



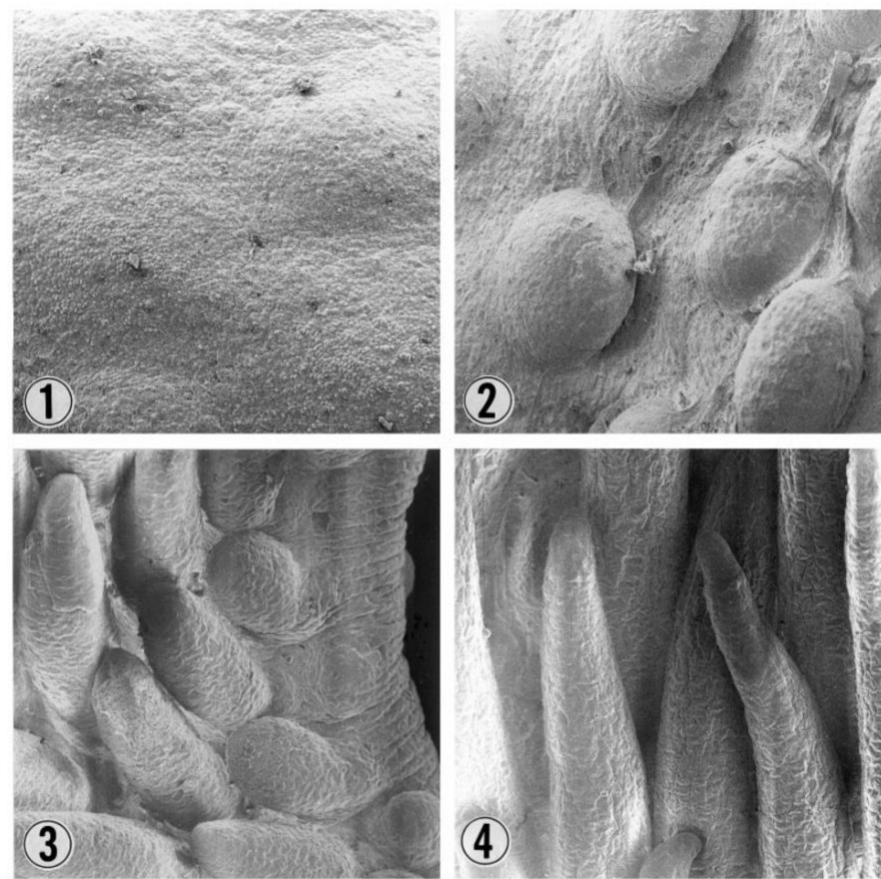
# TWISTED

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Deviantly  
growing  
feathers





Figs 1±4. Morphological development of the outgrowing embryonic feather follicle: 1, HH 34, dorsal neck; 2, HH 36, dorsal trunk; 3, HH 39, forearm; 4, HH 40, hand; SEM, all @gures~130.

Left:  
Development of the outgrowing embryonic feather follicle: Fi. 1, Hamburger-Hamilton (HH) stage 34, back-neck; 2, HH 36, back-torso; 3, HH 39, forearm; 4, HH 40, hand.

Below:  
Fig. 6. Section of the early protruding embryonic feather follicle, HH 34, showing centrally ingrowing primary capillaries (long arrow) derived from early dermal blood vessels (short arrow); a multilayered epidermis and early connective tissue fibres are visible.

Fig. 7. Longitudinal section of the freshly emerged embryonic feather follicle with its young papilla (P) epithelial roll marked with arrows), HH 42.

Fig. 8. Immunohistochemical demonstration of α-keratin in the growing embryonic feather follicle, HH 40-41, clear positive responses in the covering sheath and barbule cells (B), negative responses in the ramicels (R) and papilla (P).

**Assumed is that twisted feathers are not caused by a deficiency or excess of a particular nutrient. Twisting and doubling of feathers is for as far as I know more something that happens in the process of feather development. A few musings on why a feather may be twisted.**

#### First feathers first

Chicken feathers begin in the embryo when the feather papillae are formed. When something goes wrong during their formation, there are consequences later on. A small deviation in the construction and arrangement and precise function of the cells can have major consequences in adult feathers. The cells must be correctly arranged in the papilla. The cells that will form the quill and the cells that will form the barbs. If a doubling or rotation takes place, even if it is very small, the feather will not be formed correctly. Figures 1-8 show how meticulously the cells are arranged. By showing you this, it is clear how little needs to go wrong to cause an incorrectly growing or malformed feather.

#### After birth

... a chick may get injured. For example, by pecking of other chickens. If such an event affects the arrangement of the feather follicles,

there is a chance the feather will be deformed. This is not something that happens often, however, it is possible. Just think of tails being pecked to pieces into the flesh due to feather pecking (cannibalism). Injury can also be a reason for deformed feathers in later life.

