

# Initial levels of maternally derived antibodies predict persistence time in offspring circulation

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**Abstract** Vertebrate mothers transmit antibodies to offspring that provide humoral immunity early in life. The duration of protection provided by maternal antibodies varies considerably among species and has not been widely examined in birds. Determination of the length of maternal protection can be a useful predictor of when young are most likely to be susceptible to infection. The duration of maternal antibody protection was determined in Japanese Quail (*Coturnix japonica*) by immunizing females with keyhole limpet hemocyanin (KLH) and then collecting blood samples from offspring. Maternal antibodies remained detectable in offspring circulation for an average of 14 days (range 3–28). The duration of persistence was predicted by antibody levels as measured in maternal circulation, within egg yolks, or measured in offspring shortly after hatch. Thus, the primary benefit to offspring of high concentrations of maternal antibodies is likely to be an extended period of maternal protection during early growth and development.

**Keywords** Antibody persistence · Ecological immunology · Immunocompetence · Maternal antibody transmission · Maternal effects · Passive immunity

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## Introduction

Maternal antibody transmission provides the primary form of humoral immune defense for young vertebrates (Brambell 1970; Grindstaff et al. 2003). Antibody concentration and diversity are generally correlated between mothers and their offspring early in life (Graczyk et al. 1994; Bollen and Hau 1999; Gasparini et al. 2002; Grindstaff 2008). The concentration and diversity of antibodies transmitted may consequently influence the disease resistance and survival probability of offspring (Smith et al. 1994; Sahin et al. 2003; Al-Natour et al. 2004). Maternal antibodies are rapidly catabolized from offspring circulation after birth or hatch (Brambell 1970; Grindstaff et al. 2003). Passively transmitted antibodies are cleared at an exponential rate from offspring circulation (Nicoara et al. 1999). Offspring with higher initial levels of maternal antibodies generally retain maternal immunity longer than offspring with low initial levels of maternal antibodies (Smith et al. 1994). Young generally lose maternal protection before mature adult levels of humoral immunity are generated endogenously (Solomon 1971; Klasing and Leshchinsky 1999). Thus, offspring may be most susceptible to infectious diseases during this period after maternal antibodies are catabolized and before their own immune systems have fully differentiated (Hasselquist and Nilsson 2009). If development of active immunity is independent of the loss of maternal antibodies, offspring with low initial maternal antibody levels may be at an increased risk of infection for a longer period of time than offspring with higher initial maternal antibody levels.

In order to predict when juveniles will be most susceptible to disease, it is important to know both how long maternal antibodies remain in offspring circulation and when active immunity of offspring develops. To determine

the persistence period of maternal antibodies in Japanese Quail (*Coturnix japonica*), I immunized adult females with the novel antigen keyhole limpet hemocyanin (KLH) and collected blood samples to measure decay of KLH-specific antibodies in offspring circulation.

## Methods

### Study species

Thirty pairs of adult Japanese Quail were obtained from a randomly bred control line at Purdue University. All birds were the same age. They were maintained on the same photoperiod (16L:8D) and fed the same diet (quail layer crumbs; Wayne Animal Nutrition) for the duration of the experiment. Adults were housed in a quail battery breeder (Georgia Quail Farms) with one breeding pair per cage. Therefore, parentage of eggs was unequivocally assigned. The length, breadth, and mass of each egg at the time of collection were also recorded. Females were weighed at the beginning of the study.

### Maternal immunization

I collected blood samples (approximately 400  $\mu$ l) via the brachial vein before immunization to screen for background levels of binding to keyhole limpet hemocyanin (KLH). Females were then immunized via the semitendinosus muscle with 0.2 cc of KLH (Calbiochem 374805) emulsified in Freund's incomplete adjuvant (Sigma F5506) at a concentration of 1 mg KLH/ml (Casto et al. 2001). This novel antigen was used to control for prior exposure and to assess antibody response without inducing illness. Twenty days after the primary immunization, females were given a secondary immunization of KLH at the same dose. I immunized individuals twice to increase the magnitude of the antibody response and to induce a class shift in the response from IgM to IgY (Roitt et al. 1998). Blood samples were collected 10 days after the secondary immunization to quantify the maternal secondary antibody response to KLH.

