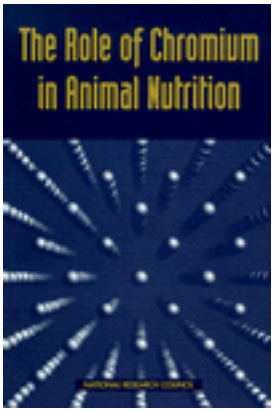

The Role of Chromium in Animal Nutrition

Committee on Animal Nutrition, National Research Council

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THE ROLE OF CHROMIUM IN ANIMAL NUTRITION

Committee on Animal Nutrition

Board on Agriculture

National Research Council

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Preface

The United States feed industry manufactures thousands of tons of supplements and feed additives each year. It has been suggested that chromium should become a constituent in certain nutritional supplements for animals. Chromium sources, other than chromium picolinate (CrPic) for swine, however, have not been approved officially as feed additives for food-producing animals in the United States mainly because there has been a lack of consensus among animal nutritionists that chromium is an essential nutrient and because scientifically based dietary requirements for chromium have not been established for food-producing animals.

Chromium is active biologically as a component of glucose tolerance factor (GTF), which enhances tissue sensitivity to insulin and glucose utilization. Results of research and clinical investigations have shown that human patients who receive parenteral nutrition and those who are type II diabetics respond favorably to chromium supplementation. There also is evidence showing that dietary chromium is beneficial for people undergoing physical or metabolic stress.

Although definitive data showing the effects of dietary chromium on metabolism, health, and performance of food-producing animals are relatively meager, sufficient evidence is available which, when taken together with observations made with humans, indicates that chromium may be an essential nutrient for animals. Some research results also indicate that chromium supplementation of practical diets of food-producing animals is beneficial. Because of the uncertainties associated with the role of supplemental chromium in animal diets, the Board on Agriculture's Committee on Animal Nutrition of the National Research Council conducted a review and an evaluation of the scientific literature on the use of

supplemental chromium in diets of livestock and laboratory animals. The committee's charge was to conduct a thorough review of the scientific literature on chromium, determine whether chromium should be classified as an essential nutrient, and analyze the effects of chromium as a supplement of practical animal diets. A steering committee, composed of members of the Committee on Animal Nutrition, was formed in April 1996. The following individuals were responsible for the respective sections of the report: Donald C. Beitz and Ronald L. Horst, introduction and metabolic role of chromium; Karin M. Wittenberg, ruminants and horses; Jerry L. Sell, swine and poultry; George C. Fahey, laboratory animals and rabbits; and Delbert Gatlin III, fish.

The report begins with a discussion of the absorption, transport, and deposition of chromium in humans and animals in Chapter 1. Current knowledge of the role of chromium in metabolism of humans and animals is described in Chapter 2. Chapter 3 provides a comprehensive review of data describing the effects of supplemental dietary chromium on cattle, sheep, swine, poultry, horses, rats, rabbits, and fish. The information reviewed for each species is summarized from the perspective of whether there is sufficient evidence to conclude that chromium is an essential nutrient and whether supplementation of practical animal diets with chromium is needed.

Jerry L. Sell, *Chair*
Steering Committee

Acknowledgments

The Committee acknowledges many scientists who provided input during the report preparation process. Numerous individuals supplied information and materials that were used during the Committee's deliberations. We extend special thanks to Nancy Arth for her assistance with preparation of the report manuscript. In addition, the secretarial assistance of Ann Shuey and Mary Cochran, Iowa State University, is gratefully acknowledged.

Contents

TABLES

The Role of Chromium in Animal Nutrition

Executive Summary

For approximately 40 years, chromium has been considered by many nutritionists as an essential nutrient for humans and animals. Chromium, which exists in nature mostly in the trivalent form (Cr^{+3}), is thought to be essential for activating certain enzymes and for stabilizing proteins and nucleic acids. Its primary role in metabolism, however, is to potentiate the action of insulin through its presence in an organometallic molecule called glucose tolerance factor (GTF). Evidence for the importance of chromium has been obtained primarily from research and clinical investigations with humans and laboratory animals. People who receive parenteral nutrition and those who are type II diabetics respond well to chromium supplementation. Research also has shown that supplemental dietary chromium is beneficial for humans and laboratory animals undergoing various stresses.

Research with animals has confirmed that chromium from dietary organic complexes, such as chromium picolinate (CrPic), chromium nicotinate (CrNic), and high-chromium yeasts, is absorbed more efficiently than is chromium from chromium chloride ($CrCl_3$). Some research data indicate that chromium is an essential nutrient for food-producing and laboratory animals. Responses of animals to chromium supplementation of practical diets, however, have varied, and the role of chromium in animal metabolism has not been clearly established. Presumably, chromium would function as a component of GTF, as suggested for humans and laboratory animals.

RUMINANTS

Bioavailability of chromium contained in commercial feeds for ruminant animals is not known. Further efforts need to be directed toward the determination of chromium concentrations and chromium bioavailability in feedstuffs and chromium supplements offered to ruminant animals. A range of chromium supplements including CrNic, $CrCl_3$, CrPic, chelated chromium, and high-chromium yeast have been used in ruminant studies. Few comparative studies have been conducted, and thus, little is known about the relative bioavailability of these sources for ruminants. Performance responses observed to date are limited to work conducted with CrPic, high-chromium yeast, and chelated chromium.

The literature does not support a general recommendation for chromium supplementation of commercial ruminant diets. Research efforts, however, have identified two situations in which chromium supplementation might have commercial application: newly arrived feedlot cattle and first lactation dairy cattle during the transition period.

Three out of eight studies conducted with newly arrived feedlot cattle subjected to the stresses of transportation, mixing, and handling have demonstrated a positive performance response in the initial weeks following arrival. Lower morbidity and plasma cortisol concentrations due to chromium supplementation have been reported; however, the responses have not been consistent. Efforts to establish enhanced immunity in response to vaccination or to foreign proteins have not shown consistent results, although blastogenesis in peripheral blood mononuclear cells cultured with T-lymphocyte mitogens was greater in cattle fed chromium-supplemented versus unsupplemented diets.

The transition period, including late lactation, parturition, and early lactation, is known to cause metabolic stress in dairy cows. There is evidence that chromium supplementation during this transition period can improve performance of first-lactation cows. This response was not observed in multiparous cows offered similar levels of supplemental chromium. Improved performance may be associated with a shift in ketone body metabolism, because lower circulating ketone concentrations in cows consuming chromium-supplemented diets have been observed in several studies. There also is evidence that chromium provided to first-lactation, postpartum cows will decrease sensitivity to insulin. As was observed with stressed, newly arrived feedlot animals, blastogenic responses of the peripheral blood mononuclear cells obtained from chromium-supplemented, early lactation cows were enhanced.

Controlled studies will be required to establish the specific role of chromium in cattle undergoing stress. These studies are essential to establish recommendations for rates of chromium supplementation where its use may be appropriate.

Work involving sheep suggests that chromium supplementation exerts subtle effects on carbohydrate and lipid metabolism. The significance of these effects, however, is not clear.

NONRUMINANTS

Responses of growing-finishing swine to supplemental dietary chromium have been inconsistent. Improvements in growth rate of swine as a result of supplementing diets with 200 to 500 µg Cr/kg as CrPic or 500 µg to 5 mg Cr/kg as $CrCl_3$ were reported in 11 of 31 studies. Similarly, feed efficiency was improved by chromium supplementation of diets in 8 of 31 studies. Favorable effects of added chromium on selected carcass traits have been observed. When supplemental dietary chromium was used, increases in carcass leanness (muscling) were reported in 9 of 24 experiments and decreases in carcass fat were observed in 11 of 26 experiments. Research to determine the influence of added dietary chromium on reproductive performance has been meager, and the results have varied (litter size was increased in one experiment, but reproductive traits were unaffected in a second study). Information on the metabolic changes that may be caused by supplementing swine diets with CrPic also is limited. Three experiments provide evidence to suggest that supplemental CrPic induces a hypoglycemic response or improves insulin efficiency in swine. *Although responses of swine to supplemental chromium have been inconsistent, there is an increasing amount of evidence indicating that chromium may favorably alter metabolism of swine under some circumstances, with resultant improvements in growth rate, carcass traits, and reproductive performance. The need for chromium supplementation of practical swine diets, however, depends on the chromium status of the animals, the amount of bioavailable chromium in the feedstuffs, and exposure of the animals to certain environmental stresses. Thus, a decision to use supplemental chromium in practical swine diets must be based on the potential benefits in individual circumstances versus the cost of supplementation.*

Evidence has been obtained that supplemental chromium at 20 mg/kg of diet as $CrCl_3$ increases the rate of glucose utilization by livers of chicks and poults in vivo and in vitro. The effects of added dietary chromium on growth rate and feed efficiency of growing poultry have differed, with improvements reported in 4 of 11 experiments. An improvement in performance of young poultry was observed when $CrCl_3$ was used to supply 20 mg Cr/kg of diet. Supplemental dietary chromium as CrPic also has been reported to decrease mortality and cholesterol in serum and egg yolks and to alter glucose metabolism of chickens. Research with poultry has shown that supplemental dietary chromium can be used to alleviate some of the toxic effects of vanadium in growing chicks and laying hens.

The evidence available today, although meager, indicates that supplemental dietary chromium can affect metabolism and well-being of poultry. Additional information is needed, however, to describe the circumstances in which supplemental dietary chromium can be used to greatest advantage.

A limited amount of research has been conducted to determine the effects of chromium supplementation on performance and metabolic responses of horses.

The meager amount of research conducted with horses has not shown any definitive benefits of dietary chromium supplementation.

Chromium has been shown to be essential for glucose metabolism in rats under controlled experimental conditions in which body stores of chromium were depleted. Mainly on this basis, chromium was addressed in the National Research Council's publication, *Nutrient Requirements of Laboratory Animals*, as a potentially beneficial dietary constituent for rats. A dietary requirement per se, however, was not given, and no benefit from chromium supplementation of practical rat diets was indicated.

Research with rabbits indicates that cholesterol and plaque content of the vascular system was decreased by supplemental dietary chromium and that chromium metabolism can be modified, depending on the dietary carbohydrate source. There is, however, insufficient information on which to base conclusions or recommendations concerning the need to supplement rabbit diets with chromium.

Investigations into the influences of supplemental dietary chromium on fish have been limited. Some studies report no effects of dietary chromium supplementation on growth or tissue chromium distribution in several fish species. Other studies, however, show that chromium supplementation of diets, especially those containing glucose, causes significant increases in weight gain, energy deposition, and liver glycogen of fish and alters postprandial plasma glucose concentrations. Thus, it seems that chromium plays a role in glucose metabolism of fish as has been reported for some other animals and humans. Specific mechanisms by which chromium influences dietary carbohydrate utilization of fish, however, have not been elucidated.

CONCLUSIONS

The Committee on Animal Nutrition has reached the following conclusions.

(1) It is not possible to make specific recommendations as to dietary form and concentration of chromium supplementation for cattle, poultry, and swine because

- *there are insufficient comparative data for the determination of relative bioavailabilities of chromium from supplemental sources;*
- *only meager data are available from titration studies designed to determine supplemental chromium concentrations that are most effective for cattle, poultry, and swine; and*
- *there have been no studies designed or conducted to determine dietary chromium requirements of cattle, poultry, or swine.*

(2) Supplementing practical diets with trivalent sources of chromium might be beneficial for the health and well-being of cattle during times of stress; however, the factors that affect the efficacy of supplemental chromium and the dietary chromium concentrations required have not been determined.

(3) Chromium supplementation of swine diets, beginning at an early age and continuing through the finishing period, could improve carcass leanness and subsequent reproductive efficiency, but these responses to chromium are likely to be inconsistent until the factors that affect the efficacy of dietary chromium inclusion are more clearly defined.

(4) Information in the scientific literature on the need for supplemental chromium in practical diets of fish, horses, sheep, rabbits, and rats is too sparse to allow conclusions.

(5) Although most research on potential toxicity of trivalent forms of dietary chromium has been conducted with rats and chickens, the results show that the concentrations of trivalent chromium typically added to diets of food-producing animals are safe and nontoxic.

(6) Additional research is needed to determine the bioavailability of chromium contained in feed ingredients and to obtain more definitive data on the comparative bioavailability of chromium from dietary supplements.

(7) Research should be designed to create reproducible signs of chromium deficiency in animals, which would facilitate the establishment of dietary chromium requirements by way of appropriate titration studies.

Successful determination of dietary requirements will depend partly on establishment of procedures for chromium analysis of feedstuffs and diets that are sensitive and that will yield reproducible results. Research also should be planned to control and measure the impact of factors suspected of contributing to inconsistent responses of animals to chromium supplementation of practical diets. This research would make it possible to document the circumstances in which supplemental dietary chromium could be used to best advantage.

1

Introduction

Chromium (Cr) has been considered an essential nutrient for humans and animals for approximately 40 years, and there are excellent reviews that detail several aspects of its function in nutrition (Anderson, 1987, 1988; Borel and Anderson, 1984; Prasad, 1978; Underwood, 1977). Chromium exists in nature mostly in the trivalent (Cr^{+3}) form. Chromium (Cr^{+3}) has been shown to have antioxidative properties in vivo (Tezuka et al., 1991), and it is integral in activating enzymes and maintaining the stability of proteins and nucleic acids (Borel and Anderson, 1984). Its primary metabolic role, however, is to potentiate the action of insulin through its presence in an organometallic molecule called the glucose tolerance factor (GTF). Schwarz and Mertz (1957, 1959) first isolated GTF from pork kidney (1957) and brewer's yeast (1959), and it is believed to consist of Cr^{+3}, nicotinic acid, glutamic acid, glycine, and cysteine (Toepfer et al., 1977). Without Cr^{+3} at its core, GTF is inactive. Most chromium in animal tissues is present in GTF.

In addition to GTF in yeast and animal tissues (Anderson, 1987), bovine colostrum contains at least five low-molecular-weight, chromium-containing substances (Yamamoto et al., 1988). One has biological activity and is an ionic complex consisting of chromium with aspartate, glutamate, glycine, and cysteine in a molar ratio of 5:4:2:1, and no detectable carbohydrate. A similar and biologically active chromium-containing substance has been found in rabbit liver (Yamamoto et al., 1989). Although relatively rare, signs of chromium deficiency are likely related to its interactions with insulin, and they include impaired glucose tolerance, elevated concentrations of insulin, glycosuria, impaired growth, decreased longevity, elevated concentrations of cholesterol and triacylglycerols,

increased aortic plaques, brain disorders, decreased fertility, and peripheral neuropathy (Borel and Anderson, 1984).

ABSORPTION, TRANSPORT, AND CONTENT IN ANIMAL TISSUES

Chromium is absorbed primarily in the small intestine. The most active site of absorption in rats seems to be the jejunum, with less efficient absorption occurring in the ileum and duodenum (Chen et al., 1973). Inorganic forms, such as that present in chromic chloride ($CrCl_3$) (as heptahydrate) and chromic oxide (Cr_2O_3), are absorbed poorly. The average absorption of Cr^{+3} has been estimated at 0.5 percent. The efficiency of absorption, however, is related inversely to dietary intake. Anderson (1987) reported that approximately 2 percent of dietary chromium was absorbed in humans consuming approximately 10 μg/day, whereas absorption efficiency was decreased to 0.5 percent when their intake was >40 μg/day. Abnormal absorption has been reported in insulin-requiring diabetics. Doisy et al. (1976) reported that insulin-requiring diabetics absorbed two to four times more chromium than was absorbed by normal subjects. The authors hypothesized that insulin-requiring diabetics are chromium deficient and develop an adaptive increase in absorption to help offset the deficiency.

Almost all sources of chromium in the Earth's crust are in the trivalent state. There are, however, manufactured forms ($K_2Cr_2O_7$, K_2CrO_4, and Na_2CrO_4) that exist in the hexavalent (Cr^{+6}) state. These forms are more soluble than is Cr^{+3} and, when administered directly into the intestine, are absorbed three to five times better than Cr^{+3} (Anderson, 1987). When taken orally, however, most of the Cr^{+6} is believed to be reduced to Cr^{+3} before reaching sites of absorption in the small intestine (Doisy et al., 1976). The reasons for the low availability of inorganic sources of Cr^{+3} are many and probably are related to formation of insoluble chromic oxide, binding to natural chelating agents in feedstuffs (such as phytate), and interference by ionic forms of other elements (zinc, iron, and vanadium) (Borel and Anderson, 1984), slow or no conversion of inorganic chromium to the bioactive form (Ranhotra and Gelroth, 1986), and suboptimal amounts of nicotinic acid (Urberg and Gemel, 1987). The content of total chromium in the diet, therefore, probably bears little relationship to its effectiveness as biologically active chromium.

