"ASSESSMENT OF THE EFFECT OF TWO DIFFERENT DOSES OF ZINC SUPPLEMENTATION ON NEURODEVELOPMENT AND GROWTH IN LATE PRETERMS : A HOSPITAL BASED RANDOMISED CONTROLLED TRIAL"

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DR DNYANESH D K M.D., L.L.B
Professor & Head
Department of Pediatrics
J N Medical College
KAHER
Belagavi, Karnataka

Dr. Dnyanesh D.K
PROFESSOR & HEAD
DEPARTMENT OF PEDIATRICS
J. N. Medical College, Belagavi

Date: 07/03/2025
Place: JNMC, Belagavi

DR N S MAHANTASHETTI M.D.
Principal
J N Medical College
KAHER
Belagavi, Karnataka

PRINCIPAL
JAWAHARLAL NEHRU MEDICAL COLLEGE
BELAGAVI

KAHER BELAGAVI J.N. MEDICAL COLLEGE

Date: 07/03/2025
Place: JNMC, Belagavi

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(Recognized by National Medical Commission, New Delhi)

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Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 | 0831 - 2470759 | www.jnmc.edu | rincipal@jnmc.edu

Ref No: MDC/PG/ Date: 06-03-2025

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Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BM0122009
Postgraduate Student,
2022-23 Batch,
Department of Paediatrics
J. N. Medical College, Belagavi.

ETHICAL CLERANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : domc@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 105 **Date: 07/04/2023**

To,
(REG NO: BM0122009)
PG Student in Paediatrics
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

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(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS

BBB	–	Blood-Brain Barrier
BERA	–	Brainstem Evoked Response Audiometry
CGA	–	Corrected Gestational Age
CNS	–	Central Nervous System
CPAP	–	Continuous Positive Airway Pressure
CTR1	–	Copper Transporter 1
Cu/Zn SOD	–	Copper-Zinc Superoxide Dismutase
DMT1	–	Divalent Metal Transporter 1
EEG	–	Electroencephalogram
ELBW	–	Extremely Low Birth Weight
ERK	–	Extracellular signal-regulated kinase
GA	–	Gestational Age
GABA	–	Gamma-Aminobutyric Acid
GI	–	Gastrointestinal
HINE	–	Hammersmith Infant Neurological Examination
HNNE	–	Hammersmith Neonatal Neurological Examination
ICC	–	Intraclass Correlation Coefficient
KMC	–	Kangaroo Mother Care
LBW	–	Low Birth Weight
LTP	–	Long-Term Potentiation
LSCS	–	Lower Segment Cesarean Section
MDI	–	Mental Development Index
MRI	–	Magnetic Resonance Imaging
MT	–	Metallothioneins

NF-κB	–	Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells
NICU	–	Neonatal Intensive Care Unit
NVD	–	Normal Vaginal Delivery
NSC	–	Neural Stem Cell
p53	–	Tumor Protein 53 (a transcription factor involved in cell cycle regulation)
PDI	–	Psychomotor Development Index
RCT	–	Randomized Controlled Trial
RDA	–	Recommended Dietary Allowance
RNA	–	Ribonucleic Acid
SOD	–	Superoxide Dismutase
VLBW	–	Very Low Birth Weight
ZnFPs	–	Zinc Finger Proteins
ZnT1	–	Zinc Transporter 1
ZnT2	–	Zinc Transporter 2

ABSTRACT

Background: Zinc is essential for growth and neurodevelopment, yet preterm neonates are at risk of deficiency. This randomized controlled trial evaluates the effects of higher-dose zinc supplementation (2 mg/kg/day) versus a lower dose (0.5 mg/kg/day) on growth and neurodevelopment in preterm neonates up to three months corrected gestational age (CGA).

Methods: Preterm neonates were randomized into two groups and assessed for anthropometric growth, serum zinc and copper levels, and neurodevelopmental outcomes using the Hammersmith Infant Neurological Examination (HINE).

Results: By three months CGA, the higher-dose zinc group had significantly greater weight ($p = 0.018$), length ($p = 0.002$), and head circumference ($p = 0.009$) gains, along with better HINE scores ($p < 0.001$). Serum zinc levels were higher ($p < 0.001$), but serum copper levels decreased significantly ($p < 0.001$), suggesting potential zinc-induced copper depletion.

Conclusion: Higher-dose zinc supplementation enhances growth and neurodevelopment in preterm neonates but may require concurrent copper monitoring. Further research is needed to determine the optimal dosage and long-term effects.

Keywords: Zinc, preterm neonates, growth, neurodevelopment, supplementation, RCT.

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INTRODUCTION

Zinc is an indispensable element and is vital various day to day functions, including immune regulation, cellular processes, enzymatic reactions, and neurological development.[1] It is particularly important during infancy, a phase characterised by rapid physical and cognitive growth. Its defictis cause global health malady especially among neonates and infants, as inadequate levels can cause impaired growth, weakened immune defences, and hindered brain development.[2,3] While the effects in older children and adults are well-established, its specific impact during the neonatal period remains less explored. Investigating their effect of different dosages of supplementation of zinc to determine growth and neurodevelopment is crucial for improving infant health outcomes.[4]

Neonatal zinc deficiency may arise due to low maternal zinc levels during pregnancy, preterm birth, or declining zinc content in breast milk. Since the placenta transfers the highest amounts of zinc during the third trimester, preterm infants are particularly susceptible.[5] Additionally, infants born in regions where maternal diets lack sufficient zinc are at greater risk.[6] Zinc deficiency can contribute to delay in growth, dysfunction of immune system, and greater infection risk like diarrhoea and pneumonia.[7,8]

Beyond its role in physical health, zinc is vital for brain development, as it influences neurogenesis, synaptic function, and neurotransmitter activity.[9]

Insufficient zinc levels in early period of life known to be associated with cognitive delays, motor impairments, and attention deficits.[10] Given the rapid brain development occurring during infancy, a deficiency during this crucial period may have lasting neurodevelopmental consequences. [11]

NEED FOR STUDY

Despite evidence supporting zinc supplementation in childhood, limited data exist on its specific effects in neonates, particularly concerning different dosing regimens. Most studies assess general supplementation benefits but do not compare dosage variations, leaving uncertainty about whether higher doses lead to better outcomes or pose risks, such as micronutrient imbalances.

To address this gap, this study employs a single blind, randomized controlled trial (RCT) to evaluate the effects of two different doses of zinc supplementation on growth and neurological development in neonates. By systematically assessing anthropometric indicators, cognitive function, and developmental milestones, this research aims to provide evidence-based insights into optimal dosing strategies for neonatal zinc supplementation. Findings could help refine nutritional guidelines and shape public health policies to improve early childhood health outcomes.

AIMS AND OBJECTIVES

Primary Objective:

1. To assess the effect of zinc supplementation on neurological development and growth of late preterm neonates

Secondary Objective:

1. To determine association of serum zinc levels and morbidities
2. To assess the correlation of serum copper to serum zinc levels

REVIEW OF LITERATURE

Preterm neonates are those babies who are born before 37 weeks period of gestation and pose major global issue on health. They are classified based on gestational age, with each category posing unique medical challenges and risks. These classifications include:

Table 1. Classification of preterm infants

Category	Period of gestation
Late Preterm	34 w - 36w+6days
Moderate Preterm	32 w - 33w+6days
Very Preterm	28w - 31w+6days
Extreme Preterm	<28 w

The incidence of adverse health outcomes increases as gestational age decreases, with the most premature infants facing the highest risks. Prematurity is a important reasons of neonatal mortality worldwide, accounting forinfant deaths. Data from 2020 indicates that nearly 2.4 million neonates died globally, with preterm birth being a major contributing factor. This equates to more than 6,500 neonatal deaths per day, highlighting the urgency for improved preventive measures and care strategies. (12)

Beyond immediate survival challenges, preterm infants are prone to long-term complications. Many experience respiratory difficulties, developmental delays, sensory involvements such as vision and hearing loss, and cognitive disabilities.

These health challenges impact not only the child but also impose a strain on families and healthcare systems.[13,14]

Given the serious consequences of preterm birth for individuals and societies, improving neonatal care, strengthening prenatal health services, and implementing global health interventions are crucial. Advancing preventive strategies and enhancing neonatal care practices can help lower the prevalence of preterm births and improve health outcomes, thereby reducing the overall public health impact. [6,15]

Zinc Deficiency

Establishing accurate reference ranges for serum zinc concentrations across different age groups is essential for diagnosing and managing potential deficiencies. The following table summarizes the normal serum zinc levels for various populations: Zinc deficiency in preterm neonates is considered as<50 µg/dL.[16-20]

Table 2 : Serum levels in Zinc populations

Age Group	Normal Range (µg/dL)	Normal Range (µmol/L)
Neonates (Cord Blood)	50–150	7.6–23.0
Preterm Neonates	71.9 – 143.9	11-12
Term Neonates (0–6 months)	65.4 – 111.2	10–17
Infants & Children	70–120	10.7–18.4
Adults	70–150	10.7–23.0

Zinc Deficiency and Neonatal Health

Zinc deficiency remains a concern, particularly in regions with limited resources where proper nutrition is often lacking. The World Health Organization (WHO) estimates that about 17% of people worldwide are affected, with neonates being among the most susceptible. Research by Prasad et al. and Wessells et al. indicates that in low- and middle-income countries, neonatal zinc deficiency is observed in approximately 20% to 30% of cases. [21,22] In contrast, wealthier nations report lower prevalence rates, though preterm and low birth weight (LBW) infants continue to be at higher risk. [23]

In India, a study by Sharma et al. (2019) stated 32% of neonates exhibited suboptimal serum zinc levels, with a higher prevalence in those born to mothers with poor nutritional status.[24] Similarly, research from sub-Saharan Africa identified a 27% prevalence of zinc deficiency along neonates.[25] LBW infants are particularly vulnerable to this micronutrient deficiency. Some studies have estimated that up to 20% of LBW neonates suffer from zinc insufficiency, highlighting the global burden of the condition, especially in regions with limited healthcare access.

Zinc and Early Childhood Development

Infancy is a crucial phase of growth and development, necessitating optimal nutritional support. It is a micronutrient that supports numerous biological functions.

Research indicates that neonates are born with limited bioavailable zinc reserves, which are crucial for metabolic and cellular functions. Due to this restricted zinc pool, deficiency during the neonatal period can lead to early-onset growth failure, even when overall tissue zinc concentrations appear within normal limits. Ensuring an adequacy of bioavailable zinc is therefore essential to support proper growth and neurodevelopment during this crucial stage of life. [26]

Essentiality of Zinc

It is a vital element involved in numerous biological processes at both the cellular and molecular levels. It provides structural stability and helps in catalyses of various enzymes of the metabolic pathways.[27] Zinc is essential for nearly 10% of the human proteome, demonstrating its significant role in maintaining physiological functions.

Zinc contributes to a range of cellular processes, including:

- Transcription Factors: It is essential for regulation of gene, binding to transcription factors that control DNA transcription into RNA. This mechanism influences cellular growth, differentiation, and environmental responses.
- Structural Proteins: It helps stabilize proteins and membranes, preserving cellular architecture and functionunder varying conditions.
- Enzymes: Zinc serves as a cofactor for several essential enzymes, including:
- Metalloproteases: Enzymes that facilitate protein breakdown, essential for tissue remodeling, wound healing, and cell signaling. Zinc primarily functions as a cofactor for over 300 metalloenzymes, most of which are bound to metallothioneins (MT). [28,29]
    - Nitric Oxide Synthase: that which regulate blood flow and immune function.
    - Superoxide Dismutase (SOD): protect cells from oxidative stress and damage.

Zinc's diverse functions underscore its indispensability in maintaining cellular health, immune function, wound healing, and oxidative stress prevention.

Role of Zinc in Neonatal Growth

It is essential for neonatal growth, a phase marked by rapid physical and physiological development. It plays a crucial role in:

- Protein Synthesis: Supports cellular growth, tissue repair, and organ development during infancy.
- Carbohydrate & Lipid Metabolism: Ensures adequate energy supply to sustain the high metabolic demands of neonatal growth.
- DNA & RNA Synthesis: Regulates cellular division and differentiation, facilitating proper cell formation and function across different body systems.

A RCT by Lönnerdal et al. (2017) found that zinc supplementation significantly enhances weight gain in neonates, especially infants are more vulnerable to zinc deficiency due to factors such as illness, prematurity, and inadequate dietary intake.(30).

Zinc and Its Role in Brain Development

It plays a important role in brain development and function. It regulates biological processes critical for brain structural integrity and cognitive function.[31] During early brain development, zinc supports neurotransmission, synaptic plasticity, neuronal differentiation.[32] Additionally, zinc contributes to gene regulation, cell survival, and the formation of neural structures involved in memory, motor control, and higher cognitive functions. [33]

Zinc is not uniformly distributed throughout but is densely concentrated in specific regions of brain where it supports distinct neurological functions. These regions include:

- Hippocampus – Essential for learning and memory, zinc regulates synaptic plasticity, particularly in induction of long-term potentiation (LTP), which is crucial for memory encoding and retrieval.[34]
- Cortex – needed for decision-making, planning, and abstract thinking. Zinc supports neuronal growth and differentiation, ensuring cognitive development. [35]
- Basal Ganglia – Involved in motor control and procedural memory. Zinc plays a role in neuronal activity regulation, influencing movement coordination. [36]
- Amygdala – A key region for emotional processing, zinc affects emotional regulation and stress responses. [37]
- Olfactory Cortex – Crucial for sensory processing, particularly in the interpretation of olfactory signals.[38]

These brain regions, integral to cognitive, emotional, sensory, and motor functions, are highly sensitive to zinc levels. Adequate zinc availability during foetal stage of rapid brain growth and synapse formation is needed for adequate neurological outcome and function.

It is needed neurotransmission, the process governing neuronal communication. It plays a important role in brain signaling, influencing learning, memory, behaviour, and emotional regulation.[39] As a cofactor for multiple enzymes including glutamate and gamma-aminobutyric acid (GABA), which are critical for excitatory and inhibitory signaling in the brain. [34]

- Glutamate – The primary excitatory neurotransmitter, glutamate facilitates neuronal activation and is essential for cognitive function and synaptic plasticity.[40]

- GABA – The major inhibitory neurotransmitter, GABA counterbalances excitatory signals, playing a vital role in mood regulation, anxiety control, and neural stability.[41]

Zinc ensures a dynamic balance between excitatory and inhibitory neurotransmission, which is critical for maintaining neuronal excitability and cognitive processing.[42]

Beyond neurotransmitter synthesis, zinc modulates ion channels that regulate neuronal excitability. These channels control the movement of ions, which are essential for synaptic transmission and generation of action potential. [43]

- Calcium Channels – Zinc regulates its influx, which is crucial for neurotransmitter release and synaptic signaling. [44]

- Sodium & Potassium Channels – These channels help maintain the resting membrane potential and action potential propagation. [45]

Zinc's role in synaptic plasticity and long-term potentiation (LTP) is important for memory formation and learning (46). By influencing LTP, zinc supports the strengthening of synaptic connections, a fundamental process in cognitive development.

Zinc is integral to multiple biological processes within the CNS. Zinc influence a process by which synapses together weaken 04 strengthen over time based on the neuronal activity which is essential for learning and memory function.[47]

Additionally, zinc is vital for neuronal development, influencing t\differentiation of neural stem cells (NSCs) into astrocytes, oligodendrocyte and neurons key components for proper brain structure and function.[48,49] By modulating these pathways, zinc promotes the survival of newly differentiated neurons, facilitating their integration into neural networks essential for brain function. [50,51]

The blood-brain barrier (BBB) is a specialized membrane that carefully control the movement of particles between the bloodstream and the brain, ensuring a stable environment for neural cells.[52] BBB shields the brain from harmful toxins, pathogens, and fluctuations in blood composition. Zinc contributes in formation, maintenance, and repair of the BBB, ensuring that essential nutrients, like glucose, a mino acids, can efficiently enter brain while preventing influx of harmful substances. [53]

Zinc influences the expression and function of zinc transporters, particularly ZnT1 and ZnT2, which regulate zinc homeostasis at the BBB. These transporters help control the movement of zinc, ensuring that adequate amounts are transported into the brain while preventing excessive accumulation, which could lead to neurotoxicity (54). Any dysregulation of zinc levels—whether due to deficiency or excess—can compromise BBB integrity, leading to increased permeability and heightened susceptibility to neurological disorders. [34]

Moreover, zinc acts as an antioxidant, helping to reduce oxidative stress, which can otherwise lead to BBB dysfunction. By stabilising cell membranes, zinc c reinforcing its protective role.[55] Additionally, zinc acts as an antioxidant and mitigate effects of neuroinflammation and oxidative damage.[56]

Neurogenesis refers to the formation of new neurons within the CNS. It unfolds in several critical stages, including the proliferation of neural stem cells, their migration to specific brain regions, differentiation into specialized neural cells, and their subsequent survival as functional parts of existing neural networks. [55]

Step 1: Neural Stem Cell (NSC) Proliferation and Migration

The first step in neurogenesis involves the proliferation of NSCs, which divide to generate a pool of precursor cells. These precursor cells then undergo migration to their designated locations within the brain. There are two primary migration routes: radial migration, where cells travel along radial glial fibers toward the surface of the developing brain, and tangential migration, where cells move parallel to the brains surface, commonly seen in interneurons. This migration ensures that neural cells reach the appropriate areas to form functional brain structures.[57]

Step 2: Neuronal Differentiation and Integration

Once the precursor cells reach their intended locations, they differentiate into specialized types of neural cells, including neurons, astrocytes, and oligodendrocytes. The differentiation process is regulated by molecular signals, including those facilitated by zinc. Newly formed neurons must integrate into the existing neural networks, establishing synaptic connections to ensure proper brain function. This integration is critical for the development of functional and mature brain structures.[58]

Step 3: Role of Zinc in Neurogenesis

It is a vital regulator throughout the neurogenesis process. Zinc is concentrated in regions of the brain, such as dentate gyrus in hippocampus, which are actively engaged in neurogenesis. These zinc-dependent proteins such as zinc finger

proteins (ZnFPs) direct NSCs toward a neuronal fate, ensuring proper neural development.[34]

Step 4: Zinc's Impact on Neural Stem Cell Proliferation

During the perinatal stage, zinc is needed for the proliferation of NSCs. Zinc deficiency can impair this process, limiting the formation of new neurons. Studies have shown that zinc supplementation promotes NSC proliferation in various brain regions, which includesubventricular zone, emphasizing the necessity of adequate zinc levels for early brain development. Zinc also plays a role in transcription factor regulation, particularly NF-κB and AP-1, which influence cell division and differentiation during neurogenesis. [59]

Step 5: Zinc's Role in Adult Neurogenesis

In adults, neurogenesis is more restricted, occurring primarily in the hippocampus. Zinc continues to regulate neurogenesis by activating p53-dependent pathways, which influence NSC proliferation and survival. Zinc deficiency in adult brains leads to decreased neurogenesis, particularly memory formation. Zinc deficiency inhibits proper neurogenesis and neuronal survival. [60]

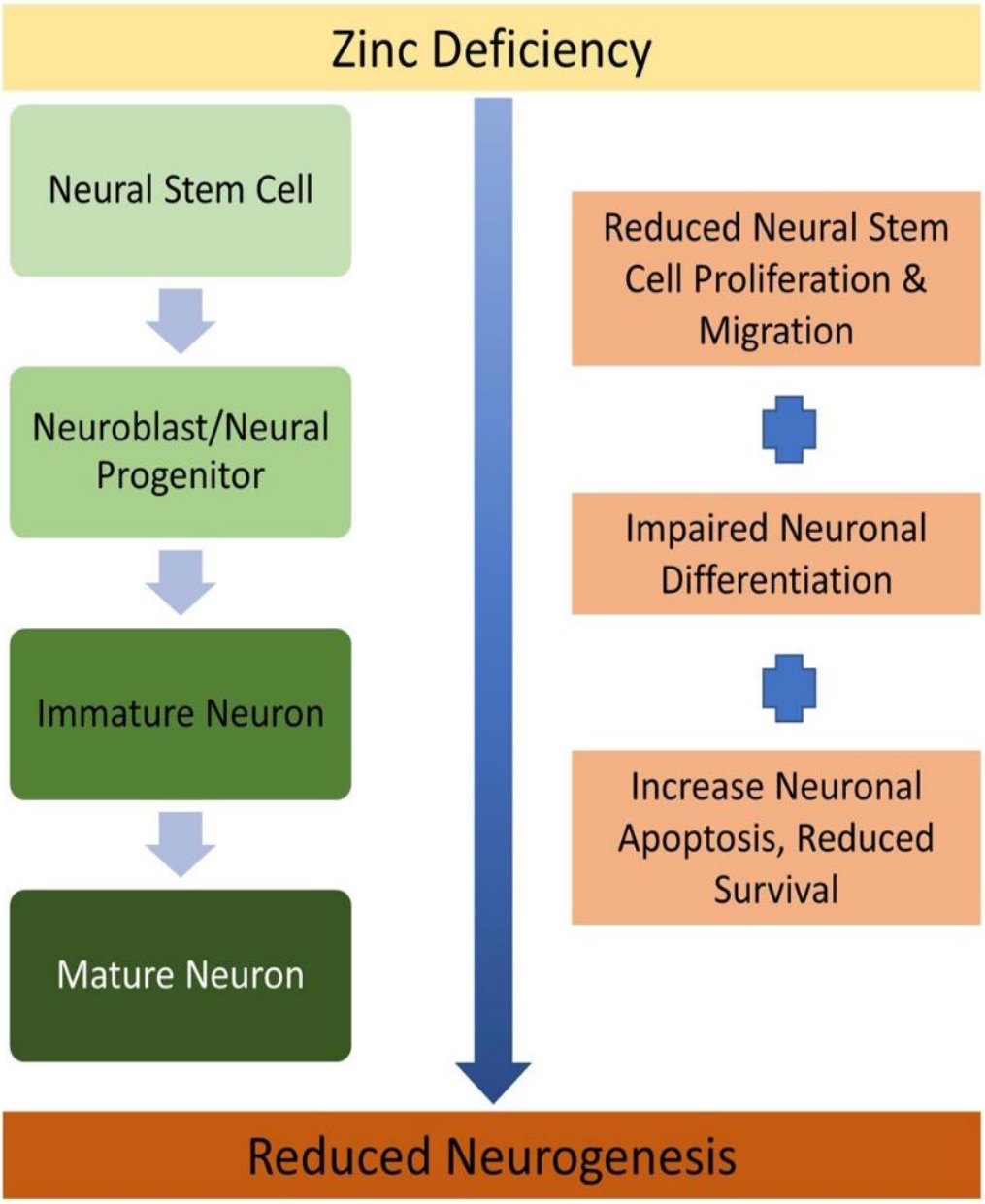
Step 6: Neuronal Differentiation in the Adult Brain

Zinc deficiency impairs this process, leading to a decrease in number oWhen zinc levels are insufficient, apoptosis is triggered in neural precursors, leading to reduced neurogenesis and cognitive impairments. [56]

In conclusion, Each stage of neurogenesis is intricately regulated by zinc, from proliferation of NSC to their differentiation and survival as mature neurons. Zinc's role is fundamental for brain development, especially during critical periods such as prenatal and early postnatal stages. [59]

Zinc deficiency during early brain development can lead to a decreased number of neurons, which is essential for memory and spatial learning. Conversely, zinc supplementation has been shown to enhance neurogenesis and promote neuronal differentiation, supporting the formation of functional neural circuits. These findings underscore the importance of zinc in sustaining neurogenesis throughout life, reinforcing its crucial role in cognitive development and brain function.

Figure 1: Zinc deficiency and neurogenesis



Body Zinc Status and Assessment

Body zinc status refers to the availability of zinc which is necessary for optimal biological functions of body. Several factors influence an individual's zinc status, including dietary intake, consumption of phytates, gastrointestinal health, and the efficiency of zinc excretion and reabsorption.[61] Despite understanding these contributing factors, many aspects of zinc metabolism and status assessment remain unclear. [62]

There are several methods used to assess zinc status, such as measuring zinc levels in biological components including blood, urine, hair, feces, sweat, and saliva.[63]

While these biomarkers can provide valuable insights into zinc levels, no single biomarker offers a complete and accurate measure of overall body zinc status.[23]

Serum Zinc in Term and Preterm Infants

Zinc status in, both term and preterm, varies significantly due to differences in growth rate, metabolic demands, and nutritional intake. In term infants, serum zinc levels typically range from 10–17 µmol/L (65.4–111.2 µg/dL) at birth, comparable to those in healthy adults. These levels peak at birth, decline over the first four months, and then stabilize as dietary zinc intake from breast milk or formula compensates for the initial depletion.

In contrast, preterm infants exhibit higher serum zinc levels at birth, typically 11–22 µmol/L (71.9–143.9 µg/dL), reflecting their increased zinc demands for accelerated growth and development. However, serum levels experience a sharp

decrease within the first 6 to 12 weeks, highlighting the critical need for zinc supplementation to prevent deficiency.

Very low birth weight (VLBW) and extremely low birth weight (ELBW) have even higher initial zinc levels (12–24 µmol/L [78.5–156.9 µg/dL] in VLBW and 13–26 µmol/L [85.0–169.9 µg/dL] in ELBW infants), but experience a more pronounced drop due to their higher metabolic demands and limited zinc stores at birth (64-66).

Table 3 : Serum Zinc levels and trends in neonatal population

Neonatal Population	Serum Zinc (µmol/L)	Converted Serum Zinc (µg/dL)	Key Trends
Term Infants	10–17	65.4 – 111.2	Peaks at birth, declines over 4 months, then stabilizes
Preterm Infants	11–22	71.9 – 143.9	Higher at birth, declines significantly over 6–12 weeks
Preterm (Very Low Birth Weight)	12–24	78.5 – 156.9	More pronounced decline, higher risk of deficiency
Preterm (Extremely Low Birth Weight)	13–26	85.0 – 169.9	Highest risk of depletion due to rapid growth demands

Dietary Zinc Requirements for Infants

The dietary reference values for zinc is for ensuring optimal growth, immune function, and neurological development throughout different life stages. The Recommended Dietary The Recommended Dietary Allowance (RDA) provides a guideline for daily zinc intake to meet the nutritional requirements.[67]

The RDA for zinc in infants and young children are as follows:[68]

- **0–6 months of age :** 1.5–2 mg/day
- **7–12 months of age :** 3–8 mg/day
- **1–3 year of age :** 4–9 mg/day (68)

These intake recommendations help maintain optimal enzymatic function, immune resilience, and cognitive processes during early childhood. [23]

Preterm infants require greater zinc due to their accelerated postnatal growth and limited in-utero zinc stores. Studies suggest that the estimated zinc requirement for preterm neonates falls at 0.4–0.5 mg/kg/day to support proper neurodevelopment, immune function, and overall metabolic stability.[69] Without adequate zinc supplementation, preterm infants face an increased risk of growth faltering, impaired neurocognitive development, and immune dysfunction. [66]

Zinc Reserves in Neonates

At birth, newborns possess significant zinc reserves, accounting for approximately 25% of their total body zinc. These reserves are mainly stored in the liver, bound to metallothioneins, with most accumulation occurring during last trimester. During the first few months of life, liver zinc levels gradually decline,

reaching stability around four months, providing an essential source of zinc when dietary intake is inadequate.[70]

Zinc Content in Human Breast Milk

Beast milk serves as the main source of zinc for neonates particularlyin colostrum, the nutrient-dense milk produced shortly after birth, averaging around 8 mg/L. However, this level drops significantly within the first week of lactation, decreasing to nearly half by day seven. In the subsequent months, the decline continues more gradually, reaching approximately 2 mg/L by two months, 1 mg/L by six months, and 0.5 mg/L by twelve months.[71-73]

Though breast milk is generally sufficient however as the volume of breast milk intake increases, the declining zinc concentration may become marginally inadequate. This makes it essential to introduce complementary feeding at six months to sustain adequate zinc intake for optimal growth and development. The gradual introduction of nutrient-rich solid foods ensures that an infant continues to receive adequate zinc beyond the exclusive breastfeeding period.[74]

Maternal Zinc Deficiency

Worldwide, approximately 82% women who are pregnant are considered at risk of deficiency.[75] Despite this, maternal plasma zinc levels do not directly relate with zinc concentrations in breast milk. Research indicates that even when maternal zinc levels are less, it maintains relatively stable zinc concentrations, suggesting biological mechanisms that regulate its transfer.[76] Furthermore, maternal factors such as age, parity (number of previous births), and smoking habits appear to have minimal impact on zinc levels in breast milk.[77]

Its deficiency in pregnancy has profound effects on fetal development, particularly the immune system. Studies suggest that inadequate zinc levels may lead to smaller immune organ sizes (e.g., thymus, spleen) and impair the function of key immune cells, which includes lymphocytes and neutrophils. Consequently, infants born to zinc-deficient mothers may have weaker immune responses, making them more susceptible to infections. [78]

Beyond immune effects, maternal zinc status influences epigenetic regulation, impacting gene expression without altering DNA sequences. DNA methylation of zinc transporter genes (e.g., ZnT5) has been associated with gestational length, with lower zinc levels linked to shorter pregnancies. A Studies show that zinc supplementation increases histone evels in cultured neuronal cells, potentially affecting neurological and immune system development in infants.[79,80]

Zinc Content in Infant Formula

The zinc content of infant formulas varies by brand and composition, typically ranging between 0.11 mg to 0.57 mg per 100 mL. These formulas are designed to provide a nutrient profile comparable to human milk, but their zinc content may differ depending on whether they are cow's milk-based or soy-based. Although many sources have higher zinc concentrations than breast milk, their bioavailability is typically lower.This is due to differences in zinc-binding compounds and absorption efficiency between human milk and formula.[81,82]

To compensate for these differences, manufacturers often fortify infant formulas with additional zinc to ensure sufficient amounts to meet their RDA. This fortification helps bridge the bioavailability gap and ensures that formula-fed infants obtain adequate zinc for healthy growth and development. [83]

Zinc Needs in Premature Infants

Premature infants have significantly greater zinc requirements due to several physiological factors that influence zinc transfer, storage, absorption, and excretion.[23]

1. Insufficient Maternal Zinc Transfer

Approximately 60% of fetal zinc is transferred towards end of pregnancy, making it a critical period for fetal zinc acquisition. Preterm infants, who miss part or all of this transfer, are at risk of deficiency. This inadequate maternal transfer can lead to impaired growth, immune dysfunction, and neurodevelopmental delays in preterm neonates. It puts premature infants at risk for metabolic and physiological challenges. [35,84]

2. Increased Zinc Demand in Premature Infants

Premature neonates require higher zinc intake due to their rapid growth and metabolic demands. However, the content in breast milk may not be adequate to fully meet the increased demands. The decline in zinc in human milk, combined with the already low baseline levels in preterm neonates, increases the risk of growth retardation and compromised immune function. Zinc supplementation is often necessary to support their higher physiological needs. [85]

3. Reduced Zinc Storage Capacity

The liver is main site for storage of zinc in the body, but premature infants have smaller liver sizes, resulting in lower zinc reserves at birth. Unlike term neonates, who accumulate hepatic zinc reserves in late gestation, preterm infants begin life with limited zinc stores. This lack of storage reduces their ability to

maintain adequate zinc levels, particularly during periods of illness or insufficient dietary intake. [86]

4. Underdeveloped Gastrointestinal System and Reduced Zinc Absorption

Premature infants have an immature gastrointestinal (GI) system, which negatively affects zinc absorption. The shortened intestinal length and reduced enzymatic activity contribute to lower zinc uptake from enteral feeding. Additionally, the presence of phytate and other inhibitors in infant formulas can further hinder zinc bioavailability. These factors make it challenging for preterm infants to absorb sufficient zinc, exacerbating deficiency risks.[87,88]

5. Negative Zinc Balance in Early Life

The combination of inadequate maternal zinc transfer, increased metabolic demands, limited storage capacity, and poor absorption leads to a negative zinc balance in preterm infants. This condition arises when zinc losses (via urine, stool, and sweat) exceed zinc intake, leading to persistent deficiency. Studies indicate that preterm neonates experience a negative zinc balance for up to two months postnatally, during which they are particularly susceptible to deficiency-related complications such as poor weight gain, immune dysfunction, and developmental delays.[89]

6. Increased Zinc Losses Through Excretion

Premature infants experience significant zinc loss through multiple excretion pathways, including:

- Intestinal losses (due to immature gut function)
- Renal excretion (higher urinary zinc output due to immature kidneys)

- Sweat losses (elevated in preterm neonates)
- Shedding of epithelial cells (contributing to further depletion)

These cumulative losses exacerbate zinc depletion and make dietary supplementation essential for maintaining adequate zinc levels.[90]

7. Increased Risk of Zinc Deficiency in Preterm Infants on Parenteral Nutrition (PN)

Many preterm neonates rely on PN due to feeding difficulties or medical complications. However, standard PN formulations may lack adequate zinc content, further increasing the risk of deficiency. Inadequate zinc levels may lead to delayed growth, impaired wound healing, and weakened immune function. Thus, careful monitoring and individualised zinc supplementation are essential to prevent zinc deficiency-related complications in premature infants.[91,92]

Interaction of Serum Zinc and Copper

Zinc and copper are elements which interact closely in various physiological processes, including enzymatic functions, immune response, and neurological development. Their balance is crucial for maintaining homeostasis, as an excess of one mineral can adversely affect the metabolism and absorption of the other. [93,94]

They compete for absorption in the gastrointestinal tract through shared transport mechanisms.[95] Consuming excessive amounts of zinc, especially beyond 50 mg per day, can interfere with copper absorption and may result in Cu deficiency.[96,97] This occurs because zinc induces metallothionein production, a protein with a high affinity for copper, sequestering it within enterocytes and reducing its systemic availability. [98,99]

This imbalance disrupts essential enzymatic functions, vital antioxidant enzyme. Disruption of zinc or copper levels impairs oxidative stress regulation, increasing susceptibility to cellular damage and neurodegeneration.[100,101] Moreover, cytochrome c oxidase (CCO), a copper-dependent enzyme vital for mitochondrial respiration, is negatively impacted by excess zinc, leading to metabolic dysfunction.[102]

Clinically, prolonged high-dose zinc supplementation has been linked to secondary copper deficiency, presenting as anemia, neutropenia, immune suppression, and neurological impairments, such as ataxia and myelopathy.[103,104]

Maintaining an optimal zinc-to-copper ratio (10:1) is essential for preventing such deficiencies.[105-108].

