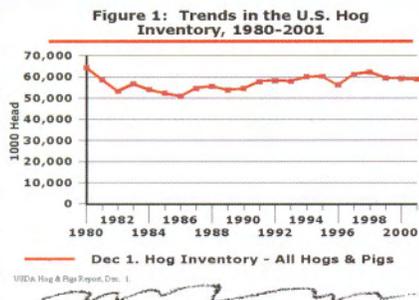


"Beyond the Hogs and Pigs Report...A Few Other Interesting Numbers Reported by NASS"

Dr. Laura Cheney, MSU Ag Economics

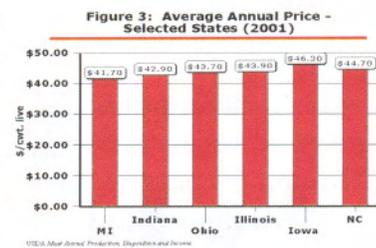
Many of us are familiar with the *Hogs & Pigs Report* published by the National Agricultural Statistics Service (NASS) of the USDA. Although once a quarterly report, NASS began collecting and publishing the document on a monthly basis in 2000. Most of the information in the monthly reports focuses on production numbers – for example, total hog inventory or total number of hog operations (see figures 1 & 2).



Many of us may be less familiar with another report issued by NASS, *Meat Animals Production, Disposition and Income*. Published yearly, this particular report is also loaded with tables and numbers and there is always interesting information found within the report. For instance, cash receipts from marketings of meat animals were more than \$50 billion in 2001. Approximately 76% of this amount (\$40 billion) can be attributed to the cash receipts of cattle and calves, 23% (\$12.5 billion) to hogs and pigs, and 1% (\$398 million) to sheep and lambs. Production numbers in 2001

were up for hogs and pigs, but down for both cattle and calves and sheep and lamb.

The *Meat Animals Production, Disposition and Income (MAPDI)* report also presents annual average prices per hundred pounds liveweight. NASS reports that the U.S. annual average price per 100 pounds liveweight increased from \$42.30 to \$44.30 between 2000 and 2001. Figure 3 shows this average annual price for Michigan (\$41.70) and other key states. Just as an aside, Hawaii's average price topped the list at \$83.80/cwt. and Alaska came in second at \$61.00/cwt. The lowest price reported was for New Jersey at \$35.60.



Inshipments of animals into States for feeding or breeding increased by about 2.4 million in 2001, from 24.5 million to 26.9 million. Michigan inshipped 280 thousand animals in 2001, up from 275 thousand in 2000. Iowa was the largest "importer" of animals, inshipping nearly 12 million animals in 2001. Iowa, Minnesota, Illinois, Missouri and Indiana were the only states to import over a million feeder pigs. Ron Plain (University of Missouri) reports that in 2001, about 27% of U.S. pigs were fed out in a different state than where they were born. In 1991, it was only 5% and only Iowa imported more than a million feeder pigs.

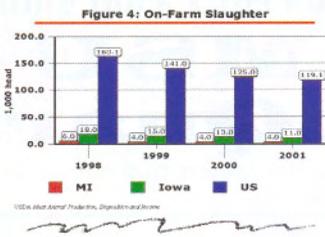
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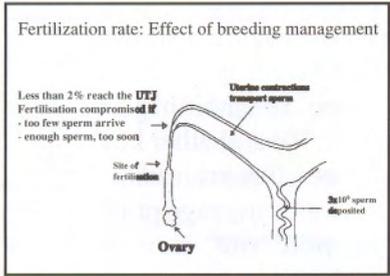
Numbers for farm slaughter, those animals slaughtered on farms primarily for home consumption, also show up in the MAPDI report. These numbers continue to decline. In 2001, just under 120,000 head were slaughtered on farm, almost a 150,000 decline from ten years earlier. Figure 4 shows on-farm slaughter for Michigan, Iowa and the U.S.



Overall, the *Meat Animals Production, Disposition and Income* report provides another tool for tracking structural change and trends in the U.S. pork industry. For those who enjoy analyzing numbers and who are interesting in knowing such trivia as which state has the highest average hog price, the report can be found each spring on-line at <http://usda.mannlib.cornell.edu/reports/nassr/livestock/>.

