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Feline allergic diseases: introduction and proposed nomenclature

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Background – Feline allergic diseases present as challenging problems for clinicians, not least because of the number of reaction patterns of the feline skin, none of which are specific for allergy. Furthermore, there is some controversy over the nomenclature that should be used in their description.

Objectives – To review the literature, assess the status of knowledge of the topic and the extent to which these diseases could be categorized as atopic in nature, and make recommendations concerning nomenclature.

Methods – Atopic diseases in humans and cats were researched. A comparison then was made of the essential features in the two species.

Results – There were sufficient similarities between human atopic diseases and the manifestations of feline diseases of presumed allergic aetiology to justify the use of "atopic" to describe some of the feline conditions affecting the skin, respiratory and gastrointestinal tract. However, none of the allergic skin diseases showed features consistent with atopic dermatitis as described in man and the dog.

Conclusions and clinical importance – The term "Feline Atopic Syndrome" (FAS) is proposed to encompass allergic diseases of the skin, gastrointestinal tract and respiratory tract, and "Feline atopic skin syndrome" (FASS) proposed to describe allergic skin disease associated with environmental allergies. We are not aware of any adverse food reactions in cats that are attributable to causes other than immunological reactions against the food itself. We therefore propose an aetiological definition of "Food Allergy" (FA) to describe such cases.

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Introduction

Research into feline skin diseases of presumed allergic aetiology has lagged far behind that in allergic dogs. In part, this may be due to the fact that canine atopic dermatitis (AD) has striking similarities with its human counterpart, and much research has focussed on the canine disease as an experimental model of the latter. Another issue that sets feline dermatology apart from that in other species is that inflammatory skin diseases of the cat present with a spectrum of reaction patterns, some of which appear to be unique for this species, and the fact that each reaction pattern may in turn have a wide range of inciting causes. Thus, one cannot expect that any particular feline condition will show similar manifestations to the disease homologues in dogs or in people. There also is a lack of agreement regarding the nomenclature used to describe feline skin diseases, with some favouring an aetiological approach,[1] whereas other authors prefer to employ a terminology that as far as possible parallels that used in humans and dogs.[2] Furthermore there is a paucity of data on the spontaneous hypersensitivity disorders affecting the gastrointestinal and respiratory tracts, although the experimental model of feline asthma has been well-characterized.[3]

This paper, the first in a series on feline allergic skin diseases, is timely, as it reviews the relevant published literature on these topics. This introduction commences with a historical review of the essential features of human allergic diseases, which is followed by an assessment of the extent to which the various feline allergic disorders can be considered as equivalent clinicopathological entities. Where the use of the same descriptors does not appear justified, alternative terminology is proposed. The ensuing three papers review the current knowledge regarding the immunopathogenesis of allergic diseases affecting the feline skin and lungs, their clinical signs and diagnostic features, and, finally, the therapeutic options.

Atopic diseases of man

1 The definition of "atopy" and the nature of skin-sensitizing antibody

Much of the terminology and our basic understanding of allergy in humans resulted from pioneering work undertaken in the 1920s and 1930s. The term "atopy", taken from the Greek meaning "strange disease", was introduced in 1923 by Coca and Cooke[4] to describe two diseases that they believed had much in common, namely asthma and hay-fever – or allergic rhinitis. The characterization of AD was attributed to Sulzberger,[5] and in 1934, Coca included this condition under his definition of "atopic".[6] The essential features of an atopic disease were defined as a familial predisposition to allergic disease affecting the skin, respiratory and/or gastrointestinal tract. The discovery of their association with skin-sensitizing antibodies resulted from the earlier seminal studies of Prausnitz and Küstner.[7] The latter was exquisitely sensitive to cooked fish and not to raw fish. The antibody responsible did not fix complement, did not precipitate with antigen and was not able to passively sensitize guinea pig skin. However, it was able to sensitize the skin of a nonallergic human recipient. Following the injection of Küstner's serum intradermally into the arm of Prausnitz, a wheal-and-flare reaction developed on subsequent challenge with cooked fish antigen. Prausnitz himself suffered from seasonal hay-fever and showed strong prick-test reactivity to ryegrass. However, paradoxically his serum was not able to sensitize the skin of Küstner in a similar manner.

