VITILIGO IN CHICKENS

May 2023 - Text: Sigrid van Dort
Photos: provided by breeders worldwide & research papers see links (doi)

This article is about coloured chickens getting colourless feathers. This can be a part of the feather or the whole feather. It manifests in different ways: a few colourless feathers each moult or all the feathers come back without colour instead of coloured or reverting to the default colour. Sometimes there are other problems too.

First: it's not hysterical mottled
When you have a chicken that keeps getting 'whiter' you quickly think of mottled suddenly becoming visible. It is indeed true that chickens heterozygous for mottled, which already have some white feathers on the head, can get more 'stray' white feather points after moulting compared to the year or years before. Vitiligo in chickens, however, is different from mottled. Yes, mottled chickens can get vitiligo as well, yet there is super or hysterical mottled. The pigment stop at the start of feather growth doesn't switch off to form only a white feather tip, pigment is delayed in entering the feather.
It is frequently seen in Serama and also in other breeds like Cochin bantams. In general breeders select for the expression of mottled throughout the years. See also my article on mottled.
Vitiligo is a different beast.

History of vitiligo in chickens
It was geneticist Robert Smyth Jr who had a line of chickens with vitiligo, at least some of them. He started breeding with them to see how it would inherit. At some point, he had 70% of the chickens turning completely white at maturity. We are talking about the 1980s.
The group of chickens still exists at the University of Arkansas and is used for study all over the world.

A partridge Brahma hen with vitiligo. Chicken from a friend of Mellissa Mathe, Australia.

Several lines have since emerged with the SL101 line showing pigmentless feathers earliest, at 6 to 10 weeks of age.

In addition, two more lines of Smyth chickens are kept, the Brown Leghorn line BL101 in which there is relatively limited occurrence of vitiligo (about 2%) which is seen as being

susceptible to vitiligo and the other line: Light Brown Leghorn (LBL101) in which no vitiligo susceptibility is present.
This susceptibility should not be seen as in our chickens, at random.
Because it is possible to induce vitiligo by an injection of a stuff with a difficult name *(5-azacytidine, a DNA methylation inhibitor)*. This is a test developed in the late 1990s. This is only for research in which everything in the chickens is as much the same as possible except susceptibility to vitiligo. This is because you also need a 'control group' if you want to study something, to compare with.

Vitiligo in humans and chickens
In humans, this is loss of pigment in the skin, also known as 'vitiligo'. This can be cosmetically negative especially in darker skin, think spots in your face. It is very noticeable, especially in summer and particularly if you have very dark skin.

Fortunately, there are now role models to help with acceptance. Both for the person who has it and for the outside world. It usually starts during adolescence and early adulthood. In humans and also in chickens, it can be only part of the skin or feathers without pigment. In humans and slightly more often in chickens, pigment can return.

Also something that goes along with vitiligo but not necessarily is alopecia. People with vitiligo are 4x more likely to also have bald patches than those without vitiligo. In chickens, this looks different. Both vitiligo and alopecia have an autoimmune component in which corticosterone has a corrective effect. But you don't use that stuff for fun.

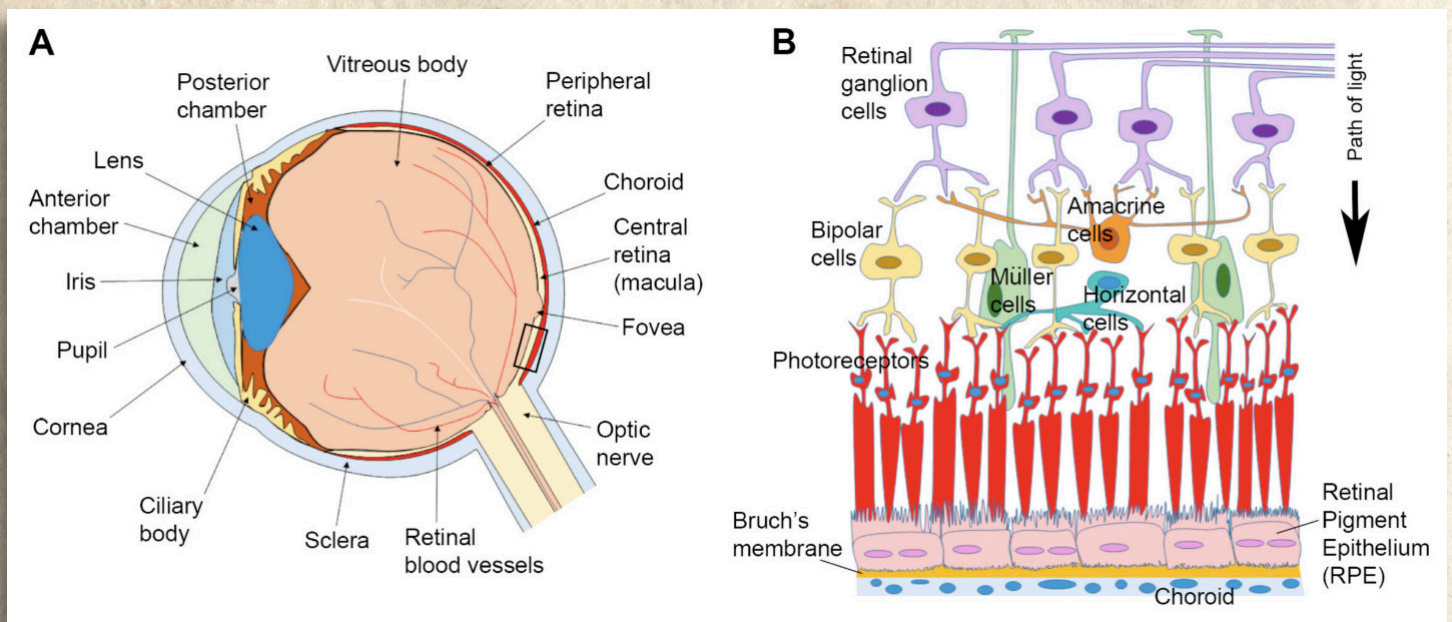
Because of the (psychological) impact of vitiligo in humans (it is not known if chickens have a problem losing their colour), there has been a lot of research into what is actually going on.
To be complete, there are a lot more animals that have vitiligo (loss of pigment), such as horses, pigs etc.

