

Color Abnormalities

The expression “a white blackbird” is hardly so paradoxical as it might seem; and indicates as well established a fact as that “blackberries are red when they are green.”

— Elliott Coues (1868)

Blackbirds are especially prone to abnormal coloration (Sage 1962), but how we classify such birds is a subject of much disagreement, confusion, and misunderstanding. If “a white blackbird” were entirely white, but its eyes and skin were normally pigmented, would it be albinistic, leucistic, or something else? Ask a birder, aviculturist, and ornithologist, and you might get three different answers. That’s not to say all birders would give one answer, while aviculturists and ornithologists would give others.

In fact, birders, aviculturists, and ornithologists alike are confused and disagree about how these and other aberrant coloration terms should be used. I began researching these terms after years of being confused myself. What I found was a nomenclature muddier than the one for molts and plumages prior to the system proposed by Humphrey and Parkes (1959). And the mess is neither confined to the study of birds nor to North America. I discovered examples of conflicting terminology in the study of all other vertebrate groups and in other parts of the world as well.

There are four major confounding factors: (1) many terms used for birds were originally proposed for mammals, which display fewer pigment types; (2) many terms were coined before we knew much about the mechanisms behind the conditions they describe; (3) because color abnormalities occur unpredictably in the wild, they are difficult to study, so we still know little about them; and (4) general misuse and misinformation. Take the debate over definitions of the term *partial albinism*, for example. Although the term has been used at least since the mid-1800s (Coues 1868) for the abnormal absence of pigmentation from parts of the plumage, many insist that albinism is an all-or-nothing condition, that a bird can be no more partially albinistic than a mammal can be partially pregnant (Buckley 1982). Yet the rationale for this restricted usage has never been satisfactorily explained. It may be that only the condition which results in the complete absence of pigment should be called albinism. Looking to human pathology, for which the term was coined, I

Jeff N. Davis

H. T. Harvey & Associates
423 West Fallbrook Avenue
Suite 202
Fresno, California 93711
jdavis@harveyecology.com

in Birds

A Proposed Nomenclature for Birders

found that it is applied more broadly in that discipline. Several forms of human albinism were described early in the 20th century (Pearson et al. 1911–1913)—which, by the way, Pettingill (1956) adapted for birds—and today many types are widely recognized, most involving retention of some pigmentation (Spritz 1994). Regardless of whether albinism should be broadly or narrowly defined, it does not account for more than one pigment type and is therefore inadequate for color anomalies in birds.

The history of the term *leucism* is just as convoluted and controversial. It was first applied exclusively to the particular condition that results in an all-white plumage or pelage and normally pigmented eyes and skin (Rensch 1925). Several decades later it was used for the abnormal condition that results in the dilution of plumage pigments (Harrison 1964). And more recently it has been applied to everything from an entirely white plumage to a single white feather (Buckley 1982). Harrison's usage appears to be the most widely recognized today, but many still favor Buckley's definition. No matter which definition you prefer, however, the term *leucism* falls short in its usefulness for classifying color abnormalities in birds because it does not account for loss of pigment in unfeathered areas.

In addition to the lack of consistency in use of abnormal color terms, another problem stands out: The traditional terms describe

The effects of carotenism vary widely. In this **Northern Cardinal** it has resulted in the total loss of carotenoids from parts of the plumage. *Texas Hill Country, Texas; January 2005. © John Westbrook.*





Pigmentation

Understanding the chemical and physical elements responsible for coloration in birds is essential for making sense of abnormal color terminology. Natural colors in feathers arise from the deposition of pigments, the development of keratin nanostructures, or both during feather elaboration, usually during molt (Lucas and Stettenheim 1972). Other colors are acquired through soiling, staining, sun bleaching, or abrasion, mostly adventitiously but sometimes deliberately. Most colors, though, result from the combination of pigments or pigments and feather structures. The coloration of unfeathered areas may change on shorter timescales.