This phenomenon was further investigated by Coca and Grove[8] who introduced the term "reagin" for this skin-sensitizing antibody. They confirmed that it was heat labile and that the skin-sensitizing ability was largely lost after heating the serum to 56°C for 30 min. They further showed that the skin of 11% of individuals was wholly nonreceptive to passive sensitization, and a further 5% were only partially receptive. There were difficulties, therefore, in using what came to be known as the Prausnitz–Küstner (or PK) test for quantitative studies. Much effort was expended over the ensuing four decades in characterizing further the nature of the reagin, and in determining to which antibody class it belonged. Finally, in the late 1960s the painstaking work of the Ishizakas, a husband and wife team, showed that it belonged to a hitherto undescribed antibody class that they designated γE, or as it later became known, immunoglobulin (Ig)E.[9]

2 Extrinsic and intrinsic atopic diseases

Only a few years after the discovery of IgE it became clear that not all cases of asthma were associated with elevated allergen-specific IgE,[10] and similar observations were made in relation to AD and rhinitis. This has led to the definition of two distinct variants of the three atopic diseases – "extrinsic", which is associated with elevated IgE levels to environmental and/or food allergens, and "intrinsic" which has no detectable IgE sensitization.[11] These also have been referred to respectively as "allergic" and "nonallergic". The pathogenesis of the latter is unclear, although as higher activation of all inflammatory pathways assessed – including Th2 – has been shown in the latter,[12] the term "nonallergic" appears to lack justification. These variants show not only immunological differences, but also differing clinical spectra.[11] In humans, it is estimated that 16–45% of cases of AD are intrinsic,[13,14] 10–33% of cases of asthma are intrinsic[15,16]and, likewise, 9–42% patients with rhinitis lack any association with IgE.[17]

3 The atopic march

Patients can present with more than one manifestation of atopic disease at the same time, and there is a tendency for atopic individuals to first exhibit signs of AD in childhood, and then progress to develop asthma and/or allergic rhinitis.[18] In one study conducted in the UK, 100 infants from atopic families were followed over a 22 year period.[19] By 1 year of age, 20% of the children had developed AD, and the incidence had declined to 5% by the end of the study. Over the same period, the incidence of allergic rhinitis increased from 3% to 15%, and the

proportion of patients that developed a wheeze indicative of asthma increased to 40%.[19] However, the legitimacy of the term "atopic march" has recently been questioned and differing factors have been suggested to play a role in the changing spectrum of the diseases over time.[20]

To what extent do feline allergic diseases satisfy criteria as being atopic in nature?

1 Is there evidence of a genetic basis?

The study of feline genetics is in its infancy, yet several pieces of evidence suggest a possible genetic basis for feline allergic diseases.

The first is contained in a report of dermatitis and vomiting with accompanying eosinophilia in eight of 26 (31%) individuals in an inbred colony of cats from Hungary which was attributed to food allergy.[21] The clinical signs in all eight resolved on feeding a hypoallergenic diet and a relapse was noted in four cases following dietary challenge.

The second was a description of three 12-month-old domestic short hair cat littermates, all of whom were reported to rub their faces, lick their abdomens, and bite and nibble their legs.[22] The condition had gradually worsened from the onset at 6 months of age. Upon presentation, the facial whiskers were bent and broken, and the commissures of the mouth were erythematous. There was a mild ceruminous otitis externa, thinning of the hair on the ears and ventral abdomen, and focal areas of hair loss on the extremities. One cat was more severely affected with crusting lesions on the face with linear excoriations, and a severe ceruminous otitis externa. The condition was unresponsive to a hypoallergenic diet trial. A year later intradermal tests (IDTs) revealed multiple sensitivities, and all three showed a good response to allergen-specific immunotherapy (ASIT), with minor relapses at the height of the pollen season. These features are entirely compatible with a diagnosis of atopy. The mother of the cats also was reported to suffer seasonal outbreaks of crusting and scabs on the head and neck, yet further investigations were not permitted.