The inheritance of vitiligo in chickens
Vitiligo is polygenic, meaning it requires multiple genes (and modifiers) and an innate susceptibility. The same applies to feather abnormalities that can occur in vitiligo chickens, they are two different things that may well be related in terms of autoimmunity.
It is also possible that there is an interaction in feather development.
It is usually confined to the large flight feathers and later some ruffled feathers.
Total baldness (alopecia universalis) is rarely seen earlier than in adult chickens. The characteristic feature here is: short (less than half a centimetre) underdeveloped feathers. Once normal feather growth resumes during moult, the feathers dry up, stop growing, and often break. This sounds like the 'wing patch' in lavender, the dried-up pin feather. The difference is that in vitiligo(alopecia areata and in bad cases universalis) no more feathers grow at some point. This is not the case in lavender (isabel) wing patch, which grows back every year in the form of dry pin feathers.

The chicken with vitiligo
In addition to the things mentioned above, loss of pigment, more problems occur in chickens with vitiligo. For instance, feather quality can be poor in some birds. The feathers may look dry, ragged, or poorly developed. In chickens with vitiligo, this is called 'alopecia areata or universalis'. It is a small percentage having bad feathers and the word 'bad' also varies.

Viligo and impaired vision
Furthermore, they can become visually impaired or blind, this is called 'retinal dystrophy'. About 40% of vitiligo chickens go blind, that is, those from the Smyth line. This is due to the destruction (through inflammatory processes) of the pigment cells in the choroid (located at the back of the eye). In the study of the eyes of vitiligo chickens, it became clear that the whole autoimmunity system turns against melanocytes, including in the eyes. Want to know more about this how pigment has anything to do with the eye search wiki 'Retinal pigment epithelium'. See *illustration* below.

Illustration: doi.org/10.3390/cells7020016,- https://www.mdpi.com/2073-4409/7/2/16/htm



Blind chicken since 1 x vitiligo. Feather colour came back the next moult, but eyesight not. Chicken was a few years old when vitiligo hit. She is now 10 years old and living with Serama and doing well, there is still some colour vision. Chicken from Katherine Ellsworth USA.

With the immune system primarily sending T- and helper cells wherever there are melanocytes, this takes place not only in the feather papillae but also in the choroid of the eye. The cells there begin to swell and the dendrites retract, the pigment granules (melanosomes) are irregular in shape and cells clump (for this phenomenon and what it looks like under the microscope, see: Delayed-Amelanotic (DAM or Smyth) Chicken: Melanocyte Dysfunction In Vivo and In Vitro, link at the end of the article).

The inflammatory reaction damages the eye, including around the optic nerve and 'pecten'. According to wiki the pecten or pecten oculi is a comb-like structure of blood vessels belonging to the choroid in the eye of a bird. It is a non-sensory, pigmented [there you go] structure that projects into the vitreous humor from the point where the optic nerve enters the eyeball. We don't have that thing.

In totally blind chickens, the whole retina was damaged, in partially sighted chickens only the choroid was partially damaged. In chickens where vitiligo also affects the eyes, the colourlessness of the feathers goes hand in hand with the reduction in vision. The study is mentioned at the end.

Hypothyroidism
Furthermore, vitiligo chickens can get hypothyroidism, this impacts the feathers, which become silky and can be longer than normal. This occurs mostly in hens. In test pairs, in the F2 bred for feather abnormalities one hen was bald after a year, which can be called alopecia due to the autoimmune reaction.

See more nerdie stuff last pages

Why does the Smyth chicken or your chicken get vitiligo?
These chickens have certain retrovirus genes in their genome (endogenous retrovirusses), and exactly the ones that cause immune problems. Pieces of retroviruses are in all DNA (in humans more than in chickens also called 'junk DNA') so also in yours.
They are small pieces of DNA from a virus in a cell, which a virus can 'write back' to RNA, hence 'retro' because exactly the opposite of putting RNA into a cell and letting it do the multiplication.
This virus DNA has been there from a very long time, several million years ago, so no worries.

These retrovirus genes are not just bad, they allow for a lot of genetic diversity and thus the possibility of adaptations.

Pieces of virus DNA which ended up (i.e. a few million years ago) in the DNA of the ancestor of today's chicken are not always wrong, it just depends on what piece of what virus.
Like for instance the avian leukosis virus, something an average chicken breeder gets the creeps of because tumours etc and a certain death, usually near adulthood or failing health forever. However, a piece of this virus in the chicken's DNA can also be useful, such as causing slow feathering (alv or avian leukosis virus 21). ALVs are used for gene identification too because they appear in many genomes, you can use them as so-called 'markers' to see where in the genome something is that you want to examine.

Getting back to vitiligo, the Smyth line chickens have many of those ancient endogenous viral fragments

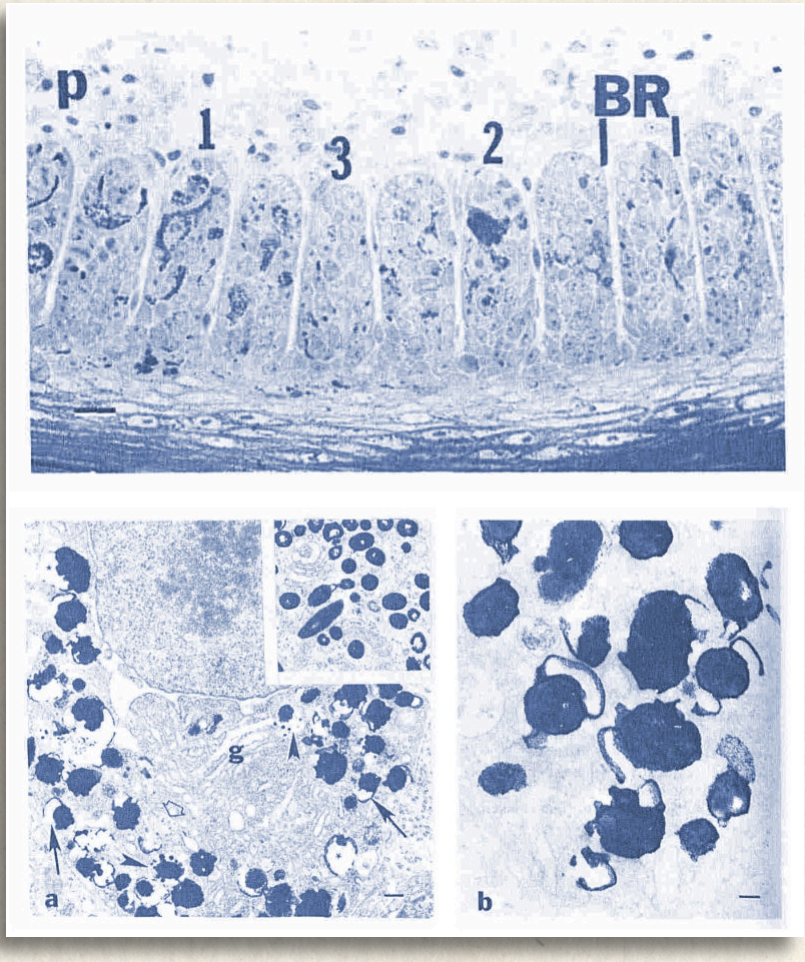
A young Chabo cock with short lasting vitiligo in 1st adult feathers. Acquired for testing by Udo Ahrens, Germany. He was not viable, zero chicks from him.

in their genes, and exactly those, which affect the immune system. A wrong influence in this case. This high number of ev genes may have to do with the huge inbreeding i.e. relatedness and therefore homozygous recessive genes and their modifiers (whichever they are) of the Smyth chickens. All sub-lines of Smyth chickens also have similar genetic similarities.