“Improving Fertility of Artificially Inseminated Sows”
Roy Kirkwood, DVM, Ph.D., Extension Swine Veterinarian, Michigan State University

In order to maximise sow fertility, current artificial insemination recommendations are that 3×10^9 live sperm be inseminated each time. The semen dose is deposited into the cervix and then the sperm have to reach the uterus, travel along the length of each uterine horn (approximately 3 ft), and finally cross the junction of the uterus with the oviduct (the uterotubal junction or UTJ). The sperm that reach the oviduct will form the sperm reservoir. Transport of sperm to the UTJ is performed by contractions of the uterine muscles (Fig. 1).



Why do we inseminate so many sperm? Of the 3×10^9 sperm deposited in the cervix, less than 2% will reach the oviduct. This is because during and after AI an average of 25% of the inseminated sperm will leak from the sow. Of the sperm that stay in the sow, some will be trapped in the cervix and most will be trapped in the uterus and destroyed by the sow’s immune system.

Once the sperm enter the oviduct they enter a resting phase until near the time of ovulation. During this resting phase sperm do continue to die, but at a relatively slow rate. However, if the time between sperm entering the oviduct and ovulation is too long, then too many sperm will have died and the low number left will cause a poor fertilization rate. Therefore, the aim for successful AI is to have a sufficient number of sperm in the sperm reservoir so that, even with a slow die-off, enough remain at the time of ovulation to maximize fertilization rate.

One reason for a small sperm reservoir at the time of

ovulation is poor timing of insemination due to poor estrus detection. For accurate estrus detection, and appropriately timed AI, there is no substitute for good boar contact. In the absence of a boar the ability to detect estrus, and the apparent duration of estrus in sows that are detected, will be reduced (Table 1). Good boar contact means that there should be at least 2 minutes of head-to-head contact while the breeding personnel apply back pressure to the sow. When reviewing boar contact, note the sow’s ability to make easy boar contact (eg. does the sow feeder size/position make contact difficult). Also, once a sow shows standing estrus, she will stand for only about 15 minutes and will then become refractory to boar stimulation. Therefore, it is usually recommended that the boar should be in front of no more than 5 sows at one time (or, alternatively, no more than the number of sows that can be bred in about 10 minutes). In addition to accurately detecting estrus, the presence of a boar during insemination stimulates uterine contractions and so sperm transport towards the sperm reservoir. If a boar is not present uterine contractions may be weak, leading to reduced sperm transport and a smaller sperm reservoir. This will reduce sow fertility.

	Boar present	Boar absent
Estrus detection rate (%)	90	52
Duration of estrus (d)	1.4	0.7

Hemsworth et al. 1984

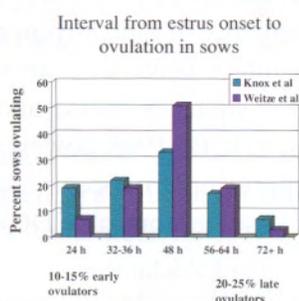
Having accurately detected the onset of estrus, it has been shown that an optimum fertilisation rate will be obtained by AI during the 24-hours before ovulation. If this is achieved, the farrowing rate and litter size are improved (Table 2). However, when does ovulation occur?

(Continued on page 3)

	Farrowing Rate (%)	Litter size (total)
Before ovulation		
24-36 hours	69	11.8
0-24 hours	92	13.2
After ovulation		
0-12 hours	76	12.3

Nissen et al. 1997

In an average sow population, ovulation can occur at any time from less than 24 hours to more than 72 hours after estrus onset (Fig. 2). Sows ovulating by 24 hours after estrus onset are called “early ovulators” while sows ovulating at more than 48 hours after oestrus onset are called “late ovulators”. This does not help us to decide when ovulation is likely to occur, but the distribution does give some information.



- You can expect about 10% of the sow population to be early ovulators and these sows will stand to be bred only once. The single mating of these sows is not a problem because the single insemination will be in the 24-hours before ovulation. Therefore, an analysis of records should show 5 to 15% of sows receiving only one insemination (in the PigCHAMP performance monitor, the percent multimate should be >85% and <95%). If, for example, 25% of sows are inseminated only once, then estrus detection must be evaluated. On the other hand, if only 1 or 2% of sows receive a single insemination, then breeding management is likely too aggressive (ie. sows are being bred after the end of estrus). If sows are bred late (ie. after ovulation) the eggs will be too old when fertilized and so fertility will be lower. Additionally, the ability of the uterus to combat infection is lower after ovulation. Therefore, a late mating is a risk factor for uterine infection, which will also result in poor fertility. A herd with a vulval discharge problem needs to urgently evaluate its breeding management.