Data derived from reports of case series also have identified some breed predispositions. In a multicentre study of 588 pruritic cats, 381 were diagnosed as suffering from a hypersensitivity dermatosis (HD).[23] They were first subdivided into those suffering from flea allergy dermatitis (FAD) (n = 146) and nonflea HD (n = 235), with the latter group comprising food HD and nonflea/nonfood HD. Pure-bred cats (Siamese, Persian, Abyssinian and Maine coon) were significantly over-represented in the latter group as compared with the former, which the authors interpreted as indicating a possible genetic basis for this group of diseases. The second report from Australia described 45 cases with signs compatible with AD, all of which failed to respond to flea control and hypoallergenic diet trials.[2] Compared to the base clinic population, domestic mixed breeds, Abyssinian and Devon rex were predisposed. In a further report of 194 cases of AD seen at a teaching hospital, Abyssinians, Himalayans and Persians were over-represented,[24] and the Abyssinian also was implicated in a report from Germany[25] which described five related Abyssinian cats that developed cardiomyopathy, three of which that also developed a pruritic dermatitis. Although this was not fully characterised, it was compatible with AD. In two cases, the skin disease was accompanied by episodes of rhinitis and conjunctivitis and the cats showed peripheral eosinophilia. The same two cats developed anaphylaxis following both vaccination and administration of penicillin.

2 Is there evidence for the involvement of IgE?

The most definitive evidence for the involvement of IgE in a feline allergic disease comes from an early description (1968) of a cat presented with concomitant dermatitis and enteritis.[26] An IDT was positive to cow's milk antigen and its serum yielded a positive PK test. Hypoallergenic diet trials and subsequent challenges confirmed the diagnosis of food allergy. The cat belonged to a veterinarian, and the immunological workup was performed by two other veterinarians who were amongst the leading immunologists of the day. One wonders how many similar cases have occurred over the years, but were not fully characterized owing to the lack of requisite expertise.

The role of IgE is discussed in detail later in this series where data from studies of cats with suspected allergic dermatitis (excluding flea allergy and mosquito bite hypersensitivity) and asthma are examined. Parameters assessed as being suggestive of the involvement of IgE include responses to atopy patch tests, the incidence of positive IDT and positive serology for allergen-specific IgE (compared to control populations), the effects of allergen avoidance and also the response to ASIT, which has long been regarded as a classical feature of IgE-mediated allergic diseases. The stated overall conclusion is that: "the evidence reviewed in this paper is supportive of the role of IgE – albeit not strongly so." However, if an intrinsic form of allergic dermatitis and/or asthma were to exist in the cat, one would not expect 100% correlation with the presence of allergen-specific IgE.

3 Is the spectrum of allergic diseases in cats similar to the atopic diseases of man, and has an "atopic march" been shown to exist in this species?

Cats suffering from dermatitis of presumed allergic origin exhibit varying presentations – yet none of them can be termed "classic" for AD when compared to the human and canine diseases. This perhaps stems from the limited spectrum of reaction patterns exhibited by cats, with apparently identical presentations arising from a wide range of unrelated causes. They also may suffer from enteritis that sometimes appears to be allergic in origin, and asthma is frequently encountered in clinical practice. Although the aetiology of the latter is controversial and could in some instances be intrinsic, a model of allergic asthma has been developed in cats, which closely parallels the spontaneous disease of humans.[3] As a further example, a case of seasonal allergic rhinitis has been described in a Japanese domestic cat whose clinical signs were strikingly similar to those seen in seasonal rhinitis (or "hay-fever") in humans,[27] and the serum was

positive for IgE against Japanese Cedar (*Cryptomeria japonica)* both by serology and PK testing.

