Iron Nutrition and Anaemia in Piglets: a Review

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Abstract

Man has known that iron plays an important role in health and disease for considerable time (Loosli, 1978). Bryan (1931) stated that documented therapeutic use of iron date back as far as 1500BC. Iron was recognised as vital nutrient for animal (Boussingault, 1872). Braasch (1981) was the first to describe anaemia in nursing pigs that were being reared in confinement in Germany. However, he didn’t equate the anaemia with iron deficiency but instead with management. Anaemia in nursing pigs was first link to iron deficiency by (McGowan and Chrichton, 1924).

Iron is a very vital component of every living thing. Bothwell et al., (1988) estimated that a 70kg adult human has a whole body iron of 60-70 parts per million (ppm). The concentration in piglets at birth is 20-30 ppm (Venn et al., 1977). Of this concentration in the piglet, 475 is associated with blood, 1.6% in the spleen, 15% in the liver and th remaining 44% is found in body tissues (Thoren-Tolling, 1995). Following the neonatal period around 80% in the pig is associated with haemoglobin (Hb) {National Research Council, 1979}. Th majority of body iron is bound to proteins as heme complexes or nonheme complexes. The most common heme complexes are Hb and myoglobin, while the common nonheme complexes are of two storage forms viz: feritin and haemosiderin, and one transport form called transferin.

Key words: Iron, Nutrition, Piglet and Anaemia

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Introduction

Iron Absorption

Iron absorption can take occur throughout the gastrointestinal tract (GIT) with the two most common sites of absorption being the duodenum and the jejunum. The common theory in iron absorption, known as the mucosal block theory, is that only enough iron to meet the animal's need is absorbed (Hahn et al., 1943).

Two of the determinants of this need are iron status and erythropoietic demand. The basis of this theory is that iron is taken up by the mucosal cells in one of three forms, Ferrous, Ferric and as part of an organic compound. Upon absorption the ferrous form is oxidised in the ferric form for incorporation into ferritin. As mucosal cells become saturated with ferritin, absorption ceases until ferritin can be converted into transferritin for removal into the plasma. This process involves reduction of the iron in ferritin into the ferrous form where it moves to the cell surface and is oxidised before incorporation into transferritin. As ferritin level is diminishing in the mucosal cells, iron absorption increases. Therefore, there is an inverse relationship between mucosal ferritin level and iron absorption.

Iron deficient animals absorb dietary iron into the mucosal cells and convert the majority into transferritin while iron adequate animals converts only a small portion of the absorbed iron into transferritin for transport into plasma (Conrad and Crosby, 1993).

Factors Affecting Iron Absorption and Bioavailability

Several factors affect iron absorption, this include, age, iron status, species, dosage level, and other nutrient components of the diet both organic and inorganic.

(Furugouri and Kawabata, 2006), using labelled ferric citrate and showed that piglets exhibited active absorption of iron up to 180 hours of age.

Specie difference

(Chausow and Czarnecki-Maulden, 1988) noted specie differences while completing a Hb repletion study with beef liver and ferrous sulphate using cats and chicks. Iron in beef liver fed to cats was 350% as available as ferrous sulphate while only 90% as available in chicks.

Dose factor

Works done with dosage levels indicate higher absorption efficiencies with lower dosage levels. (Pfau et al., 1977), demonstrated that the absorption efficiency of iron from either Hb or ferrous sulphate to have an inverse relationship to dosage level.

Iron Status Factor:

The physical or chemical status of iron also affects absorption. Iron from animal sources is readily available than iron from plant sources (Morris, 1987). This is due to the large proportion of heme iron (Hb) in the animal source. Heme iron is absorbed as an intact porphyrin complex whereas nonheme iron must be removed from its protein-bound complexes prior to absorption (Morris, 1987).

Nutritive and Non-Nutritive Elements factor:

Various nutritive and non-nutritive elements within the diet have been shown to affect iron absorption and thus, bioavailability. (Wadell and Sell, 1964) illustrated decrease iron (Fe) absorption in chicks associated with increasing dietary concentration of either calcium, phosphorus or both. Phosphorus has been hypothesized to affect Fe absorption through the formation of insoluble ferric phosphate and phytate (Underwood, 1981).

Bradley et al., (1983) illustrated that dietary Cu concentration in the range 120-240ppm, led to decrease liver Fe concentration up to 50-60% through possible impairment of absorption. (Gipp et al., 1974), concluded that Cu, when fed diet at 250 ppm not only reduce Fe absorption but could invoke Fe-deficient anaemia in pigs.

Zinc has also been shown to have antagonistic effect on Fe utilization. (Settlemire and Matrone, 1997a and 1997b) concluded that Zn impact on Fe utilization in two ways: By impairment of Fe incorporation into ferritin and by decreasing the life span of Red blood cells leading to increase Fe requirements.

Dietary manganese in excessive levels leads to reduction in Hb (Baker and Halpin, 1991).
While the aforementioned minerals have been shown to have a detrimental effect on Fe absorption and bioavailability, other nutritive factors have been shown to be beneficial. Three of these include the amino acids histidine, lysine and cysteine. (Van Campen and Gross 1999), shows that these amino acids form chelates with the ferric Fe, thus keeping the iron in solution.