Melanins

Melanins are the most prevalent pigments in birds. They produce almost all of the black, brown, gray, rufous, and buff shades and patterns, and they come in two general types—eumelanins and phaeomelanins—although a third type, erythromelanins, has been hypothesized in birds on purely genetic grounds (Harrison 1965). Eumelanins are more common and predominate in black and brown feathers, whereas phaeomelanins are less common and predominate in chestnut and rufous feathers. All melanin-containing feathers that have been analyzed contain both eumelanins and phaeomelanins, although one may make up only a tiny percentage of the total melanin content (McGraw et al. 2004, McGraw 2006). The relative proportion of these two pigment types, therefore, determines most colors. The head color of the male Brown-headed Cowbird, for instance, results from a mix of eumelanins and phaeomelanins, whereas the black breast bands of the Killdeer are pigmented primarily by eumelanins, and the ruddy throat of the Barn Swallow owes its color mainly to phaeomelanins (McGraw et al. 2004, McGraw 2006).

Carotenoids

The next most common bird pigments are carotenoids, which are responsible for most of the yellows, oranges, and reds. Carotenoids are deposited into single, contiguous patches, never exhibiting the complex patterns such as mottling or barring that are common with melanins. Birds cannot synthesize carotenoids. Instead, they obtain them from their diet, either by directly consuming carotenoid-containing algae or plants or by ingesting prey such as insects, crustaceans, or fish that harbor carotenoids that they themselves obtained from their diet. Carotenoids are often deposited in plumage or unfeathered areas without being

This totally amelanistic **Cliff Swallow** was associating with several normally colored juveniles. Because of its impaired visual acuity, this bird likely perished soon after it was photographed. Its eye was pink in life but appears dark in this photograph. *Sierra Valley, California; June 2006. © Tom Grey.*

what pigments remain, not those that are affected by the mutation or change. Shouldn't the terminology reflect what happened to the pigments rather than the appearance of the resulting plumage? Let's look at erythrism and xanthochroism, for example. These terms refer to abnormal red and yellow coloration, respectively, but they tell us little about the origin of those colors. Erythrism, for instance, may result from the under- or over-expression of one or more types of pigment. Using the traditional terminology, erythrism can arise from schizochroism, leucism, dilution, complete albinism, partial albinism, incomplete albinism, imperfect albinism, melanism, or carotenism. Confused yet?

A transparent scheme in which the terms tell us the history of the pigments and thus the source of the aberration would be less confusing and a lot more informative. Most of the traditional terms are inadequate for such a scheme, however, because they are vague, don't account for multiple pigment types, and have no stable meaning. Therefore, because of their ambiguity, limitations, and instability, I recommend abandoning them in favor of more precise terms. I hope that by offering simple definitions for unambiguous terms I might unify birders by creating an agreeable and consistent lexicon. If some still prefer different terminology, then I urge them to provide explicit definitions.

Even with unambiguous terminology, however, the exact condition may be hard or impossible to identify in the field. This is because different abnormalities can produce similar effects, more than one abnormality can occur in the same individual, and it is often impossible to know exactly what has happened to the pigments in a wild bird.



Partial amelanism in this **Steller's Jay** is due to some environmentally induced cause. This bird lost most of the melanin in its plumage, bill, legs, and feet over the course of a single molt. Although blue is a structural color, it relies on the presence of melanin, so the blue also disappeared. *Boulder County, Colorado; September 2004. © Bill Schmoker.*

altered chemically. The yellow feathers of the Yellow-breasted Chat, for example, result from the deposition of unaltered dietary lutein (McGraw 2006). Yet in other species, such as Northern Cardinal and American Goldfinch, ingested carotenoids are modified, producing divergent colors (McGraw et al. 2001).

