TRACE ELEMENT ALTERATIONS IN NEONATAL INFECTIONS

Thesis Submitted In Partial Fulfillment

Of Master Degree In Pediatrics

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بسم الله الرحمن الرحيم

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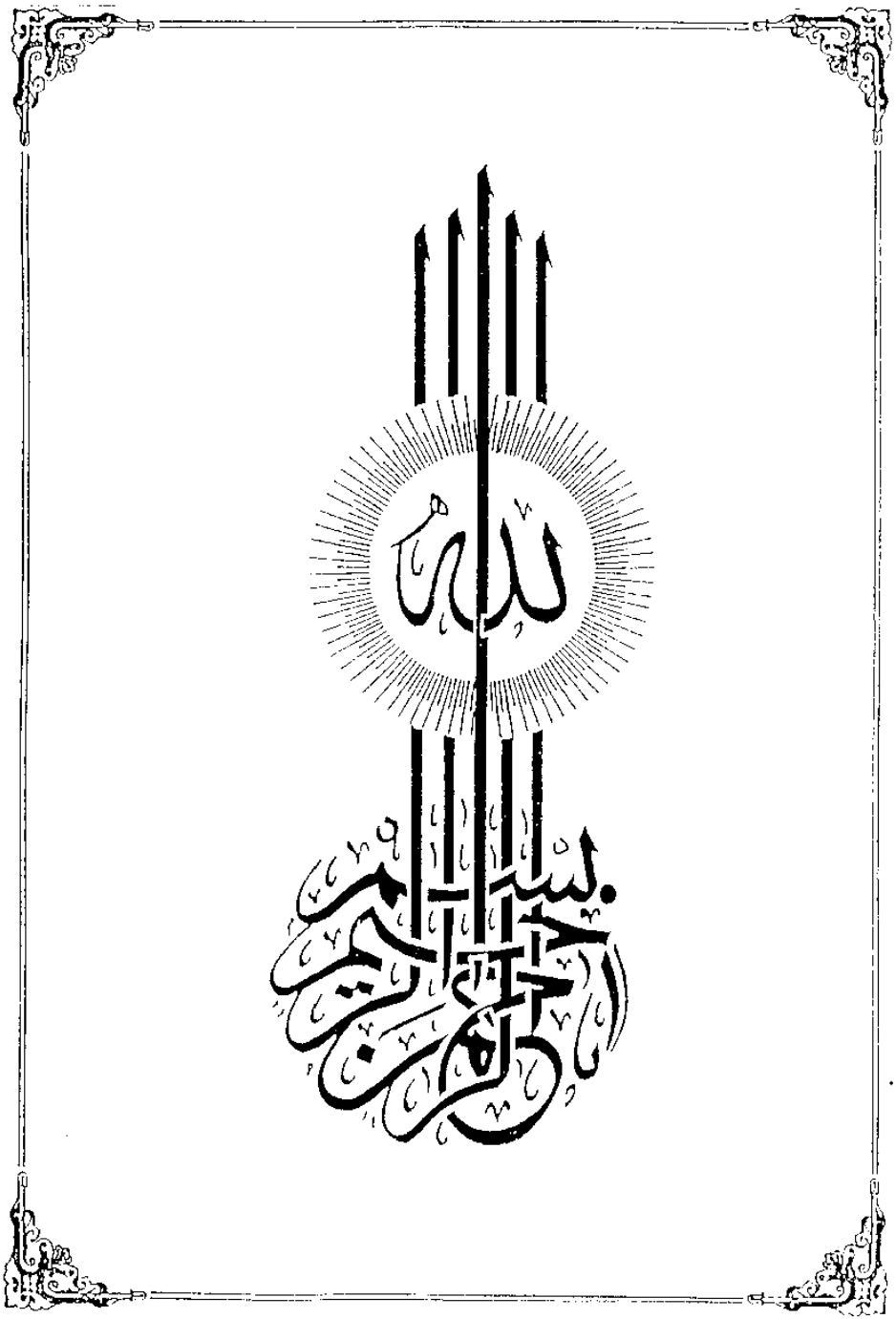
صدق الله العظيم
(سورة البقرة الآية ٣٢)



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LIST OF ABBREVIATIONS

Atomic Absorption	:	A.A.
Complete blood count	:	CBC
Concanavalin A	:	Con A.
Copper	:	Cu
Group B Streptococcus	:	GBS
Hemoglobin	:	HB
Interleukin-1	:	IL-1
Interleukin-2	:	IL-2
Lactoferrin	:	LF
Natural Killer	:	NK
Phytohemagglutinin	:	PHA
Pokeweed mitogen	:	PWM
Polymorphnuclear leukocytes	:	PMNs
Zinc	:	Zn

INTRODUCTION AND AIM OF THE WORK

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Trace elements are inorganic ions present in tissues in a minute quantity, micrograms to picograms per gram of wet organ.

The essential trace elements are: iron, iodine, cobalt, copper, manganese, molybdenum, selenium, chromium, fluorine, silicon, nickel, zinc, tin and vanadium.

Critical biological functions in animals are disrupted by deprivation of essential trace elements resulting in discrete deficiency states (Ulmer, 1980).

Zinc forms part of the enzymes involved in protein synthesis. Its deficiency is manifested mainly by anorexia and inanition and through this action protein synthesis is consequently altered (Dórea and Araújo, 1988).

Zinc deficiency in humans is known to alter immunity (Hansen et al., 1982).