### Egg collection

Eggs were collected from females throughout the experiment. A subset of eggs laid between 12 days and 3 months after the maternal secondary immunization ( $43 \pm 8$  days post-immunization) were incubated in a commercial incubator (TX-7 incubator; Stromberg's Chicks and Gamebirds Unlimited No. TX7) to control for any post-laying environmental differences. Every third egg laid by a female was frozen intact and reserved for antibody analyses. To

determine the antibody levels in eggs as compared to antibody levels in mothers and newly hatched offspring, I quantified antibody levels in one egg from each female laid within the same week as the egg her chick hatched from.

### Offspring measurements

At hatching, all chicks were housed together in a brooder (Georgia Quail Farms 0534) for approximately 2 weeks. Quail were then moved to larger cages (Morton Jones SBC362) and group housed. Blood samples were collected from offspring within 10 days of hatch and approximately every week thereafter for 2–3 weeks to assay maternally derived KLH-specific antibody levels and their persistence in offspring circulation. Maternal antibody persistence was measured in only the first hatched chick from each female to avoid pseudoreplication. The final sample size for maternal-offspring analyses was 24 due to a failure of some females to produce viable eggs.

### KLH ELISA

Specific antibody responses to KLH were quantified using an enzyme-linked immunosorbent assay (ELISA) (Demas et al. 1997). ELISA plates were coated with 200  $\mu$ l of dialyzed KLH at a concentration of 0.5 mg/ml. Plates were incubated overnight at 4°C to coat. The next day, after washing, plates were blocked with 5% milk powder diluted in PBS and again incubated overnight at 4°C. On the third day, the plates were washed and plasma samples diluted 1:40 in PBS-Tween were added to the wells in duplicate. Diluted plasma samples (100  $\mu$ l/well) were incubated on the plate for 3 h at 37°C. Positive and negative controls were also added in duplicate to all plates. The positive control was a pool of Quail plasma with high antibody responses to KLH. The negative control was a pool of plasma from Quail that had not been immunized with KLH. Finally, I included at least one blank well on each plate that contained only PBS-Tween without plasma as a measure of background binding. During the incubation, the secondary antibody (alkaline phosphatase conjugated anti-chicken IgG; Sigma A9171) was diluted 1:1,000 in PBS-Tween. After the plates were washed, 150  $\mu$ l of the diluted secondary antibody were added to all the wells. The plates were then incubated at 37°C for 1 h. At the completion of the incubation, 150  $\mu$ l of substrate buffer were added to all wells after washing. Fifteen minutes after addition of the substrate buffer, the plates were transferred to a BioRad Benchmark microplate reader (catalog no. 170-6850) and read using a 405-nm wavelength filter. I first calculated the mean blank value for each plate and subtracted this value from all of the other absorbances to account for non-specific binding. To minimize inter-assay variability, the mean

optical density for each sample was expressed as a percentage of its plate positive control optical density for statistical analyses. The average intra- and inter-assay coefficients of variation were 2 and 10%, respectively.

To quantify KLH-specific antibody levels in egg yolks, I used a similar ELISA procedure as described above for plasma samples. Because egg albumen thaws more quickly than yolk, eggshells and albumen of frozen eggs were easily separated from the yolk. Next, 0.2 g of homogenized yolk were added to 1 ml of PBS-Tween. Diluted yolks were vortexed thoroughly with glass beads to facilitate mixing. The diluted yolk samples were then used in the ELISA assay described above at a dilution of 1:40. Positive and negative controls for yolk assays were prepared from pooled positive and negative eggs, respectively, rather than pooled plasma. For yolk samples, the average intra- and inter-assay coefficients of variation were 5 and 3%, respectively.