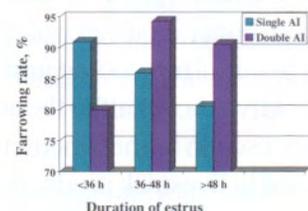
- You can expect about 20 to 25% of sows to be late ovulators. For maximum herd fertility, these sows should be bred on day 3 after estrus onset in order to have sperm deposited in the 24 hours before ovulation.

If a records analysis indicates only 5 or 10% of sows being bred on day 3, fertility may be reduced due to many sows effectively being inseminated too early relative to ovulation. However, if 40% of sows are bred on day 3 then breeding management may be too aggressive and fertility may suffer.

Although we know that the time of ovulation relative to estrus onset is variable, we also know that ovulation occurs at about 70 to 80% of the way through the estrous period. Further, we know that sows having a short wean-to-estrus interval (3 to 5 days) are likely to have a long estrous period (be late ovulators) and the breeding of these sows can be delayed if required. In contrast, sows having a long wean-to-estrus interval (>5 days) are likely to have a short estrous period (be early ovulators) and these sows should be bred immediately.

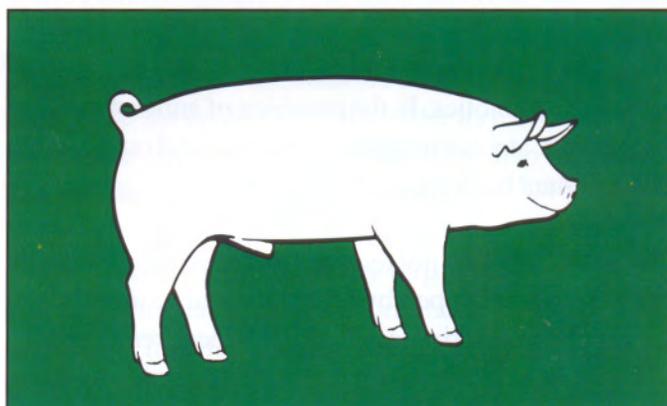
The effect on fertility of incorrect timing of AI is illustrated in Figure 3. Sows in this study were bred only once at 24 hours after estrus detection or received a second AI the following day. Some sows had a short duration of estrus (early ovulators) and a second AI caused a 10% reduction in farrowing rate.

Effect of short estrus duration and AI frequency on farrowing rate of sows



Tarocco and Kirkwood 2000

The longer the duration of estrus the more likely the sows were to be late ovulators. Therefore, for single inseminated sows, the longer the duration of estrus the longer was the interval between insemination and ovulation and so fertility was reduced. The conclusion is that maximum fertility requires good timing of insemination and good timing of insemination requires good estrus detection.



What is an antibiotic?

Many people have the idea that antibiotics are chemicals that are compounded in the laboratory. On the contrary, antibiotics are substances that are produced by certain bacteria and are found in nature. An antibiotic works by blocking a critical step of the bacterial life cycle. A strain of bacteria capable of producing an antibiotic has an edge in competition with other bacteria. Naturally, the bacteria that produces the antibiotic is resistant to its effects.

Scientists find new antibiotics by examining soil samples. They cultivate bacteria found in the soil and then check to see if the soil bacteria are able to inhibit the growth of bacteria that cause disease in animals. If so, the soil bacteria can be grown in large quantities, the antibiotic harvested from it, and processed into a commercially usable form.

How are antibiotics used?

The discovery of antibiotics changed the face of medicine, both human and veterinary, because antibiotics provided a powerful new tool against infections that previously were deadly. About half of the antibiotics produced in the U.S. are used in human medicine. Most of the other half are used in livestock production with a smaller amount used in fruit and crop production. In livestock, antibiotics are also used to treat clinical outbreaks of disease, but in addition they may be used at very low doses to improve growth and feed conversion.

What is antibiotic resistance and how does it develop?

Bacteria, like other organisms are constantly undergoing mutation. The vast majority of mutations are lethal, but occasionally a mutation occurs that confers some benefit and these bacteria survive. Some bacteria that mutate acquire the gene that gives them the ability to resist the effects of antibiotics. In the presence of antibiotics these bacteria are at a competitive advantage and outgrow the non-resistant bacteria.

