What’s in a name? Nomenclature for colour aberrations in birds reviewed

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Received 4 February 2021; revised 15 June 2021; published 10 September 2021

http://zoobank.org:pub:C2148206-C801-4A2F-94AB-48000FF0F59C

Summary.—A review is presented of the seven commonest types of colour aberrations in birds together with suggestions for a standardised universal nomenclature to identify and distinguish these aberrations. These aberrations are: Leucism (congenital absence of melanin-producing cells), Progressive Greying (progressive loss of melanin-producing cells), Albino (total absence of melanin due to lack of the key enzyme), Brown (incompletely coloured melanin), Ino (even less completely coloured melanin), Dilution (altered deposition of melanin) and Melanism (altered distribution of melanin). It is proposed that these terms should be based not only on the resulting plumage but also should distinguish the underlying processes resulting in the aberrant pigmentation. By reviewing previously used terms for colour aberrations, and cross-referencing these with my proposed terminology, errors in earlier names are pointed out, and resulting in a more comprehensive nomenclature for colour aberrations found in wild birds.

‘There has been some confusion in the past in the recording and description of variant plumages … and this paper represents an attempt to clarify some parts of it.’ (Harrison 1963a)

Colour aberrations in birds have been my main research for more than 30 years, during which I have learnt that identifying and naming these aberrations still present problems for ornithologists. An important reason for this is probably that the appearance of similar heritable aberrations (mutations) may differ radically between species, and sexes and ages of the same species, all depending on the normal pigmentation. This, plus the fact that the individual might be too far away or moving too quickly to see well, often makes it difficult to distinguish the different aberrations from each other. The main reason for the difficulties, however, may be the unfamiliarity with the different forms in combination with the numerous terms used seemingly randomly for these colour aberrations.

When attempting to identify an aberration, it is important to know exactly how the original plumage colour of the relevant species should look! With that in mind, observe in what way the colour (pigmentation) is changed. However, having done that, what do we call it?

Knowledge of melanin pigmentation development and genetics has been gained mostly via studies of domesticated species such as mice and chickens. Genetic studies in mammals, especially mice, are probably the most thorough (Lamoreux et al. 2010, Eizirik & Trindade 2021). From an early stage, it became obvious that different mammal species had a similar series of heritable coat colour variants (Searle 1968). This marked the launch of scientific comparative genetics in mammals, and one result was that similar mutations were allocated the same name in all species. The similarity was based on the relevant gene action on the pigmentation process, and not necessarily on the appearance of the final coat colour, as this can differ between species. Establishing some uniformity in the nomenclature of mutations in birds, however, has so far been greatly neglected.
In fact, a standardised nomenclature for melanin mutations will cover almost all colour mutations in birds. For example, names for supposed carotenoid mutations, like Xanthochroism, Flavism and Erythrism, are often applied to birds that are afflicted by a melanin mutation. Any reduction of melanin results in the underlying carotenoids becoming clearer (Fig. 1). So, instead of an increase of carotenoid pigment, a predominantly yellow plumage is often the result of decreased melanin, making a term like Flavism an incorrect name for this aberration (© Pieter van den Hooven).

I have been working for many years (van Grouw 2006, 2010, 2013) to achieve a comprehensive nomenclature for colour aberrations in wild birds, as have others during the last almost 175 years (Table 1).
I use traditional terminology, mostly from the earlier period in genetic science, but try to define and clarify original meanings. I will highlight areas of disagreement with earlier terminology based on the resulting appearance by considering the underlying reasons for the aberrations. Different mutations with a similar phenotypic result were often lumped under a single name. Finally, some earlier terms, like ‘Leucism’, have become disassociated from their original meaning (white feathers), leading to confusion, with names randomly used to identify aberrant-coloured birds. The name, however, should not be based on the resulting plumage alone, as the underlying processes vary. It is important to bear in mind, however, that almost all aberrations can be identified only by their appearance (phenotype), as breeding tests and/or feather analyses are often impossible with wild birds. By considering the underlying processes, a more comprehensive nomenclature for colour aberrations in wild birds will be presented.

Where unreferenced statements concerning colour aberrations in birds (inheritance, pigmentation) are made herein, they are based on personal findings during unpublished research into this subject over the last 30 years, involving both practical breeding experiments with captive birds and examination of >4,000 aberrant-coloured specimens in museum collections.

**Melanin mutations**

Melanins are the most common pigments in birds and can be distinguished in two forms: eumelanin and phaeomelanin. Depending on concentration and distribution within the feather, eumelanin is responsible for black, grey and/or dark brown colours, whereas phaeomelanin is responsible for warm reddish brown to pale buff. Together, both melanins can produce a wide range of greyish-brown colours. Besides melanins, two other common pigments in birds are carotenoids and psittacins (the latter only in parrots). These pigments are collectively responsible for most yellow (and green), orange and red plumage. Birds cannot synthesise carotenoids themselves; these must be acquired via the diet. Parrots, however, can synthesise psittacine, so any yellow, orange and red/pink in these species is not diet related.

Due to two very popular bird species in captivity, mutations in carotenoids (Canary *Serinus canaria*) and psittacins (Budgerigar *Melopsittacus undulatus*) have been well studied. Known inheritable abnormalities in these pigments are: (1) total absence; (2) decreased concentration (Fig. 2); (3) change in type (colour)—normally from red to orange or yellow, but not vice versa; and (4) change in distribution, or Carotenism as it is sometimes termed, and often goes together with an increase in concentration (Fig. 3). In parrots...
it often occurs also in combination with an altered melanin distribution.