Melanins and carotenoids are not the only types of pigments to bestow these colors on birds, however. Among the numerous uncommon to rare pigments in birds, porphyrins, for example, contribute to the brown and rufous hues in the feathers of owls, goatsuckers, and gallinaceous birds; psittacofulvins color the red, orange, and yellow

plumage of parrots (With 1978, Stradi et al. 2001); and pterins are responsible for the red, orange, and yellow irises of pigeons, owls, starlings, and blackbirds (McGraw et al. 2004). In addition, the keratin microstructure of feathers, together with pigments, produces blues, greens, and iridescent colors (Lucas and Stettenheim 1972). With knowledge of the types and distribution of pigments in a given species, it is usually possible to predict the appearance of potential color abnormalities.

What is Abnormal?

Another factor that has led to some confusion is that two of

the traditional terms, melanism and erythrism, have been applied to normal coloration in polymorphic species. Dark Red-tailed Hawks, for example, have been called melanistic, and reddish Eastern Screech-Owls have been labeled erythristic. Although the mechanisms that result in these plumages may be the same as those that result in abnormal coloration, it is best to avoid these terms for the sake of clarity and instead call such plumages morphs or variants. The question, then, is where do we draw the line? I think we can all agree that a dark

Red-tailed Hawk is normal and a dark male Northern Harrier would be abnormal. Trickier perhaps are pink Ring-billed Gulls, estimated to make up 1–2% of the population in Washington state (Hardy 2003), dark Common Murres at about 0.1% in California (Sibley 2000), and so-called “green morph” Pine Siskins at about 1% in North America (McLaren et al. 1989). What we call those is open to debate, but I’d consider them abnormal and apply the appropriate terminology. As you can see, determining what is abnormal is a bit arbitrary. The important point is that, when evaluating an individual bird for color abnormalities, we need to be aware of differences in color due to such “normal” influences as polymorphism, geographic variation, age, feather wear, and lighting conditions.



The pied appearance of this **Northern Parula** is a common result of partial amelanism. Rector, Pennsylvania; October 2000. © Powdermill Avian Research Center.

Total amelanism is the absence of all melanin from the plumage, eyes, and skin.

In species that lack carotenoids and more uncommon pigments, the condition results in an all-white plumage, pink eyes, and light, often pink, skin. The unfeathered areas usually appear pink due to the normally pigmented hemoglobin within the blood vessels near the surface of the skin. The condition is caused by a genetic mutation that blocks the production or activity of tyrosinase, a key enzyme in the synthesis of melanins. More than 50 mutations of the tyrosinase gene have been found in humans (Spritz 1994), but the number in birds is unknown. Because it is recessive, total amelanism appears only when a bird inherits the mutated gene from both parents. Autosomal and sex-linked versions

are about equally frequent (Buckley 1982). Because tyrosinase has no influence on the formation of carotenoids or other pigments, the colors they produce will still be present. A male Pileated Woodpecker with this condition, for example, would have a white body, wings, and tail, and a red pileum and submoustachial stripe.

In wild populations the frequency of total amelanism is controlled by various external factors. Due to the absence of melanin in the eyes, birds with this condition are light-sensitive and have reduced visual acuity. As a result, they more readily fall victim to predators, traffic, and other hazards, and may have difficulty locating food. Their all-white plumage also may make them more conspicuous to predators. Most probably die soon after independence at fledg-

Proposed Terminology

Color abnormalities result from changes in the concentration or distribution of pigments that are normally present, changes in the types of pigments themselves, or changes in the microstructure of feathers. The conditions described below include only the first two types. Although color abnormalities from changes in the microstructure of feathers are well known by some parrot breeders, they are unknown in wild birds.



Although partial amelanism most often affects only the plumage, it has mainly affected the bill, legs, and feet in this male **Anna's Hummingbird**. Note the retention of some melanin in the toenails and the abnormally white eye-ring. Nuevo, California; September 2002. © William Zittrich.

ing. Those that survive beyond independence may have difficulty establishing their social status if related plumage signals are lost and may have difficulty obtaining a mate if color patterns necessary for sexual display are absent.

Non-melanic plumage is also less structurally resilient than melanin-containing plumage (Bonser 1995), which could lead to reduced fitness, especially in birds such as woodpeckers in which the stability of certain feather groups—the rectrices in their case—are critical for foraging or other essential life functions. And finally, for most species, normal breeding dispersal over wide areas would reduce the probability of suitable matings required to perpetuate the mutant gene.

