Dominant white chickens are the sweetest, scientists say...

Dominant white and its alleles
Dominant white is considered to have most influence on chicken colour researchers say (Smyth 1990). Bateson was the first who did research for dominant white (1902) according to Mendel’s Law. Dominant white has its symbol I (capital i) because it inhibits pigment. Dominant white only alters feather colour and has no influence on for instance eye colour.

The underlying gene causing dominant white is PMEL17, this gene codes the formation of pigment in the pigment cells. A wild type coloured chicken has a normal functioning PMEL17 gene. A dominant white chicken has a mutated PMEL17 gene. The same is for the alleles of dominant white: dun colour and smoky (this colour is not present in the fancy). Dun colour and smoky have a different location of the mutation in the protein strain of which the PMEL17 gene consists. Smoky is a funny mutation because its capable to jeopardize the effect of the mutation causing dominant white plumage, it partly restores the formation of black pigment thus a percentage of black pigment is still possible to be given to the feather (ceratine producing cell). When heterozygous (impure I/i+), dominant white can only inhibit the production of black pigment (eumelanine). When homozygous (both parents had a mutated PMEL17 gene) red pigment is inhibited as well by the pigment cells, resulting is a chickens which is mostly white (but not totally, you see the small leaks as splashes).

There are three mutations of PMEL17 which all three have a different effect on pigment cells during the formation of pigment:
1. dominant white mutation: formation of black pigment is jeopardized hetero- and homozygous also red pigment won’t be made
2. dun colour mutation: red pigment is made, but black pigment in a different
way resulting in a cool dark brown shade when heterozygous and homozygous in a cool beige shade. 
3. smoky mutation (recessive to I, partly dominant to i+): red pigment is unaltered, smoky inhibits the effect of dominant white on black pigment resulting in a greyish shade (a kind of darker version of lavender).

More on this: The Dominant White, Dun and Smoky colour variants in chicken are associated with insertion/deletion polymorphisms in the PMEL17 gene

**Pigmentation and behaviour**

Pigmentation and behaviour have in several species a direct link to each other. In general can be stated that darker pigmented animals are more aggressive and show a more strong corticosteroid reaction compared to less or non-pigmented animals. Corticosteroid is a stress related hormone. It is probably not a total coincidence that the first signs of domestication has to do with the more docile behaviour of white animals and their handling of stress and therefore more easy adaption to life in captivity. In the 50s Belyaev did a test with foxes which were selected for tameness. After only a few generations the tame lines started to loose pigmentation. Coat colour also affects behaviour in domesticated mink (sables) and rats. In a lot of animals the difference in behaviour was observed depending on their coat or feather colour, and white is the result of domestication or the other way around, because white animals are more suitable to domesticate and therefore they were selected for this colour.

**Pleiotrophy or the by-effect of PMEL17**

The pleiotrophic effect of pea-comb (P) on the rest of the chicken you might know (Genetics of Chicken Extremes, Silkies and Silkie bantams). The amount of comb tissue is a lot less compared to other comb types, the chicken has a dewlap, the wattles are shrunken compared to p+ and there grow no feathers on the keel ridge of the breast. See also the article on www.chickencolours.com What wattles? about non bearded Silkies with a wrong comb (walnut) by which they don’t have wattles as in the SOP is asked for.

There were more people who found it convenient the industrial Leghorns were docile animals compared to other breeds (read: colours) although they were just as others easy victims of feather picking. Although its not very wild chicken like to be housed with thousands in a barn or cage with five without any privacy or room to exploit normal chicken behaviour, because wild chickens live in small groups consisting of birds of different age and a mean rooster as boss, the white layers seem not to bother much after thousands of years of domestication. So they differ a lot.

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A few scientists were curious about the How and Why of these friendly white Leghorns compared to their more heated coloured counterparts and they concluded it was due to the pleiotrophic effect of PMEL17, which causes the dominant white mutation and which is the reason why dominant white chickens are sweet and less aggressive. By the way, PMEL17 is analogous of the silver gene in mice (GP100). And because silver is already used as gene in chickens, the chicken version of mouse-silver is called PMEL17 and not silver.