In Canaries all four forms are known (Perez-Beato 2008) although (3), the change in type (from yellow to orange / red), is due to past hybridisation with Red Siskin Spinus cucullatus (Lopes et al. 2016). Also among parrots (not only Budgerigar) all four forms occur (Martin 2002). The change in type (colour), however, is very rare and recorded in only a few species, whereas the others are more widespread throughout the different species. A total absence of psittacin in parrots is commonly known as Blue; a decreased concentration as Par-blue among breeders, and the change in distribution of psitacop, together with an increased concentration and an altered melanin pattern, is termed Opaline (Martin 2002). For carotenoids, categories 1–3 can also be diet-related. In parrots, categories 3–4 can also occur due to poor physical health. Compared to melanin mutations, carotenoid / psittacin mutations are rare. In this paper I therefore focus on melanin mutations.

Melanin is produced by specialised skin cells known as melanin cells or melanocytes, which develop from melanoblasts formed in the ‘neural crest’—the embryonic spinal cord. Normally, melanoblasts migrate at an early embryonic stage to the mesodermal layers of the skin. Finally incorporated in the skin and feather follicles, melanoblasts develop into melanin cells to provide the feather cells with melanin. The chemical process to produce melanin in the melanin cells is termed melanin synthesis and the final melanin pigment is deposited, via dendritic processes, into the growing feather cells (Crawford 1990). Heritable changes (mutations) in this process may produce aberrant-coloured plumage. In general, different melanin mutations can be divided into four major categories (Lamoreux et al. 2010): (1) defects in the development of melanin cells (White Spotting); (2) defects in melanin synthesis (Albinism); (3) defects in the melanin deposit into the feathers (Dilution); and (4) defects in the type of melanin produced (Melanism).

Lastly, the loss of pigment resulting in aberrant plumage can also be caused by external factors. The commonest is dietary imbalance, which normally shows as a mixture of normal pigmentation disrupted by its absence in individual feathers (van Grouw 2018). Other causes resulting in a lack of pigment in parts of the plumage are former injuries / traumas with melanin cell damage as an effect. These aberrations are not included in my proposed nomenclature (Table 2).

**Defects in the development of melanin cells (White Spotting)**

Two distinct groups of mutations fall into this category; those in which melanin cells are absent in the skin from the outset, and mutations in which the melanin cells progressively disappear, or become less productive. The first I term **Leucism**, from the Greek leukos (= white), and the second **Progressive Greying** (see below). In Leucism the lack of melanin is
Proposed nomenclature for the most common colour aberrations in birds.

| Colour aberration                        | Effect on melanins                                                                 | Effect on plumage and skin colour                                                                 |
|------------------------------------------|-----------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------|
| **Defects in the development of melanin cells (White Spotting)** |                                                                                  |                                                                                                   |
| Leucism                                  | Total absence of both melanin pigments in either parts or all of the plumage and skin due to a neural crest disorder resulting in the congenital absence of melanin cells from some or all of the skin areas. | All-white plumage all over or all-white feathers mixed with normal-coloured ones. Pink bill and feet or normal-coloured bill and feet depending on where in the skin the cells are missing. Always melanised eyes. In partial Leucism white feathers are often in patches and bilaterally symmetrical divided over the plumage. |
| Progressive greying                      | Total absence of both melanin pigments in either parts or all of the plumage (and skin) due to progressive loss of melanin cells in some or all of the skin areas with age. | All-white plumage all over or all-white feathers mixed with normal-coloured ones. Pink bill and feet or normal-coloured bill and feet. Always normal-coloured eyes. In an early stage white feathers are often randomly spread in the plumage. |
| **Defects in melanin synthesis (Albinism)** |                                                                                  |                                                                                                   |
| Albino                                   | Total absence lack of both melanins in feathers, eyes and skin due to the heritable absence of the enzyme tyrosinase in the pigment cells. | All-white plumage all over, red eyes and pink feet and bill.                                      |
| Brown                                    | Eumelanin changed in colour (qualitative reduction) due to incomplete melanin synthesis. Phaeomelanin unaffected. | Original black is brown, original reddish/yellowish brown unaffected. Plumages bleaches rapidly further in the (sun)light. Eyes normal and feet and bill slightly lighter than normal. |
| Ino                                       | Reduction of melanin and remaining melanin changed in colour (qualitative reduction) due to incomplete synthesis of both melanins. | Original black is very pale brown/cream to dark brown, original reddish/yellowish brown hardly visible to slightly paler. Eyes, feet and bill pinkish to hardly noticeably different. |
| **Defects in the melanin deposit into the feathers** |                                                                                  |                                                                                                   |
| Dilution                                  | Normal-coloured melanin is deposited in an abnormal clumped fashion in the feather cells (both melanins or eumelanin alone). | Original black is bluish- or silvery-grey. Original reddish/yellowish brown is buff/cream or unaffected. Eyes normal and in most forms of Dilution feet and bill are normal-coloured too. |
| **Defects in the type of melanin produced** |                                                                                  |                                                                                                   |
| Melanism                                  | Aberrant production and distribution into the feather cells of normal melanin (not necessarily an increase of pigment). | Increase of black and/or reddish brown, or an altered pattern (the latter not necessarily darker). Eyes, feet and bill normal. |

