Why Tracer Metal Amino Acid Chelates?

During the 1950's animal scientists first noticed that in spite of ingestion, frequently, trace minerals were not absorbed or metabolized. The minerals being put in the animals' mouths were being recovered in the feces. That observation stimulated research to ascertain the reason(s) for that malabsorption.

Scientists discovered that before the animal could absorb the mineral from an inorganic salt form, it had to be chelated or complexed in the stomach and intestines. This chelation process was very inefficient due to the numerous chemical reactions in the gut that tended to interfere with the chelation process and subsequent absorption. For example, in non-anemic monogastric animals, uptake of iron from the diet was reported to be as low as 3%.[1]

Scientists concluded that if the animal required minerals be chelated before absorption, why not pre-chelate them prior to feeding them. Animal scientist discovered how to make nutritionally functional chelates. This was the beginning of the manufacturing and testing of Amino Acid Chelates.

Discoveries were made showing that amino acid chelates could change the metabolisms of the animals. Performance limits originally prescribed were discarded as new data emerged. Reproduction efficiency improved. Immune systems were enhanced resulting in reductions in morbidity. Growth rates were accelerated. Feed conversions were improved. Newborn mortality was reduced. And the list went on.

The initial reaction to these observations was skepticism because synthetic EDTA chelates had already been shown to be generally ineffective in improving mineral nutrition.[2] Most nutritionists of the period erroneously considered all chelated minerals to be the same just as many of today's nutritionists mistakenly think that metal amino acid chelates, metal proteinates and metal amino acid complexes are the same because they are grouped together as organic minerals.

The early skeptics forced scientists to conduct additional mineral absorption and metabolism studies. Some of the more convincing studies used radioactive isotopes. Animals were fed single doses of radioactive amino acid chelates or inorganic minerals and subsequently sacrificed. The absorption and tissue deposition of the amino acid chelated minerals were then compared to that obtained with metal salts. Other isotope studies dealt with placental transfer of chelates or the targeting of minerals to specific tissue sites. Table 1 combines and summarizes some of the general metabolism data .[3]

Table 1. Radioactive mineral deposition in animal tissues from various mineral sources following a single oral dose of each (corrected counts/minute)

	^{45}Ca (7 day post tx)		^{65}Zn (4 hr post tx)		^{59}Fe (72 hour post tx)		^{54}Mn (14 day post tx)	
	Cl_2	AAC	Cl_2	AAC	Cl_2	AAC	Cl_2	AAC
Bone	3682	5772					350	780
Muscle	614	1206	2.41	3.88	2	54	800	660
Heart	642	932	6.42	6.32	63	151	370	1190
Liver	664	742	5.15	8.65	136	243	760	1070
Brain	698	804	1.22	2.41	31	130	620	1170
Kidney	686	730	5.45	8.55	2	327	470	600
Lung	676	648					720	330
Blood Serum	8	31			700	1797		
RBC	18	13			742	2076		
Whole Blood	27	44	0.90	1.64	1335	4215		

In the early days of trace mineral chelation, Dyer, of Washington State University, wrote, "The science of chelation as it relates to the nutrition of domestic animals is new. This phenomenon, much more than techniques heretofore used, offers possibilities of regulating the amount of a given metal at the cellular level."[4] Brady, et ah, of Michigan State University, in referring to their research with iron amino acid chelate added, "We found this material effective in maintaining pig hemoglobin when fed to the sow."[5] At the "International Pig Veterinary Society Congress" it was reported that fetal absorption of ^{59}Fe via placental transfer from the mother to the fetus was 5.7 times greater when she was given a single dose of iron amino acid chelate compared to ferrous sulfate.[6] Wolter, in France, studied iron fortification of animal feeds with various metal salts and then wrote that the iron amino acid chelate was more effective because his data showed that 55% of the dose crossed the placenta compared to only 2% from ferrous sulfate.[7] And finally, Professor Eric Underwood reported that amino acid chelates promoted piglet growth and hemoglobin formation with corresponding increases in liver iron stores in newly farrowed pigs.[8]

As metal amino acid chelates became accepted by nutritionists, companies began to offer their own forms of "organic minerals". In order to clarify these offerings, the Association of American Feed Control Officials (AAFCO) has defined seven general categories of organic minerals: Metal amino acid complexes, Specific Amino acid complexes, metal propionates, mHtba Chelates, metal polysaccharide complexes, metal proteinates, and metal amino acid chelates.

