

# CORNELL POULTRY POINTERS

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Barb Smagner, Managing Editor

## THE EVER-RECURRING THREAT OF EMERGING DISEASES

After a successful battle against H7N2 avian influenza virus (AIV) in Pennsylvania, the poultry industry in North America faces two new challenges: velogenic viscerotropic Newcastle disease (VVND) in California, H5N2 AIV in New Jersey, and Pullorum disease in Canada.

### **Velogenic Viscerotropic Newcastle Disease (VVND)**

VVND, a highly pathogenic form of Newcastle disease, causes up to 100% mortality in susceptible flocks, and is difficult to control solely with live-virus vaccines. VVND is a constant scourge of the poultry industry in the Middle East, Mexico, Central and South America. Poultry producers in these countries are forced to use a combination of inactivated oil-emulsified and live-virus vaccines on their chicks, sometimes as early as 1 day of age. The vaccine's cost and the respiratory problems associated with more aggressive live NDV vaccines, take a heavy toll on the cost of egg and poultry production. This is why it is so important to eradicate VVND. The last time California faced VVND, in 1973, the cost of control efforts was close to \$56 million.

VVND emerged in a small flock of chickens in downtown Fresno, California. The disease was promptly identified by the Fresno Branch of the California Veterinary Diagnostic Laboratory System. The rapid response in diagnosis and destruction of affected chickens averted wider dissemination of the disease, and loss of national and international trade of poultry products for California.

According to "Lab Notes" from the California Veterinary Diagnostic Laboratory System, on May 24, 1998, 10 chickens from a 48-bird backyard flock of game chickens began showing signs of disease. The Fresno Branch of the California Veterinary Diagnostic Laboratory System received the sick chickens on May 26, and by May 29, the diagnosis of VVND had been made.

### CONTENTS

- 1) The Ever-Recurring Threat of Emerging Diseases
- 2) Value-Added Eggs - A Golden Opportunity for the Egg Industry
- 6) Way West, Down Under, and Up North
- 7) Poultry Reality Check Needed
- 11) Developments in Research

The chickens on the premises were placed under hold order by the California Department of Food and Agriculture. By June 10, the premises had been depopulated, cleaned, and disinfected.

### **H5N2 Avian Influenza Virus (AIV)**

While the Pennsylvania poultry industry successfully wrestled with H7N2 AIV, a game bird farm in Princeton, NJ, broke with H5N2 AIV. The disease was diagnosed on October 13, 1998, by the University of Pennsylvania New Bolton Center. The flocks currently in the farm: 32,000 pheasants, 25,000 quail and 5,000 chukar partridges are under quarantine. Since the nearest commercial flock is 5 miles away, the chance of air transmission is greatly reduced, although transmission by mechanical means (humans, trucks, crates, etc.) is still a possibility.

The New Jersey isolate is of low pathogenicity, but it has, as other H5N2, the potential to mutate to a more pathogenic form. During the 1983 Pennsylvania and 1994 Mexico outbreaks, low pathogenicity H5N2 AIV mutated to a very virulent form, causing heavy mortality and loss of production.

### **Pullorum disease (PD)**

In October 1998, PD was detected in 1,050 chickens in 10 Canadian flocks that were humanely destroyed with governmental compensation to the owners. PD has been but eradicated from the developed countries. Its resurgence endangers Canada's international trade of poultry products. PD causes high mortality in young chicks, and was one of the diseases that impeded the development of the poultry industry before it was eradicated. In order to maintain its international trade British Columbia will have to test more than 20,000 chickens to meet the terms of an international agreement that calls for a voluntary,

three-year exporting ban of poultry products if the disease is not eradicated within one year from the time it was discovered.

These outbreaks underline the importance of disease surveillance, rapid diagnosis and rapid response in eradication efforts by official agencies. However, poultry producers have to do their part by tightening biosecurity at the farm. Viruses are transmitted before a diagnosis is made. Your chickens may get infected even before the chickens where the virus originated show signs of disease!

Always verify that trucks and crates brought into the farm have been washed and disinfected, do not allow unnecessary visitors into the farm, and have clean boots and coveralls available for maintenance personnel, and visitors.

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## **VALUE-ADDED EGGS - A GOLDEN OPPORTUNITY FOR THE EGG INDUSTRY**

Although the egg is a rich source of almost all of the nutrients which are essential for humans and is among the cheapest source of animal protein on the market, it has been unfairly blamed for its cholesterol content for many years. The per-capita consumption of an egg during

the 1940's was about 320 in the U.S. Through the years, the per-capita consumption of eggs has continuously reduced and reached its lowest level of about 235 eggs in 1995. It has been only during the past few years that the per-capita egg consumption has slightly increased and approached a value of about 240 in 1998. Two factors have contributed to reduced egg consumption by Americans throughout the years. These include changes in life-style and the negative publicity surrounding the cholesterol content of eggs. It is now a fact that the old value of 270 mg cholesterol per large-sized egg was an overestimation and a more real value for egg cholesterol is about 190 - 210 mg. This, together with educating the consumers about the nutritional value of eggs has resulted in the negative publicity surrounding eggs to diminish greatly in recent years. In fact, the results of a recent study by Hu et al., which were published in the April 21, 1999 issue of JAMA (Journal of the American Medical Association 281:1387-1394), suggested that consumption of up to one egg per day is unlikely to have substantial overall impact on the risk of coronary heart disease (CHD) or stroke among healthy men and women. Additionally, with the availability of further processing of egg products which is compatible with our current life-style, the future consumption of eggs appears to be very promising in the coming years.

During the past decade, it has become clear that the nutritional value of an egg can be enhanced even further by its enrichment with certain nutrients including omega-3 fatty acids, vitamin E, beta-carotene, and others. The objective of this article is to provide you with some information regarding the chemistry, production, and health implications of omega-3-enriched eggs.

### What are the omega-3 fatty acids?

Simple lipids or fats are esters of fatty acids with certain alcohols (primarily glycerol). Simple fats are made up of one molecule of glycerol and three molecules of fatty acids. The fatty acids can be divided into two classes; saturated and unsaturated fatty acids. As the name implies, the saturated fatty acids do not have any unsaturated bonds in their structures. An example for these types of fatty acids are palmitic acid and stearic acid. A molecule of palmitic acid has 16 and a molecule of stearic acid has 18 carbon units in their structures, respectively. In short forms, the symbol of C16:0 is used to show palmitic and the symbol of C18:0 is used to show stearic acid. The "0" signifies that these fatty acids do not have any unsaturated bonds in their molecules (see Fig. 1). On the other hand, the unsaturated fatty acids may have one or more double bonds in their structures. Due to this, they are called unsaturated fatty acids. When the number of double bonds in a molecule of fatty acid is more than a couple, they are usually called polyunsaturated fatty acids (PUFAs).