*Melanin aberrations do not affect carotenoid pigments which, if present in the relevant species, remains present.*

A result of the congenital and heritable absence of melanin cells from some or all of the skin where they would normally provide the growing feather with colour (carotenoid pigments, if present in the relevant species, remain). The extent of white can vary, from just a few feathers (partially leucistic, Fig. 4A) to all-white plumage (100% leucistic, Fig. 4B); the skin also lacks melanin for individuals in the latter category. Partially leucistic birds may have a normal-coloured bill and feet, depending on where the colourless patches occur, but all such birds have melanin-pigmented eyes. The embryonic origin of melanin pigments at the back of the eyeball is different from the rest of the body; eye pigments are formed mainly from the outer layer of the optic cup (Lamoreux et al. 2010) and, as leucism affects only the migration of melanocytes originating from the neural crest, it has no influence on eye pigmentation with an optic cup origin. So, in Leucism iris colour may be affected, the rest of the eye is normally melanised and therefore the pupils are black.
The white pattern in partially leucistic birds is often patchy and bilaterally symmetrical due to the way the melanoblasts migrated to the rest of the body in the early embryonic stage, leaving certain areas without melanin cells. The pattern can be caused by a delay in the migration of melanoblasts from the neural crest to the skin. Due to this, some melanoblasts reach certain parts of the body where the skin is too far developed to incorporate them, resulting in these parts lacking colour. Another possibility is that, from the outset, insufficient melanoblasts develop in the neural crest, and therefore not all parts of the body are provided with melanin cells. Whatever the cause, in the commonest forms of Leucism in birds the parts of the body furthest away from the neural crest are left without melanin cells, resulting in the face, the wingtips, the feet and the belly being affected (Fig. 4A). The white pattern in Leucism is static, i.e., it occurs already in juvenile plumage and the amount and pattern of white feathers does not change with age. A good example of a heritable form of Leucism established in a wild bird population was found in Common Ravens *Corvus corax* on the Faeroes (van Grouw 2014).

Leucism can be defined as a neural-crest disorder resulting in lack of melanin in all or parts of the plumage and skin. Another name used for Leucism is Piebaldism. As in certain animal species ‘piebald’ is the name of a specific white-spotting gene, I prefer Leucism, a term introduced by Rensch (1925) for all-white birds as a result of 100% Leucism (see also Figs. 4B, 28). Although Rensch defined it accurately; ‘an abnormal absence of melanin pigment resulting in white feathers but with normal coloured eyes’, the definition has taken a twist since 1925 into ‘an abnormal reduced concentration of melanin resulting in ‘washed-out’ colours’ (Hess 2011), resulting in the term Leucism nowadays being often incorrectly used for aberrations in the category Dilution. Also, Harrison (1985), at least, added to the confusion: ‘Partial loss of pigment, affecting all the colours present and reducing them in intensity, is rare. It is called ‘dilution’ by bird breeders and ‘leucism’ in scientific writing, although the latter term is also used at times for various form of schizochroic loss of single pigments which makes the plumage appear paler’.

Although Leucism is very common in domestic and captive birds, it is rather rare in the wild. Far more common causes for lack of pigmentation in feathers are aberrations causing a progressive disappearance of melanin cells, which in mammals is often called ‘Progressive Greying’. Whereas the result of Leucism is present at birth, Progressive Greying is a
condition that becomes visible after the bird reaches a certain age. This is, however, generally not related to ‘being old’ (geriatric greying); it can start at any time after the normally pigmented juvenile plumage is fully developed. From the onset of the condition, the bird gains an increasing number of white feathers with each moult (see Appendix 1). In the early stages, these are usually randomly spread over the bird (Fig. 5), and in certain cases (almost) the entire plumage can become white (Fig. 6).

Eurasian Jackdaws *Corvus monedula*, however, seem to be an exception, with the loss of pigment apparently related to old age (Fig. 7). In this case, the number of white feathers increases rather slowly compared to Progressive Greying in other species. In addition, the presence of some pigment in many of the affected feathers suggests a decreased activity of the melanin cells, rather than their disappearance altogether (geriatric greying in humans is likewise the result of decreased melanin cell activity). Further research is required to establish if older Jackdaws can indeed lose pigment due to their age.

The causes of many forms of Progressive Greying are still unknown. There are certainly straightforward heritable forms (van Grouw & Hume 2015) and a nice example is also found in the domestic Canary *Serinus canaria* (see Appendix 2). Pigment disorders such as vitiligo (pigment disease in humans) may also be responsible for some of the heritable forms of progressive loss of melanin cells, but for most forms in birds a straightforward genetic base appears to be lacking (van Grouw 2018).
Progressive Greying is a term used mainly in mammalian genetics for mutations that develop white hairs due to the progressive loss of melanin cells. Due to the progressive loss of melanin cells birds can develop feathers without melanin, making it sensible to use the same term for these aberrations in birds. Others have suggested Progressive Depigmentation, which is also appropriate.

Although the causes for different forms of Progressive Greying are not always clear, juvenile plumage is always normally pigmented, and the loss of melanin can start at any point after juvenile plumage is fully grown. Without knowing the bird’s history or breeding tests, it is often impossible to determine the true nature of the form of Progressive Greying in a wild bird, but Progressive Greying as a group of aberrations is certainly the commonest cause of white feathers in wild birds (van Grouw 2012, 2013, 2018).

