

Doctoral (PhD) dissertation theses

**Examination of pedicle diseases in deer
populations in Hungary**

**Lakatos István
Kaposvár
2025**



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2025

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Scientific publications related to the topic of the dissertation Hiba! A könyvjelző nem létezik.

1. LIST OF ABBREVIATIONS

| | |
|--------------|---|
| AAB | Aberrant antler beams |
| AANY | Aberrant antler protrusion |
| AASZ | Aberrant antler stem |
| ABKS | Antler-skin junction structure |
| ACN | <i>Acetonitrile</i> |
| ACSN | Aseptic bone necrosis |
| AF | <i>Aflatoxin</i> |
| DNS | <i>Deoxyribonucleic Acid</i> |
| ANOVA | <i>Analysis of Variance</i> |
| ASC | <i>Antlerogenic Stem Cell</i> |
| CC | <i>Capreolus capreolus</i> |
| CE | <i>Cervus elaphus</i> |
| CIC | <i>International Council for Game and Wildlife Conservation</i> |
| CT | <i>Computed Tomography</i> |
| DD | <i>Dama dama</i> |
| DICOM | <i>Digital Imaging and Communications in Medicine</i> |
| DON | <i>Deoxinivalenol</i> |
| EDC | <i>Endocrine Disrupting Chemicals</i> |
| ELISA | <i>Enzyme-Linked Immunosorbent Assay</i> |
| ER | <i>Estrogen Receptor</i> |
| FB1 | Fumonizin B1 |
| FGF-4 | <i>Fibroblast Growth Factor</i> |
| GAM | <i>Generalized Additive Model</i> |
| GCV | Overall cross-validation score |
| RH | Gonadotropin-releasing hormone |
| HPLC | <i>High Performance Liquid Chromatography</i> |
| HRP | <i>Horseradish Peroxidase</i> |
| HT-2 | <i>Fusarium trichotecén</i> mikotoxin |
| ICI | <i>IntraCranial Infection</i> |
| IFN | <i>Interferon</i> |
| IGF-I | <i>Insulin-like Growth Factor I</i> |
| IL | <i>Interleukin</i> |
| KN | Compensatory growth |
| LH | <i>Luteinizing Hormone</i> |
| MAPK | <i>Mitogen-Activated Protein Kinase</i> |
| MKCS | Morphological control group |

MS *Mass Spectrometry*
MT *Mykotoxin*
MTNY *Hungarian trophy registry*
MTR *Hungarian trophy register*
NCBI *National Center for Biotechnology Information*
NGS *Next Generation Sequencing*
NK *Natural Killer*
NTD *Neural Tube Defect*
OD *Optical Density*
OTA *Ochratoxin A*
PAS *Periodic Acid–Schiff*
PBS *Phosphate-Buffered Saline*
PCD *Pedunculitis Chronica Deformans*
PCR *Polymerase Chain Reaction*
PDJ *Peduncular-Dermal Junction*
PPR *Parathyroid Hormone-Related Peptide Receptor*
PTHrP *Parathyroid Hormone-related Protein*
RA *Retinoic Acid*
RANKL *Receptor Activator of Nuclear Factor Kappa-B Ligand*
RAPS *Rose, Antler, Pedicle, Skull*
ROS *Reactive Oxygen Species*
SOOS *Spike-on-one-side*
TGF *Transforming Growth Factor*
TMB *Tetramethylbenzidine*
TRAP *Tartrate-Resistant Acid Phosphatase*
TRKCS *Trophy register control group*
UTCI *Universal Thermal Climate Index*
VGE *Game management unit*
ZEA *Zearalenone*

2. BACKGROUND AND OBJECTIVES OF THE RESEARCH

One of the fundamental tasks of wildlife management and nature conservation is to maintain the health and vitality of wild animal population. In Hungary, diseases of the antler base in deer species pose increasing challenges, which not only result in a decline in aesthetic and hunting value but can also affect the animals' welfare, reproductive success, and chances of survival. As a dynamically developing and regressing bony structure, antlers respond sensitively to an animal's physiological condition and environmental stimuli; therefore, alterations to the antler base can serve as valuable indicators of hidden health problems or environmental stress factors.

The growing incidence of antler base diseases in Hungarian deer populations is a source of concern for game managers and researchers. These alterations are often multifactorial, complicating accurate diagnosis and the development of effective intervention strategies. Currently, comprehensive data on the prevalence, severity, and possible predisposing factors of these diseases are lacking.

In January 2017, I began my work as a district chief gamekeeper in Kapos-Tolna district no. 403. After thoroughly familiarizing myself with the area, I encountered antler base and reproductive problems in fallow deer. Discussions with specialists revealed that many theories exist regarding the background of the disease. The hypothesis of bacterial infection and the resulting condition was the most dominant, which led to the commonly used term "antler base rot." However, a review of national and international scientific literature made it clear that the symptoms of the antler base alteration observed here have not been accurately described, the true extent and epidemiology of antler base deformities remain unclear, and their causes have not been clearly elucidated. Without precise descriptions, it is impossible to distinguish between pathological changes and physiological variants. Moreover, traditional trophy evaluation methods exclude such cases showing deformities, as the affected samples do not meet the standard criteria. Consequently, few people recognize or report this condition, and its actual prevalence is unknown. In September 2017, a new research group was formed at my initiative to study the phenomenon in greater detail.

3. OBJECTIVES

1. The aim of this study was to provide an accurate morphological description and definition of antler and antler base abnormalities to enable their recognition and differentiation from other causes, such as injuries acquired during mating combats, hormonal or nutritional deficiencies, or alterations caused by injuries sustained during the velvet stage.
2. To explore the latest scientific findings and cellular processes related to antler regeneration and development.
3. To establish collections of samples and control groups that allow not only multidisciplinary examination opportunities but also analysis of environmental impacts.
4. To investigate the role of infectious agents identified in previous research and reproduce their effects using new methodological approaches. To examine the relationship between antler base disease and fatal inflammatory processes in the skull.
5. To study the exposure to mycotoxins, which are already known to be of significance in livestock management, in fallow deer.
6. To examine the effects of certain mycotoxins on the sex hormone balance of male fallow deer.
7. To study the occurrence of mycotoxins in fallow deer fetuses during early stages of pregnancy.
8. To produce results that can be applied in wildlife management practices. To present the research at an international level. To continuously inform domestic wildlife managers, offering a comprehensive presentation of the significance of the antler-problem.

4. MATERIAL AND METHOD

4.1. Ethical permission

According to the statement of the Institutional Animal Welfare Committee (NAIK MBK MÁB 004-09/2018), the study did not qualify as an animal experiment, as the researchers collected samples from the carcasses of legally harvested fallow deer stags. Consequently, ethical handling regulations do not apply to this study. Carcasses were made available for sampling by authorized game managers, fully complying with all ethical and legal requirements. Furthermore, the decision issued by the Tolna County Government Office (TO-04H39/405-2/2020) on 05.06.2020 authorized the culling of antlered fallow deer outside the hunting season for research purposes.

4.2. Research areas

Sampling was carried out in seven game management units (GMU1: Gyöng; GMU2: Törökkopány; GMU3: Gúth; GMU4: Kocsola; GMU5: Tamási; GMU6: Mészkemence; GMU7: Kelebia) in the forested areas of the Great Hungarian Plain and the South Transdanubian Hills.

4.3. Sample and data collection

The classic CIC (International Council for Game and Wildlife Conservation) trophy evaluation includes measuring age, main beam, and brow tine length, so we were able to use this data from the Hungarian Trophy Register (MTNY). From these, we formed a trophy register control group (TRKCS) from 2,912 fallow deer antler evaluation sheets, excluding those marked as "deformed," "damaged beam," or "diseased beam." The Hungarian Trophy Register (MTR) from our study region, Tolna County, contained 2,924 fallow deer records from 2017 to 2020, which included data on age, brow tine, and beam length. I examined 50 abnormal fallow deer (*Dama dama*) trophies collected in the Central European counties of Somogy and Tolna in Southwest Hungary between 2017 and 2021. A morphological control group (MCG) was used for comparison. The MCG included 24 trophies from Gúth (<https://nyirerdo.hu/guthi-erdeszeti/>), where no antler base abnormality was reported during the same period. In addition, five roe deer (*Capreolus capreolus*) and three red deer (*Cervus elaphus*) trophies were analyzed. Drawing on previous experience, by providing a detailed description and comparison of 24 healthy Morphological Control Group (MKCS) and 50 abnormal trophies, we identified those structural elements whose examination allows for the detection of antler base abnormality. The RAPS characteristics were primarily determined qualitatively, based on descriptive analysis.