Zinc Deficiency and Comorbidities

Literature suggests that zinc supplementation improves growth outcomes in malnourished and preterm infants.[90] With positive correlation zinc supplementation decreases with the duration and severity of diarrheal diseases and respiratory infections in infants. [73,109]

Furthermore, it is needed for brain development, synaptic function, and neurotransmission. Low zinc levels are linked to delayed cognitive development, impaired motor skills, and behavioral disorders. Preterm infants with deficiency are prone to poor neuromotor outcomes and developmental delays.[111] Zinc is also necessary for maintaining gut integrity and enzyme activity. Deficiency contributes to increased intestinal permeability, leading to malabsorption and persistent diarrhea.[112]

Dermatological manifestations of zinc deficiency include skin and mucosal lesions, such as acrodermatitis enteropathica, a severe form characterised by dermatitis, alopecia, and diarrhea. Milder deficiencies result in delayed wound healing, skin rashes, and dermatitis. [6] Inc also contributes to iron metabolism, and its deficiency has been linked to anemia. A lack of zinc is often accompanied by deficiencies in other essential micronutrients, such as iron, vitamin A, and copper, increasing overall health risks. [113]

PREVIOUS STUDIES

Limited randomized controlled trials (RCTs) have examined these effects of zinc supplementation on the growth and neurodevelopment of infants:

- **Mathur and Agarwal** conducted an RCT with 100 preterm neonates, revealing that zinc supplementation enhanced alertness and attention at 40 weeks CGA while reducing hyper-excitability up to three months corrected age. Additionally, the zinc group exhibited increased serum alkaline phosphatase levels.[114]

- **Alshaikh et al.** performed a meta-analysis of eight RCTs involving 742 infants, showing significant improvements in weight and length z-scores with supplementation. No changes were seenin head circumference or overall developmental scores. While the evidence for growth benefits was moderate, findings related to neurodevelopment remained uncertain.[115]

- **El Sadek et al.** studied 80 preterm infants and found that those supplemented with zinc showed improvements in weight, length, serum zinc, and hemoglobin levels. Higher developmental scores was recorded in this group.[116]

- On the other hand, **Nissensohn et al.** conducted a meta-analysis that did not establish a clear link between zinc intake and mental or motor development. Some studies suggested minor negative effects on motor development over short durations, with variations depending on dosage and intervention length. (117)

MATERIALS AND METHODS

SOURCE OF DATA

Newborns born at Dr. Prabhakar Kore Hospital and Medical Research Center, Belagavi, Under KLE University, during the study period were included in the study.

STUDY DESIGN

Randomised Controlled Trial, single centre, single blinded study

STUDY DURATION

One Year study period from September 2023- August 2024.

STUDY PLACE

Dr. Prabhakar Kore Hospital and Medical Research Center, Belagavi, under KLE University.

SAMPLING TECHNIQUE

Random Sampling

SAMPLE SIZE

The sample size was calculated with the following formula

Formula used for sample size calculation is

$$n = \frac{2\left(Z_{1-\beta} + Z_{1-\alpha/2}\right)^2 (1+(m-1)\rho)\sigma^2}{md^2}$$

n - Sample size required

$\mathbf{Z_{1-\alpha/2}}$ -the critical value to the chosen significance level (α). For a two-tailed test with α = 0.05, the critical value is $Z_{1-\alpha/2}$ =1.96).

$\mathbf{Z_{1-\beta}}$ - The critical value for the desired power of the study (e.g., 1−β=0.8 corresponds to a power of 80%, and $Z_{1-\beta}$ =0.84).

ρ - The intraclass correlation coefficient (ICC), representing the similarity of individuals within a cluster. A higher ρ means more similarity within clusters.

$\mathbf{\sigma^2}$ -The variance of the outcome measure.

m - number of participants per cluster

d - The minimum detectable difference (effect size) between groups

A 5% significance level corresponds to a critical value of $\mathbf{Z_{1-\alpha/2}}$ **= 1.96**.

An 80% statistical power (**1−β = 0.80**) corresponds to $\mathbf{Z_{1-\beta}}$ **= 0.84**.**ρ =0.3:** Correlation between outcome variables at different time points

m=4: Number of time points (head circumference checked 4 points)

σ=10.108: Pooled standard deviation

d=50.38−45.59=4.79: Meaningful difference

Substituting these values in the above formula and adjusting for 10% attrition

Thus, a total of **70 participants (35 per group)** was determined to ensure adequate power for detecting the specified difference.

INCLUSION CRITERIA

- Infants born late preterm with low birth weight
- Received breast milk exclusively during the study period.
- Infants who take enteral feeds by 3rd day of life

EXCLUSION CRITERIA

- Infants with congenital anomalies/ malformations
- Infants who underwent GI surgery
- Infants kept nil per oral for longer than 3 days of life or on formula feeds.

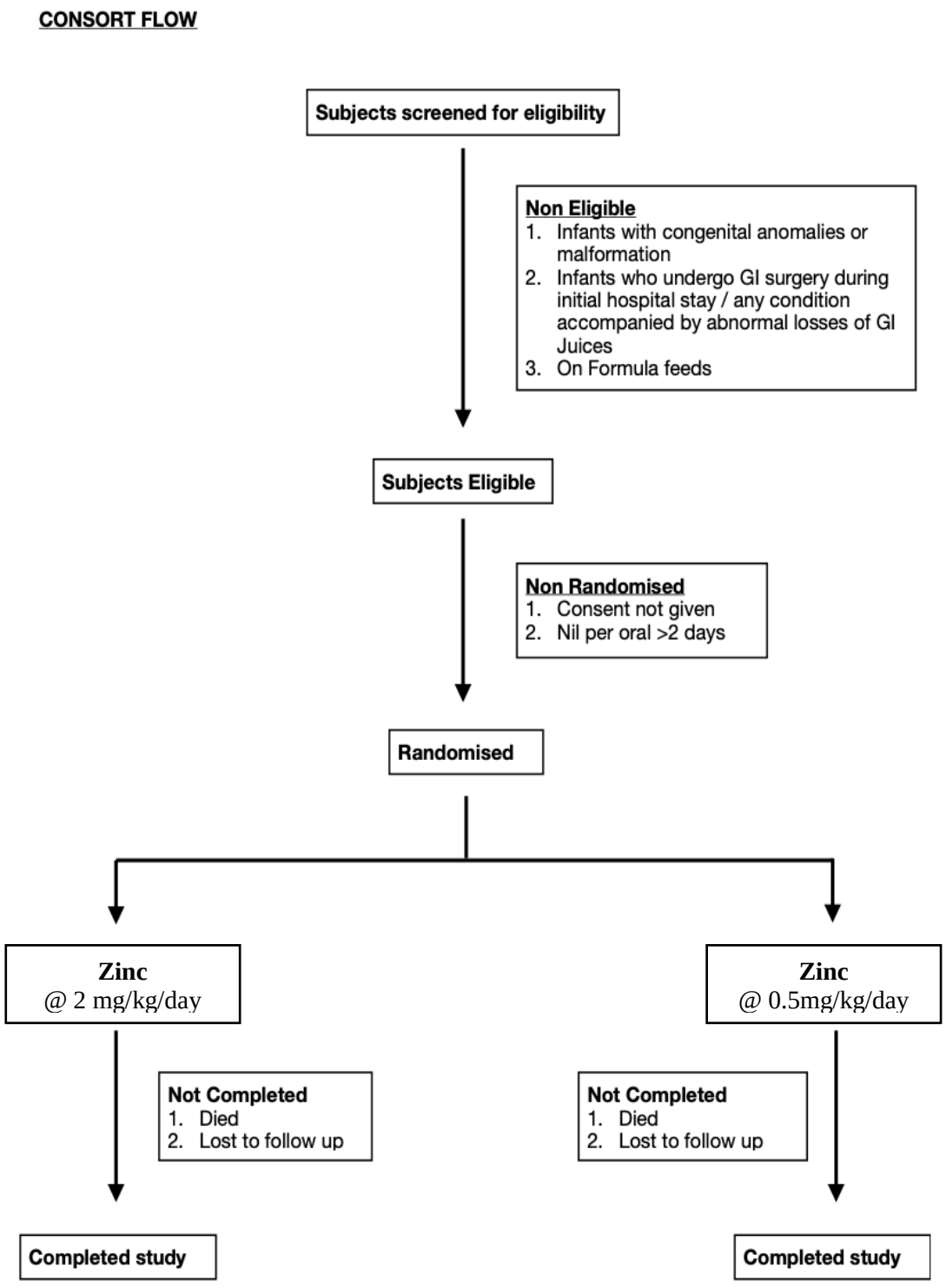
ETHICAL CLEARANCE

The study received ethical approval from the JNMC Institutional Ethical Committee (Ref No: MDC/JNMCIEC/105) on April 7th , 2023.

C.T.R.I REGISTRATION

The study was registered with the Clinical Trial Registry of India (CTRI) before sample collection and received approval under the registration number CTRI/2023/06/053625.

Figure 2: Consort flow for Screening of Neonates



INFORMED CONSENT

Parents of eligible neonates were briefed about the study's nature, and written informed consent was obtained. Consent forms were available in English and the region's major languages, including Kannada, Marathi, and Hindi (ANNEXURE 1).

STUDY PROTOCOL

The RCT was conducted at Dr. Prabhakar Kore Hospital, Belagavi, which provides specialized care for preterm neonates meeting the eligibility criteria. Data were recorded using a pre-designed pro-forma (ANNEXURE 2) and analysed statistically.

DATA COLLECTION PROCEDURE

Eligible neonates were enrolled and their data recorded using a detailed, structured pro-forma. Participants were randomized into two study groups. Randomization assignments were kept in sealed, opaque envelopes numbered sequentially and opened only after consent was obtained.

- **Group 1:** Received Zincovit Drops at 2 mg/kg/day.
- **Group 2:** Received Zincovit Drops at 0.5 mg/kg/day.

Both groups received multivitamin drops until three months of corrected gestational age. Mothers were trained in supplement administration, with instructions to repeat the dose if vomiting occurred within 30 minutes of administration. Compliance was monitored via log sheets and remaining drug volume.

MEASUREMENTS

1. ANTHROPOMETRY

Measurements taken at birth, discharge, 40 weeks CGA, and 3 months CGA:

They included Occipitalfrontal circumference, weight and length.

- **Occipitofrontal circumference:** measurement of head circumferences done using a Schorr tape with an accuracy of 1 mm, by placing it over the occipital protuberance and just over supraorbital ridge and the glabella in front, once being positioned correctly it was pull tight to compress the hair and the skin, but not too tight causing injury to the baby. Three measurements were taken for each baby and mean of the three values was taken as final value.

- **Nude weight:** The infant's weight was measured on Essae-BS-250 electronic weighing scale with precision of 0.001 kg. Infant was weighed naked with no clothing or diaper after making sure that scale was placed on flat, hard, even surface with proper calibration. Three readings were noted and mean of the three readings was taken as final value.

- **Length (crown-to-heel):** The infant's length was measured on an infantometer board after placing it on a horizontal and level surface. Three measurements for each baby were taken and mean of the three values was taken as final value after measuring it to nearest 0.1cm.

2. BLOOD TESTS

- Basal serum zinc levels within 48 hours of life.
- Serum zinc and copper levels at 3 months CGA.

3. NEUROLOGICAL ASSESSMENTS

- At 40 weeks CGA, neurological examination was done based on **Hammersmith Neonatal Neurological Examination Scale.**
- At 3 months CGA neurological examination was done based on **Hammersmith Infant Neurological Examination Scale.**

4. ADDITIONAL MONITORING

- Feeding patterns, diarrhoea, respiratory distress, fever, lethargy, vomiting, and comorbidities.
- Neurodevelopment interpretation with complementary investigations as needed (e.g., cranial ultrasound, CT/MRI, fundus examination, EEG, BERA).

SPECIMEN COLLECTION AND PROCESSING

1. Sample Collection:

- 2 ml of blood was drawn into a yellow-topped vacutainer, labeled, and transported to the Hi-Tech of the hospital.

Fig 3 : The samples collected are labelled for identification and transported to the laboratory immediately.

2. Sample Processing:

Fig 4: The sample is centrifuged for 10mins at 3500 rpm

- After clotting, The blood sample was spun in a centrifuge at 3500 RPM for 10 minutes using an Eppendorf Centrifuge 5702R to separate the serum

- The serum was transferred to a red-topped vacutainer and stored in a -20°C ice-linedrefrigerator.

3. Analysis of Zinc and Copper Levels:

- **Zinc Levels**: Estimated using the *High-Q Zinc ML Kit* by Pariksha Biotech Pvt Ltd, which uses the 5-Br-PAPS method which is based on the colorimetric reaction where the zinc ion react with reagent to form a colored complex, which is then measured by Erba CHEM -5 Plus.

- **Copper Levels**: Estimated using the *High-Q Copper ML Kit* by Pariksha Biotech Pvt Ltd, employing the Di Brom-PAESA method, which also relies on a colorimetric reaction to measure copper levels Erba CHEM -5 Plus in the sample.

Fig 5 : Erba CHEM - 5 Plus

Zinc Analysis

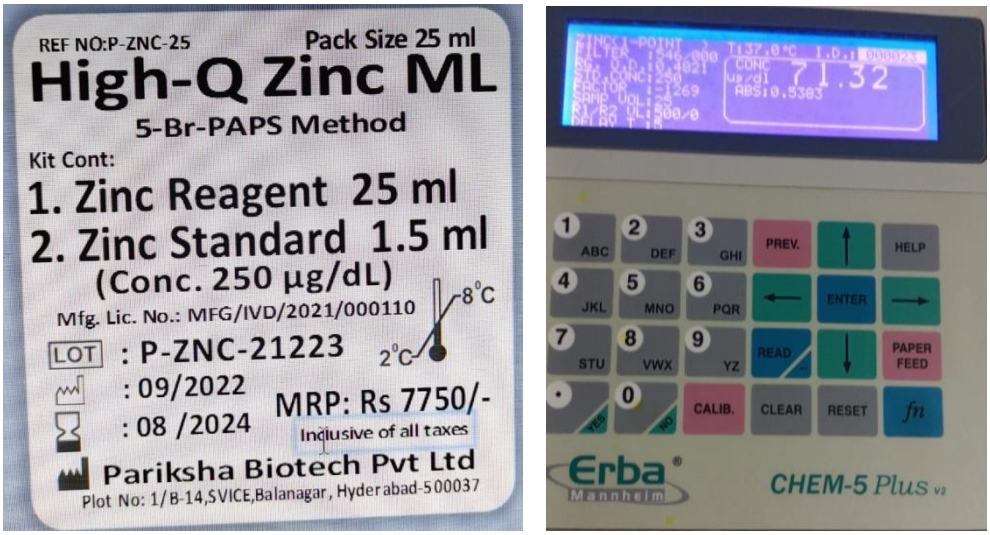
Zinc concentration is measured by its reaction with Nitro-PAPS in an alkaline solution, creating a colored complex. The colour intensity reflects the amount of zinc in the sample. The zinc estimation uses CHEMCHEK kits, which include two reagents: R1 (Borate Buffer, Salicylaldoxime, Dimethylglioxime) and R2 (Nitro-PAPS). These are mixed in a 4:1 ratio to prepare the working reagent. The reagents are stored at 2–8°C and are stable for 4 weeks after opening.

For the analysis, 1000 µL of working reagent is added to cuvettes, followed by distilled water, zinc Standard, and the sample. After incubating for 10 min at 30°C.

Zinc Concentration (µg/dL) = $\frac{\text{Absorbance of Standard}}{\text{Absorbance of Sample}} \times 20$

To convert values from µg/dL to µmol/L, multiply by a factor of 0.153.

Fig 6 : Zinc kit and analysis



Copper Analysis

Copper concentration is measured by the Di-Br-PAESA method, where copper reacts with the chromogen Di-Br-PAESA, creating a colored complex. The Copper Reagent kit contains two reagents: R1 (Acetate Buffer) and R2 (Di-Br-PAESA). These are mixed in equal parts to prepare the working reagent. The reagents are stable for 4 weeks.

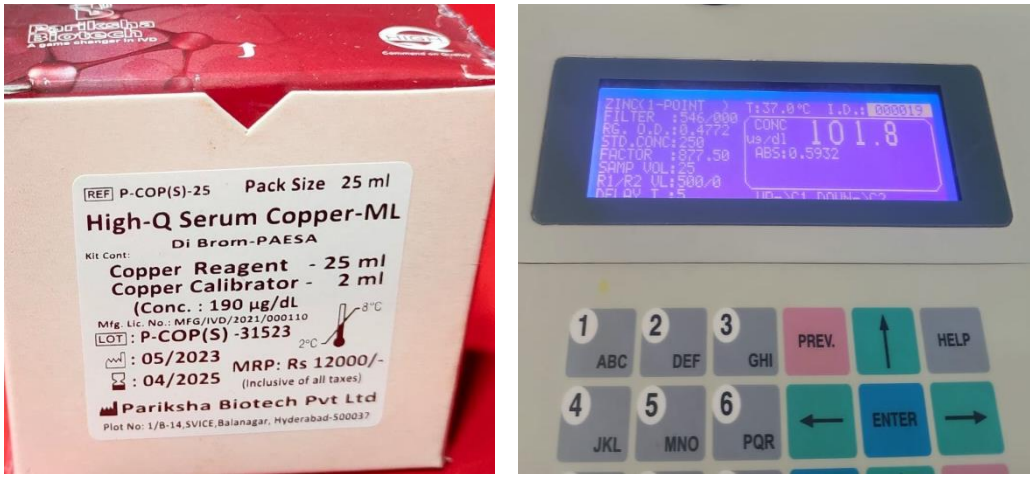
For the analysis, 1000 µL of working reagent is added to cuvettes, followed by distilled water, Copper Standard, and the sample. After incubating for 10 min at 30°C.

Absorbance is measured at 580 nm. Copper concentration is calculated using the formula:

$$\text{Copper Concentration (µg/dL)} = \frac{\text{Absorbance of Standard}}{\text{Absorbance of Sample}} \times 20$$

To convert values from µg/dL to µmol/L, multiply by a factor of 0.1574**.**

Fig 7: Copper kit and analysis



STATISTICAL ANALYSIS

The data collected was coded, organized, and stored in a Microsoft Excel spreadsheet. Descriptive statistics were used to summarize the data, with mean and standard deviation calculated for quantitative variables, while categorical variables were presented as frequencies and proportions. Data visualization techniques included bar charts, pie charts, and box plots.

To assess the relationship between explanatory variables and categorical outcomes, cross-tabulation and percentage comparisons were performed, with the chi-square test used to determine statistical significance. Independent sample t-tests were applied to compare quantitative variables between two groups, whereas paired sample t-tests were used for within-group comparisons of paired quantitative data.

A p-value of less than 0.05 was considered statistically significant. Statistical analysis was conducted using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY).

RESULTS

The study was conducted from September 2023 to August 2024 in the Neonatal Intensive Care Unit (NICU) of the Department of Paediatrics at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, affiliated with Jawaharlal Nehru Medical College, Belagavi.

During the study period, a total of 2,928 births were recorded, of which 393 were classified as late preterm. Among these, 328 neonates with a birth weight of less than 2,500 grams required admission to either the NICU or the Kangaroo Mother Care (KMC) unit.

Fifty-two newborns were excluded due to congenital malformations, birth asphyxia, or being nil per oral for more than three days. Additionally, 276 neonates were not enrolled due to parental refusal to consent or formula feeding.

In total, 112 infants were enrolled, and analyzed in the study. However, 35 infants were lost to follo-up after discharge, including 10 lost after reaching 40 weeks corrected gestational age and 27 by three months corrected gestational age.

Ultimately, 75 infants were successfully followed up until three months corrected gestational age, and their data were included in the final analysis.

Figure 8: CONSORT diagram for screening and enrolment of infants.

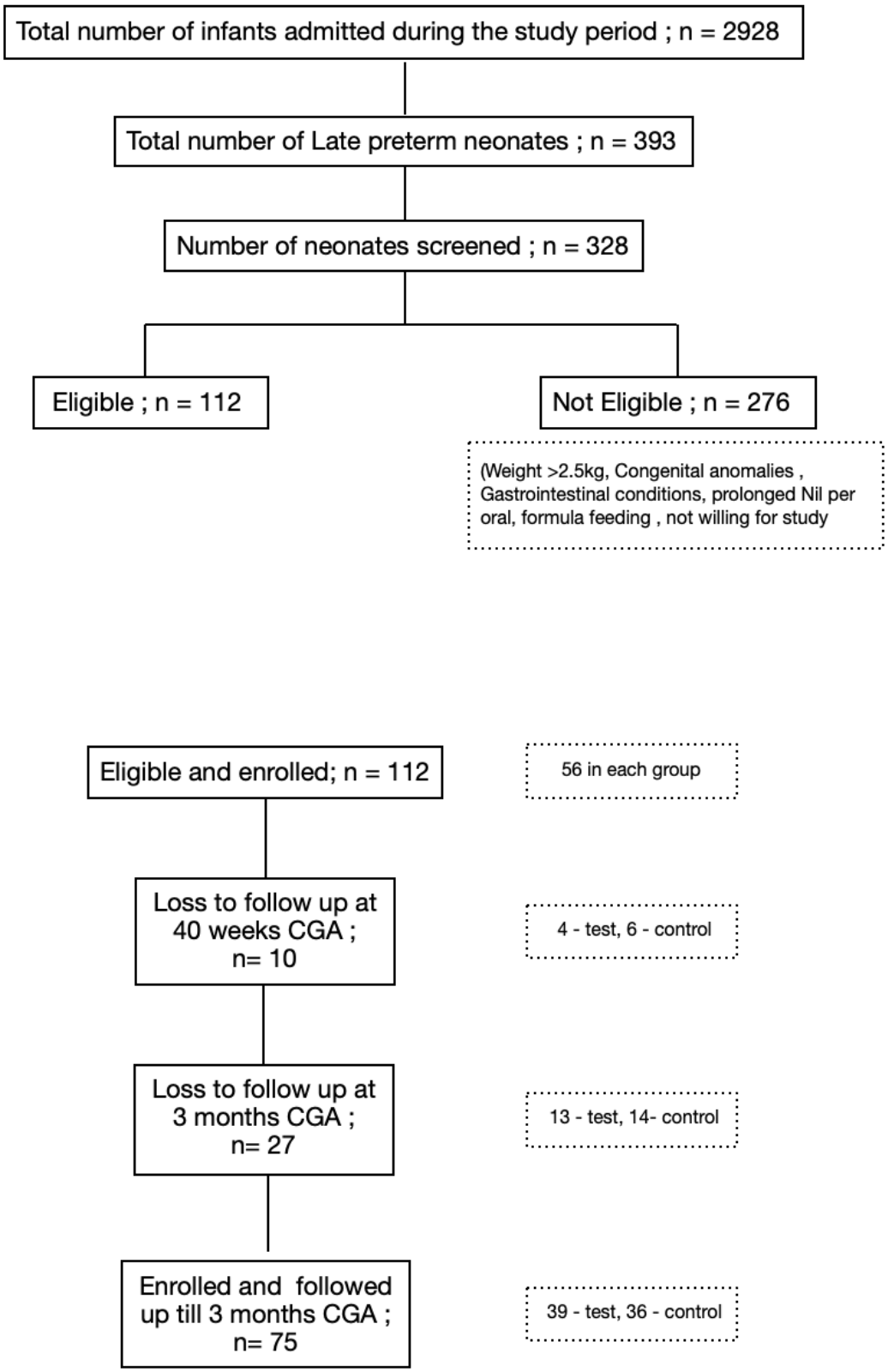


Table 4:Distribution of study groups in our samples (n=75)

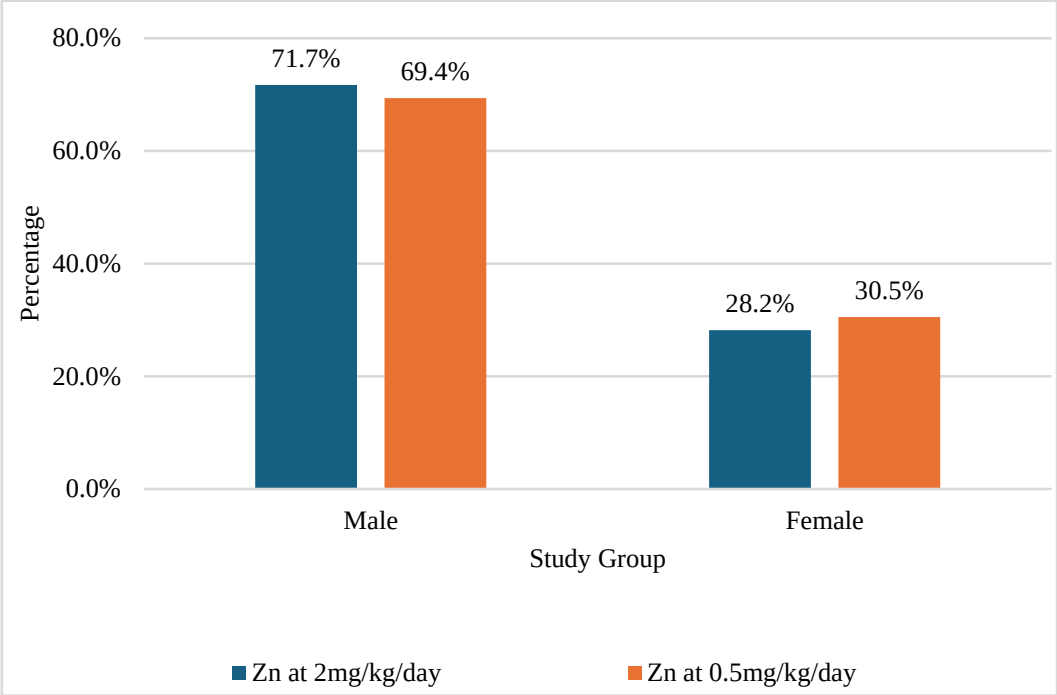
Study Group	Frequencies	Percentage
Zn at 2mg/kg/day	39	52%
Zn at 0.5mg/kg/day	36	48%

Table 5: Distribution of gender in our samples (n=75)

Sex	Frequencies	Percentage
Female	22	29.3%
Male	53	70.7%

The overall study sample consisted of 75 participants, with 53 males (70.7%) and 22 females (29.3%)

Figure 9: Cluster bar chart of comparison of sex between study group (N=75)



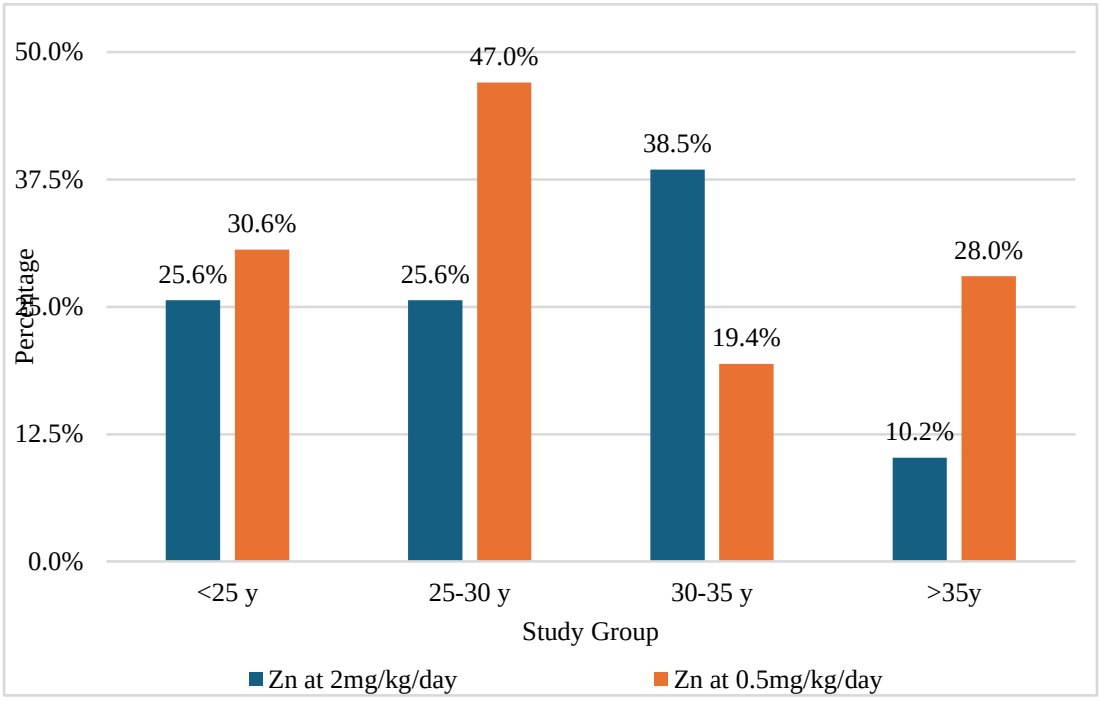
The test group has 71.7% males and 28.2% females, while the control group has 69.4% males and 30.5% females. Both groups show male predominance with no significant difference in gender distribution (χ^2 = 0.024, p = 0.876), indicating effective randomization and minimal risk of gender-related bias

Table 6 : Distribution of the mother's age group (N=70)

Age Group	Frequency	Percentage (%)
<25 years	22	29.33%
25-30 years	27	36.00%
30-35 years	21	28.00%
>35 years	5	6.67%

The 25-30 years age group has the highest frequency (36%), indicating that most mothers fall within this range.The <25 years and 30-35 years groups have similar distributions, around 29% and 28%, respectively. Only 6.67% of mothers are above 35 years, suggesting fewer pregnancies occur in this age range.