Defects in melanin synthesis (Albinism)

The enzyme tyrosinase, naturally present in melanin cells, catalyses melanin synthesis, but due to inheritable causes (mutations) it can become absent or less active, with no or incomplete melanin synthesis as a result (Lamoreux et al. 2010). Although the usually black melanin granules can range from pale cream / beige-coloured to dark brown when synthesis is incomplete, in medical science any mutation affecting normal melanin synthesis is defined as ‘albinism’. There is, however, just one true Albino, all other mutations can be categorised as forms of albinism but they are not Albino.

The term ‘albino’ was first used by the Portuguese in the early 18th century for albino people among the blacks in Africa. In birds, Albino, from the Portuguese albo and Latin albus, meaning white, can be defined as a total lack of melanin in feathers, eyes and skin due to the hereditary absence of the enzyme tyrosinase in the pigment cells. The result is an all-white bird or, depending on the species, one coloured by carotenoids / psittacin alone (Fig. 8). The red or pinkish hue that can be seen in the eyes and skin is caused by blood vessels, visible through the colourless tissue (Fig. 9). Due to the absence of tyrosinase in an Albino, melanin cannot be produced, thus the concept of a ‘partial albino’ is false.

Albino birds are rarely seen in the wild, although the mutation is not uncommon and occurs quite frequently in most populations. The reason for the apparent scarcity is that the absence of melanin in the eyes makes them highly sensitive to light, with a poor depth of
vision. It is mainly their poor eyesight, rather than their white plumage, that makes albinos vulnerable, and most die soon after fledging (Fig. 10).

Another common mutation affecting melanin synthesis is called \textit{TYRP1b}, after the enzyme ‘tyrosinase-related protein 1’ which is involved in normal melanin synthesis (Lamoreux \textit{et al.} 2010). This mutation was formerly called ‘brown’ (hence the \textit{b} in the name) and is responsible for less active tyrosinase (Kobayashi \textit{et al.} 1998). Other names for this mutation in medical science are Rufous Albinism and Brown Albinism (Manga \textit{et al.} 1997). For our purpose \textbf{Brown} perfectly reflects its effect on the pigment: incomplete melanin synthesis causes the eumelanin to remain dark brown instead of becoming black (Fig. 11).
Phaeomelanin, where it is present in the relevant species, is unaffected (Fig. 12). Just after hatching, birds with the mutation Brown have plum-coloured eyes, but these are virtually indistinguishable from normal-coloured eyes by adulthood. Likewise, in Brown, the bill and feet are only slightly paler than in normal-coloured individuals.

Brown is the commonest but also probably the most misidentified heritable aberration in birds (van Grouw 2012, 2013). It is caused by a single recessive and sex-linked genetic mutation, which is identical in all bird species (but is not sex-linked in mammals), and therefore in the wild mainly females with this mutation are encountered. In records, Brown is known by many different names: albino, fawn, isabella, leucistic, schizochroistic, erythristic, cinnamon and pale morph are only the most common, but the same terms are also often used for mutations in the category Dilution. A possible reason for the difficulty in identifying this mutation in wild birds is that incompletely oxidised eumelanin caused by the mutation Brown is very light sensitive and bleaches rapidly in sunlight (Figs. 13–14). Within a couple of months, fresh, but aberrant, Brown plumage can become nearly white, making correct identification challenging.

Many other mutations affecting melanin synthesis are caused by variations of the gene \textit{SLC45A2}, which codes for the protein ‘solute carrier family 45 member 2’ in the melanin cells, and acts as a regulator in melanin synthesis. Although the precise function of \textit{SLC45A2} is unknown, it probably transports molecules necessary for normal melanin synthesis (Domyan et al. 2014). Several different mutations (alleles) of the \textit{SLC45A2} gene, which is located on the sex-chromosome in birds, are recorded in many different species. These mutations have different effects on the final melanin pigmentation; in some hardly any melanin is produced resulting in near-white plumage, whilst in others it is only slightly paler than normal. What all appear to have in common is that at least some of the melanin present is incompletely oxidised and therefore (much) paler than normal. Also, in all pale forms, the colour of the eyes and skin is to some degree also affected.

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Figure 12. Brown in Great Crested Grebe \textit{Podiceps cristatus}, Drachten, the Netherlands, July 2018. The mutation Brown affects eumelanin alone, not phaeomelanin, so the originally black (eumelanin) crest and fringes to the ruff are now brown, but the reddish-brown (phaeomelanin) flanks and in the ruff is unchanged. Contrary to popular belief, aberrant individuals often survive well and many also find a partner and breed (© Auke Terluin)

Figure 13. Brown in European Goldfinch \textit{Carduelis carduelis}, UK, January 2014; the bleaching effect of sunlight on aberrant brown eumelanin is already clearly visible (e.g., the tertials and primary tips). Melanin mutations do not affect the carotenoid pigments so the red and yellow are unchanged (© Graeme Conduit / British Trust for Ornithology)
The near-white form is often termed Sex-linked Imperfect Albinism (Figs. 15–16), due to its inheritance and because the plumage is nearly white. In medical science it is known as Oculocutaneous Albinism type 4 (Gunnarsson et al. 2007). For the darker (less pale) forms, many names are used, frequently based on those used in aviculture for that particular mutation in a certain species. One of these names, unfortunately used also in science, is...
Diluted (Domyan et al. 2014), which incorrectly suggests a mutation in the category Dilution (defects in the melanin deposited in the feathers). As plumage colours caused by SLC45A2 mutations bleach rapidly in sunlight, these birds often appear even paler than that caused by the mutation itself (Fig. 17).