Justification for the existence of an "atopic state" in the cat would be strengthened if more than one of these possible atopic diseases were seen in the same patient, and if there was evidence of an "atopic march". Concomitant skin and gastrointestinal disease was seen in the two reports of food allergy noted above – one in a single cat[26] and one in a colony of cats[21] – and also in five of 22 (23%) diet-responsive cases in a study from New Zealand.[28] In all of these cases the gastrointestinal and dermatological signs both responded completely to the dietary change. However, partial responses to a hypoallergenic diet also may be encountered. In one early report, five of 90 cats (6%) evaluated with a possible diagnosis of atopy responded partially to the diet trial indicating concomitant reactivity to foods and environmental allergens,[29] and in one of the case series noted above, food allergy accompanied six of 45 (13%) of cases diagnosed with AD.[2] Also in a retrospective analysis of 194 cases accorded a diagnosis of AD at a veterinary teaching hospital, nine cats (4.5%) were adjudged to have concomitant food allergy.[24]

Concomitant dermatological signs and upper or lower respiratory tract disease also have been reported. Rhinitis was noted in five of 10 cases of atopy in one case series,[29] and conjunctivitis was reported in two of 45 cases (4.4%)[2] and six of 100 (6%)[22] cases diagnosed as AD and nonflea/nonfood HD, respectively, in two other papers. Lower respiratory signs diagnosed as probable or definite asthma accompanied AD in three of 45 (6.6%) and six of 100 (6%), respectively, in the two case series noted earlier,[2,21] and also in one recent case report.[30] In another publication, a series of cats seen by the cardiopulmonary service of a university teaching hospital for evaluation of probable asthma, and stated to be free of skin disease, were referred to the dermatology service for performance of IDTs and for allergy-specific IgE serology. Upon dermatological examination "a number" (not quantified) had to be removed from the study, as signs compatible with allergic skin disease were observed.[31] It is possible, therefore, that the co-existence of signs involving more than one system might be more common than is reported, as some cases may have been denied a sufficiently rigorous workup. Nevertheless, at this time, there is no indication that an "atopic march" occurs in this species.

Conclusions and proposed nomenclature

From the literature reviewed above, it can be concluded that the feline diseases of presumed allergic aetiology have some features comparable to those seen in the human atopic diseases and canine AD. Strong evidence of a genetic basis is missing thus far – the state of feline genetics research has not yet permitted the necessary investigations. Despite this, the fact that cats can suffer from the triad of allergic dermatitis, allergic enteritis and asthma, often in combination and with some evidence for the involvement of IgE, provides justification for designating these as likely atopic diseases. More detailed in-depth investigations are needed in order to assess the existence or otherwise of intrinsic variants that would explain the lack of a stronger association with IgE. Bearing all of these limitations in mind, the following terminology is proposed:

Feline atopic syndrome (FAS)
This description encompasses allergic dermatitis associated with environmental allergens, food allergy and asthma that may be associated with IgE antibodies. Food allergy and flea allergy can both either mimic and/or contribute to this syndrome, and their potential role must be assessed before deciding on the therapeutic approach.

Feline atopic skin syndrome (FASS)
An inflammatory and pruritic skin syndrome of cats manifested by a spectrum of reaction patterns, none of which are specific for this syndrome, and that may be associated with IgE antibodies to environmental allergens. Food allergy and flea allergy can both either mimic and/or contribute to this syndrome, and their potential role must be assessed before deciding on the therapeutic approach.

Feline asthma
An eosinophilic inflammatory disease affecting the bronchioles and leading to spontaneous reversible bronchoconstriction and airway remodelling, manifested by acute respiratory distress or chronic coughing and expiratory wheezing, and that may be associated with IgE antibodies to inhaled allergens.

Intrinsic and extrinsic diseases
The definitions applied to FASS and to feline asthma do not preclude the possibility that extrinsic and intrinsic (in which no relevant IgE reactivity is demonstrable) variants of both may exist with intrinsic FASS being analogous to atopic-like dermatitis of dogs.