The unsaturated fatty acids can be divided into three classes or families: 1.) *Omega-9 family* of fatty acids. The *parent fatty acid* of this family is oleic acid which is non-essential and can be synthesized in the animal's body. This fatty acid is designated with the symbol of C18:1, n-9, which means it has 18 carbon atoms and one unsaturated double bond between carbon numbers 9 and 10, when the carbon atoms are counted from the CH<sub>3</sub> terminal (see Fig. 1). 2.) *Omega-6 family* of fatty acids. The *parent fatty acid* of this family is linoleic acid which is an essential fatty acid for animals, because it cannot be synthesized in the body. Fatty acids of these family are called omega-6, because the first double bond is located between carbon numbers 6 and 7 when the

carbon numbers are counted from the CH<sub>3</sub> terminal. Linoleic acid designated with the symbol of C18:2, n-6, 9, which means it has 18 carbon atoms and two unsaturated double bonds between carbon numbers 6 and 7 and carbon numbers 9 and 10, respectively, when the carbon atoms are counted from the CH<sub>3</sub> terminal (see Fig. 1). 3.) *Omega-3 family* of fatty acids. The *parent fatty acid* of this family is linolenic acid. Fatty acids of this family are called omega-3, because the first double bond is located between carbon numbers 3 and 4 when the carbon numbers are counted from the CH<sub>3</sub> terminal. Linolenic acid designated with the symbol of C18:3, n-3, 6, 9, which means it has 18 carbon atoms and three unsaturated double bonds between carbon numbers 3 and 4, carbon numbers 6 and 7 and carbon numbers 9 and 10, when the carbon atoms are counted from the CH<sub>3</sub> terminal (see Fig. 1).

The parent fatty acids of omega-6 (linoleic acid) or omega-3 (linolenic acid) cannot be synthesized by the animal or human body. However, when these fatty acids are provided in the diet, the body has proper enzymatic systems for converting them to fatty acids with longer carbon chain and more unsaturated bonds. For example, linolenic acid (C18:3, n-3, 6, 9) can undergo repeated steps of desaturation and elongation (by proper enzymes; desaturases and elongases) and converted to arachidonic acid, a fatty acid with 20 carbon and four unsaturated double bonds (C20:4, n-6, 9, 12, 15). Similarly, linolenic acid (C18:3, n-3, 6, 9) can undergo repeated steps of desaturation and elongation and converted to EPA (eicosapentaenoic acid; C20:5, n-3, 6, 9, 12, 15), DPA (docosapentaenoic acid; C22:5, n-3, 6, 9, 12, and 15), and DHA (docosahexaenoic acid; C22:6, n-3, 6, 9, 12, 15, 18). As will be discussed in the following paragraphs, it is now well established that the omega-3 fatty acids and, particularly EPA, DPA, and DHA are important for

human health. However, it appears that the capability of an animal and human bodies in converting the parent omega-3 fatty acid (linolenic acid) to long-chain omega-3 fatty acids (EPA, DPA, and DHA) is limited. In fact, the reason that various kinds of fish and fish oils are rich sources of EPA, DPA, and DHA is not because they can convert linolenic acid to these PUFAs in their body, but is due to consuming various kinds of marine algae which are rich sources of these long-chain PUFAs.

### Why omega-3 fatty acids are important for human health

The potential health benefit of omega-3 fatty acids in the human diet has drawn attention since the original publication of Dyerberg et al. (1974; Am. J. Clin. Nutr. 28:958) reporting a link between dietary omega-3 fatty acid consumption and decreased incidence of cardiovascular disease in Eskimos. The results of more recent studies have suggested that omega-3 polyunsaturated fatty acids (PUFAs) such as EPA, DPA and DHA are important for brain development in children, normal functioning of the nervous system, preventing platelets aggregation, reducing serum triglycerides and very low density lipoproteins (VLDL) that promote clogging of the arteries, reducing cardiovascular problems and heart attacks, among others. It should be noted that the omega-6 family of PUFAs are also important for various physiological functions of the body, although they are structurally, metabolically, and functionally different than the omega-3 family of PUFAs. Prostaglandins which act like hormones and are important in reproduction, muscle contraction, transmission of nerve impulses, and control of blood pressure, are formed from arachidonic acid which this, in turn, is formed from linoleic acid, the parent fatty acid of the omega-6 family. The human diet not only

should have sufficient quantities of both the omega-3 and the omega-6 families of PUFAs, a proper ratio between the intake of these two families of fatty acids also appear to be important for good health. The proper ratio of omega-6 to omega-3 PUFAs in the human diet appears to be in the range of 1:1 to 4:1. The current ratio in the human diet in the developed world is about 10:1 or even greater. This, in part, is due to reduced consumption of marine sources and vegetables which are rich sources of omega-3 fatty acids and increased consumption of processed vegetable oils and cereal grains which are rich sources of omega-6 fatty acids. Due to this, a great demand exists for food sources which are enriched with omega-3 families of fatty acids. Although the current U.S. lists of the recommended daily allowances (RDAs) do not contain omega-6 nor omega-3 requirements, the possibility of establishing RDAs for these fatty acids appears to be forthcoming in the near future.

### **How to enrich eggs with omega-3 fatty acids**

An egg has about 10% fat that is exclusively located in the yolk. Although the fat content of the egg cannot be changed noticeably by dietary manipulation of nutrients, including adding fat to the diet, it has been well established that the fatty acid composition of an egg very closely is under the influence of fat composition of the diet consumed by laying hens. Increasing the sources of saturated fats in the diet would increase the percentage of saturated fatty acids in the egg lipids and, similarly, increasing the unsaturated sources of fats in the diet would increase the percentage of unsaturated fatty acids in the egg. Additionally, the levels of a number of vitamins such as vitamin E and some of the trace elements in the egg can be increased readily by increasing their concentration in the

layer's diet. Not surprising, enrichment of eggs with these nutrients will increase the cost of production. Nevertheless, the health consciousness segment of American families are ready to pay considerably more for such eggs. Such a high price not only can compensate for the higher costs of production, it can also be a profit-making venture for the egg industry.