Figure 10: Cluster bar chart of comparison of mothers age between study group (N=75)



Maternal age distribution differs between groups, with the control group having more mothers aged 25-30 years (47.0%) and >35 years (28.0%), while the test group has more in the 30-35 years range (38.5%). Younger mothers (<25 years) are similarly represented (25.6% vs. 30.6%). Although not statistically significant (χ^2 = 6.95, p = 0.074), these variations may impact neonatal outcomes. Maternal age should be considered a covariate in further analyses to account for potential confounding effects on nutrition, birth weight, and neurodevelopment.

Table 7: Distribution of the income (N=75)

Group	Mean ±SD (₹)	P value
Zn at 2mg/kg/day	65.95 ±57.64	
Zn at 0.5mg/kg/day	54.53 ±34.08	0.296

The mean family income per month in the test group was ₹65.95 ± 57.64, while in the control group, it was ₹54.53 ± 34.08. A p = 0.296 suggests that socioeconomic status, as measured by family income, was comparable between the two groups and unlikely to have influenced the study outcomes.

Table 8: Distribution with Corrected gestational Age in our samples (N=75)

Corrected Age Range	Frequency	Percentage (%)
34w - 34w+6d	40	53.33%
35w - 35w+6d	21	28.00%
36w - 36w+6d	14	18.67%

Table 9: Comparison of mean of GA corrected age at birth between study group (N=75)

Parameter	Study Group (Mean± SD)		P value
	Test (N=39)	Control (N=36)	
GA Corrected age at Birth	35 ± 1.01	34.87 ± 0.8	0.549

Figure 11: Cluster bar chart of comparison of GA between study group (N=75)

The distribution of gestational age was similar between the test and control groups across all categories.. These findings suggest an even distribution of gestational age across both groups, minimizing the potential for gestational age to act as a confounding variable in the study outcomes.

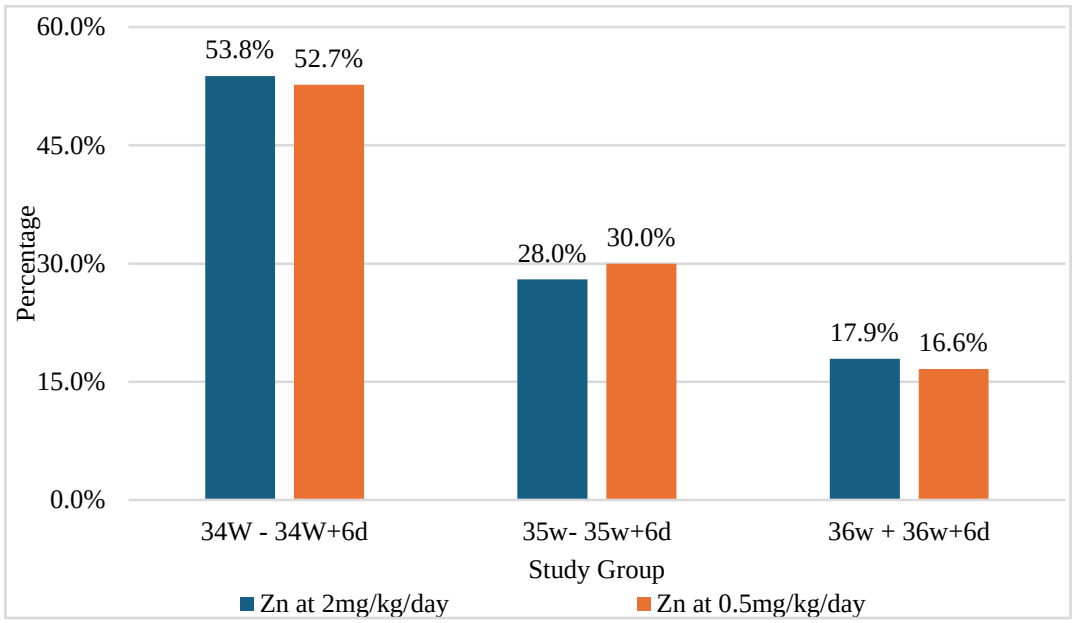


Figure 12 : Pie chart of singleton/multiple in the study population (N=75)

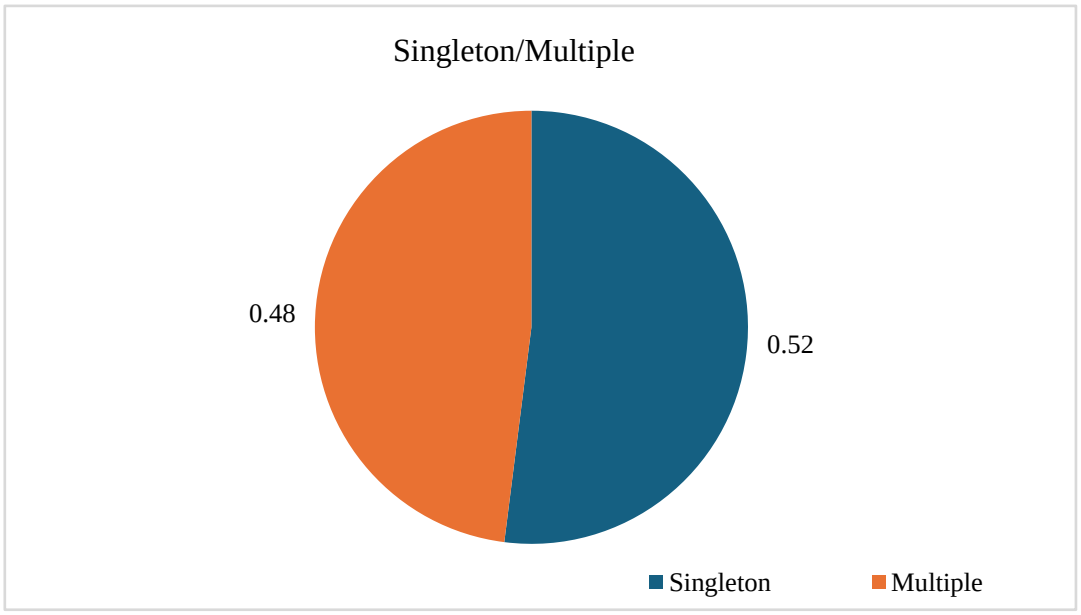


Table 10: Comparison of singleton/multiple between study group (N=75)

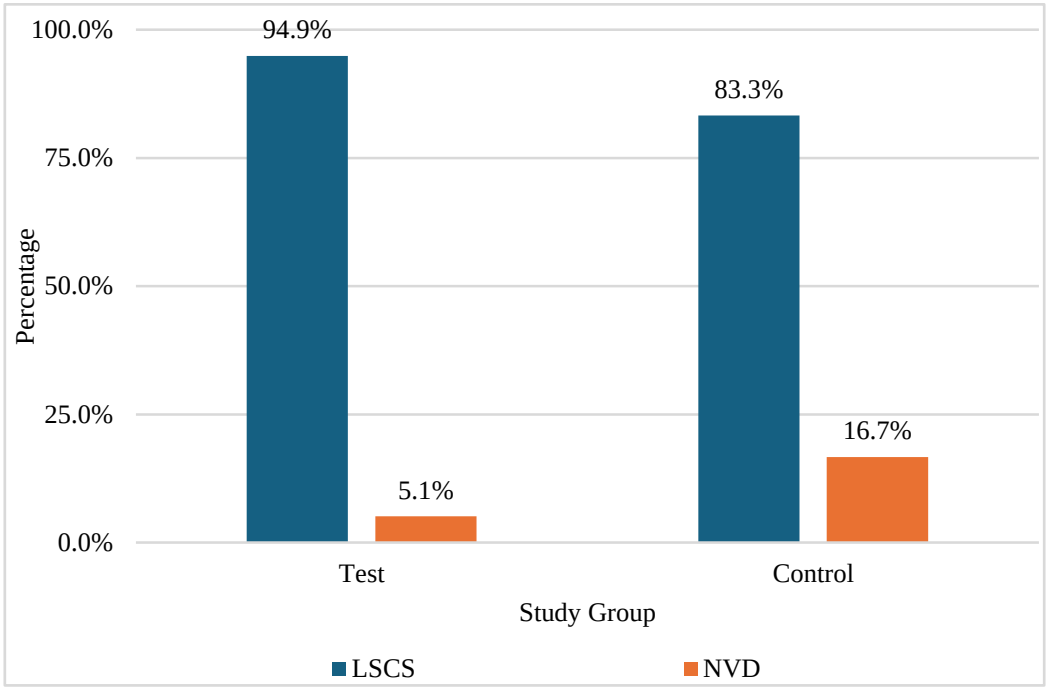
Singleton/Multiple	Study Group		Chi square	P value
	Test (N=39)	Control (N=36)		
Singleton	16 (41.03%)	23 (63.89%)	3.920	0.048
Multiple	23 (58.97%)	13 (36.11%)		

The distribution of singleton and multiple births differed significantly between the test and control groups ($\chi^2 = 3.920$, $p = 0.048$). Singleton births were greater in the control group (63.89%) than in the test group (41.03%). In contrast, multiple births were observed more frequently in the test group (58.97%) compared to the control group (36.11%). The statistically significant p-value (0.048) indicates a potential imbalance in birth type distribution.

Distribution of Mode of delivery in study population (N=75)

The data on the mode of delivery indicates that the majority of neonates (86.7%) were delivered via Lower Segment Cesarean Section, while only 13.3% were delivered through Normal Vaginal Delivery. The high prevalence of LSCS suggests that a significant proportion of cases required surgical intervention, which may be attributed to maternal or fetal complications, multiple pregnancies, or other obstetric indications.

Figure 13: Cluster bar chart of comparison of mode of delivery between study group (N=75)



The distribution of delivery modes in both the test and control groups showed a higher prevalence of lower segment cesarean section (LSCS) deliveries in both groups with 94.9% in group with higher zinc dose compared to 83.3% in the other. The test group had a lower rate of NVD (5.1% vs. 16.7%). However, the chi-square test ($\chi^2 = 1.54$, $p = 0.214$) showed no statistically significant difference between groups.

Figure 14: Pie chart of Distribution of amniotic fluid status in samples (n=75)

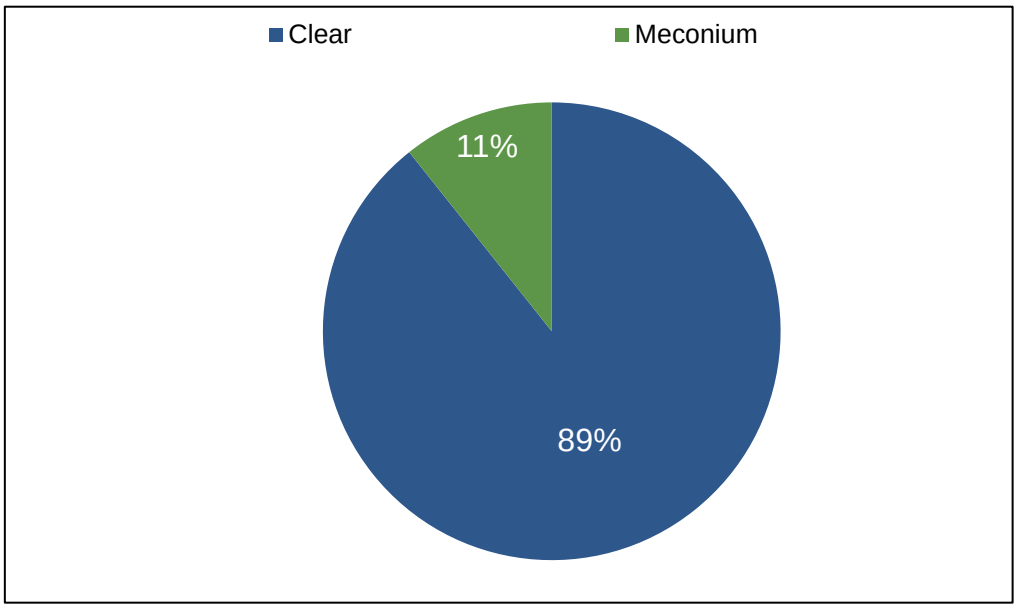


Table 11: Comparison of amniotic liquor status between study group (N=75)

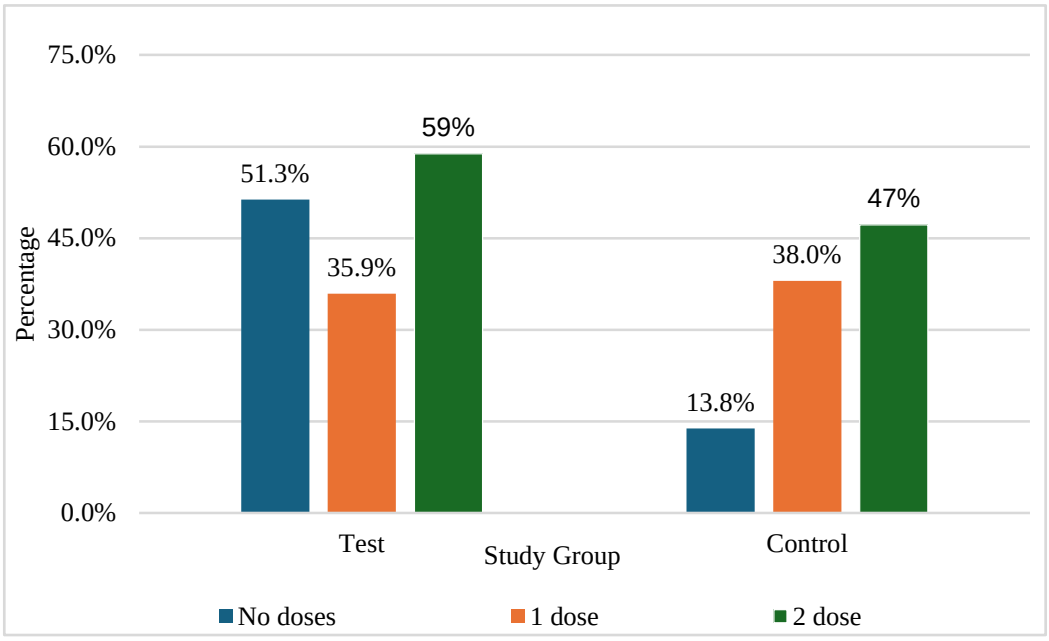
Amniotic Liquor Status	Study Group		Chi square	P value
	Test (N=39)	Control (N=36)		
Clear	37 (94.9%)	30 (83.3%)	1.54	0.214
Meconium	2(5.1%)	6(16.7%)		

In the test group, 94.9% of cases had clear amniotic fluid, while 5.1% had meconium-stained fluid. In comparison, the control group had 83.3% clear amniotic fluid and 16.7% with meconium staining. Although the test group had a lower subjects of meconium-stained fluid in relation to the control group, statistical analysis ($\chi^2 = 1.54$, $p = 0.214$) indicates that this difference is not statistically significant.

DISTRIBUTION OF ANTENATAL STEROIDS

The distribution of antenatal steroid doses among participants shows that the majority (53.33%) received the full two-dose regimen, which is the recommended course for fetal lung maturation. A significant proportion (37.33%) received only one dose, while a smaller subset (9.33%) did not receive any antenatal steroids.

Figure 15: Cluster bar chart of comparison of antenatal steroids between study group (N=75)

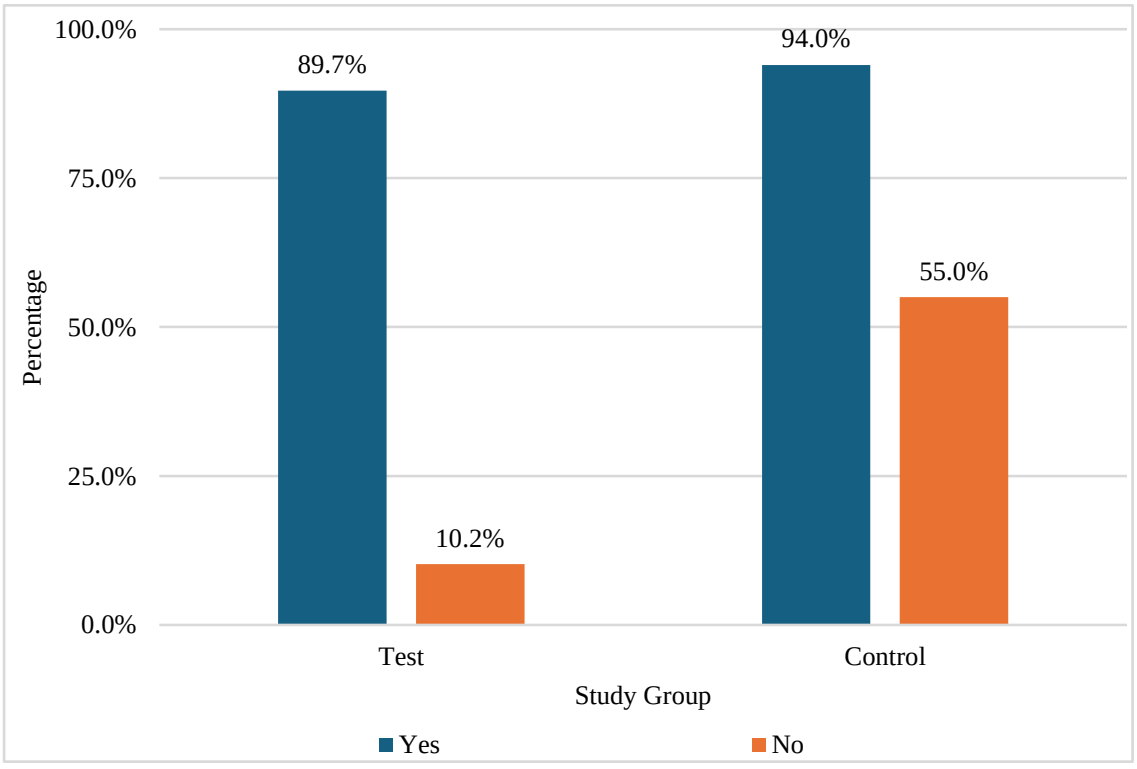


The distribution of antenatal steroid doses between groups shows notable differences. In the test group, 51.3% of participants did not receive any antenatal steroid doses, whereas only 13.8% in the control group were in this category. A single dose was administered to 35.9% of the higher dose recipients and 38.0% of the lower dose , showing a relatively similar proportion. However, a higher of participants in the control group (47.2%) received the full two-dose regimen compared to 58.8% in the test group. Chi-square analysis (χ^2 =15.45, p = 0.0004) was conducted to determine the statistical significance of these differences.

Table 12: Comparison on neonates who cried at birth in our sample (N= 75)

Cried at Birth	Frequency (n)	Percentage (%)
Yes	71	94.67%
No	4	5.33%

Figure 16: Cluster bar chart of comparison of cried at birth between study group (N=75)



The analysis of neonatal crying at birth showed no ssignificamce between the test and control groups ($\chi^2 = 0.105$, $p = 0.746$). Most neonates in both groups cried at birth, while a smaller percentage did not cry (5.56% and 10.26%, respectively). However, due to the low number of neonates who did not cry at birth, this result should be interpreted cautiously and not regarded as definitive.

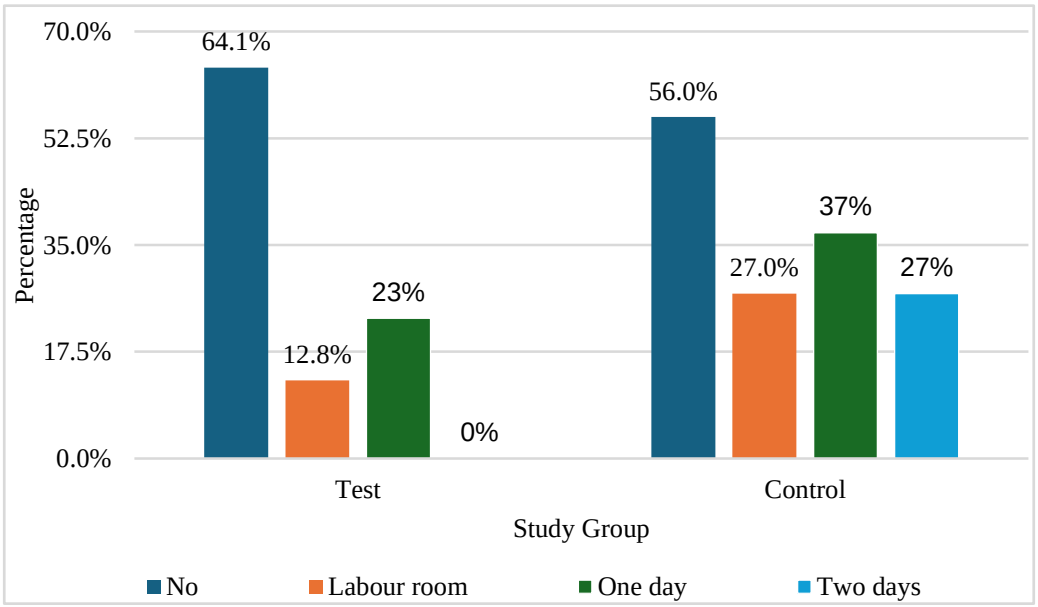
BAG AND MASK VENTILATION

100 % of babies who didn't cry at birth (that is 4 babies) required Bag and Mask Ventilation each randomisation group had 2 babies.

Table 13: Comparison on neonates who required CPAP in our sample (N= 75)

CPAP Usage	Frequency (n)	Percentage (%)
No	45	60.00%
Labour Room	8	10.67%
One day	21	28.00%
Two days	1	1.33%

Figure 17: Cluster bar chart of comparison of CPAP between study group (N=75)



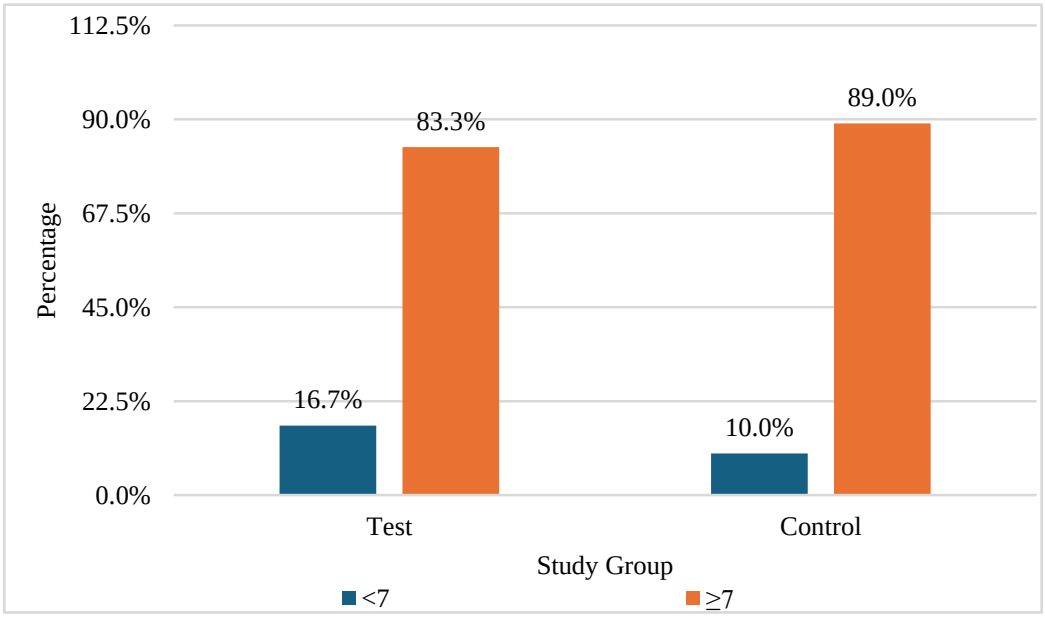
The requirement for CPAP differed significantly between the test and control groups ($\chi^2 = 21.27$, $p < 0.001$). A higher number of neonates in the test group did not require CPAP support (64.1%) compared to the control group (56.0%). Additionally,

none of the neonates in the test group required CPAP for two days, whereas 27% of the low dose recepients needed it for this duration. The low dose recipients requiring CPAP in the labour room (27% vs. 12.8%) and for one day (37% vs. 23%).

Table 14: Comparison on APGAR at 1 minute in neonates in our sample (N= 75)

Category	Frequency	Percentage (%)
<7	10	13.33%
≥7	65	86.67%

Figure 18 : Cluster bar chart of comparison of APGAR at 1 minute in neonates between study group (N=75)



The Apgar scores at birth were comparable between thel groups with chi-square test result ($\chi^2 = 0.144$, $p = 0.704$)

Comparison on APGAR at 5 minute in neonates in our sample (N= 75)

All babies had APGAR ≥7in both test and control group

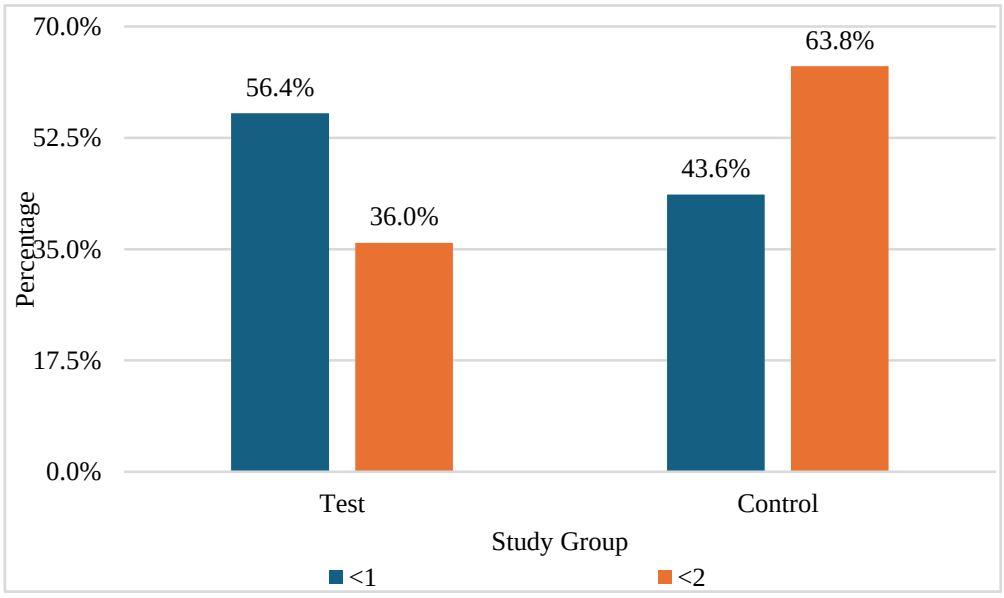
Mechanical Consequence

No babies in any group had any postural deformity or mechanical consequence of birth such as facial paralysis , brachial plexus paralysis, fracture of clavicle etc

Table 15: Comparison on initiation of breastfeeding in neonates in our sample (N= 75)

Category	Frequency	Percentage (%)
<1 day	35	46.67%
<2 days	40	53.33%

Figure 19: Cluster bar chart of comparison of Breast milk between study group (N=75)

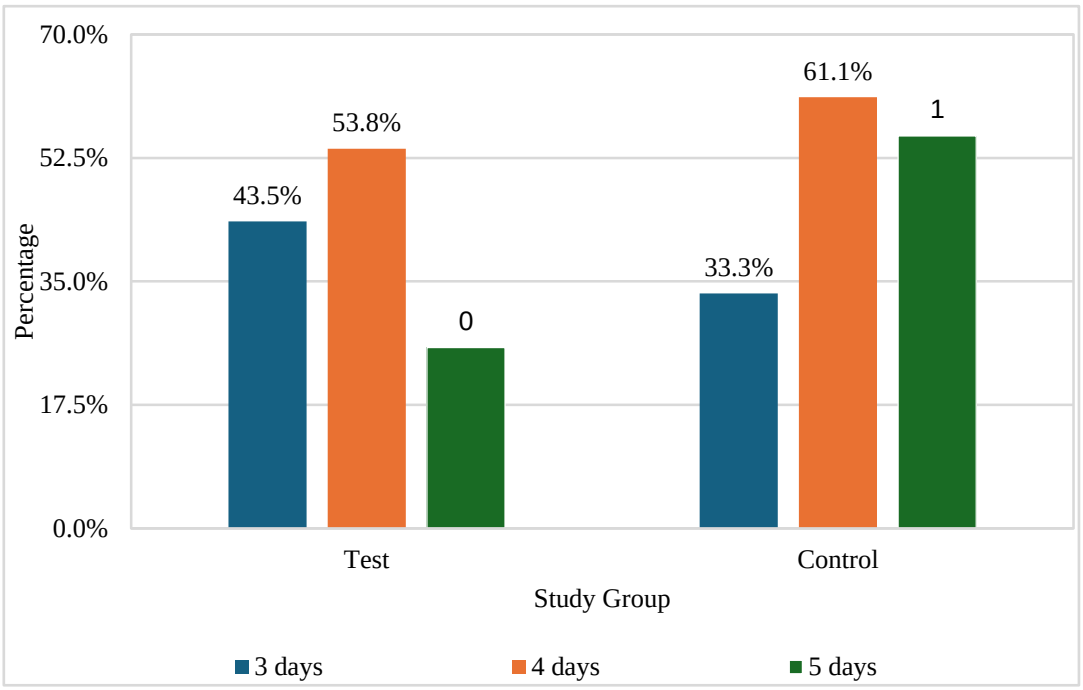


In the test group, 56.4% of neonates initiated breastfeeding within one day, whereas 36.0% started after one day. In the control group, only 43.6% initiated breastfeeding within one day, while 63.8% started after one day. T

Table 16: Comparison on initiation of enteral supplementation in neonates in our sample (N= 75)

Day	Frequency	Percentage (%)
3	29	38.67%
4	43	57.33%
5	3	4.00%

Figure 20 : Cluster bar chart of comparison of initiation on Enteral Supplementation between study group (N=75)

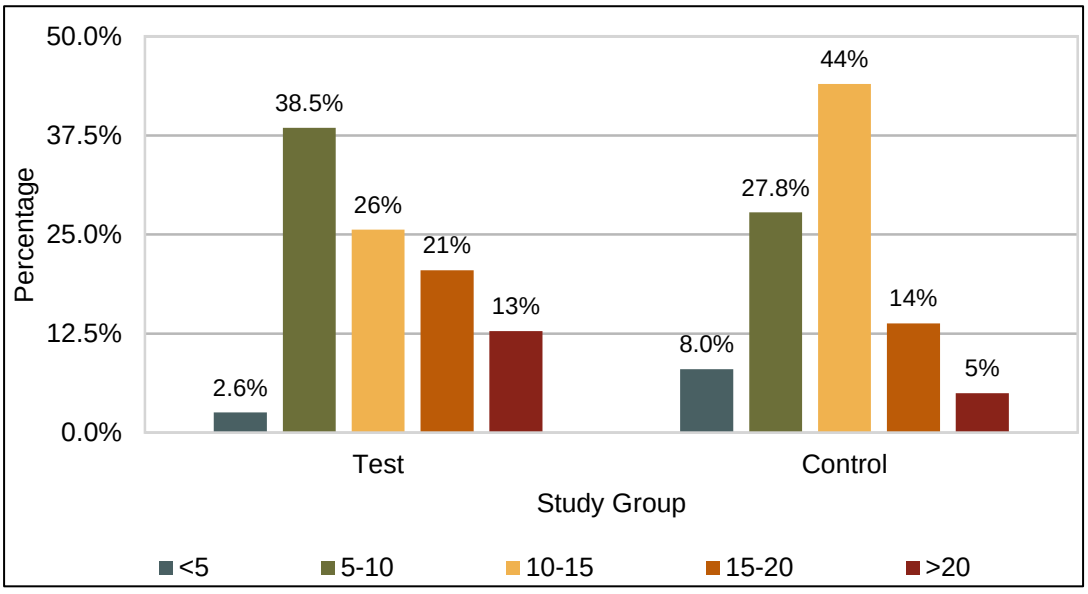


In our study, enteral supplementation was initiated earlier in the test group, with 43.5% starting by day 3 compared to 33.3% in the control group. By day 4, 53.8% of the test group had begun enteral feeds versus 61.1% in the control group, while initiation was delayed to day 5 in only 2.56% of group 1 compared to 5.56% in the group 2.