Feline food allergy
This aetiological diagnosis refers to any clinical manifestations, including those of FASS, that are attributable to immunological reactivity to an ingested food item.

Specifically excluded from the atopic designation are feline flea allergy dermatitis and mosquito-bite hypersensitivity.

Note: In this and subsequent papers in this series, allergen-specific immunotherapy (abbreviated as ASIT) refers to treatment with a series of allergen injections whose composition is based upon results of IDT and/or allergen-specific IgE serology.

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Résumé

Contexte – Une nouvelle anomalie congénitale de la tige pilaire ressemblant au phénotype nu des rongeurs est décrite dans une portée de quatre chats européens (DSH). Les données relatives aux anomalies de la tige pilaire et des follicules pileux sont rares en médecine vétérinaire.

Objectifs – Décrire et comparer les anomalies structurelles de ces chats avec d'autres dystrophies félines et d'autres mammifères.

Sujets – Une portée de chats DSH avec alopécie progressive non-inflammatoire.

Méthodes – L'évaluation histopathologique, la microscopie électronique à transmission et l'analyse des éléments par rayons X définissaient les changements pilaires et cutanés des chats nés alopéciques. Les données ont été comparées aux archives de chats normaux et de souris mutantes *Dsg4*^{lahJ} et *Krt75*^{tm1Der}.

Résultats – La microscopie électronique à lumière et à balayage des poils a révélé des défauts de forme de l'extrémité de la tige pilaire en pointe ou en lance. Les données histopathologiques consistaient en des tiges pilaires enflées, initialement au dessus de la matrice du bulbe pilaire et ensuite retrouvé dans les parties distales des follicules pileux télogènes, semblables à ceux observés chez les souris mutantes *Dsg4*^{lahJ} *Krt75*^{tm1Der} . La microscopie électronique à transmission de la tige pilaire et des follicules pileux a révélé une perte de la structure normale des poils de garde chez les chats alopéciques. Il y a avait une diminution statistiquement significative du contenu en sulfure juste au dessous des défauts des tiges pilaires (trichothiodystrophie).

Conclusions et importance Clinique – une forme rare d'alopécie congénitale résultant en une dystrophie folliculaire est décrite chez le chat, comparable aux changements de la tige pilaire et du follicule pileux

décrits dans plusieurs souches de souris mutantes avec une mutation génétique unique des gènes des molécules d'adhésion ou de kératine.

Resumen

Antecedentes – se describe una nueva anomalía congénita del pelo que se asemeja al fenotipo de pelo lanceolado de los roedores en una camada de cuatro gatos domésticos de pelo corto (DSH). Los datos relacionados con los trastornos del pelo y los folículos siguen siendo escasos en medicina veterinaria.

Objetivos – Describir y comparar anomalías estructurales en estos gatos con otras distrofias capilares en gatos y otros mamíferos.

Animales – una camada de gatos DSH con alopecia no inflamatoria progresiva.

Métodos – evaluación histopatológica, por microscopía electrónica de barrido y de transmisión y el análisis de elementos basados en rayos X definieron los cambios en el pelo y la piel de los gatos nacidos con alopecia. Los hallazgos se compararon con datos de archivo de gatos normales y ratones mutantes de pelo lanceolado (*Dsg4*^{lahJ}) y queratina 75 (*Krt75*^{tm1Der}).

Resultados – la microscopía óptica y electrónica de barrido de los pelos reveló defectos en la punta del cabello en forma de lanza o punta de lanza. Los hallazgos histológicos fueron pelos hinchados, inicialmente por encima de la matriz del bulbo piloso y luego encontrados en las partes distales de los folículos pilosos telógenos, similares a los observados en ratones mutantes *Dsg4*^{lahJ} *Krt75*^{tm1Der}. La microscopía electrónica de transmisión del pelo y los folículos pilosos mostró una pérdida en la estructura normal de los pelos primarios en los gatos alopécicos. Hubo una disminución estadísticamente significativa en el contenido de azufre justo por debajo de los defectos en los tallos del cabello (tricotiodistrofia).