For detailed morphological and histological examinations, I collected the entire heads of five fallow deer specimens with abnormal antler morphology. Additionally, with special permission from the Hunting Authority, I sampled five fallow bucks in the velvet stage, all displaying pronounced antler abnormalities, which were subjected to detailed pathoanatomical analysis, including the antler base, the surrounding cranial bone, and associated soft tissues.

The proximal, approximately 20 cm long section of the antler—or, in cases of obvious abnormality, the entire antler—along with the surrounding skull cap, including all soft tissue and the brain, was excised and fixed in 10% formalin for at least one week. These samples were cut

into 4 mm thick slices using an industrial meat bandsaw. The visibly altered areas were excised for microscopic examination. At the Department of Pathology at SZTE, we applied electric decalcification using a Tissue-tek TDE 30 Decalcifier system (ref.:1428; Sakura Finetek Europe, Alphen aan den Rijn, Netherlands) for three weeks under continuous supervision. Samples that could not be sectioned after this period were further treated in either a 70% ethanol solution containing 5% phenol for one month or in Q path DC3 (VWR ref.:09128300, Vienna, Austria) solution for three weeks.

Following decalcification, we used conventional histopathological techniques (embedding in paraffin blocks and preparing 5-micrometer sections) and cytochemical stains such as H&E, PAS, Warthin-Starry, Gram, Giemsa, Ziehl-Neelsen, and Wade-Fite to detect possible pathogens. In four cases, for one-year-old trophies, we sawed parallel, 4 mm thick slices from the antler bases, and after decalcification, we performed microscopic examinations of the slices.

From more than a hundred fallow bucks, and since reproductive problems were also observed in areas affected by antler abnormalities, I also collected samples from fallow does. The individuals were culled by authorized hunters during their usual hunting activities in the Pantanal. From 2019, over a period of three years, we also examined fallow bucks with diseased antler bases that were culled during the antler-shedding season, with special permission. During field dressing on site, I first collected 50 ml of blood from the pulmonary artery, and after coagulation, I transferred the serum into 1.5 ml Eppendorf tubes. I collected samples of the muscle, liver, kidney, stomach contents, feces, and testis or female reproductive organs, as well as skin and connective tissue samples from around the antler base. The internal organ samples were stored frozen, whereas the skin and connective tissue samples were kept refrigerated.

To determine the toxicological and hormonal parameters, during the 2019 and 2020 hunting seasons, we collected blood, liver, and muscle samples from 58 fallow deer stags showing antler abnormalities and, as controls, from 31 healthy stags.

Fallow deer doe hunts were conducted during the regular hunting season between December 2020 and January 2021. The liver and uterus, along with the developing fetuses, were removed within 3 h after the shot and stored frozen until analysis. Analyses were performed on liver samples from 70 does and 72 fetuses, as two does were carrying two fetuses. The fetuses were grouped according to age into three categories: one month, two months, and three months.

4.4. Radiological examinations

Computed tomography (CT) scans were taken of 16 aberrant trophies and two complete heads, including soft tissues, using a single-slice GE CT/e model scanner. The examination was performed by Dr. Attila Arany-Tóth at the University of Veterinary Medicine. The scanning parameters were as follows: helical mode, 512 × 512 matrix, 1 mm slice thickness, 1 mm image interval, 120 kVp, 20-120 mA, and bone convolution kernel. Transverse images of the skull were obtained perpendicular to the hard palate. The area examined extended from the occipital bone to the middle region of the orbit, including the antlers. On the DICOM images, the bony structures were evaluated using a window width of 4000 and window level of 400 HU.

4.5. Microbiological tests

Between 2018 and 2020, we examined skin and connective tissue samples excised from around the pedicles of 42 fallow deer with problematic antler bases using traditional culturing methods at the National Institute of Animal Health, Japan. In 2020 and 2024, we analyzed them using next-generation sequencing (NGS) at the Virology Laboratory of the University of Pécs.

4.6. Toxicological immunoassay studies

The examinations were carried out in the laboratories of the Reproductive Biology and Toxicology Group of the Institute of Genetics and Biotechnology at MATE. For the quantitative determination of mycotoxins, we used Enzyme-linked Immunosorbent Assay (ELISA)-based competitive immunoassay measurements. Mycotoxins were extracted from fecal, muscle, and liver samples using organic solvent extraction. ELISA measurements were performed using a Thermo Labsystems Multiskan EX ELISA reader.

To measure the zearalenone content, we used the Ridascreen ELISA kit (R-Biopharm, No.).

Aflatoxins in fecal samples were analyzed according to feed analysis standards based on a previously validated method. We applied a method specifically developed for animal organs and tissues to muscle and liver samples.

To determine the amount of deoxynivalenol, we used a quantitative competitive immunoassay kit (Toxi-Watch; Soft Flow Kft, Pécs).

For the determination of fumonisin B1 (FB1), we applied the Ridascreen (R-Biopharm, No.) ELISA kit.

For the quantitative determination of T-2 and HT-2 toxins, we used the Bio-Shield T2/HT-2 ELISA kit (Prognosis Biotech, Larissa, Greece), strictly following the manufacturer's instructions.

4.7. Serum steroid analyses

During hormone analysis, we followed the manufacturer's guidelines. Serum samples were collected during estrus and measured in triplicate using NovaTec Immundiagnostica (Dietzenbach, Germany) 17-beta-estradiol (Cat No: DNOV003), progesterone (Cat No: DNOV006), and testosterone (Cat No: DNOV002) kits.

4.8. Statistical analysis of antler characteristics

The presence of morphological abnormalities—rose, antler, antler base, and rose (RAAK)—was recorded using a value of 1 (present) and 0 (absent) separately for each side. A2.1 and A3.1 were classified as present if the length of the brow tine and antler beam was lower than the lower 20% and 5% quantiles, respectively, of the distributions for the corresponding age and side in the TRKCS. Unless stated otherwise, pairwise comparisons of means were conducted using two-sample Wilcoxon tests with Holm's multiple testing correction. Calculations were performed using the R language and environment (R Core Team (2023)); figures were plotted using ggplot2 and

ggpubr (Kassambara, 2023), ggraph (Pedersent, 2024), and pheatmap (Klode, 2019) (Sükösd et al., 2025).

4.9. Statistical analysis of hind and fetus data

We calculated the median total mycotoxin concentration in the livers of fallow deer does and fetuses. We used Kruskal-Wallis ANOVA (followed by Dunn's post-hoc test for multiple comparisons) to test for differences in mycotoxin levels between does and fetuses, and the Mann-Whitney test to assess the difference in toxin levels between does and fetuses. The Kruskal-Wallis test was used to compare mycotoxin concentrations between the different fetal age groups. To analyze the hypothetical relationship between the mycotoxin levels of fallow deer does and their fetuses, we performed generalized additive models (GAM) using the R "mgcv" (ver. 1.7-23) package. GAMs are suitable because they can detect linear or nonlinear relationships between a given response and relevant predictor variables. In our case, the mycotoxin concentration in the fetuses was considered the response variable, while the does' mycotoxin levels and their interactions were the predictors. All statistical tests were performed using R v. 4.2.3.

5. RESULTS AND DISCUSSION

5.1. Description of the symptoms of chronic antler inflammation causing deformity

To systematize the characteristics of aberrant trophies, we developed a detailed classification that examined 22 different features. These features can be divided into four main areas.