#### Genetic basis

Twisted feathers are not investigated in ways of test-breeding and see how it inherits. Nor are split feathers, doubled feathers, absence of feathers like split wing or open wing missing the axial feather. These abnormalities (if so) might be caused by multiple recessive genes (Crawford) in a certain configuration. If such a trait isn't a breed characteristic and it suddenly happens, it would be sufficient to change the combination of the parents to less related. Not all siblings will have twisted feathers, it is just one or two of them, the (un)lucky one. If all have abnormalities, given it is caused by the magic bunch of recessive genes becoming homozygous, then you might speak of inbreeding (depression). Otherwise these genes wouldn't come together after all. When heavily inbreeding, you can fix preferred traits and unwanted things simultaneously. This isn't a judgement, it is an observation.

#### The Keratin Thing

Besides the origination and development of where feathers will grow, there is another determining factor for the later feather which is assumed of less influence in twisted feathers: the keratin that makes up the feather. There are alpha and beta keratin, this has to be produced by the right cells in the right way and also in different shapes depending on whether it is a philoplume, down feather, contour feather or a hard feather for tail and wings. A defect in keratin formation can have a genetic cause or an environmental cause. Think of exposure to toxins, disease, lack of nutrients, etc. This is an extremely diffuse area that you can't really do anything without knowledge. Research sources referring to industrial poultry can be of use if you are familiar with all the nutrients and what they affect. In this case, it is not a single animal showing abnormality in feather growth, there will be several.

#### Finally

If something happens in your chicken's development you don't want and it's something structural you can't rule out based on your genetic knowledge, then deduction of reasons is a way. So there are three possible reasons: a mutation in the

embryonic stage, cause could be genetic or a developmental defect, time to buy a lottery ticket because you have hit the jackpot by creating a combination of recessive genes through inbreeding, or due to an injury after birth.

Sources pictures & text:  
Embryonal feather growth in the chicken, Wilfried Meyer, G. Baumgärtner  
Cornification, morphogenesis and evolution of feathers, Lorenzo Alibardi, DOI: 10.1007/s00709-016-1019-2

#### Dessert: get intimidated

The paper of Lorenzo Alibardi 2016 on cornification, morphogenesis and evolution of feathers, makes your head spin. If you thought you knew all about chickens, descend from your cloud as fancy chicken breeder. To give you an indication of what processes take place before there is a feather on your chicken and what genes and types of keratin are needed for this, a picture that I don't understand the content of and which nicely illustrates that a 'feather' is not a simple tool. In fact, it is amazing that most chickens have well-shaped feathers!

Fig. 2 Schematic drawing showing the gene organization of IF keratins (a,b) and CbetaPs (c, d) with an indicative map distribution of the main proteins in a feather (e). a Chromosome locus for the alpha-keratin type I cluster (the locus for type II can be in the same or in different chromosomes according to the species) with only few genes indicated with their transcriptional directions 5'→3' (arrows). b An example of a gene containing eight coding exons (black) separated by introns (In, pale). c Chromosomal locus containing the EDC, where between the S100A9/loricrin (LOR) and proline-rich protein/cornulin/S100A11 (PRP, CRNN) genes are localized those for the different types of CbetaPs (CβPs cluster comprising in the order from left to right in 5 to 3 direction, claw, feather, beak CPs). d The gene structure for CbetaPs, consisting in one intron (In, pale blue) and two exons (black) with the coding region (Cr) only within the second exon. e The expression/localization so far determined for IF keratins and different CbetaPs with their specific chromosomes (indicated as feather keratins by Ng et al. 2012, 2015; Greenwold et al. 2014; Wu et al. 2015)