One approach for increasing the concentration of omega-3 fatty acids in the egg is by the use of flaxseed in the layer's diet. Flaxseed is a rich source of unsaturated fats; it contains about 38% fat and 50% of this fat is in the form of linolenic acid (C18:3, n-3, 6, 9). Adding flaxseed to a hen's diet greatly increases the concentration of linolenic acid in the egg yolk lipids. The results of several studies have indicated that the addition of graded levels of flaxseed to the layer's diet can increase proportionally the concentration of linolenic acid in the egg yolk lipids. It appears that the concentration of linolenic acid (expressed as percentage of total fatty acids in egg yolk lipids) can be increased from a very low and negligible level of 0.26% in hens fed a diet without flaxseed to 2.3, 4.2, 6.8 and 8.9% due to adding flaxseed at levels of 5, 10, 15, and 20%, respectively, to the diet (Caston and Leeson, 1990, *Poultry Sci.* 69:1617-1620; Scheideler and Froning, 1996, *Poultry Sci.* 75:1221-1226). In the report of Scheideler and Froning (1996, *Poultry Sci.* 75:1221-1226), DPA was increased from a nondetectable level for hens fed the control diet to .099% and .175% for hens fed 10 and 15% flaxseed, respectively. The corresponding increases for DHA was from 0.52% to 1.735 and 1.795%. In the same study, the level of DHA was increased from 0.52% for hens fed the control diet to 2.87% for hens fed 1.5% menhaden fish oil. The percentage increase in the level of linolenic acid as observed by Scheideler and Froning (1996, *Poultry Sci.* 75:1221-1226) was

consistent with other reports (Caston and Leeson, 1990, *Poultry Sci.* 69:1617-1620; Jiang et al., *Poultry Sci.* 70:2467-2475). While some of the researchers recommended grounding flaxseed prior to its use in the diet due to its low digestibility by the birds, others observed a similar performance due to the use of ground as compared to whole seed in the diet. It has been suggested that the advantage of using whole as compared to ground seed can be due to the reduced possibility of lipid oxidation during the feed storage period. Apparently the brown variety of flaxseed is more favorable than the golden variety for use in the layer's diet. Another factor that may limit the use of flaxseed at 10-15% level is due to the possibility of development of fishy flavor in boiled eggs from hens fed flaxseed. Aymond and Van Elswyk (1995, *Poultry Sci.* 74:1358-1394), however, observed reduced egg production in hens fed 15% flaxseed. When flaxseed is used in the diet, the level of antioxidant should be kept at the maximum allowable level and the level of vitamin E should also be increased to reduce the chance of oxidative rancidity of fat during the feed storage period. Increasing the vitamin E content of the diet also has the advantage of increasing the vitamin E content of the egg and as a result, its nutritional value for humans. The major omega-3 fatty acid which is accumulated in yolk triglyceride fraction of yolk lipids, due to the consumption of flaxseed is linolenic acid, although as the above information indicates the concentration of DPA (C22:5, n-3) and DHA (C22:6, n-3) also are increased somewhat in the egg yolk lipids. According to Hargis and Van Elswyk (1993, *World Poult. Sci. J.* 49:251-264), the consistency of incorporation of linolenic acid from flaxseed into the egg helps to ensure a reliable and consistent egg product for human consumption in the designer food market.

As was mentioned before, an egg has about 10% fat, and a 60 g egg contains about 6 g fat. Because about 85% of the fat in yolk lipids is made up of fatty acids, the weight of fatty acids in a 60 g egg is about 5.1 g. Using the total omega-3 fatty acids values of 0.8, 6.034, and 8.77% for hens fed 0, 10, and 15% flaxseed (from the study of Scheideler and Froning 1996, *Poultry Sci.* 75:1221-1226), the total omega-3 fatty acids in a 60 g egg is calculated to be about 41, 308, and 447 mg per egg from hens fed 0, 10, and 15% flaxseed, respectively. These translate to about a 7-10% increase in omega-3 fatty acids concentration of the egg due to the use of 10-15% flaxseed in the layer's diet. These values are, of course, approximations and should be determined at each production site based on egg weight, lipid content of egg and actual percentage of omega-3 fatty acids in the egg. According to Leeson and Summers (1997, *Commercial Poultry Nutrition*) consumption of two such modified eggs each day provides adults with most of their daily recommended allowance of linolenic acid.

We previously mentioned another approach for enrichment of eggs with omega-3 PUFAs is by the use fish oils in the layer's diet. When expressed as the percentage of total fatty acids, canola and flaxseed oils have 12 and 53.3% linolenic acid (C18:3, n-3), respectively, with no detectable levels of long-chain omega-3 fatty acids. On the other hand, menhaden and herring oils have about 1% linolenic acid but a total of about 13-14% EPA, DPA, DHA. Although the use of fish oils have the advantage of enrichment of eggs mainly with long-chain members of omega-3 fatty acids (EPA, DPA and DHA) that are specifically involved in human health, the maximum level of fish oils that can be used in the layer's diet is limited due to production of eggs with fishy flavor. Additionally, the quality of fish oil is another factor

which needs to receive serious consideration for use in the layer's diet.

Still, another approach for enrichment of eggs with long-chain PUFAs of omega-3 family is the use of microalgae in the laying hen diet. Expressing as the percentage of total fatty acids, various kinds of microalgae may have up to 50% of their total fatty acids in the form of EPA, DPA, and DHA. In fact, microalgae can be produced that would be rich only in a specific kind of PUFAs, e.g., EPA, DPA, or DHA. Consequently, the use of microalgae have the potential to enrich eggs with higher levels of one or several PUFAs of omega-3 family. Currently, several laboratories are working in this area and it appears that the use of algal oils or their biomass in animal feeds for enrichment of meat and eggs with omega-3 PUFAs is forthcoming in the near future.

It should be noted that similar approaches as outlined above for enrichment of eggs with omega-3 fatty acids can be used for enrichment of broiler meat. The results of a recent study (Lopez-Ferrer et al., 1999, *Poultry Sci.* 78:356-365) have indicated that the use of relatively high levels of fish oil during the first few weeks and replacing the fish oil with flax oil during the last couple of weeks prior to slaughtering can have the potential to enrich the broiler meat with omega-3 fatty acids and particularly EPA and DHA.