I term all mutations of the SLC45A2 gene Ino, a name also used in European aviculture for the pale form (Sex-linked Imperfect Albinism) in many captive finches. Although Ino mutations (from the Greek or Latin Ine = ‘belonging to’ or ‘like’) can be categorised as a form of albinism, they are not Albino, and many are far from white. For the palest form, which is rather easy to recognise, the name Ino is sufficiently accurate. Darker forms can be termed Dark Ino as, without breeding tests, the actual mutation involved is impossible to establish.

In Ino the eyes are reddish due to the reduction of melanin, but the eyesight of an Ino bird, even in the palest forms, is much better than that of an Albino. Any adult wild bird with ‘white’ plumage and reddish eyes is probably Ino, not Albino (Fig. 18). As the...
inheritance of all Ino forms is recessive and sex-linked, mainly females will be found in the wild.

There are other mutations on other genes known to affect melanin synthesis and these often resemble Ino mutations in appearance (Fig. 19). As mentioned earlier, mutations in wild birds can be identified almost only by their appearance (phenotype) whilst their genotype is unknown. However, as these can be broadly categorised as ‘melanin synthesis-affecting mutations’, recording such birds as Ino is not wholly incorrect.

**Defects in the melanin deposit into feathers (Dilution)**

Many different genes and mutations are known in the category Dilution (from the Latin *dilutior* meaning ‘paler’ or ‘weaker’). One thing they have in common is that the aberrant colour is caused by an abnormal transport of the melanin pigment granules from...
the melanin cell into the feather cells. Instead of being uniformly deposited, the pigment granules clump together, resulting in blue-greyish tones where the original colour was black. So, the melanin pigment itself is normal-coloured but, due to the way it is distributed into the feather cells, we perceive it differently. This can be compared with a black-and-white photo in a newspaper; a high concentration of black ink dots close together are perceived as black, whilst fewer black dots in the same-sized area appear grey.

Although there are many different Dilution mutations, and each one dilutes colour in a slightly different way, such mutations are rare compared to those in the category Albinism. Dilution can be separated into two main forms. The commonest is a dilution of both eumelanin and phaeomelanin (Fig. 20). Black feathers turn greyish, and reddish or yellow-brown tracts become buff or cream-brown. The degree of dilution within a single mutation can vary individually, but most mutations cause a visible melanin reduction of c.50%. All birds with this form of dilution look like a pale, washed-out version of their normal counterparts, and can be termed Pastel (from the Latin *Pastellus*, a pale, delicate colour). The second form is a dilution of eumelanin alone, with phaeomelanin unaffected (Fig. 21). Black feathers turn grey, but reddish or yellow-brown ones remain unchanged. This form of Dilution can be called Isabel (from the Latin *isabellinus*; greyish yellow). In species with only eumelanin in their plumage it is impossible to distinguish a dilution mutation as being Isabel or Pastel (Figs. 22–23). In species with both melanins, e.g. sparrows, the phaeomelanin often seems to be even brighter in colour due to the reduction of the overlying eumelanin.

Dilution mutations affecting only the phaeomelanin are rare, and aberrant plumage with apparently diluted phaeomelanin alone is often caused by mutations in the Albinism category. As aberrantly coloured wild birds can be identified only by their phenotype, it must be taken for granted that misidentifications occur.

**Defects in the type of melanin produced (Melanism)**

**Melanism**, from the Greek *melanos* (= dark-coloured), is usually defined as an increased amount of dark pigmentation (melanin). A melanin cell is able to produce both melanins, but not simultaneously. It can, however, rapidly switch from producing one to the other.
Switching from eumelanin to phaeomelanin, and the reverse, is genetically determined. Via a mutation the melanin cell can be instructed to produce only one of the two pigments producing completely eumelanised or phaeomelanised plumage (van Grouw 2017). Timing of melanin production can also be affected and can change original plumage markings and patterns (van Grouw 2017). Melanism therefore does not necessarily imply an increase of dark pigment, but may be the result of a changed distribution in the same quantity of melanin. Consequently, a better definition of Melanism would be, a condition characterised by abnormal deposits of melanin in skin and feathers. ‘Abnormal deposit’ covers both changed distribution and an increased amount.

To summarise, the appearance of melanistic birds is mostly darker than normal, but not always. Melanism can affect birds’ plumage in three ways: (1) all of the plumage is darker and appears blackish (eumelanin), dark brown (both melanins) or reddish brown (phaeomelanin); (2) normally dark markings are bolder and noticeably ‘overrun’ their typical boundaries (the rest of the plumage is often somewhat darker as well (Figs. 24–25); and (3) the normal pattern and pigment distribution is changed, but the plumage is not necessarily darker (it can even be brighter).

Earlier nomenclature

Many names for the different colour aberrations have been proposed during the last 175 years. These names, however, were seemingly randomly used by others to identify aberrations in published records, creating much confusion.