Now you want to know what those virus fragments are, not that you can do anything with them, well, they don't know so which ones and where they are on the genome. They do know that the virus fragments are located together like a cluster (inbreeding result). Perhaps they know by now (have read studies up to December 2022), however, I have not been able to find anything, maybe looking in the wrong place (ncbi etc.). How important is this to know, not so, for a hobbyist.

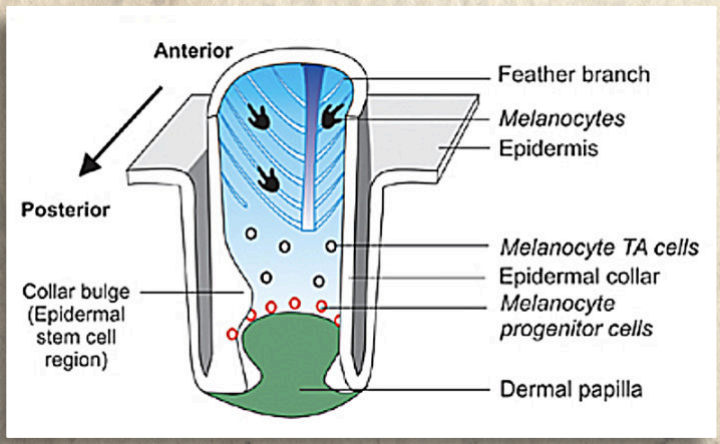
What happens to the pigment in a chicken with vitiligo?
The chicken is born with normal functioning pigment production, the whole system is working as it should. And then at some point under the microscope, abnormally shaped melanocytes can be seen in the epithelial barb ridge (collar) of the feather papilla, where the pigment cells are.
The pigment cells deliver the pigment granules (melanosomes) via dendrites (a kind of extensions) to the keratin cells which then put the colour into the keratin of the feather (the barbs).

What they saw? The pigment cells were thickened and malformed and the dendrites (legs for pigment transport) were shorter and also irregular in shape. The ability to deliver pigment from the pigment cell



WHAT HAPPENS IN THE VITILIGO FEATHER

Top photo: Cross-sectional segment of a regenerating feather from an adult bird. The barb ridges display several melanocyte stages of degeneration: 1= normal, dendritic melanocyte; 2= melanocyte without dendrites so no communication with the keranocytes; and 3= absence of melanocytes. BR = barb ridge, p = pulp. Bar = 2 um.
Bottom left: Zoomed-in section of a melanocyte (pigment cell) with weirdly shaped pigment granules (melanosomes). a. Section of a melanocyte from a pigmented regenerating feather of an adult Smyth line (SL) chicken. This cell contains many abnormal melanosomes with pigmented extensions (arrows), small pigmented vesicles (arrowheads), and electron-lucent chambers (asterisks); also pre-melanosomes (open arrow), and a well-developed Golgi apparatus (g).
Bar = 0.3 um. Inset: Normal melanosomes from regenerating feather of an adult e+ bird for comparison. Bottom right: Magnification same as left. b= Fine structural (weird) details of Smyth line chicken melanosomes. Bar = 0.1 um
From: "Delayed-Amelanotic (DAM or Smyth) Chicken: Melanocyte Dysfunction In Vivo and In Vitro, 1985



Parts of the feather papilla of a growing feather. Illustration: DOI: 10.1126/science.1230374

to the keratin cell had been reduced. Again later, there were no or hardly any melanocytes present and also less colour in the feathers.

Before the absence of pigment in the feathers was visible, there were already deformed pigment granules (melanosomes) with accumulations and therefore weird protrudings and a kind of tail with extremely high amounts of pigment, i.e. deformed. Remember how black pigment is rod-

shaped and red pigment is roundish? (See inset picture ‘a’ previous page where you can see both rod shaped and spherical pigment).
Anyway, these early vitiligo pigment granules were a mess, irregularly shaped like a bunch of grapes.
There were also partly already broken down pigment-producing cells, by the immunesystem, forming blobs.
Indeed, the abnormal pigment granules (melanosomes) triggered the body's clean-up mechanism because 'it's not right'. Now there is nothing in this story to indicate that vitiligo has anything to do with the funcioning or non-functioning of the immune system. More on this now.

What happens in the immune system of a chicken with vitiligo?
To move on to cleaning up the misshapen pigment granules and also the pigment cells (melanocytes); this mechanism of the body to clean up what is wrong is part of the immune system. Think of immunotherapy in cancer, your own immune system is sent to destroy the malformed cells. This is the same system that kicks in with vitiligo.
How does this happen?

You probably know the immune cells called T-cells, they clean up the 'bad guys'. They respond to the alarm, this can be in different ways, such as after previous infection or by vaccination with the antigen or during a virus attack.
T-cells are also called killer cells, they are a kind of white blood cells. And there are also T-helper cells, they release cytokines and play a role in the adaptive immune system (also called acquired, the system next to the immune system by birth which is the innate). Those guys shouldn't go berserk, because then you die from your own immune response (cytokine storm), but that's another story.

Loads of both these T-cells were found in the growing feathers, including the feather pulp from which 'feather' is made by the keratin cells. Many T-cells were also present in the dying and half-degraded pigment cells but also substances and cells related to an inflammatory event (interferon, T- and B-cells, and macrophages which gobble up faulty cells, the rubbish trucks of the remnants of one's own cells, too). In short, it's a mess in a vitiligo chicken, around and in its feathers...

And then what?
Then there is no more colour in the feathers because the immune system attacks anything that is not okay.
A while before war breaks out in Pigmentistan, an increased amount of white blood cells circulates in the juices (other systems < goes too far here), so there is already an inflammatory response going on a week or two before things go visibly wrong.

Soooo, the immune system causes death and destruction of pigment cells. The only way to stop it is to suppress the immune system. Vitiligo is caused by 'anti-melanocyte auto-antibodies'.
It is not known how this war against melanocytes starts, i.e. what the trigger is. In other words, it is known that there is a CD in the player but how the player is triggered to hear the music (vitiligo) is not known.
Besides, the auto-antibodies recognise not only bird, but also mammalian tyrosinase (a substance needed to make pigment, no-tyrosinase = no colour in feathers.