Total amelanism has been called albinism, total albinism, complete albinism, perfect albinism, pure albinism, true albinism, and all white.

Partial amelanism is the abnormal absence of all melanin from parts of the plumage, skin, eyes, or all three areas.

This is the most frequently observed color abnormality. Its expression can vary from a white spot on a single dark feather to completely white plumage. It can also affect unfeathered areas, though some melanin usually remains in the eyes (van Grouw 2006), distinguishing it in extreme cases from total amelanism. Carotenoid pigments, if present, will still be visible, as in yellow Evening Grosbeaks (Hudon 1997). The condition can result from a failure to deposit melanin (Hutt 1949) or



Even though the overall color of this hypomelanistic **Common Grackle** is diluted, the basic color pattern is still evident. In this case the concentration of both melanin types has been reduced. York, South Carolina; January 2006. © Bill Hilton, Jr.

possibly—if similar to related conditions in humans—from a diminished level of tyrosinase (Spritz 1994). There are both hereditary (genetic) and non-hereditary (environmental) forms. Hereditary forms can be recessive or dominant, sex-linked or autosomal (Enders and Post 1971). Environmental factors such as malnutrition, toxin ingestion, injury, disease, parasites, shock, and old age can induce non-hereditary forms of partial amelanism. Hormones may also play a role. In an early study of House Sparrows (Keck 1933), the black bill of breeding males faded to horn color when the birds were castrated, and the black coloration was restored when the birds were injected with testosterone. It



Although the overall appearance of this hypomelanistic **Gray Catbird** is washed out, the rufous undertail coverts, which are colored mainly by phaeomelanins, are retained. Rector, Pennsylvania; September 2001. © Powdermill Avian Research Center.





Aumelanism can reveal normally hidden carotenoids, as it has in this partially aumelanistic **Red-winged Blackbird**. Churchville, Pennsylvania; February 2005. © Adrian Binns.

is unknown to what extent hormones might regulate the expression of melanins in wild birds, however. In cases of hereditary partial amelanism, the white plumage pattern is consistent from one molt to the next, but in non-hereditary cases it is often reversible if the cause, e.g., poor nutrition, is corrected. Hereditary forms may persist in a population for decades. Partially amelanistic Seaside Sparrows and sharp-tailed sparrows, for example, have been present in the Long Island area since the 1890s (Enders and Post 1971).

Although partial amelanism has been reported in numerous families of birds, it tends to be more frequent in some than in others, more common in social or communally breeding birds such as blackbirds and House Sparrows, more common in small isolated populations than large continuous populations, and more prevalent in cities and smaller towns than in rural areas (Sage 1962, Bensch et al. 2000).

Because partially amelanistic individuals usually have melanin in their eyes, they are not hampered by reduced eyesight. Depending on the extent of amelanism, however, they may suffer from some of the same effects as totally amelanistic individuals. Environmentally induced forms suggest other problems, such as malnutrition and disease, which might reduce survival and reproduction. The partially amelanistic birds in one study experienced a similar lifetime reproductive success as normally colored birds



Eumelanin is retained only in the eyes of this partially aumelanistic **Common Redpoll**. Note that the red carotenoids of the crown are unaffected. Near Wasilla, Alaska; March 2005. © Ruth Ann and Doug Lloyd.

lution is variable, ranging from slightly pale to nearly white plumage, but distinct patterns such as tail bands and eye lines are usually retained. Wear and sun bleaching may



Only phaeomelanin remains in the plumage of this partially aumelanistic **Ruby-throated Hummingbird**, but its eyes and bill are still black, indicating retention of eumelanin in those areas. Apex, North Carolina; August 2005. © Susan Campbell.



Eumelanin remains only in the eyes of this partially aumelanistic **Western Sandpiper**. The bill, legs, and feet are colored with phaeomelanin, as is much of the plumage, although faintly so. Carmel, California; September 2003. © Gary Nielsen.