Until the second half of the 20th century insufficient knowledge of feather pigmentation and possible aberrations resulted in the latter being mistakenly identified and named as species. With recent DNA work many of these ‘new species’ have been unmasked (e.g. Collinson et al. 2017, Kirschel et al. 2018, Schweizer et al. 2020, see also van Grouw 2010,
2017 for more examples). Hachisuka (1926), in his description of the melanistic Common Pheasant *Phasianus colchicus*, which he correctly recognised as an aberration (mutation), highlighted the problem and noted that such cases would always prove misleading unless a definitive nomenclature was developed. He proposed that all mutations should be distinguished by the term ‘mutation’, abbreviated as ‘mut’, within the scientific name. The melanistic Common Pheasant was therefore named by Hachisuka (1926) *Phasianus colchicus*
mut. \textit{tenebrosus} (\textit{tenebrosus} = dark), claiming that this was the first time that a mutant form had been correctly described. Although Hachisuka favoured naming mutations in scientific names, the nomenclature of mutations has never been regulated by the \textit{International code of zoological nomenclature} (ICZN 1999, and previous incarnations). However, even were the Code to accept this recommendation, the naming of the causative aberration would nevertheless have presented problems (see Fig. 26).

The earliest published attempt I could find to seriously categorise the different colour aberrations was by Frauenfeld (1853), who divided them into several groups, of which the most important were ‘true Albino’ (Leucochromatism from the Greek \textit{leukos} for ‘white’, and chromatism from the Greek \textit{khroma} for ‘colour’), ‘bleached colours’ (Chlorochromatism from the Greek \textit{khloros} for ‘green’ or ‘yellow’), ‘colour variety’ (Allochromatism from the Greek \textit{allokotos} for ‘unusual’; Melanism and Leucism were included here) and ‘discolouring with age’ (Geraiochromatism from the Greek \textit{geras} or \textit{girateia} for ‘old age’). Interestingly, the latter category shows that Frauenfeld had noticed Progressive Greying aberrations; a phenomenon that was to remain undocumented for another century (Fig. 27).

Subsequently, Frauenfeld (1873) revised his list of categories to include ‘blackish colours’ (Melanochromatism) and ‘reddish colours’ (Erythrochromatism), probably based on von Pelzeln (1865), who divided colour aberrations into two groups, Albinism and Melanism, based on a decrease or increase of pigment in the plumage. Within Albinism von Pelzeln (1865) distinguished ‘complete Albinism’ (all-white plumage with red eyes), ‘incomplete Albinism’ (overall plumage paler than normal but original patterns still visible) and ‘partial Albinism’ (parts of the plumage are white but the rest is normal). He made similar divisions within Melanism: ‘complete Melanism’ (all-black plumage), ‘incomplete Melanism’ (overall darker plumage but original patterns still visible) and ‘partial Melanism’ (only some parts of

Fig 26. Brown in Blue Tit \textit{Cyanistes caeruleus}, Wageningen, the Netherlands, May 2020. Even if Hachisuka’s (1926) recommendation for scientifically naming mutations had been accepted, there still would be the problem of which names to use for the different mutations; here \textit{C. caeruleus} mut. \textit{brunneus}, or \textit{C. c. mut. erythraeus}, or something else? (© Harvey van Diek)

Figure 27. Progressive Greying in Common Coot \textit{Fulica atra}, Leuten, the Netherlands, April 2018; the progressive loss of pigment, resulting in increasing numbers of white feathers, was first recognised by Frauenfeld (1853) but until recently largely ignored by ornithologists (© Erwin van Laar)
the plumage are black, whilst the rest is normal-coloured). Von Pelzeln (1865) distinguished a fourth category within Melanism, Erythrism (overall reddish-brown plumage).

P. Pavesi (in Picchi 1903), like von Pelzeln (1865), divided aberrations into two groups based on pigmentation decreases and increases, and used the terms ‘Hypochromatism’ (from the Greek _hupó_ for ‘under’) and ‘Hyperchromatism’ (from the Greek _hupér_ for ‘over’).

Rensch (1925), who proposed a set of terms to clear ‘the rather chaotic confusion in terminology for colour aberrations’ (‘geradezu chaotische Verwirrung in der Terminologie dieser Abweichungen’), also divided, following von Pelzeln and Pavesi, the different aberrations into two main categories based on the amount of pigment, and used the same terms—Hypochromatism and Hyperchromatism. In the first category, the decrease of pigment, he included (1) Albinism; comprised of albino (with red eyes), partial albino, and leucism (Fig. 28), (2) Schizochromatism and (3) Chlorochromatism. The second category, Hyperchromatism, for the increase of pigment included (1) Melanism, divided into eumelanism and phaeomelanism and (2) Lipochromatism. The term Schizochromatism (from the Greek _schizo_ = ‘split’, ‘separation’ or ‘division’) was introduced by Haecker (1908) for aberrations in which one pigment was absent while the other was unaffected. Haecker gave as an example two specimens of Black Woodpecker _Dryocopus martius_. One was fully white but still had red on the head, whilst the other was normally black-coloured but with white on the head where usually it is red. In the first bird the melanin was absent but carotenoid was still present (Fig. 29), and in the second bird these were reversed.

C. J. O. Harrison (1963a,b), a former curator at the Natural History Museum, London, distinguished two forms of Schizochromatism, which he called Schizochroism. Both Black Woodpeckers mentioned by Haecker (1908) are an example of the first of Harrison’s categories, which he termed melano-carotenoid schizochroism, the white bird being
non-melanic and the black bird with a white head patch lacking carotenoid. As melanin aberrations do not affect the carotenoid pigment, at least three different aberrations could have caused the appearance of the red-headed white woodpecker: Albino, Leucism and Ino. Although producing a similar appearance, the nature of these aberrations is totally different and they should not be grouped under the same name. Harrison’s (1963b) second category was ‘melanic schizochroism’ in which the two types of melanin are separated into non-eumelanic and non-phaeomelanic forms, which he termed Fawn and Grey variants, respectively. These ‘Fawn variants’, however, are not the result of separated melanins as will be demonstrated later in this paper.