AAFCO stated that a metal amino acid complex "is the product resulting from complexing a soluble metal salt with a specific amino acid."[9] This definition requires that the metal ion be ionically or covalently bonded to molecules on the backbone, or in some instances a side chain, of a specific amino acid with the metal being at the end of

the chain. By the very nature of their chemical structures, metal amino acid complexes can have low stability constants. Complexes may not protect the mineral from involvement in absorption-interfering chemical reactions. Intestinal absorption of a metal amino acid complex has not been elucidated. Most biochemists believe, however, that minerals from amino acid complexes are absorbed similarly to inorganic mineral salts.

AAFCO has defined a metal proteinate as "the product resulting from the chelation of a soluble salt with amino acids and/or partially hydrolyzed protein."[10] This definition requires that a metal proteinate always contain partially hydrolyzed protein. Partially hydrolyzed protein means that the proteinaceous ligand must be larger than a peptide. Furthermore, if amino acids are present the definition requires that more than one amino acid form the other portion of the ligand. This makes the amino acid portion of the ligand a peptide because the definition requires more than one amino acid. Thus a metal proteinate must be a mineral that is chelated with both a small peptide (amino acids) and partially hydrolyzed protein or, if no amino acids are present, it is a metal chelated to partially hydrolyzed protein. Intact absorption of a metal proteinate is unlikely since metallic molecules that are larger than 1,000 daltons are not absorbed intact.[11] A metal proteinate may have to be digested before any part of it can be absorbed. Digestion of the proteinate increases the chance of releasing the mineral from the proteinate molecule and exposing it to interfering chemical reactions within the gut prior to absorption. Like the metal amino acid complexes, absorption of a true metal proteinate has never been elucidated. Since there is no uniform or reproducible molecular structure (because a proteinate employs an undefined peptide and/or undefined hydrolyzed protein), the task of describing how a metal proteinate is absorbed is extremely difficult.[12]

Chemically speaking it is highly questionable if a true metal proteinate can be formed as defined by AAFCO due to stereochemistry constraints. In order for a chelate to be formed (which a proteinate, by definition, must be), the ligand (chelating agent) must contain donor atoms and a heterocyclic ring structure with the metal as the closing member.[13] No company currently claiming to make metal proteinates has ever offered any scientific proof that its products are actually chelated though direct evidence of the ring formation and chelation bonds between the metal and the ligand.

AAFCO defines the metal amino acid chelate as "the product resulting from the reaction of a metal ion from a soluble metal salt with a mole ratio of one mole of metal to one to three (preferably two) moles of amino acids to form coordinate covalent bonds. The average weight of the chelate must not exceed 800."[14]This definition requires that the metal must meet the minimal chelation requirements mentioned above and be chelated with up to three individual amino acids which are the ligands. No peptides or hydrolyzed protein can be used. The definition specifies the type of bonding that must occur for the formation of a heterocyclic ring (a basic chemical criterion of chelation).

And finally, it limits the molecular size of the chelate which means an amino acid chelate does not require additional digestion in order to be absorbed. It is absorbed into the mucosal cell intact due to its small molecular size. Following mucosal absorption, most of the chelate is metabolized in the intestinal tissue and the minerals transferred to tissues and organs as they normally would be.[15,16] Metal amino acid chelates are the only one of the seven classes of organic minerals that has its absorption mechanism defined in the literature. Furthermore the metal amino acid chelates are the only chelates with structures that have been elucidated by electron paramagnetic resonance spectrometry (EPR), x-ray diffraction spectrometry, infrared spectrometry (IR), Fourier-transformed infrared spectrometry (FT-IR), nuclear magnetic resonance spectrometry (NMR), scanning electron microscopy (SEM), mass spectrometry (MS), photoelectron spectroscopy, and thermal decomposition.[17,18]

