DARK BROWN EGGS
EXPLAINED

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Dark brown egg inheritance compared with an orchestra

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White Leghorn hens laying white eggs. Rhode Island Reds laying dark brown eggs. The difference comes down to one crucial gene and the precise timing of its activity. This gene is autosomal (= not sex linked).

The lead instrument: ALAS1

The ALAS1 gene is the conductor's baton. It controls the opening movement of a complex biochemical piece: the synthesis of protoporphyrin IX (Pp IX), the pigment that colours brown eggs. In White Leghorns, the ALAS1 conductor barely raises the baton, actually just not. The gene is expressed at very low levels. Little pigment is made. The eggs stay pale white. In dark brown egg laying Rhode Island Reds, ALAS1 is expressed at high levels. The conductor is vigorous and commanding driving up the volume to 6 (where Marans are 11). Pp IX synthesis begins in earnest!

The orchestra assembles: timing matters

The synthesis happens in a specific location: the shell gland, the organ that calcifies the eggshell. The timing is critical. At 4 hours after oviposition (when the previous egg was laid), the next egg hasn't entered the shell gland yet. No calcification has begun. Pp IX levels are low across all hen types. At 16 hours, rapid calcification is underway. This is when the orchestra reaches its crescendo. In dark brown egg layers, Pp IX concentrations in the shell gland peak at around 85 nanomoles per gram. In White Leghorns however, they remain at roughly 24 nanomoles per gram > a three-fold difference. The dark brown egg laying hens are producing pigment at a much higher rate. At 22 hours, calcification is nearly complete. Pigment deposition is at its maximum. The brown eggs are already darkening visibly.

The supporting sections: transport genes

Three additional genes act as the supporting orchestral sections, moving the pigment where it needs to go:

- **SLC25A38:** transports glycine into mitochondria, feeding the initial synthesis reaction
- **ABCG2:** exports protoporphyrin IX out of the mitochondria into the cell cytoplasm
- **FLVCR1:** helps export pigment out of the cell entirely, into the uterine fluid where it will coat the eggshell.

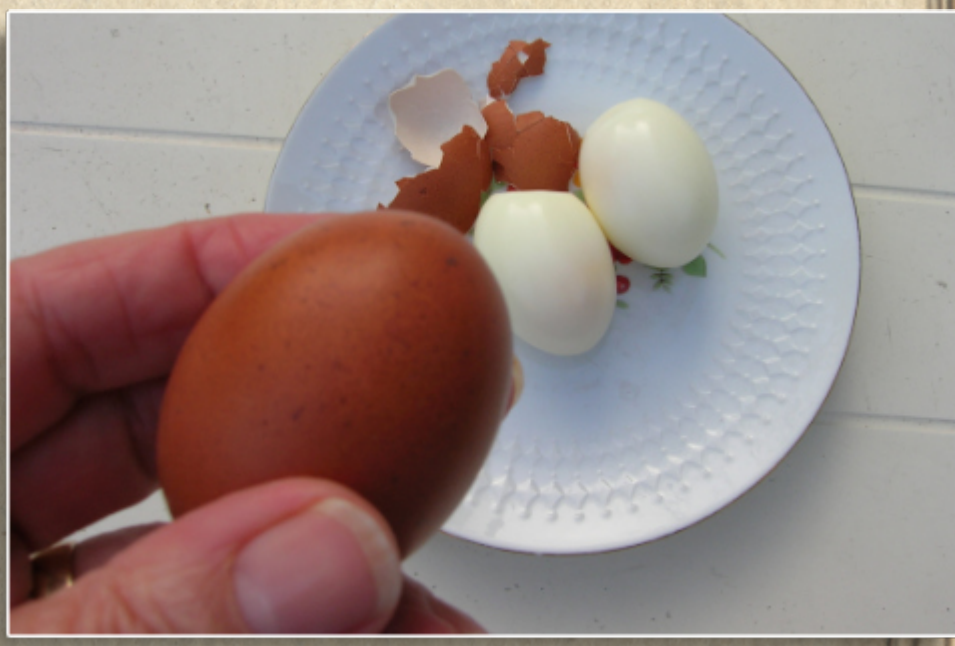

Photo: Marans bantam eggs. Photo: Elly Vogelaar (NL)

Each plays its part. High expression of ALAS1 means nothing if the transporters cannot move the pigment efficiently. In the dark brown egg laying hens, these supporting genes are also elevated, ensuring the pigment flows through the system.

The rhythm section: metabolic signals
Prostaglandins (chemical messengers) appear to orchestrate the final release. In dark brown egg laying hens at 22 hours, specific prostaglandins (15-dehydro-prostaglandin E1 and prostaglandin G2 2-glyceryl ester) are upregulated in the uterine fluid. These molecules seem to trigger the final surge of pigment deposition onto the shell surface, creating the dark brown colour that becomes visible.

The genetic score: silent mutations with effects
The researchers identified seven genetic variants (SNPs) in ALAS1 that associated with egg brownness. All were ‘silent’ (they didn't change the amino acid sequence). Yet they still mattered because they altered how quickly the gene's instructions were translated into protein. Subtle changes in codon usage can affect translation speed, protein folding, and ultimately, protein function. The orchestra's musicians can play the same notes, but at different tempos, creating different overall effects.

The final performance
In White Leghorns: the conductor is absent, the orchestra is quiet, little pigment is made. Pale white eggs are the result.
In the dark brown egg laying Rhode Island Reds: the conductor (ALAS1) is engaged, the supporting sections (transporters) work efficiently, the rhythm section (prostaglandines) provides timing cues, and the genetic variants ensure optimal translation.
The result is a coordinated synthesis and deposition of pigment onto a calcifying shell, producing the characteristic dark brown egg, imo the visible inheritance from Marans bloodlines.
Both are functional eggs. The difference is purely in gene expression levels and the coordinated activity of multiple genes at precise developmental stages.

Sex-linkage in brown egg colour: A claim awaiting evidence
The inheritance of dark brown egg colour in chickens is frequently described by breeders as sex-linked, yet this assertion lacks support in published genetic research. Controlled studies examining brown egg genetics—including recent transcriptomic and SNP analyses—consistently identify autosomal genes (particularly ALAS1 on chromosome 12) as the primary regulators of egg pigmentation.
The sex-linkage claim may arise from sex-specific expression patterns, linkage with other traits or observational bias in small-scale breeding programmes.
However, it remains unproven without formal genetic pedigree analysis and segregation data.
Until controlled crosses demonstrate sex-linked inheritance patterns, autosomal control of this trait is favoured by the evidence.

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Research paper: 2022, **Identification of crucial genes and metabolites regulating the eggshell brownness in chicken.** https://link.springer.com/article/10.1186/s12864-022-08987-7?getft_integrator=wiley