Complexing chromium to organic molecules also can influence availability. For example, oxalate enhanced the absorption of chromium in rats, whereas EDTA and citrate did not (Chen et al., 1973). Other synthetic organic forms, such as chromium nicotinate (CrNic) and chromium picolinate (CrPic), also have been used as readily available sources of chromium. Olin et al. (1994) reported that absorption of chromium by rats during the first 12 hours after oral administration was greatest for CrNic and least for $CrCl_3$, with absorption from CrPic ranking intermediate. Anderson et al. (1996b) determined the relative bioavail-

ability of nine different organic and inorganic forms of chromium by measuring the incorporation of chromium into tissues of rats fed these various chromium sources. They demonstrated that chromium incorporation into tissues is highly dependent upon the form, with the greatest incorporation occurring from chromium dinicotinic acid diglycine cysteine glutamic acid, CrPic, chromium acetate, chromium potassium sulfate, and glycine chromium complexes. They also concluded that chromium chloride was a very poor source of chromium and that raising animals in stainless steel cages did not result in significant changes in tissue chromium concentrations. Naturally occurring chromium complexes also are known for their relatively high biologic availability. Experiments with rats, for example, suggest that 10 to 25 percent of the chromium in brewer's yeast is absorbed (Underwood, 1977). Good natural sources, in addition to brewer's yeast, include dark chocolate, black pepper, and some processed meats (Hunt and Stoecker, 1996; Toepfer et al., 1973).

Once absorbed, chromium circulates in plasma at a concentration of 0.01–0.3 μg/L (Anderson, 1987). These plasma concentrations are much lower than those reported before 1980, largely because of the increased sensitivity of chromium analysis made possible by use of the graphite furnace in conjunction with atomic absorption spectrophotometry (Mertz, 1993b). As described by Mertz (1993b), detection of such low serum concentrations requires strict control of contamination by performing sample preparation and analysis in ultraclean experimental rooms and by constant quality control with the use of standard reference materials. Plasma concentrations of chromium can be lower during infection and glucose loading (Borel and Anderson, 1984).

Circulating chromium is associated with the β-globulin portions of plasma and, in physiologic concentrations, is transported to tissues bound to transferrin and possibly as a component of GTF (Prasad, 1978). Chromium, therefore, can have significant effects on serum iron transport. Ani and Moshtaghie (1992), for example, demonstrated that intraperitoneal injections of $CrCl_3$ in rats can significantly decrease serum iron, total iron-binding capacity, and ferritin. At superphysiologic concentrations, chromium binds nonspecifically to other plasma proteins (Prasad, 1978).

Plasma is cleared of chromium within a few days of administration (Anderson, 1987). Whole-body chromium, however, is cleared in rats at a much slower rate and has been expressed as a three-compartment model with half-lives of 0.5, 6, and 83 days (Borel and Anderson, 1984). Some tissues, such as bone, testes, and epididymides, retain chromium longer than do the heart, lung, pancreas, or brain. Unlike some other elements (e.g., calcium and magnesium), it seems that no equilibrium exists between tissue stores of chromium and plasma. Concentrations in plasma are, therefore, a poor indicator of chromium status. Total body chromium concentrations decrease with age, which is reflected in a decrease in tissue uptake. In pharmacokinetic experiments, older mice had less ability to

concentrate chromium in several tissues than did younger animals. The reasons for these differences in tissue uptake are unknown.

Absorbed chromium is excreted mainly in the urine. Small amounts, however, are lost in hair, perspiration, and bile. The 24-hour urinary excretion rate for normal human subjects is reported to be 0.22 μg/day (Borel and Anderson, 1984), which is consistent with the relatively low absorption rate (approximately 0.5 percent) and typical daily chromium consumption rates (62–85 μg/day). Urinary excretion of chromium has been shown to increase after oral loading of glucose in healthy patients and is higher in human diabetics (Borel and Anderson, 1984). Stress and exercise also can result in increased urinary chromium excretion. Anderson (1988) reported that, in adult males, a six-mile run resulted in a fivefold increase in urinary chromium excretion within two hours after running, and the total urinary chromium excreted during the day of the run was more than twice that on the next day. Severely traumatized patients also excrete several times more chromium than do normal subjects. Urinary chromium excretion is, therefore, probably not a good indicator of dietary chromium status.

2

Chromium and Metabolism

Chromium (Cr) potentiates the action of insulin via the glucose tolerance factor (GTF) (Mertz, 1993a). Although the way in which this potentiation occurs has not been determined, Mertz et al. (1974) hypothesized that chromium forms a complex with insulin and insulin receptors to facilitate the response of insulin-sensitive tissues. Anderson et al. (1990) demonstrated that suboptimal intake of chromium by humans leads to detrimental changes in glucose, insulin, and glucagon status of subjects with slightly impaired glucose tolerance. Research results presented by Govindaraju et al. (1989) did not support the postulate that trivalent Cr^{+3} serves to assemble insulin and its receptor through metal-sulfur bonding, but indicated that chromium stabilizes the structure of insulin and affects its state of aggregation to influence the biopotency of the hormone.

In summarizing results of several experiments with humans, rats, mice, and other species, Anderson (1994) presented a list of physiological and biochemical symptoms of chromium deficiencies that strongly suggest chromium is an essential nutrient (Table 2-1).

Other indications suggesting that chromium is essential in the diets of food-producing animals are presented later in this report. Because there is no accurate measure of chromium status, daily chromium requirements for animals, including humans, have been difficult to define. One measure of sufficiency has been to determine whether glucose tolerance is improved with chromium supplementation. Moreover, the form of dietary chromium determines biological activity, and GTF in brewer's yeast has the highest bioavailability (Toepfer et al., 1973). A range of chromium intake between 50 and 200 μg/day is recommended for adult humans (National Research Council, 1989).

TABLE 2-1. Signs and Symptoms of Chromium Deficiency

Function	Species
Impaired glucose tolerance	Human, rat, mouse, squirrel monkey, guinea pig
Elevated circulating insulin	Human, rat, pig
Glycosuria	Human, rat
Fasting hyperglycemia	Human, rat, mouse
Impaired growth	Human, rat, mouse, turkey
Hypoglycemia	Human
Elevated serum cholesterol and triacylglycerols	Human, rat, mouse, cattle, pig
Increased incidence of aortic plaques	Rabbit, rat, mouse
Increased aortic intimal plaque area	Rabbit
Neuropathy	Human
Encephalopathy	Human
Corneal lesion	Rat, squirrel monkey
Ocular eye pressure	Human
Decreased fertility and sperm count	Rat
Decreased longevity	Rat, mouse
Decreased insulin binding	Human
Decreased insulin receptor number	Human
Decreased lean body mass	Human, pig, rat
Elevated percentage body fat	Human, pig
Enhanced humoral immune response	Cattle
Morbidity	Cattle

Source: Anderson, 1994

CARBOHYDRATE METABOLISM

The first suggestion that chromium participates in carbohydrate metabolism in animals was the report of Schwarz and Mertz (1957). They observed that GTF, which was shown later to contain chromium (Schwarz and Mertz, 1959), was deficient in animals with impaired glucose tolerance, and that supplemental chromium improved glucose tolerance (Schwarz and Mertz, 1959). Although GTF seems to contain nicotinic acid, glycine, glutamic acid, and cysteine in addition to chromium, synthetic complexes have markedly less insulin-potentiating activity than does the naturally occurring complex (Anderson et al., 1978). Thus, the exact structure of the native insulin-potentiating complex has not been determined. Glucose uptake, glucose use for lipogenesis, glucose oxidation to carbon dioxide, and glycogenesis increase because of the addition of chromium plus insulin to animal tissues (Anderson, 1987). Chromium alone was ineffective. Also, the low-molecular-weight, chromium-binding substance present in milk enhanced glucose oxidation and lipogenesis from glucose (Yamamoto et al., 1988, 1989). The effect of the substance on glucose metabolism was decreased markedly when chromium was removed.

Chromium supplementation of several human patients receiving total parenteral nutrition and afflicted with a variety of disorders in glucose metabolism, such as diabetes-like symptoms, caused glucose metabolism to return to normal (Jeejeebhoy et al., 1977). Because of this benefit of supplemental chromium, the American Medical Association recommends daily supplementation of total parenteral nutrition solutions with 10 to 15 μg of Cr^{+3} to stable adults with intestinal fluid losses. There also have been observations of a syndrome resembling diabetes mellitus being cured by chromium supplementation, indicating that a decreased sensitivity of peripheral tissues to insulin is the primary biochemical lesion in chromium deficiency (Anderson et al., 1996a). Chromium increases or potentiates the activity of insulin but does not substitute for the anabolic hormone.

Anderson (1987) cited numerous case studies with humans in which glucose tolerance and other measures of glucose metabolism were improved with chromium supplementation. Moreover, supplemental dietary chromium (200 or 1,000 μg/day) had beneficial effects on cholesterol, glycosylated hemoglobin, glucose, and insulin in blood of humans with type II diabetes (Anderson et al., 1996a). Improvements were not always observed, probably because chromium status was adequate without chromium supplementation or because other etiological factors were involved.

LIPID METABOLISM

Numerous studies suggest that chromium is necessary for normal lipid metabolism and for minimizing rates of atherogenesis. For example, rats and rabbits fed low-chromium diets had greater concentrations of serum cholesterol and aortic lipids and exhibited greater plaque formation (Abraham et al., 1982a,b). Chromium supplementation decreased cholesterol concentrations. Newman et al. (1978) reported that humans who died of coronary artery disease had low chromium concentration in aortae but not in other tissues. Increases in high-density lipoprotein (HDL) cholesterol (Anderson, 1995; Riales and Albrink, 1981); and decreases in total cholesterol, low density lipoprotein (LDL) cholesterol, and triacylglycerols (Anderson, 1995; Lefavi et al., 1993) in humans have been reported to occur after chromium supplementation. Anderson (1987) indicates that the effects of chromium supplementation on blood lipids in humans are not always consistent; effects on lipid metabolism seem independent of effects on glucose metabolism (Lefavi et al., 1993). Blood lipids of humans with the greatest concentrations of blood cholesterol and triacylglycerols decrease the most after chromium supplementation. Because many factors cause elevated blood lipids, only those hyperlipemic individuals with marginal chromium status would be candidates for improvements in clinical status by chromium supplementation.

PROTEIN METABOLISM

Because of the role of insulin in amino acid uptake by animal tissues, chromium is predicted to interact with protein biosynthesis. Roginski and Mertz (1969) reported that chromium supplementation increased amino acid incorporation into heart proteins and amino acid uptake into tissues of rats. No other studies of an effect of chromium on protein synthesis or turnover have been reported.

NUCLEIC ACID METABOLISM

Chromium in the trivalent oxidation state seems to be involved in the structural integrity and expression of genetic information in animals. The bonding of chromium to nucleic acids is tighter than is that of other metal ions (Okada et al., 1982). Chromium protects ribonucleic acid (RNA) against heat denaturation. Moreover, chromium seems to concentrate in the nuclei of animal cells. Supporting the hypothesis that it affects gene function, chromium has been shown to enhance RNA synthesis in mice in vitro (Okada et al., 1982) and in vivo (Okada et al., 1983). With the use of the regenerating rat liver model, nucleic-acid-enhancing activity was associated with a 70,000 dalton protein that contained 5 to 6 chromium ions (Okada et al., 1984).

STRESS

Chromium status of animals seems to be influenced by physiological, pathological, and nutritional stresses. For example, exercise (Anderson et al., 1982) and trauma (Borel and Anderson, 1984) increased urinary chromium of humans and thereby could contribute to chromium deficiency. Symptoms of chromium deficiency are aggravated by a low-protein diet, exercise, blood loss, and infection (Mertz and Roginski, 1969; Roginski and Mertz, 1969). The intriguing possibility that supplemental chromium increases longevity and retards aging by improving immune function and enhancing resistance to infectious diseases is being investigated (Burton et al., 1996). Several studies summarized by Burton (1995) indicate that supplemental dietary chromium for market-transit-stressed feedlot calves and periparturient and early-lactation dairy cows improves milk production, immune status, and health.

CHROMIUM TOXICITY

Chromium salts are strong oxidizing agents used, for example, in alloying and tanning, and in the manufacture of rust- and corrosion-resistant paints. Workers exposed to high concentrations of these compounds suffer from chromium toxicity, and their symptoms include eczematous dermatitis, ulceration of skin,

lung cancer, gastroenteritis, nephritis, and hepatitis. Chromium toxicity is primarily associated with exposure to hexavalent Cr^{+6} compounds rather than to Cr^{+3} compounds, which have relatively low toxicity. The difference in toxicity between the two forms arises because of the relative ease with which Cr^{+6} is absorbed by cells. Unlike Cr^{+3}, Cr^{+6} is taken up easily, probably through an ion transport system (Jennette, 1979). Thus, extracellular reduction of Cr^{+6} to Cr^{+3} currently is perceived as a protective action (DeFlora and Wetterhahn, 1989). Inside the cell, reduction of Cr^{+6} could be a mechanism for detoxification as well as for activation (Alexander, 1993). Some of the chromium species formed become long-lived coordinated complexes that can migrate from the cytoplasm to the nucleus and damage the deoxyribonucleic acid (DNA) therein. Several enzymes and low-molecular-weight compounds participate in the reduction process (Alexander, 1993). Ascorbate and thiol-reducing-containing reductants, such as glutathione and cysteine, are likely nonenzymatic reductants (Alcedo and Wetterhahn, 1990; DeFlora and Wetterhahn, 1989; Standeven and Wetterhahn, 1991). Among enzymatic proteins, the endoplasmic P450 and the ethanol-inducible P450IIE1 have been shown to reduce Cr^{+6} in vitro (Mikalsen et al., 1991). Hexavalent chromium also can penetrate the mitochondrial membrane and be reduced (Ryberg and Alexander, 1984, 1990). Although Cr^{+6} can react at several places within the cell, the toxic effects seem specific. Excess cellular Cr^{+6} causes a dramatic depression of mitochondrial oxygen consumption, evidently because of an inhibition of the α-ketoglutarate dehydrogenase that supplies the respiratory chain with reduced nicotinamide adenine dinucleotide (Ryberg and Alexander, 1990). Several DNA lesions also can be observed after Cr^{+6} exposure; for example, DNA strand breaks, DNA interstrand cross-links, DNA-protein cross-links, and nucleotide derivatives caused by reactive oxygen species can be formed and cause abnormal phenotypes (Alexander, 1993).

For livestock, the National Research Council (1980) set the maximum tolerable concentrations of chromium in the diet at 3,000 ppm for the oxide and 1,000 ppm for the chloride form. Both forms are present as Cr^{+3}. Hexavalent forms are more soluble than are Cr^{+3} forms, and they are at least five times more toxic.

3

Chromium in Animal Nutrition

Information obtained on the nutritional essentiality and physiological function of chromium (Cr) has been less definitive for animals than it has been for humans. The experimental approaches and criteria used to evaluate the influence of supplemental dietary chromium on cattle, sheep, swine, poultry, horses, rats, rabbits, and fish are discussed in this chapter.

RUMINANTS

Lactating Cows

Assessment of performance or metabolic responses to chromium supplementation in lactating dairy cattle has focused on the transition and early-lactation cow. Results of a study with periparturient and early-lactation dairy cows indicated that chromium supplementation influences the immune response in stressed cattle (Burton et al., 1993). Twenty cows fed a basal diet containing an unspecified quantity of chromium were compared with those fed 5.5 mg supplemental chromium per day as chelated chromium for 6 weeks before calving, and 9.98 mg/day for the first 16 weeks of lactation. Humoral immune responses were assessed by measuring antibodies either to ovalbumin or to human red blood cells. The chromium-supplemented cows had greater antiovalbumin antibody response profiles than did unsupplemented cows. Differences in antibody titers to human red blood cells were not evident. These results suggest that supplemental chromium influences production of IgG, but not IgM antibodies. Cows fed supplemental chromium had greater mitogen-stimulated blastogenic responses

by peripheral blood mononuclear cells prepartum and at calving than did those of controls, also indicating a greater cell-mediated immune response because of chromium supplementation. Burton et al. (1996) conducted a subsequent study to determine whether chromium affected cytokine production by activated peripheral blood mononuclear cells. They observed lower concentrations of interleukin-2, interferon-γ, and tumor necrosis factor-α in the culture supernatants of mitogen-stimulated mononuclear cells from chromium-supplemented cows than from unsupplemented cows in one or more of four sample times during the trial. This observation suggests that chromium can affect blastogenic activity by altering cytokines production during the early stages of peripheral blood mononuclear cell activation.

Results of experiments involving 74 Holstein cows suggested differences in response between primiparous and multiparous cows fed diets supplemented with 0.5 mg Cr/kg dry matter (DM) as amino acid chelated chromium (Yang et al., 1996). Supplemental chromium increased milk yield of primiparous cows by 13.2 percent in Experiment 1 ($P = 0.06$) and 7 percent in Experiment 2 ($P < 0.05$) during the first 16 weeks of lactation. Supplemental chromium had no effect, however, on milk yield of multiparous cows when compared with that of cows consuming unsupplemented prepartal diets (containing 0.79 and 1.23 mg Cr/kg DM) and unsupplemented postpartal diets (containing 1.01 and 1.60 mg Cr/kg DM) in Experiments 1 and 2, respectively. Although primiparous cows tended to increase their dry matter intake (DMI) in the first 4 to 8 weeks of lactation when fed supplemental chromium, DMI was not influenced by treatment. In one of the experiments, supplemental chromium decreased mean serum nonesterified fatty acid (NEFA) and β-hydroxybutyric acid (β-HBA) in cows of three or more parities. Results of adrenocorticotrophic-releasing hormone and gonadotropin-releasing hormone (ACTH and GnRH) stimulation-response tests on primiparous cows suggest that chromium-supplemented cows experienced a greater stress with their increased milk production.