Some aberrations, like true Albino (with red eyes) were identified correctly by earlier authors and all of them used the same term. Most of the suggested names, however, as already demonstrated above, also covered different aberrations with similar effects but of a totally different nature (see Table 1). Another example is Erythrism which, since von Pelzeln introduced it, was used by others for any aberration causing a colour more reddish or reddish brown than normal. The mutation Brown, certain forms of Dilution, Ino and Melanism, and an increase of red carotenoid, however, can all cause more reddish-looking plumage. Harrison (1963b) also had an opinion on erythrism, confusing matters further. According to him ‘erythristic plumage is one in which the normal eumelanin, and possibly phaeomelanin also, is replaced by a third chestnut-red melanin’. There is, however, no third melanin and, as shown below, Harrison was confused by the brown form of eumelanin, mistaking it for phaeomelanin. Based on the mutation Brown (see below) in Carrion Crows Corvus corone (which he called ‘Fawn’; the ‘non-eumelanic’ form of Schizochroism), Harrison (1963b) incorrectly opined that the plumage of crows Corvus contains both eumelanin and phaeomelanin. A corvid, however, lacking eumelanin will be white, and not brown, as they have no underlying phaeomelanin. The mutation Brown prevents eumelanin from being fully synthesised and so the plumage does not become black but remains brown (Fig. 11).

Two years later, Harrison (1965) was still convinced of the existence of an ‘unnamed chestnut-red melanin’ writing, ‘Irrespective of its biochemical relation with other melanins the chestnut-red melanin exists as a visible and well-defined entity and it seems preferable to have some term by which to refer to it. In the apparent absence of other names, I would suggest that it should be referred to as “Erythromelanin”, since it is the pigment usually present in the colour variants which we know as “erythristic” forms.’ Based on the crow example and others given by Harrison (1963b, 1965; from specimens at NHMUK) he indeed confused the brown form of eumelanin for phaeomelanin, and wrongly assumed that phaeomelanin was the ‘erythromelanin’.

Recently Davis (2007) published an overview of the many pigment abnormalities in birds and proposed a nomenclature in an attempt to finally establish uniformity. An important prerequisite was that terminology should reflect what happens to the pigments rather than the resulting plumage appearance. Davis therefore introduced what he believed to be a new terminology, to avoid confusion between historical and current interpretations, by incorporating a prefix. ‘Amelanism’ (prefix a- meaning ‘not’ or ‘without’) as a preposition for the absence of melanin, ‘hypomelanism’ for decreased pigment concentration, and ‘hypermelanism’ for increases. However, this was not new at all: as mentioned above, P. Pavesi (in Picchi 1903) had already used ‘Hypochromatism’ and ‘Hyperchromatism’ to divide aberrations with decreased or increased pigmentation, respectively, as had Rensch (1925).

**Conclusions**

The mutation Brown is probably the commonest heritable colour aberration in birds, but its importance, or even existence, has been overlooked. Depending on the author,
Brown has been included in Chlorochromatism, Schizochroism (Fawn) or Erythrism (terms also often used for mutations in the category Dilution, see Table 1). Davis (2007) referred to Brown as ‘Aeumelanism’ and stated, correctly, that it is inherited as a sex-linked, recessive trait. He defined it as ‘the abnormal absence of eumelanin from the plumage, skin, eyes, or all three areas’. In this mutation the number of eumelanin pigment granules is unchanged, but the pigment’s colour is altered due to incomplete synthesis. Because eumelanin is present, Aeumelanism is also an inappropriate term for this mutation. Davis (2007) further introduced the term Amelanism, partial (1) or total (2), which he defined as: the absence of all melanin from (1) parts of the plumage, skin, eyes, or all three areas [Leucism and Progressive Greying] and (2) from all plumage, eyes and skin [Albino]. The absence of all melanin results in white feathers (carotenoid pigments, if present in the relevant species, remain present) but, although the final appearance is broadly similar—white plumage—the nature and genetic background are different. To explore the different causes and occurrences of plumage irregularities correctly it is vital to distinguish aberrations by their causes, not their appearance (see Fig. 28).

A simple scheme to identify and name the commonest colour aberrations in birds is a must. Which names are used is less important provided they are universal, easy to use and cover the aberration appropriately. Many names proposed by earlier workers (e.g., Chlorochromatism, Schizochroism, Erythrism, Amelanism) lump multiple aberrations together, each with a separate cause and genetic background. Furthermore, names like Aeumelanism and Schizochroism are inappropriate for the aberration they refer to (Brown), as they incorrectly suggest an absence of eumelanin. As stated, it is vital to distinguish

Figure 30. Mutation affecting melanin synthesis in Barn Swallow Hirundo rustica, Steensel, the Netherlands, June 2013; as eumelanin is not developed (synthesised) these parts of the plumage are white, but phaeomelanin is normally developed. This may or may not be a mutation genetically related to Brown, but without breeding tests this will remain unknown. Like this mutation, many others can affect coloration, but all are far rarer in wild birds than those described herein, and therefore not covered by the presented nomenclature (© Theo van de Mortel)
aberrations by their causes, not their appearance, which may give erroneous indications as to the real occurrence of certain aberrations. Although the names I use perhaps do not explain the cause, they effectively distinguish the differing genetic backgrounds. My list is not exhaustive, but it covers the mutations most likely to be encountered by birdwatchers (Table 2). Other mutations affecting bird coloration are far rarer in wild birds (Fig. 30) and therefore not covered herein.