Because there are fundamental differences in the molecular structure of each of the organic mineral sources described above, it follows that there are also differences in their bioavailabilities under a variety of conditions. For example, in a beef cattle study copper bioavailability from an amino acid chelate , a metal amino acid complex manufacturer, and a proteinate manufacturer in the presence of copper antagonists was investigated. Three groups of 9 each, Angus steers with a mean weight of 318 kg were given 20 ppm of copper daily as an amino acid complex or as copper proteinate or as copper amino acid chelate in conjunction with 10 ppm Mo, 0.35% S and 750 ppm Fe for 120 days. At 0, 30, 60, and 120 days blood and liver samples were obtained from each animal and assayed for Cu, Fe, Mo, and ceruloplasmin activity. The copper antagonists initially affected absorption of all three sources of copper; however, by 60 to 90 days the animals receiving the copper amino acid chelate were able to overcome these effects whereas the copper complex or proteinate were still negatively affected. Only the copper amino acid chelate significantly increased ceruloplasmin activity ($p = 0.086$) and serum copper levels ($p = 0.08$) from the initial decreases of these two measures.[19]

In a split herd study, Black Pied Danish and Fresian cows were divided into two groups.[20] All of the cows received the same feed and feed supplements. As they calved, the heifer calves were given the same weaning and grower feeds except for the source of part of the mineral supplement. The treatment group received 0.04 g Cu, 0.08 g Mn, and 0.16 g Zn/kg feed as amino acid chelates whereas the other group received the same minerals as inorganic metal salts. These supplements were fed daily throughout the study period. The choice of minerals was based on the fact that copper, zinc, and manganese contribute to the development and function of superoxide dismutase (SOD) in the immunological system.[21] In the spring, all of the calves were bled for the first time and their IgM and IgG (IgGi and IgG2) immunoglobulins levels measured. (IgG and IgM immunoglobulins make up approximately 80% of all the immunoglobulins produced by cattle and thus are a reasonably reliable measure of the degree of potential immunity that the animal has developed.) Morbidity and mortality were also recorded from calving to that point in both groups in the spring. In the fall, the calves were again bled and IgM and IgG immunoglobulin measured. Morbidity and mortality from the date of the first bleeding to the second bleeding was recorded in both groups. Table 2 summarizes the results.[20]

Table 2. Immunity, morbidity, and mortality in calves fed Cu, Zn, Mn amino acid chelates or metal sulfates

	Inorganic Mineral	Amino Acid Chelate
Spring IgM	0.62 mg/ml[a]	1.10mg/ml[b]
Spring IgG	1.65mg/ml[a]	2.43 mg/ml[b]
Spring Morbidity	67%	11%
Spring Mortality	22%	2%
Fall IgM	0.91 mg/ml[c]	1.76mg/ml[d]
Fall IgG	3.75mg/ml	4.43 mg/ml
Fall Morbidity	54%	0%
Fall Mortality	17%	0%

[a] The differences are significant at p<0.05 [cd] The difference is significant at pO.001

At parturition, a certain amount of immunity is conferred on the calves by their mothers. As they grow older, their own immune systems begin to develop. Those calves receiving the amino acid chelates had 77.4% and 47.3% higher IgM and IgG values, respectively, in the spring than the inorganic mineral supplemented calves. In the fall, the IgM value for the chelate supplemented group was 93% higher than the inorganic mineral group. While both groups had higher IgM values in the fall compared to their spring values which is a normal result of a developing immune system, the chelate group improved 60% whereas the inorganic mineral group only improved 46.7%. Subsequent statistical analysis of covariance revealed that there were no differences between spring and fall values within treatments for either IgM or IgG ($P < 0.05$). The difference in the IgM values in the spring between the two treatments was significant ($P = 0.004$) as was the difference in the spring IgG values ($p = 0.042$). The difference in IgM values in the fall was also significant ($p = 0.001$). The difference in IgG values as a result of treatments was not significant ($p = 0.523$). These data make it very clear that feeding metal amino acid chelates can have a positive effect on the immune system which is also suggested by lower morbidity, lower mortality, and greater immune response when challenged by vaccinations, etc.