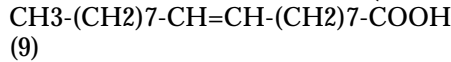
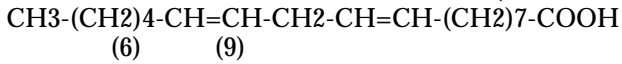
## Other opportunities

Eggs can be enriched easily for a number of vitamins and minerals such as vitamins E, A, B2, B12, folic acid, biotin, manganese and iodine, among others, by enrichment of the layer's feed with these nutrients (Nabor, 1979, *Poultry Sci.* 58:518-528 and 1993, *J. Appl. Poult. Res.* 2:385-393). Some forms of minerals such as organic selenium or chromium may be more efficiently used to enrich eggs with these minerals than their inorganic forms. Enrichment of eggs with beta-carotene and conjugate fatty acids (CLA) are other opportunities which are forthcoming. The egg industry should keep themselves abreast of the latest developments in these areas and use these golden opportunities for further enrichment of eggs. This not only is expected to increase the per-capita egg consumption, but could be a money generating venture for the egg industry.

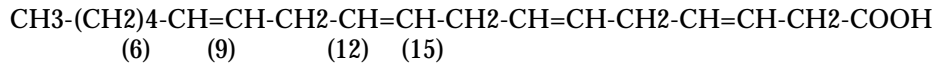
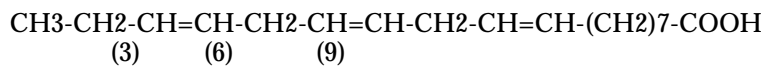
K. Keshavarz  
Department of Animal Science  
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**Figure 1.**

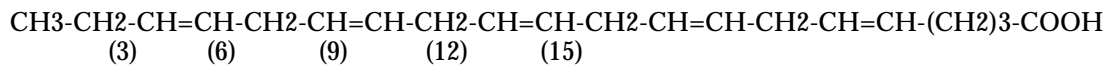
## THREE FAMILIES OF UNSATURATED FATTY ACIDS:

**1. OMEGA-9 FAMILY OF FATTY ACIDS (Parent fatty acid is oleic acid, C18:1, n-9)****2. OMEGA-6 FAMILY OF FATTY ACIDS (Parent fatty acid is linoleic acid, C18:2, n-6, 9)**

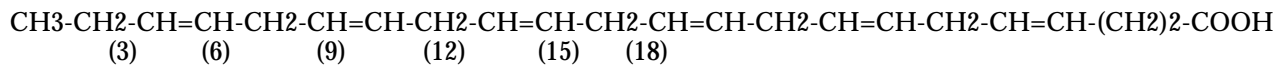
ARACHIDONIC ACID (C20:4, n-6, 9, 12, 15)

**3. OMEGA-3 FAMILY OF FATTY ACIDS (Parent fatty acid is linolenic acid, C18:3, n-3, 6, 9)**

EICOSAPENTAENOIC ACID (EPA = C20:5, n-3, 6, 9, 12, and 15)



DOCOSAHEXAENOIC ACID (DHA = C22:6, n-3, 6, 9, 12, 15, 18)




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## WAY WEST, DOWN UNDER, AND UP NORTH

**Newcastle Disease Outbreaks**

Could Newcastle disease (ND) be on the rise again? This appears to be the case, as countries which have been ND-free are once more battling a virulent form of this devastating disease.

In May 1998, a virulent form of ND was diagnosed in a flock of backyard chickens in California. It was only thanks to the rapid response of the California State Government and the United States Department of Agriculture, that this deadly disease was stopped. Thus, the U.S. poultry industry suffered no repercussions or losses in poultry export markets due to this incident.

This year, in April, Australia started its own battle to control a virulent form of ND. The disease was first detected more than two weeks ago on Mangrove Mountain Ridge, New South Wales. In order to stop the virus from spreading any further, the Australian Government destroyed one-and-a-half million chickens. Unfortunately, the highly contagious ND virus (NDV) has already been found on another chicken farm on the New South Wales central coast.

The Australian Government is very concerned about the problem, for if Newcastle disease should become endemic they will be unable to continue their ban on importation of poultry products from countries where ND is present. This importation of poultry products from countries with lower production costs will prove a serious challenge to Australia's own producers. A second very serious concern relates to the wild-bird population. If ND were to affect their wild birds, Australia may lose the very lucrative bird-watchers tourism that it now enjoys.

While Australia continues its fight to save its poultry industry and wild-bird population, Canada has also reported an outbreak of virulent ND in a flock of farmed ducks in Quebec. If uncontrolled, Canada stands to lose an international poultry market worth millions of dollars. The total cost of ND to its economy will depend only on the amount of time it takes Canadians to stamp out the disease.

**PROTECT YOURSELF AGAINST NEWCASTLE.**

**TAKE A MOMENT TO REVIEW BIOSECURITY IN YOUR FARM. LET'S NOT HAVE AN EXOTIC DISEASE IN NEW YORK STATE.**

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## POULTRY REALITY CHECK NEEDED

**Industry Veteran says the poultry industry faces a major disease crisis if present management practices continue.\***

The poultry industry in the United States has enjoyed decades of increasing progress and prosperity. Few in our industry have experienced a catastrophic outbreak of disease or endured the pain of seeing tens of thousands of dead birds hauled from houses in front-end loaders.

Those of us who have, as we did during the 1968 outbreak of Newcastle disease in the U.K., carry

it with us for a lifetime. The memories can't fail to serve as a reminder that the bacteria and viruses against which we do battle daily are ancient, opportunistic and crafty.

For those reasons, and in light of what we are seeing every day across the industry, it seems an appropriate time to step back, assess the evolution of our standards and practices and ask ourselves if we aren't cracking the door open for just such a disaster in the United States.

If we discover that this is true, and we believe that it is, we're apt to find that it was done bit-by-bit, procedure-by-procedure and operation-by-operation - driven by economics in the name of cost-cutting. Unfortunately, whether money is saved or not, biological principles do not change.