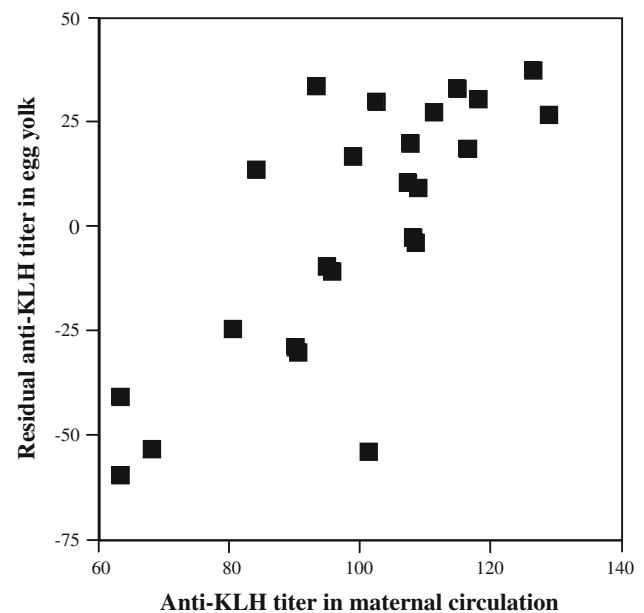
### Statistical analyses

Before data were analyzed, I checked for normality of residuals and homogeneity of variance. Data were analyzed using SAS version 9.1. The time elapsed between maternal immunization and egg collection significantly negatively correlated with anti-KLH titers measured in egg yolk ( $r = -0.42$ ,  $P = 0.04$ ). Therefore, the number of days post-immunization was included as a covariate in models of the relationship between maternal or egg antibody levels and antibody levels in offspring to control for effects of changes in antibody titers during the sampling period.

### Results

KLH-specific antibodies were detected in all egg yolks (range 11–143, mean =  $95 \pm 7$  SE). KLH antibody levels measured in maternal plasma and egg yolks were positively correlated ( $F_{1,23} = 31.10$ ,  $P < 0.0001$ ) (Fig. 1). However, antibody titers measured in maternal circulation were significantly greater than antibody titers measured in offspring circulation ( $t = 10.90$ ,  $df = 46$ ,  $P < 0.0001$ ). Yolk KLH antibody titer was not related to yolk mass ( $F_{1,23} = 1.94$ ,  $P = 0.18$ ), the number of eggs laid by a female ( $F_{1,23} = 2.10$ ,  $P = 0.16$ ), or female body mass ( $F_{1,23} = 0.60$ ,  $P = 0.45$ ).

Maternally derived KLH-specific antibodies were detected in the earliest samples from all offspring and initial antibody levels varied widely among offspring (range 9.28–95.67, mean =  $37 \pm 4.4$  SE). KLH-specific antibodies in offspring plasma are known to be of maternal origin because KLH was a novel antigen for these Quail, and offspring were not immunized with KLH. Prior to immunization, adult Quail did not have detectable levels of



**Fig. 1** Relationship between anti-KLH antibody titers measured in maternal circulation during the secondary response and anti-KLH titers measured in egg yolks produced by females at least 12 days after the secondary immunization. To control for the effect of changes in antibody titer with sampling date, the residuals of the relationship between days post-immunization and anti-KLH titer in egg yolks are plotted. Only one egg was measured from each female

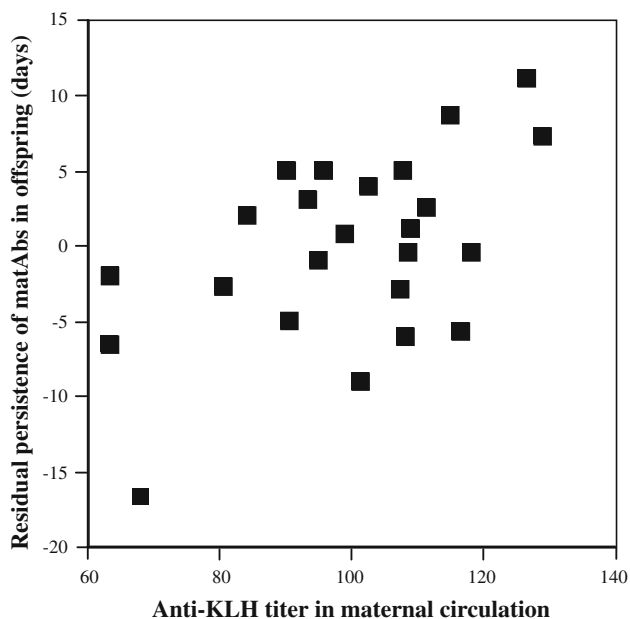
KLH-specific antibodies in circulation. Therefore, any KLH-specific antibodies must have been derived from the mother. Furthermore, KLH-specific antibody levels measured in maternal plasma and in offspring plasma were positively correlated ( $F_{1,23} = 8.28$ ,  $P = 0.009$ ). Male and female offspring did not differ significantly in maternally derived antibody levels ( $t = 1.67$ ,  $df = 22$ ,  $P = 0.11$ ).