In a confirming study, researchers at Oklahoma State University fed 199 mg Cu, 296 mg Mn, and 699 mg Zn/day as either the amino acid chelate or as CuSC>4, ZnS04, and MnO to the university cattle herd for 75 days. At the beginning of the study, blood samples were obtained from each animal and erythrocyte SOD determined. This activity was statistically the same for both groups. At the conclusion of the study, blood samples were again

obtained and erythrocyte SOD determined. The mean erythrocyte SOD was 59.3 units/ml packed cells for the chelate fed cows and 56.8 units/ml packed cells for the inorganic mineral fed group. The 4.4% increase in SOD in the amino acid chelate group was significant ($p < 0.05$).[22]

Several investigators have reported improvements in milk production in dairy cows when amino acid chelates are included in the diet. For example, a herd of Holstein dairy cows with a rolling herd average of 23,500 pounds of milk at the start of the trial were divided into two groups. Both groups received a mineral supplement containing 0.95% Ca, 1.15% K, 0.34% Mg, 424 ppm Cu, 855 ppm Mn, and 1640 ppm Zn/kg feed as either amino acid chelates or inorganic mineral salts (K. was an amino acid complex in the chelate group). During the first and second lactations there were no significant differences between the two groups. By the third lactation, however, there was a significant difference in body conditions between treatments ($p < 0.027$) and a significant difference ($p < 0.005$) in milk production (79.4 pounds, inorganic versus 88.5 pounds, chelate). The reason for the difference by the third lactation period was due to a much higher body condition score as seen in Figure 1. The inorganic mineral group did not have a comparable body condition when compared to the amino acid chelate group. A lower body condition score negatively affects milk production in high producing herds."[23]

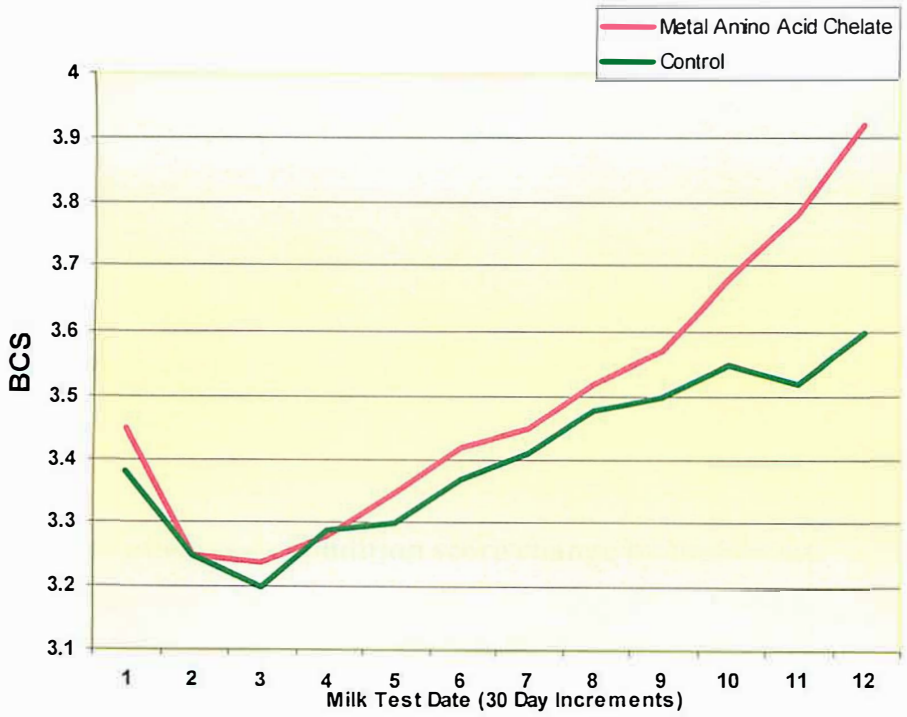

Figure 1. Third lactation body condition score change by treatment.