With the growing sophistication of computers, we as an industry have become increasingly obsessed by numbers. Pitting our scientists and managers against a constantly moving target of lower costs, we have demanded change in every phase of the business. Those who find a more efficient (i.e. cheaper) way to get a job done receive the praise, while those who preach caution, risk being seen as "out of step" or "unwilling to cooperate."

An examination of virtually all the changes made in the past decade shows that they've come in the guise of convenience and efficiency, but they are, in fact, cost-cutting measures. Few, if any, decisions have been made solely for the sake of avian health or the long-term protection of the industry. The balance between the two has been lost; the scale is now weighted almost entirely on the cost-cutting side. And, therefore, on the side of microorganisms - much longer on this earth than humans!

### Putting off paying the price

Until now, we have paid no great price for this unbalanced approach

to management. We have slept well believing that we can vaccinate our problems away. "Haven't we always?" we ask.

Adding to our lack of fear is the fact that warning signs do not emerge during day-to-day work within our individual arenas. Take the grower, for example. He sees no measurable problem because he's decreased the downtime in his houses. Nor does the hatchery manager feel he's taking a unacceptable risk by changing the delivery of vaccine from a slow, labor intensive method to something faster and more efficient. Our industry has not always helped - often turning a blind eye to the effect that antibiotics and bag systems may have on vaccine titers.

### Changes are cumulative

The warning that I'm laying on the table, however, is that we have taken too many small steps in too many areas without examining the situation from an industry-wide perspective. It's the cumulative effect of what we are doing that may be leading us to disaster. Later in this article, I will enumerate a list of changes, any one of which seems insignificant, but when viewed as a whole demonstrates clearly the dangerous imbalance between cost-cutting and health-support we've come to accept.

### Trusting technology

The larger risk we face because of the smaller risks we take is that our weapons against disease will lose their power. Take a look at the history of Marek's disease in the U. S. When there was no control in the late '60's and early '70's, we were accustomed to condemnation figures in the 10 to 20 percent range. Had that continued, there is little doubt that the industry would not be where it is today. But HVT Type III vaccine was developed and we grew accustomed to much lower figures.

However, HVT started to fail in the U. S. in the '80's. This loss of control was once again met by technology, with the introduction of Type II vaccines. Once again, no wide-spread, catastrophic outbreaks occurred and cost-cutting management practices continued.

In the early '90's, we began seeing changes in the Marek's virus. No longer so handily controlled by Type II and III vaccines, we turned to Rispons clones. Results were not encouraging.

Lately, we've turned to original Rispons CVI 988, which has been used in combination with Type III vaccines in Europe for many years. In layers and breeders, this combination appears successful due to clean house policies. However, the trend among pullet growers not to clean out houses may overshadow any protection benefits of the Rispons/HVT combination. In broilers where the combination has not been used widely overseas, the hoped-for panacea may fail to materialize under early challenge. Knowing that the vaccine has never been tested under U. S. conditions, few of us in the biologics industry will be surprised.

As I write this, even more disturbing changes are being observed in the Marek's virus. In the Carolinas and on the Shore, this strain shows the characteristic of early viremia against which the Rispons/HVT combo may not protect under the present management conditions. These Marek's isolates of the '90's have much quicker viremia than we have dealt with in the past. If these strains become the norm, there is no guarantee that anything in our arsenal can control them. We are firing all our bullets on Type I, II and III.

### Technology's shortcomings

It's comforting to assume that the skilled scientists in the biologics industry will race forth in the nick of

time with a vaccine that will once again save the day. With utmost faith in our ability to do whatever we wish without paying a price, we continue to take risks, putting off the day when the bill for our shortsightedness will fall due.

I am not alone in my belief that we may be closer to that day than we want to admit. Some of our best scientists agree that we may be running out of ammunition. Hard as we may try, no one will develop the perfect vaccine. If faced with a true crisis, we probably couldn't get a perfect vaccine licensed in time to save us even if we had it. That is the reality of the government regulations under which we now operate.

### Cost savings or risk taking?

Cost saving/risk taking changes are taking place across the board. Finger pointing is out of the question because they are occurring industry-wide. The biologics industry itself must shoulder some responsibility. For years we have urged producers to "leave disease control to us while you manage your companies for greater profits."

### What are we doing?

This list of practices is by no means complete but is included to illustrate how pervasive risky changes have become.

More to the point, it is included to illustrate the foolhardiness of comparing the industry of 1995 with the industry of 1960, 1970, or 1980. Our practices—dozens of them—have changed significantly and we must factor them in when making any assessment of risk.

### We're getting relaxed about procedures once considered essential

**Shorter downtime.** The financial advantages of shorter downtime are obvious. From allowing more growout periods/year to eliminating the need for more houses, this new

practice saves money. The price: When downtime is less than 14 days, cleaning and disinfectant programs are reduced; the opportunity for contamination is increased.

**Biosecurity.** We're getting relaxed about procedures once considered essential. Since it hasn't caused a problem yet, we think it never will. The price: We face far greater risks now than in the past. We are a world business. A man can be on a foreign farm in the morning and on a U. S. farm that afternoon. It is doubtful many questions would be asked at our farm gates.

**Litter quality.** Litter is a problem. Litter chips are expensive and it's hard to dispose of them or even get people to take them when they're discarded with today's increased ecological awareness. With the advent of the nipple drinker, litter stays drier, it looks better to the naked eye and we don't top dress as regularly as in the past. When looked at from a financial point of view, we've changed for the better. The price: In actuality, dry litter is a hospitable climate for bacteria and viruses, especially Marek's disease, allowing dander to circulate. While we've escaped consequences so far, the effect on just-vaccinated chicks could be catastrophic allowing for more virulent and rapidly multiplying strains of Marek's disease to emerge and survive.

**Vaccine delivery.** There has been a number of moves toward more economical routes of vaccine delivery. At the hatchery, Marek's was formerly administered from individual bottles; stirrers were used to maintain the consistency of titer within each dose. Now the vaccine comes from a common supply, which is both faster and less labor intensive. ILT deliver has moved from eyedrop to water, not because the water route is superior but because the labor cost is less. The price: Titrers are less consistent, not all birds are receiving the correct dosage. Hand-in-hand with a biologics industry that wants to keep customers happy, we have

changed from what's best for bird health to what works economically. We have had confidence in herd immunity, which has been enough in the past, but we're not addressing the reality that we face serious consequences if we fall below the critical mass.

