterial oxygen levels too. Keeping the animals for 24 hours on pasture will quickly reduce clinical symptoms to a subclinical level.

The visible morphological changes are primarily located in the small airways and spread reactively to the alveoli and major air passages (Kaup et al., 1990a,b). Lesions may be focally, but functional changes may manifest themselves well throughout the bronchial tree. Bronchial lumina may contain a variable amount of exudate and may be plugged with debris. The epithelium is infiltrated with inflammatory cells, mainly neutrophil granulocytes. Furthermore, epithelial desquamation, necrosis, hyperplasia and non purulent peribronchial infiltrates may be seen. Fibrosing peribronchitis spreading in neighbouring alveolar septa was reported in severely diseased animals (Kaup et al, 1990b). The extent of these changes in the bronchioles is related to decrease of lung function, but changes may be distinctly focal in nature (Kaup et al., 1990b). Especially the function of Clara cells is important for the integrity of the bronchioles. Mildly diseased animals show loss of Clara cell granules next to goblet cell metaplasia even before inflammatory changes occur in the

bronchioles. This together with the ultrastructural alterations found by Kaup et al. (1990b) supports the idea of the damaging effects of dust and LPS. In severely affected horses Clara cells are replaced by highly vacuolated cells. Reactive lesions may be seen at the alveolar levels. These include necrosis of type I pneumocytes, alveolar fibrosis and variable degree of type II pneumocyte transformation. Furthermore, alveolar emphysema with an increase in Kohns' pores can be present. These structural changes may explain the loss of lung compliance in horses with severe RAO.

Whether there is any causal relation between RAO and IAD is not yet established (Robinson 2001; Anonymous 2003). In both disorders, however, a poor climate in the stables plays a role. It could be theorized that IAD eventually may result in RAO, but Gerber et al. (2003a) suggest there is no direct relation between IAD and RAO. In RAO the hyperreactivity induced by histamine nebulization or to air allergens is manifold more severe than in IAD, where only a mild bronchial hyperreactivity often can be shown.

Since long time, based on observations made on members of generations of horse families, it was believed that RAO has a hereditary component. Just recently Ramseyer et al. (2007) provided very strong evidence of an inherited predisposition to RAO on the basis of findings in two groups of horses. The same research group could demonstrate that mucin genes are likely to play a role too (Gerber et al., 2003b) and that the IL4RA gene located on chromosome 13 is a candidate for RAO predisposition (Jost et al., 2007). The results gathered so far suggest that RAO seems to be a polygenic disease. Using segregation analysis for the hereditary aspects of the pulmonary health status for two stallion families, Gerber et al. (2009) showed that a major gene plays a role in RAO. The mode of inheritance in one family was autosomal dominant, whereas in the other horse family RAO seems to be inherited in an autosomal recessive mode.

3.3.2.3 Silicosis

Pulmonary silicosis results from inhalation of silicon dioxide (SiO₂) particulates. It is uncommon in horses; only in California a case series has been published. Affected horse showed chronic weight loss, exercise intolerance, and dyspnoea (Berry et al., 1991).

4. Conclusion

It may be questioned whether our pets, especially dogs, cats and horses are to be considered as victims of or "Sentinels" for air pollution. They are actually victims of human activities, just like man himself. On the other hand, the dog, horse and cat breeds, as we know them today, were all bred by man during and after the process of domestication. If the horse (*equus caballus*) had not been domesticated by man, it would have become extinct long ago. The counter trade of this help is that horses have to adapt themselves to what they become offered by man. Feed, shelter, veterinary care, but also misuse and exposure to health compromising factors. Hence, horses like other companion animals and production animals are exposed to the same environmental factors as man and thus may serve as "Sentinels for environmental risks". Due to their shorter life span, dogs and cats may express health problems by adverse environment during life or at post mortem at an earlier moment than man. Horses may display chronic effects of dust inhalation that are useful observations in comparative medicine. In the opinion of the authors, the combination of veterinary and human medical epidemiological data is a very powerful tool to identify environmental risk factors for man and its animal companions.

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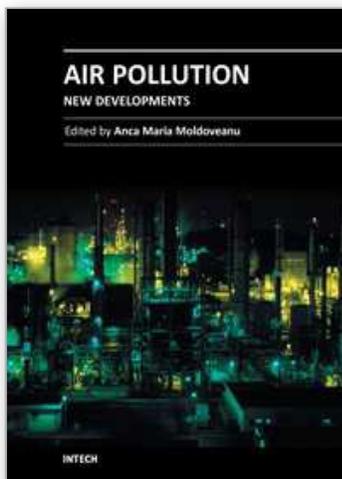
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Today, an important issue is environmental pollution, especially air pollution. Due to pollutants present in air, human health as well as animal health and vegetation may suffer. The book can be divided in two parts. The first half presents how the environmental modifications induced by air pollution can have an impact on human health by inducing modifications in different organs and systems and leading to human pathology. This part also presents how environmental modifications induced by air pollution can influence human health during pregnancy. The second half of the book presents the influence of environmental pollution on animal health and vegetation and how this impact can be assessed (the use of the micronucleus tests on *TRADESCANTIA* to evaluate the genotoxic effects of air pollution, the use of transplanted lichen *PSEUDEVERNIA FURFURACEA* for biomonitoring the presence of heavy metals, the monitoring of epiphytic lichen biodiversity to detect environmental quality and air pollution, etc). The book is recommended to professionals interested in health and environmental issues.

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