Table 17: Comparison on days in hospital till discharge in neonates in our sample (N= 75)

Range	Frequency	Percentage (%)
<5	4	5.33%
5-10	25	33.33%
10-15	26	34.67%
15-20	13	17.33%
>20	7	9.33%

Figure 21: Cluster bar chart of comparison of days in hospital till discharge in neonates between study group (N=75)

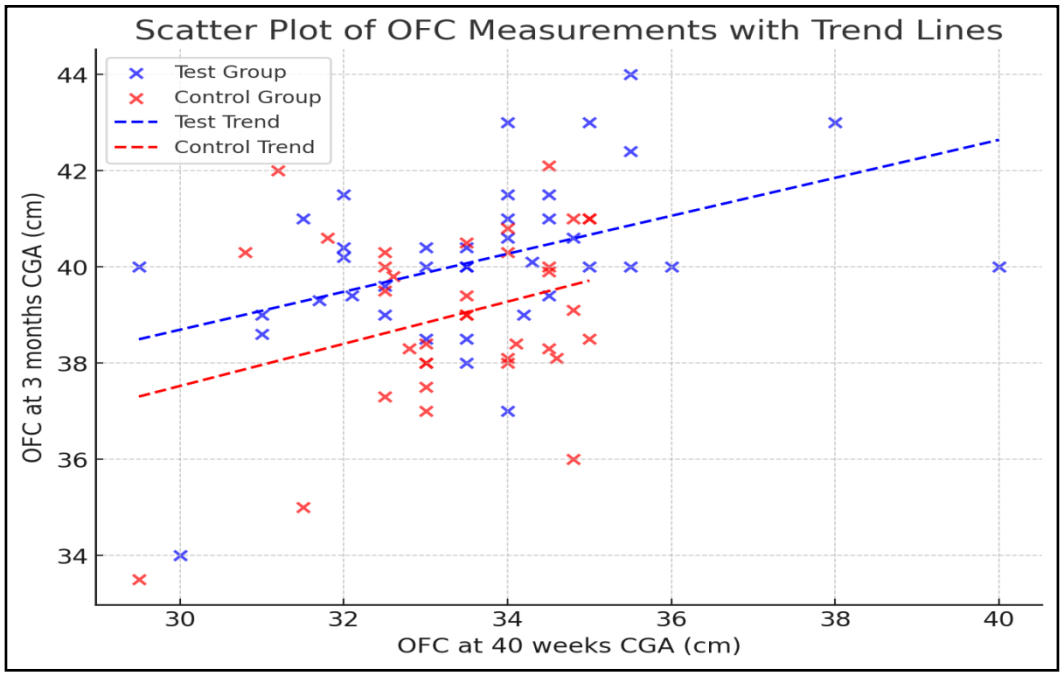


Hospital stay durations were similar between groups. In the test group, 38.46% stayed 5-10 days, and 25.64% stayed 10-15 days. In the control group, 27.78% stayed 5-10 days, while 44.44% stayed 10-15 days. Shorter stays (<5 days) were 2.56% (test) vs. 8.33% (control), while longer stays (>20 days) were 12.82% (test) vs. 5.56% (control). A chi-square test ($\chi^2 = 5.25$, $p = 0.262$) showed no significant difference.

Table 18: Comparison of mean of OFC between study group(N=75)

OFC (cm)	Study Group (Mean± SD)		P value
	Test (N=39)	Control (N=36)	
At Birth	30.7 ± 1.65	30.69 ± 1.24	0.986
At Discharge	31.46 ± 1.57	31.61 ± 1.91	0.722
At 40 weeks CGA	33.63 ± 1.99	33.41 ± 1.3	0.577
At 3 months CGA	40.13 ± 1.77	39 ± 1.84	0.009

Figure 22: Scattter Plot of OFC measurements between study group (N=75)



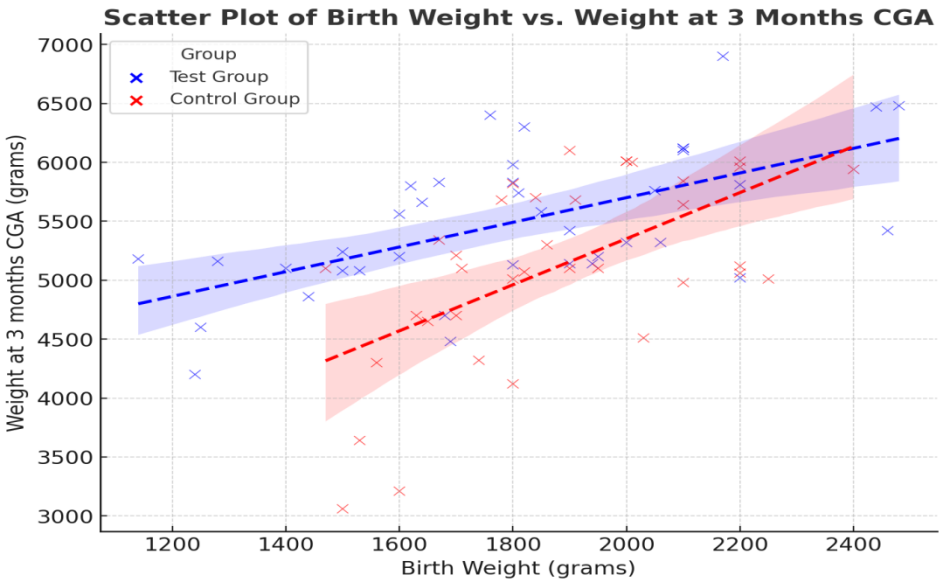
The occipitofrontal circumference (OFC) measurements of preterm neonates in the test and control groups were similar at birth indicating no significant difference. This similarity persisted at discharge (31.46 ± 1.57 cm vs. 31.61 ± 1.91 cm, p = 0.722) and at 40 weeks CGA (33.63 ± 1.99 cm vs. 33.41 ± 1.3 cm, p = 0.577), suggesting that early postnatal head growth was comparable between the groups. However, by 3 months CGA, the test group, which received a higher zinc dose (2 mg/kg/day), exhibited a significantly larger OFC (40.13 ± 1.77 cm) compared to the control group (39 ± 1.84 cm), with a p-value of 0.009.

In the scatter plot analysis the test group (blue) shows a steeper trend, suggesting that the group that received higher dose had a relatively higher OFC growth over time .This statistically significant difference suggests that increased supplementation may have contributed to improved postnatal brain growth, as OFC is a key marker of neurodevelopment in preterm infants.

Table 19: Comparison of mean of Weight between study group(N=75)

Weight (g)	Study Group (Mean± SD)		P value
	Test (N=39)	Control (N=36)	
At Birth	1806.15 ± 337.53	1877.22 ± 237.21	0.299
At Discharge	1806.28 ± 287.6	1855.14 ± 258.22	0.443
At 40 weeks CGA	2390.09 ± 761.1	2598.33 ± 545.85	0.181
At 3 months CGA	5498.21 ± 596.21	5113.92 ± 778.48	0.018

Figure 23 : Scattter Plot of weight measurements between study group (N=75)



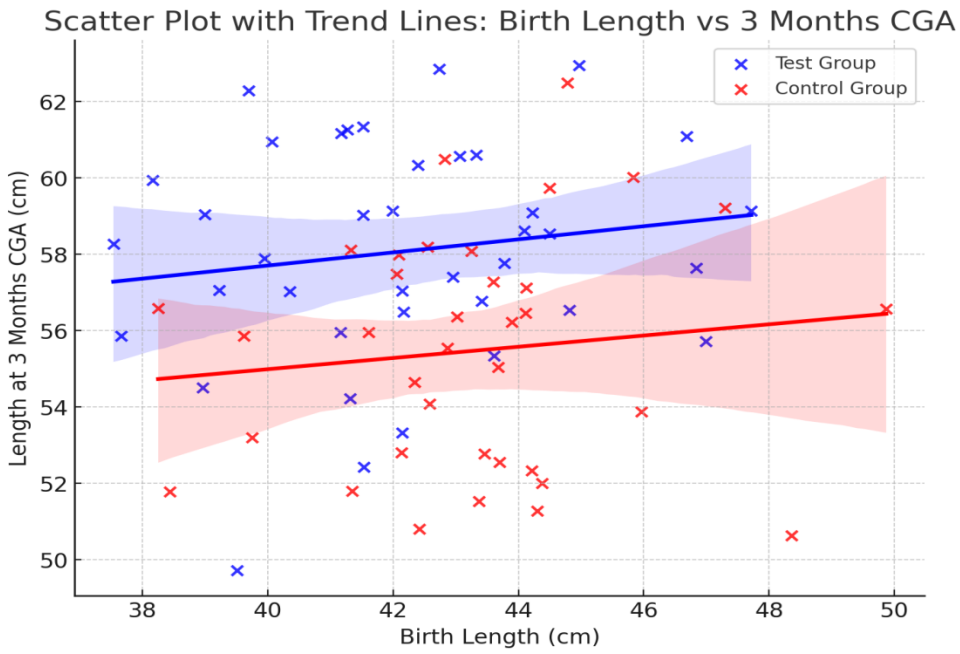
The longitudinal weight analysis of preterm neonates in the test and control groups showed no significant differences at birth (1806.15 ± 337.53 g vs. 1877.22 ± 237.21 g, p = 0.299), discharge (1806.28 ± 287.6 g vs. 1855.14 ± 258.22 g, p = 0.443), or 40 weeks corrected gestational age (2390.09 ± 761.1 g vs. 2598.33 ± 545.85 g, p = 0.181). However, by 3 months CGA, the test group exhibited a significantly higher mean weight (5498.21 ± 596.21 g) compared to the control group

(5113.92 ± 778.48 g, p = 0.018). Scatter plot analysis showed a steeper weight gain trajectory in the group receiving higher dose suggesting a potential role of supplementation in enhancing postnatal growth.

Table 20: Comparison of Length between study group(N=75)

Length (cm)	Study Group (Mean± SD)		P value
	Test (N=39)	Control (N=36)	
At Birth	42.77 ± 2.67	43.44 ± 2.61	0.273
At Discharge	43.73 ± 2.55	44.63 ± 2.42	0.121
At 40 weeks CGA	46.53 ± 3.7	46.94 ± 3.1	0.601
At 3 months CGA	58 ± 3.16	55.65 ± 3.12	0.002

Figure 24 : Scattter Plot of length measurements between study group (N=75)



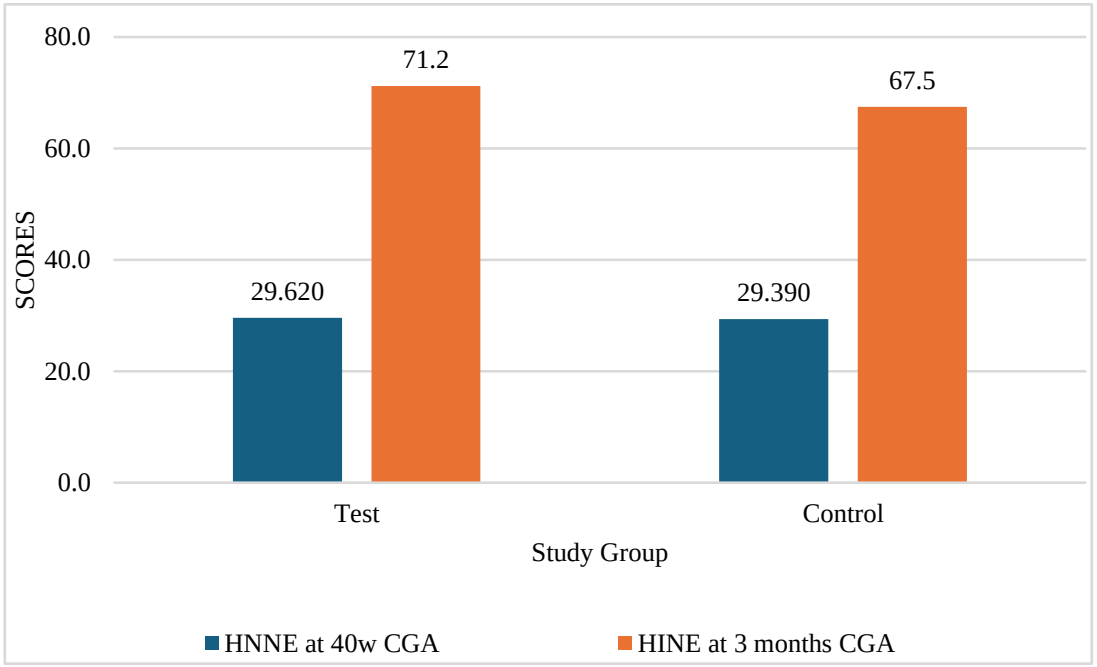
The longitudinal analysis of length measurements in the test and control groups revealed no significant differences at birth (42.77 ± 2.67 cm vs. 43.44 ± 2.61 cm, p = 0.273), at discharge (43.73 ± 2.55 cm vs. 44.63 ± 2.42 cm, p = 0.121), or at 40 weeks corrected gestational age (46.53 ± 3.7 cm vs. 46.94 ± 3.1 cm, p = 0.601).

However, by 3 months corrected gestational age, the test group exhibited a significantly greater length (58.00 ± 3.16 cm) compared to the control group (55.65 ± 3.12 cm, p = 0.002). Scatter plot analysis demonstrated a steeper growth trajectory in the test group, suggesting a positive impact of supplementation of zinc on postnatal linear growth.

Table 21: Comparison of mean of HNNE and HINE between study group (N=75)

Parameter	Study Group (Mean± SD)		P value
	Test (N=39)	Control (N=36)	
HNNE At 40 weeks CGA	29.62 ± 2.23	29.39 ± 2.22	0.661
HINE At 3 months CGA	71.23 ± 1.4	67.5 ± 1.76	<0.001

Figure 25 : Cluster bar chart of comparison of neurological scores in neonates between study group (N=75)

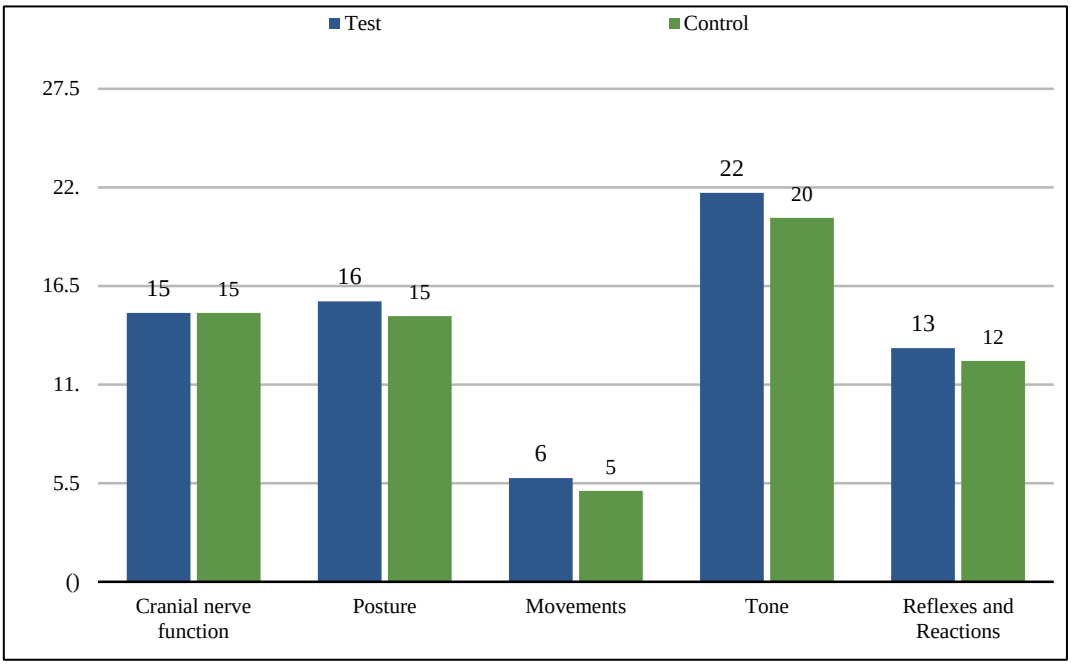


The neurological assessment scores of groups were evaluated at two different time points using the HNNE at 40 weeks corrected gestational age and theHINE at 3 months CGA. At 40 weeks CGA, the HNNE scores were comparable between the test group (29.62 ± 2.23) and the control group (29.39 ± 2.22), with a p-value of 0.661, indicating no statistically significant difference. However, at 3 months CGA, the test group demonstrated significantly higher HINE scores (71.23 ± 1.4) compared to the control group (67.5 ± 1.76), with a p-value of <0.001, suggesting a meaningful improvement in neurological outcomes for the test group. This data implies that while both groups had similar neurological performance in the early neonatal period, the test group exhibited significantly better neurological development by 3 months CGA.

Table 22 : Correlation of HINE parameters at 3 months CGA (N=75)

HINE Component	Zn @ 2mg/kg/day (Mean ±SD)	Zn @0.5mg/kg/day (Mean ±SD)	t-statistic	p-value
HINE at 3 months CGA	71.23 ±1.4	67.5 ±1.76	8.72	<0.001
Cranial Nerve Function	15.00 ±0.00	14.89 ±0.31	1.80	0.083
Posture	15.63 ±0.66	14.82 ±1.02	3.57	<0.001
Movements	5.78 ±0.42	5.07 ±0.26	7.95	<0.001
Tone	21.69 ±1.15	20.29 ±0.94	5.20	<0.001
Reflexes & Reactions	13.03 ±1.09	12.32 ±0.90	2.75	0.008

Figure 26: Cluster bar chart of comparison of HINE parameters at 3 months CGA (N=75)



Our findings indicate that the test group exhibited significantly better neurodevelopmental outcomes at 3 months CGA ompared to the control group. Higher HINE scores, improved posture, tone, reflexes, and reactions in the test group suggest a positive impact of the intervention. The lack of significant difference in cranial nerve function (p = 0.083) implies that this specific domain remained unaffected.

Table 23: Comparison of mean of Serum zinc and Serum copper between study group (N=75)

Parameter	Study Group (Mean± SD)		P value
	Test (N=39)	Control (N=36)	
Serum Zinc at birth	64.24 ± 8.56	68.32 ± 7.35	0.031
Serum Zinc at 3 months CGA	117.9 ± 12	90.4 ± 8.02	<0.001
Serum copper at 3 months CGA	113.43 ± 21.27	135.77 ± 12.07	<0.001

Figure 27 : Cluster bar chart of comparison of serum zinc levels at birth vs 3 months CGA between study group (N=75)

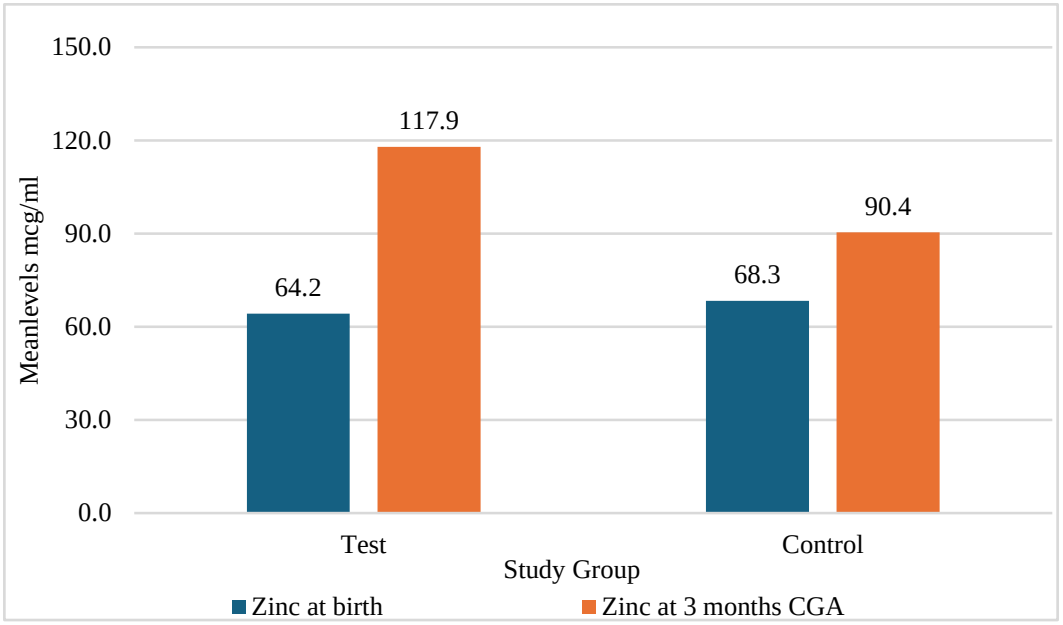
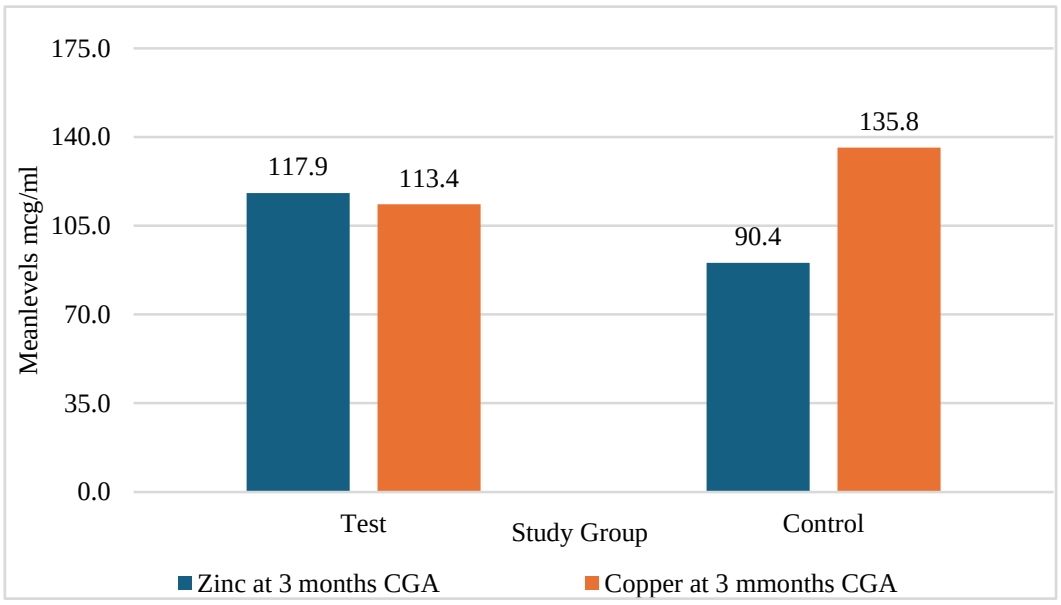


Figure 28: Cluster bar chart of comparison of serum zinc levels at 3 months CGA vs serum copper levels at 3 months CGA between study group (N=75)



Serum zinc and copper levels differed significantly between groups. At birth, zinc levels were lower in the test group (64.24 ± 8.56 µg/dL) than in controls (68.32 ±

7.35 µg/dL, p = 0.031), but by 3 months CGA, they were higher (117.9 ± 12 vs. 90.4 ± 8.02 µg/dL, p < 0.001), likely due to supplementation. Conversely, copper levels at 3 months CGA were lower in the test group (113.43 ± 21.27 µg/dL) than in controls (135.77 ± 12.07 µg/dL, p < 0.001), possibly reflecting differences in metabolism or regulation.

Table 24: Correlation of Serum Zinc at Birth between and anthropometry parameter in the study population (N=75)

Pairs (At Birth CGA)	Pearson Correlation	P-value
Overall (N=75)		
Serum Zinc vs OFC	0.185	0.113
Serum Zinc vs Weight	0.281	**0.015**
Serum Zinc vs Height	0.282	**0.014**
Test (N=39)		
Serum Zinc vs OFC	0.144	0.182
Serum Zinc vs Weight	0.230	0.159
Serum Zinc vs Height	0.541	0.077
Control (N=36)		
Serum Zinc vs OFC	0.292	0.141
Serum Zinc vs Weight	0.245	0.157
Serum Zinc vs Height	0.529	0.069

The evaluation of serum zinc levels at birth in 75 infants revealed a meaningful connection with birth weight (r=0.281, p=0.015) and birth length (r=0.282, p=0.014), while no significant link was found with head circumference

(r=0.185, p=0.113). When analyzing the test and control groups separately, there was a moderate correlation between zinc levels and birth length, but it did not reach statistical significance (test group: r=0.541, p=0.077; control group: r=0.529, p=0.069). The associations between zinc levels and both birth weight and head circumference were weaker and not statistically significant in either group.

Table 25: Correlation of Serum Zinc at 3 months CGA between and anthropometry parameter in the study population (N=75)

Pairs (3 months CGA)	Pearson Correlation	P-value
Overall (N=75)		
Serum Zinc vs OFC	0.230	**0.026**
Serum Zinc vs Weight	0.262	**0.024**
Serum Zinc vs Height	0.373	**0.001**
Test (N=39)		
Serum Zinc vs OFC	0.130	**0.039**
Serum Zinc vs Weight	0.091	0.082
Serum Zinc vs Height	0.195	0.159
Control (N=36)		
Serum Zinc vs OFC	0.037	0.833
Serum Zinc vs Weight	0.079	0.652
Serum Zinc vs Height	0.153	0.381

At 3 months CGA , serum zinc levels showed significant positive correlations with OFC (r=0.230, p=0.026), weight (r=0.262, p=0.024), and height (r=0.373,

p=0.001) in the overall group, indicating a potential role in postnatal growth. In the test group, zinc correlated weakly but significantly with OFC (r=0.130, p=0.039), while associations with weight and height were not significant. No significant correlations were seen in group receiving lower dose. These findings suggest zinc may contribute to growth, particularly in height, though other factors likely influence outcomes.

Table 26: Correlation of Serum Zinc at 3 months CGA between and Copper , HNNE and HINE in the study population (N=75)

Pairs (3 months CGA)	Pearson Correlation	P-value
Overall (N=75)		
Serum Zinc vs Serum Copper	-0.584	**<0.001**
Serum Zinc vs HNNE at 40 Weeks	0.155	0.188
Serum Zinc vs HINE	0.684	**<0.001**
Test (N=39)		
Serum Zinc vs Serum Copper	-0.369	0.021
Serum Zinc vs HNNE at 40 Weeks	0.265	0.103
Serum Zinc vs HINE	0.283	**0.040**
Control (N=36)		
Serum Zinc vs Serum Copper	-0.115	0.512
Serum Zinc vs HNNE at 40 Weeks	0.064	0.714
Serum Zinc vs HINE	0.066	0.708

Serum zinc and copper showed a strong inverse correlation in the overall group ($r = -0.584$, $p < 0.001$), indicating a competitive interaction. This negative relationship was weaker but significant in the test group ($r = -0.369$, $p = 0.021$) and nonsignificant in controls, suggesting an effect of zinc supplementation.

Serum levels of zinccorrelated strongly with the HINE score at 3 months CGA ($r = 0.684$, $p < 0.001$), supporting its role in neurodevelopment. This association was moderate in group receiving higher dose ($r = 0.283$, $p = 0.040$) but nonsignificant in controls, further pointing to the impact of supplementation. However, no significant correlation was found between zinc and HNNE scores at 40 weeks CGA, suggesting its effects on neurodevelopment may become more apparent over time.

CORRELATION AMONGST CONFOUNDING FACTORS

Table 27: Comparison of mean of HNNE at 40 weeks and HINE 3 month CGA between gender (N=75)

Parameter	Gender (Mean± SD)		P value
	Male (N=53)	Female (N=22)	
HNNE at 40 weeks CGA	29.45 ± 2.36	29.64 ± 1.87	0.746
HINE at 3 months CGA	69.21 ± 2.45	70 ± 2.41	0.204

The comparison of neurological assessment scores between male and female neonates showed no statistically significant differences. At 40 weeks corrected gestational age, the mean HNNE score was 29.45 ±2.36 for males and 29.64 ±1.87 for females (p = 0.746). Similarly, at 3 months corrected gestational age, the mean HINE score was 69.21 ±2.45 for males and 70 ±2.41 for females (p = 0.204). While females had slightly higher mean scores. Both are not significant hence indicating that gender did not substantially influence neurological outcomes in this study population.

Table 28 : Comparison of mean of HNNE at 40 weeks and HINE 3 month CGA between Liquor (N=75)

Parameter	Liquor (Mean± SD)		P value
	Clear (N=68)	Meconium (N=7)	
HNNE at 40 weeks CGA	29.62 ± 2.21	28.43 ± 2.15	0.178
HINE at 3 months CGA	69.68 ± 2.35	67.14 ± 2.34	0.008

Neurological assessment scores varied by amniotic fluid type. At 40 weeks CGA, HNNE scores were slightly lower in the meconium-stained liquor group (28.43 ± 2.15) than in the clear liquor group (29.62 ± 2.21), While the difference was not statistically significant (p = 0.178), HINE scores were considerably lower in the meconium group by 3 months CGA (67.14 ± 2.34) than in the clear liquor group (69.68 ± 2.35) (p = 0.008), suggesting that meconium exposure may negatively impact early neurological outcomes.

Table 29 : Comparison of mean of HNNE at 40 weeks and HINE 3 month CGA between Cried at birth (N=75)

Parameter	Cried at birth (Mean± SD)		P value
	Yes (N=71)	No (N=4)	
HNNE at 40 weeks CGA	29.61 ± 2.21	27.75 ± 1.5	0.103
HINE at 3 months CGA	69.49 ± 2.36	68.5 ± 4.12	0.434

Neurological assessment scores showed no significant difference between infants who cried at birth and those who did not. At 40 weeks CGA , the HNNE score was lower in non-crying infants (27.75 ± 1.5) compared to those who cried (29.61 ± 2.21), though this difference was not significant (p = 0.103). Likewise, at 3 months CGA, HINE scores were slightly lower in non-crying infants (68.5 ± 4.12) than in those who cried (69.49 ± 2.36), with no significant difference (p = 0.434). However, given the very small number of neonates who did not cry at birth, these results should be carefully analysed.

Table 30: Comparison of mean of HNNE at 40 weeks and HINE 3 month CGA between CPAP (N=75)

Parameter	CPAP (Mean± SD)		P value
	Yes (N=29)	No (N=46)	
HNNE at 40 weeks CGA	29.34 ± 1.56	29.61 ± 2.55	0.619
HINE at 3 months CGA	69 ± 2.42	69.72 ± 2.46	0.219

The neurological assessment scores did not differ significantly between infants who required CPAP support and those who did not. At 40 weeks CGA, the mean HNNE score 29.34 ± 1.56 in the CPAP group and 29.61 ± 2.55 in the non-CPAP group ($p = 0.619$). Similarly, at 3 months CGA, the mean HINE score was 69 ± 2.42 in the CPAP group and 69.72 ± 2.46 in the non-CPAP group ($p = 0.219$). Although the CPAP group had slightly lower scores, these differences were not statistically significant, suggesting that CPAP use does not substantially impact early neurological outcomes.

Table 31: Correlation of HNNE at 40 weeks CGA between and APGAR parameter in the study population (N=75)

Pairs	Spearman rank Correlation	P-value
HNNE at 40 weeks CGA vs APGAR 1	0.093	0.428
HINE at 3 months CGA vs APGAR 1	0.187	0.145
HNNE at 40 weeks CGA vs APGAR 5	0.128	0.278
HINE at 3 months CGA vs APGAR 5	0.109	0.218

In our study, all had an APGAR score above 5 at 1 minute and greater than 7 at 5 minutes. Spearman rank correlation analysis showed weak and non-significant associations between neurological assessment scores and APGAR scores at both time points. The correlation between HNNE at 40 weeks CGA and APGAR at 1 minute was 0.093 ($p = 0.428$), while its correlation with APGAR at 5 minutes was 0.128 ($p = 0.278$). Similarly, the correlation between HINE at 3 months CGA and APGAR at 1 minute was 0.187 ($p = 0.145$), and with APGAR at 5 minutes was 0.109 ($p = 0.218$). These results suggest that early APGAR scores may have limited predictive value for neurological outcomes at 40 weeks and 3 months CGA in this study population.