Further studies with prepartum and early-lactation cows confirmed that supplementation with 5.5 mg Cr/day as chelated chromium during the dry period, and with 10 mg Cr/day during the initial six weeks of lactation, tends to improve milk yield in the initial stages of lactation for primiparous cows, but not for multiparous cows (Subiyatno et al., 1996). Glucose tolerance tests conducted on cows two weeks prepartum and two weeks postpartum showed no effect of Cr supplementation on multiparous cow plasma glucose and insulin basal or peak levels, clearance rates, or insulin-to-glucose ratios; however, the prepartum plasma insulin-to-glucose ratio and triglyceride and insulin concentrations were decreased ($P < 0.10$) by supplemental chromium in primiparous cows. Insulin sensitivity of postpartum primiparous cows was reduced ($P < 0.10$) due to chromium supplementation. Subiyatno et al. (1996) also studied the effects of propionate loading on a second group of primiparous cows maintained on the same feeding program. Effects of chromium supplementation were most apparent at two weeks postpar-

tum, causing reduced basal but higher peak serum propionate levels, elevated basal and peak glucose levels, reduced basal and peak insulin levels, and a lower insulin-to-glucose ratio ($P < 0.10$). These responses were not evident when the test was repeated at six weeks postpartum; however, reduced basal serum NEFA levels were observed for supplemented cows following propionate infusion.

Chromium supplementation has, relatively consistently, improved milk production of primiparous cows in the first four to six weeks of lactation when chromium supplementation was initiated in late gestation and continued into the first weeks of lactation. Primiparous cows can be more resistant to insulin than multiparous cows during late gestation (McClary et al., 1988). Supplementation with chromium seems to improve insulin sensitivity for these primiparous animals before calving, if one can assume that the ratio of insulin to glucose can be used as a crude indicator. After calving, however, supplemental chromium seems to reduce insulin sensitivity and increase plasma triglycerides. Subiyatno et al. (1996) suggested that this may relate to the ability of the liver to mobilize triglycerides as very low density lipoproteins during negative energy balance. Recently completed work by Besong et al. (1996) supports this theory. Decreased plasma β-HBA and liver triglyceride concentrations were observed 30 days after calving in cows supplemented with 0.8 mg Cr/day as chromium picolinate (CrPic). A summary of experimental results with lactating cows is presented in Table 3-1.

Preweaned Calves

Newborn calves offered 1.0 mg Cr/kg DM as CrPic in either the milk replacer or calf starter for a 53-day study did not show a performance response (DePew et al., 1995). Glucose responses to an intravenous glucose tolerance test and an intravenous propionate load test also were not affected by treatment. Prefeeding blood glucose and insulin levels were not affected by dietary treatment during the course of the trial, but there was some indication that prefeeding NEFA concentrations were decreased in calves given chromium-supplemented diets. Further work to determine the effects of dietary chromium on metabolic criteria during the first weeks of life have not been conducted. One-week-old bull calves, however, were used to determine the effect of either organic or inorganic chromium on cell-mediated and humoral immune response (Kegley et al., 1996). Calves were fed a milk replacer containing 0.31 mg Cr/kg DM for an 87-day trial. Chromium was supplemented at 0.4 mg/kg DM as either chromium nicotinate (CrNic) or as chromic chloride ($CrCl_3$). Animal performance criteria were not affected by chromium supplementation. Immunoglobulin G, IgM, and total antibody titers to porcine red blood cells also were not affected by dietary treatment. Chromium supplementation did not influence blastogenic response of lymphocytes when either T-cell-stimulating or thymus-dependent B-cell mitogens were used. Intradermal injection of phytohemagglutinin resulted in greater skinfold thickness responses for calves fed either supplemental chromium source.

TABLE 3-1. Influence of Supplemental Dietary Chromium on Lactating Dairy Cows[a]

Reference	Dietary Cr Concentration per kg DM Unless Stated Otherwise (Source)	Lactation Stage and Duration of Experiment	Feed Intake	Milk Yield
Burton et al. (1993)	Basal diet = unknown; Cr diet = basal + 5.5 mg (chelated Cr).	20 Holstein cows 6 wk pre- to 16 wk postpartum	ND	ND
Besong et al. (1996)	Basal diet = unknown; Cr diet = basal + 0.8 mg (CrPic).	24 Holstein cows 30 d pre- to 60 d postpartum	↑ DMI.	↑ milk yield.
Burton et al. (1996)	Basal prepartum diet = unknown; Cr prepartum diet = basal + 5.5 mg/d; Basal postpartum diet = unknown; Cr postpartum diet = basal + 10 mg/d (chelated Cr).	19 Holstein cows 6 wk pre- to 16 wk postpartum	ND	ND
Subiyatno et al. (1996)	Basal prepartum diet = 0.79 mg; Cr prepartum diet = basal + 5.5 mg/d; Basal postpartum diet = 1.23 mg; Cr postpartum diet = basal + 10 mg/d (chelated Cr).	12 Holstein cows - 4 PP and 8 MP 6 wk pre- to 2 wk postpartum	No effect.	No effect.
Subiyatno et al. (1996)	Basal prepartum diet = 1.01 mg; Cr prepartum diet = basal + 5.5 mg/d; Basal postpartum diet = 1.60 mg; Cr postpartum diet = basal + 10 mg/d (chelated Cr).	12 PP Holstein cows 6 wk pre- to 6 wk postpartum	No effect.	↑ 10% $P<0.10$

Milk Components	Body Weight and Change	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	ND			↑ anti-OVA titer (IgG). No anti-HRBC response (IgM). ↑ con-A mitogen stimulated blastogenic response of peripheral mononuclear cells.
No effect.	ND	No effect on plasma glucose, NEFA, and serum insulin. ↓ plasma β-HBA and liver triglyceride on d 30 postpartum.		
ND	ND	ND	ND	↓cytokines in culture supernatants of mitogen-stimulated mononuclear cells.
ND	No effect.		↓ plasma triglycerides, ↓ peak insulin, ↓ insulin:glucose ratio in prepartum PP cows, and ↑ plasma triglycerides and ↑ insulin:glucose ratio in postpartum PP cows during glucose tolerance test $P<0.10$.	
ND	ND		↑ serum glucose peak + AUC, ↑ propionate peak + basal concentration, and ↓ insulin:glucose + glucagon:glucose ratios during postpartum propionate infusion $P<0.10$.	

TABLE 3-1. Continued

Reference	Dietary Cr Concentration per kg DM Unless Stated Otherwise (Source)	Lactation Stage and Duration of Experiment	Feed Intake	Milk Yield
Yang et al. (1996)	Basal prepartum diet = 0.79 mg; Cr prepartum diet = 1.29 mg; Basal postpartum diet = 1.01 mg; Cr postpartum diet = 1.51 mg (amino-acid-chelated Cr).	34 Holstein cows - 12 PP and 22 MP 6 wk pre- to 16 wk postpartum	No overall effect. Cr-fed PP cows had 15% greater DMI in first 4 wk of lactation.	PP cows - 13.2% increase *P*=0.06; MP cows - no effect.
Yang et al. (1996)	Basal prepartum diet = 1.23 mg; Cr prepartum diet = 1.93 mg; Basal postpartum diet = 1.60 mg; Cr postpartum diet = 2.10 mg (amino-acid-chelated Cr).	40 Holstein cows 18 PP and 22 MP 6 wk pre- to 16 wk postpartum	No overall effect. Cr-fed PP cows tended to have a higher DMI in first 8 wk of lactation *P*=0.08.	PP cows - 7% increase; MP cows - no effect.

[a]Definitions of abbreviations used in table:
AUC - area under curve
β-HBA - beta hydroxybutyric acid
con-A - concanavalin A
Cr - chromium
CrPic - chromium picolinate
d - day
DM - dry matter
DMI - dry matter intake
HRBC - human red blood cells
LH - luteinizing hormone
MP - multiparous
ND - not determined
NEFA - nonesterified fatty acids
OVA - ovalbumin
PP - primiparous
wk - week

Milk Components	Body Weight and Change	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
No effect.	PP cows lost more weight, *P*=0.07; MP cows - no effect.	No effect on NEFA or β-HBA.		No effect on reproduction.
No effect.	No effect.	PP cows - no effect. MP cows - serum NEFA and β-HBA were reduced when 3+ parities. Serum glucose, cortisol, progesterone, and LH not affected.		No effect on reproduction.

Antibody titers to *Pasteurella hemolytica* were not affected by chromium supplementation. Serum cortisol concentrations were not influenced by treatment on day 7 of the trial; however, calves fed chromium-supplemented diets had lower cortisol concentrations and higher white blood cell counts 5 days after infection with infectious bovine rhinotracheitis virus (IBR). These differences disappeared by day 12 after infection. A summary of experimental results is presented in Table 3-2, which also presents results of studies on growing-finishing cattle.

Growing-Finishing Cattle

The first research results suggesting a beneficial effect of chromium supplementation to the diet of cattle were reported by Chang and Mowat (1992). Average daily gain (ADG) and ADG/DMI of 108 transportation-stressed, Charolais-cross calves, provided with 0.4 mg Cr/kg DM as high-chromium yeast, were improved by 30 and 27 percent, respectively, during the initial 28 days after arrival at the feedlot, compared with stressed calves not receiving chromium-supplemented feed. This benefit of supplemental chromium was not observed when calves received a long-acting antibiotic within 48 hours of arrival at the feedlot. The measured chromium concentration in the control diets (corn silage with urea-corn or with soybean meal) of this study was 12.12 mg/kg DM.

Selecting 96 steers from the above group of 108 calves, Chang et al. (1992) fed diets with 0 or 0.2 mg Cr/kg DM from high-chromium yeast during the growing period, followed by diets with 0 or 1.87 mg Cr/kg DM during the finishing period. Chromium supplementation had no effect on performance during either period. Chromium supplementation, however, decreased serum cortisol in all calves and increased serum IgM and total immunoglobulin in calves fed corn silage diets supplemented with soybean meal. The immune response was not evident for calves fed a urea-corn grain supplement. Carcass characteristics were not altered as a result of chromium supplementation during the growing-finishing phase. Results of this series of experiments suggest that chromium might be essential to cattle during periods of stress.

A second trial with transportation-stressed calves showed a 27 percent increase in ADG, but no feed efficiency response, when 0.2 or 1.0 mg Cr/kg DM as high-chromium yeast was added to a diet containing 0.16 mg Cr/kg DM (Moonsie-Shageer and Mowat, 1993). No performance response was observed for calves fed an intermediate concentration (0.5 mg/kg DM) of chromium in this 30-day trial. Unlike the earlier trial, chromium supplementation reduced morbidity from days 2 to 30, and the 0.2 mg Cr/kg supplementation rate was most effective. Rectal temperatures were lower for the first 5 days after arriving in the feedlot, and elevated serum calcium and magnesium were evident in the first week but not thereafter. Increased hemagglutinating antibody titers to human red blood cells on day 14 suggested an improved humoral immune response in stressed feeder calves fed chromium supplements. Serum cortisol levels were

decreased in calves fed supplemental chromium by the end of the 28-day trial. Chromium supplementation had no effect on concentrations of potassium, cholesterol, globulin, glucose, protein, urea, and alkaline phosphatase in serum, and it had a transitory effect on serum albumin.

Feedlot-adjusted steers offered a supplement of 1 mg Cr/kg DM in a chelated form did not show improved ADG, DMI, or feed efficiency but had decreased serum glucose and cortisol during a 56-day trial (Mowat et al., 1993). Results of this study confirmed that the responses observed in previous studies conducted by Chang and Mowat (1992) were not caused by the yeast component of the chromium supplement, but rather were related to chromium. A second trial with newly arrived steer calves compared high-chromium yeast with chelated chromium and showed decreased morbidity for the chelated-chromium-treated calves relative to controls; calves receiving the high-chromium yeast had an intermediate response. Chromium supplementation did not affect calf weight gain in the 35-day trial, but feed efficiency was improved with diets containing the high-chromium yeast and one of the two chelated forms of chromium. Serum cortisol was not affected by treatment in the study (Mowat et al., 1993). The chromium concentration of the unsupplemented diet was 0.16 mg/kg in each trial.

In another study, calves fed a lower level of supplemental chromium (0.14 mg added Cr/kg DM as chelated chromium) did not have improved ADG, DMI, or feed efficiencies in a 49-day trial using corn-silage-based diets (Wright et al., 1994). Morbidity was not decreased in calves; however, the number of illness relapses for calves receiving the chromium supplement was smaller. The authors indicated that the failure to demonstrate improved calf performance with chromium supplementation in this trial could have been related to the fact that the calves were sick early in the trial, allowing little opportunity for chromium to exert its effect. Chromium concentrations in the unsupplemented diets were reported to be 0.32 mg/kg DM for the first 28 days and 1.05 mg/kg DM thereafter. Serum total and free triiodothyronine and plasma cortisol were not influenced by chromium supplementation. Chromium-supplemented calves receiving vaccinations had greater plasma ascorbate concentrations than did calves receiving vaccinations only, or no vaccinations and no supplemental chromium, suggesting a link between immune function and dietary chromium.

To determine a link between immune function and dietary chromium, Burton et al. (1994) tested whether supplemental chromium affected antibody responses of stressed feedlot cattle to clinically relevant antigens (IBR and parainfluenza-3 [PI-3]) contained in a commercial vaccine. Steers were offered 0.5 mg Cr/kg DM as chelated chromium in a corn-silage-based diet containing 0.16 mg Cr/kg DM. Chromium supplementation increased the magnitude of the peak antibody response to IBR but had no effect on anti-PI-3 antibody titers.

Chang et al. (1994) supplemented the diet of transportation-stressed steers with 0.14 mg Cr/kg DM as chelated chromium for 49 days. They reported that blood lymphocytes obtained from morbid steers fed supplemental chromium

TABLE 3-2. Influence of Supplemental Dietary Chromium on Cattle[a]

Reference	Dietary Cr Concentration per kg DM (Source)	Body Weight at Start and Duration of Experiment	Improved Growth Rate	Improved Feed Efficiency
PREWEANED CALVES				
DePew et al. (1995)	Basal diet = unknown; Cr diet = basal + 1.0 mg (CrPic).	42 newborn Holstein calves 39 kg 53-d trial	No effect.	No effect.
Kegley et al. (1996)	Basal diet = 0.31 mg; CrNic = 0.71 mg; $CrCl_3$ diet = 0.71mg.	1-wk-old Holstein bull calves 87-d trial	No effect.	No effect.
WEANED, STRESSED CALVES				
Chang and Mowat (1992)	Basal diet = 12.12 mg; Cr diet = 12.52 mg (Cr yeast).	108 calves - 245 kg 28-d trial transportation stressed	No overall effect. ↑ in calves not receiving antibiotics.	No overall effect. ↑ in calves not receiving antibiotics.
Moonsie-Shageer and Mowat (1993)	Basal diet = 0.16 mg; Cr diets = 0.36, 0.66, 1.16 mg (Cr yeast).	84 steers - 236 kg 30-d trial transportation stressed	↑ for 2 of 3 Cr-supplemented diets.	No effect.
Mowat et al. (1993)	Basal diet = unknown; Cr yeast diet = basal + 0.5 mg; chelated Cr diet = basal + 0.5 mg; chelated Cr, Zn, Cu + Mn for 1^{st} 7 d = basal + 0.5 mg.	72 steers - 233 kg 35-d trial mixing/transportation stressed	No effect.	↑ for Cr yeast and one of chelated Cr diets.
Bunting et al. (1994)	Basal diet = 0.53 mg; Cr diet = 0.90 mg (CrPic).	10 Holstein steers - 98 kg 58-d trial not stressed	No effect.	No effect.
Bunting et al. (1994)	Basal diet = 0.34 mg; Cr diet = 0.71 mg (CrPic).	14 Holstein heifers - 122 kg 56-d trial not stressed	No effect.	No effect.

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	ND	No differences in blood glucose or insulin ↓ NEFA until wk 2.	No difference in response to i.v. glucose tolerance test or i.v. propionate load test.	
ND	ND	Short-term ↓ serum cortisol following IBR infection.		No response to porcine RBC. No T-cell or B-cell mitogen-induced blastogenic response of lymphocytes. ↑ skin fold thickness with i.d. injection of PHA.
ND	ND	ND	ND	
ND	ND	↓ cortisol by d 28. ↑ serum Ca and Mg in first wk. ↑ serum albumin in wk 1 and 3.	ND	↓ rectal temperature for first 5 d. ↓ morbidity ↑ IgG titer on d 14.
ND	ND	No effect.	ND	↓ morbidity.
ND	ND	↓ plasma cholesterol in wk 4 only. No effect on plasma NEFA, glucose, insulin, creatinine, urea N, total protein.	↑ rate of glucose clearance during i.v. glucose tolerance test. No difference for insulin challenge test.	No difference in GH or GHRH. No difference in N balance.
ND	ND	↓ plasma cholesterol in wk 6 only. No effect on plasma NEFA, glucose, insulin, creatinine, urea N, or total protein.	↑ rate of glucose clearance during i.v. glucose tolerance test. No difference for insulin challenge test.	No difference in GH or GHRH. No difference in N balance.

