Dilute Down Lethal: A New Lethal Mutation in Japanese Quail

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Abstract: “Dilute down lethal (DDL)” is a new mutation of Japanese quail (*Coturnix japonica*). Neonatal plumage of the DDL mutant is the same as the wild type in pattern, but its coloration is slightly lighter than that of the wild type. In addition to the down color abnormality, some DDL chicks have bent digits. All DDL chicks die within three days of age. Genetic analysis revealed that the DDL mutation is controlled by an autosomal recessive gene. The proposed gene symbol is *ddl*.

Key words: Japanese quail, lethality, mutation

In September 1994, we found an unusual chick showing dilution of plumage color and morphological abnormality in the digits, among the progeny from a pair of wild-type Japanese quail (*Coturnix japonica*) maintained at our laboratory. The same pair, thereafter, continued to produce similarly abnormal progeny. All abnormal chicks died within 3 days of age. The abnormality was conjectured to be a recessive mutation with complete lethality. In Japanese quail, there are several mutations that have the same primary plumage pattern as the wild type but show dilution in color [2]. The present trait seemed to be different from all of them. We therefore named the trait “dilute down lethal (DDL)”

General care of quail is described elsewhere [7]. We separated the male and female of the original pair that produced the first abnormal chicks, and mated them to wild-type controls to obtain F₁ birds. Subsequently, we mated the F₁ birds among themselves avoiding sib matings and selected carrier matings by a progeny test. In these proven carrier matings, we examined the seg-

regation ratio of normal and abnormal offspring, and analyzed the ratio by chi-square test. We also investigated the morphology of the abnormal chicks in comparison with the wild type. For the observation of the skeleton, we stained the bone and cartilage with alizarin red S and alcian blue 8GX (Wako Pure Chemical Industries, Ltd., Osaka, Japan), respectively, based on the methods described by Tsudzuki and Wakasugi [8] for the bone and by Watson [10] for the cartilage.

Morphology: Neonatal plumage of the DDL chick was slightly lighter than the wild type in color, although it had the same striped pattern as the wild type (Fig. 1). The brownish stripes of the wild type were replaced by slightly lighter brownish or yellowish colored stripes in the mutant chick. In the wings and legs of the mutant, blurry black markings replaced the dense black markings of the wild type. In addition to the down color abnormality, some DDL chicks (25%, 15/60) had bent digits (Fig. 1, arrow). In spite of this abnormal external appearance, the phalanges themselves were

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Table 1. Incidence of the dilute down lethal mutant in carrier matings

No. of eggs set	No. of eggs fertile	No. of dead embryos ^{a)}			No. of chicks hatched		Expected ratio ^{b)}	χ^2	P
		1-10 d.	11-18 d.		NOR	DDL			
			NOR	DDL			NOR : DDL		
312	306	15	14	11	206	60	3 : 1	0.056	.90>P>.80

^{a)}d=days of incubation, NOR=normal, DDL=dilute down lethal mutant. ^{b)}Based on simple autosomal recessive inheritance.

Fig. 1. Dorsal view of wild-type (left) and mutant (right) chicks. The mutant down color is slightly lighter than the wild type. An arrow shows a bent digit.

morphologically normal.

Mode of inheritance: Table 1 shows the segregation ratio of normal and DDL individuals in the progeny of carrier matings. Of 291 progeny, 220 were normals and 71 were DDLs. The sex ratio was normal in both normal and DDL individuals. This segregation ratio was in good agreement with the expected 3:1 ratio ($\chi^2=0.056$, $.90>P>.80$) based on the hypothesis that the DDL trait is controlled by an autosomal recessive gene. We propose the symbol *ddl* for the mutant gene controlling the DDL trait.

Of the 220 wild-type progeny, 14 (6%) died at the late embryonic stage, whereas among the 71 DDL progeny 11 (15%) died at the same stage. That is, the mortality of the DDL mutant is significantly higher at the late embryonic stage than the wild type ($\chi^2=5.697$, $.025>P>.010$). Besides, all of the 60 DDL chicks that hatched died within 3 days of age. It is therefore concluded that the *ddl* gene has a completely lethal effect.

The cause of the lethality is not yet known. In macroscopic anatomy, there were no abnormalities in the brain or viscera of the DDL mutant. Moreover, the DDL mutant showed no specific clinical signs before death.

In Japanese quail, approximately 10 recessive mutants are accompanied by lethality [2], the majority of which are chondrodystrophic embryonic lethal mutants with abnormally shaped beaks and/or short shanks [1, 3-6, 9]. However, in the DDL mutant, the beaks were of normal shape, and the legs of normal length.

The DDL mutant is similar to the light down lethal (LDL) mutant [7] in having a slightly lighter down color than the wild type and showing lethality at the chick stage, but the two are distinguishable by the following three features: (1) the down color of the LDL is more reddish than that of the DDL; (2) the LDL has a normal body shape, but the DDL sometimes shows abnormality in the digits; and (3) lethality occurs within three days of age in the DDL, but within three weeks in the LDL. There is no mutation in chickens and turkeys that is similar to the DDL mutation of Japanese quail.

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