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REVIEW ARTICLES

# *The Origin of Pilonidal Sinus Disease – 10 Wrong Theories and one Recent Discovery.*

Marius Dettmer [1, 6, 7], Konstantinos Degiannis [2, 6, 7], Myriam Braun-Münker [3, 7], Dietrich Doll [4, 6, 7], Matthias Maak [5, 7 *]

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## *Abstract*

***Introduction:*** In the last two centuries, many theories have been proposed to explain the origin of pilonidal sinus disease (PSD) – congenital and acquired.

***Materials and Methods:*** A PubMed literature review was conducted and looked at different proposed theories on the origin of PSD; this overview was then compared to research results from more recent studies.

***Results:*** Initially it was postulated, that PSD was of embryonic origin. This however changed during World War II as more 78.000 American soldiers were diagnosed and treated for PSD. Thereafter, the perception of the origin of PSD changed to an acquired one. New data has shown that short hair fragments, which have fallen from the scalp may be the origin of PSD – therefore disproving the theory of folliculitis and fatty gland obstruction.

***Conclusion:*** These new findings may explain why recurrences/new diseases occur within follicle-free areas – such as scars and without any preceding infection. This may aid in the prevention of PSD.

***Keywords:*** Pilonidal sinus disease, genesis, Prevention, short hair fragments.

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* ***Corresponding author:***
*Matthias Maak*
✉ *matthias.maak@kreiskrankenhaus-hoechstadt.de*

1 *Department of Trauma Surgery and Orthopaedics, Reconstructive and Hand Surgery and Burn Medicine, German Armed Forces Central Hospital Koblenz, Koblenz, GERMANY*
2 *Department of Trauma, Hand and Reconstructive Surgery, University Hospital of Saarland, Saarland University, Homburg, GERMANY*
3 *Fulda University of Applied Sciences, Fulda, GERMANY*
4 *Department of Procto-Surgery and Pilonidal Sinus, St. Marienhospital Vechta, Academic Teaching Hospital of the MHH Hannover, Vechta, GERMANY*
5 *Department of Surgery, University Hospital Erlangen Friedrich-Alexander University (FAU) Erlangen-Nuremberg, Erlangen, GERMANY.*
6 *Vechtaer Research Institute VIFF eV, Vechta, GERMANY*
7 *Deutsche Forschungsgruppe Pilonidalsinus, Vechta, GERMANY*

## *Introduction*

Pilonidal sinus disease (PSD) was first described 186 years ago as a "lump" in the lower back region of a captain's young son. [1]. Despite being injected with silver (Ag) and mercury (Hg) solutions[1], healing was dismal. A small

[1] In the past, mercury was used in several forms for antimicrobial purposes. Some of the most common forms of mercury-based disinfectants were as follows:

1. Mercury (I) chloride (Hg2Cl2): Also known as mineral calomel, was a widely used antiseptic and disinfectant in the 19th and early 20th centuries as well as in the treatment of syphilis. It was used to treat wounds, as a preservative for biological specimens, as a disinfectant for medical and dental instruments, and to rid the body from "impurities". It frequently caused accidental poisonings.
2. Mercury (II) chloride, mercury bichloride (HgCl2): This form of mercury was commonly used as an antiseptic and as a topical treatment for skin conditions.
3. Mercury (II) oxide, mercuric oxide (HgO): Mercury oxide was used as a disinfectant for air and surfaces, and was also used as a component in some cosmetics and soaps. It is a highly toxic substance which can be absorbed by inhalation of aerosol, by ingestion or through skin.

nest of hair was discovered by the physician and thereafter removed – this resulted impressive healing. Most common theories at that time were as follows:

*Congenital theories postulated*

As this disease occurred in the midline, it was consecutively thought that pilonidal sinus was an acquired disease.

In 1860, *Luschka* postulated that a pilonidal sinus was a remnant of the embryonic preen gland in human beings [2]. In 1878, *Feré* proposed, that a pilonidal sinus developed between 3rd and 6th embryological week in utero, when the neural tube and skin ectoderm close in the midline[3]. Through faulty closure, skin remnants like hair follicles would be transferred into the depth, allowing hair to grow and develop into larger hair nests.