Left: Julie Locke’s Padua hen in the UK, turned almost white the next moult. Above Peter Williams’s story of a black Hamburg hen in Australia becoming more white every moult. All breeds, all chickens whatever the make can get vitiligo all over the world.

Grace Worthy's gold blue-spangled (yes, very rare colour only in Australia?!) Hamburg hen. She has light legs, Hamburgs have slate legs which is white skin with dermal pigment. The others do have slate legs, her id+ is gone. Vitiligo also attacks the pigment cells in the legs so skin, horn and the eyes. She is almost white now, the blow photos are taken for this article.

This is what recessive white is based on. Oops, that's another story…

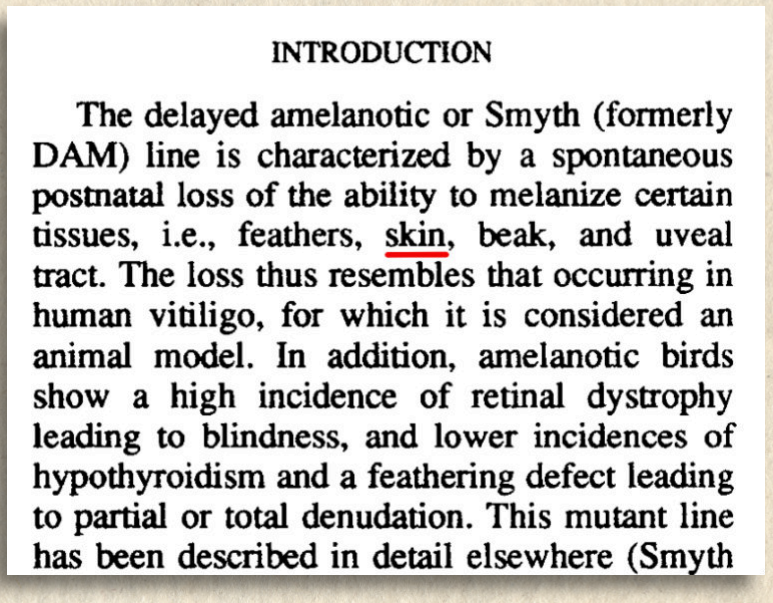
Triggers outside the body that can cause vitiligo
Besides the defect in the pigment cell and the autoimmune reaction to it, both of which disrupt pigment and then make it partially, occasionally or completely impossible to produce colour, there is also an environmental factor that can trigger vitiligo if a chicken is sensitive.
Vaccination with turkey herpes virus against Marek is a trigger.
Without a vaccination, 20% of Smyth chickens have vitiligo, with marek vaccination this rises to 70-95% with vitiligo.

HVT (herpes virus turkey) is a vaccine widely used in the industry against Marek's disease virus serotype-1 (MDV-1 since 1968).
This vaccine is live herpes virus from turkeys of the conventional strain (FC126). This vaccine is used for short-lived chickens in whom it works for a while.
In longer-lived hobby chickens, this vaccine is not sufficient because MDV-1 has become more virulent.

Hobby chickens are mostly vaccinated with a bivalent vaccine (HVT-1 and -2, plus serotype 1 CVI988/Rispens vaccine). The HVT vaccine is a cell-free, freeze-dried vaccine and is therefore cheaper and easier to handle as it does not need to be stored in liquid nitrogen.
Both vaccines, the old-fashioned and the new, are so called live vaccines that actively replicate and give a natural infection so antibodies are made. Chickens are not turkeys, they don't get sick from it but do get antibodies against Marek.

The viruses in these vaccines multiply in the feather follicle and virus particles are continuously spread in the environment through dander. Looking at these flakes of skin can tell if the vaccination was successful (added benefit).
Modern Marek vaccinations are a live vaccine (including the freeze-dried ones).

Here's the thing: chickens susceptible to vitiligo (the Smyth line and maybe your backyard chicken too) get a reaction to Marek vaccination because it... you feel it coming... acts on the feather follicle (Marek loves feathers).
Chickens susceptible to vitiligo get a severe immune response in the feather follicles due to Marek vaccination. This local immune reaction then also spreads to the pigment cells (melanocytes). The melanocytes are in a battleground (with the harmless Marek virus to which the immune system reacts which is exactly what is intended) and if they already had a tendency to be malformed and the immune system was already lurking to eliminate them, there is now a double immune response. In the susceptible chicken, this can trigger vitiligo if it was already dormant (young animals get a Marek vaccination).
Incidentally, in vitiligo-sensitive humans, a herpes virus can trigger this.



INTRODUCTION

The delayed amelanotic or Smyth (formerly DAM) line is characterized by a spontaneous postnatal loss of the ability to melanize certain tissues, i.e., feathers, skin, beak, and uveal tract. The loss thus resembles that occurring in human vitiligo, for which it is considered an animal model. In addition, amelanotic birds show a high incidence of retinal dystrophy leading to blindness, and lower incidences of hypothyroidism and a feathering defect leading to partial or total denudation. This mutant line has been described in detail elsewhere (Smyth

How is vitiligo in feathers reversible?
Some chickens regain their colour at moulting. How is this possible if melanocytes (pigment cells) are destroyed by the immune system = vitiligo?
This is possible because melanoblasts (the precursor cells from which a melanocyte is later formed) are not attacked and destroyed by the immune system. This means that from the reserve cells, the melanoblasts in the dermis, new melanocytes move up into the collar of the feather papilla and start producing (hopefully not misshapen) pigment granules there. If the new pigment cells make well-formed pigment and, therefore, they themselves do not deform and therefore have no shrinking dendrites and can keep contact with the keratin-forming cells, the immune system will not kick in and the pigment can re-enter the feather as normal.