- Rose abnormalities (3 features: R1-R3)
- Antler abnormalities (6 features: (A1, A2, A2.1, A3, A3.1, A4)
- Antler base abnormalities (7 features: P1-P7)
- Skull abnormalities (6 features: S1-S6)

This system is called the RAPS features. Since combinations of these can also indicate the severity of the condition, I have defined four stages of disease progression (Figure 1), which may serve as a starting point for a future, generally accepted classification that could be applied to larger populations.

| RAPS grade | I. | II. | III. | IV. |
|------------|---|---|--|---|
| Rose |  |  |  | X |
| | Granularity absence < 1cm | Deformity | Granularity absence > 1cm | |
| Antler |  |  |  |  |
| | Supernumerary tine | Brow tine length difference | Main tine difference | Lack of antler |
| Pedicle |  |  |  |  |
| | Proximal surface pitting | Deformity/Shortening/Widening | Free/irregular casting plane, fistula | Lack of pedicle |
| Skull |  |  |  |  |
| | Pitting around the pedicle | Spongy remodeling/pedicle dislocation | Fistula | Suture/skull opening |

1. figure. Explanation of RAPS points

By examining RAPS characteristics, the severity of chronic pedicle inflammation causing deformity (pedunculitis chronica deformans, PCD) can be effectively identified and assessed.

- The abnormalities were classified as follows.
- The rose can be categorized into three degrees of severity.
- Antler, Pedicle, Skull: four degrees of severity were distinguished for each category.

To examine morphological differences in trophies, an evaluation system consisting of 22 criteria was used, which takes into account the characteristics detailed in Table 1. This comprehensive protocol ensures accurate and professional assessment of abnormalities.

1.table. Detailed description of the characteristics of RAPS

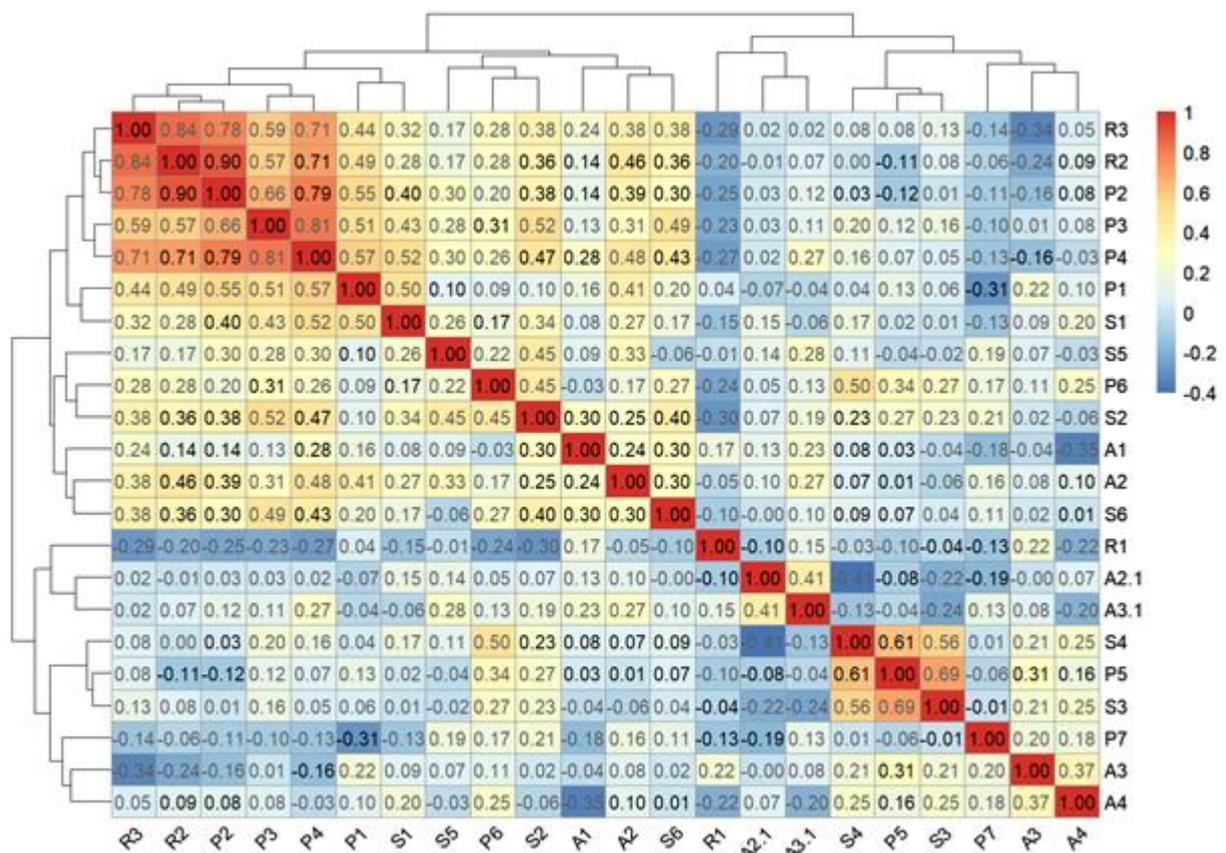
| | | |
|----------------|-------------|---|
| Rose | R1 | The discontinuity of the rose beading is longer than 5 mm but less than 1 cm. |
| | R2 | Rose deformity (can also be assessed on the castrated antlers) |
| | R3 | Loss of rose graininess greater than 1 cm. |
| Antler | A1 | An extra branch originating from the rose, which may indicate an extra antler bud. |
| | A2 | Visible difference in length between the two sides of the eye branch (several cm). |
| | A2.1 | Measurable difference from the trophy registry control group (TRKCS), which was formed from data from the Hungarian Trophy Registry, after excluding cases with records indicating antler stem abnormalities. |
| | A3 | The visible difference in length of the antler stems relative to each other (several cm). |
| | A3.1 | Measured difference compared to TRKCS |
| | A4 | Absence of antlers or only the presence of abnormal antler stems |
| Pedicle | P1 | Unevenness (rust-like roughness) on the proximal surface of the antler shaft (distally physiological) |
| | P2 | Deformity of the antler shaft, i.e. a visible difference of more than 4 mm between the two diameters of the regular circle (D and d) |
| | P3 | Shortening of the antler shaft beyond physiological limits |
| | P4 | Widening of the antler shaft beyond normal |
| | P5 | Antler fistula |
| | P6 | A free casting surface from which no antler bone protrusions protrude |
| | P7 | More than half of the antler stem's shedding plane is in or below the plane of the skull |
| Skull | S1 | The roughness extends to the surface of the skull |
| | S2 | Spongy transformation of the skull bones, where the cavities formed are larger than the intertrabecular spaces in the spongy bone |
| | S3 | Separation of the suture between the skull bones |
| | S4 | Separation of cortical bones |
| | S5 | The mouth of the fistula opens on the skull, away from the antler base. |
| | S6 | The center of the antler stem has shifted significantly, typically towards the brow ridge. |

RAPS features were classified into 4 grades based on the severity of their pathomorphological abnormalities (**Table 2**).

2. table. Severity levels of RAAK features

| | |
|------------|--------------------------------------|
| I. grade | R1, A1, P1, S1 |
| II. grade | P2, R2, A2, A2.1, P2, P3, P4, S2, S6 |
| III. grade | R3, A3, A3.1, P5, P6, S4 |
| IV. grade | A4, P7, S3, S4 |

Using the small (d) and large (D) axes of the antler bases (modeled as ellipses), I examined whether PCD caused elliptical distortion in the antler base. I quantified ellipticity based on the distance of the foci from the origin: (e), and by analyzing the eccentricity (distance between foci/D). Both measurements showed significantly different averages between the diseased and healthy cohorts (2-sample Wilcoxon test, p-values: 4.371×10^{-7} and 1.426×10^{-4}). A higher degree of ellipticity was observed in diseased specimens. I compared the frequency of RAAK characteristics between the healthy Morphological Control Group (MKCS) and groups with aberrant antlered fallow deer. In the MKCS from the northern control area (Guth), I examined 1056 morphological characteristics across 24 trophies, of which 16 (1.52%) showed anomalies. In contrast, among the aberrant fallow deer trophies from our study area (50 trophies, 2200 characteristics examined), 989 pathological features (44.95 %) were detected. Of the 22 characteristics, 20 were statistically significant discriminators (Pearson's chi-square test, **Figure 2**).