TABLE 3-2. Continued

Reference	Dietary Cr Concentration per kg DM (Source)	Body Weight at Start and Duration of Experiment	Improved Growth Rate	Improved Feed Efficiency
Burton et al. (1994)	Basal diet = 0.16 mg; Cr diet = 0.66 mg (chelated Cr).	55 crossbred calves - 233 kg 36-d trial transportation and handling stressed	ND	ND
Chang et al. (1994)	Basal diet = unknown; Cr diet = basal + 0.14 mg (chelated Cr).	24 steers - 258 kg 49-d trial mixing/transportation stressed	ND	ND
Wright et al. (1994)	Basal diet (first 28 d) = 0.32 mg; Basal diet (last 18 d) = 1.05 mg; Cr diet = basal + 0.14 mg (chelated Cr).	132 steers - 250 kg 49-d trial mixing/transportation stressed	No effect.	No effect.
Chang et al. (1995)	Basal diet = 1.41 mg; Cr yeast diet = 2.50 mg; $CrCl_3$ diet = 2.50 mg; $CrCl_3$ and niacin diet = 2.50 mg (Cr yeast or $CrCl_3$).	135 steers - 236 kg 56-d trial mixing/transportation stressed	No effect.	No effect.
Kegley and Spears (1995)	Basal diet = 0.66 mg; $CrCl_3$ diet = 1.06 mg; Cr yeast diet = 1.06 mg; CrNic = 1.06 mg.	125 steers - 215 kg 56-d trial mixing/transportation stressed	No effect.	No effect.
Mathison and Engstrom (1995)	Basal diet = unknown; Basal diet, no stress = unknown; Cr diet, stress = basal + 0.57 = 0.83; Cr diet, no stress = basal + 0.69 - 0.83 (chelated Cr).	192 steers - 262 kg 28-d trial mixing/transportation stressed	No effect.	No effect.
Wright et al. (1995)	Basal diet = 0.32 mg; Cr diet = 0.56 mg (chelated Cr).	72 steers - 250 kg 28-d trial mixing/transportation stressed	ND	ND
Chang et al. (1996)	Basal diet (first 28 d) = 0.32 mg; Cr diet (last 18 d) = 1.05 mg; Cr diet = basal + 0.14 mg (chelated Cr).	66 steers - 250 kg 49-d trial mixing/transportation stressed	ND	ND

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	ND			↑ peak antibody titer to IBR. No effect for PI-3 vaccination.
ND	ND			↑ blastogenic response in lymphocytes to con-A for morbid calves.
ND	ND	No effect on serum total and free triiodothyronine. No effect on cortisol. ↑ plasma ascorbate post-vaccination.		
ND	ND	Cr - yeast ↑ serum Fe and total iron-binding capacity in healthy calves. No effect for serum cortisol, serum glucose, plasma ascorbate, plasma protein, ammonia.		
ND	ND	↑ serum insulin for CrNic.		↑ serum IgG. ↑ skin inflammatory response to PHA. ↑ blastogenic response in lympho-cytes to PHA (T-cell).
ND	ND	ND	ND	No effect on morbidity.
ND	ND	↓ serum haptoglobulin on d 7.		
ND	ND			↑ d 28 titer for BVD. No response for IBR, PI-3, BRSV, *P. hemolytica.*

TABLE 3-2. Continued

Reference	Dietary Cr Concentration per kg DM (Source)	Body Weight at Start and Duration of Experiment	Improved Growth Rate	Improved Feed Efficiency
Chang et al. (1996)	Basal diet = 1.45 mg; Cr diet = 2.20 mg (Cr yeast).	54 steers - 236 kg mixing/transportation stressed	ND	ND
Kegley et al. (1997)	Basal diet = unknown; Cr diet = basal + 0.4 mg (CrNic).	48 steers - 263 kg 56-d trial transportation stressed	↑ (P<0.10).	No effect.
GROWING-FINISHING CATTLE				
Chang and Mowat (1992)	Basal diets - urea and corn = 12.03 mg; SBM = 12.09 mg; Cr diets = urea + corn - basal + 0.2 mg; SBM - basal + 0.2 mg (Cr yeast).	96 steers - 280 kg 70-d trial	No effect.	No effect.
Chang et al. (1992)	Basal finishing diet = 1.87 mg; Cr finishing diet = 2.07 mg (Cr yeast).	96 steers - 377 kg 68-d finishing trial	No effect.	No effect.
Mowat et al. (1993)	Basal diet = 0.16 mg; Cr diet = 1.16 mg (chelated Cr).	12 steers - 260 kg 56-d trial	No effect.	No effect.
Mathison and Engstrom (1995)	Basal diet = unknown; Cr diet = basal + 0.5 mg (chelated Cr).	190 steers - 294 kg feed lot adjusted, fed until finished	No effect.	No effect.

[a]Definitions of abbreviations used in table:

BVD - bovine viral diarrhea
BRSV - bovine respiratory syncytial virus
con-A - concanavalin A
Cr - chromium
$CrCl_3$ - chromium chloride
CrNic - chromium nicotinate
CrPic - chromium picolinate
d - day
DM - dry matter
GH - growth hormone
GHRH - growth hormone releasing hormone
IBR - infectious bovine rhinotracheitis virus
i.d. - intradermal
IgG - immunoglobulin G
IgM - immunoglobulin M
i.v. - intravenous
ND - not determined
NEFA - nonesterified fatty acids
OVA - ovalbumin
PHA - phytohemagglutination
PI-3 - parainfluenza-3
RBC - red blood cells
SBM - soybean meal
wk - week

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	ND			No response for BVD, IBR, PI-3, BRSV, *P. hemolytica*. ↑ titer to OVA.
ND	ND	↓ serum IgG after stress.		No difference in rectal temperature or antibody response after IBR challenge. No antibody response difference to porcine RBC.
ND	ND	↓ cortisol. ↑ urea. ↓ protein.	ND	Animals fed corn silage and SBM had ↑ IgM and total Ig. Not observed for animals fed corn silage urea-corn grain.
No effect.	No effect.	ND	ND	↑ liver Cr for cattle fed urea (not SBM) in growing phase.
ND	ND	↓ cortisol and glucose.	ND	ND
No effect.	No effect.			No effect on morbidity.

responded more to concanavalin A stimulation than did lymphocytes from steers not receiving added chromium. Added chromium did affect performance of the steers.

Growing Holstein steer and heifer calves were offered either a control diet (containing 0.53 or 0.34 mg Cr/kg DM, respectively, for steers and heifers) or the control diet plus 0.37 mg Cr/kg DM as CrPic for heifers (Bunting et al., 1994). Calf ADG, DMI, feed efficiency, and nitrogen balance were not influenced by dietary treatment. There were transient declines in plasma cholesterol for both heifers and steers; however, average plasma cholesterol concentrations were not affected by treatment. Plasma NEFA, glucose, insulin, creatinine, urea nitrogen, and total protein were not affected by treatment. Calves receiving supplemental chromium showed a 27 to 40 percent more rapid clearance of plasma glucose and a shorter plasma glucose half-life during an intravenous glucose tolerance test than did controls. Calves also were subjected to an intravenous insulin challenge test, and heifers, but not steers, had faster glucose clearance rates and shorter plasma glucose half-lives when fed supplemental chromium. These data suggest that chromium affects carbohydrate metabolism by enhancing the response of tissues to insulin.

The potential interaction between supplemental inorganic chromium as $CrCl_3$ and niacin was investigated by Chang et al. (1995). Unsupplemented and chromium-yeast-supplemented diets were included as controls. Newly arrived steers were offered an alfalfa-silage-based diet containing 1.41 to 1.47 mg Cr/kg DM, with chromium supplemented at 0.75 mg/kg DM. There were tendencies toward increased weight gain and improved feed efficiency in the initial 28 days for calves fed the inorganic chromium-plus-niacin treatment, with no improvements for calves receiving chromium-yeast or inorganic chromium alone. Calf morbidity and response to antibiotic treatment were not influenced by dietary treatment. Serum iron and total iron-binding capacity were increased for healthy calves receiving the inorganic chromium-plus-niacin treatment. No consistent response to treatment was observed for serum cortisol or glucose concentrations nor for plasma ascorbate, protein, urea, and ammonia concentrations.

Interest generated from the work conducted at the University of Guelph, Ontario, led to similar studies at other sites in North America. Three chromium supplements, $CrCl_3$, high-chromium yeast, and CrNic, were compared with respect to growth, glucose metabolism, and immune responses of stressed feeder calves (Kegley and Spears, 1995). Chromium was added at 0.4 mg/kg DM to a corn-silage-based diet containing 0.66 mg Cr/kg DM. Steer performance was not influenced by treatment during this 56-day study. Serum cortisol did not respond to treatment. Serum insulin concentrations were greater in cattle receiving the CrNic than in cattle receiving the other chromium supplements at 15 and 30 minutes after glucose infusion; and they were greater (at 30 minutes after infusion) than were those in animals fed control diets. Steers fed high-chromium yeast tended to have a greater inflammatory response to an intradermal injection

of phytohemagglutinin than did control steers. The inflammatory response of steers fed the other chromium supplements was significantly less than that of steers fed high-chromium yeast. Kegley and Spears (1995) concluded that the form of dietary chromium affects its function in cattle. Moreover, CrNic increased total serum IgG, and peripheral lymphocytes isolated from these steers had a greater blastogenic response to phytohemagglutinin than did lymphocytes from steers fed diets supplemented with $CrCl_3$.

Mathison and Engstrom (1995) evaluated the effect of chromium supplementation on rate and efficiency of weight gain and morbidity of steer calves during the initial 28 days in a feedlot under well-managed or stress-imposed conditions. Supplemental chelated chromium resulted in total chromium intake ranging from 0.69 to 0.83, and from 0.57 to 0.83 mg/kg DM, respectively, for unstressed and stressed calves during the 28-day trial. Chromium supplementation did not affect any of the criteria measured in this trial. A subsequent growing-finishing trial in which a diet of barley silage and barley grain was supplemented with chromium at 0.5 mg/kg DM showed no improvement in DM intake, growth, feed efficiency, carcass characteristics, or morbidity in steers as a result of chromium supplementation.

Acute-phase response, as indicated by serum haptoglobulin and total hemolytic complement activity, was measured in 72 steer calves that had been stressed by going through a sale barn marketing process (Wright et al., 1995). Calves fed a corn-silage-based diet containing 0.32 to 1.05 mg Cr/kg DM supplemented with 0.14 mg Cr/kg DM as chelated chromium had lower serum haptoglobulin concentrations 7 days after arriving at the feedlot than did the calves whose feed was not supplemented. Morbidity among the calves was greatest at that time. By day 14, however, no effects of chromium supplementation on serum haptoglobulin were observed, and the increase in complement activity observed with the control calves was not evident with chromium-supplemented calves.

Chang et al. (1996) worked with the same calves to determine the effect of a diet supplemented with 0.14 mg Cr/kg diet on antibody response to vaccination for IBR, PI-3, bovine respiratory syncytial virus (BRSV), bovine viral diarrhea (BVD), and *P. hemolytica* on days 0 and 21 after arrival at the feedlot. Blood samples were taken on days 0, 28, and 35 for determination of antibody titers. Supplemental chromium increased the titer for BVD only on day 28 and caused no effect on antibody responses to other vaccines.

Chang et al. (1996) conducted a second trial with another group of market-transport-stressed steer calves to determine the effect of 0.75 mg Cr/kg DM as chromium yeast on antibody responses to IBR, PI-3, BRSV, BVD, and *P. hemolytica*. The basal diet for this trial contained 75 percent alfalfa silage and 1.45 mg Cr/kg DM. No changes in antibody titers were caused by chromium supplementation. Calves also were injected with 2 mg ovalbumin at vaccination (days 0 and 21). Supplemental chromium enhanced the primary response to this

protein antigen and tended to increase the secondary response as well. Results of these two experiments (Chang et al., 1996) and those obtained by Burton et al. (1994) illustrate the inconsistent immune responses observed in stressed calves fed chromium supplements.

Kegley et al. (1997) observed that calves fed 0.4 mg Cr/kg DM as CrNic for 56 days before transportation, handling, fasting, and vaccination stress, tended to have improved ADG for the subsequent 80-day feeding period. Serum total IgG was decreased, but rectal temperature and antibody response to IBR or porcine red blood cells were not affected by chromium supplementation.

Sheep

Some of the earliest work to suggest a requirement for chromium by ruminants was related to the investigation of a stimulatory factor in cane molasses (Britton et al., 1968). Lambs were fed a molasses-free control diet, a molasses supplement, a molasses ash supplement, or 0.037 mg Cr/day as $CrCl_3$. Lambs fed the molasses ash and chromium-supplemented diet had increases in nitrogen utilization similar to those of lambs receiving the molasses supplement and greater than lambs fed the control diet. They did not exhibit improved ability to digest fiber, as had been observed for the molasses-supplemented group.

Supplementation of 0.25 mg Cr/kg DM as CrPic did not influence ADG, DMI, or nitrogen utilization of female lambs (38 ± 2.7 kg body weight) that were fed a basal diet containing less than 1 mg Cr/kg DM for 85 days (Kitchalong et al., 1995). Prefeeding and three-hour postprandial blood samples collected from lambs on three occasions during the study showed similar plasma albumin, total protein, urea nitrogen, glucose, insulin, glucagon, T_3, and T_4 concentrations, irrespective of dietary chromium concentration. A 17 percent decrease in plasma total cholesterol was observed in the chromium-supplemented lambs during the first week but not at weeks 7 or 11. Plasma NEFA concentrations were 21.7 percent less, and the extent of decline in NEFA after feeding was substantially less for lambs fed the chromium-supplemented diet. Carcass data indicated no treatment effects on carcass weight or on heart and kidney weights expressed as a percentage of body weight of the lambs. Chromium-supplemented lambs had lower lean carcass yields and lower liver weights, and they tended to have less fat over the tenth rib ($P = 0.09$) and decreased pelvic fat ($P = 0.15$) than did their control counterparts. Samsell and Spears (1989), using low- and high-fiber diets containing 0.175 and 0.295 mg Cr/kg diet DM, respectively, noted that supplemental chromium (0.01 mg/kg DM as $CrCl_3$) did not influence plasma glucose or insulin concentrations and did not affect plasma glucose clearance when lambs were given a glucose tolerance test. A 48-hour fasting period resulted in reduced plasma glucose concentrations for lambs fed the low-fiber diet. A second study conducted by

Samsell and Spears (1989) showed that serum NEFA was decreased by chromium addition when lambs were fed the high-fiber diet.

In contrast to results with female lambs (Kitchalong et al., 1995), Samsell and Spears (1989) observed that supplementing 0.01 mg Cr/kg DM as $CrCl_3$ reduced fasting plasma glucose concentrations in male lambs fed a low-fiber diet. The 48-hour fasting period for the Samsell and Spears (1989) study was much longer than that imposed by Kitchalong et al. (1995).

Williams et al. (1994) evaluated the effects of supplementing 0 or 0.80 mg Cr/kg DM as CrPic to the diet of 29 kg lambs exposed to thermal stress. Lambs received a basal diet of corn silage and soybean meal (78 to 16 ratio, DM basis) at 2.7 percent body weight. Basal diet chromium levels were not determined. Chromium supplementation did not influence nitrogen, potassium, or zinc retention by lambs. The authors reported a yeast culture by chromium interaction for serum glucose concentrations in lambs.

DePew et al. (1996) fed diets with or without 0.5 mg supplemental Cr/kg DM as CrPic to lambs for 42 days. Chromium supplementation did not affect performance of the lambs, and no effects of chromium were observed for plasma glucose, NEFA, triglycerides, cholesterol, or insulin when lambs were fed or fasted.

Sano et al. (1996) conducted an isotope study to determine the effect of supplemental chromium on whole-body kinetics of glucose, lactate, and propionate in rams fed a high-grain diet. This study showed an increase in the percentage of glucose derived from propionate but no other differences in plasma concentrations or turnover rates of glucose. Rams were given either a control diet with an undetermined amount of chromium or the control diet with 0.5 mg supplemental Cr/kg DM as chelated chromium.

The work of Samsell and Spears (1989), Kitchalong et al. (1995), and Sano et al. (1996) suggested that chromium supplementation exerts subtle effects on carbohydrate and lipid metabolism in sheep. The significance of these effects, however, is not clear. The basal diet chromium concentrations were not always provided by the researchers, and the chromium supplementation concentrations used in the work conducted with sheep ranged from 0.01 to 0.80 mg/kg DM. The results of studies involving sheep are summarized in Table 3-3.