Conclusión e importancia clínica – en gatos se describe una forma poco común de alopecia congénita que da como resultado distrofia folicular, similar a los cambios en el folículo piloso y el tallo del pelo descritos en varias cepas de ratones mutantes con mutaciones de un solo gen en moléculas de adhesión o genes de queratina.

Zusammenfassung

Hintergrund – Es wird eine neue angeborene Haarschaft Abnormalität bei einem Wurf von vier Hauskatzen (DSH) beschrieben, die dem lanzenförmigen Haar Phänotyp von Nagern gleicht. Daten über Haarschaft- und Follikelstörungen bleiben in der Veterinärmedizin rar.

Ziele – Eine Beschreibung der Strukturabnormalitäten bei diesen Katzen und ein Vergleich mit anderen Haardystrophien bei Katzen und anderen Säugern.

Tiere – Ein Wurf von Hauskatzen mit einer progressiven nichtentzündlichen Alopezie.

Methoden – Mittels histopathologischer Evaluierung, Raster- und Transmissionselektronenmikroskopie, und Röntgen-basierter Elementanalyse wurden die Haar- und Hautveränderungen bei Katzen, die mit einer Alopezie geboren worden waren, definiert. Die Befunde wurden mit archivierten Daten von normalen Katzen und lanzenförmigen Haaren (*Dsg4*^{lahJ}) und Keratin 75 (*Krt75*^{tm1Der}) von mutanten Mäusen verglichen.

Ergebnisse – Die Licht- und Rasterelektronenmikroskopie der Haare zeigte Lanzen- oder Speer-Kopf geformte Defekte der Haarspitzen. Die histologischen Befunde zeigten geschwollene Haarschäfte, ursprünglich oberhalb der Haarwurzelmatrix und später auch in den distalen Teilen der telogenen Haarfollikel, ähnlich denen bei *Dsg4*^{lahJ} *Krt75*^{tm1Der}mutanten Mäusen. Die Transelektronenmikroskopie von Haarschaft und Haarfollikeln zeigte ein Verschwinden der normalen Struktur der Deckhaare bei haarlosen Katzen. Es bestand eine statistisch signifikante Abnahme des Schwefelgehaltes unmittelbar unter den Defekten in den Haarschäften (Trichothiodystrophie).

Schlussfolgerung und klinische Bedeutung – Eine seltene Form einer angeborenen Alopezie, resultierend aus einer follikulären Dystrophie wird bei Katzen in einer ähnlichen Form beschrieben, wie es bereits bei einigen mutanten Mausstämmen mit einer Einzelgenmutation in Adhäsionsmolekülen oder Keratingenen publiziert worden war.

要約

背景 – げっ歯類の槍状の毛の表現型に似た新しい先天性毛幹異常が4頭のドメスティック・ショートヘア（DSH）の同腹子で記述されている。毛幹および毛包の障害に関連するデータは、獣医学ではまだ不足している。

目的 – 本研究の目的は、これらの猫の構造異常を説明し、猫や他の哺乳類の他の毛髪ジストロフィーと比較することであった。

動物 – 進行性の非炎症性脱毛症を伴うDSH猫の同腹子。

方法 – 組織病理学的評価、走査型および透過型電子顕微鏡法、およびX線ベース元素解析により、脱毛症で生まれた猫の毛および皮膚の変化が定義された。調査結果は、健常猫と披針形の毛（Dsg4^{lahJ}）およびケラチン75（Krt75^{tm1Der}）変異マウスからのアーカイブデータと比較された。

結果 – 毛髪の光学顕微鏡および走査型電子顕微鏡検査により、毛先の槍または槍の頭の形をした欠陥が明らかになった。組織学的所見は、Dsg4^{lahJ} Krt75^{tm1Der}変異マウスで観察されたものと同様に、最初は毛球マトリックス上にあり、後に休止期毛包の遠位部分に見られた、膨張した毛幹であった。毛幹および

