

Chickens *Do Not* Receive Growth Hormones: *So Why All the Confusion?*

Naturally occurring hormones, such as estrogen, progesterone, and testosterone, are essential for various physiological processes in humans and animals. Throughout history, these naturally occurring hormones have been making their way into the environment, posing the risk of contamination.

The World Health Organization (WHO) has identified certain environmental contaminants as a global concern. These contaminants are capable of harming reproduction and development by altering endocrine functions in humans and wildlife. As the human population grows and livestock production becomes more concentrated, the quantity and concentration of hormones within localized areas increases.

As a result, many of us in the poultry field hear the same question with increasing frequency: “Why do you put hormones in the feed to make chickens grow so big and fast?” The fact that the question begins with “why” instead of “do” indicates the level of confusion and misunderstanding of the consuming public. **The truth is no hormones have been allowed in poultry production for more than 50 years.** Hormone use in poultry production was banned in the United States in the 1950s.

Why the Confusion?

Some of the confusion and misunderstanding may stem from the fact that the poultry and beef cattle industries operate under different regulations. While growth hormone use is banned in poultry production, it is a perfectly legal and accepted practice in the beef cattle industry.

The Food and Drug Administration (FDA) first approved growth hormones to increase growth, feed efficiency, and carcass leanness of beef cattle in 1956. Currently, there are five hormones (progesterone, testosterone, estrogen, zeranol, and trenbolone acetate) approved as growth implants for cattle (Archibeque et al., 2007). Trenbolone acetate and zeranol are synthetic hormones, and the USDA Food Safety Inspection Service routinely monitors for residues of these products to ensure the safety of the beef supply.

Progesterone, testosterone, and estrogen are naturally occurring hormones in both humans and animals. These hormones are necessary for normal development, growth, and reproduction. Additionally, humans may ingest steroid hormones as part of hormone replacement therapy and in birth control pills. Of these hormones, estrogen currently tends to receive the most attention. Estrogen occurs naturally in both males and females and plays a role in sexual development, reproduction, and behavior (Swyers, 2011). Federal regulatory monitoring of estrogen, progesterone, and testosterone is not possible because it is not possible to tell the difference between hormones used for treatment and those produced naturally by the body.

Unlike poultry (which receive no added growth hormones), most beef cattle fed in the United States do receive a growth-promoting hormone implant, usually when they enter a feedlot. These implants generally are given to beef cattle in the form of a pellet that is surgically implanted under the skin on the back of the animal’s ear. (The



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ear is used because ears do not enter the food supply.) Over the course of a 100- to 120-day period, the implant slowly dissolves and releases the hormone.

These implants are important in the beef cattle industry because animals spend 100–200 days in the feedlot. Recognized benefits of the implants include decreasing the number of days on feed, improving carcass yields, increasing efficiency of the cattle feeding process, and keeping beef prices low for the consuming public. However, **growth implants can only be used in beef cattle**. There are no steroid hormones approved for growth purposes in poultry, dairy cattle, veal calves, or pigs.

We have mentioned hormone growth *implants* several times but have not discussed *feeding* hormones to animals. There's a reason for that. Regardless of what you may have read or been told, **growth hormones added to chicken feed would NOT be effective**. This is one of many reasons chickens do not receive hormones and another possible source of confusion.

Hormones exist in two different chemical forms: they can be steroids or proteins. *Steroid* hormones are active in the body when taken orally. For example, birth control pills are steroid hormones that can be taken orally and remain effective even after passing through the digestive tract. However, *protein* hormones are broken down in the stomach and extensively metabolized after leaving the gut—they lose their ability to act in the body when ingested. Therefore, to have an effect on the body, protein hormones must be injected. This is why most feedlot cattle receive growth hormone as a pellet injected under the skin of the ear instead of as a supplement added to the feed.

Growth hormones are proteins, similar to insulin that is used to treat diabetes. As people with diabetes well know, no oral form of insulin exists that can be taken to avoid all those insulin injections. Insulin taken in oral form would be broken down in the digestive tract just as any other protein is broken down, and would not be effective. If protein growth hormones were given orally to chickens via the feed, they would be broken down in the digestive tract and rendered ineffective.

Therefore, like insulin in humans, growth hormones given to chickens would have to be injected to be effective. And to further complicate matters, research indicates that, to be administered successfully, **chickens would need to receive growth hormone injections several times each day** (Czarick and Fairchild, 2012). This undertaking is logistically impossible. Most broiler growers have 20,000 or more chickens in each house and numerous houses on the farm. There is no way to catch each chicken in every broiler house numerous times a day and give it a hormone injection. Besides, modern broilers have been genetically selected by primary breeder companies to grow to their physiological limit. The fact is, chickens grow as fast as they

should naturally, without the use of growth hormones. Additionally, using hormones to force chickens to grow too quickly would cause increased leg problems and even early death.

Reasons for Rapid Growth

There are three main reasons for the rapid growth rate we see in today's commercial poultry, but none of them are related to hormones.

The first, mentioned previously, is the **success of primary breeder companies** in selecting the best birds for growth and performance. For the past several decades, geneticists have been able to cut roughly one day per year off the time it takes to reach a specified target weight. They have benefited from the short generation interval (lifespan) of the chicken, allowing them to make huge strides in a short period of time. Genetic improvement in the pork and beef industries comes much slower because of the increased generation interval and the time it takes to recognize genetic variation and improvement.

Second is research related to **nutritional requirements of the bird**. We now know exactly what we should be feeding different genetic strains, and birds are kept to specific target weights in terms of energy, protein, vitamins, and minerals to optimize performance and growth.