2. figure. Correlations between RAPS characteristics of the 50 examined fallow deer (*Dama dama*)

Fisher's exact test ($p < 0.0001$) confirmed that the RAAK characteristics were highly distinct between the groups. Antler abnormalities are generally considered unilateral lesions. However, my research indicated bilateral involvement stemming from a systemic disease affecting the entire organism. The observed asymmetry likely reflects varying environmental influences, primarily infection and secondary trauma. Histological and radiological analyses revealed that this phenomenon aligned with the surface form of chronic osteomyelitis, which often spread to the adjacent surfaces of the skull (S1, 64%). Rose deformity (R2, 48%) and the absence of rose granules (pearls) larger than 1 cm (R3, 53%) were also common, appearing in approximately half of the cases. In this study, R2 was significantly associated with the visible pedicle length difference (A2, 76%), whereas R3 was correlated with shortened pedicle (A2.1, 76%) and main beam (A3.1) lengths. Furthermore, both R2 and R3 were significantly associated with all pedicle and skull abnormalities, indicating that more severe rose deformities may serve as indicators of subdermal issues. Pedicle shortening and widening are physiological events associated with aging. Based on subjective assessment, we judged shortening (P3) to be more pronounced than normal in 61% of pathological cases and widening (P4) in 63%. However, pedicle deformity, defined as a deviation from a regular circular shape (P2), was observed in more than half of the cases (60%), making it a useful marker for disease detection in fallow deer but not in roe or red deer. In extreme cases, we observed lateral displacement of the center of the remaining pedicle, which often extended to the brow ridge (S6, 31%). In my work, "spikes" (a not precisely defined term in hunting circles) were defined as Aberrant Antler Processes (AANY) to distinguish pathological forms from the unbranched antlers of healthy yearling stags. If no bone-based antler projection was formed at a given site or only an AANY was present, the case was classified as "antler deficient" (A4, 40%).

A formation was considered an antler only if the main beam length exceeded that of the brow tine projection and at least a rudimentary rose was visible on the antler. Our CT scans revealed that these structures were generally solid, with only one instance of cavitation. In cases where the pedicle growth centers were less affected, more pronounced growth could occur through the incorporation of unused nutrients and minerals, which became available in greater quantities due to reduced osteoblast numbers in other areas. I have termed this process Compensatory Growth (CG). C may manifest as a significant difference in brow tine length, as a longer than normal brow tine is expected on the less affected side.

The observed skull abnormalities, such as the unevenness of the cranial surface around the antler base (S1, 64%), spongy transformation of the skull (S2, 30%), and opening of the cortical plates of the cranial bones (S6), suggest that these atypical resorptive and proliferative processes cannot be attributed to trauma or irregular scarring resulting from imperfect healing. Morphological evidence of secondary healing of chronic osteomyelitis is also indicated by the formation of fistulas, which were detectable at the antler base in 16% of cases (P5) and on the skull in 10% of cases (S4). The opening of sutures (S3, 20%) could theoretically be the result of injury; however, this cannot occur in isolation, as it would have to be accompanied by a fracture of the adjacent skull and bone displacements. Similarly, the detachment of the external cortical plate from the underlying bone layer due to trauma would have to involve a significant displacement. In contrast, CT scans of an aberrant trophy showed that the gap between the cortical plates (S5, 23%) followed the contour of the skull, suggesting that this malformation could not be the result of injury. The sharp fracture lines, whether with or without displacement, and the absence of callus formation, at least in the absence of partially broken-off antler base structures (such as an antler base with a regular circular cross-section but missing a segment), are further evidence that these abnormalities do not originate from injury.

5.2. The occurrence of aberrant trophies in different cervid species

The ratio of aberrant trophy samples to the population density of the studied area is presented in three tables. Between 2017 and 2021, the occurrence of aberrant trophies among wild deer species in southern Hungary showed a significant increase compared with the previous two decades. The occurrence of aberrant trophies was significantly higher in fallow deer (*Dama dama*) than in roe deer (*Capreolus capreolus*) and red deer (*Cervus elaphus*). In the case of fallow deer, there were ten times as many problematic trophies as there were among roe deer (50 vs. 5) and approximately 16 times as many as there were among red deer (50 vs. 3).

Based on the data adjusted for population density, it appears that fallow deer are even more affected by these anomalies, 34 times more frequently than roe deer and 50 times more frequently than red deer.

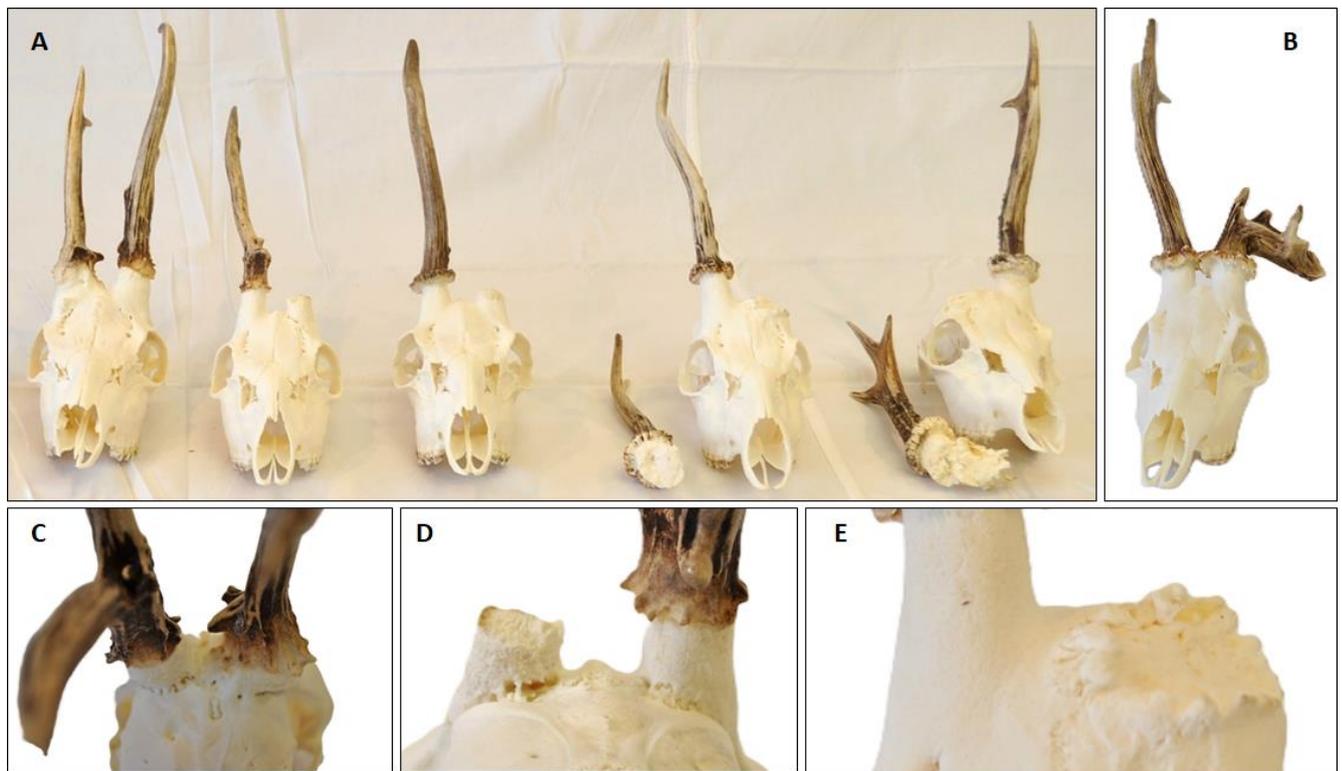
3. table. Ratio of the number of samples with aberrant trophies to the stock density of the studied area.

| | Species | | |
|--|-----------------------|------------------|----------------------------|
| | <i>Cervus elpahus</i> | <i>Dama Dama</i> | <i>Capreolus capreolus</i> |
| Abridgment | CE | DD | CC |
| Investigation of bucks in the region (2017-2020) | 5701 | 1912 | 6424 |
| Samples | 3 | 50 | 5 |
| % | 0,053 | 2,62 | 0,08 |
| Species / DD | 50 | 1 | 34 |

We identified the characteristics of chronic pedicle inflammation causing deformity in the trophies of five roe deer (*Capreolus capreolus* (CC) in my Cervidae trophy collection (Figure 3). This relatively small sample may suggest that CC is ten times less affected than DD is. Moreover, values corrected for population density data indicate an involvement rate 22 times lower. This observation does not reflect the actual proportion in the CC population, since normal trophies also exhibit great morphological variability, which significantly hinders the recognition of aberrant trophies, and trophies displaying obvious abnormalities are also excluded from the evaluation process (<https://www.cicukteb.com/uk-species/index>).