**You don’t have to read this:** PMEL17 is a type 1 integral membrane protein which is present in the pigment cell and its a component of the fibrillar strips on which melanin is polymerized. It is in linkage group E22C19W28 in which are three genes located ERBB3, TUBAL1 and GLI. E22C19et cetera is homologous
to chromosome 10 of the mouse (silver) and 12 of humans (red hair). PMEL17 plays a crucial role in the normal development of black pigment (eumelanosomes). The proteolytic (=protein) splice (see wiki: proteolysis) and processing of PMEL17 guides the restructuring from early melanosomes from amorphous vesicles to elongated fibrous structures. When PMEL17 has mutated by kicking out some nucleotides from the protein chain or by shuffling them, the process of black pigment formation will be different or absent.

**The test birds**

For this behavioural research the scientists used F6 crosses from Red Jungle Fowl and white Leghorns. These F6 consisted of white (I/I) and wild type (I+/I+). The F6s were scored for two factors. The first factor was activity and social behaviour and the second factor was aggressive behaviour. The second factor tests were done with mature birds which also have hormonal reasons to act the way they act. PMEL17 did not affect hormone levels. Negative social experiences (like feather pecking) were avoided during growing up, they were housed individually immediately after hatching and could see and hear each other. They had large wire cages and shavings on the floor and unlimited access to feed and water. Their days were 12 up, 12 down. They came together in one room (test space) during the tests. It can be said that 53.2% of the variations in behaviour (more calm, less aggressive, more social behaviour) can be explained as pleiotrophic effect of PMEL17.

The test were done twice (chick test was lost), as growers and as adult chicken. The behavioural tests covered various aspects of social behaviour, exploration behaviour, fear, aggression tests, intruders tests (roosters) and the tendency to peck feathers, which were all scored. Also their weight was taken on different ages and blood tests were done for corticosterone and testosterone. Except the aggression tests and the immobility test (fear) there were no humans in the test rooms, and behaviour was registered by camera and the tape was later analysed.

A test example was the aggression test. A hand covered with cloth moved to and fro in the cage for 60 seconds according to a standardized movement. Behaviour of the rooster was recorded on video and the frequency of aggressive behaviour(s) scored afterwards from tape. Also differences in behaviour were noted: coming to the hand with hackle spread, only following the movements of the hand with the head, is the rooster coming to the hand till less than 20 centimetres or does it give one aggressive peck or did he jump on the hand. This all during the 60 seconds. Describing all tests is boring, check out the original paper. The outcomes of the aggression tests are in the table on the right.

There is a clear difference between the wild type and dominant white F6 birds. Behaviour of both groups can be split up in three factors of which respectively two could be interpreted from activity/explorative behaviour of the chicken, and the aggressive behaviour of the chicken. This means that individuals differ in personality traits. The score of the first factor suggests that chickens which scored high on explorative behaviour (the surroundings) were more social and less fearful. The second factor suggests that the more aggressive chickens also were more fearful.

There should be made a difference between offensive and defensive aggressive behaviour, which makes a lot of difference concerning the underlying factors. Defensive aggression is described as an attack to protect the mortal body and
has to do with fear. Defensive aggression was measured during the aggression test and wild type roosters showed more aggressive behaviour compared to white roosters. Offensive aggression is described as a reaction on a threat from supposed important resources (feed, territory, hens). This was tested during the intruder test. Differences in colour and behaviour were seen during the hand test, but not during the intruder test, by which can concluded that PMEL17 has merely influence on defensive aggressive behaviours. By the way, testosterone levels in wild type and white roosters were the same.

The white feather colour had obvious influence on behaviour. The supports earlier research which stated that PMEL17 had influence on social behaviour and explorative behaviour concluding PMEL17 has influence on personality traits.

Next to PMEL17, individual characters played a role; the personality traits of a chicken as variation within its colour group, but these were not of statistical importance.

More details, read the paper: Genotype on the Pigmentation Regulating PMEL17 Gene Affects Behaviour in Chickens Raised Without Physical Contact with Conspecifics

**Conclusion:** dominant white chickens are the sweetest, regardless breed, compared to coloured birds of the same breed.

![Graph](image)

*Fig. 3. The average factor scores (±SEM) on the first two factors extracted from the principal component analysis (PCA): (a) scores of wild type and white genotype on factor 1 and (b) scores of females and males of both genotypes on factor 2.*