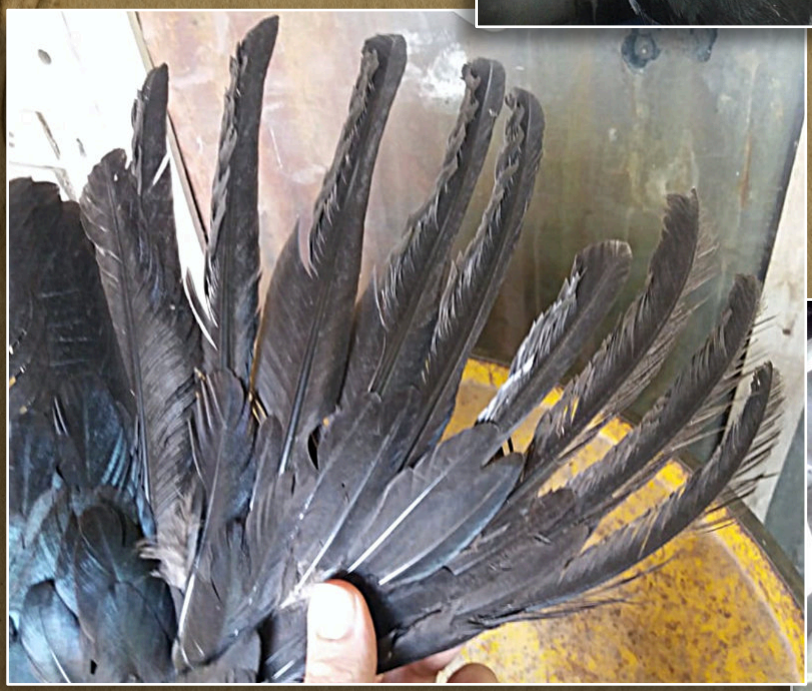
Slate coloured legs turn white in some vitiligo chickens
It took a while (2 days of searching/reading) to understand that not only the melanocytes in the feather papillae are attacked and destroyed. The melanocytes in the skin, eye and horn (beak and nails) are also attacked. I found this in a 1988 study.
Modern literature from 2020 onwards does not address this, only the eye, because blind, but not the skin like that of the legs.
And the Smyth chickens have no dermal pigment, they have ordinary yellow Leghorn legs. Hence it seemed strange that some vitiligo hobby chickens also lacked pigment in the legs (id+, slate legs). Even though they are not albinos, no red eyes and not born colourless, nor are they feather albinos which in chickens is recessive white where only the feathers have lost colour.
It is usually the case when looking for something specific as a hobby chicken breeder, when you search for exactly what you need: 'vitiligo and leg colour in chickens' < you won't find it. Hobby chicken breeders look for details which are not always scientifically researched.
It is no accidental serendipity that it now turns out that the legs also lose pigment, you just have to read everything you find from 1983 onwards. Above is the snippet from the 1988 study mentioning the fact that the skin too can become devoid of pigment. So, that's cleared up too.

Karen Johns' Indian game hen in Australia.
Colour is switched off, then switches on as in mottled but that isn't the case. New melanocytes are made and they are not attacked by the immune system because the pigment is not deformed.
In other feathers colour is switched off completely.

The third photo shows how the pattern should look without vitiligo messing everything up.

Brandi Suits McKnight's brown Leghorn, USA. A hen born in 2019 flipping from colour to completely white, then partial colour and back to colour again. After that, today she is half colour half white, the photo above scratching the dirt.

Jo Gunn in Australia has a father (below) and a son (right). They have dark legs, vitiligo didn't hit the dermal pigment (id+). The melanocytes flip on and off (white mid-sickles son) during feather growth.Thanks to the fast generating of melanocytes from the precursor cells, colour is restored as soon as the attack by the immune system is over. Sort of a single event cuckoo/barred (B).



Giò Gallo Brahma from Italy and his 'black' Livorno. The black hen with vitiligo shows affected wing feathers. The almost white 'black' Livorno cock with vitiligo has a sister, she too has vitiligo (this isn't mottled or exchequer). They have a son who is completely black. More on inheritance in the text.

NERDIE STUFF

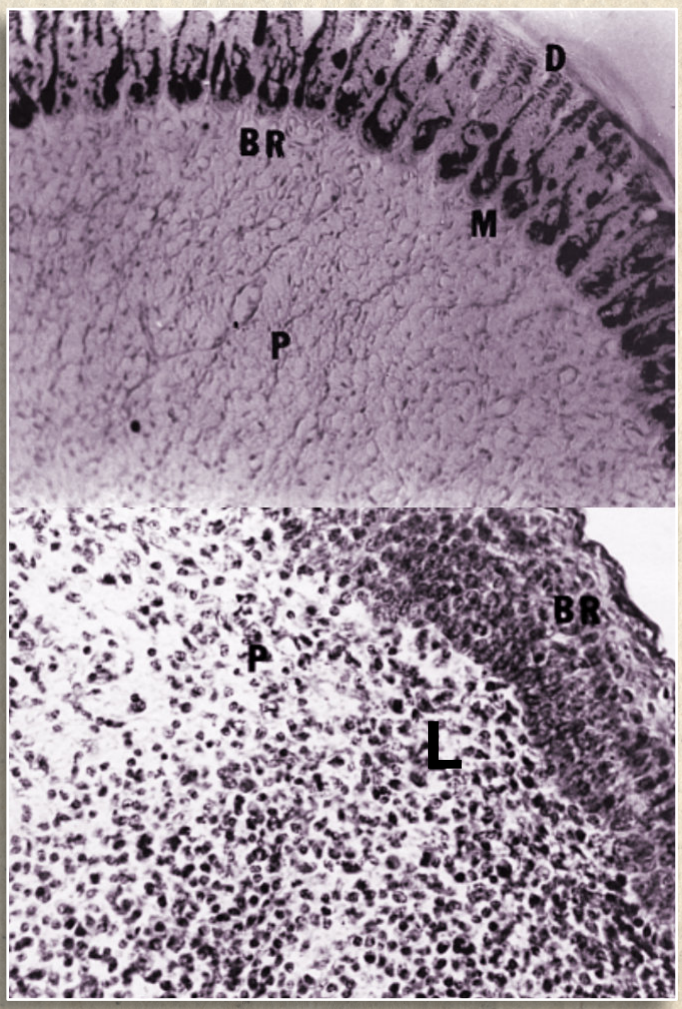
Bad feathers
From Smyth's 1999 study with nice old-fashioned understandable photos of what it looks like.
There must already be a visible feather abnormality in the vitiligo chickens, it's a matter of using your eyes. If you can make a pair of chickens with bad feathers and vitiligo then after two generations you get very bad feathers at 17.4%, which is virtually bald.

Can you reverse it with injections of corticosteroids (as in alopecia in humans?), it has been tried. Two months of applying ointment (0.05%) only gave some swelling of the spot where the feathers should be. So no result. Then injection, four out of five bald test chickens died 10 days after the injections. The fortunate one that survived regained feathers and pigment in the feathers two months after the last injections. Unfortunately, the effect lasted only a few weeks, and the chicken went white and bald again.

This experiment was probably done during moult, because chickens do not grow body feathers for 6 or more years like the hair on our heads.
The difference between humans and chickens is that chickens only have alopecia if they also have vitiligo. The Smyth Line 101, which is the first to become white, does not have alopecia.

Top photo: 14-wk-old male from the Smyth 103 subline showing the basic feathering defect that precedes the varying degrees of alopecic feather loss caused by vitiligo.

Left: a pair of one year old Smyth chickens: male and female exhibiting severe alopecia areata. These birds won't have feathers anymore following their next feather moult.