The antibiotic does not technically cause the resistance, but allows it to happen by creating a situation where an already existing variant can flourish. Whenever antibiotics are used, there is selective pressure for resistance to occur.

How serious is the problem of antibiotic resistance?

It's a huge problem in human medicine. Antimicrobial resistance is becoming a factor in virtually all hospital-acquired infections. Many physicians are concerned that several bacterial infections soon may be untreatable.

Strains of *Staphylococcus aureus* resistant to methicillin and other antibiotics are endemic in hospitals. Infection with methicillin-resistant *S. aureus* (MRSA) strains may also be increasing in non-hospital settings. A limited number of drugs remain effective against these infections. Increasing reliance on vancomycin has led to the emergence of vancomycin-resistant enterococci, bacteria that infect wounds, the urinary tract and other sites. Until 1989, such resistance had not been reported in U.S. hospitals. By 1993, however, more than 10% of hospital-acquired enterococci infections reported to the Center for Disease Control (CDC) were resistant.

Streptococcus pneumoniae causes thousands of cases of meningitis and pneumonia, and 7 million cases of ear infection in the United States each year. Currently, about 30% of *S. pneumoniae* isolates are resistant to penicillin, the primary drug used to treat this infection. Many penicillin-resistant strains are also resistant to other antimicrobial drugs.

In sexually transmitted disease clinics that monitor outbreaks of drug-resistant infections, doctors have found that more than 30% of gonorrhea isolates are resistant to penicillin or tetracycline, or both.

Strains of multidrug-resistant tuberculosis (MDR-TB) pose a particular threat to people infected with HIV. Drug-resistant strains are as contagious as those that are susceptible to drugs. MDR-TB is more difficult and vastly more expensive to treat, and patients may remain infectious longer due to inadequate treatment.

Diarrheal diseases cause almost 3 million deaths a year – mostly in developing countries, where resistant strains of highly pathogenic bacteria such as *Shigella dysenteriae*, *Campylobacter*, *Vibrio cholerae*, *Escherichia coli* and *Salmonella* are emerging. Recent

(Continued on page 5)

outbreaks of *Salmonella* food poisoning have occurred in the United States. A potentially dangerous “superbug” known as *Salmonella typhimurium* DT109 resistant to ampicillin, sulfa, streptomycin, tetracycline and chloramphenicol, has caused illness in Europe, Canada and the United States. Throughout the human medical community, antibiotic resistance is a huge problem. It is common in outpatient clinics, is seen even more frequently in the general hospital wards and is found at the highest rates in the intensive care wards. Because antibiotics are one of the foundations of modern medicine, essential in surgery, cancer chemotherapy and organ transplants, the continuing rise in the number of bacteria resistant to antibiotics is a cause of great concern. The USDA reports that about 70% of bacteria that cause infections in hospitals are resistant to at least one of the drugs most commonly used to treat infections.

Its not a major problem in livestock

The range of antibiotics currently available for use in animals is relatively limited. Furthermore, most of those used in livestock production have been available for several decades yet they remain effective. Diagnostic laboratories regularly monitor the sensitivity to antibiotics of organisms isolated from sick animals and while some resistance has been found the situation is not nearly as critical as in human medicine.

How does the development of antibiotic resistance in animals fit together with its development in humans?

The Direct Route

People take drugs. The maximum likelihood of developing resistance in bacteria that infect humans occurs when people directly consume antibiotics. In fact over half of all the antibiotics sold in the U.S. are directly consumed by people. Most scientists agree that the increase of bacterial resistance to antibiotics in humans is largely the result of over-reliance on antibiotics in human medicine. According to the CDC, America’s 287 million people consume 235 million doses of antibiotics annually. The World Health Organization estimated that 40% of that use is unnecessary. And according to the Centers for Disease Control and Prevention, 20 to 50% of all outpatient antibiotic prescriptions and 25 to 45% of antibiotics prescriptions in hospitals are inappropriate

A study reported in the April, 1994, New England Journal

of Medicine, showed that increase in antibiotic resistance parallels increase in antibiotic use in humans. Researchers examined a large group of cancer patients given antibiotics called fluoroquinolones to prevent infection. Between 1983 and 1993, the percentage of such patients receiving antibiotics rose from 1.4 to 45%. During those years, the researchers isolated *E. coli* bacteria annually from the patients, and tested the bacteria for resistance to 5 types of fluoroquinolones. Between 1983 and 1990, all 92 *E. coli* strains tested were easily killed by the antibiotics. But from 1991 to 1993, 11 of 40 tested strains (28%) were resistant to all 5 drugs.