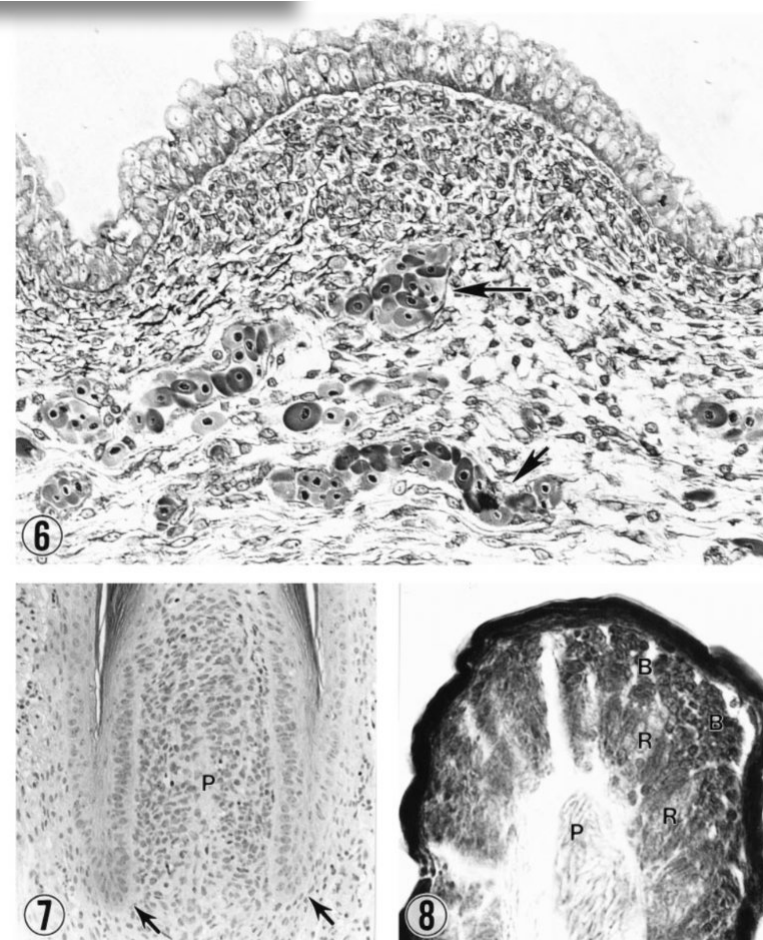
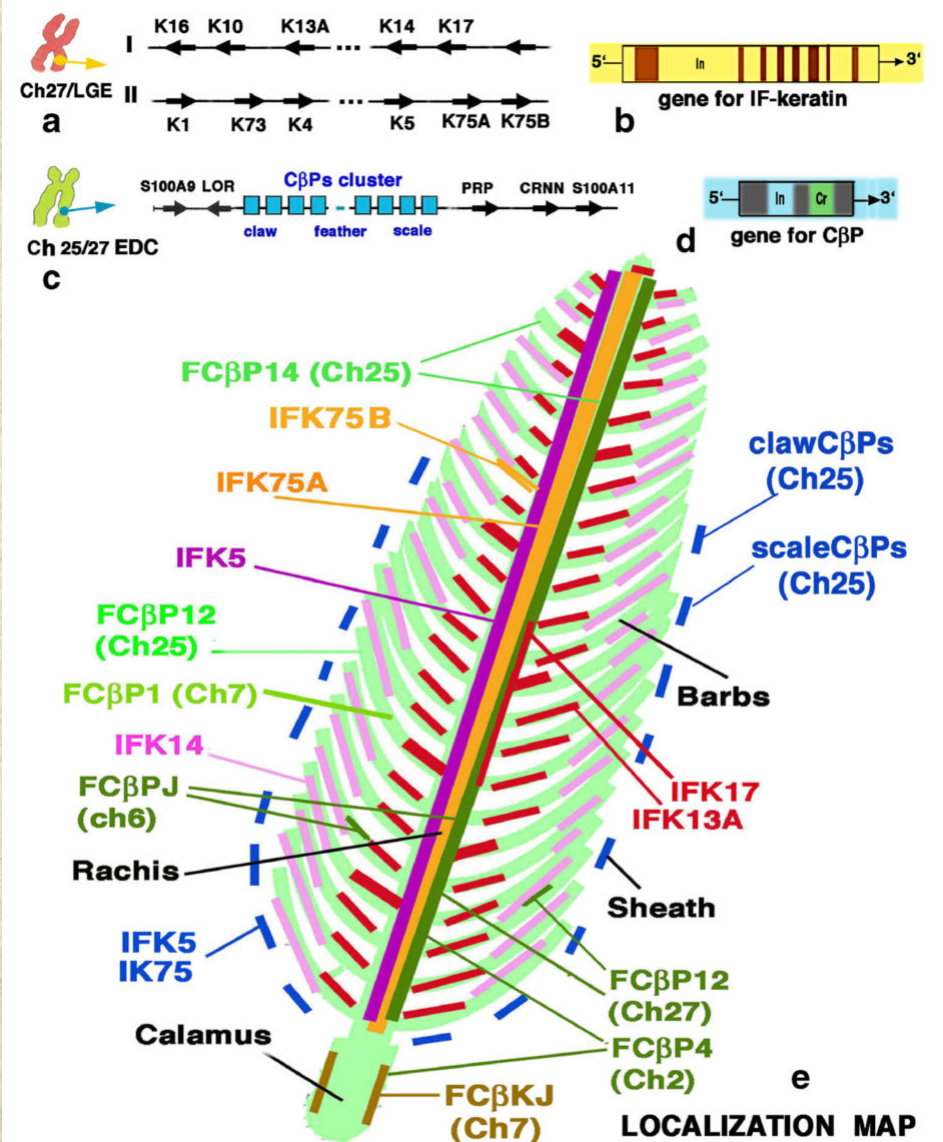


Fig. 6. Sagittal section of the early protruding embryonic feather follicle, HH 34, with centrally ingrowing primary capillary supply (long arrow), derived from early dermal blood vessels (short arrow); a multilayered epidermis and early connective tissue fibres are visible. Technovit embedding, PAMS staining, ~375.  
Fig. 7. Longitudinal section of the freshly inserted embryonic feather follicle with its young papilla (P) (epithelial roll marked with arrows), HH 42. Technovit embedding, H.E. staining, ~245.  
Fig. 8. Immunohistochemical demonstration of α-keratin in the growing embryonic feather follicle, HH 40±41, distinctly positive reactions in the covering sheath and the barbule cells (B), negative reactions in the rami cells (R) and the papilla (P). Paraffin section, PO staining, ~600.