In 1882, *Lannelongue* described a ligament between the pilonidal sinus area and the coccyx[4]. As the body grew, the distance between skin and coccygeal bone increased. The idea was, that ligamental traction would generate a small concave area, which would finally develop into PSD.

Other scientists like *Madelung* (1885) suggested, that a pilonidal sinus was a "error in nature" where insufficient resorption of the in-utero human tale had occurred [5]. *Tourneaux* (1887) blamed remnants of neuro-cutaneous tracts for the formation of pilonidal sinuses [6].

***Congenital theories answered***

As time passed on, the embryological theories were subsequently disproven. Answering Luschka with his preen gland theory, the preen gland is only seen in birds and not in mammals[2]. The faulty closure theory with the inclusion of hair follicles in the depths would have been proven by histology by identifying hair follicles – which could never be seen. Only loose hair without follicles could be seen in the pilonidal sinus cavities until now. Caudal traction as a reason for developing PSD would have been very evident in the very tall people; this has proven not to be true. If the faulty resorption of the human tail would be a reason for PSD, a pilonidal sinus would be seen from birth on. In reality, pilonidal sinuses mostly arise new in previous healthy people during puberty. Neurocutaneous tracts, as proposed by *Tourneaux*, have never been seen in histology.

***Acquired theories postulated***

As World War II passed by, more than 78.000 American soldiers were diagnosed with PSD between 1941 and 1945 [7], and a further 530.701 between 1944 and 1951, as reported by *Buie* 1952 [8]. As the US Army was the first army to introduce mechanized warfare with the introduction of Jeeps, PSD was immediately attributed to driving in cars, hence coining the name "Jeep's Disease"-[9].

*Patey and Scarff* (1946) believed, that bumpy rides on hard seats led to hair being suctioned into small gluteal injuries [10]. in 1953, *Hueston* proposed that obstruction and infection of hair follicles to be responsible - hair with its complete root would lodge itself into a deeper skin area [11]. As Palmer in 1959 realized, PSD was more common during puberty [12]. As increased muscle development is typical during puberty, he proposed gluteal traction onto the skin to be the main reason for invagination and pilonidal sinus development. The most unconventional theory until now was from *Ahmad* (2005), who proposed that *"wrong sexual thoughts"* would activate special sweat glands in the gluteal area, which would lead to a PSD [13].

***Acquired theories answered***

Regarding *Buie´s "Jeep´s disease"* and related driving of cars – it became evident post World War II, that marching soldiers had the same pilonidal incidence as driving soldiers; so *"Jeep´s disease"* was observational bias, as during war, young male soldiers tend to be in close quarters, therefore being prone to PSD [14].

The suction theory of hair by *Patey and Scarff* seemed appropriate, but did not answer the question of anatomical region: most gluteal muscle and most of suction is generated close to the anus, where the intergluteal fold is deepest [15]. But PSD does not arise here; in contrast, a pilonidal sinus in close proximity to the anus is very rare [16]. Most cases of PSD arise cranial to the intergluteal fold and in the proximal third of the intergluteal fold, where the intergluteal fold is shallow.

The obstruction and infection of hair follicles as proposed by *Hueston* in 1953 is just a nice theory and never been observed. These obstructed follicles resulting in hair moving into the deeper skin from here, especially as the hair with a root is large and blunt tipped, therefore impeding movement into deeper tissues. Furthermore, 1/5th of the PSD cases is seen in women.

The large majority of them don't have any telogenic hair present in the upper third of the intergluteal fold or above; so, there is no hair from here to be inflamed or to move into the skin. Furthermore, pilonidal sinus is seen in scars and areas without any hair follicle.

This shouldn't be possible due to *Hueston* and his obstruction/folliculitis theory. Gluteal traction of hair follicles, as *Palmer* proposed, should generate most of the PSD in sporty people, who develop a larger and more powerful gluteal muscle. In fact, we see the opposite: less cases of PSD in sporty people.