Copper is a component of several enzymes including cytochrome C oxidase, superoxide dismutase etc... Copper deficiency syndrome in infants causes: psychomotor retardation, hypotonia, sideroblastic anaemia, etc... (Shaw, 1988).

Many of the features of copper deficiency in infants can be explained on the basis of deficiency of particular copper enzymes (Shaw, 1988).

Infections are a frequent and important cause of morbidity and mortality in the neonatal period. A variety of organisms can cause neonatal infection e. g. bacteria, viruses, fungi, protozoa, chlamydia and mycoplasma.

The presenting clinical manifestations in the neonate with infection may be subtle and may mimic the features of other common diseases during this period. As a result, the diagnosis of infection is often missed or delayed until the process has become widespread (Overall, 1987).

This thesis aims at studying the serum concentrations of copper, zinc and iron in neonatal infections.

Plasma protein analysis will help to reveal the magnitude of inflammatory reaction.

REVIEW OF LITERATURE

ZINC

Biological Importance of Zinc:

Zinc is an essential element for plants, animals, and humans. It is a constituent of a number of enzymes involved in major metabolic pathways and functions, e. g Carbonic anhydrase, carboxypeptidase, alkaline phosphatase, lactic, malic and alcohol dehydrogenase, insulin, and most of the enzymes involved in DNA replication, repair, and transcription (Orten and Neuhaus, 1982).

The retina contains a zinc metalloenzyme, retinene reductase, which is required for the reconstitution of retinene (vitamin A aldehyde) during rhodopsin cycle (Tyler, 1979).

Insulin forms complexes with zinc, which makes it possible for crystalline zinc insulin to be prepared during insulin purification.

Zinc insulin complexes are also present in the β cells of the pancreas, and there is evidence suggesting that zinc is used in these cells to store and release insulin as required (Davies, 1972).

Zinc deficiency in humans is known to alter immunity. Thymic atrophy, lymphopenia, with alterations in the various

subsets of lymphocytes, and anergy to delayed hypersensitivity skin testing have been documented to accompany human zinc deficiency and return to normal with zinc supplementation (Hansen, 1982).

Zinc Metabolism:

- Absorption:

Approximately 20 to 30 percent of ingested dietary zinc is absorbed (Prasad, 1983).

Zinc absorption is affected by the body size, the level of zinc in diet and the presence in the diet of other potentially interferring substances, such as phosphate, phytate, fiber, and other chelating agents (Spencer et al., 1980).

It has been proposed that a low molecular weight ligand in the intestinal lumen, and presumably of pancreatic origin, is directly involved in that phase of zinc absorption, which is concerned with the uptake of zinc from the lumen into mucosal cells (Evans et al., 1975).

Absorption of zinc in the newborn is believed to be facilitated by zinc-binding ligands in the breast milk which diminishes after a few days and zinc-binding ligands in the gut mucosa which takes some days to be fully developed (Shaw, 1979).

Cousins (1979) attempted to explain the data on the mechanism of zinc absorption at the level of intestinal mucosal cells. A portion of the dietary zinc entering the lumen of the small intestine is transported across the mucosal brush border membrane by a process probably requiring ATP. Within the intestinal cells, newly acquired cytoplasmic zinc, equiliberates with "zinc pool" and is either transferred to high molecular weight proteins and metallothionine or to the plasma (Prasad, 1983) (Fig. 1).

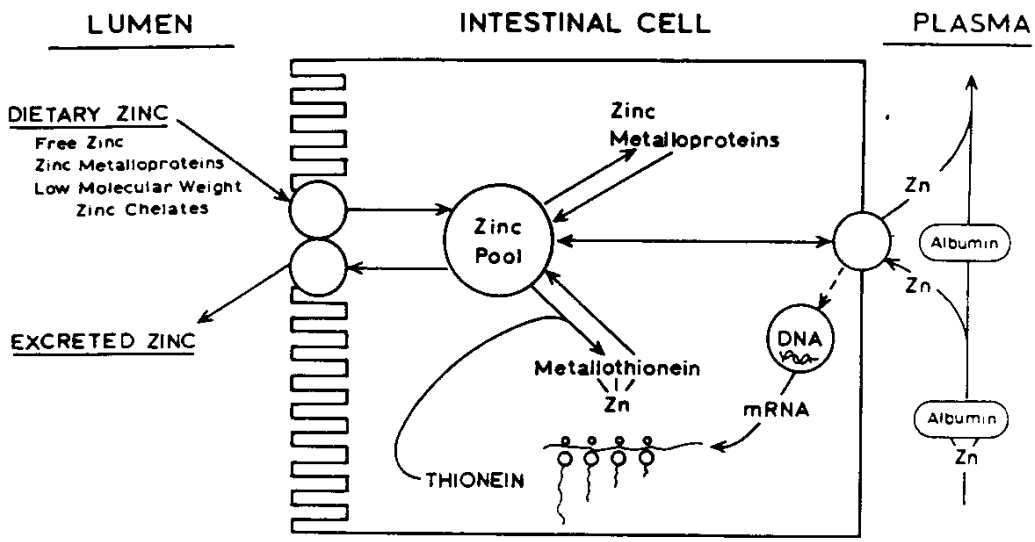

Figure 1.

Regulatory pathway of dietary zinc processing by intestinal cells. (Dashed line denotes induction of metallothionein mRNA. Thionein is a metal-free metallothionein) (Cousins, 1979).