Milk quantity is only one aspect of milk production. Quality is equally important. Investigators at the University of Bologna also conducted a split herd study.[24] For 221 days, each group received a mineral supplement containing 23.4 mg Fe, 0.1 mg Cu, 4.8 mg Zn, and 4.4 mg Mn/kg of feed as either the amino acid chelate or as inorganic mineral salts. Consumption was regulated by hand feeding. The total average milk production was 2% higher for the chelate group. Protein content in the milk was significantly ($p < 0.01$) higher in the chelate group compared to the inorganic mineral group (3.06% versus 2.09% for the inorganic). Somatic cell count was significantly less ($p < 0.05$) in the chelate group (297.8 versus 326.8 for the inorganic).[24] Butterfat was significantly higher ($p < 0.05$) for the chelate group.In substantiating their findings on the effect of amino acid chelates on butterfat, Formigoni, et al., reported on a Wisconsin dairy where the chelates were supplemented, removed, and subsequently reintroduced. Figure 2 summarizes the influence of those chelates on butterfat production.

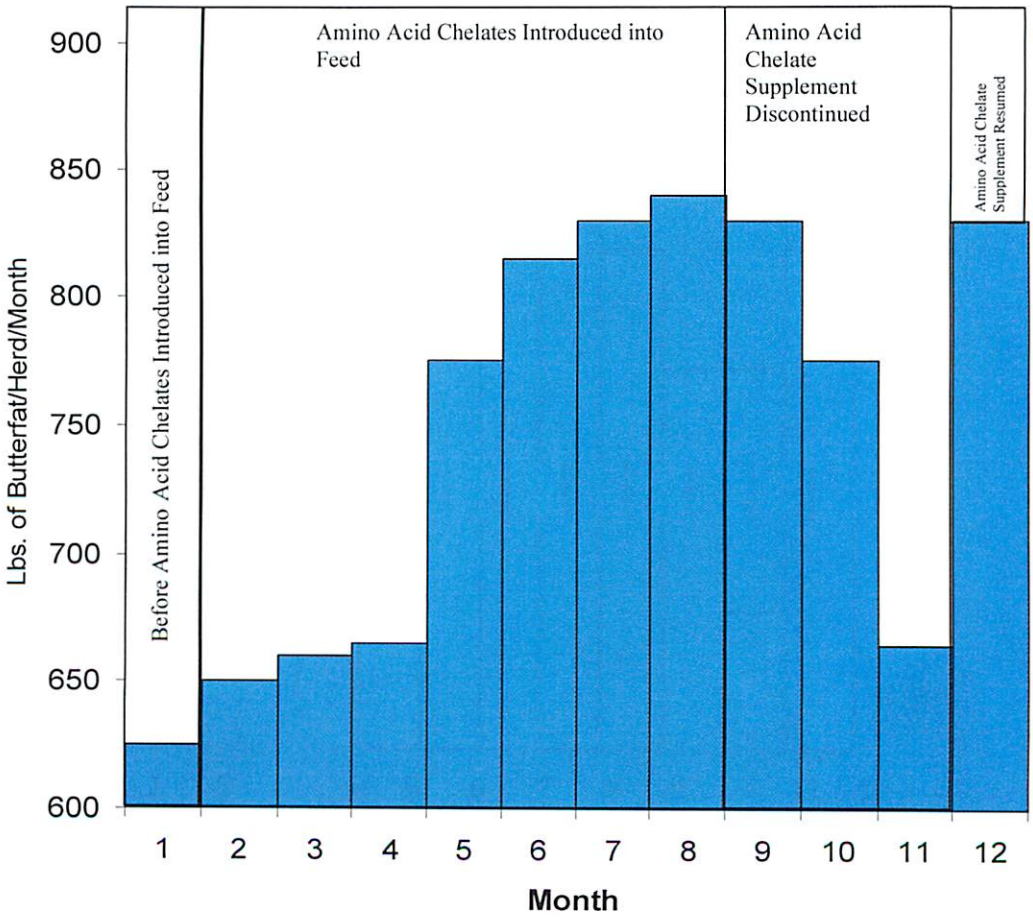

Figure 2. The Effect of amino acid chelates on butterfat production.