Table 32: Correlation of HNNE at 40 weeks CGA between and anthropometry parameter in the study population (N=75)

Pairs (40 weeks CGA)	Pearson Correlation	P-value
HNNE at vs OFC	-0.120	0.307
HNNE at Weight	0.004	0.971
HNNE at Height	-0.083	0.479

The table displays the Pearson correlation between Hammersmith Neonatal Neurological Examination scores at 40 weeks corrected gestational age and three anthropometric measurements: occipitofrontal circumference, weight, and height. The correlation between HNNE and OFC is -0.120 (p = 0.307), reflecting a weak negative association that is not statistically significant. Similarly, the correlation between HNNE and weight is 0.004 (p = 0.971), indicating virtually no relationship, and the correlation between HNNE and height is -0.083 (p = 0.479), again a weak negative correlation lacking statistical significance. These findings suggest that HNNE scores at 40 weeks CGA are not meaningfully associated with OFC, weight, or height.

ASSOCIATION OF ZINC WITH COMORBIDITIES

None of the neonates from either group required hospital readmission or physician consultation for diarrhea, respiratory illness, skin lesions, failure to thrive, retinopathy of prematurity , or necrotizing enterocolitis post-discharge. This emphasizes the absence of any adverse outcomes in out study population.

DISCUSSION

Multiple studies have investigated the impact of enteral zinc supplementation on the growth and neurodevelopment of preterm infants, considering differences in gestational age, birth weight, dosage, duration, and assessed outcomes. Birth weights ranged from <1500g to 2500g, and gestational ages spanned 24–37 weeks. Zinc supplementation was initiated at different time points, from the first day of life (Ragab, 2014) to several weeks postnatally (Friel, 1993; Aminisani, 2011), with dosages between 2 mg/kg/day to 10 mg/day, administered via solutions, fortified formulas, or multivitamins. Duration varied from six weeks to six months, with most studies maintaining supplementation for at least two to three months.[118-120]

Growth parameters which were primary Studies such as (Diaz-Gomez, 2003; Islam, 2010; Ragab, 2014) was associated with enhanced weight gain and increased head circumference growth.Neurodevelopmental impact was assessed using standardized scales like the Griffiths Developmental Scale and Amiel-Tison Score (Mathur, 2015; Terrin, 2013), with mixed results. Some studies also assessed neonatal morbidities (Ragab, 2014; Kumar, 2011), but zinc's protective effects were inconsistent.[118-123]

This RCT evaluates Zinc supplementation in 75 preterm neonates at Dr. Prabhakar Kore Hospital, Belagavi. Infants were randomized into two groups receiving either 2 mg/kg/day or 0.5 mg/kg/day of zinc until three months corrected gestational age. Neurodevelopmental outcomes were assessed using the HNNE at 40 weeks CGA and the HINE at 3 months CGA.

A total of 75 infants completed the study, with 39 participants (52%) receiving a higher zinc dose of 2 mg/kg/day (Group 1) and 36 participants (48%) receiving a

lower zinc dose of 0.5 mg/kg/day (Group 2). This distribution ensured a nearly equal allocation between the two groups, allowing for a comparative analysis of the effects of different zinc supplementation doses on growth and neurodevelopmental outcomes in preterm neonates.

The doses for supplementation were chosen based on the study by Friel JK (69), which investigated a low dose of 0.5 mg/kg/day, and the ESPGHAN guidelines (2022) (124), which recommend 2 mg/kg/day for optimal enteral zinc intake in preterm infants. These references provide the scientific basis for the selected dosages, ensuring alignment with established research and clinical guidelines.

SOCIO-DEMOGRAPHIC DETAILS

The socio demographic characteristics of the enrolled infants, including age at enrolment, gender distribution, maternal education, and socioeconomic background, were comparable to previous studies on preterm neonates and zinc supplementation.

Gender Distribution

Among 75 participants, 53 were males (70.7%) and 22 females (29.3%), with a similar distribution between groups: 71.7% males in the test group and 69.4% in the control group. The difference was not statistically significant ($\chi^2 = 0.024$, $p = 0.876$), indicating successful randomization and reducing the potential for gender-related bias. This aligns with previous studies reporting male predominance in preterm cohorts such as Gupta et al., 2017 and Shah et al., 2020. [125,126]

Maternal Age

Most mothers (36%) were aged 25–30 years, followed by <25 years (29%) and 30–35 years (28%), with only 6.67% above 35 years. The control group had more

mothers aged 25–30 years (47.0%) and above 35 years (28.0%), while the test group had more aged 30–35 years (38.5%). Differences not significant ($\chi^2 = 6.95$, $p = 0.074$), but maternal age may influence neonatal outcomes. Findings align with Patel et al. ;2019 (127), who reported 40% of mothers aged 25–30 years.

Socioeconomic Status

The mean family income was ₹65.95K ± 57.64 in the test group and ₹54.53K ± 34.08 in the control group ($p = 0.296$), indicating comparable socioeconomic backgrounds, consistent with studies in India and South Asia (Kumar et al., 2018; Banerjee et al., 2016) (128,129).

Maternal Education

Mothers with bachelor's or higher degrees were slightly more in the test group (68.4%) than the control group (61.1%). They were not significant reducing the likelihood of education-related bias. Findings align with Gupta et al. (2017) and Patel et al. (2020). [125, 127]

Parental Presence

All fathers were present at home at enrollment, ensuring consistent parental involvement, adherence to supplementation, and compliance with follow-up assessments, minimizing variability in parental support as a confounding factor.

BIRTH CHARACTERISTICS

Gestational Age

Gestational age distribution was comparable between groups, with most neonates born at 34–35 weeks, minimizing its role as a confounding factor. Similar balance was observed in previous trials on zinc supplementation in preterm neonates.[119,120]

Singleton vs. Multiple Births

A significant difference was noted in singleton vs. multiple births ($\chi^2 = 3.920$, $p = .048$), with multiple births more frequent in the 2 mg/kg/day group.Multiple pregnancies are known to elevate the risk of preterm birth, and various health complications in neonates (Ragab et al., 2014; Islam et al., 2010)., potentially affecting study outcomes due to different nutritional needs and responses to zinc.[118,121]

Mode of Delivery

Lower Segment Cesarean Section (LSCS) was highly prevalent (86.7%), consistent with prior studies on preterm neonates (Mathur et al., 2015; Kumar et al., 2011). LSCS, often indicated for fetal distress, maternal comorbidities, and multiple gestations, can increase neonatal respiratory risks, but its even distribution across groups minimizes confounding effects.[115,128]

Neonatal Resuscitation & Respiratory Support

Apgar scores were comparable between groups ($\chi^2 = 0.144$, $p = .704$), aligning with studies showing no immediate effects of zinc on neonatal adaptation (Diaz-Gomez et al., 2003; Ragab et al., 2014). However, CPAP requirement differed significantly ($\chi^2 = 21.27$, $p < .001$), with fewer neonates in the 2 mg/kg/day group requiring CPAP and none needing it for more than one day. In contrast, 27% of the control group required CPAP for two days.[118,119]

Antenatal Steroid Exposure

Antenatal steroid exposure was imbalanced (χ^2 = 15.45, p = .0004), with 51.3% of the 2 mg/kg/day group receiving no steroids vs. 13.8% in the control group. Given steroids' role in reducing neonatal morbidity (Roberts & Dalziel, 2017), this disparity could have influenced respiratory outcomes.[130] Future studies should ensure balanced steroid exposure to minimize confounding.

POSTNATAL CHARACTERISTICS

Birth-Related Injuries and Postural Deformities

In our study, none of the neonates in either group exhibited postural deformities or birth-related mechanical injuries, such as facial paralysis, brachial plexus injury, or clavicle fractures. These findings suggest that mode of delivery and perinatal factors did not contribute to birth trauma in our cohort. Given the association of instrumental deliveries and difficult labor with birth injuries (Akangire et al., 2016), the absence of such complications in our study indicates effective obstetric management and a low risk of mechanical birth trauma among participants.

Breastfeeding Initiation

There was a higher protion of neonates in the test group (56.4%) initiated breastfeeding within the first day compared to the control group (43.6%). Conversely, delayed initiation (beyond one day) was more in the control group (63.8%) than in the test group (36.0%). Chi-square analysis was significant with (χ^2 = 5.26, p = 0.0218), suggesting that neonates receiving the intervention were more likely to begin breastfeeding earlier than those in the control group. Early initiation of breastfeeding is associated with improved neonatal outcomes, including enhanced immune protection and better weight gain (Victora et al., 2016). [132]

Initiation of Enteral Supplementation

Early enteral feeds in preterm neonates is crucial for gut maturation, reduced feeding intolerance, and improved overall outcomes. In our study, enteral supplementation was initiated earlier in the test group, with 43.5% starting by day 3 compared to 33.3% in the control group. By day 4, 53.8% of the test group had begun enteral feeds versus 61.1% in the control group, while initiation was delayed to day 5 in only 2.56% of the test group compared to 5.56% in the control group.

The significant statistical difference ($\chi^2 = 4.79$, $p = 0.0286$) indicates better feeding readiness in the test group, which has been associated with improved intestinal maturation and function (Berseth et al., 2010; Neu & Pammi, 2017). These findings highlight the potential benefits of targeted nutritional strategies in enhancing feeding tolerance and supporting growth in preterm infants.[133,134]

GROWTH

Occipitofrontal Circumference

OFC growth was comparable at birth, discharge, and 40 weeks CGA, but by 3 months CGA, the 2 mg/kg/day zinc group showed significantly greater growth (40.13 ± 1.77 cm vs. 39 ± 1.84 cm, $p = 0.009$). This suggests zincs role in postnatal brain development, as OFC is a key neurodevelopmental marker in preterm infants.

Consistent with our findings, Friel et al. (2013) reported improved head circumference growth with zinc supplementation. However, some studies found no significant impact. Taneja et al. (2017) observed no effect on OFC despite improved plasma zinc levels, while Mazariegos et al. (2010) suggested factors like overall nutrition and inflammation may influence outcomes.

The observed OFC increase suggests zinc at 2 mg/kg/day may enhance postnatal brain growth. Given its association with long-term cognitive and motor development (Uauy et al., 2015), further follow-up is needed to ascertain sustained neurodevelopmental benefits.

Weight

Weight parameter didn't show potential difference in birth, discharge, or 40-week CGA weights between groups. However, by 3 months CGA, the test group (2 mg/kg/day zinc) had a significantly higher mean weight (5498.21 ± 596.21 g vs. 5113.92 ± 778.48 g, $p = 0.018$), with a steeper weight gain trajectory.

Similar to our findings, studies by Friel et al. (2013) and Islam et al. (2018) reported improved weight gain with zinc supplementation (120,121). Abdul-Karem et al. (2016) also found enhanced weight gain in preterm neonates receiving zinc.[135] However, Taneja et al. (2017) found no significant effect, likely due to differences in nutritional status and supplementation duration, [136]

The significant weight gain in our study suggests that higher-dose zinc supplementation may enhance postnatal growth. Since early weight gain is linked to better neurodevelopmental outcomes (Uauy et al., 2015), further long-term studies are needed. [137]

Length

Length measurements were similar at birth, discharge, and 40 weeks CGA. However, by 3 months CGA, the test group had significantly greater length (58.00 ± 3.16 cm vs. 55.65 ± 3.12 cm, $p = 0.002$), with a steeper growth trajectory.

Zinc is crucial for skeletal growth, bone mineralization, and protein metabolism (Prasad, 2012) (21). Research by Raghavendra et al. (2014) and Castillo-Durán et al. (2018) also documented enhanced linear growth following zinc supplementation.[138,139] Similarly, Darlow et al. (2019) reported a significant increase in length at 3 months among preterm infants receiving ≥2 mg/kg/day of zinc. In contrast, Bhutta et al. (2016) found no measurable impact, which may be attributed to differences in baseline nutritional status.[140,141] The observed length gain suggests that adequate zinc intake may enhance skeletal development. Since early linear growth predicts long-term neurodevelopmental and metabolic outcomes (Victora et al., 2010), further follow-up is warranted.[132]

NEUROLOGICAL ASSESSMENT

Neurological assessments using the HNNE and HINE showed significantly improved neurodevelopmental outcomes in neonates receiving higher zinc supplementation (2 mg/kg/day). While HNNE scores at 40 weeks corrected gestational age (CGA) were comparable between groups ($p = 0.661$), by 3 months CGA, the test group exhibited significantly higher HINE scores ($p < 0.001$), suggesting zinc may enhance early neurological development.

Zinc role in synaptogenesis, myelination, and neurotransmission is well known(Prasad, 2012). Prior studies have linked adequate zinc intake to improved motor and cognitive function in preterm Darlow et al. (2019) similarly reported better neuromotor outcomes with higher zinc supplementation.[21,111,140,142]

Neurodevelopmental Outcomes at 3 Months CGA

- **Higher HINE Scores**: The test group showed significantly better global neurological function.
- **Improved Motor Control**: Better posture, tone, reflexes, and reactions suggest enhanced neuromuscular development.
- **Cranial Nerve Function**: No significant difference ($p = 0.083$), indicating cranial nerve maturation may follow a different trajectory.

Zinc is essential for synaptic plasticity, neuronal differentiation, and myelination (Tamura et al., 2013).[111] Studies link zinc deficiency to delayed myelination and neuromotor development (Mocchegiani et al., 2015), while supplementation improves neuromotor function in preterm infants (Ghavami et al., 2020).[143,144] The lack of effect on cranial nerve function is consistent with prior research, as zinc primarily influences motor pathways and synaptic development (Krebs, 2013). [145]

ZINC - COPPER INTERACTION

The evaluatio underscores the metabolic effects of increased zinc supplementation in preterm neonates. At birth, serum zinc levels were notably lower in the test group to the control group ($p = 0.031$). However, by 3 months CGA the test group demonstrated a significantly higher mean serum zinc level ($p < 0.001$), indicating efficient absorption and utilization of the supplemented zinc (2 mg/kg/day).

The marked increase in serum zinc levels in the test group supports previous findings that zinc supplementation effectively raises plasma zinc concentrations in preterm infants by Brown et al.[90] A study by Friel et al. (2016) also reported a

similar trend, where preterm neonates receiving higher zinc doses demonstrated significantly improved serum zinc status and associated benefits in growth and neurodevelopment. [120]

Conversely, serum copper levels followed an inverse pattern, with significantly lower values in the test group at 3 months CGA ($p < 0.001$). This observation aligns with the well-documented interaction between zinc and copper metabolism. High zinc intake can induce metallothionein synthesis in enterocytes, which preferentially binds copper, reducing its systemic absorption (Plum et al., 2010).[146] Given that copper is critical for hematopoiesis, mitochondrial function, and neuronal development, this decrease may have physiological implications, warranting careful monitoring in long-term supplementation strategies (Lönnerdal, 2021.[95]

Our findings suggest that while zinc supplementation enhances serum zinc levels, it may simultaneously impact copper homeostasis. This emphasizes the need for balanced micronutrient supplementation to avoid potential deficiencies or imbalances that could affect neonatal health outcomes.

Serum Zinc at Birth and Anthropometric Parameters

Our study identified a positive correlation between serum zinc levels at birth and both birth weight ($p = 0.015$) and birth length ($p = 0.014$), supporting zinc's role in fetal growth through its involvement in cellular proliferation, DNA synthesis, and protein metabolism (Tamura et al., 2013). However, no significant correlation was observed with occipitofrontal circumference (OFC), indicating that neurocranial development may rely on other micronutrients such as copper, iron, and essential fatty acids (Cusick & Georgieff, 2016).[146]

Subgroup analysis revealed moderate but statistically insignificant correlations between serum zinc and birth length in both the test group ($r = 0.541$, $p = 0.077$) and the control group ($r = 0.529$, $p = 0.069$). This could be attributed to sample size limitations or individual variations in zinc metabolism (Hambidge et al., 2011).[147] While previous research has linked zinc deficiency to intrauterine growth restriction (Uauy et al., 2015),[137] our findings suggest that fetal growth is influenced by multiple maternal and environmental factors.

Serum Zinc at 3 Months CGA and Anthropometric Parameters

At 3 months CGA, serum zinc levels were significantly correlated with OFC ($p = 0.026$), weight ($p = 0.024$), and height ($p = 0.001$), supporting zinc's role in postnatal brain and skeletal growth (Prasad, 2013; Wessells & Brown, 2012).[148,149] The strong correlation with length aligns with evidence on zinc's role in bone metabolism and protein synthesis (Hambidge et al., 2011).[147]

Subgroup analysis revealed a weak but significant correlation between serum zinc and OFC in the test group ($p = 0.039$), while correlations with weight and length were not significant. The control group showed no significant associations, suggesting zinc's impact may depend on baseline nutritional status and interactions with other micronutrients (Goldenberg et al., 2014). [150]

Neurological Outcomes and Zinc's Role in Neurodevelopment

Serum zinc levels demonstrated a highly significant positive correlation with the HINE score at 3 months CGA in the overall cohort ($r = 0.684$, $p < 0.001$), indicating a potential role of zinc in early neurodevelopment. This association aligns with zinc's known involvement in synaptic plasticity, neuronal differentiation, and

myelination—key processes essential for neurological maturation in preterm infants (Takeda, 2013; Mocchegiani et al., 2001).[144, 151] Interestingly, while the correlation remained statistically significant but moderate in the test group ($r = 0.283$, $p = 0.040$), it was not significant in the control group ($r = 0.066$, $p = 0.708$), reinforcing the hypothesis that zinc supplementation may enhance neurodevelopmental outcomes.

In contrast, the correlation between serum zinc levels and the HNNE score at 40 weeks CGA was weak and did not reach statistical significance in the overall cohort ($r = 0.155$, $p = 0.188$), the test group ($r = 0.265$, $p = 0.103$), or the control group ($r = 0.064$, $p = 0.714$). This finding suggests that zinc's impact on neurodevelopment may become more apparent over time rather than immediately after birth. This aligns with existing evidence that zinc's effects on neuronal function and synaptogenesis require time to manifest (Levenson, 2005).

Preterm neonates are at higher risk of zinc deficiency due to inadequate stores at birth and increased postnatal demands, which can negatively impact cognitive and motor development (Bhatnagar & Taneja, 2001).[152] The stronger correlation between serum zinc and HINE scores at 3 months CGA, but not with HNNE scores at 40 weeks, suggests that adequate zinc levels may be more critical for post-neonatal rather than immediate newborn neurological development.

These findings have important clinical implications, suggesting that zinc supplementation in preterm infants may support better neurodevelopmental outcomes, particularly in the first few months of life. However, the observed inverse relationship with serum copper highlights the need for careful micronutrient balance to avoid potential deficiencies or toxicities.

CORRELATION DATA

Gender and Neurological Outcomes

Neurological assessment scores showed no significant gender differences. At 40 weeks CGA, the mean HNNE score was 29.45 ± 2.36 (males) vs. 29.64 ± 1.87 (females) ($p = 0.746$). At 3 months CGA, the mean HINE score was 69.21 ± 2.45 (males) vs. 70 ± 2.41 (females) ($p = 0.204$). While females had slightly higher scores, the differences were not statistically significant, indicating that sec did not substantially impact early neurological outcomes.

Some studies suggest better neurodevelopmental outcomes in female preterm infants, possibly due to estrogen-related neuroprotection and differences in brain maturation (Raznahan et al., 2010; Claas et al., 2011).[153,154] However, others, including Spittle et al. (2017), align with our findings, reporting no significant gender-based differences in early assessments.[155] The uniform nutritional and medical care in our study may have contributed to these results.

Impact of Amniotic Fluid Type on Neurological Outcomes

At 40 weeks CGA, neonates born through meconium-stained liquor had lower HNNE scores (28.43 ± 2.15) compared to those with clear amniotic fluid (29.62 ± 2.21), though this difference was not statistically significant ($p = 0.178$). However, at 3 months CGA, HINE scores were significantly lower in the meconium-stained liquor group (67.14 ± 2.34) than in the clear liquor group (69.68 ± 2.35, $p = 0.008$), suggesting a negative impact of meconium exposure on early neurodevelopment.

Meconium-stained amniotic fluid (MSAF) is associated with hypoxia, inflammation, and oxidative stress, which can impact neonatal brain development (Ghidini & Spong, 2001)(156) . Even in non-MAS cases, in utero meconium exposure is associated with higher perinatal complications, including neonatal encephalopathy and lower Apgar scores, which may contribute to neurodevelopmental delays (Klinger & Kruse, 2020).[157]

Our findings support these concerns, suggesting that MSAF exposure may have delayed neurological effects despite the absence of immediate impairments at 40 weeks CGA. These delays may stem from cumulative postnatal effects of hypoxia, inflammation, or perinatal stressors.

Impact of Birth Cry on Neurological Outcomes

Our study found no significant differences in neurological scores between neonates who cried at birth and those who did not. At 40 weeks CGA, non-crying infants had slightly lower HNNE scores (27.75 ± 1.5) compared to those who cried (29.61 ± 2.21, $p = 0.103$). At 3 months CGA, non-crying infants had lower HINE scores (68.5 ± 4.12) than those who cried (69.49 ± 2.36, $p = 0.434$), but the difference was not statistically significant.

A newborn's first cry is a critical indicator of neurological function, signaling effective respiratory transition, oxygenation, and intact brainstem activity (Perlman & Risser, 1996).[158] Non-crying neonates face higher risks of birth asphyxia and hypoxic-ischemic encephalopathy (HIE), both associated with adverse neurodevelopmental outcomes (Volpe, 2019).[159] However, our study suggests that many non-crying neonates recover well without severe hypoxic injury.

Previous research indicates that transient perinatal distress does not always lead to long-term neurological deficits, especially with prompt resuscitation and postnatal support (Lawn et al., 2013).[160] Our findings align with these observations, though the small sample size of non-crying infants limits definitive conclusions.

Impact of CPAP Support on Neurological Outcomes

Our study found no significant differences in neurological scores between neonates who required CPAP support and those who did not. At 40 weeks CGA, the mean HNNE score was slightly lower in the CPAP group (29.34 ± 1.56) than in the non-CPAP group (29.61 ± 2.55, $p = 0.619$). At 3 months CGA, the HINE score was 69 ± 2.42 in the CPAP group and 69.72 ± 2.46 in the non-CPAP group, with no significant difference ($p = 0.219$).

CPAP is a widely used reducing the need for intubation-related complications. While severe respiratory distress and prolonged mechanical ventilation are linked to poorer neurodevelopment (Schmidt et al., 2014), CPAP itself is generally considered safe with minimal long-term neurological impact (Laptook et al., 2021). [161,162]

Our findings are consistent with previous research. Fischer et al. (2018) reported no significant differences in motor and cognitive development at two years of age between preterm infants treated with CPAP and those who underwent early intubation.[163] Similarly, the SUPPORT trial found no notable differences in neurodevelopmental outcomes at 18–22 months among infants randomized to CPAP versus mechanical ventilation (Finer et al., 2010).[164]

Association between APGAR Scores and Early Neurological Outcomes

In our study, all neonates had an APGAR score above 5 at 1 minute and greater than 7 at 5 minutes, indicating favorable early adaptation to extrauterine life. Spearman rank correlation analysis between APGAR scores and neurological assessment scores showed weak, non-significant associations:

At both 40 weeks CGA and 3 months CGA, the correlations between HNNE/HINE scores and APGAR scores at 1 minute and 5 minutes were weak and not statistically significant (all p-values > 0.05). This suggests no strong relationship between APGAR scores and the neurological assessments at these time points.

The APGAR score is a well-established clinical tool for assessing a newborn's immediate condition after birth, particularly in cases of perinatal asphyxia. However, its utility in predicting long-term neurodevelopmental outcomes remains debated. Low APGAR scores (<5 at 5 minutes) are linked to higher risks of cerebral palsy, neurodevelopmental delay, and cognitive impairment (Thorngren-Jerneck & Herbst, 2001; Moster et al., 2002). [165,166]

Normal APGAR scores (as in our study) have a limited ability to predict subtle neurodevelopmental differences (Casey et al., 2001).[167]A meta-analysis by Perlman & Risser (1996) found that in preterm infants, APGAR scores alone were poor predictors of long-term neurodevelopment compared to other clinical factors.[158]

Laptook et al. (2009) also concluded that while extremely low APGAR scores correlate with neurological impairment, a normal score does not guarantee normal neurodevelopment.[162]

Association between Hammersmith Neonatal Neurological Examination (HNNE) Scores and Anthropometric Measurements

Understanding the relationship between early neurological function and physical growth provides insight into how anthropometric parameters influence neurodevelopment in preterm infants. Our study examined the Pearson correlation between Hammersmith Neonatal Neurological Examination (HNNE) scores at 40 weeks corrected gestational age (CGA) and three key anthropometric measures: occipitofrontal circumference (OFC), weight, and height.

- HNNE and OFC: A weak negative correlation was found ($r = -0.120$, $p = 0.307$), suggesting no significant association between neurological scores and head circumference at this stage.

- HNNE and Weight: A negligible correlation ($r = 0.004$, $p = 0.971$) indicates that weight does not have a measurable impact on early neurological function as assessed by HNNE.

- HNNE and Height: Another weak negative correlation ($r = -0.083$, $p = 0.479$) suggests that length is not significantly linked to early neurological outcomes.

These findings suggest that HNNE scores at 40 weeks CGA are not strongly influenced by early growth parameters, indicating that neonatal anthropometry alone may not serve as a reliable predictor of neurological function at this stage.

Birth weight and head circumference have been linked to later cognitive and motor development (Franz et al., 2018).[168] However, early growth may not necessarily correlate with neurological function as assessed by HNNE . Simic et al. (2020) found that while postnatal growth restriction predicts long-term

neurodevelopmental impairments, early standardized neurological assessments like HNNE are more influenced by perinatal complications and brain maturity than by simple anthropometric measures.[169]Luttikhuizen dos Santos et al. (2013) emphasized that neurodevelopment in preterm infants results from a complex interplay of factors including nutrition, environmental stimulation, and medical care, rather than just growth parameters.[170]

ASSOCIATION OF ZINC WITH COMORBIDITIES

Our study found that none of the neonates from either group required hospital readmission or physician consultation for diarrhea, respiratory illness, skin lesions, failure to thrive, retinopathy of prematurity (ROP), or necrotizing enterocolitis (NEC) post-discharge.

Zinc is known for its essential role in immune function, intestinal integrity, and overall growth. Previous studies have indicated that zinc deficiency can predispose infants to infections, gastrointestinal disturbances, and poor growth outcomes.[109-111] However, in our cohort, even among the control group that did not receive higher-dose zinc there was no observed increase in these adverse health outcomes. This may suggest that standard nutritional support, including breast milk and multivitamins, was sufficient to meet the neonates' zinc requirements.

Additionally, contrast with previous studies that have shown an association between zinc deficiency and increased susceptibility to infections and gastrointestinal disorders. The absence of NEC cases in both groups is particularly notable, as zinc deficiency has been linked to gut barrier dysfunction, which can contribute to NEC development. The lack of ROP cases also aligns with evidence suggesting that adequate zinc levels support retinal development.

These results highlight the potential role of routine neonatal care, nutritional interventions, and close follow-up in minimizing post-discharge morbidity in preterm neonates. However, given the relatively small sample size and the controlled hospital environment in which our study was conducted, they should be interpreted with caution.

STRENGTHS OF THE STUDY

- The study's randomized design strengthens its validity by minimizing bias and ensuring balanced baseline characteristics between groups. Minor differences in initial anthropometric status were observed, but the significant differences in outcomes suggest a true effect of zinc supplementation.

- Our study assessed multiple outcomes, including growth parameters, neurodevelopmental scores, serum zinc and copper levels, providing a holistic evaluation of zinc's effects.

- The findings hold significant implications for preterm infant nutrition, particularly in resource-limited settings where zinc deficiency is prevalent. Given that zinc supplementation had no significant side effects, this intervention could be safely integrated into neonatal care protocols.

- No significant side effects, such as vomiting, diarrhea, abdominal cramps, or loss of appetite, were reported in the zinc group. Studies indicate that zinc overdose is rare with doses <25 mg/day (Terrin et al., 2013). The absence of side effects reinforces the safety of zinc supplementation at the studied dose (2 mg/kg/day).

LIMITATIONS OF THE STUDY

- As the study was conducted at a single center, its findings may not be fully applicable to neonatal populations with varying demographics, nutritional statuses, or healthcare environments.
- Neurodevelopmental assessment using HNNE and HINE scales at 3 months corrected age provides only an early indication of neurological outcomes. Long-term effects on cognition, motor skills, and behaviour may require extended follow-up
- Despite monitoring, actual adherence to supplementation may vary, and parental involvement introduces potential inconsistencies.
- Only serum zinc and copper levels are analyzed, without considering other micronutrients (e.g., iron, selenium, or vitamin A) or individual variations (due to genetic or gastrointestinal factors) in zinc metabolism.
- The study followed participants up to three months corrected age, limiting the ability to assess long-term neurodevelopmental outcomes.
- Although our study monitored feeding patterns, breastfeeding exclusivity, and complementary feeding, detailed nutritional intake data (e.g., caloric and protein intake) were not rigorously quantified.
- Antenatal steroid use and multiple births were not evenly distributed between groups. While statistical adjustments were made, these factors could have influenced respiratory adaptation and growth outcomes. Future studies should consider larger sample sizes and stratified randomization to address these imbalances.

RECOMMENDATIONS

- Consider multicenter trials to improve generalizability across diverse neonatal populations.
- Incorporate long-term assessments to capture effects on cognition, motor skills, growth, and immunity.
- Use electronic monitoring or regular parental counseling to ensure adherence to supplementation.
- Standardize data collection on maternal nutrition, breastfeeding, and socioeconomic factors to better adjust for their impact.
- Include additional micronutrients and explore genetic or gastrointestinal factors affecting zinc metabolism.

CONCLUSION

Socioeconomic & Demographic Factors

There were no notable differences in maternal age, level of education, or household income between the groups. Additionally, all neonates had their fathers living at home.

Pregnancy & Birth Factors

Gender, gestational age, mode of delivery, and amniotic fluid status were similar between groups. However, multiple births were more frequent in the test group, and fewer test group neonates received the full antenatal steroid regimen.

Birth & Resuscitation Factors

APGAR scores were comparable between groups. CPAP use was higher in the control group, while no birth-related complications were reported in either group.

Postnatal Factors

Early breastfeeding initiation was more frequent in the test group but not significantly different. Hospital stay duration was similar between groups.

Serum Zinc & Copper Levels

Serum zinc levels were initially lower in the test group at birth but increased by 3 months corrected gestational age. Meanwhile, serum copper levels remained lower in the test group throughout the study period.

Neurodevelopmental Outcomes

HNNE scores at 40 weeks CGA showed no differences between groups. However, HINE scores are greater in the test group at 3 months CGA.

Growth Parameters at 3 Months CGA

The test group showed greater improvements in head circumference, weight, and length.

Correlation Analysis

At birth, serum zinc levels showed a positive correlation with birth weight and length. By 3 months corrected gestational age, they were significantly linked to head circumference, weight, length, and HINE scores in the test group.

Additional Findings

HNNE and HINE scores remained consistent across gender, amniotic fluid status, neonatal crying at birth, CPAP use, and APGAR scores but were higher in neonates with clear amniotic fluid.

SUMMARY

This randomized controlled trial evaluates the impact of a higher zinc supplementation dose (2 mg/kg/day) compared to a lower dose (0.5 mg/kg/day) on postnatal growth and neurodevelopment in preterm neonates up to three CGA. The study groups were well-balanced in terms of socioeconomic and demographic factors, birth resuscitation measures, and early postnatal characteristics. Baseline serum zinc levels were similar between groups, ensuring outcomes were likely due to zinc supplementation rather than pre-existing variations.