Summary

Availability of chromium contained in commercial diets fed to ruminant animals is not known. Further efforts need to be directed toward the determination of chromium concentrations and chromium availability in feedstuffs and chromium supplements offered to ruminant animals. A range of chromium supplements including CrNic, $CrCl_3$, CrPic, chelated chromium, and high-chromium yeast have been used in ruminant studies. Comparative studies have been limited and thus little is known about the relative availability of these sources for

TABLE 3-3. Influence of Supplemental Dietary Chromium on Sheep[a]

Reference	Dietary Cr Concentration per kg DM (Source)	Body Weight at Start and Duration of Experiment	Improved Growth Rate	Improved Feed Efficiency
Britton et al. (1968)	Basal diet = unknown; Cr diet = 0.037 mg/d and basal (molasses ash or $CrCl_3$).	Growing lambs	ND	ND
Samsell and Spears (1989)	Basal diet, low fiber = 0.175 mg; Basal diet, high fiber = 0.295 mg; Cr diet, low fiber = 0.185 mg; Cr diet, high fiber = 0.305 mg; ($CrCl_3$).	16 lambs - 45 kg 28-d trial	ND	ND
Samsell and Spears (1989)	Basal diet, low fiber = 0.175 mg; Basal diet, high fiber = 0.295 mg; Cr diet, low fiber = 0.185 mg; Cr diet, high fiber = 0.305 mg; ($CrCl_3$).	16 lambs - 50 kg 28-d trial	ND	ND
Williams et al. (1994)	Basal diet = unknown; Cr diet = basal + 0.8 mg (CrPic).	24 lambs - 29 kg heat stressed	ND	No effect.
Kitchalong et al. (1995)	Basal diet = <1 mg; Cr diet = basal + 0.25 mg (CrPic).	24 lambs - 38 kg 85-d trial	ND	ND
DePew et al. (1996)	Basal diet = unknown; Cr diet = basal + 0.5 mg (CrPic).	24 lambs - 33 kg 42-d trial	No effect.	ND
Sano et al. (1996)	Basal diet = unknown; Cr diet = basal + 0.5 mg (chelated Cr).	6 rams 28-d trial	Yes - no statistics provided.	ND

[a]Definitions of abbreviations used in table:

Cr - chromium
$CrCl_3$ - chromium chloride
CrPic - chromium picolinate
d - day
DM - dry matter
FFA - free fatty acids
h - hour
HDL - high density lipoprotein
i.v. - intravenous
K - potassium
N - nitrogen
ND - not determined
NEFA - nonesterified fatty acids
wk - week
Zn - zinc

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	ND			↑ N balance.
ND	ND	No effect on plasma glucose, insulin, total or HDL cholesterol.	No effect on glucose clearance during glucose tolerance test. ↓ fasting (48 h) glucose for low fiber diet.	
ND	ND	↓ serum FFA at 3 h post feeding. No effect on plasma glucose, or insulin.		
ND	ND	No effect on plasma glucose.		No effect on N, K, Zn balance, N or ability to digest fiber.
ND	↓ pelvic fat in wethers, $P = 0.15$.	No effect on total cholesterol, albumin, total protein, T_3 and T_4, glucose, urea N. ↓ NEFA.	No effect on glucose clearance rates during tolerance tests. ↑ insulin and ↓ glucose in wk 2 but not wk 10 of i.v. insulin challenge test.	No effect on N balance.
ND	ND	ND	No effect on fed or fasting plasma glucose, NEFA, triglycerides, cholesterol, insulin.	
ND	ND			

ruminants, although it should be noted that performance responses observed to date are limited to work conducted with CrPic, high-chromium yeast, and chelated chromium.

The literature does not support a general recommendation for chromium supplementation of commercial ruminant diets. Research efforts, however, have identified two situations in which chromium supplementation might have commercial application: newly arrived feedlot cattle and first-lactation dairy cattle during the transition period.

Three of eight studies conducted with newly arrived feedlot cattle subjected to the stresses of transportation, mixing, and handling have demonstrated a positive performance response in the initial weeks following arrival. Reduced morbidity and plasma cortisol levels due to chromium supplementation have been reported; however, the responses have not been consistent. Efforts to establish an enhanced immune response using response to vaccination or to foreign proteins have not shown consistent results, although blastogenesis in peripheral blood mononuclear cells cultured with T-lymphocyte mitogens was greater in cattle fed chromium-supplemented versus unsupplemented diets.

The transition period, including parturition, early lactation, and late lactation, is known to cause metabolic stress in dairy cows. There is evidence that chromium supplementation during this transition period can improve performance for first-lactation cows. This response was not observed for multiparous cows offered similar levels of supplemental chromium. Improved performance may be associated with a shift in ketone body metabolism, as reduced circulating ketone levels in cows consuming chromium supplemental diets have been observed in several studies. There is also evidence that chromium supplementation of first-lactation, postpartum cows will reduce sensitivity to insulin. As was observed for the receiving feedlot animals, chromium supplementation enhanced blastogenic responses of the peripheral blood mononuclear cells obtained of early-lactation cows.

Controlled studies will be required to establish the specific role of chromium for cattle undergoing stress and to establish recommendations for rates of chromium supplementation where its use may be appropriate.

NONRUMINANTS

Growing-Finishing Swine

Most of the recent research on dietary chromium supplementation for swine has focused on performance and carcass characteristics of growing swine. An early study by Steele et al. (1977), however, showed that a synthetic, chromium-containing glucose tolerance factor (GTF) potentiated the hypoglycemic response in swine after an intravenous insulin challenge, showing that the chromium-containing substance was biologically active, as had been described for humans

and rats by Mertz et al. (1974). Subsequent research evaluated the effects on swine of supplemental dietary chromium, as inorganic $CrCl_3$ or as organically bound CrPic. Page et al. (1990) fed diets containing 0, 30, or 60 mg Cr/kg as $CrCl_3$ to growing swine for 43 days. They observed no influence of chromium on growth rate or carcass traits, including backfat thickness, loin eye area, or percentage muscling. Concentrations of several constituents of blood serum also were unaffected. In a subsequent study, Page et al. (1992a,b) fed swine of two genotypes diets containing 0 or 200 μg Cr/kg as CrPic for 102 days. Average daily gain and feed intake were increased by dietary CrPic, whereas feed efficiency, carcass dressing percentage, and concentrations of several constituents of blood serum were unaffected. Feeding 200 μg of Cr/kg decreased tenth-rib fat and increased loin eye area and percentage of carcass muscling. Serum cholesterol concentration also was reduced by dietary CrPic. Page et al. (1992a) reported that the favorable effects of CrPic on swine carcass traits and serum cholesterol occurred irrespective of genotype. In a follow-up study, Page et al. (1992b) found that feeding CrPic did not affect pork quality in terms of tenderness (shear force) or cooking loss.

In 1993, Page et al. reported the results of three experiments in which chromium, supplied as CrPic, was evaluated at concentrations ranging from 25 to 800 μg/kg of feed. The basal diet contained 735 μg Cr/kg. Effects of CrPic on weight gain and feed efficiency were inconsistent among experiments. In two experiments, feeding 100 or 200 μg Cr/kg decreased tenth-rib backfat and increased loin eye area and percentage of muscling. A comparison of $CrCl_3$ and CrPic as dietary sources of chromium in one experiment showed that CrPic was effective in improving carcass traits, whereas $CrCl_3$ was not. Lien et al. (1993) fed diets containing 0, 200, 400, or 800 μg Cr/kg as CrPic to swine and reported no effects on ADG or feed efficiency. Feeding 400 or 800 μg Cr/kg reduced backfat thickness and serum glucose concentration. Serum insulin concentrations were directly related to dietary CrPic, and serum lipid concentration was reduced by either 200 or 400 μg CrPic/kg of feed; serum cholesterol was not affected.

Berrio et al. (1995) conducted in vitro studies on red blood cell ghosts and adipocytes obtained from swine fed 0 or 200 μg supplemental Cr/kg of diet as CrPic. They reported that insulin binding was increased with red blood cell ghosts obtained from swine fed 200 μg Cr/kg of diet in two experiments. Insulin binding to adipocytes from swine fed 200 μg Cr/kg of diet also was increased in one experiment. These observations indicated that chromium may play a role in insulin action in swine.

Evock-Clover et al. (1993) evaluated dietary CrPic for swine treated or not treated with pituitary-derived porcine somatotropin (pST). Feeding 300 μg Cr/kg of diet did not affect rate of growth, feed efficiency, or carcass composition of swine reared from 30 to 60 kg body weight, irrespective of pST treatment. Dietary CrPic, however, decreased serum insulin and glucose concentrations and normalized the increase in glucose and insulin resulting from pST treatment. No

interactions between dietary CrPic and pST were observed. In a subsequent study, Evock-Clover and Steele (1994) fed 300 μg Cr to swine reared from 60 to 90 kg body weight. Dietary CrPic had no effect on growth rate, feed efficiency, or nutrient partitioning. Feeding CrPic increased the percentage of carcass protein of swine treated with recombinant pST.

The influence of 200 μg Cr/kg of diet, supplied as $CrCl_3$, CrPic, or CrNic, on immune response of young swine was evaluated by Van Heugten and Spears (1994). Dietary chromium increased weight gain and feed intake of swine not challenged by an injection of *E. coli* lipopolysaccharide (LPS). Chromium, however, did not prevent the adverse effects of LPS on swine performance, and no clear-cut effects of chromium supplementation on other measures of immune response were discernible.

Boleman et al. (1995) conducted an experiment to evaluate the effects of feeding 200 μg Cr/kg of diet as CrPic on growth and carcass composition of swine and on sensory characteristics of pork. Feeding CrPic reduced weight gain and feed intake of swine reared from 30 to 103 kg. Carcass data showed that feeding CrPic during the late growing and finishing periods decreased body fat and increased the percentage of muscle of swine. Cooking loss, drip loss, shear force, and sensory characteristics of meat were not affected by chromium treatment. Smith et al. (1994) reported that 200 μg Cr/kg of diet as CrNic had no effect on weight gain and feed efficiency of swine but that backfat thickness was decreased by chromium treatment.

Harper et al. (1995) reported that feeding 200 μg Cr/kg of diet as CrNic improved ADG of weaning pigs and of swine during the finishing period. Chromium supplementation also reduced backfat thickness but had no effect on longissimus muscle cross-sectional area. Harris et al. (1995) conducted a similar study and found that feeding 200 μg Cr/kg of diet as CrPic did not affect performance or carcass characteristics of swine fed either adequate or low-protein diets.

Amoikon et al. (1995) conducted two experiments to determine the effects of dietary chromium on performance, glucose tolerance, and plasma metabolites of swine. Feeding 200 μg Cr/kg of diet as CrPic to swine weighing 21 to 25 kg for 19 or 21 days had no effect on weight gain or feed efficiency. Concentrations of plasma cholesterol were increased, whereas plasma NEFA, urea nitrogen, and insulin concentrations were decreased in swine fed chromium. During a glucose tolerance test, the glucose disappearance rate increased and glucose half-life decreased in swine fed chromium; insulin kinetics were not affected. Dietary chromium did not affect plasma growth hormone concentrations.

Mooney and Cromwell (1995) reported that when 200 μg Cr (CrPic)/kg was fed to swine throughout the growing-finishing periods, gain in body weight, gain in muscle mass, and accretion rate of muscle increased, whereas gain in fat and fat accretion rate decreased. The net effects of dietary chromium were swine carcasses that contained an increased percentage of muscle and a decreased percentage of fat. Similar observations were reported by Lindemann et al. (1995b)

in that the inclusion of 200 μg Cr/kg of diet as CrPic increased muscling and decreased backfat of swine. In contrast, Ward et al. (1995) reported that feeding eight sources of chromium, including CrPic and $CrCl_3$, at 200 μg Cr/kg of diet for 77 days had no effect on carcass traits and did not affect growth rate or feed efficiency. Determination of the concentrations of several serum constituents revealed only one inconsistent effect of dietary chromium: Reagent grade CrPic reduced serum NEFA, whereas a commercial source of CrPic had no effect on serum NEFA. Ward et al. (1994) measured the effect of dietary chromium on insulin binding by hepatic plasma membranes in vitro. Feeding swine 200 μg Cr/kg of diet as CrPic for 42 or 64 days did not affect in vitro insulin binding by hepatic plasma membranes.

Wenk et al. (1995) compared the effect of 500 μg of Cr/kg of diet given as $CrCl_3$, CrPic, or high-Cr yeast on swine that weighed 27.4 kg each at the start of the experiment. The experiment was terminated when each pig weighed 106.5 kg. Supplementing the diet with 500 μg Cr/kg as $CrCl_3$ improved rate of gain and feed efficiency, and there was a trend for improved performance by swine fed CrPic or high-Cr yeast. No effects of supplemental Cr were observed for carcass fatness.

Several reports of studies on the effects of feeding supplemental chromium to growing swine were presented at the 1996 meeting of the American Society of Animal Science. Mooney and Cromwell (1996) fed 200 μg Cr/kg of diet as CrPic to barrows of medium-lean-gain and high-lean-gain genetic backgrounds. Dietary chromium did not affect performance when barrows were reared from 21 to 104 kg body weight. In addition, supplemental chromium did not improve carcass characteristics, chemical composition, or accretion rates of carcass protein or fat, regardless of the genetic potential of the swine. LeMieux et al. (1996) report that supplemental CrPic did not alter performance of weaning pigs whether fed with or without supplemental betaine or zinc. Harper and Kornegay (1996) also observed no effects of dietary CrPic supplementation on performance or carcass quality of swine grown from weaning to 102 kg body weight in the presence or absence of 5 percent dietary fish meal. In contrast, Lien et al. (1996b) reported improvements in rate of gain of swine fed 200 μg Cr/kg of diet as CrPic for 95 days. This effect was accompanied by reductions in serum triglycerides and low-density lipoprotein cholesterol and by increases in serum insulin, creatinine, and high-density lipoprotein cholesterol. Chromium picolinate did not affect backfat thickness. Dietary $CrCl_3$ (200 μg Cr/kg) did not elicit the responses observed with CrPic. Yi et al. (1996) fed gilts averaging 73 kg each in body weight 105 μg Cr/head as CrPic during a 160-day growing period. Carcass data obtained when gilts weighed 114.5 kg showed that supplemental chromium increased loin eye area and percentage lean but did not affect other carcass traits, compared with carcasses of swine given no supplemental dietary chromium. Supplemental chromium had no effect on corpus luteum numbers in the gilts.

Min et al. (1997b) fed 20 kg swine diets supplemented with 0, 100, 200, or

400 μg Cr/kg as CrPic for 103 days. They reported that rate of gain was not affected by dietary chromium, but feed efficiency was improved slightly by feeding 200 or 400 μg Cr/kg of diet, and carcass fat was reduced by 200 μg Cr/kg of diet. In vitro tests also showed that lipolytic activity and protein synthesis of adipocytes obtained from swine fed 200 μg Cr/kg of diet were increased over that of the control groups. Serum triglycerides were decreased by feeding 200 or 400 μg Cr/kg of diet, but glucose, insulin, cholesterol and NEFA concentrations in serum were unaffected. Min et al. (1997a) fed 60 kg swine a diet supplemented with 200 μg Cr/kg as CrPic in combination with daily injections of pST. Treatment with pST improved weight gain and feed efficiency during the 52-day experiment. Carcass fat also was decreased by pST. Chromium supplementation did not alter the effects of pST on performance or carcass traits, nor did chromium affect lipogenic or lipolytic activity of adipose tissue of the swine.

In an experiment involving 1,000 pigs, Lindemann and Purser (1997) fed diets supplemented with 0 or 200 μg Cr/kg as CrPic. They reported that chromium supplementation improved weight gain and feed efficiency of barrows but not of gilts. Percentage lean in the carcass of barrows also was increased by supplemental chromium. Responses of barrows to chromium supplementation were evident whether 200 μg Cr/kg was fed throughout the growout period or whether 200 μg Cr/kg was fed in the first grower diet followed by 100 μg Cr/kg in the next three grower diets. Crow et al. (1997) did not observe any effects of feeding diets containing 100, 200, or 500 μg Cr/kg as CrPic on performance or carcass composition of swine reared from 45 to 109 kg body weight. They did, however, report that the blood insulin-to-glucose ratio increased linearly as dietary chromium increased. Crow and Newcomb (1997) also found that weight gain, feed efficiency, loin eye area, and backfat thickness were not influenced by supplementing diets with 200 μg Cr/kg as CrPic. In this experiment, no effect of supplemental chromium on blood insulin-to-glucose ratio was detected.

Mooney and Cromwell (1997) compared the effects of dietary sources and concentrations of supplemental chromium (200 or 400 μg Cr/kg as CrPic versus 5 or 25 mg Cr/kg as $CrCl_3$) on performance and carcass traits of swine. They reported that 200 μg Cr/kg as CrPic increased ADG and feed intake and increased lipid accretion rate in the carcass compared with feeding no supplemental chromium. Other carcass traits, however, were not affected by chromium source or concentration. Results of a second experiment showed that inclusion of either 200 μg Cr/kg as CrPic or 5 mg Cr/kg as $CrCl_3$ had no effect on performance or backfat thickness of swine but both chromium sources increased loin eye area. Percentage of muscle tissue in carcasses and accretion rate of muscle tissue were increased and percentage fat was decreased in swine fed supplemental chromium, with CrPic being more effective than $CrCl_3$. No changes in blood metabolites occurred as a result of supplemental chromium in either experiment.

