

Effect of post-hatch feed deprivation on chick development

Dr. Inge van Roovert-Reijrink

Around day 18 of incubation eggs are transferred from the setter to the hatcher. Very often the first chicks hatch during day 19 of incubation, while the last chicks hatch 30 to 36 hours later. To have maximum hatchability, chicks are taken from the hatcher after a total incubation period of 512 hours (21 days and 8 hours).

Post-hatch chicks are selected, counted, and the viable chicks are transported to the farm. The extra time post-hatch needed for handling and transport depends on the flock size, necessary handling, and distance between hatchery and farm. As a consequence, a period without feed and water of 40 hours is no exception.

Chicks seem to be able to compensate for a relatively short period of delayed feed access post-hatch. When the period without feed and water post-hatch is not longer than 24 h, differences in development can be seen during their early life, but performance and

health status of the chicks can be the same during later life, as shown in several studies (Bhanja et al., 2009; Lamot et al., 2014).

However, when the period without feed and water post-hatch exceeds 24 h, which is common in commercial practice, this can have long lasting effects. In the current article the effect of delayed access to feed post-hatch on growth performance, yolk utilization and intestine development, immune system, and muscle development will be discussed according to the latest published research on this topic.

Growth performance

Delayed access to feed and water post-hatch results in delayed onset of growth. This means that chicks that do not receive feed immediately post-hatch will have a lower body weight upon arrival at the broiler farm compared to chicks that have access to feed immediately.

In the study of Bhanja et al. (2009) chicks subjected to feed deprivation beyond 24 h (32-48 hours) post-hatch could not compensate for their delayed start in growth by 5 weeks of age in comparison to chicks that had access to feed within 24 h post-hatch. Feed deprivation for 48 h was even more detrimental to growth than 24-40 h. Juul-Madsen et al. (2004) also showed that chicks deprived of feed for 48 h had a lower body weight than chicks fed immediately post-hatch during the complete grow out period (42d). The 24 h feed deprived chicks had already compensated for the delay in growth by d 8 while the 48 h feed deprived chicks did not seem to catch up till the end of the grow out period (42d) indicating that feed deprivation for more than 24 h post-hatch is harmful in relation to the chick's ability to grow. Simon et al. (2015) also showed that chicks with access to feed and water immediately post-hatch showed a significant higher body weight than chicks with 72 h feed deprivation throughout the whole experimental period (42d).



It can be concluded that feed deprivation post-hatch for a period longer than 24 h has long lasting effects on growth performance because chicks are limited in their ability to compensate for the delay in onset of growth.

Yolk utilization and intestine development

As was previously shown by Noy et al. (1996), Bhanja et al. (2009) also showed that residual yolk uptake was significantly higher in chicks that were fed immediately post-hatch compared to feed deprived chicks for 48 h. At 72 h post-hatch, residual yolk weight of chicks with immediate access to feed was significantly lower than of chicks that were feed deprived for only 8 h. At 72 h post-hatch the residual yolk of chicks that were deprived of feed for 24 h or longer contained more lipid and less protein than chicks deprived of feed shorter than 24 h. This suggests that other nutrients are used from the yolk when external feed is available for the chicks than when no external feed is available.

Bhanja et al. (2009) did not find an effect of early feeding on relative weights of the proventriculus and gizzard. However, liver, pancreas, and jejunum weight were significantly higher in chicks fed during the initial 24 h period compared to chicks that were feed deprived for 32 to 48 h post-hatch. The ileum was significantly heavier in chicks fed immediately compared to those deprived of feed for 48 h. The small intestinal segments like jejunum and ileum were significantly longer in chicks fed during the first 24 h period compared to 32 to 48 h post-hatch, which was in agreement with results of Maiorka et al. (2003).

Geyra et al. (2001) also concluded that early access to feed post-hatch is important for optimal early intestinal development. They showed that the jejunum seemed to be the most sensitive of the intestinal segments. Feed deprivation between 0 and 48 h post-hatch decreased crypt size, the number of

crypts per villus, crypt proliferation, villus area, and the rate of enterocyte (intestinal absorptive cells) migration in the duodenum and jejunum. All of these imply a reduced absorptive function of the intestines, meaning that once feed intake starts, uptake of nutrients from the gut may be impaired in delayed fed chicks.

