Albinism In Florida Green Turtle (*Chelonia mydas*) Hatchlings: Ratio-Based Evidence Of Basic Mendelian Recessiveness

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On 21 July 2017, a nesting green turtle (*Chelonia mydas*) measuring 97.5 cm in curved carapace length laid a clutch of eggs in Juno Beach, Florida USA. This turtle was fitted with two inconel flipper tags on the forelimbs (left: EEN126, right: EEN128) and a passive integrated transponder (PIT) tag in the right front flipper (989001001238560). On 3 September, her clutch of 98 hatchlings emerged from the nest that was being monitored for a study on maternal transfer of cheloniid herpesvirus 5 (ChHV5). To our surprise, 23 albino turtles made up a portion of those 98 hatchlings (Fig. 1). Upon excavation of the clutch, we observed seven unhatched eggs, three of which contained albino embryos, and ten dead pipped eggs/hatchlings, three of which were albino; 25% of the clutch was phenotypically albino:

$$\frac{\text{23 live albinos + 3 dead albino embryos + 2 dead pipped albinos}}{\text{98 hatched eggs + 7 unhatched eggs + 10 dead pipped eggs}} =$$

$$\frac{\text{29 albinos}}{\text{115 total eggs}} = 25.2\%$$

With sea turtles, it is not uncommon to find one or two hatchlings with pigmentation anomalies from a single clutch (Pritchard 1979). Finding numerous hatchlings with pigment abnormalities is much rarer. The number of albino hatchlings produced from this nest nearly mirrors a nest of loggerhead (*Caretta caretta*) hatchlings from 1994 in Brazil that produced 22 albino hatchlings out of 98 total hatchlings (Marcovaldi *et al.* 1995). The Brazilian loggerhead nest had a hatching success of 87.5% and an incubation period of 52 days. The green turtle nest from our study incubated for 45 days, with a hatching success of 85.2%, which is higher, in comparison, than the average hatching success of 27 other nests from the ChHV5 viral transfer study (mean ± SD = 66.0% ± 22.6%), suggesting that albinism, in this case did not impact developmental success.

In birds and snakes, albinism is an autosomal recessive trait (Sage 1962; Bechtel & Bechtel 1989) that when inherited results in enzymatic deficiencies of melanin metabolism during development (Erickson & Kaefer 2015). A similar genetic pattern has been