Reproductive activity was also recorded in this university herd study." The inorganic mineral group exhibited first heat 2 days earlier than the chelate group (54.7 days for the chelate and 52.9 days for the inorganic minerals). The period from parturition to insemination was one day longer (67.4 versus 66.4 days), but the number of first service pregnancies postpartum was significantly greater ($p < 0.05$) for the amino acid chelate group (45 for the chelate versus 15 for the inorganic group). Furthermore, the total number of days from parturition to conception was significantly less ($p < 0.05$) for the chelate group (102 days versus 127 days). And finally, the number of inseminations per confirmed pregnancy was less (2.0 for the chelate compared to 2.7 for the inorganic mineral source).[24]

In an attempt to understand why the metal amino acid chelates had such a positive effect on bovine reproduction, a study was conducted by investigators at the University of Maryland using the university herd. Forty pregnant heifers were divided into two groups according to expected parturition date. Beginning 30 days before expected calving, each animal received a mineral supplement as the amino acid chelates of Mg (2.4%), Fe (1.0%), Mn (0.5%), Cu (0.1%), Zn (1.4%), and K (4.5%) as an amino acid complex or equivalent amounts of metal as inorganic salts. The mineral supplement was top dressed on the feed of each cow. The percent of metal was the calculated percentage of mineral in the feed. From calving until 80 days postpartum, the amount of supplement administered/day was doubled. Second calf conception averaged 45 days earlier in the chelate treated group. Thirty days following calving 64% of the chelate group was ready to be rebred due to greater follicular activity ($p < 0.05$). Uterine tissue biopsies revealed that periglandular fibrosis was significantly less ($p < 0.005$) in the chelate group compared to the animals that received equivalent amounts of minerals as inorganic mineral salts. The fibrosis, or scarring of the endometrial tissue, is considered a pathological response when the tissue does not regenerate properly and affects conception. While the role of each mineral in the regeneration of endometrial tissue has not been fully elucidated, it is clear that there is a positive correlation between mineral bioavailability and tissue regeneration and that metal amino acid chelates with their greater bioavailability had a highly significant ($p < 0.005$) positive effect.[25,26]

Metal amino acid chelates have also resulted in significant metabolic responses in swine. Early studies demonstrated that when iron amino acid chelate was fed to gestating sows, their piglets were approximately 6.7% heavier at birth.[27] Widdowson reported that if fetal nutrition were optimum, the number of cells per tissue or organ would be greater and the mature size of each of these cells would be larger than if fetal nutrition were marginal.[28] Radioisotope research using amino acid chelates has shown significantly greater placental transfer of those minerals from the amino acid chelate sources compared to inorganic minerals.[6] This led to the deduction that heavier birth weights observed in piglets from mothers fed the amino acid chelates was due to better

fetal mineral nutrition. In a confirming study, investigators at Michigan State University assayed the tissues from 12 newborn piglets farrowed from sows that had received 50 ppm of supplemental Fe as either iron amino acid chelate or ferrous sulfate beginning three weeks before expected farrowing. Hemoglobin was 12.2% higher, plasma iron 9.8% higher, liver iron 34.2% higher, spleen iron 8.6% higher, and muscle iron 3.2% higher in the chelate group than in the sulfate group.[29]

Researchers at Iowa State University have reported that a 1 pound heavier pig at birth will result in a 7.78 pound difference at weaning.[30] Widdowson reported that this is due in part to the fact that cell numbers and mature cell size in tissues and organs from well nourished fetuses while larger at birth generally remain larger throughout post-natal life, if all other factors are equal.[28] To demonstrate this concept, in a split herd study, one half of the sows were fed 500 ppm of iron amino acid chelate (50 ppm Fe) daily during gestation while the other half received 50 ppm Fe daily as ferrous sulfate.[31] At birth piglets from sows fed the iron amino acid chelate were 6.7% heavier. All of the pigs in both groups were fed similarly from farrowing to market. Each piglet was weighed at weaning (4 weeks), at 9 weeks, and at 5 *V%* months when all were marketed. At 5 *V2* months, they were still over 5% heavier. The chelate group of pigs were heavier throughout life as Table 3 shows.

Table 3. Mean weights from birth to market of pigs from sows fed iron amino acid chelate or ferrous sulfate pre-parturition.