Kornegay et al. (1997) conducted four experiments to determine the influ-

ence of supplemental CrPic on nutrient digestibility and retention in swine. Animals weighed 26.3, 13.2, and 30.9 kg at the start of Experiments 1, 2, and 3, respectively. Data obtained from the three experiments, each using 12 barrows, were pooled for statistical evaluation. Supplementation of diets with 200 μg Cr/kg of diet as CrPic improved dry matter digestibility and absorption of nitrogen as compared with those of swine fed no supplemental chromium. Carcass analysis also showed that supplemental chromium increased the area of the longissimus muscle. In Experiment 4, 200 μg Cr/kg of diet again improved dry matter digestibility and nitrogen absorption during the finishing period. Although there were no effects of supplemental chromium on nitrogen retention in the four experiments, the data showed that dietary nutrient availability was improved when chromium was added to the diet of swine.

The reasons for the inconsistencies in responses of growing swine to dietary chromium supplementation observed by several researchers are not known. Anderson (1994) and Mowat (1994) suggested, on the basis of studies with humans and cattle, that animals respond most favorably to supplemental chromium when exposed to stress, and that the inconsistent responses to chromium reported in the literature were related to poorly controlled stress conditions. Results of recent research with swine in which limited floor space was used to cause stress, however, have not supported their suggestion. Siberio et al. (1996a) used 21-day-old pigs in a 35-day feeding study to determine the influence of dietary chromium, supplied by a high-chromium yeast, on immune status when swine were provided with up to 59 percent less than the recommended amount of floor space. They reported that feeding 500 μg Cr/kg of diet, with or without supplemental copper, had no effect on performance or immune response of young swine reared in a crowded environment. Siberio et al. (1996b) also conducted a 15-day trial with 21-day-old pigs in which the animals were fed diets supplemented with 0, 500, or 1,000 μg Cr/kg of diet to determine the effect on retention of copper or zinc, which has been shown to increase during stress. Stress was imposed by providing only 0.15 m^2 of floor space per pig. Supplemental chromium, provided as high-chromium yeast, had no effect on retention of copper or zinc by the stressed baby pigs.

Ward et al. (1997) used a complete factorial arrangement of two supplemental chromium concentrations (0 and 400 μg/kg of diet as CrPic), two dietary lysine (protein) concentrations (80 and 120 percent of levels recommended by National Research Council, 1988) and two floor space allowances (0.025 and 0.035 m^2/kg body $weight^{0.67}$) in a study with growing swine. Supplemental chromium improved weight gain and feed efficiency of swine fed the 80 percent lysine diet but slightly impaired performance of swine fed the 120 percent lysine diet. There were no indications that supplemental chromium alleviated the adverse effects of the stress of inadequate floor space on pig performance. An interaction between supplemental chromium and floor space, however, was ob-

served in that thickness of tenth-rib fat of swine reared in inadequate floor space (0.025 m^2/kg body weight$^{0.67}$) was increased by chromium supplementation.

Sows

As a follow-up to feeding 200 μg Cr/kg of diet as CrPic to growing swine, Lindemann (1995b) continued feeding 200 μg Cr/kg to gilts through two parities. They found that sows fed chromium had more total pigs born, more live pigs born, and more pigs at 21 days than did sows fed an unsupplemented diet. They also observed that changes in serum insulin concentrations and insulin-to-glucose ratios in the sows indicated an improvement in the efficiency of insulin action as a result of chromium supplementation.

In another study, Lindemann et al. (1995a) used gilts that had received no previous dietary chromium supplementation. The treatments evaluated were no supplemental chromium, 200 μg Cr/kg of diet as CrPic during nonlactation only, 200 μg Cr/kg during lactation only, or 200 μg Cr/kg during both periods. No effects of supplemental CrPic were observed on total pigs born, live pigs born, or pigs alive at 21 days. The authors surmised that these results, which did not support those of Lindemann et al. (1995b), provided evidence of the need for chromium supplementation early in the gilt's life (from 18 kg body weight).

No information describing chromium toxicity for swine was found in the literature.

Summary

Reported responses of growing-finishing swine to supplemental dietary chromium have been inconsistent (Table 3-4). Statistically significant ($P \leq 0.10$) improvements in growth rate as a result of supplementing diets with 200 to 500 μg Cr/kg as CrPic were reported in 11 of 31 studies. Similarly, feed efficiency was improved by adding CrPic to diets in 8 of 31 studies. In addition to reports of improved performance of swine, favorable effects of added chromium on selected carcass traits have been observed. When supplemental dietary $CrCl_3$ or CrPic was used, increases in carcass leanness (muscling) were reported in 9 of 24 experiments and decreases in carcass fat were reported for 11 of 26 experiments. Research to determine the influence of added dietary CrPic on reproductive performance has been meager and the results were inconsistent: Litter size was increased in one experiment but reproductive traits were unaffected in a second trial. There also is limited information on metabolic changes that could be caused by supplementing swine diets with CrPic. Results of several experiments have shown that supplemental CrPic induced a hypoglycemic response or improved insulin efficiency in swine and, in some instances, in vitro lipogenic and lipolytic activities of adipocytes and livers were altered when swine were fed supplemental dietary chromium. *Although responses of swine to supplemental chromium*

have been inconsistent, there is an increasing amount of evidence indicating that chromium may favorably alter metabolism of swine under some circumstances, with resultant improvements in growth rate, carcass traits, and reproductive performance. The need for chromium supplementation of practical swine diets, however, depends on the chromium status of the animals, the amount of bioavailable chromium in the feedstuffs, and exposure of the animals to certain environmental stresses. Thus, a decision to use supplemental chromium in practical swine diets must be based on the potential benefits in individual circumstances versus the cost of supplementation.

Poultry

Interest in chromium as a dietary supplement for poultry dates to Hill and Matrone's 1970 study reporting that adding chromium to chick diets decreased the toxicity of dietary vanadium. They found that the growth-depressing effects of 20 mg vanadium/kg of diet decreased as dietary chromium (as $CrCl_3$) concentrations were increased from 500 to 2,000 mg Cr/kg of diet, although total recovery of growth rate was not achieved. They also found that rates of oxidative phosphorylation (P:0 ratio) of chick liver tissue were reduced by vanadium and that the in vitro effects of vanadium on the P:0 ratio could be prevented by including chromium in the medium at a 10:1 ratio to vanadium. Hafez and Kratzer (1976) confirmed the observations of Hill and Matrone (1970) that inclusion of 1,000 mg Cr (as $CrCl_3$) alleviates most of the slower growth and mortality of chicks fed diets containing 50 or 100 mg of vanadium/kg of diet.

Interest in dietary chromium for laying hens was stimulated when Jensen et al. (1978a,b) reported a favorable effect of chromium (as $CrCl_3$) on albumen quality (Haugh unit score) of eggs. Jensen et al. (1978b) suggested that dietary chromium may be necessary for maintaining the normal physical state of the egg albumen. This suggestion was supported by data showing that 5 mg Cr/kg of diet as $CrCl_3$ prevented the adverse effect on Haugh unit score of 10 mg vanadium/kg of diet. In subsequent research, Jensen and Maurice (1980) were unable to show that chromium fed in the absence of dietary vanadium was essential for optimal albumen quality of eggs, although they confirmed that chromium alleviated the deleterious effects of vanadium on albumen quality. They also reported that dietary chromium had no effect on glucose tolerance of White Leghorn hens, contrary to what has been reported for humans and rats (Mertz, 1992).

Not all research done with laying hens has shown that dietary chromium overcomes the adverse effects of vanadium on albumen quality. Sauveur and Thapon (1983) evaluated $CrCl_3$ and a natural source of chromium and reported that neither source influenced the adverse effects of 5 or 30 mg vanadium/kg of diet on albumen quality. Similarly, Benabdeljelil and Jensen (1989, 1990) were unable to show that 5 to 50 mg Cr/kg of diet improved albumen quality of eggs produced by hens fed 10 or 30 mg vanadium/kg. The reasons for the disparate

TABLE 3-4. Influence of Supplemental Dietary Chromium on Swine[a]

Reference	Concentration of Supplemental Dietary Cr per kg (Source)	Body Weight at Start and End and Duration of Experiment	Improved Growth Rate[b]	Improved Feed Efficiency
GROWING PIGS				
Steele et al. (1977)	Tested a Cr-containing GTF	6, 30 kg pigs/trt for in vivo study. Adipose tissue samples from 8 pigs for in vitro study.	ND[b]	ND
Page et al. (1990)	30, 60 mg ($CrCl_3$)	45 pigs; Start = 61.7 kg; 43-d trial.	No	No
Page et al. (1992a,b)	200 µg (CrPic)	50 pigs; Start = 21.9 kg; 102-d trial.	Yes	No
Evock-Clover et al. (1993)	300 µg (CrPic)	24 barrows; Start = 30 kg; End = 60 kg.	No	No
Lien et al. (1993)	200, 400, 800 µg (CrPic)	32 pigs; Start = ~50 kg; End = ~100 to 110 kg; 75-d trial.	No	No
Page et al. (1993)	Experiment 1 and 2 = 25 to 800 µg (CrPic); Experiment 3 = 200 µg (CrPic or $CrCl_3$); Basal diet = 735 µg Cr.	236 pigs in 3 experiments; Start = 37.8, 30.5, 22.4 kg; 73-, 83-, and 98-d trials.	Inconsistent among 3 experiments.	Inconsistent among 3 experiments.
Evock-Clover and Steele (1994)	300 µg (CrPic)	50 barrows; Start = 20 kg; End = 90 kg.	No	No
Smith et al. (1994)	200 µg (CrPic)	64 pigs; Start = 34 kg; End = 102 kg.	No	No
Van Heugten and Spears (1994)	200 µg ($CrCl_3$, CrPic, CrNic)	96, 21-d-old pigs; 31-d trial.	Yes	Yes
Ward et al. (1994)	200 µg (CrPic)	Exp. 1:42 to 84 d; Exp. 2:56 to 120 d.	No	No

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	ND	ND	Yes	
No	No	None	ND	
Yes	Yes	Reduced cholesterol, increased triglycerides.	ND	Cooking qualities of pork not affected.
No	No	Reduced insulin and glucose, normalized serum glucose and insulin in pST pigs.	ND	
ND	Yes	Reduced glucose and lipid, increased insulin.	ND	
Yes	Yes	ND	ND	
No	No	ND	ND	
ND	Yes	ND	ND	
ND	ND	ND	ND	No consistent benefit for immune response.
ND	ND	ND	ND	Reduced insulin binding by hepatic plasma membranes in vitro.

TABLE 3-4. Continued

Reference	Concentration of Supplemental Dietary Cr per kg (Source)	Body Weight at Start and End and Duration of Experiment	Improved Growth Rate[b]	Improved Feed Efficiency
Berrio et al. (1995)	0 and 200 µg (CrPic) Isolated adipocytes from subcutaneous adipose tissue and red blood cell ghosts.	Not stated.	ND	ND
Boleman et al. (1995)	200 µg (CrPic)	24 pigs; Start = 20 or 55 kg; End = 103 kg.	No; reduced growth.	No
Harper et al. (1995)	200 µg (CrPic)	144 pigs in 3 experiments; Start = 7.3 kg; 35-d trial.	Yes	Yes
Harris et al. (1995)	200 µg (CrPic)	120 pigs; Start = 10.4 kg; End = 109.7 kg.	No	No
Lindemann et al. (1995a,b)	200 µg (CrPic)	Two experiments; Start = 40.9 and 14.5 kg; End = 98 and 98 kg.	No	Yes
Mooney and Cromwell (1995)	200 µg (CrPic)	14 pigs; Start = 27 kg; End = 109 kg.	Yes	No
Ward et al. (1995)	200 µg (CrPic plus seven other Cr sources)	128 crossbred pigs; Start = 18.1 kg; 77-d trial.	No	No
Wenk et al. (1995)	0 and 500 µg Cr from $CrCl_3$, CrPic, or high-Cr yeast	40 gilts; Start = 27.4 kg; End = 106.5 kg.	Yes, with $CrCl_3$. Also, tended to improve with other Cr sources.	Yes, with $CrCl_3$. Also, tended to improve with other Cr sources.
Harper and Kornegay (1996)	200 µg (CrPic)	96 pigs; Start = 5.9 kg; End = 10.2 kg.	No	No
LeMieux et al. (1996)	200 µg (CrPic)	Two experiments; Start = 5.2 and 6.3 kg; End = 9.8 and 12.0 kg; 21-d trials.	No No	No No

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	ND	ND	ND	Cr increased in vitro binding of insulin to RBC ghosts in two experiments and to adipocytes in one experiment.
Yes	Yes	ND	ND	Cooking qualities of pork not affected.
No	Yes	ND	ND	
No	No	ND		
Yes	Yes	ND	ND	
Yes	Yes	ND		
No	No	Slight, inconsistent, e.g., reagent CrPic reduced NEFA but commercial sources did not affect NEFA.	ND	
No	No	ND	ND	
No	No	ND	ND	Factorial with 5% fish meal.
ND	ND	ND	ND	Factorial arrangement with betaine and zinc oxide.
ND	ND	ND	ND	

TABLE 3-4. Continued

Reference	Concentration of Supplemental Dietary Cr per kg (Source)	Body Weight at Start and End and Duration of Experiment	Improved Growth Rate[b]	Improved Feed Efficiency
Lien et al. (1996a,b)	200 µg (CrPic or $CrCl_3$)	36 pigs; Start = ~36 kg; 95-d trial.	Yes with CrPic, not $CrCl_3$.	No
Mooney and Cromwell (1996)	200 µg (CrPic)	Two experiments; 40 medium- and high-lean genotypes and 60 medium-lean gentoypes; Start = 21 kg; End = 104 kg.	No	No
Siberio et al. (1996a)	500 µg (high-Cr yeast)	Start = 21 d, 7.3 kg; 35-d trial.	No	No
Siberio et al. (1996b)	500 and 1,000 µg; Basal diet = 1,440 µg (high-Cr yeast).	21 barrows; Start = 6 kg; 15-d trial.	No	No
Yi et al. (1996)	105 µg Cr/head during 160 days (CrPic).	17 gilts; Start = 73 kg; End = 114.5 kg; 160-d trial.	ND	ND
Crow and Newcomb (1997)	0 and 200 µg (CrPic)	224 pigs; (96 barrows, 128 gilts); Start = 25 kg; End = 109 kg.	No	No
Crow et al. (1997)	0, 100, 200, or 500 µg (CrPic)	191 pigs; (95 barrows, 96 gilts); Start = 45 kg; End = 109 kg.	No	No
Kornegay et al. (1997)	200 µg (CrPic)	Three experiments, 12 barrows in each experiment; Start = 26.3, 13.2, and 30.9 kg in Experiments 1, 2, & 3, respectively.	No	ND
	200 µg (CrPic)	12 barrows in switch-back design; Start = 82 kg.	ND	ND

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
ND	No	CrPic reduced triglycerides and LDL cholesterol; increased insulin, creatinine, HDL cholesterol.	ND	
No	No	ND	ND	
ND	ND	ND	ND	No effect on immune response.
ND	ND	ND	ND	No effect on retention of copper or zinc by stressed pigs.
Yes, increased loin eye area and lean percentage.	No	ND	ND	No effect of Cr on corpus luteum numbers.
No	No	No	ND	No effect of Cr on blood insulin-to-glucose ratio.
No	No	Yes, insulin-to-glucose ratio increased as dietary Cr increased.	ND	
Increased longissimus muscle area.	No	ND	ND	Improved DM digestion and absorption of nitrogen. No significant effect on nitrogen retention.
ND	ND	ND	ND	Improved DM digestion and increased nitrogen absorption ($P <$ 0.06). No effect on nitrogen retention.