Commenting briefly on the proposal of *Ahmed* (2005) - there are no special sweat glands found in the buttocks. PSD patients sweat less than average individuals [17, 18], have the similar testosterone levels as other people, and their sexuality is adequate in relation to their pubertal age behavior [19].

While being widely used in the past, mercury is now known to be highly neurotoxic and not used anymore for inner application. Instead, better and more efficient alternative wound disinfectants such as PVP-iodine, chlorhexidine, silver sulfadiazine, etc. are used. Nowadays, thiomersal (sodium salt of an organic mercury compound) is still used as a preservative in cosmetic and pharmaceutical products.

***The most likely reason for Pilonidal Sinus Disease***

So, if we see a PSD more often in some families, in people that have been excised largely before and around puberty: What are the reasons for that acquired disease with an obvious family trait?

*Bosche, et al.* proved that the pilonidal sinus nests contained between 0 and 403 loose hairs; and on light microscopy they discovered that 75% of the hair in the pilonidal sinus were without root [20]. Most of the hair was cut at one or both sides, and the mean length of the hair in the sinus nest was less than 1 cm [20].

They additionally proved that stronger hair was significantly more present in PSD patients when compared to normal patient population [21]. Scientific forensic biologists from the Munich Police could successfully prove that most of the hair in the nest resembled hair from the head, and not from the intergluteal region [22]. In fact, occipital hair was present in this region immediately at the end of any haircut, despite regular protective measurements [23].

Interestingly, *Gosselink* – using Electron microscopic methods - could depict hair just entering the skin in 2017, thus proving that sharp hair with "right" directional scales was the culprit, that penetrated into deeper skin [24, 25]. Ruptured hair, as well as hair with its root, are too large and too bulky at the end to enter the skin, which could be demonstrated by *Bosche* [20].

***Consequences of the short hair fragment genesis of PSD – an outlook***

What are the wider reaching consequences of this new findings related to sharp hair fragments being the culprit of PSD?

- We have to think about how to reduce the amount of sharp hair fragments being present in the intergluteal fold. Wet haircuts are better than dry haircuts; and scissor cuts are better than machine cuts, as they produce less sharp hair fragments.
- The hairdresser gowns and other protective measures have to be improved.
- Every male should have a shower after going to the hairdresser, washing out the small hair fragments between neck and gluteal muscles area.
- In patients with lots of hair growing in the intergluteal area, depilation treatment of the intergluteal area can be of use. Intergluteal hair is able to catch and hold sharp hair fragments from the top; it keeps them in the intergluteal fold. As longer this sharp hair fragments stay in the intergluteal fold, as larger is their change to be drilled into the skin through intergluteal movements when walking. Regular use of depilatory cream and razor depilation adds 50% to the postoperative recurrence rate, as proven by *Petersen and others* [26]. Today laser depilation seems to be the best way to enable a long-term depilation needing no further compliance than the 5-10 sessions. Laser depilation works best in dark haired patients, which are more prone to PSD.
- In term of future research, we need to identify the persons at risk, the ones that have a family history of PSD or who have 2-5 years´ earlier disease than others. Shampoos or formulas need to be developed which may permanently soften the hair, with the effect of lesser short hair injections. Could a protective spray covering the intergluteal skin prevent further injections of hair in the glabella/intergluteal region? Alternatively, a skin treatment which improves penetration resilience of the skin to the small hair fragments could theoretically lead to the same positive effects. Until now, perforation resilience of the skin has not been investigated so far, which is an interesting area of research. Another research topic which will be published on soon will tell us if the glabella sacralis region just above the intergluteal fold has major influence of the amount of hair entering the intergluteal fold.

There are numerous questions to be answered. One hundred- and ninety-years following Mayo's findings, there is reason to be optimistic that PSD prevention can be improved within the next 10 years.

## Conclusion

These new findings may explain why recurrences/new diseases occur within follicle-free areas – such as scars and without any preceding infection. This may aid in the prevention of PSD.

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