	Amino Acid Chelated Iron Fed Sows	**Ferrous Sulfate Fed Sows**	**Weight Difference**	**% Difference**
Birth weight	3.15 lbs 1.43 Kg	2.95 lbs 1.34 Kg	0.2 lbs 0.09 **Kg**	6.87%
4 week weight	15.00 lbs 6/82 Kg	14.40 lbs 6.55 Kg	0.6 lbs 0.27 **Kg**	4.2%
9 week weight	48.70 lbs 22.14 Kg	46.20 lbs 21.00 Kg	2.5 lbs 1.14 **Kg**	5.4%
5 *Vi* month weight	219.70 lbs 99.96 Kg	208.50 lbs 94.77 Kg	11.2 lbs 5.09 **Kg**	5.4%

Larger pigs at farrowing have lower mortality rates. Based on a 1948 litter study, investigators at Iowa State University concluded that piglets weighing under 2 pounds (0.9 Kg) have a 42% survival rate, whereas piglets weighing 3 pounds (1.4 Kg) or more have an 82 to 88% survival rate, all other factors being equal.[30] Feeding pregnant sows amino acid chelates during gestation will reduce piglet mortality because the piglets are generally larger at birth. For example, a group of second parity sows were divided into three groups.[32] The first group of sows, the control group, did not receive any supplemental iron during gestation and their piglets received a supplemental iron from

farrowing to weaning. The second group of sows did not receive supplemental iron during gestation but their piglets were injected with iron dextran at 1 day post-farrowing. The third group of sows received 250 ppm of supplemental iron as the amino acid chelate daily for the last 30 days of gestation, but their piglets did not receive any additional supplemental iron from farrowing to weaning. All sows received the same gestation feed which met NRC requirements except in the chelate group, which was were given the additional 250 ppm of iron amino acid chelate (25 ppm Fe) as noted above. At farrowing the piglets from the sows that were fed iron amino acid chelate weighed 4.6% more than the control piglets and 6.03% more than the piglets that subsequently received iron dextran injections. The heavier birth weight of the chelate group is probably a result of the additional iron the pregnant sows received and transferred to their fetuses during the last 30 days of gestation. At weaning they were still 18.1% heavier than the controls which received no supplemental iron from farrowing to weaning and 5.4% heavier than the group which were injected with iron dextran. The mortality in the group receiving no supplemental iron, was 20.1%. The iron dextran group had a mortality rate of 18.3% and the piglets from the mothers that had been fed the iron amino acid chelate had a mortality rate of 6.5%. The heavier birth weights had a significant ($p < 0.05$) and positive effect on piglet mortality even though the piglets from the chelate supplemented sows were marginally anemic when their hemoglobin levels were compared to the iron dextran group. The control group was very anemic. This study demonstrated, however, that piglet mortality was more a function of piglet birth weights than hemoglobin levels, and the birth weights were affected by the feeding iron amino acid chelate during gestation.

When fed to sows, metal amino acid chelates have been shown to influence conception, reproduction, and lactation. In a series of studies conducted at the University of Bologna, the effects of the metal amino acid chelates compared to inorganic mineral salts were investigated. In the first study, 50 gestating sows were divided into 2 groups of 25 each. At 3 weeks before expected farrowing and continuing through lactation, each animal received 5% Mg (chelate or oxide), 12.5 mg Fe (chelate or sulfate), 2.5 mg Mn (chelate or oxide), 6.0 mg Zn (chelate or sulfate) and 0.3 mg Cu (chelate or sulfate)/kg feed. As Table 4 shows, in the group receiving the amino acid chelates there were less stillbirths, fewer open days to estrus, and a higher conception rates on the first service, all of which have a significant economic impact on swine production. Subsequent statistical analysis of the data in Table 4 indicated that all of the differences were highly significant.