Vitamin C or ascorbic acid also has been shown to have beneficial effects on Fe absorption. (Greenberg et al., 2007) shows that iron-deficient rats had increased efficiency of absorption of Fe when Vit C was given with the Fe supplement. (Van Campen, 2002) earlier investigated the effects of histidine and Vit C supplementation on Fe absorption and concluded that Vit C was more effective in increasing Fe retention. This author hypothesized that this was due to the ability of Vit C to act as both reducing and chelating agent.

**Piglet Anaemia**

Piglet anaemia also called Iron deficient anaemia is a hypochromic-microcytic anaemia generally associated with young, rapidly growing piglets deprived of Fe in their diet or from their environment. The most common parameter to indicate Fe-deficient anaemia is haemoglobin concentration.

When a piglet is born, it has sufficient iron to last for only 3–7 days and so must obtain sufficient iron from elsewhere. Under natural conditions, baby pigs may obtain sufficient iron from the soil, but most pigs today are farrowed and reared on concrete floors indoors and thus have no access to soil. However, some soils contain very little iron, or iron in a form that is chemically bound and not available to the pigs.

Indoors the pig has no access to iron other than to the sows' milk (which is deficient) until it starts to eat creep feed. The pig is born with a normal level of haemoglobin in the blood of 12-13g/100ml and this rapidly drops down to 6-7g by 10 to 14 days of age. A shortage of iron results in lowered levels of haemoglobin in the red cells, (anaemia), a lowered capacity for the carriage of oxygen around the body and an increased susceptibility to disease (BSAS, 2003).

**Iron Need in Piglets**

The newborn pig contains approximately 50mg of Fe at birth, mostly in the form of Hb (Venn et al 1977). The neonatal pig has been determined to have a requirement of 7-16mg of Fe/day for normal growth (Venn et al 1977). This requirement can be expressed in another way as 21mg/kg body weight gain (Braude, et al., 1987). Due to minimal concentration of Fe in sows milk (1mg/l), neonatal pigs reared in confinement require supplementation Fe in order to overcome the susceptibility to anaemia (Brady et al., 1978). Maximum Hb levels are produced in neonate at 14 days of age when piglets are supplemented with either 100 or 150mg of iron dextran at birth. Maximum growth rate was achieved through supplementation of 100mg in the form of injectable Fe dextran to piglets weaned at three weeks (Zimmerman et al., 1989).

Fe requirement as concentration of diet decreases with age and weight due to decrease in the blood volume/unit weight and higher Fe intake. The Fe requirement for pigs 1-5 and 20-50kg live weight are 100 and 60ppm respectively, which is equivalent to Fe intake of 25 and 114mg (National research Council, 1988).

**Deficiency Symptoms of Iron**

The most common parameter to indicate Fe-deficient anaemia is haemoglobin concentration. In 1979, the National Research Council created a classification system by which pigs could be categorized based on the extent of the anaemia by their Hb concentration, measured in grams /decilitre as follows:

i. Pigs with Hb levels 10 or above are normal.

ii. Hb level of 9 is the minimum level for optimum performance.

iii. Hb level of 8 indicates a borderline anaemia.

iv. Hb level of 7 is the level in which anaemia retards growth.

v. Hb level of 6 is considered severe anaemia and 4 as severe anaemia with increased mortality.

The first sign of Fe-deficient anaemia is roughness of hair coat and lost of pigmentation of mucus membrane. The skin is wrinkle, pigs exhibit listlessness, characterized by drooping of the head and ears combine with lack of appetite and reduce weight gain.
In severe cases, pigs may be identified by dyspnea, increase heart and respiratory rates, the animal present systolic murmurs due to reduced blood viscosity; pigs die suddenly due to anoxia.

Affected pigs have higher prevalence for subcutaneous edema in the neck, shoulder and limb areas (Conrad, 1980). Anaemic pigs show higher susceptibility to infectious diseases (such as pneumonia, influenza and GIT disorders) because a dietary Fe level is directly related to antibody production (Luke and Gordon, 1980).

**Treatment and Prevention of Piglet Anaemia**

Several management approaches have been postulated and they include:

> A shovelful of clean earth given daily and sprinkled with iron sulfate. Alternatively, ashes can be used and sprinkled with a copper–iron solution.

> Various oral mixtures can be used and are placed on the back of the tongue; these are best given within 36 hours of birth to be effective. Iron can also be provided in piglets’ drinking water, with a dispenser placed in the creep area.

> Iron sulfate paste can be painted onto the sows teats every 2–3 days.

> Use of iron licks or blocks.

> Administration of 200mg (i/m) of iron dextran as a single dose is sufficient.

The easiest method is to give the piglet an injection of 150-200mg of iron dextran in either a 1 or 2ml dose. Iron is best given from 3 to 5 days of age and not at birth, because a 2ml dose at birth causes considerable trauma to the muscles.

Iron can also be given orally but this method is time consuming and the pig must be treated on 2 or 3 occasions at 7, 10 and 15 days of age.

> Oral pastes available ad lib have been used but the uptake within any litter is variable and a few piglets remain anaemic.

**Reference**