During the 4-week sampling period post-hatch, the titer of KLH-specific antibodies declined in the plasma samples of all offspring. By 30 days post-hatch, none of the offspring had detectable levels of KLH-specific antibodies in circulation. To estimate the persistence of maternally derived antibodies in offspring circulation, I fit exponential decay curves to the antibody titers over the first month. Prior research in both humans and Blue and Gold Macaws (*Ara ararauna*) has established the use of exponential decay curves to describe the decline of maternally derived antibodies in offspring circulation (Linder et al. 2000; Lung et al. 1996). In this study, declines in offspring antibody titers tended to be better explained by exponential decay curves, rather than linear functions ( $t = 1.90$ ,  $df = 23$ ,  $P = 0.07$ ; exponential  $R^2 = 0.91 \pm 0.03$ ; linear  $R^2 = 0.88 \pm 0.03$ ). The fit of exponential curves was weakest when initial antibody levels in young were low ( $r = 0.44$ ,  $P = 0.032$ ). Maternally derived antibodies persisted in offspring circulation on average  $14 \pm 1.4$  days and the period of persistence ranged from 3 to 28 days.

The number of days chicks maintained detectable levels of KLH-specific antibodies in circulation was predicted by levels of KLH antibodies quantified in maternal circulation during the secondary response ( $F_{1,23} = 8.52$ ,  $P = 0.0082$ ) (Fig. 2), in egg yolks produced within a week of the egg the chick hatched from ( $F_{1,23} = 20.16$ ,  $P = 0.0002$ ), and by the initial KLH antibody levels measured in chick circulation ( $F_{1,23} = 50.07$ ,  $P < 0.0001$ ).

## Discussion

As found in other studies (Gasparini et al. 2002; Grindstaff et al. 2005; Grindstaff 2008), maternal, egg, and early offspring antibody levels were positively correlated. Therefore, any environmental perturbation that affects antibody levels in maternal circulation will have effects across generations on early offspring immunity. Furthermore, maternal antibodies were catabolized from offspring circulation within 30 days post-hatch. Because initial antibody levels were a strong predictor of the number of days chicks maintained detectable KLH-specific antibody levels in circulation, the primary benefit to offspring of receiving high maternally derived antibody levels is likely to be an increased period of protection from infection before their own immune systems mature.



**Fig. 2** Relationship between anti-KLH antibody titers measured in maternal circulation during the secondary response and the persistence in days of KLH-specific antibodies in offspring circulation. To control for the effect of changes in antibody titer with sampling date, the residuals of the relationship between days post-immunization and persistence time of maternal antibodies (matAbs) are plotted. Antibody titers were measured in only one offspring per female

Differentiation of the specific, adaptive immune response is largely antigen-driven; therefore, it is poorly developed in young animals with little previous exposure to antigens. Young are, as a consequence, primarily dependent on maternally derived antibodies and non-specific innate immune responses for protection (Klasing and Leshchinsky 1999; Seto 1981). However, during development, innate immune responses and activation of the inflammatory response are associated with growth suppression (Klasing 1997). Stimulation of the immune system through either natural infections or experimental immunizations has repeatedly been demonstrated to reduce growth rates in young animals (Klasing et al. 1987; Soler et al. 2003; Brommer 2004; Grindstaff 2008).

The presence of protective levels of specific maternal antibodies allows offspring to resist infection without simultaneously stimulating the innate immune response with its associated detrimental effects on growth (Klasing and Leshchinsky 1999; Grindstaff 2008). Thus, young with high levels of maternal antibodies may benefit both from an increased period of protection from infection and the ability to maintain growth rates after infection (Grindstaff 2008). The strong relationship between initial maternal antibody levels and the period of persistence of maternal antibodies also indicates that young with initially high levels of maternally derived antibodies will maintain protection against infection throughout a greater portion of the growth period than young with initially low antibody levels. In wild populations of birds, offspring of mothers with higher circulating antibody levels should be better insulated from local diseases, and thus better able to maintain growth rates if infected.

High levels of maternally derived antibodies may also have direct effects on the development of the offspring immune response. Several recent studies, including a few conducted with birds; suggest maternally derived antibodies may have persistent effects on the offspring immune response (reviewed in: Boulinier and Staszewski 2008; Hasselquist and Nilsson 2009). In Black-legged Kittiwakes (*Rissa tridactyla*), maternally derived *Borrelia burgdorferi* antibodies enhanced the offspring antibody response to *Borrelia* (Gasparini et al. 2006). In Song Sparrows (*Melospiza melodia*), maternal, but not paternal, immunization with tetanus toxoid enhanced offspring tetanus-specific antibody responses (Reid et al. 2006). In Pied Flycatchers (*Ficedula hypoleuca*), offspring of mothers immunized with *Salmonella* lipopolysaccharide (LPS) exhibited elevated antibody production in comparison to the offspring of control, non-immunized mothers (Grindstaff et al. 2006). In contrast, maternally derived antibodies have also been demonstrated to suppress offspring antibody responses. In particular, vaccination studies have reported blocking effects of maternal antibodies. Maternal antibodies may