Immune system

The immune system of a chicken consists of many complex components. Roughly, the first line of defense is a physical barrier between the chicken and the outside world such as the skin and mucous layers. Any pathogen that manages to penetrate those barriers will be encountered by cells that will attack unknown bodies such as viruses and bacteria indiscriminately. This part of the immune system is called the innate immune system as it is present from birth. The second part of the immune system is called the adaptive immune system and it partly consists of maternal immunity provided through the yolk, and partly of immunity that will have to develop post-hatch. Important components of the adaptive immune system are antibodies, which are proteins that can neutralize bacteria and viruses; as well as B-cells and T-cells (lymphocytes), which are involved in antibody production and even have a memory function so they can recognize previously encountered pathogens with a quick response.

Feed deprivation immediately post-hatch not only affects growth performance and intestine development, but can also be harmful for the development of the complex immune system. Juul-Madsen et al. (2004) showed that feed deprivation post-hatch affected immune parameters in the blood such as: the relative expression of B-cell antigens (BU-1 molecules) on the surface of B-cells, the ratio of different types of T-cells (CD4/CD8), which assist other components of the immune system and are able to

destroy virus and tumor infected cells, and lastly the expression of cell surface proteins that are able to recognize pathogens (relative expression of MHC class II positive leukocytes). Chicks that were feed deprived the first 48 h post-hatch had a lower relative expression of B-cell antigens (BU-1 molecules) on the surface of B-cells from d 5 to d 42. For the other two mentioned immune variables, the immediate fed chicks had the lowest value in comparison to chicks that had access for feed after 24 or 48 h. These results are complicated and there is no straightforward explanation. Juul-Madsen et al. (2004) suggested according to these results that feed deprivation for 48 h post-hatch was unfavorable to the viability and immune performance of broilers whereas deprivation for 24 h appears to be acceptable for growth and normal immunological performance.

Simon et al. (2015) showed that early life feeding strategy and housing conditions influenced the chick's response to an immune challenge that they received on d28. Chicks housed on the floor (normal situation for many broilers) and which had delayed access to feed post-hatch (72h), showed the highest antibody titers against HuSA, which is an unknown human protein that a chicken's immune system will respond to as though being infected. These chicks also showed the strongest sickness response and poorest performance in response to the applied immune challenge.

Intestinal development occurs in concert with the development of the gut associated lymphoid tissue (GALT). The GALT is a component of the mucosal immune system and provides protection against pathogens encountered by the gut. In the study of Bar Shira et al. (2005), GALT activity was determined by antibody production, distribution of B and T cells (lymphocytes) in the gut, expression of lymphocyte specific genes, and distribution of B and T cells in



the cloacal bursa. Their findings showed that while development of GALT in the foregut (duodenum, jejunum, and ileum) was only slightly and temporarily delayed by feed deprivation (72h). This was also confirmed by Simon et al. (2014), who also did not find a clear effect of early feeding on immune maturation in the foregut (ileum) in comparison to 72 h feed deprivation post-hatch.

However, Bar Shira et al. (2005) showed that development and functionality of GALT in the hindgut was impaired during the first 2 weeks of life. During this critical 2 week period, chicks with delayed access to feed might be more vulnerable to environmental pathogens than the immediate fed chicks.