**Summer programs.** It saves money to cut back on vaccines during the summer "when we don't need it." The price: Viruses don't recognize summer. By neglecting immunization, we take the pressure off and provide an opportunity for them to regroup. A number of "summer programs" has backfired this year, especially in areas of high Marek's and bronchitis challenge. The cost savings of summer programs is surpassed by mortality and performance loss.

**Vaccine dilution.** We've been doing it for years; we probably always will. The biologics industry has been complicitous, assuring growers that problems that crop up will vanish if they simply switch to another brand. And, of course, it appears to work for a time (while adequate dosages are reinstated). It is considered "standard" practice to push for a minimum protective dose. The price: How low can we go? How low should we go? We cannot possibly answer these questions accurately if we consider cost alone. It is our duty and responsibility to factor in the risk we assume by taking the pressure off the viruses.

### Time to take stock

These are only a few examples. It is the responsibility of each of us to look at our own segment of the industry and identify all the places we have sacrificed for the sake of savings. The overall total of how much we've changed should shock us into action.

### A change for the better

It would be impractical to suggest that we ditch every innovation of the last 10 years. No one in the real

world expects that to happen or would even recommend it. But here are things **WE MUST DO** if we are to protect the industry that has brought so much prosperity to so many.

**WE MUST RESTORE** the balance between science and economics. We cannot afford to continue every cost-cutting measure now in place and add new ones the minute they become available. If vaccines are being cut (or even occasionally discarded, as rumor has it), then more attention must be given to management programs and litter quality. If litter quality is compromised, we must be more diligent about proper vaccination. In other words, we must balance one decision against another, admitting that the 100 percent saving route is too dangerous to travel.

**WE MUST LOOK** at our practices from a company-wide perspective. No one thing we are doing spells trouble. Only by examining the totality of our choices can we assess the impact of each individual decision.

**WE MUST RESTORE** the balance of power between our scientists and our accountants. It is my opinion that many of our best scientists harbor the fears expressed here. If their voices have been weakened, they must be strengthened and their scientifically-based concerns respected, as they were before the numbers became such a driving force.

**WE MUST RESPECT** the viruses and bacteria that we fight. With the advent of penicillin, some scholars predicted that bacteria would be a thing of the past by 1990. How wrong they were. Anyone who reads the daily newspaper realizes that in human health, there are some areas in which we are in worse shape than ever. Tuberculosis is more rampant in some of our prisons and inner cities than in the Dark Ages.

There is a lesson here for our industry. The past two or three decades represent a fraction of a moment compared to the millions of

years the viruses we are fighting have managed to survive. It would be complacent and arrogant to assume that we can easily prevail against them because, for a brief point in time, we've managed to do it.

**WE MUST CONSIDER** the cost of catastrophe. Such words as "I don't care what it takes, we've got to get this stopped," are heard only after a problem has erupted. It is our responsibility to recognize that our past cost-cutting may have caused some of our most costly setbacks.

**WE MUST PRESERVE** the power of our vaccines and pharmaceuticals. It is better to err on the side of caution than to err on the side of optimism when relying on technology to develop more potent weapons.

### We must decide

Now is the time to decide. We can go on with business as usual, hoping for the best as we charge headlong toward lower costs. Or we can begin making the prudent moves needed to restore a balance between economics and long-range avian health. We can pay now or we can later. But it should be known and it must be said, one way or another we will pay.

[Article is by Ken Rudd, Merial-Select Laboratories, Inc., A Merck & Rhône-Poulenc Company and was printed with permission from Ken Rudd.]

### \*About Ken Rudd

Ken Rudd wears several hats in the poultry industry. He was trained as a microbiologist, obtained his first job with Glaxo in the UK and did research with human influenza, small animals and poultry which led to work in the production of avian and human vaccines.

His next job was with Duphar, then part of Phillips Electronics, which needed someone with experience to work in marketing and tech service. Another move carried

him to South Africa and Rainbow Chicks, where he was health services manager for Cape Province. Salsbury in South Africa needed technical service and sales help and Ken joined the company, which soon brought him to the U. S. as poultry production manager.

He spent 13 years with Salsbury Laboratories, including working in sales in California. He has been with Merial-Select Laboratories for ten years where he has been director of marketing and vet services. He also is global marketing manager for Rhône-Poulenc's avian business unit worldwide, which includes Merial-Select Laboratories, Inc., Gainsville, GA.

Ken has been in the poultry business since 1958 and has been in avian service on every continent. With these years of experience in which he has seen both the good and bad in poultry health, it seems appropriate for him to put together the accompanying words about the status of the American poultry industry.

**Broiler Performance Data (Region)  
Live Production Cost**

	SW	Midwest	Southeast	Mid-Atlantic	S. Central
Feed cost/ton w/o color (\$)	121.52	114.35	129.41	129.84	125.8
Feed cost/lb. meat (¢)	11.59	11.19	12.61	13.07	12.43
Days to 4.6 lbs.	45	45	46	46	46
Med. cost/ton (¢)	2.44	2.10	3.24	3.04	2.72
Chick cost/lb. (¢)	3.92	3.30	3.71	3.59	3.65
Vac. Med. cost/lb. (¢)	0.08	0.03	0.22	0.10	0.16
WB & 1/2 parts condemn. cost/lb.	0.25	0.24	0.31	0.34	0.41
% Mortality	4.42	4.49	5.58	5.71	6.32
Sq. Ft. @ placement	0.76	0.74	0.76	0.74	0.82
Lbs./Sq. Ft.	0.32	7.48	6.68	7.32	6.62
Downtime (days)	16	11	12	12	13
Data for week ending 4/17/99.					

**Broiler Performance Data (Company)  
Live Production Cost**

	Average Co.	Top 25%	Top 5 Co.'s
Feed cost/ton w/o color (\$)	126.33	119.26	112.32
Feed cost/lb. meat (¢)	12.29	11.38	10.97
Days to 4.6 lbs.	46	46	46
Med. cost/ton (¢)	2.81	1.68	1.30
Chick cost/lb. (¢)	3.84	3.47	2.98
Vac. Med. cost/lb. (¢)	0.71	0.03	0.04
WB & 1/2 parts condemn. cost/lb.	0.32	0.20	0.13
% Mortality	5.37	4.08	4.31
Sq. Ft. @ placement	0.76	0.75	0.71
Lbs./Sq. Ft.	6.68	6.78	7.39
Downtime (days)	13	13	11
Data for week ending 4/17/99.			