TABLE 3-4. Continued

Reference	Concentration of Supplemental Dietary Cr per kg (Source)	Body Weight at Start and End and Duration of Experiment	Improved Growth Rate[b]	Improved Feed Efficiency
Lindemann and Purser (1997)	0, 200 µg from start to finish, or 200 µg in first diet followed by 100 µg in final three diets (CrPic).	1,000 pigs; Start = 26 kg; End = 117.4 kg.	Yes, of barrows not gilts.	Yes, of barrows not gilts.
Min et al. (1997a)	0, 4 mg pST/head daily, and 4 mg pST plus 200 µg Cr (No Cr treatment alone).	72 pigs; Start = 60 kg; End = 105 kg; 52-d trial.	Yes, with pST, but Cr had no additional effect.	Yes, with pST but Cr had no additional effect.
Min et al. (1997b)	0, 100, 200, 400 µg (CrPic)	32 gilts, 32 barrows; Start = 20 kg; 103-d trial.	No	Indicated ($P \leq 0.10$) with 300 and 400 µg.
Mooney and Cromwell (1997)	Exper. 1—0, 200, or 400 µg (CrPic) or 5 or 25 mg ($CrCl_3$).	35 pigs, individually penned; Start = 19.6 kg; End = 43.2 kg; 35-d trial.	Yes, by 200 µg from CrPic ($P<0.07$).	No
Mooney and Cromwell (1997)	Exper. 2—200 µg (CrPic) or 5 mg ($CrCl_3$).	42 pigs, individually penned; Start = 19 kg; End = 109 kg.	No	No
Ward et al. (1997)	400 µg (CrPic)	64 barrows, 64 gilts; Start = 27.2 kg; Dietary lys = 80 or 120% of NRC; Floor space = 0.035 m^2/kg $BW^{0.67}$ versus 0.025 m^2/kg $BW^{0.67}$.	Yes, with 80% lys diet, not with 120% lys diet.	Yes, with 80% lys diet, not with 120% lys diet.
SOWS				
Lindemann et al. (1995a)	200 µg (CrPic)	73 gilts at start. During nonlactation periods of three parities, or during lactation periods of three parities, or during lactation and nonlactation periods of three parities.	No effect on litter size.	ND

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
Yes, of barrows not gilts.	ND	ND	ND	
No effect of Cr.	Yes, with pST but Cr had no additional effect.	ND	ND	No effect of Cr on lipogenic or lipolytic activity in adipose tissues.
No	Yes, with 200 µg.	No	ND	Increased lipolytic activity of adipocytes with 200 µg. Net protein retention by hepatocytes increased by 200 and 400 µg, mainly in gilts.
No	No	ND	ND	200 µg from CrPic increased ($P<0.07$) lipid accretion in carcass.
Yes, by both Cr courses.	Yes, by both Cr courses, with CrPic most effective.	No	ND	Accretion rate of carcass protein was increased and that of carcass fat was decreased by Cr.
No	Cr increased tenth-rib fat of pigs reared with inadequate floor space ($P<0.07$).	Minimal effects on metabolites and hormones.	ND	Cr did not overcome effect of stress.
ND	ND	ND	ND	

TABLE 3-4. Continued

Reference	Concentration of Supplemental Dietary Cr per kg (Source)	Body Weight at Start and End and Duration of Experiment	Improved Growth Rate[b]	Improved Feed Efficiency
Lindemann et al. (1995b)	200 µg or 500/1,000 µg (CrPic)	During growing period and two parities. During growing period only. 39 gilts used.	Increased litter size.	Indicated $P<0.10$

[a]Definitions of abbreviations used in table:

BW - body weight	LDL - low density lipoprotein
Cr - chromium	lys - lysine
CrNic - chromium nicotinate	ND - Not determined
CrPic - chromium picolinate	NEFA - nonesterified fatty acids
DM - dry matter	pST - porcine somatotropin
d - day	RBC - red blood cells
GTF - glucose tolerance factor	wk - week
HDL - high density lipoprotein	

[b]Only statistically significant responses ($P \leq 0.05$) are indicated unless stated otherwise.

Increased Carcass Muscling	Reduced Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response or Improved Insulin Efficiency	Other
Yes	Yes	Reduced serum insulin and insulin:glucose ratio.	ND	

results obtained by these studies as compared with those of Jensen and Maurice (1980) and Jensen et al. (1978a,b) are not evident.

The influence of chromium supplementation on rate of egg production by laying hens also has varied. When $CrCl_3 \cdot 6H_2O$ was used as the supplemental source of chromium, no effects on egg production were observed (Benabdeljelil and Jensen, 1989, 1990; Jensen and Maurice, 1980; Jensen et al., 1978b). However, Southern and Page (1994) reported that including 100 or 200 μg Cr/kg of diet as CrPic enhanced egg production in one of three experiments with White Leghorn laying hens. Lien et al. (1996a) fed diets containing 0, 200, 400, or 800 μg Cr/kg as CrPic and observed no chromium effect on egg production. Although weights of eggs and egg yolks were not affected by dietary CrPic in the Lien et al. (1996a) study, cholesterol concentration in egg yolks and in serum of hens was related inversely to dietary CrPic concentration.

Kim et al. (1997) fed diets containing 0, 200, 400, or 800 μg Cr/kg as CrPic to laying hens for 6 weeks. Supplemental chromium did not affect rate of egg production, but average egg weight was increased and serum cholesterol was decreased for hens fed 400 μg Cr/kg of diet. Dry matter and protein digestibilities were improved when diets were supplemented with 200, 400, or 800 μg Cr.

Working with chicks, Cupo and Donaldson (1987) determined the effects of chromium and vanadium on glucose metabolism and lipid synthesis. They reported that chromium (as $CrCl_3$) and vanadium increased the rate of glucose utilization of liver tissue 16 percent and 33 percent, respectively. Feeding 20 mg Cr/kg or 20 mg vanadium/kg, alone or in combination, enhanced incorporation of ^{14}C glucose into liver fatty acids. They concluded that chromium and vanadium do not always act antagonistically in biologic processes.

Results of research with growing poultry designed to determine the influence of dietary chromium on performance have varied. Baker and Molitoris (1975) found that omission of chromium from a purified, crystalline amino acid diet did not affect growth or feed efficiency of chicks. Steele and Rosebrough (1979, 1981), however, reported that inclusion of 20 mg Cr/kg of diet as $CrCl_3$ increased growth rates of poults fed corn-soybean-meal diets from 7 to 21 or from 1 to 21 days of age, respectively. Steele and Rosebrough (1981) also reported that added dietary chromium accelerated the rate of glucose uptake by chick liver incubated in vitro. Rosebrough and Steele (1981) observed a stimulation of poult growth when 20 mg of chromium were included in a 23 percent crude protein starter diet but not when the diet contained 30 percent crude protein. Supplemental chromium also increased the activity of hepatic glycogen synthetase of poults.

Because of the interest in modifying chromium content of foods, Anderson et al. (1989) fed diets containing 25, 100, or 200 mg Cr/kg of diet as $CrCl_3$ to turkeys for five weeks. Chromium content of tissues increased as dietary chromium increased, with liver tissue accumulating the greatest concentrations.

Because research to determine the influence of dietary CrPic on young poultry was not done until the 1990s, few data are available. Liarn et al. (1993) fed

diets containing 0, 100, 200, 300, or 400 µg Cr/kg as CrPic to broiler chickens from 1 to 56 days of age. The basal starter, grower, and finisher diets used in this study contained 2,600, 1,400, and 71 µg Cr/kg, respectively, as determined by laboratory analysis. Supplemental chromium had no effect on rate of gain, feed efficiency, or carcass composition. In vitro activity of hepatic ATP citrate lyase was increased when 100 or 200 µg Cr/kg of diet were fed but activity of hepatic fructose-1,6-diphosphatase was not. Ward et al. (1993) reported that diets containing 200 or 400 µg Cr/kg of diet as CrPic did not affect weight gain, feed intake, feed efficiency, or protein, fat, or ash content of muscle of 5- to 19-day-old broiler chicks. Feeding 200 µg Cr/kg, however, tended to increase the protein concentration in whole bodies of chicks. Ward and Southern (1995a) determined growth, organ weight, and selected plasma metabolites of broilers as influenced by dietary CrPic in concentrations of 0, 400, 1,600, and 16,000 µg Cr/kg. In one experiment, CrPic did not affect weight gain, feed intake, feed efficiency, plasma insulin, glucagon, NEFA, or liver, heart, or abdominal fat pad weights. Generally, weights of most body parts were not affected consistently by dietary CrPic. Rate of gain and feed efficiency of chicks improved during the last 7 days of a second experiment of 49 days duration. In a subsequent study, Ward and Southern (1995b) found that feeding a diet containing 200 µg of Cr/kg as CrPic did not influence weight gain or feed efficiency of broilers from 4 to 8 weeks of age. Also, plasma glucagon and NEFA and glucose clearance rates in 11-week-old broilers were not affected by dietary CrPic.

Kim et al. (1996a) fed diets supplemented with 0, 100, 200, 400, 600, or 800 µg Cr/kg as CrPic to broiler chickens from 1 to 42 days of age. Supplemental chromium did not affect growth rate or feed efficiency. Carcass fat was decreased by feeding 100 or 200 µg Cr/kg of diet. Two hundred µg Cr/kg of diet also decreased serum cholesterol, and HDL cholesterol of serum increased as dietary chromium increased. Percentage mortality of the chickens was inversely related to dietary chromium supplementation.

Information is meager on chromium toxicity for poultry. Dietary concentrations of chromium ranging from 3 to 1,000 $CrCl_3$ mg/kg caused no adverse effects on growing chicks (Hill and Matrone, 1970; Baker and Molitoris, 1975). Similarly, 30 or 100 mg Cr/kg as Na_2CrO_4 and 100 mg Cr/kg as K_2CrO_4 had no deleterious effects on growth and health of chicks to 21 days old (Romoser et al., 1961; Mertz and Roginski, 1975). Feeding a diet containing 2,000 mg Cr/kg as $CrCl_3$ decreased growth in chicks (Hill and Matrone, 1970).

Kim et al. (1996a) fed diets supplemented with 0, 800, 1,600, or 2,400 µg Cr/kg as CrPic to broiler chickens from 1 to 42 days of age. No signs of chromium toxicity were observed. Percentage mortality decreased as dietary chromium increased, and serum glucose and cholesterol decreased in chickens fed 800 or 1,600 µg Cr/kg of diet.

Results of research with young poultry are summarized in Table 3-5 and results obtained with laying hens are summarized in Table 3-6.

TABLE 3-5. Influence of Supplemental Dietary Chromium on Young Poultry[a]

Reference	Dietary Cr Concentration per kg (Source)	Age/Duration of Experiment	Increased Growth Rate[b]
Hill and Matrone (1970)	500, 1,000, 2,000 mg ($CrCl_3$)	1- to 21-d-old chicks	Yes, only in presence of toxic concentration of S.
Baker and Molitoris (1975)	3 mg ($CrCl_3$)	1- to 27-d-old chicks.	No response with purified crystalline amino acid diet.
Hafez and Kratzer (1976)	50, 100, 200 mg ($CrCl_3$)	1- to 28-d-old chicks.	Yes, only in presence of toxic concentration of V.
Steele and Rosebrough (1979)	20, 40, 80 mg ($CrCl_3$)	7- to 21-d-old turkeys.	Yes, with 20 mg/kg in absence of added nicotinic acid.
Steele and Rosebrough (1981)	20 mg ($CrCl_3$)	640, 7-d-old turkeys; 14-d trial.	Yes
Cupo and Donaldson (1987)	20 mg ($CrCl_3$)	Four chicks per treatment; 1- to 21-d-old chicks.	No
Liarn et al. (1993)	0, 100, 200, 300, or 400 µg (CrPic) Cr in basal diet, by analysis ranged from 71 to 2,600 µg/kg.	250 1-d-old broiler chickens; 56-d trial.	No
Ward et al. (1993)	200, 400 µg (CrPic)	60, 5-d-old chicks; 14-d trial.	No
Ward and Southern (1995a)	200 µg (CrPic)	40, 28-d-old chicks; 30- and 49-d trials.	No
Ward and Southern (1995b)	400, 1,600, 16,000 µg (CrPic);	Exp. 1 - 60 chicks; Start - 5 d; Duration - 28 d. Exp. 2 - 60 chicks; Start - 3 d; Duration 49 d;	No
	400, 1,600 µg (CrPic).	5- to 54-day old chicks.	Slight from 47 to 54 days of age.

Improved Feed Efficiency	Decreased Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response	Other
Yes	ND	ND	ND	
No	ND	ND	ND	
ND	ND	ND	ND	Reduced mortality due to V toxicity.
Yes	ND	ND	ND	
Yes	ND	ND	Yes, in vitro liver assay.	Increased hepatic lipogenesis from glucose.
No	No	No effect on serum fatty acids or cholesterol.	ND	Increased rate of glucose utilization by liver in vivo and in vitro.
No	No	ND	ND	Hepatic ATP citrate lyase activity increased with 100 and 200 µg.
No	No	ND	ND	No effect on N balance.
No	ND	No effect on plasma glucagon or NEFA.	No change in glucose clearance rate.	ND
No	No	No effect on plasma glucagon, insulin, urea N, NEFA.	ND	ND
Yes	Yes, slightly.	Reduced plasma glucose and NEFA. Increased insulin. No effect on glucagon.	ND	ND

TABLE 3-5. Continued

Reference	Dietary Cr Concentration per kg (Source)	Age/Duration of Experiment	Increased Growth Rate[b]
Kim et al. (1996a)	0, 800, 1,600, or 2,400 µg (CrPic).	144, 1-d-old broiler chicks; 42-d trial.	No
Kim et al. (1996b)	0, 100, 200, 400, 600, or 800 µg (CrPic)	288, 1-d-old broiler chicks; 42-d trial.	No

[a]Definitions of abbreviations used in table:
ATP - adenosine triphosphate
Cr - chromium
$CrCl_3$ - chromium chloride
CrPic - chromium picolinate
d - day
HDL - high density lipoprotein
N -nitrogen
ND - Not determined
NEFA - nonesterified fatty acids
S- sulfur
V - vanadium
wk - week

[b]Only statistically significant responses ($P \leq 0.05$) are indicated unless stated otherwise.

Improved Feed Efficiency	Decreased Carcass Fat	Change in Serum/Plasma Constituents	Hypoglycemic Response	Other
No	No	Decrease in glucose as dietary Cr increased. Decrease in cholesterol with 800 and 1,600 µg.	ND	Mortality decreased as dietary Cr increased. No signs of toxicity observed.
No	Yes, with 100 and 200 µg.	Decreased cholesterol with greatest decrease from 200 µg Cr. Increased percentage HDL cholesterol as dietary Cr increased.	ND	Notable decrease in mortality as dietary Cr increased.

TABLE 3-6. Influence of Supplemental Dietary Chromium on Laying Hens[a, b]

Reference	Dietary Cr Concentration per kg (Source)	Age/Duration
Jensen et al. (1978b)	5 mg ($CrCl_3$)	60-wk-old hens; 6- or 16-wk trial.
Jensen and Maurice (1980)	5, 10 mg ($CrCl_3$)	Hen age not given. 4- to 6-wk trials.
Sauveur and Thapon (1983)	10 mg ($CrCl_3$)	210, 40-wk-old hens; 6- to 8-wk trials.
Benabdeljelil and Jensen (1989)	5, 10, 50 mg ($CrCl_3$)	42- and 32-wk-old hens; 4- and 6-wk trials.
Southern and Page (1994)	100, 200 µg (CrPic)	32-, 28-, and 48-wk-old hens; 4- to 8-wk trials.
Lien et al. (1996a,b)	200, 400, 800 µg (CrPic)	100, 55-wk-old hens; 35-d trial.
Kim et al. (1997)	0, 200, 400, 800 µg (CrPic). Complete factorial arrangements with 14 and 16% protein.	960, 37-wk-old hens; 6-wk trial.

[a]Definitions of abbreviations used in table:
Cr - chromium
$CrCl_3$ - chromium chloride
CrPic - chromium picolinate
d - day
DM - dry matter
ND - Not determined
wk - week

[b]Only statistically significant responses ($P \leq 0.05$) are indicated unless stated otherwise.

Effect on Egg Production	Effect on Quality of Egg Albumen
None	Increased albumen quality in presence or absence of dietary vanadium.
None	Increased albumen quality in presence of dietary vanadium in 1 of 5 experiments.
None	No effect on albumen quality in presence or absence of dietary vanadium.
None	No effect on albumen quality in presence or absence of dietary vanadium.
Increased in one of three experiments.	ND
None	Slight increase with 400 µg Cr/kg. Reduced cholesterol concentration in yolk and serum.
None. Egg weight increased with 400 µg. Improved DM and protein digestibility with 200, 400, and 800 µg. Decreased serum cholesterol with 400 µg.	ND

Summary

Research with poultry has shown that supplemental dietary chromium can be used to alleviate some of the toxic effects of vanadium in growing chicks and laying hens. Evidence also has been obtained that supplemental chromium at 20 mg/kg of diet as $CrCl_3$ increases the rate of glucose utilization by livers of chicks and poults in vivo and in vitro. The effects of added dietary chromium on growth rate and feed efficiency of growing poultry have differed, with improvements reported in four of eight experiments. An improvement in performance of young poultry was observed when $CrCl_3$ was used to supply 20 mg Cr/kg of diet. Supplementing diets with 200 µg to 1,600 µg Cr/kg as CrPic increased growth rate of chicks in only two of seven studies. Recent research with broiler chickens, however, has shown that supplemental chromium decreased mortality during periods of rapid growth.

The evidence available today, although meager, indicates that supplemental dietary chromium can affect metabolism and well-being of poultry. Additional information is needed, however, to describe the circumstances in which supplemental dietary chromium can be used to greatest advantage.

Horses

A limited amount of work has been done to determine the effects of chromium supplementation on performance and metabolic responses of horses. Daily supplementation of 5 mg chromium as chromium yeast to a diet containing an undetermined concentration of chromium for a 14-day period did not affect fasting (8 hours) concentrations of plasma glucose, insulin, cortisol, or triacylglycerols (Pagan et al., 1995). Trained thoroughbred horses fed chromium supplements tended to have lower plasma insulin concentrations 1 hour after feeding, but other plasma metabolites were not affected by chromium. Plasma glucose tended to decrease during exercise, and post-exercise concentrations of triacylglycerols were increased in horses receiving chromium supplements. Peak plasma lactate concentrations tended to be lower, and those of cortisol were lower just prior to and in the early stages of the exercise program.