I assessed five trophies on both sides and classified the frequency of anomalies into three categories: 1. frequent (70%-100%), 2. moderately frequent (40%-69%), and 3. rare (less than 39%). In two cases (CC 4 and CC 5), one antler broke off during hunting, and the bony structure showed marked weakening, resulting in an evident pathological fracture. In these cases, we were able to evaluate the broken part and compare it with the opposite side of the same tooth. Given the low number of cases, I discussed the RAPS characteristics—that is, the involvement of the burr (R), antler (A), pedicle (P), and skull (S)—collectively.

Antler abnormalities were the most common (52%), followed by involvement of the antler base (27%) and skull (23%). Interestingly, the occurrence rate of rose anomalies (R1-R3) was significantly lower (17%) than that of DD and CE. This can be attributed to the absence of antler base deformation (P2), which is an important characteristic of PCD in DD, and R2 is not independent of P2. This interspecies difference may be due to the different distributions of osteoclasts at the antler base. In the case of CC, these bone-resorbing cells were narrowly concentrated at the casting plane, resulting in an almost even separation line when they were active. In contrast, in DD, they are more dispersed throughout the antler base, creating an uneven surface.



3.figure. Trophies of roe deer with antler problems (own figure; Sükösd et al., 2025).

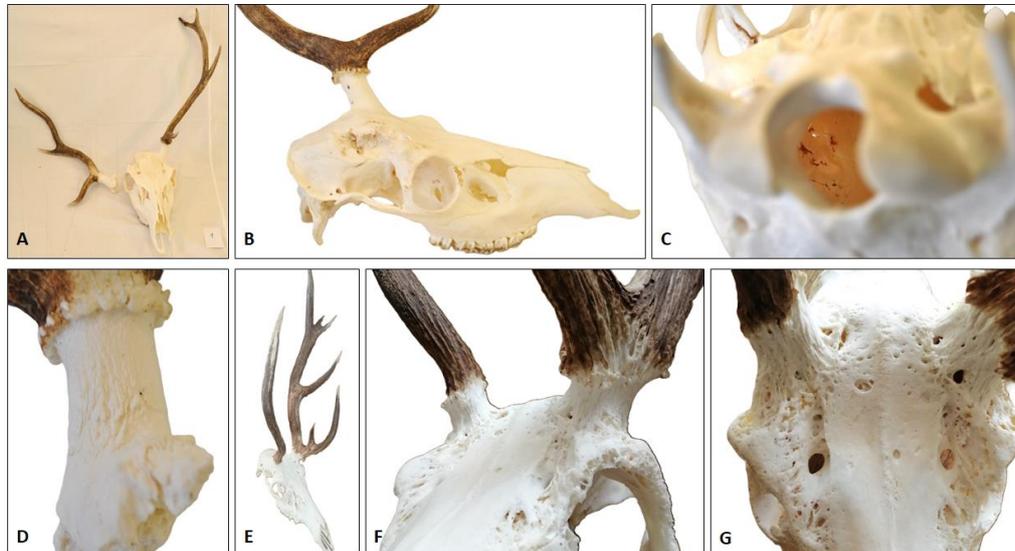
Trophy abnormalities in roe deer (*Capreolus capreolus*, CC) caused by deformity-inducing chronic pedicle inflammation (PCD). A. I ranked the five cases according to PCD severity. B. In the CC injured during the velvet stage, no abnormalities in the rose, pedicle, or skull were observed. C. Rear view of the CC1 case. (a) (b) There are no brow tines or antler beams on either side. D. Rear view of the CC2 case. Unevenness was observed on the proximal pedicle surface on both sides, most prominently on the side where early shedding occurred. On one side, a gap (fissure) formed between the pedicle and frontal bone, whereas loss of rose granulation was observed on the other side. E. On the left side of the CC4 case, a bony ridge-like edge developed at the margin of the widening pedicle base, indicating osteoblastic bone formation activity. The effect of osteoclasts does not extend beyond the pedicle, in contrast to what is seen in fallow deer (*Dama dama*, DD) or even more so in red deer (*Cervus elaphus*, CE).

Population density-adjusted data indicated a 32-fold lower prevalence in red deer (*Cervus elaphus*, CE) than in fallow deer (*Dama dama*, DD). Despite the low number of CE cases, the morphological criteria (RAAK points) for deformity-inducing chronic pedicle inflammation (PCD) showed significant overlap between the two species (**Figure 4**).

In these cases as well, at least on one side, an abnormal transformation or conspicuous weakening of the antler burr was visible. In the case of a three-year-old deer (CE 1), the right antler burr broke off during hunting, which is why it was found (P6, P7). The fracture occurred in the proximal third of the antler burr, right at the level of the skull, where both the external and internal surfaces showed spongy remodeling (S2) without any displacement of the flat bone plates (**Figure 4.c**). This relatively significant loss of bone mass proved that the holes at the base of the antler burr (clearly visible from the inside through the foramen magnum, **Figure 4. b**) could not have formed as a result of trauma because they were not located along the line of force. The roughness found on the outer skull surface (S1) around the antler burr is a clear sign of chronic osteomyelitis (see text), which is characteristic of PCD and a result of prolonged inflammation, not acute trauma. CE

1 may be an example of PCD, where damage to the antler burr began at an early stage of life, possibly even in the fetal stage. At this point, Mts may have already inhibited cartilage formation, causing aseptic bone necrosis (ACSN) and cavity formation (see text). Morphological evidence of this pathomechanism includes the intact rose, relatively minor shortening of the antler shaft (A2), roughness of the distal part of the antler burr (P1) and surface of the skull (S1), and, in severe cases, even spongy transformation of the flat bones (S2). The resulting cavity was located in the proximal third of the antler burr (similar to the one-year-old DD case, see text), weakening it, which led to the pathological fracture. The other two CE cases (CE 2 and CE 3) may be examples of the PCD pathomechanism leading to consequential scar formation due to improper wound healing and irregular bone remodeling. In these cases, this was associated with distal antler burr, rose, and antler deformation or antler loss.

One of the most striking differences between DD and CE is the extent of spongy reconstruction (**Figure 4. g**), which was not only more pronounced but also affected the frontal bones. This may be the result of osteoclast/osteoblast activity (which is most prominent at the casting plane) not being limited to the pedicle but also affecting the surrounding ectoderminally derived part of the frontal bone. Other characteristics differing from DD are the absence of pedicle deformation (P2), shortening (P3), and widening (P4), which can be explained by interspecific differences. In the case of DD, bone remodeling beneath chronic osteomyelitis shifts the balance between osteoclasts and osteoblasts in favor of the latter, as opposed to CE, where the former may be more pronounced.



4. Figure. Symptoms of deforming chronic antler disease (PCD) in red deer (*Cervus elaphus*, CE). A-D: Trophy of a two-year-old deer with a broken right pedicle, where the fracture occurred during hunting, allowing examination of both sides. The lesion may represent an early phase of PCD, in which aseptic bone necrosis leads to pathological fractures. The right antler beam was shorter and less developed than the left. B. The wide fracture surface affected most of the frontal bone and extended close to the orbital arch. C. View through the foramen magnum toward the base of the right pedicle of the atlas. Sponge-like holes were formed without displacement of the cranial bone surface. The holes were not aligned in a straight line, which would otherwise indicate a traumatic impact. D. The proximal two-thirds of the surface of the

broken pedicle showed remarkable unevenness without deformation or loss of burr granulation. E-G. The case of the five-year-old stag suggests that the inhibition of wound healing after antler shedding is a PCD-causing effect. E. The difference between the two sides is striking, with only a single aberrant antler beam developing on the right. (There is no brow tine, so it cannot be considered a true antler.) F. Complete loss of burr granulation on both sides, without shortening/widening/deforming of the pedicle, although the surface irregularity is apparent. G. Spongy transformation due to osteoclast activation is characteristic of PCD in C-E cases. On both sides, most of the frontal bone was affected, and the disease also reached the orbital arches. Unlike in fallow deer, no formation of bony ridges or widening of the pedicle was observed, which could be a sign of increased osteoblast activity.