Cross-section of a developing normal feather of the barb ridge (BR) area. Note absence of leukocytes (white blood cells) in the pulp (P) and the melanocytes (M) in the barb ridges putting melanin into the developing barb cells (D), 292 X.

Cross-section of a developing alopecia feather due to vitiligo. Cellularity - a mess of cells (L) in the pulp (P) and dying melanocytes in the barb ridges (BR), 292 X.

---

7 ways for a pigment cell to die
The melanocyte can die in vitiligo in 7 ways.
Since this is beyond our backyard knowledge so also to rewrite in donald duck language here is the study (2020) https://onlinelibrary.wiley.com/doi/10.1111/pcmr.12955
Picture from the study to show that this is quite complicated and cannot be digested by backyard chicken keepers without a 6-year university degree in molecular of all things study.

There are seven forms of death, including apoptosis, necrosis, autophagy, necroptosis, pyroptosis, ferroptosis, phagoptosis, and Anoikis,

Chantal Berthelot-Lucas from Canada has this silver black-laced Polish hen. In the first photo there is still some dark pigment higher up the front of her legs, in the recent photos, for this article (leg photo right) it is completely gone. There are still a few melanocytes trying to put pigment in the feathers.

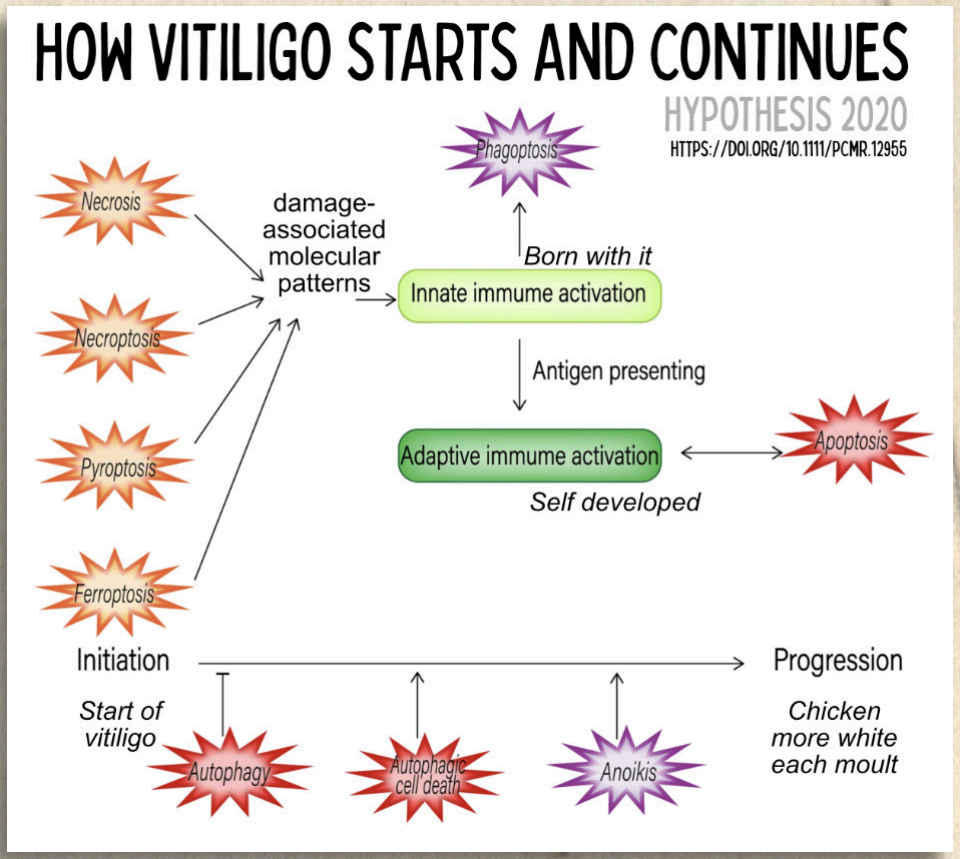
Note: when searching photos of silver laced Padua and Polish, there are several without dermal pigment. It is possible this isn't much fixed. For backyard Polish and Padua this isn't important at all of course. Original does have id+ in legs.

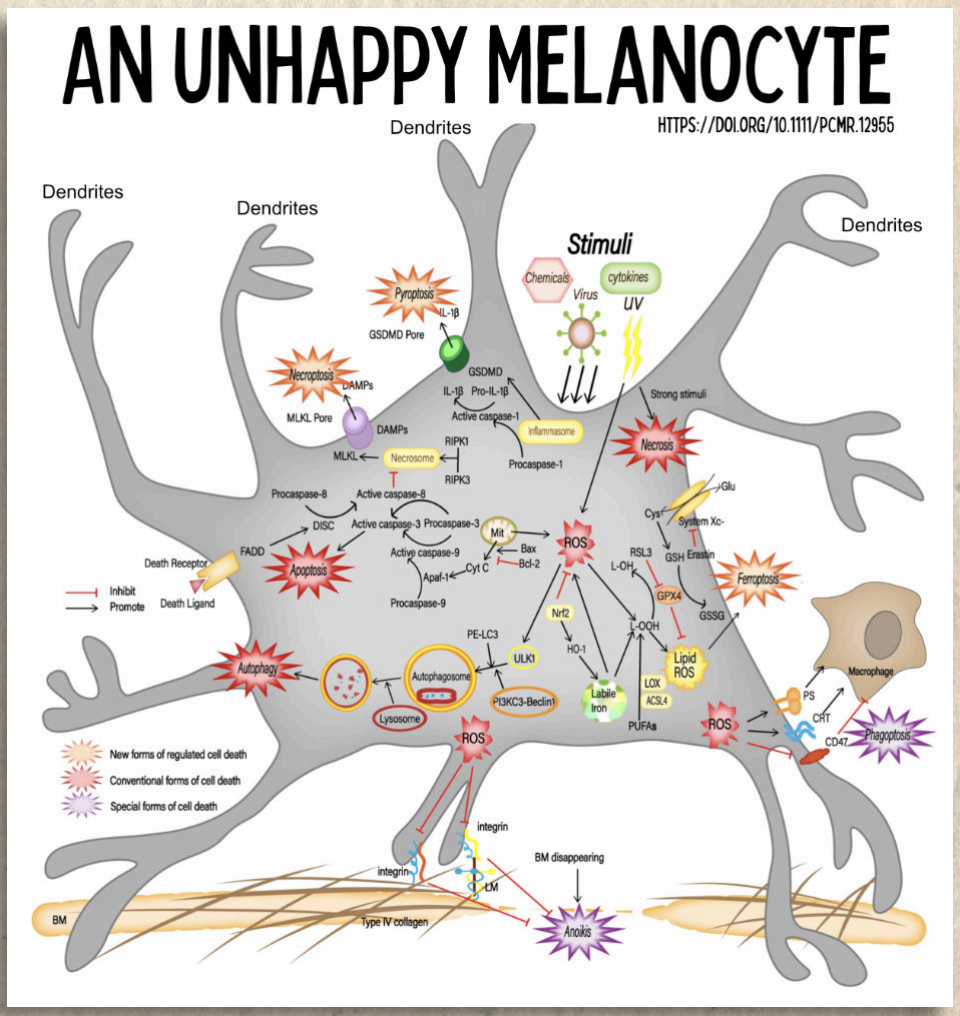
NERDIE STUFF

are likely to participate in the the process by which vitiligo develops.