block stimulation of the offspring immune response, which reduces the efficacy of the vaccination (Glezen 2003). This result has been replicated in Kittiwakes. Offspring of mothers immunized with the Newcastle disease virus vaccine produced lower antibody levels in response to the same vaccination than offspring of non-immunized mothers (Staszewski et al. 2007). The levels of maternal antibodies transmitted to offspring may reflect a dynamic balance between the benefits of providing maternal protection and potential costs of blocking stimulation of the offspring immune response, including suppression of later antibody responses.

The observation that Japanese Quail chicks catabolize maternal antibodies by 30 days post-hatch suggests that if these precocial chicks are to maintain humoral immunity, they must begin endogenous antibody synthesis by this time. Even at hatch, Quail chicks had lower antibody levels than mothers. Offspring are potentially most sensitive to infectious disease during the period when they have catabolized maternally derived antibodies and before they have the ability to synthesize antibodies at adult levels in response to infection (Rose and Orlans 1981; Smith et al. 1994; Klasing and Leshchinsky 1999). In order to predict when, and for how long, juveniles will be most sensitive to infection, both the persistence time of maternal antibodies in offspring circulation and the timing of the development of active immunity of offspring must be known (Boulinier and Staszewski 2008; Hasselquist and Nilsson 2009). Additionally, it will be important to determine whether there is any interaction between levels of maternal antibodies and the development of active immunity. The length of the sensitive period between the disappearance of maternal antibodies and the development of full capacity to produce new antibodies is also likely to differ among species as a function of local disease pressure and the developmental trajectory of offspring (e.g., precocial or altricial) (Klasing and Leshchinsky 1999).

Only a few previous studies have assessed the persistence of maternal antibodies in avian species. For example, detectable levels of maternally derived antibodies have been found in domestic chickens up to 3–4 weeks post-hatch (Rose and Orlans 1981). Levels of maternally derived immunoglobulins in domesticated ducks (*Anas platyrhynchos*) declined as early as five days post-hatch and reached minimal levels by day 14 (Liu and Higgins 1990). In Jackass Penguins (*Spheniscus demersus*), maternal anti-*Plasmodium* antibodies were undetectable by 10 weeks of age (Graczyk et al. 1994). In Blue and Gold Macaw chicks, the serum half-life of maternal anti-bovine serum albumin (BSA) antibodies was 3.85 days and BSA antibodies were undetectable in chicks by 42 days post-hatch (Lung et al. 1996). Future research should address the period of protection provided by maternal antibodies in

a broader diversity of birds, specifically in relation to the development of endogenous antibody production. This would generate opportunities to conduct comparative studies of maternal antibody transmission in relation to developmental mode.

## Zusammenfassung

Die Anfangslevel der von der Mutter erworbenen Antikörper bestimmen die Zeit der Persistenz im Blutkreislauf der Nachkommen

Wirbeltiermütter geben Antikörper an ihre Nachkommen weiter, die humorale Immunität im frühen Alter gewährleisten. Die Dauer des Schutzes durch mütterliche Antikörper variiert beträchtlich zwischen Arten und ist bei Vögeln bislang nicht umfassend untersucht worden. Eine Ermittlung der Dauer des Antikörper-Schutzes durch die Mutter kann dabei helfen herauszufinden, wann die Jungvögel wahrscheinlich am anfälligsten für Infektionen sind. Die Dauer des Schutzes durch mütterliche Antikörper wurde bei der Japanwachtel (*Coturnix japonica*) ermittelt, indem Weibchen mit Hämocyanin aus Schlitzschnecken (keyhole limpet hemocyanin, KLH) immunisiert und dann Blutproben von den Nachkommen gesammelt wurden. Die mütterlichen Antikörper blieben im Blutkreislauf der Nachkommen für durchschnittlich 14 Tage (Spannweite: 3–28) nachweisbar. Die Dauer der Persistenz ergab sich aus den Antikörper-Leveln die im Blutkreislauf der Mutter, im Eigelb oder bei den Nachkommen kurz nach dem Schlupf gemessen wurden. Daher ist es wahrscheinlich, dass der primäre Nutzen hoher mütterlicher Antikörperkonzentrationen für die Nachkommen in einer verlängerten Periode des mütterlichen Schutzes während des frühen Wachstums und der Entwicklung besteht.

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