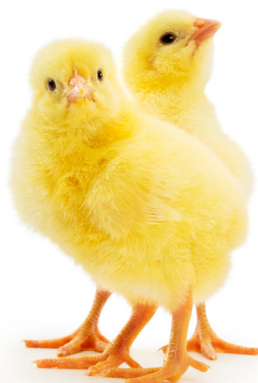
Muscle development

Although intestine growth occurs also in chicks without access to feed and water post-hatch to some extent, external feed intake is essential for muscle growth (Bigot et al., 2003). Feed deprivation post-hatch delayed pectoral muscle weight gain and the weight increase occurred only after chicks had access to feed. In contrast, the weight of the intestinal fragments of the jejunum increased in feed deprived chicks during the fasting period, but remained lower than in fed chicks even after refeeding. It seems that intestinal growth is preferential in early development in chicks compared to muscle growth (Bigot et al., 2003). Bigot et al. (2003) showed that early fed chicks increased the capacity for protein

synthesis during the first 3 d post-hatch (due a greater increase in RNA than protein levels) concomitantly with increase in DNA level, which suggested satellite cell proliferation as previously described by Halevy et al. (2000). RNA level and consequently capacity for protein synthesis were only increased after feeding in the feed deprived chicks and DNA level remained lower than in early fed chicks during the first 4 d post-hatch (Bigot et al., 2003). This may explain why it takes some time for chicks to compensate for the early loss in body weight gain, if they manage to do so at all. In the study of Halevy et al. (2000), chicks that were feed deprived directly post-hatch did not regain their body weight or breast muscle weight by d 41, however chicks that fasted between d 4-6 post-hatch had full growth compensation by d 41.

Conclusion

It can be concluded that feed deprivation for a period longer than 24 h post-hatch can have long lasting negative effects on growth performance, intestinal development, the immune system, and muscle development. A period without feed and water of 40 hours, from hatch until arrival on the farm, is no exception in current daily practice. By providing access to feed and water immediately post-hatch, the negative effects of feed deprivation are avoided and optimal development is ensured for all chicks, whether they are the first or the last to hatch.



References

Bar Shira, E., D. Sklan, and A. Friedman. 2005. *Impaired immune response in broiler hatchling hindgut following delayed access to feed*. *Vet. Immunol. Immunopathol.* 105:33-45.

Bhanja, S. K., C. Anjali Devi, A. K. Panda, and G. Shyam Sunder. 2009. *Effect of post hatch feed deprivation on yolk-sac utilization and performance of young broiler chickens*. *Asian-Aust. J. Anim. Sci.* 22:8:1174-1179.

Bigot, K., S. Mignon-Grasteau, M. Picard, and S. Tesseraud. 2003. *Effects of delayed feed intake on body, intestine, and muscle development in neonate broilers*. *Poult. Sci.* 82:781-788.

Geyra, A., Z. Uni, and D. Sklan. 2001. *The effect of fasting at different ages on growth and tissue dynamics in the small intestine of the young chick*. *Br. J. Nutr.* 86:53-61.

Halevy, O., A. Geyra, M. Barak, Z. Uni, and D. Sklan. 2000. *Early posthatch starvation decreases satellite cell proliferation and skeletal muscle growth in chicks*. *J. Nutr.* 130:858-864.

Juul-Madsen, H. R., G. Su, and P. Sørensen. 2004. *Influence of early or late start of first feeding on growth and immune phenotype of broilers*. *Br. Poult. Sci.* 45:2:210-222.

Lamot, D. M., I. B. van de Linde, R. Molenaar, C. W. van der Pol, P. J. A. Wijtten, B. Kemp, and H. van den Brand. 2014. *Effects of moment of hatch and feed access on chicken development*. *Poult. Sci.* 93:1-11.



Maiorka, A., E. Santin, F. Dahlke, I. C. Boleli, R. L. Furlan, and M. Macari. 2003. *J. Appl. Poult. Res.* 12:483-492.

Noy, Y., Z. Uni, and D. Sklan. 1996. *Routes of yolk utilization in the newly-hatched chick*. *Br. Poult. Sci.* 37: 987-996.

Simon, K., G. de Vries Reilingh, J. E. Bolhuis, B. Kemp, and A. Lammers. 2015. *Early feeding and early life housing conditions influence the response towards a noninfectious lung challenge in broilers*. *Poult. Sci.* 00:1-8.

Simon, K., G. de Vries Reilingh, B. Kemp, and A. Lammers. 2014. *Development of ileal cytokines and immunoglobulin expression levels in response to early feeding in broilers and layers*. *Poult. Sci.* 93:3017-3027.