The Indirect Route Humans live in constant contact not only with animals and plants but also with bacteria. Bacteria are everywhere: in water and soil and in the bodies of humans and other animals. The vast majority of these bacteria do not cause disease, but they may not be completely innocuous. Disease-causing bacteria have frequent contacts with the so-called commensal or harmless bacteria from humans, animals, plants, fish, soil and water. These commensal bacteria can serve as reservoirs for resistance genes, collecting them and holding them for future transmission. It is possible that a commensal bacteria could transfer its resistance to a disease-causing bacteria.

Bacteria, in environments where antibiotics are used, are able to evolve and exchange genes that confer resistance to antibiotics. Through a circuitous route a bacteria that becomes resistant to antibiotics in a farm environment can come into contact with people. First the farm animal must be exposed to antibiotics and the bacteria that normally reside in its gut must develop resistance. Then at slaughter the intestine must be accidentally cut and the contents contaminate the carcass, survive washing and chilling and be present on meat in the supermarket display case. Then if the food preparer does not cook the meat the resistant bacteria could find its way to the intestine of a person. If it occupies a person’s intestine for a long enough period of time, it has the opportunity to transfer its resistance genes to disease-causing bacteria that may also be passing through the same person’s intestine.

How often does this actually happen?

Most scientists remain unconvinced that there is a significant transfer of antibiotic resistance or antibiotic-

(Continued on page 6)

resistant bacteria from animals to man. It is conceivable that the reverse may be true as in many cases resistance levels appear to be higher in bacteria isolated from people. In each stage of the food chain that occurs after livestock or animal products leave the farm, there is opportunity for cross-contamination from and by humans to the livestock products being produced and during preparation in the kitchen.

Data like that shown in the table below has been used to make the argument that resistance in animals is transmitted to humans. Sulfonamides and tetracycline are commonly used antibiotics in both swine production and in human medicine. The presence of these antibiotics in both pig and human intestinal tracts would favor the development of resistant bacteria. One study found striking similarities in the antibiotic resistance patterns of Strep bacteria from horses in Australia, the U.K. and the U.S.A. The authors concluded that the similarity in resistance patterns was most likely due to certain antibiotics being the treatment of choice for Strep infections in horses, because the horses in these distant countries would not have been likely to have contact with each other.

Occurrence of Resistance (%) among *E. coli* Isolated from Healthy Animals, Foods and Healthy Humans in Denmark (DANMAP 97)

Antibiotic	Broilers	Cattle	Beef	Pigs	Pork	Humans
Gentamicin	0	1	0	0	2	1
Kanamycin	0	0	0	0	8	12
Chloramphenicol	0	1	3	9	5	3
Trimethoprim	3	8	3	6	3	13
Ampicillin	2	1	5	10	30	19
Enrofloxacin/Cipro	0	0	0	0	0	1
Sulfonamides	7	7	8	47	19	31
Tetracycline	2	8	15	37	28	23

What needs to be done?

Before conclusions can be drawn about the contribution of antibiotic use in livestock to antibiotic resistance in human pathogens, reliable scientific data from further research are required on the following:

- The true frequency of transfer of antibiotic resistance from animal bacteria to significant human pathogens.
- The frequency of clinically significant circumstances where it can be demonstrated reliably that antibiotic resistance, which has originated in bacteria from animals as a result of the veterinary use of antibiotics, has caused clinical problems in humans.
- The degree to which animal pathogens, other than those such as salmonella which infect a broad range of hosts, actually colonize humans.

What are our government agencies doing about the problem?

Although livestock have been estimated to contribute less than 5% to the problem of antibiotic resistance in humans, the Food and Drug Administration (FDA) and its Center for Veterinary Medicine (CVM) would prefer this contribution to be zero and so have decided to make it one of their highest priority issues.