5.3. Health status of cervids with aberrant trophies

During one hunting season, I collected samples from 58 animals with abnormal antlers and 31 control animals to assess their condition, as well as to perform toxicological and hormonal analyses. I documented the animals' physical condition based on hunting records. The physical condition of animals with deformed antlers was significantly worse. (Fisher test, $p < 6.51 \times 10^{-6}$).

Although mortality is influenced by hunting and planned culling, these factors are not independent of an animal's health condition. Our results are consistent with the findings that animals with antler deformities are often culled due to their poor health rather than the value of their trophy. Individuals with deformed antlers had a lower expected lifespan in every age group than those with normal antlers. Therefore, antler deformity should be identified as a sign of a serious illness.

5.4. Antler-skin junction structure (ABKS); structural and pathological characteristics

In diseased individuals, we observed a pronounced and noticeable separation between the soft tissues around the antler base, including the skin, subcutaneous connective tissue, and periosteum, and the underlying bone. This indicates that the tissue structure around the antler base, which is responsible for maintaining the integrity of the body's outer covering, was damaged, potentially contributing to the development of the disease. Structures passing through the body's outer covering—the first line of defense—can potentially expose the organism to pathogens from the environment entering alongside them, which, along with the dry antler, may indeed be possible. We defined the soft tissues surrounding the antler base as a distinct tissue structure, the Antler Base-Skin Coupling Structure (ABKS) (Pedunculo-Dermal Junction). We found that damage to the ABKS causes separation of the periosteum from the bone, allowing the accumulation of external contaminants, such as plant fibers, in the gap that forms. Pathogens can enter through these contaminants, leading to infection and triggering acute inflammation.

5.5. Characteristics of antler infections and bacterial pathogens

Microscopic analysis of the antler base and surrounding soft tissues revealed acute inflammation at the site. In deeper tissues, we observed chronic inflammatory cells and scar tissue. This inflammation was most prominent around the blood vessels and nerves and within scar tissue. We also found bone fragments detached from the surface of the antler base. *Trueperella pyogenes* is considered the primary pathogen responsible for inflammation of the soft tissues around the antler

base. However, a Hungarian case study identified *Staphylococcus xylosum* as a potential pathogen in this condition. *T. pyogenes*, along with other microbes, has also been detected in purulent meningoencephalitis cases within the cranial cavity, supporting its central role. In partial accordance with these findings, our study using conventional culturing methods examined 42 cases and identified *T. pyogenes* in 24 cases (57%), often in combination with other microbes. Notably, this pathogen was not detected in the remaining cases.

Our NGS analysis performed on six samples, which is also informative regarding bacteria, has shed new light on the microbial background of pedicle inflammation. Although *T. pyogenes* was detectable in all six samples examined, its relative frequency compared to that of other pathogens was strikingly low. In one case, it reached 5.2%, while in the others, it ranged between 0.1% and 1.5%. This suggests that it is present as a secondary, rather than a dominant, pathogen. It is typically found on the skin and mucosal surfaces of animals, has invasive potential, and can enter the host following minor skin injuries and proliferate in necrotic tissues. In contrast, in five out of six cases, *Fusobacterium* species were identified as the dominant bacteria, with a frequency between 39% and 78% of the microbial population, whereas in the sixth case, it was the third most common type at 19%. *Fusobacterium* species are obligate anaerobic commensals that often play a role in abscess and osteomyelitis development. It is presumed that the classical culture medium may provide a selective advantage for *T. pyogenes*; however, this is not the case with NGS. For both *T. pyogenes* and *Fusobacterium* sp. to exert their abscess-forming effect, damage to the integument is necessary. This is attributed to mating fights, which could also explain the significant difference in incidence between sexes, although it is unlikely that these injuries affect only the immediate area around the pedicle. Among our study samples, we did not find any case in which abscesses or bony lesions developed on the skull independently of the healthy pedicle, which would indicate an inoculation effect caused by head trauma. The center of the lesions was always the pedicle. In my opinion, impaired wound healing after antler shedding paves the way for pus-forming agents responsible for secondary infection.

5.6. Mycotoxicosis, hormonal disorders and their relationship with bone formation

In the absence of animal or cell culture studies examining the effects of mycotoxins on antler development and regeneration, we could only use research from other species on bone development, cell regeneration, and immune function for comparison purposes. To investigate the possible connection between antler abnormalities and exposure to mycotoxins (MTs), we measured the concentrations of the most important MTs: aflatoxins (AF), zearalenone (ZEA), deoxynivalenol (DON), and fumonisin B1 (FB1). These measurements were performed in the serum, liver, and muscle tissues of FPD-affected fallow deer and healthy control animals. Our study showed a correlation between mycotoxin and hormone levels in stags with pedicle disease.

Our analysis detected a significantly increased AF concentration in the serum ($p = 0.011$) and liver tissue ($p = 0.048$), but not in the muscle tissue ($p = 0.264$). This is consistent with the mechanism of action of AF, which involves the disruption of bone metabolism, particularly through the inhibition of protein synthesis and impairment of bone cell function. These findings suggest that AFB1 may disrupt antler formation by reducing the viability and differentiation potential of antler stem cells (ASCs).

Our measurements confirmed significantly elevated ZEA levels in both serum ($p < 0.0001$) and liver tissue ($p = 0.00025$), the latter being the organ responsible for ZEA elimination (Ropejko and Twaruzek, 2021). ZEA is a potent, relatively low-toxicity mycoestrogen; however, in utero studies in rats have shown that it delays skeletal ossification.

Our hormonal tests revealed significant differences in the serum of infected deer. One of our most important findings was the significant decrease in testosterone levels ($p < 0.0001$) and increase in estrogen levels ($p < 0.0001$), which were associated with cases exhibiting antler anomalies. The progesterone levels did not show any significant changes.

In the affected animals, the serum DON level was approximately three times higher than that in the control animals ($p = 0.021$), and the DON level in liver tissue was also significantly elevated ($p = 0.00021$). DON disrupts the delicate balance between bone formation and breakdown by inhibiting osteoblast activity while stimulating osteoclasts. This dual effect leads to bone structural weakening and fractures. DON also exerts immunomodulatory effects that are dose- and exposure-dependent. FB1 did not show any significant increase in either serum ($p = 0.48$) or liver tissue ($p = 0.49$).

We propose the following hypothesis regarding the mechanisms underlying the observed hormonal changes: the decrease in testosterone levels was linked to increased concentrations of the mycoestrogen ZEA. The rise in endogenous estrogen levels can be attributed to the compensatory activity of the hypothalamic-pituitary-gonadal axis. The synergistic effect of increased mycoestrogen ZEA and endogenous estrogen may contribute to the decline in androgen-dependent antler development.

The irregular shedding plane, which is a clear manifestation of an insufficient testosterone peak, was prominently observed among the studied antler anomalies, serving as evidence of the connection between MT effects and developmental abnormalities in the pedicle bone. This phytoestrogen effect may also explain the reproductive biology problems observed in the deer population.

We hypothesized that the cavity formation observed at the antler base of yearling stags may serve as morphological evidence of the direct cytotoxic effects of mycotoxins on bone formation. At this developmental stage, antler shedding has not yet occurred; therefore, the skin covering is still intact. Mycotoxins can enter fetal circulation from the maternal bloodstream of fallow deer through the placenta and can also be detected in maternal milk.

We assumed that these mycotoxins not only inhibit the periosteal growth centers of the antlers but also negatively affect the endochondral ossification process at the antler base, leading to the formation of a cavity partially filled with granulation tissue, that is, a lesion indicative of aseptic bone necrosis (ACSN). Despite their limitations (dried samples containing minimal tissue remnants and lack of classical formalin fixation), microscopic examination of the antler bases from yearling stag trophies confirmed evidence of necrosis and the presence of regenerative granulation tissue.

Although unilateral occurrence was observed in three-quarters of the cases, this is more likely attributable to physiological asymmetry rather than environmental effects. One case in our red deer trophy series can be traced back to the same pathomechanism: in a three-year-old stag, the

proximal part of the right antler base broke off, while the distal part and the antler itself remained essentially intact. All of these findings imply the presence of a pre-existing cavity.