(Bensch et al. 2000). In another study, however, the condition was thought to be associated with a reduced probability of survival (Ellegren et al. 1997).

Partial amelanism has been called albinism, partial albinism, semi-albinism, incomplete albinism, leucism, schizochroism, piebaldism, white spotting, abnormal white, and Pealea phenomenon, and individuals with this condition also have been dubbed pied, splashed, and variegated.

Hypomelanism is the abnormal reduction of melanin concentration from the plumage, skin, eyes, or all three areas.

This condition produces a faded appearance. The extent of pigment di-

lution is variable, ranging from slightly pale to nearly white plumage, but distinct patterns such as tail bands and eye lines are usually retained. Wear and sun bleaching may cause further whitening. Although this condition often affects only the plumage, it can affect unfeathered areas, as was the case for a Western Meadowlark in Arkansas (Hanebrink 1971). Hypomelanism can be total or partial, hereditary or non-hereditary, recessive or dominant, and can affect one or both types of melanin. If it is possible to distinguish the exact nature of pigment dilution, the appropriate modifiers may be applied: total hypomelanism, partial hypomelanism, hypoeumelanism, and so forth. Carotenoid and other non-melanin pigments, if present, will be unaffected.

The effects of this condition on survival and reproduction probably vary with the species involved and the type and extent of the condition. The hereditary form has been

shown in chickens to be associated with reduced early growth and slow utilization of yolk sac contents (Santos and Silversides 1996), factors that may affect survival. In Antarctic penguins, however, there was no evidence of reduced reproductive fitness (Forrest and Naveen 2000), and hypomelanism has persisted in a population of Black-billed Magpies in Edmonton, Alberta since at least the 1940s (Hudon 1995).

Hypomelanism has been termed leucism, albinism, partial albinism, imperfect albinism, dilute albinism, dilution, dilutism, ghosting, paling, isabellism and isabellinism—odd choices since isabelle apparently is a fairly dark color—and chlorism and chlorochroism—also odd since *chloro-* is the Greek root word for green. Individuals with the condition also have been called faded, frosted, and bleached. Hypomelanism may be indistinguishable in the field from the following two conditions.

Aeumelanism is the abnormal absence of eumelanin from the plumage, skin, eyes, or all three areas.

This condition results in a pale plumage in which the melanic areas are usually some shade of fawn, brown, or rufous. Areas of the plumage or individual feathers that have little or no phaeomelanin will be white or nearly so. This abnormality is inherited as a sex-linked, recessive trait. Because it is sex-linked it occurs more often in females, which in birds are the heterogametic sex, than in males. If other non-melanic pigments are normally present, they will be unaffected. In some cases loss of eumelanin reveals a more extensive distribution of red or yellow, which is normally masked by eumelanin. As with most of the other abnormalities, the effects of aeumelanism on survival and reproduction probably vary, and I found no specific information on this aspect in the literature.

Aeumelanism has been called schizochroism, schizochromatism, schizomelanism, imperfect albinism, and isabellism. Individuals with this condition have also been labeled buff, fawn, and cinnamon varieties.

Aphaeomelanism is the abnormal absence of phaeomelanin from the plumage, skin, eyes, or all three areas.

This condition results in a pale plumage in which the melanic areas are usually some shade of black or gray, often ash-gray. It is inherited as an autosomal recessive trait, so it is as likely in males as in females. It is much less frequent than aeumelanism. If other non-melanic pigments are normally present they will be unaffected.

Aphaeomelanism has been known as schizochroism, schizochromatism, schizomelanism, imperfect albinism, and isabellism, and individuals with this condition have also been referred to as gray or silver varieties.



This partially hypereumelanistic **Swamp Sparrow** is sporting an abnormally dark face. Long Point Bird Observatory, Ontario; May 2006. © Stu Mackenzie.