One step that the CVM is taking is to make it much tougher to get any new antibiotics approved for animals. CVM is considering whether sponsors should conduct studies on the potential impact of the use of antimicrobial drugs on “pathogen load” — the number of pathogenic organisms that are found in the animal’s intestinal tract.

The FDA is working with the Centers for Disease Control (CDC) and the United States Dept of Agriculture (USDA) to monitor the use of antimicrobials in livestock and antimicrobial resistance

In 1996, FDA, CDC and USDA started the National Antimicrobial Resistance Monitoring System (NARMS). NARMS uses data from the Centers for Disease Control and Prevention’s FoodNet system, which contains a random sampling of foodborne diseases that occur in humans around the country. It also uses the U.S. Department of Agriculture’s (USDA) Food Safety Inspection Service (FSIS) slaughterhouse samples, which give us information about pathogens in food from animals. Currently, NARMS collects data on *Campylobacter*, *Salmonella*, *Esherichia coli* O157, and *Shigella* and their susceptibility to 17 antimicrobial drugs. Results can be compared with data from previous years to look for changes in the resistance of the organisms to these drugs.

NARMS provides information on the rate of infections in humans including infections due to resistant foodborne pathogens, and information regarding the prevalence of resistant foodborne pathogens in animals. CVM can use these data to identify associations between the rate of resistant infections in humans and the prevalence of animals with resistant pathogens. NARMS and CVM have already accepted the assumption that rates of resistance in swine impact the rates in humans. While this may have some credibility for specific food borne pathogens, others will argue that because the problem of antibiotic resistance in humans increases at each step of the medical care system becoming more severe as patients move from the outpatient clinic through the hospital to the intensive care unit, other factors must be vastly more important in the development of resistance than consumption of pork.

“Pork Industry Outlook - Summer 2002”

Dr. Steve Meyer, Director of Economics, National Pork Board

The U.S. pork industry has been hammered by a number of factors this spring – most of which were unforeseen as recently as March. Price weakness was expected during the second half of this year but prices into the \$20/cwt live weight range were completely unforeseen during the spring and summer months when producers count on seasonal price strength to improve their financial position. Let’s re-cap what has happened so far in 2002 and what the remainder of the year might hold in store.

Price weakness since March has been the result of two major occurrences. The first, of course, was the embargo placed on U.S. chicken shipments by Russia. This caused chicken (especially leg quarters) to back up in U.S. plants and storage facilities. In turn, chicken prices dropped dramatically and U.S. retailers and foodservice operators found themselves in a position to leverage low-priced chicken against the prices of other meat items. Excess chicken supplies put pressure on wholesale pork and beef prices which, in turn, drove bids for hogs and cattle downward.

The second reason for this spring’s price struggles is that, beginning in April, hog supplies were 3-4 percent larger than was expected based on the March Hogs and Pigs Report. These supplies, when placed upon an already precarious wholesale demand situation, drove wholesale and live prices even lower. Packing capacity was not an issue in the price declines of this spring.

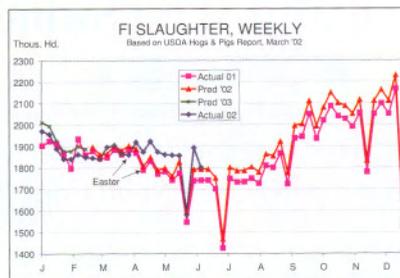
Recent weeks have seen some recovery from the price lows of late April. The hog price rally of the week ending June 7 was NOT supported by a wholesale product price increase. It appears that packers had booked sales and then found themselves short of the product needed to fill the orders. So, they chased pigs and squeezed margins. This theory was supported out by the rapid reduction in hog prices during the week ending June 14 as packers fought to get margins back up.

The most encouraging news in quite some time is the action in hog markets for the week ending June 21. As of this writing, terminal market live hog bids are above \$40/cwt live weight for the first time since the first week of March. And the rally is being driven by wholesale demand – cutout

values have improved by over \$5/cwt carcass weight since June 10.

In spite of the recent encouraging developments, though, I remain very concerned about price prospects for this fall. While a new read on hog supplies will come with USDA’s Hogs and Pigs Report on June 28 and Statistics Canada’s Pig Report in late July, I expect the slaughter projections for this fall to increase from the levels shown in the Weekly Federal Inspection Graph. Weekly slaughter totals in the 2.1 to 2.2 million head range are a definite possibility. These kind of supplies will push hog prices into the low \$20s and any upturns in weekly slaughter or reductions in slaughter capacity will result in capacity utilization rates high enough to drive prices into the \$10s.