The hypothesis how it happens
Possible roles of different forms of melanocyte death in the initiation and progression of vitiligo.
Necrosis, necroptosis, pyroptosis, and ferroptosis of melanocytes activate innate immune cells through the release of DAMPs (damage-associated molecular patterns). Autophagy facilitates the breaking of immune tolerance by working as auto-antigen. Innate immune cells then present antigens and trigger the adaptive immune system. With the recruitment of T-cells, more melanocytes are induced to apoptosis during the progression of vitiligo. The infiltration of macrophages (the cleaners) makes it possible for melanocyte phagoptosis. Anoikis could occur in the active phase of vitiligo. Anoikis is a form of programmed cell death that occurs in anchorage-dependent cells when they detach from the surrounding extracellular matrix. Usually cells stay close to the tissue to which they belong since the communication between nearby cells as well as between cells and the matrix provide essential signals for growth or survival. You can look up the other forms of cell death on wiki.

Mit, mitochondrion; ROS, reactive oxygen species; UV, ultraviolet; GSDMD, propyroptotic protein gasdermin D; DAMPs, damage-associated molecular pattern; BM, basal membrane; MLKL, lineage kinase domain-like protein; RIPK1, receptor-interacting protein 1; RIPK3, receptor-interacting protein 3; DISC, death-inducing signaling complex; FADD, death domain; Apaf-1, apoptotic protease activating factor-1; Cyt C, cytochrome c; PE-LC3, microtubule-associated protein 1 light chain 3 complex with phosphatidylethanolamine; ULK1, unc-51-like autophagy activating kinase 1; PI3KC3, class III phosphatidylinositol 3-kinase; HO-1, heme oxygenase-1; Nrf2, nuclear factor E2-related factor 2; LOX, lipoxygenases; ACSL4, Acyl-CoA synthetase long-chain family member 4; PUFAs, polyunsaturated fatty acids; Glu, glutamic acid; Cys, cysteine; GSH, glutathione; GSSG, glutathione (oxidized form); LM, laminin; PS, phosphatidylserine; CRT, cell surface calreticulin; System Xc-, glutamate/cysteine antiporter.





From: The fate of melanocyte: Mechanisms of cell death in vitiligo; 2020 (https://doi.org/10.1111/pcmr.12955)

A dark (silver pencilled) and a partridge Brahma from Beck Loy in Australia.
It seems vitiligo is more common in Brahmas who have blood from the import several years ago. One way to stop this phenomenon is to cross to enough original Australian Brahmas to 'dilute' kinship. The genes responsible for susceptibility to vitiligo seem to lie together and are therefore inherited together. That's why it possible to select for these vitiligo susceptible genes (Smyth did so). Too much inbreeding is not a good idea, as with all recessive genes and mysterious modifiers you don't want.

NERDIE STUFF

On chickens going blind with vitiligo

Of chickens with vitiligo (progressive), 5-20% become partially sighted or blind. This occurs at the same rate as feathers whitening, i.e. loss of pigment cells.
It sounds like a nightmare:

Mononuclear leukocyte presence and MHC class II expression in choroids of healthy controls and Smyth line (SL) chickens with and without vitiligo and visual impairment.
(A) Representative CD4 or CD8 IHC-stained cells in frozen sections (7 μm) of eyes from 12-week-old chickens. BL-NV: Normally sighted (NV) parental Brown line (BL) control without vitiligo (no-vit); SL-NV: normally sighted, vitiligo-prone Smyth line (SL) chicken without vitiligo; SL-IV: vision-impaired SL chickens [partially sighted (P) or blind], all SL-IV chickens had vitiligo. RPE (retinal pigmented epithelium, see picture page 2) is destroyed in blind SL-IV chickens.
(B) The appearance of eyes in an SL-NV chicken without vitiligo (top) and a vitiliginous, blind SL-IV chicken (bottom). (C) Presence (% area) of T cell subsets (γδ, CD4, and CD8), B cells, macrophages, and MHC class II+ cells in choroids of controls, SL-NV, and SL-IV (includes P and blind). At each age, mononuclear cells were identified by indirect immunohistochemistry (IHC) using chicken marker-specific mouse monoclonal antibodies, biotinylated horse-antimouse IgG secondary antibody, and Vekta-stain Elite ABC reagents (streptavidin and biotin-conjugated peroxidase).
Size-bar = 100 μm. Data are mean ± SEM; per age group, n = 6 and 3 for control and SL-NV, respectively; SL-IV: n = 0 at 1 week, n = 1 at 4 weeks, and n = 10 at 12 weeks. (A–C) Within an age group and across sample categories (control-NV, SL-NV, and SL-IV), mean levels of individual cell types without a common letter are different (P ≤ 0.05).

The End.

A few references that may be of interest:

Article on mottled: https://www.chickencolours.com/index.php/articles/

Marek vaccination latest open access: https://www.frontiersin.org/articles/10.3389/fvets.2022.873163/full