The rather simple names I use for common melanin mutations, based mainly on traditional names used in earlier genetic work appear to function well, but may be refined further. The aim here is to aid progress towards an international, usable nomenclature for colour abnormalities in wild birds that distinguishes, as far as possible, the nature and causes of each abnormality. Only then can we document the occurrence and frequency of different colour irregularities effectively. However, where an identification is uncertain, it is preferable for observers not to attempt to name the aberration in a publication, but just carefully describe and, if possible, photograph the bird, thereby placing the information on record, but preventing any misinterpretation as the result of an incorrect name.

Acknowledgements

I thank Till Töpfer and an anonymous reviewer for their constructive suggestions that improved the submitted manuscript considerably. As an article about colour would be seriously impaired without colour photographs to support the text, I am very grateful to the following for kindly supplying material: Pieter van de Hooven, Alois van Mingeroet, Jaap Denee, Rob Belterman, Bert Bruggeman, Piet Broekhof, Erwin van Laar, Bart van Beijeren, Harvey van Diek, Wim Wiijering, Theo van de Mortel, Auke Terluin, Annelies Marijnis, Jon Håkansson, Lennart Hjalmarsson, Hans-Peter Sahrhage, Stefan Schopper, Jann Hansen, Johan Bink, Billy Cannybud, Mike Yip, Steve Young, Glen Roberts, Paul Davies and Graeme Conduit / British Trust for Ornithology.

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Appendix 1: Progressive Greying in progress

Frauenfeld (1853) had already noticed that normally coloured birds can lose the pigment in their plumage later in life, in a phenomenon unappreciated by many later workers, and the majority of birdwatchers appear unaware of it, despite further evidence becoming available from ringers. In the late 1950s, Band (1956) reported that a ‘normal’ adult male Common Blackbird trapped in Lancashire on 24 November 1950 was retrapped on 4 December 1955 with ‘considerable patches of white over most of its plumage’. In response to Band’s observation, several other ringers also reported cases of ‘albinism related to age’ (Foott 1956, Spencer 1956, Hess 2011, Kirschel, A. N. G., Nwankwo, E. C. & Gonzalez, J. C. T. 2018). Investigation of the status of the enigmatic White-chested Tinkerbird *Pogonias makawai* using molecular analysis of the type specimen. *Ibis* 160: 673–680.

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Appendix 2: Progressive Greying can result in Fancy patterns

An iconic breed of Canary *Serinus canaria* is the London Fancy, distinguished by its absence of melanin in the body feathers, resulting in yellow plumage due to the presence of carotenoid alone, but fully melanised flight and tail feathers (Fig. 32). The breed disappeared in the early 1900s and breeders ever since have unsuccessfully tried to recreate the London Fancy, because the pattern was not caused by Leucism (a mutation common in the species) but by Progressive Greying. London Fancy canaries are fully melanin-pigmented when juvenile, but loss of melanin cells starts almost immediately after the bird has developed this plumage, so after its first incomplete moult the new feathers lack melanin. Canaries, like most passerines, do not moult their flight and tail feathers in their first year, so the typical pattern of the London Fancy was the result of adult feathers (without melanin) and the still present juvenile feathers (with melanin). This pattern lasts just c.8 months until the first complete moult. Afterwards a London Fancy will be (almost) all yellow. Canary breeders in 1800 knew this, but their counterparts in the 20th century did not. Recreating the breed was impossible without the particular mutation for progressive melanin cell loss, which disappeared with the London Fancy from canary aviculture. Recently, however, the mutation spontaneously re-occurred in a stud of canaries in the Netherlands, and together with the mutation the London Fancy is back. The mutation can occur in any species and is recorded in several wild birds (Figs. 33–34).

Figure 31. Progressive Greying in Common Chaffinch *Fringilla coelebs* in four successive years, Jonsered, Sweden (A) 25 March 2018, (B) 11 April 2019, (C) 16 April 2020, and (D) 31 March 2021; after 2020 the number of white feathers hardly increased (© Jann Hansen)
Figure 32. London Fancy Canary; the typical pattern of dark flight and tail feathers (melanin) and yellow body plumage (no melanin) in this distinctive breed of the domestic Canary *Serinus canaria* is the result of a heritable form of Progressive Greying in which the loss of melanin cells starts shortly after the first, fully melanised juvenile plumage is developed. The flight and tail feathers are still juvenile, whilst the rest of the plumage is adult following the first incomplete moult (© Alois van Mingeroet).

Figure 33. Heritable Progressive Greying in a first-winter Eurasian Siskin *Spinus spinus*, Liverpool, England, January 2016; loss of melanin caused by similar Progressive Greying mutation as the London Fancy Canary (© Steve Young).

Figure 34. Heritable Progressive Greying in a first-winter Blue Tit *Cyanistes caeruleus*, Schaffhausen, Switzerland, January 2019; loss of melanin caused by similar Progressive Greying mutation as the London Fancy Canary (© Stefan Schopper).