Given the direct negative effects of these mycotoxins on chondrogenesis and osteogenesis, they can also be regarded as antlerotoxic, introducing a new dimension to their well-known toxicity profiles, which already include hepato-, nephro-, and neurotoxic effects. Pathological development of the antler base, together with incomplete closure of the covering skin, creates an environment that promotes secondary bacterial infection, intracranial inflammation, and abnormal fractures of the antler base and skull. This series of events can ultimately lead to serious health consequences, including death.

5.7. Deformity-causing chronic odontogenic dermatitis (PCD) as a consequence of wound healing disorders.

After antler shedding, a wound resembling an open bone fracture is formed. In healthy animals, after healing, this leaves at most a small central scar and closes with a velvet-like skin. Acute wound healing involves blood clotting, inflammatory processes, cell proliferation, and tissue remodeling. Wound healing is a complex and vulnerable process that can stall, resulting in chronic, non-healing wounds. Common triggers in such cases include infection, chronic inflammation, immunosuppression, and circulatory disturbances, which often impede healing. Although the healing of antler wounds has been extensively studied, research has not yet focused on inflammatory cells as microscopically observable indicators of immune activity or on the morphological features associated with pathological healing. Microbial contamination of wounds is unavoidable in non-sterile environments. This poses a risk to the ossification zone, as bacteria can spread through the bloodstream and cause osteomyelitis. However, if the immune system functions properly and wound closure occurs in a timely manner, inflammation is minimal and self-limiting, reflecting natural resistance to chronic inflammation and infection. If tissue regeneration is impaired and immunosuppression occurs, as in our case due to multi-mycotoxicosis, a wound healing disorder develops. These conditions can trigger an inflammatory response that exceeds physiological levels, resulting in secondary or delayed wound healing, typically involving excessive fibrosis and scar formation. This irregular scarring disrupts critical signaling pathways between the epithelium and regenerating growth centers. Morphological examination of pathological antler pedicles and aberrant antler shafts (AASZ) has shown that these structures originate from osteoblasts and osteochondroid columns scattered within irregular scar tissue. In this state, appositional intramembranous ossification occurs in the periosteum of the antler pedicle, with scarring unevenly expanding, sometimes reaching the edge of the brow ridge. This abnormal growth process results in greater-than-normal shortening and pronounced bone loss of the antler pedicle, which ultimately leads to a “bald” condition. The etiology, pathomechanism, and morphology of the lesions we describe do not fit into any single recognized disease category, but they are similar to Garré’s sclerosing chronic, non-suppurative (i.e., not pus-forming) osteomyelitis.

Therefore, PCD can be defined as a chronic inflammation affecting the bone tissue of the antler pedicle, caused by closure disorders of the shedding wound or by abnormal endochondral ossification of the pedicle. The consequent scarring leads to disruption and structural weakening of the antler pedicle and antler regeneration, creating the possibility of pathological fractures,

superinfection, and fatal intracranial inflammation. PCD, described by the characteristics of RA, can also be observed in trophies and harvested animals (Sükösd et al., 2025).

5.8. Mycotoxin test results of fallow hinds and their fetuses

Since we know nothing about the mycotoxin exposure of wild-living animals, the first step was to demonstrate the presence of mycotoxins in deer and their fetuses. Later, this fact helped us explain the phenomenon of the first antlered fallow bucks losing their antler bases. AF was present in 70% of the deer hinds, ZEA in 41%, DON in 90%, T2/HT2-toxin in 96%, and FB1 in 84% of the samples. Mycotoxin concentrations showed high individual variability in the livers of the hinds. The minimum concentration was "0" for all the mycotoxins. For ZEA, more than half of the hind liver samples, and for AF, nearly one-third did not contain the toxins. In some cases, the maximum concentrations were also outliers. AF was present in 82% of fetuses, ZEA in 96%, DON in 98%, T2-toxin in 85%, and FB1 in 3%. Individual differences among fetuses were also significant. We always found individuals with a "0" concentration, but the proportion of "0" was lower for ZEA, AF, and DON, while for T2-toxin and especially FB1, it was higher than in the hinds. The concentrations of the three mycotoxins (AF, DON, and T2-toxin) were of similar magnitude in the hinds and their fetuses, but the maximum value of ZEA was found in the fetus and that of FB1 in the hinds, indicating that FB1 does not (or only to a very limited extent) pass through the placenta.

Comparing the mycotoxin concentrations, we rarely found significant differences between sampling sites. The data showed high individual variability in mycotoxin concentrations in both the maternal and fetal livers, even within the same study area. High individual variability obscured spatial differences. This reflects the diversity of habitats of wildlife management units, variability in individual habitat use, and spatial and temporal changes in the availability of mycotoxin sources in each sampled area.

In six of the seven sampling areas, ZEA levels were significantly higher in the fetuses than in the hinds (Mann–Whitney U test). The AF concentrations were similar, with only a difference observed in the VGE4 area. An inverse relationship was observed between DON and T2-toxin. In one sampling area, the DON levels were higher in hinds than in fetuses. In some cases, there was also a significant difference in T2-toxin concentration, with fetuses showing higher toxin levels in two areas of the brain.

Differences in mycotoxin levels among the three fetal age groups were analyzed using the Kruskal–Wallis test. We suspected a slight increase in mycotoxin concentrations with fetal age for ZEA, AF, and DON; however, these differences were not statistically significant, except for DON, where the third and most developed groups had significantly higher toxin levels than the youngest group ($H(2, N = 60) = 6.100, p = 0.047$; post hoc: $z = 2.445, p = 0.044$).

Mother-fetus data pairs were divided into three groups to analyze the relationship between mycotoxin levels in hinds and fetuses: (1) the concentration of a given mycotoxin was "0" in the hind but detectable in the fetal liver; (2) the given mycotoxin was present in the hind liver but absent in the fetus; and (3) both the hind (mother) and the fetus contained the given mycotoxin. A total of 27 mother-fetus data pairs were assigned to group 1 for ZEA, 11 for AF, 6 for DON, 8 for T2-toxin, and none for FB1; therefore, this group was excluded from the preliminary correlation analyses. The correlation analysis focused on the data from Groups 2 and 3. Considering GAM

modeling, the fit of the smoothed (nonlinear) models was better for each mycotoxin than the parametric (linear) expression. In the main effects models, the predicted nonlinear relationship between the hinds and fetuses was significant in the case of ZEA (ZEAfetus(ZEAhind): edf = 4.868, F = 7.740, p = 0.006) and AF (AFfetus(AFhind): edf = 5.699, F = 2.671, p = 0.043). For the T2-toxin concentration, the T2-toxin×FB1 interaction model was the best candidate model. Although the nonlinear interaction was significant (T2-toxinfoetus~s(T2-toxinhind, FB1hind): edf = 2.603, F = 4.024, p = 0.018), the model did not demonstrate that varying FB1 concentrations significantly influenced the positive effect of the T2-toxin concentration in hinds.

In the 1st group of mother-fetus pairs, the hinds were free of mycotoxins, but the fetuses contained them. We assumed that the previously ingested toxin had already been eliminated from the hind's liver but was still detectable in the fetus. Since we did not know the amount of prior mycotoxin intake, this group had to be disregarded during the statistical analysis; however, these data reflect the slow breakdown of different toxins in the fetus and the accumulation of toxins. In the 2nd group, the mycotoxin was present in the hind's liver but absent in the fetus. We assumed that the mother's recent mycotoxin intake had not yet penetrated the fetus. This could reflect the possible filtering capacity of the placenta or that infiltration into the fetus requires a longer period. If both the maternal and fetal livers contained the mycotoxin (group 3), we assumed a correlation between them; however, this could only be demonstrated for ZEA and AF. Owing to the known reproductive-disruptive and fetotoxic effects of MTs, potentially harmful effects in groups 1 and 2 are likely to occur in the later stages of pregnancy. This will be investigated in future studies.

5.9. Antlers as biomarkers

The annually regenerating antlers provide a unique opportunity to assess the accumulation of both organic and inorganic substances, making them valuable biomarkers for environmental monitoring, particularly in regions with high-density stag populations. According to our research, the rapid annual division of stem cells at the antler base can be highly sensitive to mycotoxins, indicating that antlers may serve as indicators of environmental mycotoxin exposure. Examining abnormal antler growth patterns can offer an easily accessible method for evaluating the regional mycotoxin burden, reflecting environmental conditions and potential exposure risks.