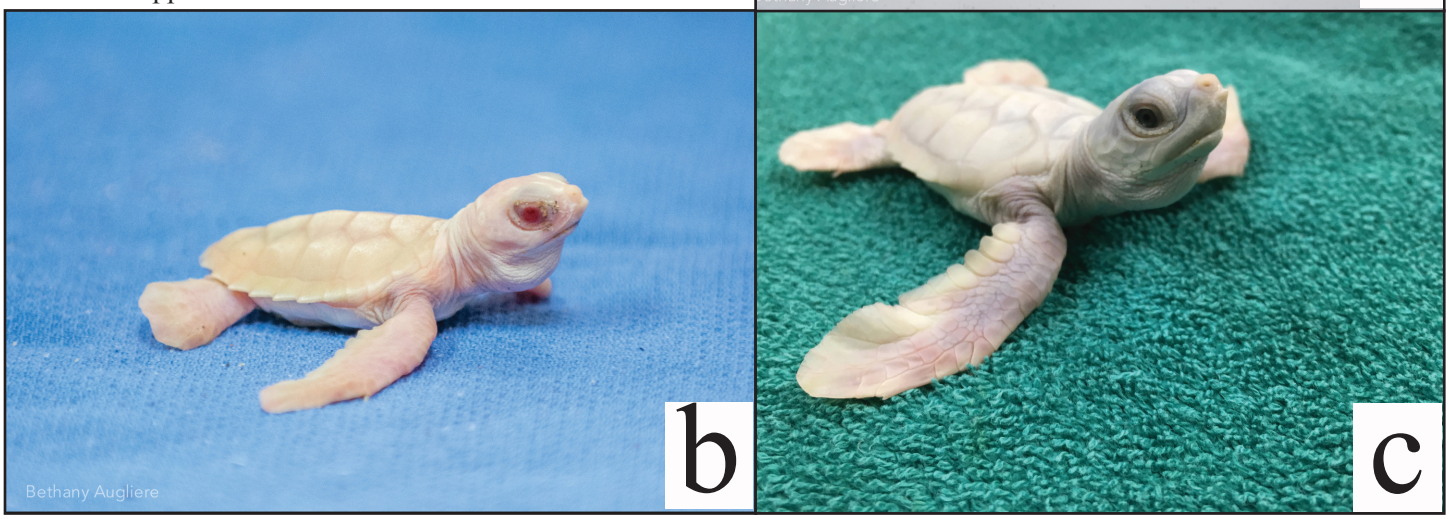

Figure 1. (a) Albino hatchlings from the same clutch; two normally pigmented hatchlings were included for comparison. Extra vertebral and lateral scutes (*i.e.*, scute abnormalities) are indicated by the black arrowheads. Photo credit: Bethany Augliere. (b) Albino hatchling green turtle. The red eyes indicate a lack of ocular melanin pigment, allowing capillary blood to be seen through the retina. Photo credit: Bethany Augliere. (c) For comparison, a leucistic hatchling green turtle from a separate clutch from 2017. This individual was the only abnormally pigmented hatchling in its clutch. Leucistic animals lack melanin and may lack other pigments in the skin, but have pigmented eyes. Photo credit: Christina Coppenrath.

	PMother	pMother
PFather	PFPM (25%): normal pigment	PFpM (25%): normal pigment, albinism carrier
pFather	pFPM (25%): normal pigment, albinism carrier	pFpM (25%): albinism

Figure 2. Hypothetical single trait Punnett square representing a possible scenario from this clutch of green turtle eggs. Based on the 3:1 ratio of normally pigmented hatchlings to albino hatchlings, this clutch most likely had a single father. "P" and "p" represent the dominant and recessive alleles for presence and absence of pigment, respectively.

predicted in sea turtles (Godfrey & Mrosovsky 1995); however, this has not been confirmed. Recently, corn snakes (*Pantherophis gutattus*) were used as model species to investigate the mutation resulting in amelanism in reptiles. This study revealed that mutation of the OCA2 gene is responsible for the pigment disorder (Saenko *et al.* 2015). Based on the ratio of albino hatchlings to total eggs in the green turtle clutch from this study, it is likely that albinism in sea turtles is a result of basic Mendelian recessiveness as indicated by a Punnett square for a single trait (Fig. 2), with 25% of the offspring carrying both autosomal recessive copies of the allele leading to the phenotypic expression of the lack of pigment. Additionally, based on the near perfect ratio of 3:1 for normally pigmented to albino hatchlings, it is likely that only one father contributed to this clutch (despite known occurrences of multiple paternity in green turtles: Ireland *et al.* 2003). Perhaps the mother and father were related, as both would have to be carriers of this rare allele. Our interpretation is hypothetical at this time and support or lack of support will depend upon genetic analyses. It also has been suggested that environmental influences including stress and habitat quality can impact the occurrence of this disorder (McCardle 2012).

Albinism has been observed in all vertebrate groups (cyclostomes: Jensen 1959; elasmobranchs: Sandoval-CaStillo *et al*. 2006; osteichthyes: Manoel *et al.* 2017; amphibians: López & Ghirardi 2011; reptiles: Clay 1935; Bechtel & Bechtel 1981; Kar & Bustard 1982; Spadola & Di Toro 2007; birds: Sage 1962; mammals: Uieda 2000), including all seven sea turtle species (Bustard & Limpus 1969; Whitmore & Dutton 1985; Hewavisenthi 1990; Godfrey & Mrosovsky 1995; Marcovaldi *et al*. 1995; Johnson *et al.* 1999; Türkozan & Durmuş 2001; Hitchins & Bourquin 2005; Sönmez & Özdilek 2011). Generally, albinism in marine turtles is rare, with less than 1% of embryos/hatchlings expressing this condition (Fowler 1979; Kaska & Downie 1999; Hitchins & Bourquin, 2005). Observations of albinism in larger life stages are extremely rare (Fletemeyer 1977; Sönmez & Özdilek 2011), which indicates little to no chance of survival in sea turtles with abnormal pigmentation. Understandably, this trait is not selected for in the wild as albino sea turtles likely suffer higher mortality rates due to the inability to camouflage (Erickson & Kaefer 2015), as well as anatomical abnormalities (*e.g.*, cleft palate, scute anomalies; Kaska & Downie 1999, Turkozan & Durmus 2001; Hitchins & Bourquin, 2005; Sönmez & Özdilek 2011; Fig. 1), disease susceptibility (Hayley-McCardle 2012), and potential effects on seafinding behavior due to abnormal retinal pathways to the brain (Sage 1962; Guillery 1986; Godrey & Mrosovsky 1995). However, an amelanistic loggerhead was observed nesting in Queensland, Australia in 1977 & 1978 (Limpus *et al*. 1979). We were unable to test the seafinding behavior of the hatchlings documented here due to lack of permits and the impending Hurricane Irma; however, this will be studied in any future encounters with albino hatchlings.

For follow up studies, blood and morphometric measurements were collected from every hatchling from this nest in an effort to determine paternity, health and morphological differences between hatchlings with and without albinism, and the gene responsible for this disorder. Those results will be published as part of a larger study on the health impacts of albinism in green turtle hatchlings.

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