Table 4. Reproductive performance in sows fed metal amino acid chelates or inorganic mineral salts

	Inorganic Minerals	Amino Acid Chelates	% Improvement
Mean number of stillbirths per	1.3^{a}	$\mathbf{0.7^{b}}$	46.2%
litter Mean days to recycle to	7.5^{a}	$\mathbf{5.0^{b}}$	33.3%
estrus Mean % conception on first	73.0^{c}	$\mathbf{94.0^{d}}$	28.8%

The differences were significant at $p<0.05$ The differences was significant at $p < 0.10$

This first study was followed by a second study. Sixty sows were split into two groups of 30 each and fed 300 ppm of iron from ferrous sulfate or 100 ppm or iron from iron amino acid chelate beginning at conception and continuing through gestation, lactation and rebreeding. The group receiving the amino acid chelates lactated one day longer than the sulfate group and had 3.9% more weaned pigs at 26 days. The open days between weaning and estrus were the same: 8.32 versus 8.33. The number of artificial inseminations/conceptions, however, was 20.3% less for the chelate group (1.82 sulfate and 1.45 chelate). This resulted in an increase of 2.21 births per year for the chelate group compared to 2.04 for the sulfate group. Factoring in the 7.8% lower births per year with the higher piglet mortality in the sulfate group, the piglets weaned/year was projected to be 16.32 for the sulfate group and 18.17 for the iron amino acid chelate group, an 11.3% increase which is not only significantly greater but also economically greater.

Switching emphasis from the effects of the metal amino acid chelate on swine reproduction to meat production, when compared to other manganese sources, manganese amino acid chelate is the only source of supplemental manganese that has been proven to significantly reduce back fat and increased lean muscle in pigs.[33,34] A series of 7 studies demonstrated that feeding 20 ppm of manganese as amino acid chelate daily to growing pigs was 14% more effective in producing lean pork than 100 ppm of manganese from manganese sulfate. The mean P2 fat depth was 14.33 mm for the sulfate group compared to 12.33 mm for the amino acid chelate. One of these studies used 35 pigs, each having an initial weight of approximately 31 kg, and which were divided into 3 groups. One was a control group.

The second group received 20 ppm of Mn as the amino acid chelate. The third group received 20 ppm Mn from another chelate source. All pigs were fed these manganese sources for 65 days. At butchering the amino acid chelate group had 8.2% less fat than the control group and 3.7% less fat than the other chelate group. Only the manganese amino acid chelate produced grade 1 pigs. The control group and the other manganese chelate group produced grades 2, 3, and 4 pigs. There were no grade 4 pigs in the manganese amino acid chelate group whereas there were grade 4 pigs in both the control group and the other chelate group. Due to leaner meat, the average price/kg deadweight of the amino acid chelate group was 4.7% more than for the control group and about 1 % more than for the other chelate group which gave a net increased return on investment of 326% for the manganese amino acid chelate group and 219% for the other chelate group when they were compared to the control group.[34]

While feeding the manganese amino acid chelate became a commercial success with the feed company that conducted the studies, no statistical analysis was reported in the above trial. Nevertheless, when the research was duplicated by French researchers who focused on the statistical side of the study, they reported that there was no significant difference in feed consumption or growth rates between groups. The differences in fat deposition based on treatment differences were, however, highly significant ($p < 0.004$).[35]

In summary, the effects of amino acid chelates on animal production represent the degree of difference between metal amino acid chelates, metal amino acid complexes, and metal proteinates and the others in the organic mineral catagory. Marketing can concoct wonderful stories about the products being similar or which product is the cheapest or what a non-scientific field trial did. Several companies claim to sell much cheaper products that are "just like" the Amino Acid Chelates chelates except they don't meet the critical chemistry requirements to enjoy the benefits that true amino acid chelates provide. The problem with these stories is that the animal can't read or understand the sales pitch, but its body knows the difference between a mineral that is already set to work versus one that must receive special handling and modifications. The measure of the value of an amino acid chelate is in the performance of the animal. It is in that arena that the metal amino acid chelates made by NuTech stand alone. When the amino acid chelate replaces the "proteinate" or amino acid complex or inorganic mineral, animal performance is almost always improved. This is seen in lower mortality, better feed conversion, improved reproduction, faster growth rates, etc.

In the final analysis, metal amino acid chelates are the least expensive mineral in the market place because the return on the investment is the greatest.

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