Over the past two decades, industrial pollutant emissions in Hungary have significantly decreased, particularly in southern regions. Given this decline, we consider it unlikely that lead or fluoride exposure is directly related to the observed antler anomalies in this study. In other words, other environmental factors may play a more significant role in the development of these anomalies than genetic factors.

6. CONCLUSIONS

Based on the examination of 22 variables related to the rose, antler, antler pedicle, and skull, we have described a new disease, a deformity-causing chronic inflammation of the antler pedicle, referred to in Latin as *Pedunculitis Chronica Deformans* (PCD). Through statistical analysis of available trophy register data and a database created from my sample collections, we identified morphological characteristics that enable veterinarians, wildlife biologists, and game managers to clearly recognize animals affected by the disease. By integrating the 22 examined variables into a unified scoring system, we developed the RAPS evaluation system, which, when supplemented with data from trophy evaluations, is suitable for recognizing PCD and distinguishing its stages of development. I recommend the application of the RAPS scoring system in official trophy evaluation processes and suggest expanding trophy evaluation data to include these variables. To support this, it is advisable to prepare a detailed guide for trophy evaluators. This opens the way for gathering precise epidemiological data and forming homogeneous groups of affected individuals, which, in turn, facilitates the identification of causal factors and may ultimately contribute to the development of potential treatment strategies.

In this study, we examined the anatomical and physiological background of PCD. In our results, we described in detail the skin-connective tissue-bone interfaces of the antler pedicle (Peduncular-Dermal Junction, PDJ) as a distinct anatomical structure, emphasizing its significance as a key factor in the development and progression of the disease. We demonstrated the occurrence of aseptic inflammatory processes at this site. According to our findings, the first step in PCD development is disruption of the PDJ. We showed that as the disease progresses, cavities can form within the antler itself, and inflammation spreading toward the cranial cavity can also cause intracranial abscesses. By applying RAPS scoring to several suspected PCD roe deer and red deer samples, we found symptoms characteristic of the disease in these species. Therefore, we assume that PCD occurs not only in fallow deer but also in red and roe deer. Further studies are needed to clarify why PCD appears less frequently and inconspicuously in these species.

This study provides a more comprehensive understanding of these deformities and lays the groundwork for future diagnosis and treatment options. Our results primarily offer detailed information to veterinary and wildlife health professionals for recognition of PCD. Simultaneously, because a similar structure can be found in the tissue connection between the tooth and periodontium, the findings may also be useful for dental practice. Previously, “antler base rot” was attributed to microbial infection. Our detailed microbiological examinations of individuals with inflamed antler bases did not confirm their primary causative role; rather, they could only be considered secondary superinfecting components. Although numerous bacteria, including pus-forming species, were detected in the samples, there was significant variation between individuals. From this, we can conclude that inflammatory bacteria play a role in the development of the disease, but they appear only in the more advanced stages of PCD after the skin-bone connection has been disrupted. We assume that abnormalities in the scar tissue that forms after antler shedding may also be responsible for the development of the disease. However, considering that we detected the initial symptoms of PCD even in first-antlered fallow deer bucks, it is assumed that the development of the disease can be traced back to more complex causes. Since the literature documents a number of effects of mycotoxins that could lead to the development of this problem, we began investigating the relationship between various mycotoxins and PCD.

Testosterone plays a role in the development of secondary sexual characteristics, including formation, antler growth, and shedding. One mycotoxin, ZEA, disrupts the production of sex hormones and thus can affect antler development. Our studies confirmed higher ZEA and decreased testosterone levels in individuals with PCD. We also detected elevated concentrations of two other mycotoxins, AFB1 and DON, in PCD patients. Both toxins have an inhibitory effect on bone growth and bone regeneration, which is why we have good reason to assume that mycotoxins may have a significant impact on the development of antler growth disorders. Numerous studies have shown that, primarily due to heat and drought, global climate change leads to increased mycotoxin production by molds, and even the appearance of new mycotoxins. This may explain the increased incidence of antler abnormalities in recent decades. An additional question to investigate is when and how mycotoxins enter the organism. To uncover this, it is necessary to thoroughly study the diets of different deer species and mycotoxin contamination of the feed they consume. Based on my initial practical experience, contaminated wildlife feed may be the primary source of mycotoxins. This is evidenced by the fact that, in some areas, after discontinuing corn feeding upon my recommendation, reproductive indicators improved. Further detailed research and reevaluation of the role of wildlife feeding are needed.

Because PCD symptoms appear at a young age, we assumed that mycotoxins might enter the offspring even during fetal life via the maternal organism. Therefore, we examined the mycotoxin levels in pregnant fallow deer and their fetuses. Based on our results, ZEA, AFB1, DON, T-2/HT-2 toxin, and FB1 were present in the majority of pregnant fallow deer hinds during the autumn-winter period, even in the best-managed populations. The great individual variability between wildlife management units and the lower differences reflects the highly variable spatial and temporal access to mycotoxin sources in the environment. The type, spatial distribution, and temporal changes in mycotoxin sources are important pieces of information for risk reduction. The spatial scale of the research should be adapted to the movement and habitat use of each species. As these mycotoxin sources are accessible to fallow deer and other wild animal populations, the negative effects of mycotoxins may be more general at the ecosystem level. We demonstrated that the studied mycotoxins, except FB1, crossed the placenta and were found in the fetus. The significantly higher mycotoxin concentrations found in fetuses, especially when the given toxin could not be detected in the mothers, indicate the potential accumulation of mycotoxins in fetuses. The mycotoxin concentration in fetuses tended to increase with age; in all cases, a slight increase was observed, but only DON was statistically significant. This highlights the risk of mycotoxin exposure during pregnancy. High mycotoxin concentrations and the simultaneous presence of various mycotoxins, even at low concentrations (multi-mycotoxin effect), may negatively affect fetal development and neonatal mortality. Further studies are needed to determine what reproductive and fetal development problems may be related to mycotoxins, but all mycotoxins that enter the fetus are known to have a disruptive and inhibitory effect on bone development, so we can rightly assume that they may cause abnormalities already in the embryonic development of the antler rudiments.

7. NEW SCIENTIFIC RESULTS

1. Through the analysis of the database created during my research, I was able to accurately identify and describe chronic antler pedicle inflammation causing deformity (Pedunculitis Chronica Deformans, PCD), thus providing a foundation for further research.
2. The RAPS scoring system I developed enables the standardized evaluation and documentation of the various stages and severity of the disease, both in wildlife management practices and during trophy evaluations.
3. For the first time in this study, the presence of PCD was detected in fallow -, red-, and roe deer populations.
4. A detailed description of the osteological and histoanatomical features of PCD is provided. This description can serve as a basis for diagnosing PCD at various developmental stages in veterinary practice.
5. I demonstrated that the development of PCD begins with the disruption of the skin-connective tissue-bone junctions of the antler pedicle (peduncular dermal junction, PDJ).
6. I also demonstrated that in the advanced, purulent stage of PCD, numerous inflammation-causing microbes may appear in the tissues, but the exclusive effect of any single bacterial species could not be confirmed. I found evidence that the initial symptoms of PCD can occur without microbial infections.
7. My examinations supported the potential role of mycotoxins in the etiology of antler pedicle disease. I showed that individuals with PCD have lower testosterone levels, while aflatoxin B1, zearalenone, and deoxynivalenol levels are higher than those in healthy individuals. I also explain the possible mechanism of action based on the literature.
8. I demonstrated that all the examined mycotoxins can be present in the liver of fallow deer and that several mycotoxins can be present in a single individual simultaneously. My results highlight the considerable spatial, temporal, and individual variation in the occurrence of mycotoxins.
9. My research is the first to prove that AFB1, ZEA, DON, and T2-toxin cross the placenta of pregnant fallow does and can accumulate in the fetus, potentially causing fetal damage.

8. OWN PUBLICATIONS

Scientific publications related to the topic of the dissertation

Journal articles

1. Szőke, Zsuzsanna ; Babarczi, Bianka ; Mézes, Miklós ; Lakatos, István ; Poór, Miklós ; Fliszár-Nyúl, Eszter ; Oldal, Miklós ; Czéh, Árpád ; Bodó, Kornélia ; Nagyéri, György ; Ferenczi Szilamér.
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