By three months CGA, infants receiving the higher zinc dose exhibited significantly greater growth in key anthropometric parameters, including weight, length, and head circumference. These findings highlight the essential role of zinc in promoting postnatal growth, likely through its involvement in cellular proliferation, immune function, and metabolic processes necessary for early development.

Neurodevelopmental assessments using the Hammersmith Infant Neurological Examinationshowed that infants in the higher-dose zinc group had superior scores, indicating enhanced neurological maturation. Zinc is known to contribute to neuronal differentiation, synaptic plasticity, and myelination—processes critical for early brain development. Furthermore, a positive correlation between serum zinc levels and neurodevelopmental scores reinforces its role in cognitive and motor function development in preterm neonates.

However, the study also observed a notable decline in serum copper levels in the test group, raising concerns about potential zinc-induced copper depletion. Since zinc and copper compete for absorption in the intestines, excessive zinc intake may interfere with copper absorption, potentially leading to functional copper deficiency.

Given copper's importance in neurological function, blood cell production, and immune regulation, these findings highlight the need for further research on micronutrient balance in preterm infants receiving high-dose zinc supplementation.

While this study provides valuable insights, the long-term effects of zinc supplementation on neurodevelopment, growth, and immune function remain unknown. Future research should focus on extended follow-up to assess sustained neurodevelopmental outcomes, determine the optimal dosage and duration of zinc supplementation, and evaluate whether concurrent copper supplementation is necessary to mitigate potential deficiencies.

In conclusion, higher-dose zinc supplementation appears to be a promising strategy for enhancing growth and neurodevelopment in preterm neonates. However, careful consideration of micronutrient interactions, particularly zinc-copper balance, is crucial to optimizing neonatal nutritional interventions. Further research is needed to establish safe and effective supplementation protocols that maximize benefits while minimizing risks in this vulnerable population.

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ANNEXURE – I - INFORMED CONSENT FORM

KAHER's JNMC, BELAGAVI

DEPARTMENT OF PAEDIATRICS

ASSESSMENT OF THE EFFECT OF TWO DIFFERENT DOSES OF ZINC SUPPLEMNTATION ON NEURODEVELOPMENT AND GROWTH IN LATE PRETERMS : A HOSPITAL BASED RANDOMISED CONTROLLED TRIAL

Name of Student/Principal Investigator: _____

Name of Guide/Co Investigators: _____

Introduction: Zinc is second important trace element in our body after iron and it is needed in most physiological functions of the body. It is very important for the growth and neurological development. Preterm babies born less than 37 weeks gestational age have reduced zinc stores as there is less time for transfer of zinc from mother to foetus. Hence zinc supplementation after birth becomes essential.Therefore this study will be conducted with two different dosages of zinc to correlate with the growth and neurodevelopment.

Explanation of procedure:

Following the voluntary decision to enrol the neonate in this study, a detailed pro forma will be filled and the neonate will be randomised to group 1 which will receive Syrup Zinc at 2 mg/kg/day and group 2 which will receive syp zinc at 500mcg/kg/day. Supplementation will be started after 48 hours of life and will be continued till 3 months of corrected gestational age. Blood sample to estimate serum Zinc in the neonate will be taken prior to starting Zinc supplementation.

Anthropometric measurements will be taken off the neonate at birth and at the time of discharge. Mother will be asked to followup at 40th week of corrected gestational age and at 3 months of corrected gestational age. The compliance will be monitored by log book entries and amount of syrup zinc in the bottle. At 40th week of corrected gestational age- anthropometric measurements and neurological examination by Hammersmith neonatal neurological examination will be done. At 3 months of corrected gestational age anthropometric measurements, neurological examination by Hammersmith infant Neurological examination and blood sampling of zinc and copper levels will be done. This will complete the trial period.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study. Zinc supplementation used in this particular study will not cause any side effects to the baby.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator.**

Authorisation for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact:

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee for Human Subjects' Research of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waiving off any of your legal rights

KAHER's JNMC, बेलगावी

बाल रोग विभाग

सूचित सहमति प्रपत्र

न्यूरोडेवलपमेंट पर जिंक सप्लीमेंट की दो अलग-अलग खुराक के प्रभाव का आकलन और देर से पूर्व में वृद्धि: एक अस्पताल आधारित यादृच्छिक नियंत्रित परीक्षण

छात्र/प्रधान अन्वेषक का नाम:

गाइड/सह अन्वेषक का नाम:

परिचय: आयरन के बाद जिंक हमारे शरीर में दूसरा महत्वपूर्ण ट्रेस तत्व है और शरीर के अधिकांश शारीरिक कार्यों में इसकी आवश्यकता होती है। यह वृद्धि और तंत्रिका संबंधी विकास के लिए बहुत महत्वपूर्ण है। गर्भावस्था के 37 सप्ताह से कम समय में जन्म लेने वाले प्रीटर्म शिशुओं में जिंक स्टोर कम हो जाता है क्योंकि मां से भ्रूण में जिंक के हस्तांतरण के लिए कम समय होता है। इसलिए जन्म के बाद जिंक सप्लीमेंट आवश्यक हो जाता है। इसलिए यह अध्ययन जिंक की दो अलग-अलग खुराकों के साथ किया जाएगा ताकि विकास और न्यूरो विकास के साथ संबंध स्थापित किया जा सके।

प्रक्रिया की व्याख्या:

इस अध्ययन में नवजात को नामांकित करने के स्वैच्छिक निर्णय के बाद, एक विस्तृत प्रोफार्मा भरा जाएगा और नवजात को समूह 1 में रेंडमाइज किया जाएगा जो 2 मिलीग्राम / किग्रा / दिन पर एसआईपी जिंक प्राप्त करेगा और समूह 2 जिसे 500एमसीजी / एसआईपी जिंक प्राप्त होगा। किग्रा/दिन। अनुपूरण जीवन के 48 घंटों के बाद शुरू किया जाएगा और सही गर्भकालीन आयु के 3 महीने तक जारी रहेगा। नवजात में सीरम जिंक का अनुमान लगाने के लिए रक्त का नमूना जिंक अनुपूरण शुरू करने से पहले लिया जाएगा। जन्म के समय और डिस्चार्ज के समय नवजात शिशु का एंथ्रोपोमेट्रिक माप लिया जाएगा। मां को सही गर्भकालीन आयु के 40वें सप्ताह और सही की गई गर्भकालीन आयु के 3 महीने पर फॉलोअप करने के लिए कहा जाएगा। अनुपालन की निगरानी लॉग बुक प्रविष्टियों और बोतल में सिरप जिंक की मात्रा द्वारा की जाएगी। संशोधित गर्भकालीन आयु के 40वें सप्ताह में एंथ्रोपोमेट्रिक मापन और हैमरस्मिथ द्वारा न्यूरोलॉजिकल जांच की जाएगी। 3 महीने की सही गर्भकालीन आयु पर एंथ्रोपोमेट्रिक माप, हैमरस्मिथ शिशु द्वारा न्यूरोलॉजिकल परीक्षा न्यूरोलॉजिकल परीक्षा और जिंक और कॉपर के स्तर के रक्त के नमूने लिए जाएंगे। यह परीक्षण अवधि को पूरा करेगा।

अध्ययन में भाग लेने से पीछे हटना: स्वैच्छिक रूप से इस अध्ययन में भाग लेना। आप इस अध्ययन में भाग लेने या एक बार नामांकित होने के बाद भागीदारी जारी रखने का निर्णय लेने के लिए स्वतंत्र होंगे। यदि आप अपनी भागीदारी वापस लेने का निर्णय लेते हैं, तो आप ऐसा करने के लिए स्वतंत्र हैं। हालांकि, कृपया मुख्य अन्वेषक को निर्णय बताएं।

अध्ययन में भाग लेने से संभावित लाभ: इस अध्ययन में भाग लेने से आपको कोई लाभ नहीं मिलेगा। एकत्र किए गए डेटा से बड़े पैमाने पर आबादी को मदद मिलेगी।

अध्ययन में भाग लेने से संभावित जोखिम: इस अध्ययन में भाग लेने में कोई जोखिम शामिल नहीं है। इस विशेष अध्ययन में उपयोग किए गए ज़िंक अनुपूरण से शिशु पर कोई दुष्प्रभाव नहीं पड़ेगा।

गोपनीयता और गोपनीयता: आपके द्वारा एकत्र की गई जानकारी को किसी भी तरह से रोकने के लिए कोडित किया जाएगा
व्यक्ति आपको पहचानने के लिए। आपकी पहचान कभी उजागर नहीं होगी। आपसे एकत्र किए गए डेटा को गोपनीय रखा जाएगा और प्रकाशन के लिए केवल संसाधित या एकत्रित डेटा का उपयोग किया जाएगा।
वित्तीय प्रोत्साहन: इस अध्ययन में भाग लेने के लिए आपको कोई भुगतान नहीं मिलेगा।

अध्ययन के दौरान किए गए अन्वेषणों की लागत का भुगतान प्रधान अन्वेषक द्वारा किया जाएगा।

एकत्रित डेटा के प्रकाशन के लिए प्राधिकरण: एकत्रित डेटा के प्रसंस्करण के बाद प्राप्त परिणाम वैज्ञानिक उद्देश्य के लिए प्रकाशित किए जाएंगे और या वैज्ञानिक समूहों को प्रस्तुत किए जाएंगे। हालांकि, आपकी पहचान कभी उजागर नहीं की जाएगी।

यदि आपके पास अध्ययन प्रतिभागी के रूप में अपने अधिकार के संबंध में कोई प्रश्न या शिकायत है, तो आप डॉ हर्षा हेगड़े, चेयरपर्सन, एथिकल कमेटी फॉर ह्यूमन सब्जेक्ट्स रिसर्च ऑफ जेएनएमसी, 0831-2473777 एक्सटेंशन 4052 से संपर्क कर सकते हैं।

कानूनी अधिकार: इस सहमति फॉर्म पर हस्ताक्षर करके, हम आपके किसी भी कानूनी अधिकार का त्याग नहीं कर रहे हैं

KAHER's JNMC , ಬೆಳಗಾವಿ

ಮಕ್ಕಳ ವಿಭಾಗ

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ನ್ಯೂರೋ ಡೆವಲಪ್ಮೆಂಟ್ ಮತ್ತು ಬೆಳವಣಿಗೆಯ ಮೇಲೆ ಎರಡು ವಿಭಿನ್ನ ಡೋಸ್ ಝಿಂಕ್ ಸಪ್ಲಿಮೆಂಟೇಶನ್

ಪರಿಣಾಮದ ಮೌಲ್ಯಮಾಪನ

ವಿದ್ಯಾರ್ಥಿ/ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು:

ಮಾರ್ಗದರ್ಶಿ/ಸಹ ತನಿಖಾಧಿಕಾರಿಗಳ ಹೆಸರು:

ಪರಿಚಯ: ಸತುವು ನಮ್ಮ ದೇಹದಲ್ಲಿ ಕಬ್ಬಿಣದ ನಂತರ ಎರಡನೇ ಪ್ರಮುಖ ಜಾಡಿನ ಅಂಶವಾಗಿದೆ ಮತ್ತು ಇದು ದೇಹದ ಹೆಚ್ಚಿನ ಶಾರೀರಿಕ ಕ್ರಿಯೆಗಳಲ್ಲಿ ಅಗತ್ಯವಾಗಿರುತ್ತದೆ. ಬೆಳವಣಿಗೆ ಮತ್ತು ನರವೈಜ್ಞಾನಿಕ ಬೆಳವಣಿಗೆಗೆ ಇದು ಬಹಳ ಮುಖ್ಯ. ತಾಯಿಯಿಂದ ಭ್ರೂಣಕ್ಕೆ ಸತುವು ವರ್ಗಾವಣೆಗೆ ಕಡಿಮೆ ಸಮಯ ಇರುವುದರಿಂದ **37** ವಾರಗಳ ಗರ್ಭಾವಸ್ಥೆಯ ವಯಸ್ಸಿನ ಅವಧಿಗಿಂತ ಕಡಿಮೆ ಅವಧಿಯಲ್ಲಿ ಜನಿಸಿದ ಪ್ರಸವಪೂರ್ವ ಶಿಶುಗಳು ಸತುವು ಶೇಖರಣೆಯನ್ನು ಕಡಿಮೆಗೊಳಿಸುತ್ತವೆ. ಆದ್ದರಿಂದ ಜನನದ ನಂತರ ಸತುವು ಪೂರಕವಾಗಿದೆ. ಆದ್ದರಿಂದ ಬೆಳವಣಿಗೆ ಮತ್ತು ನರಗಳ ಬೆಳವಣಿಗೆಯೊಂದಿಗೆ ಪರಸ್ಪರ ಸಂಬಂಧ ಹೊಂದಲು ಈ ಅಧ್ಯಯನವನ್ನು ಸತುವಿನ ಎರಡು ವಿಭಿನ್ನ ಡೋಸೇಜ್‌ಗಳೊಂದಿಗೆ ನಡೆಸಲಾಗುವುದು.

ಕಾರ್ಯವಿಧಾನದ ವಿವರಣೆ:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನವಜಾತ ಶಿಶುವನ್ನು ದಾಖಲಿಸಲು ಸ್ವಯಂಪ್ರೇರಿತ ನಿರ್ಧಾರವನ್ನು ಅನುಸರಿಸಿ, ವಿವರವಾದ ಪ್ರೋಫಾರ್ಮಾವನ್ನು ಭರ್ತಿ ಮಾಡಲಾಗುತ್ತದೆ ಮತ್ತು ನವಜಾತ ಶಿಶುವನ್ನು ಗುಂಪು **1** ಗೆ ಯಾದೃಚ್ಛಿಕಗೊಳಿಸಲಾಗುತ್ತದೆ, ಇದು **2 mg/kg/day** ನಲ್ಲಿ **Syp Zinc** ಅನ್ನು ಸ್ವೀಕರಿಸುತ್ತದೆ ಮತ್ತು ಗುಂಪು **2 500mcg/** ನಲ್ಲಿ **syp** ಸತುವನ್ನು ಸ್ವೀಕರಿಸುತ್ತದೆ. ಕೆಜಿ **/** ದಿನ. **48** ಗಂಟೆಗಳ ಜೀವಿತಾವಧಿಯ ನಂತರ ಪೂರಕವನ್ನು ಪ್ರಾರಂಭಿಸಲಾಗುವುದು ಮತ್ತು ಸರಿಪಡಿಸಲಾದ ಗರ್ಭಾವಸ್ಥೆಯ ವಯಸ್ಸಿನ **3** ತಿಂಗಳವರೆಗೆ ಮುಂದುವರಿಯುತ್ತದೆ. ನವಜಾತ ಶಿಶುವಿನ ಸೀರಮ್ ಝಿಂಕ್ ಅನ್ನು ಅಂದಾಜು ಮಾಡಲು ರಕ್ತದ ಮಾದರಿಯನ್ನು ಸತುವು ಪೂರಕವನ್ನು ಪ್ರಾರಂಭಿಸುವ ಮೊದಲು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ. ನವಜಾತ ಶಿಶುವಿನ

ಜನನ ಮತ್ತು ವಿಸರ್ಜನೆಯ ಸಮಯದಲ್ಲಿ ಆಂಥ್ರೊಪೊಮೆಟ್ರಿಕ್ ಅಳತೆಗಳನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ. ಸರಿಪಡಿಸಿದ ಗರ್ಭಾವಸ್ಥೆಯ ವಯಸ್ಸಿನ **40** ನೇ ವಾರದಲ್ಲಿ ಮತ್ತು ಸರಿಪಡಿಸಿದ ಗರ್ಭಾವಸ್ಥೆಯ ವಯಸ್ಸಿನ **3** ತಿಂಗಳಲ್ಲಿ ತಾಯಿಯನ್ನು ಅನುಸರಿಸಲು ಕೇಳಲಾಗುತ್ತದೆ. ಲಾಗ್ ಬುಕ್ ನಮೂದುಗಳು ಮತ್ತು ಬಾಟಲಿಯಲ್ಲಿರುವ ಸಿರಪ್ ಸತುವಿನ ಪ್ರಮಾಣದಿಂದ ಅನುಸರಣೆಯನ್ನು ಮೇಲ್ವಿಚಾರಣೆ ಮಾಡಲಾಗುತ್ತದೆ. **40** ನೇ ವಾರದಲ್ಲಿ ಸರಿಪಡಿಸಲಾದ ಗರ್ಭಾವಸ್ಥೆಯ ವಯಸ್ಸು- ಆಂಥ್ರೊಪೊಮೆಟ್ರಿಕ್ ಮಾಪನಗಳು ಮತ್ತು ಹ್ಯಾಮಸ್ಮಿತ್ ನವಜಾತ ನರವೈಜ್ಞಾನಿಕ ಪರೀಕ್ಷೆಯಿಂದ ನರವೈಜ್ಞಾನಿಕ ಪರೀಕ್ಷೆಯನ್ನು ಮಾಡಲಾಗುತ್ತದೆ. **3** ತಿಂಗಳ ಸರಿಪಡಿಸಿದ ಗರ್ಭಾವಸ್ಥೆಯ ವಯಸ್ಸಿನ ಆಂಥ್ರೊಪೊಮೆಟ್ರಿಕ್ ಮಾಪನಗಳಲ್ಲಿ, ಹ್ಯಾಮಸ್ಮಿತ್ ಶಿಶುವಿನ ನರವೈಜ್ಞಾನಿಕ ಪರೀಕ್ಷೆ ಮತ್ತು ಸತು ಮತ್ತು ತಾಮ್ರದ ಮಟ್ಟಗಳ ರಕ್ತದ ಮಾದರಿಯನ್ನು ಮಾಡಲಾಗುತ್ತದೆ. ಇದು ಪ್ರಾಯೋಗಿಕ ಅವಧಿಯನ್ನು ಪೂರ್ಣಗೊಳಿಸುತ್ತದೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆಯಿಂದ ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಿಕೆ: ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆ. ಒಮ್ಮೆ ದಾಖಲಾದ ನಂತರ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಬೇಕೆ ಅಥವಾ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ಮುಂದುವರಿಸಬೇಕೆ ಎಂದು ನಿರ್ಧರಿಸಲು ನೀವು ಸ್ವತಂತ್ರರಾಗಿರುತ್ತೀರಿ. ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ಹಿಂತೆಗೆದುಕೊಳ್ಳಲು ನೀವು ನಿರ್ಧರಿಸಿದರೆ, ಹಾಗೆ ಮಾಡಲು ನೀವು ಸ್ವತಂತ್ರರು. ಆದಾಗ್ಯೂ, ದಯವಿಟ್ಟು ನಿರ್ಧಾರವನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಗೆ ತಿಳಿಸಿ.

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ಸಂಭವನೀಯ ಪ್ರಯೋಜನಗಳು: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ನೀವು ಯಾವುದೇ ಪ್ರಯೋಜನಗಳನ್ನು ಪಡೆಯುವುದಿಲ್ಲ. ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯು ಜನಸಂಖ್ಯೆಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ಸಂಭವನೀಯ ಅಪಾಯಗಳು: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ಯಾವುದೇ ಅಪಾಯಗಳಿಲ್ಲ. ಈ ನಿರ್ದಿಷ್ಟ ಅಧ್ಯಯನದಲ್ಲಿ ಬಳಸಲಾದ ಝಿಂಕ್ ಪೂರಕವು ಮಗುವಿಗೆ ಯಾವುದೇ ಅಡ್ಡ ಪರಿಣಾಮಗಳನ್ನು ಉಂಟುಮಾಡುವುದಿಲ್ಲ.

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ: ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಯಾವುದನ್ನೂ ತಡೆಯಲು ಕೋಡ್ ಮಾಡಲಾಗುತ್ತದೆ

ನಿಮ್ಮನ್ನು ಗುರುತಿಸಲು ವ್ಯಕ್ತಿ. ನಿಮ್ಮ ಗುರುತನ್ನು ಎಂದಿಗೂ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಿದ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಪ್ರಕ್ರಿಯೆಗೊಳಿಸಿದ ಅಥವಾ ಒಟ್ಟುಗೂಡಿದ ಡೇಟಾವನ್ನು ಮಾತ್ರ ಪ್ರಕಟಣೆಗಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ.

ಹಣಕಾಸಿನ ಪ್ರೋತ್ಸಾಹಗಳು: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಯಾವುದೇ ಪಾವತಿಯನ್ನು ಸ್ವೀಕರಿಸುವುದಿಲ್ಲ.

ಅಧ್ಯಯನದ ಅವಧಿಯಲ್ಲಿ ಮಾಡಿದ ತನಿಖೆಗಳ ವೆಚ್ಚವನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಪಾವತಿಸುತ್ತಾರೆ.

ಒಟ್ಟುಗೂಡಿದ ದತ್ತಾಂಶದ ಪ್ರಕಟಣೆಗೆ ಅಧಿಕಾರ: ಒಟ್ಟು ದತ್ತಾಂಶವನ್ನು ಸಂಸ್ಕರಿಸಿದ ನಂತರ ಪಡೆದ ಫಲಿತಾಂಶಗಳನ್ನು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಪ್ರಕಟಿಸಲಾಗುತ್ತದೆ ಅಥವಾ ವೈಜ್ಞಾನಿಕ ಗುಂಪುಗಳಿಗೆ ಪ್ರಸ್ತುತಪಡಿಸಲಾಗುತ್ತದೆ. ಆದಾಗ್ಯೂ, ನಿಮ್ಮ ಗುರುತನ್ನು ಎಂದಿಗೂ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ.

ಪ್ರಶ್ನೆಗಳು: ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳಿದ್ದಲ್ಲಿ, ನೀವು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ:

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ನಿಮ್ಮ ಹಕ್ಕಿನ ಕುರಿತು ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳನ್ನು ಅಥವಾ ದೂರುಗಳನ್ನು ಹೊಂದಿದ್ದರೆ, ನೀವು **JNMC** ಯ ಮಾನವ ವಿಷಯಗಳ ಸಂಶೋಧನೆಯ ನೈತಿಕ ಸಮಿತಿಯ ಅಧ್ಯಕ್ಷರಾದ ಡಾ ಹರ್ಷ ಹೆಗ್ಡೆ, **0831-2473777** ವಿಸ್ತರಣೆ **4052** ಅನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

ಕಾನೂನು ಹಕ್ಕುಗಳು: ಈ ಸಮ್ಮತಿಯ ನಮೂನೆಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ, ನಿಮ್ಮ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ನಾವು ಬಿಟ್ಟುಕೊಡುವುದಿಲ್ಲ.

KAHER's JNMC, बेलागावी

बालरोग विभाग

सूचित संमती फॉर्म

झिंक सप्लिमेंटेशनच्या दोन वेगवेगळ्या डोसच्या न्यूरोडेव्हलपमेंटवर आणि उशीरा प्रीटरम्सच्या वाढीवर परिणामाचे मूल्यांकन: एक हॉस्पिटल आधारित यादृच्छिक नियंत्रित चाचणी

विद्यार्थ्याचे/मुख्याध्यापक अन्वेषकाचे नाव:

मार्गदर्शक/सह अन्वेषकांचे नाव:

परिचय: झिंक हा आपल्या शरीरातील लोहानंतरचा दुसरा महत्त्वाचा घटक आहे आणि शरीराच्या बहुतेक शारीरिक कार्यांसाठी त्याची गरज असते. वाढ आणि न्यूरोलॉजिकल विकासासाठी हे खूप महत्वाचे आहे. गर्भावस्थेच्या 37 आठवड्यांपेक्षा कमी कालावधीत जन्मलेल्या अकाली बाळांमध्ये झिंकचे साठे कमी होतात कारण आईकडून गर्भात झिंकचे हस्तांतरण होण्यास कमी वेळ असतो. त्यामुळे जन्मानंतर झिंकची सप्लिमेंटेशन आवश्यक बनते. त्यामुळे वाढ आणि न्यूरो डेव्हलपमेंटशी संबंध ठेवण्यासाठी झिंकच्या दोन वेगवेगळ्या डोससह हा अभ्यास केला जाईल.

प्रक्रियेचे स्पष्टीकरण:
या अभ्यासात नवजात शिशूची नावनोंदणी करण्याच्या ऐच्छिक निर्णयानंतर, एक तपशीलवार प्रोफॉर्मा भरला जाईल आणि नवजात शिशुला गट 1 मध्ये यादृच्छिक केले जाईल ज्याला 2 mg/kg/day दराने Syp Zinc मिळेल आणि गट 2 ज्यांना 500mcg/ वर syp झिंक मिळेल. किलो/दिवस. सप्लिमेंटेशन 48 तासांच्या आयुष्यानंतर सुरू केले जाईल आणि 3 महिन्यांचे गर्भधारणेचे वय दुरुस्त होईपर्यंत सुरू राहील. झिंक सप्लिमेंटेशन सुरू करण्यापूर्वी नवजात मुलामध्ये सीरम झिंकचा अंदाज लावण्यासाठी रक्ताचा नमुना घेतला जाईल. जन्माच्या वेळी आणि डिस्चार्जच्या वेळी नवजात मुलाचे मानववंशीय मोजमाप घेतले जाईल. सुधारलेल्या गर्भधारणेच्या वयाच्या 40 व्या आठवड्यात आणि गर्भधारणेच्या सुधारित वयाच्या 3 महिन्यांत आईला फॉलोअप करण्यास सांगितले जाईल. लॉग बुक एंट्री आणि बाटलीमध्ये सिरप झिंकचे प्रमाण याद्वारे अनुपालनाचे परीक्षण केले जाईल. सुधारित गर्भावस्थेच्या वयाच्या 40 व्या आठवड्यात- हॅमरस्मिथद्वारे मानववंशीय मोजमाप आणि न्यूरोलॉजिकल तपासणी नवजात न्यूरोलॉजिकल तपासणी केली जाईल. 3 महिन्यांच्या गर्भधारणेचे वय मानववंशीय मोजमाप दुरुस्त केल्यावर, हॅमरस्मिथ शिशुची न्यूरोलॉजिकल तपासणी आणि जस्त आणि तांबे पातळीचे रक्त नमुने घेतले जातील. हे चाचणी कालावधी पूर्ण करेल.

अभ्यासातील सहभागातून माघार घेणे: या अभ्यासात ऐच्छिक सहभाग. या अभ्यासात भाग घ्यायचा की नावनोंदणी झाल्यावर सहभाग सुरू ठेवायचा हे ठरवण्यासाठी तुम्ही मोकळे असाल. तुम्ही तुमचा सहभाग मागे घेण्याचा निर्णय घेतल्यास, तुम्ही तसे करण्यास मोकळे आहात. तथापि, कृपया मुख्य अन्वेषकांना निर्णय कळवा.

अभ्यासात सहभागी होण्याचे संभाव्य फायदे: या अभ्यासात भाग घेतल्याने तुम्हाला कोणतेही फायदे मिळणार नाहीत. गोळा केलेला डेटा मोठ्या प्रमाणावर लोकसंख्येला मदत करेल.

अभ्यासात सहभागी होण्यापासून संभाव्य धोके: या अभ्यासात सहभागी होण्यात कोणतेही धोके नाहीत. या विशिष्ट अभ्यासात वापरल्या जाणाऱ्या झिंक सप्लिमेंटेशनमुळे बाळावर कोणतेही दुष्परिणाम होणार नाहीत.

गोपनीयता आणि गोपनीयता: तुमच्याकडून संकलित केलेली माहिती कोणत्याही प्रकारची टाळण्यासाठी कोड केली जाईल
तुम्हाला ओळखण्यासाठी व्यक्ती. तुमची ओळख कधीच उघड होणार नाही. तुमच्याकडून गोळा केलेला डेटा गोपनीय ठेवला जाईल आणि केवळ प्रक्रिया केलेला किंवा एकत्रित केलेला डेटा प्रकाशनासाठी वापरला जाईल.
आर्थिक प्रोत्साहन: या अभ्यासात सहभागी होण्यासाठी तुम्हाला कोणतेही पेमेंट मिळणार नाही.

अभ्यासादरम्यान केलेल्या तपासणीचा खर्च मुख्य अन्वेषकाद्वारे दिला जाईल.

एकत्रित डेटाच्या प्रकाशनासाठी अधिकृतता: एकत्रित डेटाच्या प्रक्रियेनंतर प्राप्त झालेले परिणाम वैज्ञानिक हेतूसाठी प्रकाशित केले जातील आणि किंवा वैज्ञानिक गटांना सादर केले जातील. मात्र, तुमची ओळख कधीही उघड होणार नाही.

अभ्यास सहभागी म्हणून तुमच्या अधिकाराबाबत तुम्हाला काही प्रश्न किंवा तक्रारी असल्यास तुम्ही डॉ. हर्षा हेगडे, अध्यक्ष, जेएनएमसीच्या मानवी विषय संशोधनासाठी नैतिक समिती, ०८३१-२४७३७७७ विस्तार ४०५२ यांच्याशी संपर्क साधू शकता.

कायदेशीर अधिकार: या संमती फॉर्मवर स्वाक्षरी करून, आम्ही तुमचे कोणतेही कायदेशीर अधिकार सोडत नाही आहोत

CONSENT STATEMENT

"I am making a voluntary decision to participate in the study "**ASSESSMENT OF THE EFFECT OF TWO DIFFERENT DOSES OF ZINC SUPPLEMENTATION ON NEURODEVELOPMENT AND GROWTH IN LATE PRETERMS : A HOSPITAL BASED RANDOMISED CONTROLLED TRIAL".** My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction."

Name of the parent: _____

Signature or left thumb impression of the participant: _____

Name of the witness: _____

Signature or left thumb impression of the witness: _____

Name of the investigator: _____

Signature of the investigator: _____

Date: _____

Place: _____

सहमति वक्तव्य

“मैं इस अध्ययन में भाग लेने का एक स्वैच्छिक निर्णय ले रहा हूँ **“**न्यूरोडेवलपमेंट पर जिंक सप्लीमेंट की दो अलग-अलग खुराक के प्रभाव का आकलन और देर से पूर्व में वृद्धि**:** एक अस्पताल आधारित यादृच्छिक नियंत्रित परीक्षण **”** नीचे मेरे हस्ताक्षर इंगित करते हैं कि मैंने भाग लेने का निर्णय लिया है और मैंने ऊपर दी गई जानकारी को पढ़ लिया है या ऊपर प्रदान की गई जानकारी मुझे उस भाषा में पढ़ दी गई है जिसे मैं सबसे अच्छी तरह समझता हूँ। मुझे प्रश्न पूछने का अवसर दिया गया था और उनका उत्तर मेरी संतुष्टि के अनुसार दिया गया है।”

प्रतिभागी का नाम (माता-पिता): _____

प्रतिभागी के हस्ताक्षर या बाएं अंगूठे का निशान: _____

गवाह का नाम: _____

साक्षी के हस्ताक्षर या बाएं अंगूठे का निशान: _____

अन्वेषक का नाम: _____

अन्वेषक के हस्ताक्षर: _____

तारीख:_____

जगह:_____

ಸಮ್ಮತಿ ಹೇಳಿಕೆ

“ನಾನು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸ್ವಯಂಪ್ರೇರಿತ ನಿರ್ಧಾರವನ್ನು ಮಾಡುತ್ತಿದ್ದೇನೆ “ ನ್ಯೂರೋ ಡೆವಲಪ್ಮೆಂಟ್ ಮತ್ತು ಬೆಳವಣಿಗೆಯ ಮೇಲೆ ಎರಡು ವಿಭಿನ್ನ ಡೋಸ್ ಝಿಂಕ್ ಸಪ್ಲಿಮೆಂಟೇಶನ್ ಪರಿಣಾಮದ ಮೌಲ್ಯಮಾಪನ” ಕೆಳಗಿನ ನನ್ನ ಸಹಿ ನಾನು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದ್ದೇನೆ ಮತ್ತು ನಾನು ಮೇಲೆ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಓದಿದ್ದೇನೆ ಅಥವಾ ಮೇಲೆ ಒದಗಿಸಿದ ಮಾಹಿತಿಯನ್ನು ನನಗೆ ಚೆನ್ನಾಗಿ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ಓದಲಾಗಿದೆ ಎಂದು ಸೂಚಿಸುತ್ತದೆ. ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವನ್ನು ನೀಡಲಾಯಿತು ಮತ್ತು ಅವುಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ.”