The concentration of both melanin types in the plumage of this partially hypermelanistic **Tufted Titmouse** is abnormally high. Somers, New York; November 2005. © Debbie and Ken Field.

Hypermelanism is an abnormally high melanin concentration in the plumage, skin, eyes, or all three areas.

This condition, which is less common than amelanism and hypomelanism, results in a darker than normal plumage. The unfeathered areas may or may not be affected, and the plumage may be totally or partially affected. The condition can involve the increase in concentration of only one melanin type. When it affects eumelanin, resulting in a blackish color, it can be called hypereumelanism. Likewise, when it affects phaeomelanin, resulting in reddish-brown color, it can be termed hyperphaeomelanism. There are hereditary and non-hereditary forms. Hereditary forms are usually autosomal and can be dominant or recessive (McGraw 2006). As with most other color abnormalities, the effects of hypermelanism on survival and reproduction vary with the species and extent of the condition. Non-hereditary forms, which can indicate malnutrition or some other pathology, may negatively affect survival. But hereditary hypermelanism can become more widespread in an isolated population—even to the degree that it replaces the normal coloration—if there is an advantage to the darker color (Sage 1962). This has occurred in the Bananaquit on some islands in the West Indies. The hypereumelanistic population, best referred to as the “black morph” because it is no longer “abnormal”, occupies a predominantly wet habitat, whereas the yellow population inhabits dry deciduous areas. Because bacteria degrade non-melanin feathers more rapidly than melanin-containing feathers, and because

there are more bacteria in wet than in dry habitats, the black morph might have a selective advantage in the wet areas (Burt and Ichida 2004). So, in this case, bacterial degradation of non-melanin feathers might explain the selective advantage of the black morph and hence retention of the mutant gene.

Hypermelanism has been known as melanism and nigristism.

Carotenism is an abnormality of carotenoid pigmentation that results from one or more of four causes: (1) change in the normal distribution or extent of carotenoid pigments; (2) increase or decrease in carotenoid concentration, resulting in a change in color or color intensity; (3) change in carotenoid pigment type and therefore a change in color; and (4) total absence of carotenoids from all or part of the plumage or skin.

This abnormality occurs only in species that normally display carotenoids in their plumage or unfeathered areas. Its expression varies extensively. It accounts for white flamingos, Western Gulls with yellow legs, Ruby-crowned Kinglets with yellow crowns, Cedar Waxwings with orange tails, orange Scarlet Tanagers, and yellow Northern Cardinals (McGraw et al. 2003). Carotenism often results from dietary factors during or just prior to molt, although it also can be caused by a genetic mutation that disrupts carotenoid metabolism (McGraw et al. 2003). The best-known case of this abnormality is that of flamingos, which turn white in captivity if a source of carotenoids is not

added to their diet. The effects of carotenism on survivorship and reproduction vary for reasons outlined previously for other abnormalities.

Carotenism has been called xanthism, xanthochroism, flavism, erythrism, cyanism, albinism, and partial albinism, and individuals with this condition have been called lutinos.

Other Color Abnormalities

Color abnormalities may also result, of course, from changes in other less common pigment types. In those cases, I suggest applying the appropriate



This carotenistic **Western Gull**, with abnormal orange rather than normal pink legs, was first reported as an out-of-range Yellow-footed Gull. *Doheny Beach State Park, California; December 2005. © Travis Cooper.*



This carotenistic **Prairie Warbler** has an aberrant orange rather than normal yellow face. *Dry Tortugas National Park, Florida; April 2005. © Larry Manfredi.*

modifiers where possible, e.g., total aporphyrinism, hyperpterinism, etc. In addition, color abnormalities may be due to hybridization; gynandromorphism, which in dimorphic species results in an individual that displays both male and female characteristics (often on either side of the midline); androgynism, which results in a female that displays male plumage or vice versa; and atavism, which results in an individual that displays an ancestral plumage character. Those conditions, however, are, mostly for space limitations, beyond the scope of this article.