毛包の透過型電子顕微鏡検査は、脱毛猫のガード毛の正常な構造の喪失を示した。毛幹の欠陥（トリコチオジストロフィー）のすぐ下で硫黄含有量の統計的に有意な減少があった。
結論と臨床的重要性 – 接着分子またはケラチン遺伝子に単一遺伝子変異を有するいくつかの変異マウス系統で報告された毛包および毛幹の変化に類似した、毛包ジストロフィーを引き起こすまれな形態の先天性脱毛症が猫で説明されている。

摘要
背景 — 在4只同窝家养短毛(DSH)猫中，发现了类似啮齿动物披针形毛发表型，这是一种新的先天性毛干异常。与毛干和毛囊疾病相关的数据在兽医学中仍然很少。
目的 — 描述这些猫的结构异常，并与猫和其他哺乳动物的其他毛发形成不良进行比较。
动物 — 患有进行性非炎性脱毛症的一窝DSH猫。
方法 — 组织病理学评价、扫描和透射电子显微镜以及基于X射线的元素分析，定义了新生脱毛猫的毛发和皮肤变化。将结果与正常猫、披针形毛发(Dsg4lahJ)和角质75(Krt75tm1Der)突变小鼠的存档数据进行比较。
结果 — 毛发的光学和扫描电子显微镜检查显示，毛尖存在披针形或矛头形缺陷。组织学发现毛干肿胀，最初在毛球基质上方，后来在终止期毛囊的远端部分发现，与在Dsg4lahJ Krt75tm1Der突变小鼠中观察到的相似。毛干和毛囊的透射电镜显示脱毛猫护毛的正常结构缺失。毛干缺损正下方的硫含量在统计学上显著降低（毛发硫营养不良）。
结论和临床重要性 — 发现了猫的罕见先天性脱发形式，由毛囊发育不良所导致，与粘附分子或角质基因中单基因突变的几种突变小鼠品系中报告的毛囊和毛干变化类似。

Resumo
Contexto – Uma nova anomalia congênita da haste pilosa semelhante ao fenótipo de pelo lanceolado dos roedores foi descrita em uma ninhada de quatro gatos domésticos de pelo curto (DSH). Dados relacionados a enfermidades da haste e folículo piloso permanecem escassos na medicina veterinária.
Objetivos – Descrever e comparar as anomalias estruturais nestes gatos com outras distrofias pilosas em gatos e outros mamíferos.
Animais – Um gato DSH apresentando alopecia não inflamatória progressiva.
Métodos – Avaliação histopatológica, microscopia eletrônica de varredura e transmissão e análise elementar baseada em raio-X foram utilizadas para caracterizar as alterações de pele e pelos em gatos nascidos com alopecia. Os achados foram comparados a dados arquivados de gatos normais e ratos com mutação de pelo lanceolado (*Dsg4*^{lahJ}) e Queratina 75 (*Krt75*^{tm1Der}).
Resultados – À microscopia de varredura e óptica, observou-se pelos com defeitos nas pontas, que se apresentavam em formato de lança ou ponta de lança. Os achados histológicos foram hastes pilosas dilatadas, inicialmente acima da matriz do bulbo piloso e posteriormente nas partes distais dos folículos pilosos telógenos, similar ao observado nos ratos mutantes *Dsg4*^{lahJ} *Krt75*^{tm1Der}.Ao microscópio eletrônico de transmissão, as hastes pilosas e os folículos pilosos demonstraram perda na estrutura normal dos pelos guardiães nos gatos alopécicos. Houve uma redução significativa no conteúdo de enxofre imediatamente abaixo dos defeitos nas hastes pilosas (tricotiodistrofia).
Conclusão e importância clínica – Uma forma rara de alopecia congênita resultante de distrofia folicular descrita em gatos é similar às alterações nos folículos pilosos e hastes pilosas em diversas linhagens de ratos com mutações de um único gene em genes de moléculas de adesão ou queratina.