ಭಾಗವಹಿಸುವವರ ಹೆಸರು (ತಾಯಿ/ ತಂದೆ): _____

ಭಾಗವಹಿಸುವವರ ಸಹಿ ಅಥವಾ ಎಡ ಹೆಬ್ಬೆರಳಿನ ಗುರುತು: _____

ಸಾಕ್ಷಿಯ ಹೆಸರು: _____

ಸಾಕ್ಷಿಯ ಸಹಿ ಅಥವಾ ಎಡ ಹೆಬ್ಬೆರಳಿನ ಗುರುತು: _____

ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: _____

ತನಿಖಾಧಿಕಾರಿಯ ಸಹಿ: _____

ದಿನಾಂಕ: _____

ಸ್ಥಳ: _____

संमती वधिान

“मी अभ्यासात सहभागी होण्याचा ऐच्छिक निर्णय घेत आहे “झिंक सप्लिमेंटेशनचा प्रभाव न्यूरोडेव्हलपमेंट आणि उशीरा मुदतीच्या वाढीवर - एक हॉस्पिटल आधारित यादृच्छिक नियंत्रित चाचणी” खाली माझी स्वाक्षरी दर्शवते की मी सहभागी होण्याचा निर्णय घेतला आहे आणि मी वर दिलेली माहिती वाचली आहे किंवा वर दिलेली माहिती मला चांगल्या प्रकारे समजेल अशा भाषेत वाचण्यात आली आहे. मला प्रश्न विचारण्याची संधी देण्यात आली आणि त्यांना माझ्या समाधानासाठी उत्तरे देण्यात आली.”

सहभागीचे नाव (आई/ वडील): _____

सहभागीची सही किंवा डाव्या अंगठ्याचा ठसा: _____

साक्षीदाराचे नाव: _____

साक्षीदाराची सही किंवा डाव्या अंगठ्याचा ठसा: _____

तपासकर्त्याचे नाव: _____

अन्वेषकाची स्वाक्षरी: _____

तारीख: _____

जागा: _____

ANNEXURE II - PROFORMA

KAHER's JNMC, BELAGAVI

DEPARTMENT OF PAEDIATRICS

ASSESSMENT OF THE EFFECT OF TWO DIFFERENT DOSES OF ZINC SUPPLEMENTATION ON NEURODEVELOPMENT AND GROWTH IN LATE PRETERMS : A HOSPITAL BASED RANDOMISED CONTROLLED TRIAL

Principal Investigator : _____

Guide : _____

Co-Guide: _____

Subject Serial No: _____ **Control Group/ Intervention Group**

Name of Infant : _____ Sex : male / female

Name of Mother:_____ Name of Father :_____

Date of Birth: _____ Gestational age:_____

IP Number :_____ Phone No:_____

ANTHROPOMETRIC ASSESSMENTS

Number	At Birth	At discharge	At 40 weeks of corrected Gestational age	At 3 months of corrected gestational age
Date of assessment				
Day of Life				
Corrected age				
Occipitofrontal circumference (cm)				
Weight (kg)				
Height (cm)				

BIOCHEMICAL INVESTIGATIONS

Serum Zinc at birth (<48h) : _____

Serum Zinc at 3 months corrected gestational age : _____

Serum Copper at 3 months corrected gestational age : _____

NEUROLOGICAL ASSESSMENT

Hammersmith Neonate Neurological Examination (40 weeks corrected gestational age): _____

Hammersmith Infant Neurological Examination (3 months corrected gestational age) :_____

[A] SOCIOECONOMIC DATA

Maternal age :_____

Maternal Education:_____

Presence of father at home:_____

Family Income per month: _____

[B] MATERNAL DATA- PREGNANCY AND BIRTH HISTORY

LMP : _____ EDD:_____

Singleton/ multiple gestation:_____

Mode of delivery : Normal vaginal delivery/ LSCS/ Assisted (Ventouse/ Forcep)

If Vaginal delivery - cephalic/ Breech :_____

If Cesarian section - indication_____

Liquor : Clear/ Meconium/ blood stained _____

Antenatal risk factors : _____

Antenatal Steroid intake (doses): _____

[C] INFANT DATA- RESUSCITATION DETAILS

Cried at birth : Yes/No Bag and mask ventilation : Yes/No

CPAP : Yes/No Intubated: Yes/No

APGAR SCORE: 1' _____ 5' _____ 10' _____

[D] GROWTH PARAMETERS AT BIRTH

Occipitofrontal circumference _____ cm _____centiles

Weight _____ g _____centiles

Length _____ cm _____centiles

[E] POSTURAL DEFORMITIES (acquired in utero or postnatally)

Skull: _____

Neck: _____

Body axis: _____

Upper limbs: Left _____Right_____

Lower limbs: Left _____Right_____

[F] MECHANICAL CONSEQUENCES OF BIRTH PROCESS

Caput succedaneum:_____ Cephalhematoma:_____ Severe cranial Moulding:_____

Facial ecchymosis: _____ Bruising from forceps (if extensive/ abnormal):_____

Facial paralysis: _____ Brachial plexus paralysis: _____

Hematomna of SCM:_____ Fracture of clavicle: _____

Others: (specify)_____

[F] ENTERAL FEEDS

Breast feeding started at:_____

Enteral supplementation started at :_____

[G] TREATMENT GIVEN

IV fluids : _____

Antibiotics : _____

Others : _____

[I] COMPLEMENTARY INVESTIGATIONS (if any, specify)

1.Cranial Ultrasound:_____

2.CT - Scan or MRI:_____

3.Optic Fundi:_____

4.EEG:_____

5.BERA:_____

6.Others:_____

[J] SIDE EFFECTS/ ADVERSE EFFECTS / MORBIDITIES

_____
_____
_____
_____
_____
_____
_____
_____
_____
_____
_____

HAMMERSMITH NEONATAL NEUROLOGICAL EXAMINATION

TONE PATTERN ITEMS

POSTURE	arms & legs extended or very slightly flexed	legs slightly flexed	leg well-flexed but not adducted	leg well flexed & adducted near to abdomen	abnormal posture: opisthotonus a) arms flexed, b) legs extended
ARM RECOIL	arms do not flex	arms flex slowly not always; not completely	arms flex slowly; more complete	arms flex quickly and completely	arms difficult to extend; snap back forcefully
ARM TRACTION	arms remain straight; no resistance	arms flex slightly or some resistance felt	arms flex well till shoulder lifts, then straighten	arms flex to approx. 100° & maintained as shoulder lifts	flexion of arms <100°; maintained when body lifts up
LEG RECOIL	No flexion	incomplete or variable flexion	complete but slow flexion	complete fast flexion	legs difficult to extend; snap back forcefully
LEG TRACTION	legs straight - no resistance	legs flex slightly or some resistance felt	legs flex well till bottom lifts up	knee flexes & remains flexed when bottom up	flexion stays when back & bottom up
POPLITEAL ANGLE	180°	≈ 150°	≈110°	≈90°	<90°
HEAD CONTROL (1)	no attempt to raise head	infant tries: effort better felt than seen	raises head but drops forward or back	raises head: remains vertical	
HEAD CONTROL (2)	no attempt to raise head	infant tries: effort better felt than seen	raises head but drops forward or back	raises head: remains vertical; it may wobble	head upright or extended; cannot be passively flexed
HEAD LAG	head drops & stays back	tries to lift head but it drops back	able to lift head slightly	lifts head in line with body	head in front of body
VENTRAL SUSPENSION	back curved, head & limbs hang straight	back curved, head ↓, limbs slightly flexed	back slightly curved, limbs flexed	back straight, head in line, limbs flexed	back straight, limbs above body

1	.5	2	.5	3	.5	4	.5	5	
3	0	9	6	60	9	12	0	1	25-27w
1	0	6	2	61	16	12	1	1	28-29w
2	0	4	2	65	17	8	0	2	30-31w
0	0	0	2	81	4	9	0	4	32-34w
0	0	0	0	6	3	90	1	0	Full term

3	1	9	9	44	9	23	2	0	25-27w
1	1	3	4	42	15	33	0	1	28-29w
1	0	8	3	42	10	36	0	0	30-31w
0	0	2	2	54	15	25	0	2	32-34w
0	0	5	2	22	3	67	1	0	Full term

3	0	17	5	51	10	14	0	0	25-27w
7	1	14	7	45	8	18	0	0	28-29w
7	2	15	4	51	7	14	0	0	30-31w
6	2	25	0	59	4	4	0	0	32-34w
0	0	1	0	22	8	69	0	0	Full term

3	0	14	4	18	5	52	0	4	25-27w
0	0	5	2	24	5	62	0	2	28-29w
0	0	10	2	34	2	50	0	2	30-31w
0	0	9	0	38	2	49	0	2	32-34w
0	0	3	1	4	1	91	0	0	Full term

3	1	17	6	35	6	27	1	4	25-27w
1	1	17	2	36	6	35	1	1	28-29w
2	0	21	8	38	5	25	0	1	30-31w
0	4	29	10	43	2	10	0	2	32-34w
0	0	0	1	12	12	72	0	3	Full term

3	0	22	8	46	6	14	0	0	25-27w
5	1	16	5	48	7	17	1	0	28-29w
2	0	15	10	53	5	15	0	0	30-31w
2	0	26	4	49	4	13	0	2	32-34w
0	0	5	5	19	20	51	0	0	Full term

3	0	17	4	46	9	21	0	0	25-27w
0	0	13	5	46	12	24	0	0	28-29w
3	0	14	2	48	13	20	0	0	30-31w
4	0	15	4	55	4	18	0	0	32-34w
0	0	0	6	26	12	56	0	0	Full term

3	0	3	5	57	11	21	0	0	25-27w
1	2	6	4	50	13	24	0	0	28-29w
1	0	2	2	63	11	21	0	0	30-31w
0	0	4	2	77	2	15	0	0	32-34w
0	0	0	4	29	15	52	0	0	Full term

3	3	27	13	36	3	15	0	0	25-27w
3	3	18	7	40	14	15	0	0	28-29w
7	3	16	5	46	7	16	0	0	30-31w
4	0	21	4	56	0	15	0	0	32-34w
0	0	9	4	44	12	31	0	0	Full term

0	0	21	11	38	11	15	4	0	25-27w
3	0	25	8	44	8	10	0	2	28-29w
3	0	22	8	47	5	14	1	0	30-31w
2	0	17	2	56	2	19	0	2	32-34w
0	0	4	5	47	16	28	0	0	Full term

FLEXOR TONE (compare arm and leg traction)		arm flexion < leg flexion	arm flexion = leg flexion	arm flexion > leg flexion; difference ≤1 column	arm flexion > leg flexion; difference > 1 colum n
FLEXOR TONE (resting posture)			arms and legs generally flexed	strong arm flexion with strong leg extension *intermittent*	strong arm flexion with strong leg extension *continuous*
LEG TONE (leg traction and popliteal angle)		leg traction > popliteal angle	leg traction = popliteal angle	leg traction < popliteal angle; difference ≤1 column	leg traction < popliteal angle; difference > 1 colum n
HEAD CONTROL (sitting)		neck extension < neck flexion	neck extension = Neck flexion	neck extension > neck flexion; difference ≤ 1 column	neck extension > neck flexion; difference > 1 colum n
NECK AND AXIAL TONE (horizontal)		ventral suspension < head lag	ventral suspension = head lag	ventral suspension > head lag; difference ≤1 column	ventral suspension > head lag; difference > 1 colum n

1	.5	2	.5	3	.5	4	.5	5	
0	0	45	0	27	<1	27	0	1	**25-27w**
0	0	40	<1	40	0	20	<1	0	**28-29w**
0	0	34	<1	47	<1	18	0	1	**30-31w**
0	0	38	<1	36	<1	24	<1	2	**32-34w**
0	0	25	3	53	0	18	0	<1	**Full term**

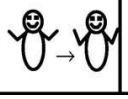
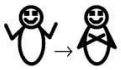
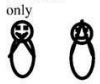
0	0	0	0	99	<1	0	0	1	**25-27w**
0	0	0	0	96	<1	3	0	1	**28-29w**
0	0	0	0	96	<1	2	0	2	**30-31w**
0	0	0	0	94	<1	2	0	4	**32-34w**
0	0	0	0	99	0	<1	0	<1	**Full term**

0	0	43	<1	34	0	21	<1	1	**25-27w**
0	0	41	0	39	<1	19	0	1	**28-29w**
0	0	38	0	36	<1	22	<1	4	**30-31w**
0	0	19	<1	50	<1	29	<1	2	**32-34w**
0	0	4	0	57	0	35	0	1	**Full term**

0	0	25	0	64	0	9	0	2	**25-27w**
0	0	17	0	70	0	13	0	0	**28-29w**
0	0	18	0	76	0	6	0	0	**30-31w**
0	0	23	0	64	0	13	0	0	**32-34w**
0	0	3	0	94	0	3	0	<1	**Full term**

0	0	20	0	39	0	35	0	6	**25-27w**
0	0	31	0	42	0	26	0	1	**28-29w**
0	0	24	0	49	0	26	0	1	**30-31w**
0	0	17	0	51	0	28	0	4	**32-34w**
0	0	24	0	58	0	18	0	<1	**Full term**

REFLEX ITEMS

TENDON REFLEX	absent	felt, not seen	seen	'exaggerated'	clonus
SUCK/GAG	no gag / no suck	weak irregular suck only: no stripping	weak regular suck some stripping	strong suck: (a) irregular (b) regular good stripping	no suck but strong clenching
PALMAR GRASP	no response R L	short, weak flexion of fingers R L	strong flexion of fingers R L	strong finger flexion, shoulder ↑ R L	very strong grasp; infant can be lifted off couch R L
PLANTAR GRASP	no response R L	partial plantar flexion of toes R L	toes curve around the examiner's finger R L		
PLACING	no response R L	dorsi-flexion of ankle only R L	full placing response with flexion of hip, knee & placing sole on surface R L		
MORO REFLEX	no response or opening of hands only	full abduction at shoulder and extension of the arms; no adduction 	full abduction but only delayed or partial adduction 	partial abduction at shoulder and extension of arms followed by smooth adduction 	• no abduction or adduction; • only forward extension of arms from the shoulders • marked adduction only  or

1	.5	2	.5	3	.5	4	.5	5	
0	0	9	0	55	7	13	3	13	**25-27w**
0	0	12	0	50	7	22	4	5	**28-29w**
0	0	24	1	52	1	13	0	9	**30-31w**
0	0	18	0	57	0	17	4	4	**32-34w**
<1	0	21	0	78	0	<1	0	<1	**Full term**

0	0	1	0	3	3	93	0	0	**25-27w**
0	0	3	0	7	0	90	0	0	**28-29w**
0	0	0	0	6	2	92	0	0	**30-31w**
0	0	4	0	10	0	86	0	0	**32-34w**
0	0	1	0	5	0	92	0	2	**Full term**

0	0	5	0	47	7	30	1	10	**25-27w**
0	0	3	1	40	8	43	1	4	**28-29w**
0	0	1	0	51	3	35	0	10	**30-31w**
0	0	7	0	53	3	30	0	7	**32-34w**
<1	0	6	0	84	0	9	0	<1	**Full term**

0	0	4	1	95	0	0	0	0	**25-27w**
0	1	5	2	92	0	0	0	0	**28-29w**
0	0	2	1	97	0	0	0	0	**30-31w**
0	0	2	2	96	0	0	0	0	**32-34w**
<1	0	2	0	98	0	0	0	0	**Full term**

5	2	12	3	78	0	0	0	0	**25-27w**
0	2	12	6	80	0	0	0	0	**28-29w**
1	0	8	8	83	0	0	0	0	**30-31w**
0	0	4	0	96	0	0	0	0	**32-34w**
1	0	18	0	81	0	0	0	0	**Full term**

0	0	13	1	61	4	20	0	1	**25-27w**
0	0	12	1	64	6	15	1	1	**28-29w**
0	0	12	1	51	3	28	0	5	**30-31w**
0	0	23	0	46	2	27	0	2	**32-34w**
0	0	1	0	20	0	79	0	0	**Full term**

MOVEMENTS AND ABNORMAL SIGNS

a

SPONTANEOUS MOVEMENT (quantity)	no movement	sporadic and short isolated movements	frequent isolated movements	frequent generalized movements	continuous exaggerated movements
SPONTANEOUS MOVEMENT (quality)	only stretches	stretches and random abrupt movements Some smooth movements	fluent movements but mono-tonous	fluent alternating movements of arms + legs; good variability	cramped synchr-onous mouthing jerky or other abnormal movement
HEAD RAISING	no movement	infant rolls head over, chin not raised	infant raises chin, rolls head over	infant brings head and chin up	infant brings head up and keeps it up

1	.5	2	.5	3	.5	4	.5	5	
0	0	15	3	28	3	51	0	0	25-27w
0	0	17	3	26	11	43	0	0	28-29w
0	0	13	0	31	8	48	0	0	30-31w
0	0	20	0	27	0	51	0	2	32-34w
<1	0	3	0	5	0	92	0	<1	Full term

0	0	16	4	42	11	23	1	3	25-27w
0	0	22	5	35	1	23	2	2	28-29w
0	0	20	6	34	2	36	0	2	30-31w
0	0	21	0	15	0	60	0	4	32-34w
2	0	5	0	<1	0	93	0	<1	Full term

0	0	36	6	34	6	14	1	3	25-27w
1	1	35	4	34	9	14	1	1	28-29w
1	1	40	5	28	1	21	1	2	30-31w
0	0	40	0	30	4	22	2	2	32-34w
<1	0	10	0	50	0	40	0	<1	Full term

b

ABN. HAND OR TOE POSTURES		hands open, toes straight most of the time	intermittent fisting or thumb adduction	continuous fisting or thumb adduction; index finger flexion, thumb opposition	continuous big toe extension or flexion of all toes
TREMOR		no trem or or trem or onl y when cry ing	tremor only after Moro or occasionally when awake	frequent tremors when awake	continuous tremors
STARTLE	no startle even to sudden noise	no spontan -eous startle but reacts to sudden noise	2-3 spontaneous startles	more than 3 spontaneous startles	continuous startles

1	.5	2	.5	3	.5	4	.5	5	
0	0	57	4	37	0	2	0	0	25-27w
0	0	64	6	28	0	2	0	0	28-29w
0	0	67	1	30	1	1	0	0	30-31w
0	0	75	2	21	0	2	0	0	32-34w
0	0	85	0	12	0	3	0	<1	Full term

0	0	43	1	29	8	16	0	3	25-27w
0	0	43	0	27	9	19	2	0	28-29w
0	0	54	0	24	3	19	0	0	30-31w
0	0	62	0	30	0	4	0	4	32-34w
0	0	88	0	12	0	<1	0	<1	Full term

22	0	40	7	20	1	10	0	0	25-27w
23	1	35	7	30	2	2	0	0	28-29w
37	1	32	1	25	1	3	0	0	30-31w
50	0	35	0	9	0	6	0	0	32-34w
<1	0	94	0	6	0	<1	0	<1	Full term

BEHAVIORAL SIGNS, VISION, HEARING

EYE APPEARANCE	**does not open eyes**		**full conjugated eye mov**	**transient nystagmus strabismus roving eye movemens sunsetting sign**	**persistent nystagmus strabismus roving eye movements dow nward deviation**
AUDITORY ORIENTATION	**no reaction**	**auditory startle; Brightens and stills; no true orientati - on**	**shifting of eyes, head might turn towards source**	**prolonged head turn to stimulus; search with eyes; smooth**	turns head and eyes towards noise every time; jerky abrupt
VISUAL ORIENTATION	**does not follow or focus on stimuli**	**stills, focuses follows briefly to the side but loses stimuli**	**follows horizontal -ly and vertically; no head turn**	**follows horizon -tally and vertically; turns head**	follows in a circle
ALERTNESS	**will not respond to stimuli**	**when awake, looks only briefly**	**when awake, looks at stimuli but loses them**	**keeps interest in stimuli**	does not tire (hyper-reactive)
IRRITABILITY	**quiet all the time, not irritable to any stimuli**	**awakes, cries some -times when handled**	**cries often when handled**	**cries always when handled**	cries even when not handled
CONSOLABILITY	**not crying consoling not needed**	**cries briefly; consol -ing not needed**	**cries; becomes quiet when talked to**	**cries; needs picking up to console**	cries cannot be consoled
CRY	**no cry at all**	**whimpe -ring cry only**	**cries to stimuli but normal pitch**		high pitched cry; often continuous

1	.5	2	.5	3	.5	4	.5	5	
6	0	0	0	74	4	16	0	0	25-27w
2	0	0	0	80	2	15	1	0	28-29w
5	0	0	0	80	2	13	0	0	30-31w
4	0	0	0	87	2	7	0	0	32-34w
7	0	0	0	92	0	1	0	<1	Full term

5	1	28	0	57	1	8	0	0	25-27w
2	0	23	10	50	6	9	0	0	28-29w
5	1	27	7	51	1	8	0	0	30-31w
3	0	14	0	73	3	7	0	0	32-34w
<1	0	30	0	50	0	20	0	<1	Full term

6	0	7	2	25	3	26	9	22	25-27w
0	0	7	1	33	7	21	15	16	28-29w
1	0	9	0	27	5	25	10	23	30-31w
0	0	10	0	42	10	38	0	0	32-34w
<1	0	7	0	41	0	51	0	1	Full term

6	0	22	1	48	3	20	0	0	25-27w
1	0	17	4	60	3	14	1	0	28-29w
0	0	21	1	43	2	33	0	0	30-31w
0	0	7	3	54	0	36	0	0	32-34w
1	0	2	0	48	0	49	0	<1	Full term

12	1	52	0	31	0	3	0	1	25-27w
16	2	47	2	27	1	5	0	0	28-29w
27	0	47	1	22	0	2	0	1	30-31w
23	0	49	0	23	0	5	0	0	32-34w
<1	0	93	0	5	0	2	0	<1	Full term

10	0	29	0	29	3	29	0	0	25-27w
17	1	19	2	29	7	22	1	2	28-29w
27	0	18	0	28	2	22	1	2	30-31w
23	0	9	0	32	2	28	0	6	32-34w
1	0	41	0	45	0	12	0	<1	Full term

11	0	11	0	78	0	0	0	0	25-27w
16	0	5	2	77	0	0	0	0	28-29w
26	1	3	1	69	0	0	0	0	30-31w
23	0	6	2	69	0	0	0	0	32-34w
<1	0	7	0	92	0	0	0	1	Full z

HAMMERSMITH INFANT NEUROLOGICAL EXAMINATION (v 08.02.19)

Name **Date of birth**

Gestational age **Date of examination**

Chronological age / Corrected age **Head circumference**

Name of assessor

SUMMARY OF EXAMINATION
Global score (max 78)
Number of asymmetries
Behavioural score (not part of the optimality score)

Cranial nerve function	**score**	**(max 15)**
Posture	**score**	**(max 18)**
Movements	**score**	**(max 6)**
Tone	**score**	**(max 24)**
Reflexes and reactions	**score**	**(max 15)**

COMMENTS

(Throughout the exam, if a response is not optimal but not poor enough to score 1, give a score of 2)

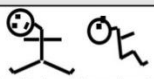
NEUROLOGICAL EXAMINATION

ASSESSMENT OF CRANIAL NERVE FUNCTION

	score 3	2	score 1	score 0	score	Asymmetry / Comments
Facial appearance (at rest and when crying or stimulated)	Smiles or reacts to stimuli by closing eyes and grimacing		Closes eyes but not tightly, poor facial expression	Expressionless, does not react to stimuli		
Eye movements	Normal conjugate eye movements		**Intermittent** Deviation of eyes or abnormal movements	**Continuous** Deviation of eyes or abnormal movements		
Visual response Test ability to follow a black/white target	Follows the target in a complete arc		Follows target in an incomplete or asymmetrical arc	Does not follow the target		
Auditory response Test the response to a rattle	Reacts to stimuli from both sides		Doubtful reaction to stimuli or asymmetry of response	No response		
Sucking/swallowing Watch infant suck on breast or bottle. If older, ask about feeding, assoc. cough, excessive dribbling	Good suck and swallowing		Poor suck and/or swallow	No sucking reflex, no swallowing		

1

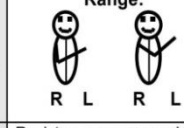
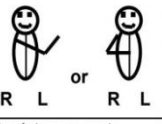
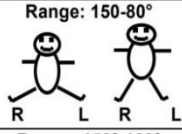
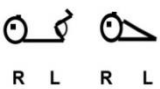
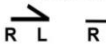
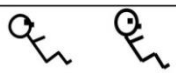
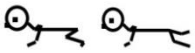
ASSESSMENT OF POSTURE (note any asymmetries)

	score 3	score 2	score 1	score 0	sc	Asymmetry / comments
Head in sitting	 Straight; in midline		 Slightly to side *or* backward *or* forward	 Markedly to side *or* backward *or* forward		
Trunk in sitting	 Straight		 Slightly curved or bent to side	 Very rounded rocketing back bent sideway		
Arms at rest	In a neutral position, central straight or slightly bent		**Slight** internal rotation *or* external rotation **Intermittent** dystonic posture	**Marked** internal rotation *or* external rotation or dystonic posture hemiplegic posture		
Hands	Hands open		**Intermittent** adducted thumb *or* fisting	**Persistent** adducted thumb *or* fisting		
Legs in sitting in supine and in standing	Able to sit with a straight back and legs straight or slightly bent (long sitting)  Legs in neutral position straight *or* slightly bent	**Slight** internal rotation or external rotation	Sit with straight back but knees bent at 15-20 °  Internal rotation *or* external rotation at the hips	Unable to sit straight unless knees markedly bent (no long sitting)  **Marked** internal rotation *or* external rotation *or* fixed extension or flexion or contractures at hips and knees		
Feet in supine and in standing	Central in neutral position Toes straight midway between flexion and extension		**Slight** internal rotation *or* external rotation **Intermittent** Tendency to stand on tiptoes or toes up or curling under	**Marked** internal rotation *or* external rotation at the ankle **Persistent** Tendency to stand on tiptoes *or* toes up or curling under		

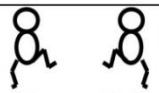
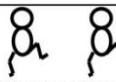
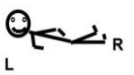
ASSESSMENT OF MOVEMENTS

	Score 3	Score 2	Score 1	Score 0	score	Asymmetry / comments
Quantity Watch infant lying in supine	Normal		Excessive or sluggish	Minimal or none		
Quality Observe infant's spontaneous voluntary motor activity during the course of the assessment	Free, alternating, and smooth		Jerky Slight tremor	• Cramped & synchronous • Extensor spasms • Athetoid • Ataxic • Very tremulous • Myoclonic spasm • Dystonic movement		

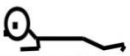
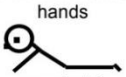
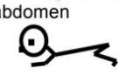
ASSESSMENT OF TONE

	Score 3	Score 2	Score 1	Score 0	sc	Asym/Co
Scarf sign Take the infant's hand and pull the arm across the chest until there is resistance. Note the position of the elbow in relation to the midline.	**Range:** 					
Passive shoulder elevation Lift arm up alongside infant's head. Note resistance at shoulder and elbow.	Resistance overcomeable 	Resistance difficult to overcome	No resistance 	Resistance, not overcomeable 		
Pronation/supination Steady the upper arm while pronating and supinating forearm, note resistance	Full pronation and supination, no resistance		Resistance to full pronation / supination overcomeable	Full pronation and supination not possible, marked resistance		
Hip adductors With both the infant's legs extended, abduct them as far as possible. The angle formed by the legs is noted.	**Range: 150-80°** 	**150-160°** 	**>170°** 	**<80°** 		
Popliteal angle Keeping the infant's bottom on the bed, flex both hips onto the abdomen, then extend the knees until there is resistance. Note the angle between upper and lower leg.	**Range: 150°-100°** 	**150-160°** 	**~90° or > 170°** 	**<80°** 		
Ankle dorsiflexion With knee extended, dorsiflex the ankle. Note the angle between foot and leg.	**Range: 30°-85°** 	**20-30°** 	**<20°or 90°** 	**> 90°** 		
Pull to sit Pull infant to sit by the wrists. (support head if necessary)						
Ventral suspension Hold infant horizontally around trunk in ventral suspension; note position of back, limbs and head.						