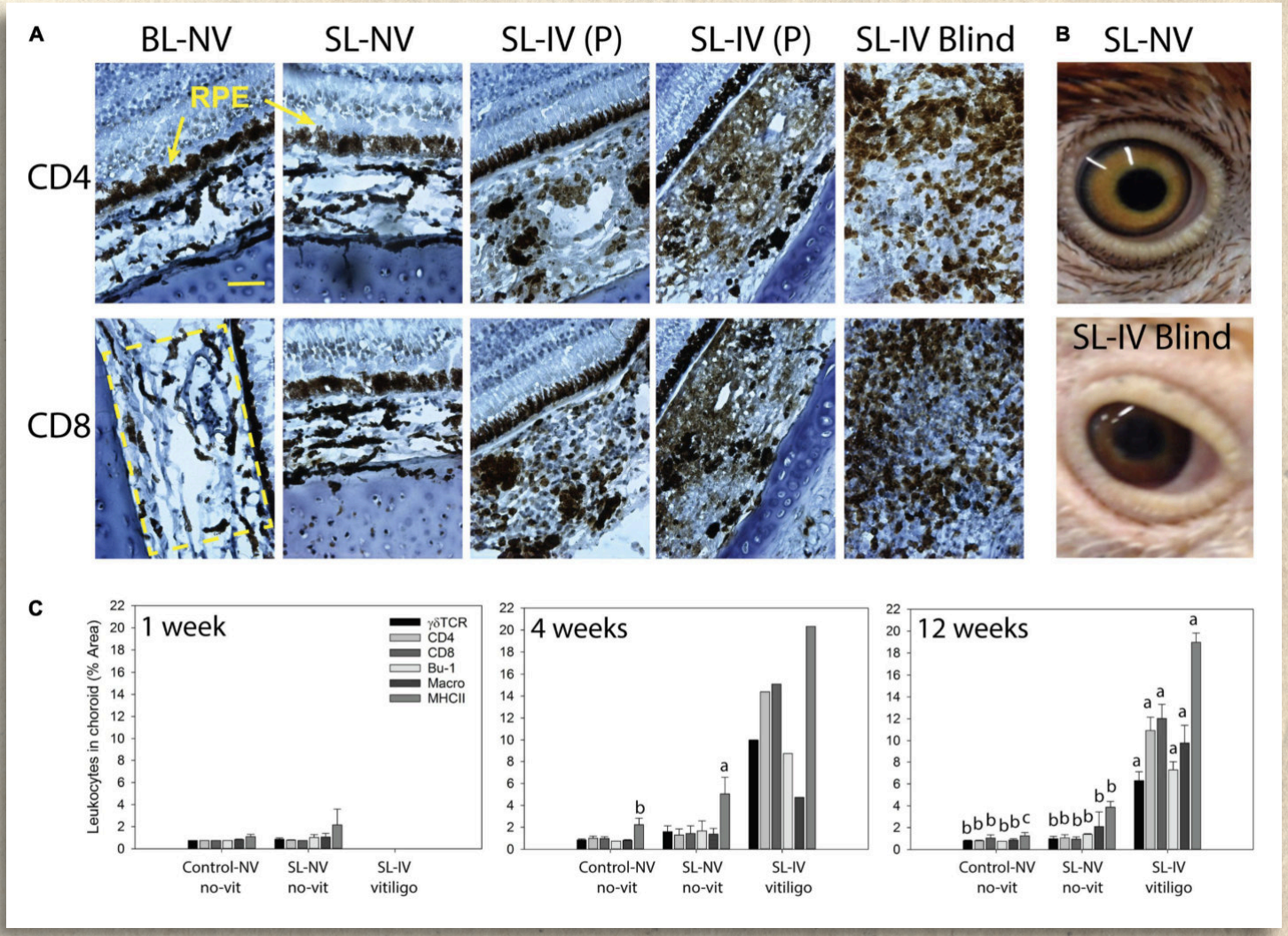
Bad feathers in vitiligo chickens, 1999: https://pubmed.ncbi.nlm.nih.gov/10674368/

A general article on auto-immune problems in chickens, 2006: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3292797/pdf/ukmss-45239.pdf

Via this link you can find a lot more articles on vitiligo and Smyth chickens, bottomscroll: https://pubmed.ncbi.nlm.nih.gov/10674368/

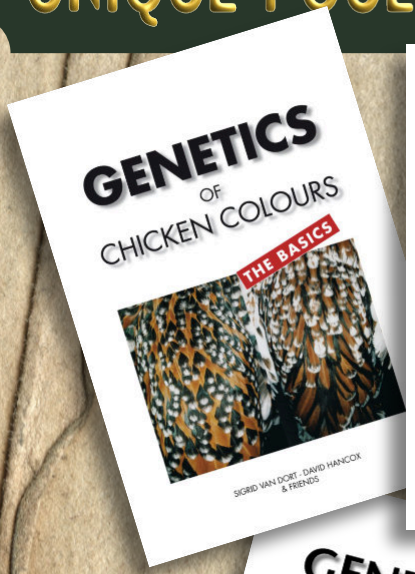
Research on going blind, 2022: https://pubmed.ncbi.nlm.nih.gov/35547230/

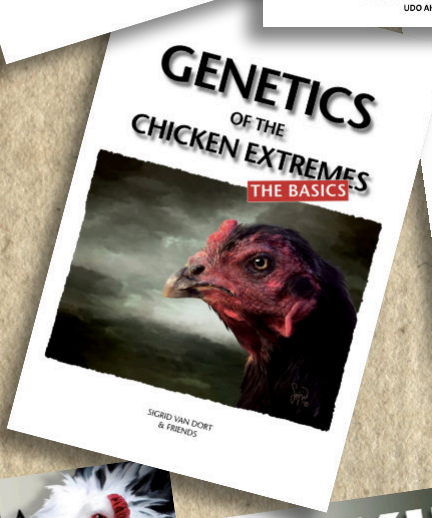
For old microscope photos what's happening, 1985, https://www.sciencedirect.com/science/article/pii/S0022202X8690850X).

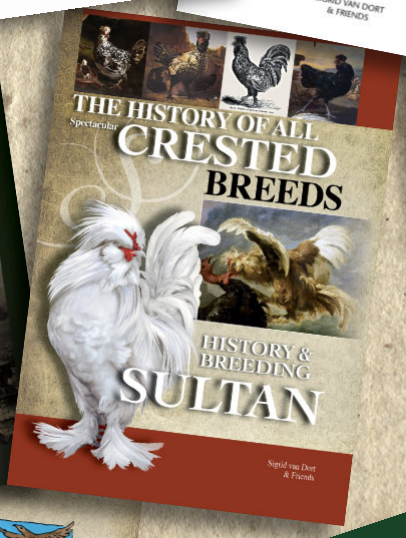


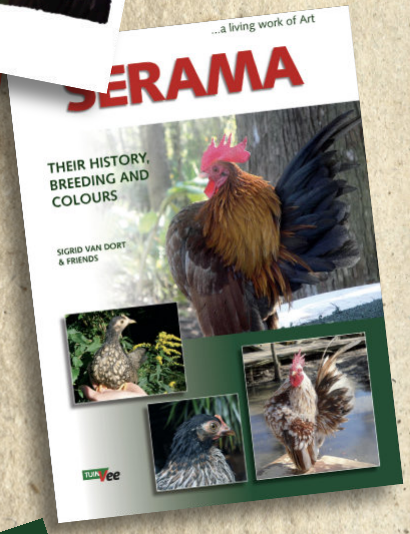
(DOI: 10.3389/fmed.2022.846100)

UNIQUE POULTRY BOOKS FOR CHICKEN ADDICTS









WWW.CHICKENCOLOURS.COM