Identification Pitfalls

Color abnormalities routinely cause identification problems. Totally amelanistic Dovekies and Black Guillemots were once described as distinct species (Coues 1868), partially amelanistic House Sparrows have been called Snow Buntings, carotenistic Red-bellied Woodpeckers—with yellow instead of red on the head—are sometimes mistaken for Golden-fronted Woodpeckers (Gerber 1986), and the list goes on. I'm sure most of us are familiar with similar cases. The important point is that we should be aware of

the effects of color abnormalities, especially when identifying possible vagrants.

What We Don't Know

There is still a lot to learn about the causes, manifestations, heritability, and effects of these abnormalities. Many aviculturists probably know more than has been reported in the ornithology and birding literature, and they should be encouraged to publish their observations. As there also is much to learn simply by studying photographs or reading descriptions of abnormally colored birds, I recommend reporting such birds whenever they are encountered, in the print literature, if possible, but also on birding e-mail lists and websites. Documenting the distribution, effects, and frequency of color abnormalities could also have conservation implications if any of these conditions arise from exposure to environmental contaminants.



I believe the simple scheme proposed above includes the

most common color abnormalities and defines them in ways that should permit widespread usage. Although the proposed terms may seem unnecessarily jargonistic, I found no plain language alternatives that are up to the job. As a final point of illustration, if I were to say that I saw a total albino male Northern Cardinal, most would think of an all-white bird. But, in fact, because "albinism" does not affect carotenoids, this bird would be red with white only around its bill. In contrast with the familiar but fuzzy term *albinism*, *amelanism* applied to this case gets right to the point: This is a bird that lacks melanin.

Acknowledgments

I am indebted to Jocelyn Hudon for his thorough review of a draft of this article; Joseph Morlan for invaluable discussions; and René Corado, Linnea Hall, Tony Hertz, Jocelyn Hudon, and Dan Singer for providing hard-to-find references.