Recently completed research at Louisiana State University (D. Thompson, personal communication) showed that feeding 5 mg chromium per head daily as CrPic did not affect metabolic or hormonal responses when adult mares were subjected to a glucose tolerance test, insulin challenge, epinephrine challenge, feeding challenge, or exercise challenge. Pokeweed mitogen tended to stimulate greater proliferation of lymphocytes in chromium-supplemented mares than it did in control mares. The meager amount of research conducted with horses, described here, has not shown any definitive benefits of dietary chromium supplementation.

Rats

Because supplemental trivalent chromium (Cr^{+3}) has been reported to enhance insulin and glucose metabolism, it has been suggested that chromium is essential for the rat and that its function is to aid in the utilization of glucose. The work of Schwarz and Mertz (1959), Schroeder et al. (1963), Schroeder (1966), Roginski and Mertz (1967), Mertz et al. (1965), Mertz and Roginski (1969), and Roginski and Mertz (1969), using highly restrictive environmental conditions, often is cited as evidence for the essentiality of chromium for the rat. Many of the chromium deficiency symptoms are typical of those associated with aging. Because of the larger number of responses associated with this process, life span should be positively affected by chromium supplementation. This effect of chromium was assessed by Evans and Meyer (1992, 1994) in rats. They found an increase in life span of greater than 25 percent due to supplementation of the diet with 1,000 μg Cr/kg as CrPic. Whether chromium contributes to a beneficial physiological function is uncertain. Specificity is questioned because other heavy metals seem to initiate similar effects (Fagin et al., 1987; Pederson et al., 1989).

Other studies have failed to show positive effects of chromium on glucose tolerance or glucose utilization in tissues of rats (Flatt et al., 1989; Holdsworth and Neville, 1990; Woolliscroft and Barbosa, 1977). Woolliscroft and Barbosa (1977) fed 6-week-old Sprague-Dawley rats 30 percent Torula yeast diets containing low chromium concentrations (30 to 100 μg/kg, as $CrCl_3$ estimated) or diets that contained 5,000 μg Cr/kg. After 6 weeks, there was no significant difference in intravenous glucose tolerance between the two groups. Flatt et al. (1989) found no significant difference in food intake, body weight gain, glycosylated hemoglobin, plasma glucose, plasma insulin, glucose tolerance, or insulin sensitivity between two groups of weaning Wistar rats fed either 30 or 1,000 μg Cr /kg of diet as $CrCl_3$ for 32 days. Differences in chromium concentrations in tissues between the two groups varied—from no change in skeletal muscle to a 44 percent reduction in the pancreas.

Holdsworth and Neville (1990) found that supplementing the diet with chromium acetate had no effect on glucose metabolism in rats. They fed weanling Wistar rats Torula yeast diets similar to those designed by Schwarz (1951) but supplemented with L-cystine, L-methionine, and L-histidine. These supplemented diets supported more rapid growth than did the original diet and contained 100 (low-chromium diet) or 1,000 μg Cr/kg (high-chromium diet). A control group was fed a commercial natural-ingredient diet. After five weeks, the rats fed the Torula yeast diets gained 30 percent less weight than did the control rats, regardless of whether supplemental chromium was used. Those fed chromium-supplemented yeast diets did not grow at a significantly greater rate than did those without supplemental chromium. The incorporation of glucose carbon into liver glycogen in the rats fed the low-chromium diet was only one-fifth that of the control rats, but was not different from that of rats given the chromium-supple-

mented yeast diet. Yeast was grown in media with or without chromium. Extracts from this yeast enhanced glucose incorporation into glycogen of hepatocytes isolated from rats fed low- or high-chromium diets regardless of whether chromium was present in the extract.

Others reported lower sperm counts in semen of rats fed low-chromium diets (<100 μg/kg) for eight months, compared with semen of rats fed high-chromium diets (2,000 μg/kg) (Anderson and Polansky, 1981). Effects of chromium supplementation on weight gain of rats are achieved only with restrictive environmental conditions (Schroeder et al., 1963). Under similar conditions, supplementation of the diet with other heavy metals, such as cadmium and lead, also enhanced initial weight gain, suggesting a nonspecific pharmacological response rather than a nutritional response (Schroeder et al., 1963).

Anderson et al. (1996b) measured the incorporation of chromium into the tissues of rats fed nine different forms of chromium. They postulated that insulin-chromium-nicotinic acid complexes stabilized with amino acids would have the greatest incorporation into tissues because these complexes would be similar to the naturally occurring, biologically active complexes that have been shown to be better utilized. Chromium absorption in conjunction with tissue chromium concentrations was determined in order to ascertain whether tissue chromium stores, and presumably status, are regulated at the level of absorption. Chromium compounds tested were chromium chloride, chromium acetate, chromium potassium sulfate, chromium trihistidine, chromium triglycine, chromium trinicotinic acid, chromium dinicotinic acid dihistidine, chromium tripicolinic acid, and chromium dinicotinic acid diglycine cysteine glutamic acid. Complexes were fed to weanling rats for three weeks at 5,000 μg Cr/kg of diet. The basal control diet was a cornstarch-based diet containing 30 μg Cr/kg. Chromium incorporation into the kidney was greatest for chromium dinicotinic acid diglycine cysteine glutamic acid (850 ng/g dry weight) followed by chromium potassium sulfate (407 ng/g), chromium acetate (397 ng/g), chromium dinicotinic acid dihistidine (394 ng/g), CrPic (368 ng/g), chromium glycine (343 ng/g), CrNic (166 ng/g), chromium chloride (74 ng/g), chromium trihistidine (49 mg/g), and control (23 ng/g). Chromium concentration of the liver was greatest for the chromium picolinate compound (50 ng/g) followed by chromium dinicotinic acid diglycine cysteine glutamic acid and chromium acetate. Liver chromium concentrations of rats fed the remaining complexes were not significantly different from those of control animals that received no added chromium. Chromium concentrations were significantly greater in the kidney than in the liver, spleen, heart, lungs, and gastrocnemius muscle. Differences in absorption of radioactive forms of chromium did not explain the differences in tissue chromium concentrations. Chromium absorption after 4 hours and retention after 24 hours were not significantly different for the forms of chromium tested. These data demonstrate that chromium concentrations are greatest in the kidney and that the form of dietary chromium significantly affects tissue chromium concentrations. Absorption of

chromium does not correlate with tissue chromium concentrations, and blood chromium is not in equilibrium with tissue chromium stores.

Although the early studies seemed to indicate that dietary chromium supplements enhanced glucose metabolism, more recent studies do not. Perhaps the duration of the experiments and the environmental conditions in the later studies were insufficient to allow chromium stores to be depleted and deficiency signs to be expressed. Signs of chromium deprivation might have been more evident if longer feeding periods or multiple-generation studies had been used.

Trivalent chromium salts, chromic oxide, and metallic chromium are not very toxic. Because of their oxidizing and protein-precipitating properties, chromium trioxide, chromates, and bichromates are potent poisons. The LD_{50} (the dose that is lethal to 50 percent of test animals) for a Cr^{+3}-nicotinic acid complex injected intravenously was 60 mg/kg of body weight in rats. The lethal single oral dose for Cr^{+6} in young rats was 130 mg/kg, whereas as much as 650 mg Cr^{+3}/kg produced no overt toxicosis (National Research Council, 1980).

Summary

Chromium has been shown to be essential for glucose metabolism in rats only under highly controlled experimental conditions in which body stores of chromium were depleted. Mainly on this basis, chromium has been listed in the National Research Council (1995) publication *Nutrient Requirements of Laboratory Animals* as a potentially beneficial dietary constituent for rats, but a dietary requirement per se was not given.

Rabbits

Chromium is present in very low concentrations in the tissues and body fluids of rabbits. Plus (1988) reported mean chromium concentrations of 0.65, 1.00, 4.90, and 0.80 ppm in rabbit liver, kidney, serum, and brain, respectively.

Chromium deficiency in rabbits has not been demonstrated. However, positive effects of chromium on cholesterol metabolism and sucrose utilization have been reported. For 135 days, Abraham et al. (1991) treated 33 Loewenstein (Yoknian) male rabbits with daily injections of distilled water; of 20 μg potassium chromate; or of 1, 5, 10, or 20 μg $CrCl_3$. The rabbits were fed a cholesterol-enriched (1 percent cholesterol) diet ad libitum. The mean serum chromium concentrations were increased by 2- to 10-fold for treated rabbits within 1 week of treatment. It also was observed that the cholesterol content in aorta, aortic weight, and aortic minimal surfaces covered by plaque were decreased by approximately 50 percent for rabbits treated with either form of chromium. Similar decreases in cholesterol-induced plaque in rabbits injected daily with potassium chromate also have been previously reported (Abraham et al., 1980, 1982a).

Moersen and Borgman (1984) studied changes in zinc and chromium me-

tabolism in rabbits fed purified diets with either corn starch or sucrose as the sole dietary source of carbohydrate. Sixteen mature New Zealand White rabbits were assigned to two dietary treatments for 14 weeks. Hair samples obtained at the beginning and end of the experiment were analyzed for zinc and chromium content. The chromium concentration in hair of rabbits fed the sucrose diet decreased by about 30 percent at the end of week 14. Similar studies with rats and humans have reported a negative chromium balance on diets high in glucose or sucrose (Roginski and Mertz, 1969; Mertz et al., 1974).

The minimal toxic concentration of chromium for rabbits is unknown. Chromium in high doses can be toxic to rabbit liver and kidney. Tandon et al. (1978) treated male albino rabbits with daily injections of either normal saline, 2 mg Cr/kg of body weight as chromium nitrate (trivalent form), or potassium dichromate (hexavalent form) for a period of six weeks. Serum urea concentration of chromium-treated rabbits increased threefold within three weeks of treatment. This change was coupled with cellular damage to liver and kidney. Serum chromium concentrations were about five times greater for rabbits injected with the hexavalent form than they were in the rabbits treated with trivalent chromium. Aerosol exposure of rabbits to Cr^{+3} or Cr^{+6} (hexavalent) at a rate of 0.9 mg/m^3 for six weeks (five days/week and six hours/day) resulted in nodular accumulations of the alveolar macrophages, with most macrophages showing enlarged lysosomes. However, gross appearance of lung tissue was normal (Johansson et al., 1986). Overall, the hexavalent form induced greater toxic effects than did the trivalent form.

Summary

Research with rabbits indicates that cholesterol and plaque content of the vascular system were decreased by supplemental dietary chromium and that the dietary carbohydrate source can modify chromium metabolism. There is, however, insufficient information on which to base conclusions or recommendations concerning rabbits' need for dietary chromium.

Fish

Investigations of the influence of chromium in fish nutrition have been limited, although more attention has been focused on this topic in recent years. Tacon and Beveridge (1982) fed rainbow trout *(Oncorhynchus mykiss)* chemically defined diets (1.56 mg Cr/kg provided by the ingredients) supplemented with 0, 1, 3, or 6 mg Cr/kg as $CrCl_3$ and observed no differences in weight gain or changes in tissue chromium distribution. In a more recent study, supplementation of practical diets with chelated chromium at 0.5 mg/kg of diet increased blood glucose clearance in rainbow trout but did not affect weight gain or protein and energy retention (Bureau et al., 1995). Hertz et al. (1989) reported that chromium supplementation of a fishmeal-soybean-meal-based diet improved glu-

cose utilization in common carp *(Cyprinus carpio)*. Increased intestinal absorption of glucose was reported in snakehead *(Channa punctatus)* at concentrations as low as 1 mM (Sastry and Sunita, 1982).

Significant effects of dietary chromium on growth and carbohydrate utilization of hybrid tilapia *(Oreochromis niloticus* x *O. aureus)* have been reported. In a study by Shiau and Lin (1993), hybrid tilapia were fed fishmeal-based diets containing either cornstarch or glucose at 40 percent dry weight with or without supplemental chromium (2 mg Cr/kg) as $CrCl_3$. The unsupplemented diets were analyzed to contain 0.4 mg Cr/kg. Weight gain and retention of dietary protein and energy in fish fed the diets containing cornstarch were not affected by chromium supplementation but were significantly greater than in fish fed diets containing glucose. Chromium supplementation of the diet containing glucose did, however, significantly increase weight gain, energy deposition, and liver glycogen of fish, and it delayed the postprandial peak in plasma glucose from two to three hours. Thus, chromium supplementation enhanced the utilization of dietary glucose but not cornstarch by hybrid tilapia. In a related study, Shiau and Chen (1993) fed hybrid tilapia fishmeal-based diets similar to those described previously, containing either cornstarch or glucose with or without supplemental chromium (2 mg Cr/kg) as $CrCl_3$, $Na_2CrO_4 \cdot 4H_2O$, or Cr_2O_3. Fish fed the diets with cornstarch, regardless of chromium supplementation or source, had significantly greater weight gain, feed intake, protein and energy retention, and body lipid compared with those fed diets with glucose. The fish fed the glucose diet without supplemental chromium showed significantly less weight gain as well as more rapid plasma glucose peak time and elevated glucose-6-phosphate activity, compared with fish fed glucose diets with supplemental chromium, regardless of source. Fish fed the glucose diet supplemented with Cr_2O_3 had weight gain, feed intake, protein and energy retention, body lipid, and phosphofructokinase activity similar to that of fish fed cornstarch diets and much greater than found in fish fed the other glucose diets. Thus, supplementation of Cr_2O_3 to a diet containing glucose improved glucose utilization by hybrid tilapia and was much more effective than were the other sources of chromium. Specific mechanisms by which chromium enhances the utilization of dietary glucose in hybrid tilapia, however, were not elucidated.

In a subsequent study, Shiau and Liang (1995) observed enhanced weight gain and feed utilization of hybrid tilapia fed diets containing cornstarch relative to fish fed diets containing glucose. Also, fish fed diets containing glucose supplemented with Cr_2O_3 at 0.5 percent exhibited increased weight gain, feed efficiency, protein efficiency ratio, protein deposition, and phosphofructokinase activity, as well as lower tissue chromium, than did fish fed a similar diet supplemented with 2 percent Cr_2O_3. The Indian major carp (*Labeo rohita)* also has been reported to exhibit growth enhancement when fed 10 mg Cr/kg of diet, but growth reduction when fed 20 and 40 mg Cr/kg; and carcass chromium was increased by chromium intake (Jain et al., 1994).

The effects of waterborne chromium also have been studied in some fish species. Chronic exposure to waterborne Cr^{+6} has been reported to inhibit Na/K-ATPase activity but not Mg-ATPase activity in several tissues of rainbow trout (Kuhnert et al., 1976). It has also been reported to increase the frequency of micronuclei induction in erythrocytes of Prussian carp (*Carassius auratus gibelio)* (Al-Sabti et al., 1994), and to alter several enzyme activities in snakehead (Sastry and Sunita, 1983), rainbow trout, and seabass (*Dicentrarchus labrax)* (Boge et al., 1988).

Summary

Investigations concerning the influences of dietary chromium on fish have been limited. Some studies reported no effect on growth or tissue chromium distribution. Others showed that chromium supplementation of diets, especially those containing glucose, caused significant increases in weight gain, energy deposition, and liver glycogen, and altered postprandial plasma glucose concentrations. Some differences in response to different sources of dietary chromium also have been reported. Specific mechanisms by which chromium influences dietary carbohydrate utilization of fish have not been elucidated.

CONCLUSIONS

Based on careful analysis of the available scientific literature, as presented in this chapter, the following conclusions can be made.

(1) It is not possible to make specific recommendations as to dietary form and concentration of chromium supplementation for cattle, poultry, and swine because

- *there are insufficient comparative data for the determination of relative bioavailabilities of chromium from supplemental sources;*
- *only meager data are available from titration studies designed to determine supplemental chromium concentrations that are most effective for cattle, poultry, and swine; and*
- *there have been no studies designed or conducted to determine dietary chromium requirements of cattle, poultry, or swine.*

(2) Supplementing practical diets with trivalent sources of chromium might be beneficial for health and well-being of cattle during times of stress; however, the factors that affect the efficacy of supplemental chromium and the dietary chromium concentrations required have not been determined.

(3) Chromium supplementation of swine diets, beginning at an early age and continuing through the finishing period, could improve carcass leanness and subsequent reproductive efficiency, but these responses to chromium are likely to be inconsistent until the factors that affect the efficacy of dietary chromium inclusion are more clearly defined.

(4) Information in the scientific literature on the need for supplemental chromium in practical diets of fish, horses, sheep, rabbits, and rats is too sparse to allow conclusions.

(5) Although most research on potential toxicity of trivalent forms of dietary chromium has been conducted with rats and chickens, the results show that the concentrations of trivalent chromium typically added to diets of food-producing animals are safe and nontoxic (National Research Council, 1980).

(6) Additional research is needed to determine the bioavailability of chromium contained in feed ingredients and to obtain more definitive data on the comparative bioavailability of chromium from dietary supplements.

(7) Research should be designed to create reproducible signs of chromium deficiency in animals, which would facilitate the establishment of dietary chromium requirements by way of appropriate titration studies.

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