**Broiler Whole Bird Condemnation (Region)**

	SW	Mid-West	Southeast	Mid-Atlantic	S. Central
% Septox	0.344	0.427	0.178	0.411	0.379
% Airsac	0.162	0.087	0.455	0.311	0.551
% I.P.	0.062	0.048	0.194	0.175	0.244
% Leukosis	0.008	0.009	0.015	0.058	0.004
% Bruise	0.007	0.009	0.018	0.014	0.015
% Other	0.014	0.016	0.049	0.024	0.016
% Total	0.597	0.597	0.910	1.013	1.209
% $\frac{1}{2}$ parts condemnations	0.401	0.364	0.316	0.332	0.421

Data for week ending 4/17/99.

**Broiler Whole Bird Condemnation (Company)**

	Average Co.	Top 25%	Top 5 Co.'s
% Septox	0.347	0.270	0.190
% Airsac	0.332	0.141	0.095
% I.P.	0.151	0.070	0.022
% Leukosis	0.025	0.011	0.003
% Bruise	0.013	0.011	0.006
% Other	0.023	0.014	0.024
% Total	0.891	0.517	0.340
% $\frac{1}{2}$ parts condemnations	0.391	0.289	0.183

Data for week ending 4/17/99.

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## DEVELOPMENTS IN RESEARCH

The following are extracts of some of the papers presented at the 1999 Southern Poultry Science Society and Southern Conference on Avian Diseases in Atlanta in January 1999. \* Environmental enrichment can improve productivity and reduce fear, depression and other harmful states. In a study (Jones and Rayner, Rosline Institute, Scotland and National Farmer's Union, London), 100 poultry producers were asked if they played music to their flocks and if they have noticed any benefits.

The results of the study indicated that 46% of the producers routinely played music. Of these, 96% said that it calmed the chickens, 52% felt that the birds were less aggressive, 20% reported healthier birds, and 16% claimed increased egg production. About 90% of the farmers who had not played music would now consider it. Chart music and easy listening were the most popular whereas heavy metal and jazz were poorly rated. Investigators concluded that playing the radio to

chickens is an easy practicable way of enriching their environment, and perhaps, helping to reduce their fear of new noises.

\* Miles and Comer (University of Florida) reported the results of an experiment which was conducted to determine if feeding a diet with 2% sodium bicarbonate for one hour during the dark period (midnight feeding) would improve shell quality in aged laying hens selected for their ability to lay eggs with good and poor shell quality. From a

population of laying hens, 320 birds were selected based on poor and good eggshell quality (shell weight). The birds of each shell quality group were fed one of four dietary treatments. Three dietary treatments with 0, 1, and 2% sodium bicarbonate were used in this experiment. The birds of T<sub>1</sub> were fed a diet with no sodium bicarbonate during the 15 h daily photoperiod. Birds of T<sub>2</sub> were fed a diet with 1% sodium bicarbonate during the photoperiod. Birds of T<sub>3</sub> were fed a diet with no sodium bicarbonate during the 15 h of daily light and the same diet from 0100 to 0200 a.m. Birds of T<sub>4</sub> were fed a diet with no sodium bicarbonate during the 15 h of daily photoperiod and a diet with 2% sodium bicarbonate during 0100 to 0200 a.m. Feeding sodium bicarbonate at any time did not influence egg weight during the 7 day feeding. In the poor shell quality group, but not in good shell quality group, feeding a diet with 2% sodium bicarbonate for one hour (during midnight) increased shell breaking strength and the percent shell when compared to the hens not receiving sodium bicarbonate during the midnight hours.

\* Scheideler et al. (University of Nebraska) reported the results of an experiment which was conducted to determine the effect of feeding two sources of corn; normal corn (NC) and high available phosphorus corn (HAPC) on performance of hens from 20 to 40 wks of age. The HAPC contained .27% total P and .17% available P (AP), while the NC contained .23% total P and .05% AP. Diets were formulated to use these corn sources to contain AP levels of .4, .35, .3, .25, and .2% AP plus 500 units phytase. Feed consumption and egg production were not significantly affected by dietary AP or corn type. Egg weight and egg mass were reduced significantly as dietary AP decreased; diets with .2% AP plus phytase had equal egg weight and egg mass to .35 and .4%

AP diets. Specific gravity was not affected by dietary treatment but percent dry shell was improved at the lower AP levels and with phytase. Dietary AP level and corn type did not have a significant effect on bone ash. Excreta total P was reduced as the dietary AP was reduced, but was higher with HAPC than the NC. Total P, Ca, Zn, Cu, and Mn digestibilities were affected by corn type and AP level. HAPC reduced the digestibilities of these nutrients as compared to NC. This negative effect was alleviated by phytase supplementation of HAPC diets for Ca, Mn, Zn, and Cu. The authors concluded that while HAPC allows for less dicalcium phosphate supplementation in layers diets, it still has the potential to bind dietary cations which can be alleviated by dietary phytase.

\* Conjugated fatty acid (CLA) is a mixture of geometric isomers of linoleic acid (cis 9, cis 12 when the double bonds are counted from the COOH terminal or cis 6 and cis 9, when the double bonds are counted from the CH<sub>3</sub> terminal). Previous studies have shown that dietary CLA increased yolk saturated fatty acids (C16:0 and C18:0) and reduced yolk levels of monounsaturated fatty acids (C16:1 and C18:1). When CLA was fed to a low-fat diet, 100% of embryonic mortality of fertile eggs happened. Pariza and Cook (University of Wisconsin) conducted an experiment to determine if CLA-induced embryonic mortality can be prevented by increasing the dietary level of oleic acid (C16:1). Treatments consisted of; T<sub>1</sub>, 0.5% corn oil, T<sub>2</sub>, 0.5% CLA-90, T<sub>3</sub>, as T<sub>1</sub> plus 10% olive oil, and T<sub>4</sub>, as T<sub>2</sub> plus 10% olive oil. Diets were fed to laying hens for 2.5 months. Hens were inseminated and eggs were collected daily and stored at 60° F for 24 hours prior to incubation. After 6 days of feeding T<sub>2</sub>, percent hatch of fertile eggs was zero, whereas the percent hatch from T<sub>1</sub>, T<sub>3</sub>, and T<sub>4</sub> were 72, 87, and 57%, respectively. When hens fed T<sub>2</sub> were

put on T<sub>1</sub>, hatchability was increased to 100% within one week. The percentage of CLA as percentage of total fatty acids of yolk for hens fed T<sub>1</sub> to T<sub>4</sub> were 0.11, 1.91, 0.8, and 0.09%, respectively. The ratio of 16:0/16:1 and 18:0/18:1 in yolk of eggs from T<sub>2</sub> were increased by about 2- and 2.5-fold, respectively, when compared to T<sub>1</sub>. Olive oil prevents the CLA-induced increase of C16:0 and C18:0 and a decrease in C18:1 in yolk lipid, but not C16:1. The results indicated that embryo mortality associated with maternal ingestion of CLA was due to the increased ratio of saturated to monounsaturated fatty acids and not due to CLA.