Literature Cited

- Bensch, S., B. Hansson, D. Hasselquist, and B. Nielsen. 2000. Partial albinism in a semi-isolated population of Great Reed Warblers. *Hereditas* 133:167–170.
- Bonsler, R.H.C. 1995. Melanin and the abrasion resistance of feathers. *Condor* 97:590–591.
- Buckley, P.A. 1982. Avian genetics, pp. 21–110 in: M. Petrak, ed. *Diseases of Cage and Aviary Birds*, second edition. Lea and Febiger, Philadelphia.
- Burt, E.H., and J.M. Ichida. 2004. Gloger's rule, feather-degrading bacteria, and color variation among Song Sparrows. *Condor* 106:681–686.
- Coues, E. 1868. Instances of albinism among our birds. *American Naturalist* 2:161–162.
- Ellegren, H., G. Lindgren, C.R. Primmer, and A.P. Møller. 1997. Fitness loss and germline mutations in Barn Swallows breeding in Chernobyl. *Nature* 389:593–596.
- Enders, F., and W. Post. 1971. White-spotting in the genus *Ammospiza* and other grassland sparrows. *Bird-Banding* 42:210–219.
- Forrest, S.C., and R. Naveen. 2000. Prevalence of leucism in pygocelid penguins of the Antarctic Peninsula. *Waterbirds* 23:283–285.
- Gerber, D.T. 1986. Female Golden-fronted Woodpecker or mutant female Red-bellied Woodpecker? *American Birds* 40:203–204.
- Hanebrink, E.L. 1971. Dilute albinism in a Western Meadowlark collected in Mississippi County, Arkansas. *The Migrant* 42:82.
- Hardy, L. 2003. The peculiar puzzle of the pink Ring-billed Gulls. *Birding* 35:498–504.
- Harrison, C.J.O. 1965. The chestnut-red melanin in schizochroic plumages. *Ibis* 107:106–108.
- Harrison, J.M. 1964. Plumage: Abnormal and aberrant, pp. 643–646 in: A.L. Thomson, ed. *A New Dictionary of Birds*. McGraw-Hill Book Company, New York.
- Hudon, J. 1995. Edmonton and its imperfect albino black-billed magpies. *Edmonton Naturalist* 23:21–23.
- Hudon, J. 1997. Non-melanic schizochroism in Alberta Evening Grosbeaks, *Coccothraustes vespertinus*. *Canadian Field-Naturalist* 111:652–654.
- Humphrey, P.S., and K.C. Parkes. 1959. An approach to the study of molts and plumages. *Auk* 76:1–31.
- Hutt, F.B. 1949. *Genetics of the Fowl: The Classic Guide to Poultry Breeding and Chicken Genetics*. Norton Creek Press, Blodgett.
- Keck, W.N. 1933. Control of the bill color of the English Sparrow by injection of male hormone. *Proceedings of the Society for Experimental Biology and Medicine* 67:315–347.
- Lucas, A.M., and P.R. Stettenheim. 1972. *Avian Anatomy: Integument, Part 2*. Agriculture Handbook 362. U.S. Department of Agriculture, Washington.
- McGraw, K.J. 2006. Mechanics of melanin-based coloration, pp. 243–294 in: G.E. Hill and K.J. McGraw, eds. *Bird Coloration, Vol. 1: Mechanisms and Measurements*. Harvard University Press, Cambridge.
- McGraw, K.J., G.E. Hill, and R.S. Parker. 2003. Carotenoid pigments in a mutant cardinal: Implications for the genetic and enzymatic control mechanisms of carotenoid metabolism in birds. *Condor* 105:587–592.
- McGraw, K.J., G.E. Hill, R. Stradi, and R.S. Parker. 2001. The influence of carotenoid acquisition and utilization on the maintenance of species-typical plumage pigmentation in male American Goldfinches (*Carduelis tristis*) and Northern Cardinals (*Cardinalis cardinalis*). *Physiological and Biochemical Zoology* 74:843–852.
- McGraw, K.J., K. Wakamatsu, S. Ito, P.M. Nolan, P. Jouventin, F.S. Dobson, R.E. Austic, R.J. Safran, L.M. Siefferman, G.E. Hill, and R.S. Parker. 2004. You can't judge a pigment by its color: Carotenoid and melanin content of yellow and brown feathers in swallows, bluebirds, penguins, and domestic chickens. *Condor* 106:390–395.
- McLaren, I.A., J. Morlan, P.W. Smith, M. Gosselin, and S.F. Bailey. 1989. Eurasian Siskins in North America: Distinguishing females from green-morph Pine Siskins. *American Birds* 43:1268–1274.
- Pearson, K.D., E. Nettleship, and C.H. Usher. 1911–1913. *A Monograph on Albinism in Man*. Draper's Co. Research Memoirs, Biometric Series 6, 8, and 9, London.
- Pettingill, O.S. 1956. *A Laboratory and Field Manual of Ornithology*, third edition. Burgess Publishing Company, Minneapolis.
- Rensch, B. 1925. Die Farbabweichungen der Vögel. *Journal für Ornithologie* 73:514–539.
- Sage, B.L. 1962. Albinism and melanism in birds. *British Birds* 55:201–225.
- Santos, G.A., and F.G. Silversides. 1996. Utilization of the sex-linked gene for imperfect albinism (S* ALS). 2. Yolk sac lipid utilization. *Poultry Science* 75:1455–1462.
- Sibley, D.A. 2000. *The Sibley Guide to Birds*. Knopf, New York.
- Spritz, R.A. 1994. Molecular genetics of oculocutaneous albinism. *Human Molecular Genetics* 3:1469–1475.
- Stradi, R., E. Pini, and G. Celentano. 2001. The chemical structure of the pigments in *Ara macao* plumage. *Comparative Biochemistry and Physiology B* 130:57–63.
- van Grouw, H. 2006. Not every white bird is an albino: Sense and nonsense about colour aberrations in birds. *Dutch Birding* 28:79–89.
- With, T.K. 1978. On porphyrins in feathers of owls and bustards. *International Journal of Biochemistry* 9:893–895.