Third, we better understand the kind of **environment the bird needs** to make the most of the genetic and nutritional potential it has. This includes providing the proper temperature, air quality, ventilation, lighting, and feeder and drinker space to obtain optimum performance. The right environment, coupled with high-quality feed and superior genetics, yields a bird that does not require and would not benefit from growth hormones.

Hormone Awareness Is Increasing

Hormones are naturally occurring chemical messages released into the blood stream by the hormone-producing organs in the bodies of all animals, including humans. This means that humans, chickens, and other animals have naturally occurring hormones in their systems at all times. As a result, steroidal hormones produced by humans and animals are constantly being excreted into the environment in their active forms.

Among the general public, there is increasing interest in and awareness of hormone levels found in the environment and the food we eat. While natural steroid hormones have always been present in the environment, they are of growing concern for many individuals. Increasing human population numbers and intensive livestock production needed to feed the population could lead to concentrated releases of hormones.

Today, hormone residues in meat are often the first concern of consumers in Europe and the United States (Sundlof, 1994). This is partly because synthetic steroid hormones used as pharmaceutical drugs have been found to affect cancer risks. For example, a synthetic estrogen drug used in the 1960s, diethylstilbestrol, was withdrawn from use after it was found to increase the risk of vaginal cancer in daughters of treated women (Gandhi and Snedeker, 2000).

Additional confusion concerning hormones stems from the fact that, for years, people didn't realize steroid hormones could come from plants. They thought this exposure came only from food of animal origin. However, many plants that are important to human nutrition contain phytoestrogens. Phytoestrogens are estrogenic compounds found in plants, including fruits, vegetables, beans, peas, and cereal grains (Swyers, 2011). And a number of foods contain hormonally active substances at concentrations exceeding those found in meat.

In fact, Hartmann et al. (1998) reported that meat does not play a dominant role in the daily intake of steroid hormones. These researchers indicated the main source of estrogens and progesterone are milk products (60–80 percent). Contribution to the hormone supply from eggs and vegetable foods were on the same order of magnitude as meat, meat products, and fish. However, Handa et al. (2010) concluded that estrogen intake from daily meat consumption cannot be disregarded as a factor governing human health. Among dietary risk factors, Ganmaa and Sato (2005) reported they were most concerned with milk and dairy products, because today's milk is produced from pregnant cows, in which estrogen and progesterone levels are markedly elevated. In contrast, Parodi (2012) reported that, upon

ingestion, only 2–5 percent of the bioactive form of estrogen survives metabolism in the intestinal mucosa and first-pass-effect of the liver. Hartmann et al. (1998) indicated the first-pass-effect of the liver inactivates about 90 percent of ingested hormones. Although hormone risks continue being debated and researched on several fronts, definitive answers are currently difficult to come by, and conflicting reports continue to add to the level of confusion among consumers.

Understanding the issue is made more difficult by the fact that **humans' natural steroid production far exceeds the daily hormonal intake values from either plant or animal foods**. Table 1 lists daily natural human hormone production in relation to amounts in birth control pills and certain other foods. Estrogen production is reported in nanograms. A nanogram is one-billionth of a gram (0.000000001), which is comparable to one blade of grass on a football field (NCBA, 2007). Children produce about 20 times more progesterone and about 1,000 times more testosterone and estrogens than they ingest in food on average per day; and children show the lowest level of steroid production among all humans (Hartmann et al., 1998).

Hormone data is limited in chickens because chickens do not receive growth hormone supplements. Therefore, unlike in the beef cattle industry, there are no synthetic hormone levels to test for in chickens. However, as mentioned previously, there are naturally occurring levels of estrogen, progesterone, and testosterone in chickens and all other animals. Handa et al. (2010) reported estrogen levels in U.S. and Japanese chicken fat samples (Table 1). Data for chicken fat were reported because estrogen levels were generally higher in fat than in meat (Handa et al., 2010).

Table 1. Daily endogenous (natural) estrogen production in relation to amounts in birth control pills and certain other foods.

Source of estrogen	Amount in nanograms	Source of estrogen	Amount in nanograms
Pre-pubertal girl, daily	54,000	3 oz soybean oil	168,000
Pre-pubertal boy, daily	41,500	3.5 oz soy protein concentrate	102,000
Adolescent girl, daily	93,000	1 cup of soy milk	30,000
Pregnant woman, daily	3,415,000	3 oz wheat germ	3,400
Non-pregnant woman, daily	480,000	3 oz eggs	2,625
Normal adult man, daily	136,000	3 oz cabbage	2,016
Low-dose birth control pill	20,000	3 oz ice cream	520
Regular-dose birth control pill	30,000–35,000	3 oz peas	340
High-dose birth control pill	50,000	3 oz potatoes	225
		3 oz steak (implanted beef animal)	1.9
		3 oz USA chicken fat	1.8
		3 oz Japanese chicken fat	1.8

Sources include Anonymous, 2010; Anonymous, 2011; Handa et al., 2010; NCBA, 2007; Swyers, 2011. See References section for full citations.

There is reason for concern regarding the impact of hormones ingested or released into the environment. As the population grows, and farm animal production increases to meet the increasing food demand, protecting the environment will become even more important. In addition, research into daily hormonal intake levels from both plant and animal origin should continue in order to monitor effects on human health and well-being. However, it is very important to remember that, on a daily basis, humans naturally produce far greater amounts of hormones than they consume in food.

Dispelling the Myths

Both humans and animals excrete hormones that have the potential of reaching the environment. Diligence with nutrient management programs for farming operations is a necessity, and we must continue to closely monitor impacts on the environment. Also, the poultry industry must do a better job of providing factual information to consumers to combat the confusion, myths, and inaccurate information that has become so prevalent regarding hormone use and chicken production. The truth is **no hormones are used in poultry production**. And even though the truth speaks for itself, the poultry industry must be vocal if we expect the message to be heard.

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