\* Dietary conjugated fatty acid (CLA) has been reported to cause hardening of yolks and the pink discoloration of white in eggs stored at a temperature less than 60° F. The objective of this study (Pariza and Cook, University of Wisconsin) was to determine if increasing dietary monounsaturated fatty acids would prevent CLA-induced changes in egg quality. Four dietary regimens were used. They consisted of 0.5% corn oil (Diet A), 0.5% CLA (Diet B), 0.5% corn oil plus 10% olive oil (Diet C), and 0.5% CLA plus 10% olive oil (Diet D). Daily collected eggs were stored at 39, 60, and 70° F for three months. Eggs from hens fed the CLA diet (Diet B) had 17-fold increase in CLA level when compared to the control, 0.11 and 1.91%, respectively. Yolk from hens fed Diet D had 66% reduction in CLA compared to Diet B. The C18:0/C18:1 in yolk from hens fed Diet B increased three-fold compared to hens fed Diets A, C, and D. Eggs from Diet B had hardened yolk when stored at a temperature of less than 39° F. Diet B also caused a pinkish white when stored at temperatures of 39 and 60° F, but not those stored at temperature of 70° F. The addition of olive oil prevented hardening of the yolk and pink discoloration of the white. Yolk and albumen of eggs stored from group B for three months

had a pH of 7.89 and 8.32, respectively. Yolk and albumen pH of hens fed Diets A, C, and D were similar (6.08 and 9.04, respectively). Eggs from hens fed CLA increased iron content in the white as compared to the control, and feeding olive oil prevented the increase in white iron. The investigators concluded that olive oil prevented the adverse effect of CLA on egg probably by restoring the ratio of C18:0/C18:1.

\* Gomez et al. (University of Nebraska) conducted an experiment to determine the effect of energy level and enzyme supplementation on performance of laying hens. Hy-Line W36 were fed the experimental diets from 40 to 50 wk of age. The experiment consisted of a 3 X 2 factorial arrangement of the treatments with three levels of energy (1255, 1286, and 1320 kcal ME/lb) and two levels of enzyme (0 and .75%; Avizyme 1500; amylase/proteinase) supplementation. Egg production and feed consumption were not influenced by dietary energy or enzyme supplementation. Enzyme supplementation had a greater tendency for production of egg mass ( $P < .08$ ), body weight gain ( $P < .06$ ), and egg specific gravity ( $P < .08$ ), and had an improved feed conversion ratio ( $P < .05$ ) than hens fed a diet supplemented with no enzyme. Enzyme supplementation increased the egg mass by .88 g/day. Feed conversion ratio was the most efficient ( $P < .05$ ) for hens fed 1286 kcal ME/lb compared to the hens fed 1255 or 1320 kcal ME/lb (2.0, and 2.01, respectively). Egg components were not influenced by enzyme or energy supplementation. In summary, this age of hens were not responding to energy by feed intake adjustment. Enzyme supplementation increased egg mass and feed conversion at all energy levels.

\* Zhang et al. (Auburn University) conducted an experiment to determine if broiler breeder hens would respond to phytase supplementation in a manner similar

to Leghorns in increasing phytate P utilization. Four dietary regimens consisting of two AP levels (.1 and .3%) and two phytase levels (0 and 300 units/kg) were fed to individually-caged breeder hens from 27 to 60 wk of age. Phytase increased egg production by 2-4% as compared to hens fed no phytase. Supplemental phytase had no effect on egg weight or specific gravity. Phytase reduced mortality by 7%, regardless of AP levels used. Phytase increased bone quality with both dietary AP levels. The results indicated that phytase supplementation would correct production problems associated with a low-AP diet. More research is needed to study the effect of phytase on fertility and hatchability.

\* In birds, vitamin A is supplied by the yolk in the embryonic stage and must be replaced by an exogenous source after hatching. Although feedstuffs contain sufficient vitamin A to fulfill normal requirements for growth, there are several factors, such as mycotoxins, reoviruses, coccidial infections and nutritional imbalances which may still cause vitamin A depletion. This, in turn, can affect growth rate. Uni et al. (Hebrew University of Jerusalem) conducted an experiment to determine the effect of severe and mild vitamin-A deficiency and vitamin A-repletion on chicken intestinal functionality and morphology. Results showed 1.) lack of vitamin A decreased weight after 13 days on the deficient diet and that compared to mice and rats, chickens respond very quickly to vitamin A deficiency. 2.) Severe vitamin-A deficiency altered the small intestine of chickens both biochemically and morphologically. The activities of a number of enzymes involved in the digestion process were reduced due to vitamin A deficiency. 3.) The repletion group showed remarkable increase in mucosal enzymatic activity only 4 days after switching to the control diet. The results of this

study suggested that administration of higher doses of vitamin A during an intestinal disease may have a positive effect on damaged mucosa and may lead to a rapid recovery of mucosal functions. The results of another report (Uni and Sklan, Hebrew University of Jerusalem) indicated that rapid rate of intestinal villi (finger-like microscopic projections located in the wall of the intestine) growth was observed from day 16 of incubation and until day 7 post-hatch, although rate of development was different in duodenum, jejunum, and ileum. Also, disaccharidase activity was found two days before hatching and activity increased ten-fold two days after hatching. Furthermore, delayed access to the first feeding after hatch, depressed villi growth and brush border enzyme activity for several days and caused regression in mucosal development. The results of this study provided more accurate information on the pattern of the small intestine development and indicates that a progressive increase at a young age, both in the absorptive area and in mucosal capacity, for hydrolysis in the broiler chick.

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