What can be done? The number of pigs is pretty well set for the remainder of 2002. The weight of those pigs and the timing of their slaughter can still be affected, however. Professors Grimes and Plain of the University of Missouri have pointed out that producers should consider speeding up marketings in the third quarter by 4-5% and then slowing marketings in the fourth quarter back to “normal” levels. This action will move 2-3 days’ slaughter from the fourth quarter to the third quarter and take 4 to 5 pounds off of average slaughter weights. Both of these outcomes should stabilize the hog markets between the quarters and represent a win-win situation for producers and packers.



Additionally, producer should put a sharp pencil to their costs and financial position and consider just how long they can stand losses of various magnitudes. The recent cash rally has not affected lean hogs futures yet but producers should watch closely for pricing opportunities which, even if they don’t guarantee profits, reduce the amount of losses that may be incurred.

Finally, producers and their organizations must do everything they can to increase pork demand. Talking to retailers about featuring pork, helping with promotional efforts and supporting checkoff-funded programs of the National Pork Board and non-checkoff initiative of the National Pork Producers Council will all help. Be involved and be heard.

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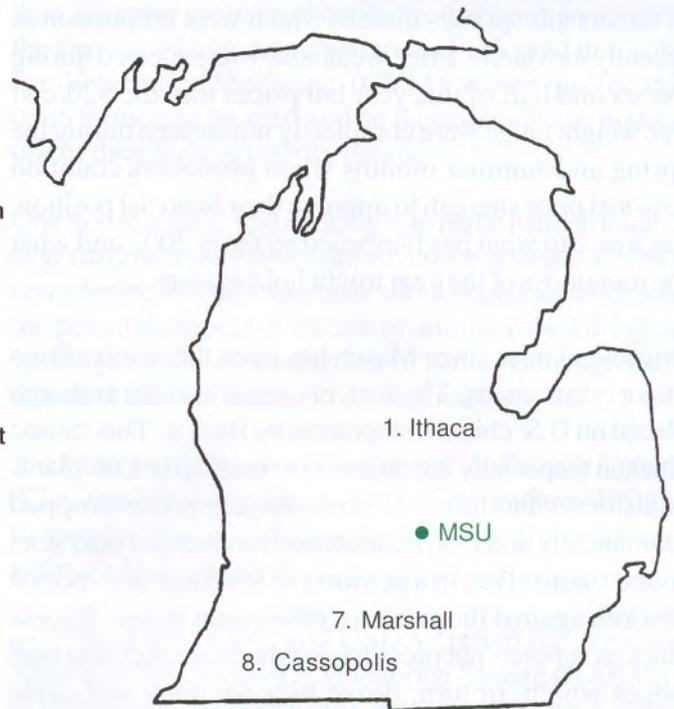
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All comments and suggestions should be directed to:

MICHIGAN STATE UNIVERSITY EXTENSION



Announcing the 2nd Annual Mid-West Swine Nutrition Conference

The Animal Sciences Departments of Michigan State University, the Ohio State University, Purdue University, the University of Illinois, and the University of Kentucky are working together to sponsor a conference for the swine feed industry. It will be held on September 4, 2002, at the Omni North Hotel in Indianapolis. Topics to be addressed include:

- A DVM Perspective of On-Farm Swine Nutrition
- New Developments Regarding the Pig's Need for Vitamins C, E and A
- Do High Levels of B-Vitamins? Nutrition and Pork Quality
- Effects of HACCP Regulations in the Feed Manufacturing Business
- Current State and Federal Environmental Regulations Regarding Animal Wastes
- Modifying Diets to Reduce Odor
- The Impact of Genetically Modified Corn, Soybean Meal and Pigs on Phosphorus Excretion
- Feeding Strategies to Enhance Growing-Finishing Pig Performance and Reduce Nutrient Excretion
- Mineral Mass Balance System

Attendance is open to anyone, including consulting nutritionists, swine practitioners and producer/nutritionists. The conference registration fee is \$100 per person through August 26 and \$150 per person after August 26 or on site. The URL for the web site is:

<http://www.ansci.uiuc.edu/conference/nutrition.htm> with more information and a registration page that may be printed, completed, and mailed.