REFLEXES AND REACTIONS

	Score 3	Score 2	Score 1	Score 0	sc	Asym / Co
Arm protection Pull the infant by one arm from the supine position (steady the contralateral hip) and note the reaction of arm on opposite side.	 Arm & hand extend R L		 Arm semi-flexed R L	 Arm fully flexed R L		
Vertical suspension hold infant under axilla making sure legs do not touch any surface – you may "tickle" feet to stimulate kicking.	 Kicks symmetrically		 Kicks one leg more or poor kicking	 No kicking even if stimulated or scissoring		
Lateral tilting (describe side up). Hold infant up vertically near to hips and tilt sideways towards the horizontal. Note response of trunk, spine, limbs and head.						
Forward parachute Hold infant up vertically and quickly tilt forwards. Note reaction /symmetry of arm responses,	 (after 6 months)		 (after 6 months)			
Tendon Reflexes Have child relaxed, sitting or lying – use small hammer	Easily elicitable biceps knee ankle	Mildly brisk bicep knee ankle	Brisk biceps knee ankle	Clonus or absent biceps knee ankle		

SECTION 2 MOTOR MILESTONES (not scored; note asymmetries)

Head control	Unable to maintain head upright normal to 3m	Wobbles normal up to 4m	Maintained upright all the time normal from 5m			**Please note age at which maximum skill is achieved**
Sitting	Cannot sit	With support at hips  normal at 4m	Props  normal at 6m	Stable sit  normal at 7-8m	Pivots (rotates)  normal at 9m	Observed: Reported (age):
Voluntary grasp – note side	No grasp	Uses whole hand	Index finger and thumb but immature grasp	Pincer grasp		Observed: Reported (age):
Ability to kick in supine	No kicking	Kicks horizontally but legs do not lift	Upward (vertically)  normal at 3m	Touches leg  normal at 4-5m	Touches toes  normal at 5-6m	Observed: Reported (age):
Rolling - note through which side(s)	No rolling	Rolling to side normal at 4m	Prone to supine normal at 6 m	Supine to prone normal at 6 m		Observed: Reported (age):
Crawling - note if bottom shuffling	Does not lift head	On elbows  normal at 3m	On outstretched hands  normal at 4m	Crawling flat on abdomen  normal at 8m	Crawling on hands and knees  normal at 10m	Observed: Reported (age):
Standing	Does not support weight	Supports weight normal at 4m	Stands with support normal at 7m	Stands unaided normal at 12m		Observed: Reported (age):
Walking		Bouncing normal at 6m	Cruising (walks holding on) normal at 12m	Walking independently normal by 15m		Observed: Reported (age):

SECTION 3 BEHAVIOUR (not scored)

	1	2	3	4	5	6	Comment
Conscious state	Unrousable	Drowsy	Sleep but wakes easily	Awake but no interest	Loses interest	Maintains interest	
Emotional state	Irritable, not consolable	Irritable, carer can console	Irritable when approached	Neither happy or unhappy	Happy and smiling		
Social orientation	Avoiding, withdrawn	Hesitant	Accepts approach	Friendly			

This is the official form for use with the Hammersmith Infant Neurological Examination.
Its content and scoring system are not to be changed. ***Main reference*** *Haataja L et al J Peds 1999;135:153-61*
For enquiries about the examination*, please contact Prof Frances Cowan f.cowan@imperial.ac.uk,*
Prof Leena Haataja leena.haataja@hus.fi or Prof Eugenio Mercuri eugeniomercuri@unicatt.it
Websi*te hammersmith-neuro-exam.com*

ANNEXURE III – MASTER CHART

SL No:	Study	Infant name	Fathers name	Date of birth	IP No	HRB No	Sex	Phone number	Maternal age	Maternal education	Family income per month (k)	LMP	EDD	Singleton/multiple	Mode of delivery	Vaginal- cephalic/ breech	Caesarean indication	Liquor- clear/ meconium/ blood	Antenatal risk factor	Antenatal steroid doses	Cried at birth	Bag and mask ventilation	CPAP	APGAR-1	APGAR-5	APGAR-10	Postural deformity	Mechanical consequences of Birth Process	Breast feeding started at	Enteral supplementation started at	Date of birth	Corrected age	Birth OFC	Birth weight	Birth height	Date of assessment at discharge	Day of life at discharge	Corrected age at discahrge	Discharge OFC	Discharge weight	Discharge height	Date of assessment at 40w CGA	Day of life at 40w CGA	OFC at 40w CGA	Weight at 40w CGA	Height at 40w CGA	Date of assessment at 3 months of CGA	Day of life at 3monthCGA	OFC at 3 monthCGA	Weight at 3 months CGA	Height at 3 months CGA	Serum zinc at birth	Serum zinc at 3 months CGA	Serum copper at 3 months CGA	HNNE at 40w CGA	HINE at 3 months CGA
1	T	B/o Trupti 1	Ramakrishna Kadannavar	03-07-23	1199898	2563	Female	9449058819	40	LLB	200	26-10-22	2/8/23	Multiple	LSCS		PIH, PPROM	Clear	PIH, hypothyroidism, PPROM	1	Yes	No	No	8	9	NA	None	None	2	4	03-07-23	34w	29	1600	41	12/7/23	8	34w+2d	30	1560	42	1/8/23	33	33	2180	44	1212/23	3m+2w	39	5200	59	58	112	98.4	30	71
2	T	B/o Trupti 2	Ramakrishna Kadannavar	03-07-23	1199898	2564	Female	9449058819	40	LLB	200	26-10-22	2/8/23	Multiple	LSCS		PIH, PPROM	Clear	PIH, hypothyroidism, PPROM	1	Yes	No	No	8	9	NA	None	None	2	4	03-07-23	34w	33	2480	47	12/7/23	8	34w+2d	34	2420	48	11/8/23	33	35	3100	50	12/12/23	3m+2w	40	6480	64	64	134	88.9	32	73
3	T	B/o Renuka	Rupesh Gurav	06-07-23	1200930	2763	Male	9945010300	28	Bcom	40	16-11-22	23/8/23	Singleton	LSCS	Cephalic	Prev 2 LSCS	Clear	HTN	2	Yes	No	Labour Room	7	9	NA	None	None	2	4	06-07-23	34w+3d	32	2000	43	18/7/23	13	36w+2d	33	1925	45	25/8/23	42	36	3220	50	24/11/23	3m	40	5320	59	65	117	99.9	30	70
4	C	B/o Kirthi	Parashuram Lamani	18-07-23	1203760	2593	Female	8147977318	26	10th	15	12-11-22	19/8/23	Sinlgleton	LSCS	Cephalic	Severe PE, oligo	Clear	PE	1	Yes	No	No	7	8	NA	None	None	1	3	18-07-23	35w+6d	30	1650	47	30/7/23	12	37w+4d	31	1645	48	22/8/23	35	33	2200	49	29/11/23	3m+ 1w	40	4650	55	87	89	137	30	69
5	C	B/o Jyoti 1	Yallapa Medar	11-08-23	1209193	2617	Male	9902468592	26	BA	24	12/12/22	14/9/23	Multiple	LSCS		Oligo	Clear	Twins	2	Yes	No	No	8	9	NA	None	None	1	3	11-08-23	35w	32	2400	47	22/8/23	11	36w+5d	33	2510	48	22/9/23	37	35	3520	51	23/12/24	3m +1w	41	5940	57	80	92	150	28	64
6	C	B/o Jyoti 2	Yallapa Medar	11-08-23	1209194	2618	Male	9902468592	26	BA	24	12/12/22	14/9/23	Multiple	LSCS		Oligo	Clear	Twins	2	Yes	No	No	8	9	NA	None	None	1	3	11-08-23	35w	33	1900	45	22/8/23	11	36w+5d	33	1750	46	22/9/23	37	35	3420	50	23/12/24	3m +1w	42	6100	58	68	90.3	172	28	68
7	T	B/o Sujata 1	Vigneshwar	24-08-23	10002443	2626	Male	8904048688	35	Engineer	200	26-12-22	2/10/23	Multiple	LSCS		Twins with AEDF	Clear	HTN	1	No	2 cycle	1	4	7	8	None	None	2	4	24-08-23	34w+2d	32	2060	45	12/9/23	18	37w	32	2020	46	3/10/23	42	34	2860	49	2/2/24	3m+3w	42	5320	63	52	125	143	27	73
8	T	B/o Sujata 2	Vigneshwar	24-08-23	10002447	2625	Male	8904048688	35	Engineer	200	26-12-22	2/10/23	Multiple	LSCS		Twins with AEDF	Clear	HTN	1	Yes	No	No	8	9	NA	None	None	2	4	24-08-23	34w+2d	27	1140	38	12/9/23	18	37w	28	1270	39	3/10/23	42	32	2080	44	2/2/24	3m+3w	41	5180	60	55	118	151	28	72
9	C	B/o Kaveri	Suraj Halagi	12-09-23	1006759	2874	Male	8951810666	28	Mcom	90	15/1/23	22/10/23	Singleton	LSCS	Cephalic	PPROM, MSL	Meconium	PPROM	1	Yes	No	1	7	9	NA	None	None	2	4	12-09-23	34w	30	1710	40	22/9/23	11	35w+3d	31	1740	43	27/10/23	49	34	2460	46	12/2/24	3m+3w	39	4320	57	60	100	149	30	66
10	T	B/o Sameena banu	Imtiaz Sayyed	14-09-23	10000387	2614	Male	9535815858	34	Engineer	120	6/12/22	12/9/23	Singleton	LSCS	Cephalic	Prev 2 LSCS	Clear	PIH	2	Yes	No	Labour room	7	8	NA	None	None	1	3	14-09-23	34w+5d	31	1500	40	29/8/23	15	36w+6d	32	1560	41	23/9/23	39	34	1960	48	28/12/23	3m+4d	43	5240	58	56	103	159	28	68
11	C	B/o Nagatai	Basaveshwar	16-09-23	10007898	2768	Female	8901367889	26	12th	15	5/11/23	12/10/23	Singleton	LSCS	Cephalic	Severe PE, EOF|GR	Clear	Severe PE	2	Yes	No	No	7	9	NA	None	None	2	4	16-09-23	36w+2d	30	1800	44	30/9/23	14	38w+2d	31	1780	45	15/10/23	29	33	3100	47	1/2/24	3m+2w	38	5010	54	71	91..4	142	30	68
12	T	B/o Priyanka	Vasant terdal	17-09-23	10007953	2912	Male	7349242507	30	BA	50	23-1-23	30/10/23	Singleton	NVD	Cephalic	AEDF	Clear	HTN	2	No	1 cycle	Labour Room	4	7	8	None	None	2	5	17-09-23	34w	29	1680	41	6/10/23	24	37w+3	30	1670	42	31/10/23	44	31	1880	43	19/1/24	3m+3w	39	4700	54	61	97.5	150	30	71
13	C	B/o Reya	Rahul Patil	27-09-23	10010162	2871	Male	6364495592	32	BA	45	17-2-23	24/11/23	Singleton	LSCS	Cephalic	MSL	Meconium		0	Yes	No	1	7	8	NA	None	None	2	4	27-09-23	34w	31	1800	45	2/10/23	5	34w+5d	32	1700	45	24/11/23	35	35	2700	47	3/3/24	3m+2w	40	5820	54	71	91.9	129	29	68
14	C	B/o Rekha	Mallikarjun BM	07-10-23	10012344	2766	Male	7337786447	30	BA	75	4/2/23	12/11/23	Singleton	LSCS	Cephalic	HTN, IVF, Short stature	Clear	HTN, IVF, Short stature	2	Yes	No	No	7	8	NA	None	None	2	4	07-10-23	34w	30	1710	44	12/11/23	25	37w+1d	31	1780	45	17/11/23	36	32	2000	45	1/4/24	3m+2w	41	5100	57	74	100	138	39	69
15	C	B/o Kavita	Vinodkumar Guruvannavar	09-10-23	10012464	2663	Male	8431861166	24	12th	10	3-2-23	10/11/23	Singleton	NVD	Cephalic	Oligo	Meconium	PPROM 22h, PTB on ATT 2 m	2	yes	No	1	7	8	NA	None	None	2	4	09-10-23	35w	32	2030	46	15/10/23	7	35w+6d	32	2060	47	13/11/23	36	33	2340	49	15/2/24	4m+1w	38	4510	54	69	99.4	142	27	65
16	C	B/o Soumya	Nagayya Kalmath	14-10-23	10014026	2665	Female	9901137835	28	BCom	60	10-2-23	17/11/23	Singleton	LSCS	Cephalic	Severe PE	Clear	PE	1	Yes	No	No	7	8	NA	None	None	2	4	14-10-23	35w	31	2200	45	23/10/23	9	36w+2d	31	2200	47	17/11/23	34	35	2200	47	3/3/24	3m+2w	41	5120	54	64	80.4	142	29	70
17	C	B/o Pranita	Sagar	14-10-23	10013934	2900	Male	8951376230	30	12th	20	30-1-23	6/11/23	Singleton	NVD	Cephalic	NRNST	Meconium	HTN	2	yes	No	No	8	9		None	None	1	3	14-10-23	36w+3d	29	2100	42	19/10/23	6	37w+2	30	1780	43	7/11/23	25	33	2060	44	12/3/24	3m	40	5640	58	70	103	132	30	66
18	T	B/o Pooja	Mahesh Shirahatti	20-10-23	10010926	2668	Female	7829678289	31	BSc	100	27/1/23	3/11/23	Singleton	LSCS	Cephalic	Prev LSCS	Clear	HTN	0	Yes	No	No	6	8	9	None	None	2	4	20-10-23	35w+3d	35	2460	48	8/10/23	6	36w+2d	35	2400	49	3/1/23	27	36	3.42	50	1/2/24	3m	40	5420	56	71	94.8	143	27	68
19	C	B/o Ayesha	Shubani Sandi	21-10-23	10015634	2899	Female	9538412526	24	BCom	75	10-2-23	17/11/23	Singleton	LSCS	Cephalic	AEDF	Clear	LOFGR, AEDF	1	Yes	No	No	7	8	NA	None	None	2	4	21-10-23	35w+4d	31	1910	43	27/10/23	6	36w+3d	31	1780	44	17/11/23	30	33	3100	47	3/3/24	3m+2w	40	5680	54	70	92	135	30	69
20	C	B/o Pradnya	Dhirendra Haridas Patil	27-10-23	10016564	2877	Male	831023882	32	BA	100	24/2/23	1/12/23	Singleton	NVD	Cephalic	Oligo with cord around neck	Meconium	None	0	Yes	No	1	6	8	NA	None	None	2	4	27-10-23	35w	27	1500	37	10/11/23	14	37w	28	1460	39	5/12/23	21	30	1740	42	26/3/24	3m+3w	34	3060	51	56	100	111	27	66
21	T	B/o Renuka T1	Gavadi Rajkumar	02-11-23	10018960	2688	Male	9844906285	36	MBA	120	24/2/23	01/12/23	Multiple	LSCS		Twins	Clear	Ectopic prev	2	Yes	No	Labour Room	7	8	NA	None	None	1	4	02-11-23	35w+6d	31	2200	44	17/11/23	15	38w	31	1860	45	1/12/24	29	32	1900	46	21/3/24	3m+3w	42	5020	59	68	121	90.2	32	72
22	T	B/o Renuka T2	Gavadi Rajkumar	02-11-23	10018047	2687	Male	9844906285	36	MBA	120	24/2/23	01/12/23	Multiple	LSCS		TWins	Clear	Ectopic Prev	2	Yes	No	No	8	9	NA	None	None	1	4	02-11-23	35w +6d	28	1250	38	17/11/23	15	38w	29	1210	38	1/2/24	29	30	1300	40	21/3/24	3m +3w	40	4600	58	69	98.3	119	31	73
23	C	B/o Aishwarya	Rahul Patil	11-11-23	10019979	2812	Female	9731426375	26	BCom	80	23/3/23	28/01/23	Singleton	NVD	Cephalic		Clear	PPROM	1	Yes	No	1	6	7	8	None	None	2	4	11-11-23	34w+1d	30	1820	44	17/11/23	6	35w	31	1780	44	26/12/23	32	31	3320	49	14/4/24	3m+3w	40	5070	55	72	91.6	128	30	70
24	C	B/o Sushma	Nandish Rotti	15-11-23	10020473	2877	Male	9972177216	24	BA	70	25/2/23	8/12/23	Multiple	LSCS	Cephalic	Singleton	Clear	Prev LSCS	2	Yes	No	No	7	8	NA	None	None	2	4	15-11-23	36w	33	2100	48	19/11/23	4	36w+4d	33	2080	48	8/12/23	28	35	3260	51	1/4/24	3m+3w	41	5840	59	74	94.9	147	30	68
25	T	B/o Komal 1	Raju dhamnekar	02-12-23	10024431	2738	Male	7353347981	26	12th	25	7-4-23	12/1/24	Multiple	LSCS		Twins	Clear	MCDA	2	Yes	No	1	7	8	NA	None	None	2	4	02-12-23	34w	31	1850	45	11/12/23	10	35w+3d	32	1820	46	9/1/24	38	34	3000	47	2/5/24	3m+3w	40	5580	60	49	122	100	31	72
26	T	B/o Komal 2	Raju dhamnekar	02-12-23	10024432	2739	Male	7353347981	26	12th	25	7-4-23	12/1/24	Multiple	LSCS		Twins	Clear	MCDA	2	Yes	No	No	8	9	NA	None	None	1	4	02-12-23	34w	31	2050	45	11/12/23	10	35w+3d	32	1840	45	2/2/24	38	34	3160	48	2/5/24	3m+3w	41	5760	60	77	112	103	28	72
27	T	B/o Vaishnavi	Kanoji Jyothibha	15-12-23	10027375	2746	Female	7353143782	28	BA	60	26/4/23	31/1/24	Singleton	NVD	Cephalic	NRNST	Clear	None	1	yes	No	No	6	8	NA	None	None	1	4	15-12-23	34w	32	1690	41	01/01/24	17	36w+6d	32	1780	43	2/2/24	40	35	2620	47	21/5/24	3m+3w	39	4480	57	70	131	108	30	73
28	T	B/o Priyanka	Omkar Madiwal	20-12-23	10028417	2777	Male	8788861009	25	12th	20	16-7-23	21/4/24	Singleton	LSCS	Cephalic	AEDF	Clear	HTN	1	Yes	No	No	7	8	NA	None	None	1	3	20-12-23	35w+2d	30	1280	40	5/1/24	17	37w+3	31	1400	41	23/1/24	35	32	1840	42	21/5/24	3m	40	5160	59	67	115	143	28	69
29	C	B/o Harshada T1	Siddharth Gogte	26-12-23	10016778	2749	Male	7888234789	44	12th	50	4/5/23	8/2/24	Singleton	LSCS		Twins	Clear	Chronic HTN, hypothyroidism,	2	Yes	No	No	7	9	NA	None	None	2	4	26-12-23	34w	31	1530	39	9/1/24	15	37w+3d	31	1720	42	6/2/24	41	35	2520	47	5/4/24	3m+2d	36	3640	53	58	78.1	119	27	64
30	C	B/o Surekha	Manjunath Bhajanti	10-01-24	10032761	2784	Male	9006788980	30	Btech	40	20-5-23	24/2/24	Singleton	LSCS	Cephalic	HELLP syndrome	Clear	PE	0	No	2 cycles	1	5	7	8	None	None	2	4	10-01-24	34w	30	1470	40	20/01/24	11	35w+4d	41	1480	41	23/2/24	34	34	2460	45	24/5/24	3m	40	5100	52	67	93.6	133	27	65
31	T	B/o Savita	Ramesh kole	15-01-24	10046307	2803	Female	9164438260	26	12th	15	6/7/23	17/4/24	Singleton	LSCS	Cephalic	Previous lscs	Clear	PPROM	0	Yes	No	No	7	9	NA	None	None	2	3	15-01-24	36w+6d	30	1950	45	27/3/24	7	37w+6d	31	2080	46	12/4/24	22	32	2700	48	15/7/24	3m	40	5200	58	68	122	92.4	30	72
32	T	B/o Saba Anjum 1	Mohd Sheik	21-01-24	10009410	2796	Female	7353960233	31	BCom	40	24-5-23	28/2/24	Multiple	LSCS		DCDA Twins	Clear	DCDA Twin	2	Yes	No	No	7	9	NA	None	None	1	3	21-01-24	34w	30	1760	42	3/2/24	14	36w	32	1820	44	1/3/24	35	34	2140	46	15/7/24	3m+2w	40	6400	60	64	132	120	33	72
33	T	B/o Saba Anjum 2	Mohd Sheik	21-01-24	10009411	2797	Female	9164871311	31	BCom	40	24-5-23	28/2/24	Multiple	LSCS		DCDA Twins	Clear	DCDA Rwin	2	Yes	No	No	7	9	NA	None	None	1	3	21-01-24	34w	31	1820	42	3/2/24	14	36w	32	1900	44	1/3/24	35	34	2200	47	15/7/24	3m+2w	41	6300	61	68	139	115	32	72
34	T	B/o Jaba	Kalicharan Tudu	24-01-24	10035808	2798	Male	6372822014	26	10th	24	11-5-23	15/2/24	Singleton	LSCS	Breech	Breech, FGR, increased resistance	Meconium	HTN	2	Yes	No	No	8	9	NA	None	None	1	4	24-01-24	36w+6d	31	2100	43	01/2/24	7	37w+6d	32	2050	44	16/2/24	22	33	2380	46	1/6/24	3m+2w	40	6120	61	58	100	112	25	72
35	C	B/o Kaveri T-1	Vittal Sampagar	30-01-24	10024692	2930	Male	7406000305	22	Bcom	50	24/4/23	29/1/24	Multiple	LSCS		PPROM	Clear	PPROM	1	Yes	No	1	7	8	NA	None	None	2	4	30-01-24	34w+1d	31	1840	45	30/12/23	17	37w+3d	32	1920	46	30/01/24	42	34	2780	48	14/5/24	3m+2w	41	5700	61	75	93.4	122	31	70

SL No:	Study	Infant name	Fathers name	Date of birth	IP No	HRB No	Sex	Phone number	Maternal age	Maternal education	Family income per month (k)	LMP	EDD	Singleton/multiple	Mode of delivery	Vaginal- cephalic/ breech	Caesarean indication	Liquor- clear/ meconium/ blood	Antenatal risk factor	Antenatal steroid doses	Cried at birth	Bag and mask ventilation	CPAP	APGAR-1	APGAR-5	APGAR-10	Postural deformity	Mechanical consequences of Birth Process	Breast feeding started at	Enteral supplementation started at	Date of birth	Corrected age	Birth OFC	Birth weight	Birth height	Date of assessment at discharge	Day of life at discharge	Corrected age at discahrge	Discharge OFC	Discharge weight	Discharge height	Date of assessment at 40w CGA	Day of life at 40w CGA	OFC at 40w CGA	Weight at 40w CGA	Height at 40w CGA	Date of assessment at 3 months of CGA	Day of life at 3monthCGA	OFC at 3 monthCGA	Weight at 3 months CGA	Height at 3 months CGA	Serum zinc at birth	Serum zinc at 3 months CGA	Serum copper at 3 months CGA	HNNE at 40w CGA	HINE at 3 months CGA
36	C	B/o Kaveri T-2	Vittal Sampagar	30-01-24	10024693	2931	Male	7406000305	22	Bcom	50	24/4/23	29/1/24	Multiple	LSCS		PPROM	Clear	PPROM	1	Yes	No	1	7	8	NA	None	None	2	4	30-01-24	34w+1d	31	1780	44	30/12/23	17	37w+3d	32	2040	46	30/01/24	42	34	3140	48	14/5/24	3m+2w	41	5680	59	59	79.1	142	30	68
37	C	B/o Preeti	Channabasayya Hiremath	31-01-24	10037524	2818	Male	9035579767	24	BA	30	4-6-23	10/3/24	Singleton	NVD	Cephalic	Oligo	Clear	PPROM	2	No	2 cycles	2	4	7	8	None	None	2	5	31-01-24	34w + 4d	31	1700	44	13/02/24	14	36w+4d	32	1860	45	1/3/24	46	34	2120	46	18/6/24	3m+2w	39	4700	57	71	89.8	144	27	65
38	C	B/o Soumya	Manjunath Chandaregi	01-02-24	10037559	2815	Male	9900227179	24	Btech	65	7-6-23	13/3/24	Singleton	LSCS	Cephalic	Severe PE, AEDF	clear	PE, anemia	0	Yes	No	1	6	8	NA	None	None	2	5	01-02-24	34w+3d	28	1670	42	16/2/24	17	36w+4d	29	1760	44	12/3/24	40	34	2420	47	1/2/24	3m+2w	38	5341	56	67	96.4	142	27	66
39	T	B/o Soumya	Mallikarjun Konji	07-02-24	10039184	2822	Male	9380085197	19	10th	20	10/5/23	14/2/24	Singleton	LSCS	Cephalic	Severe PE, LOFGR	Clear	PE	1	Yes	No	1	7	9	NA	None	None	2	4	07-02-24	35w	31	1800	43	18/2/24	11	36w+3d	32	1780	44	12//3/24	36	34	2240	46	11/6/24	3m	39	5130	57	84	122	121	31	69
40	T	B/o Bhagyashree 2	Mahesh Choukulkar	20-02-24	10042075	2849	Male	9359237635	31	BCom	18	17/6/24	22/3/24	Singleton	LSCS		DCDA Twins	Clear	Twins	2	yes	No	1	8	9	NA	None	None	1	3	20-02-24	35w+3d	30	1400	41	12/3/24	23	38w+4d	31	1600	42	22/3/24	35	31	1780	43	28/6/24	3m+1w	39	5100	52	56	126	100	28	69
41	T	B/o Bhagyashree 1	Mahesh Choukulkar	20-02-24	10042074	2850	Male	9359237635	31	B com	18	17/6/24	22/3/24	Singleton	LSCS		DCDA Twins	Clear	Twins	2	Yes	No	1	7	9	NA	None	None	1	3	20-02-24	35w+3d	32	1900	45	12/3/24	23	38w+4d	33	2000	46	22/3/24	35	34	2200	47	28/6/24	3m+1w	40	5420	54	68	102	98.3	30	69
42	C	B/o Rubina	Tanveer Mulla	02-03-24	10044385	2980	Female	9741488195	26	12th	20	3-7-23	8/4/24	Singleton	LSCS	Cephalic	AEDF	Clear	EOFGR,AEDF	1	Yes	No	No	7	8	NA	None	None	1	3	02-03-24	34w+5d	31	1560	42	11/3/24	9	36w+2d	32	1490	42	12/4/24	42	33	2320	46	19/7/24	3m+1w	38	4300	55	69	83.6	151	30	68
43	T	B/o Bharati T-1	Gurubasappa Mallar	04-03-24	10046848	3013	Male	7899007688	35	BTech	65	17/3/24	22/4/24	Multiple	LSCS		DCDA with Early onset FGR	Clear	T2 DM on Insulin, Rh neg Preg	1	Yes	No	No	8	9	NA	None	None	2	4	04-03-24	34w	28	1240	38	8/4/24	29	37w+5d	29	1400	39	26/4/24	49	30	1440	40	2/8/24	3m+10d	34	4200	56	72	128	95	33	71
44	T	B/o Bharati T-2	Gurubasappa Mallar	04-03-24	10046849	3012	Male	7899007688	35	Btech	65	17/3/24	22/4/24	Multiple	LSCS		DCDA with Early onset FGR	Clear	T2 DM on Insulin, Rh neg Preg	1	Yes	No	No	8	9	NA	None	None	2	4	04-03-24	34w	29	1440	39	8/4/24	26	37w+5d	30	1560	42	26/4/24	47	33	2600	46	2/8/24	3m+10d	39	4860	54	80	132	91.3	34	72
45	C	B/o Demannavar T-1	Siddalingappa	10-03-24	10046025	2994	Male	7483501131	32	B Tech	120	3/7/23	8/4/24	Multiple	LSCS		Cervical Encerclage in labour	Clear	DCDA twin	2	Yes	No	1	7	8	NA	None	None	2	4	10-03-24	34w+1d	31	1860	42	22/3/24	12	36w+5d	31	1820	44	23/4/24	34	34	2080	45	3/8/24	3m+2d	39	5300	52	58	88.2	132	30	68
46	C	B/o Demannavar T-2	Siddalingappa	10-03-24	10046026	2995	Male	7483501131	32	B Tech	120	3/7/23	8/4/24	Multiple	LSCS		Cervical Encerclage	Clear	DCDA twin	2	Yes	No	1	7	8	NA	None	None	2	4	10-03-24	34w+1d	31	1900	42	22/3/24	12	36w+5d	32	1840	44	23/4/24	34	33	2140	45	3/8/24	3m+2	38	5100	50	60	78.2	130	32	69
47	C	B/o Nirmala	Sunilkumar Choragi	12-03-24	10047139	2876	Male	9845929185	34	BTech	140	29/7/23	4/5/24	Singleton	LSCS	Cephalic	Severe PIH	Clear	PIH	1	Yes	No	1	7	9	NA	None	None	2	3	12-03-24	34w+2d	30	1600	40	30/3/24	18	37w	31	1480	42	19/4/24	48	32	1700	43	30/7/24	3m+2d	35	3210	48	72	91.8	113	29	67
48	C	B/o Sushma	Mahesh patil	14-03-24	10046979	2864	Male	9552652642	21	12th	10	13/7/24	18/4/24	Singleton	LSCS	Breech	Eclampsia	Meconium	Eclampsia	1	yes	No	No	6	8	NA	None	None	1	4	14-03-24	35w	32	2000	44	22/3/24	8	36w+1d	32	1840	45	16/4/24	49	35	2860	49	14/6/24	3m+2w	40	6010	59	78	95.5	122	31	67
49	C	B/o Akshata	Bahubali khanaj	16-03-24	10047625	2982	Female	9877787891	25	BA	40	15-7-23	20/4/24	Singleton	LSCS	Cephalic	Severe PE	Clear	HTN	1	Yes	No	No	7	8	NA	None	None	1	4	16-03-24	34w	30	1630	41	21/3/24	6	34w+6d	30	1420	41	1/5/24	45	34	3040	37	6/8/24	3m+5d	38	4700	55	54	62.1	143	30	64
50	C	B/o Surekha	Prashanth Bhandenkar	19-03-24	10048055	3004	Female	9035665653	21	10th	25	26-7-23	1/5/24	Singleton	LSCS	Cephalic	PPROM with trauma	Clear	PPROM with trauma to abdomen	0	Yes	No	No	7	8	NA	None	None	1	3	19-03-24	34w	32	2200	47	2/4/24	14	36w	32	2280	48	1/5/24	42	35	3240	53	18/8/24	3m+3w	38	5960	57	75	92.4	151	30	68
51	T	B/o Shilpa	Robert Nagnoor	20-03-24	10048524	2982	Male	7892068844	24	BA	50	25/7/23	30/4/24	Singleton	LSCS	Cephalic	HTN	Clear	HTN	2	Yes	No	No	7	8	NA	None	None	1	4	20-03-24	34w	29	1500	39	29/3/24	10	35w+4d	30	1520	40	30/4/24	42	34	2320	45	25/7/24	3m+ 1w	38	5080	62	61	120	150	28	72
52	T	B/o Sukanya T-1	Kadesh Nyamagoud	26-03-24	10050732	2878	Male	7019171025	23	Diploma	60	13-7-23	18/4/24	Multiple	LSCS		PIH	Clear	Twins	2	Yes	No	1	7	8	NA	None	None	2	4	26-03-24	35w+6d	34	2170	49	2/4/24	7	36w+6d	35	2190	50	28/4/24	48	38	3140	54	16/8/24	3m+2w	43	6900	66	63	109	110	29	70
53	T	B/o Sukanya T-2	Kadesh Nyamagoud	26-03-24	10050733	2877	Male	7019171025	23	Diploma	60	13/723	18/4/24	Multiple	LSCS		PIH	Clear	Twins	2	Yes	No	1	7	8	NA	None	None	2	4	26-03-24	35w+6d	32	1940	45	2/4/24	7	36w+6d	32	2080	46	28/4/24	39	40	5140	62	16/8/24	3m+2w	40	5140	62	65	134	94.6	28	70
54	C	B/o Najmina	Ashfaque Alam	31-03-24	10050798	2876	Female	6363880298	23	Bed	30	19-7-23	24/4/24	Singleton	LSCS	Cephalic	AEDF	Clear	Hypothyroidism	1	Yes	No	No	8	9	NA	None	None	2	4	31-03-24	36w+5d	32	2100	41	9/4/25	10	38w+1d	32	2140	47	24/4/24	23	33	2320	48	1/8/24	3m+1w	37	4980	57	77	91.7	127	30	69
55	T	B/o Sushma	Rajendra Patil	14-04-24	10053650	2750	Male	9448995056	23	12th	20	26-11-23	22/5/24	Singleton	LSCS	Cephalic	Oligo	Clear	Oligo	1	Yes	No	No	7	9	NA	None	None	1	3	14-04-24	34w+4d	30	1810	43	22/4/24	8	35w+6d	31	1680	44	21/5/24	37	32	2010	46	2/9/24	3m+2w	39	5740	58	59	130	101	28	72
56	T	B/o Renuka	Renuka Muniraj	29-04-24	10057617	2998	Female	9980433622	33	BCom	180	23/9/23	29/6/24	Singleton	LSCS	Cephalic	Prev LSCS	Clear	Severe PE, hypothyroidism	1	Yes	No	No	7	8	NA	None	None	2	4	29-04-24	34w+3d	29	1530	42	31/5/24	17	37w+6d	30	1520	43	31/5/24	32	34	2120	44	4/9/24	3m+2w	37	5080	53	62	122	88.4	26	72
57	T	B/o Sadhana T-1	Kishore Kudale	01-05-24	10057647	2974	Male	7349180593	27	BA	25	22/8/23	28/5/24	Multiple	LSCS		DCDA with Early onset FGR	Clear	DCDA twin	2	Yes	No	No	8	9	NA	None	None	1	3	01-05-24	36w+1d	32	2200	44	8/5/24	8	37w+3d	33	2000	45	31/5/24	25	36	3040	47	4/9/24	3m+1 w	42	5810	56	62	130	100	27	70
58	T	B/o Sadhana T-2	Kishore Kudale	01-05-24	10057647	2975	Male	7349180593	27	BA	25	22/8/23	28/5/24	Multiple	LSCS		DCDA with Early onset FGR	Clear	DCDA Twin	2	Yes	No	Labour Room	8	9	NA	None	None	1	3	01-05-24	36w+1d	31	2100	43	8/5/24	8	37w+3d	32	2000	44	31/5//24	25	35	3100	47	4/9/24	3m+1w	43	6100	58	74	135	97.4	30	71
59	T	B/o Gayatri T-1	Prahlad Patil	29-05-24	10064741	2970	Female	7774863571	24	Bed	60	23/9/23	29/6/24	Multiple	LSCS		Twins	Clear	DCDA Twin	2	Yes	No	1	7	8	NA	None	None	2	4	29-05-24	35w+6d	32	2100	42	7/6/24	8	37w+1d	33	1720	43	28/6/24	29	36	2300	45	15/10/24	3m+2w	44	6120	58	67	124	131	31	73
60	T	B/o Gayatri T-2	Prahlad Patil	29-05-24	10064742	2971	Female	7774863571	24	Bed	60	23/9/23	29/6/24	Multiple	LSCS		Twins	Clear	DCDA Twin	2	Yes	No	1	7	8	NA	None	None	2	4	29-05-24	35w+6d	31	1580	41	7/6/24	8	37w+1d	33	1940	43	28/6/24	29	33	1940	43	15/10/24	3m+2w	40	5830	54	58	120	140	30	73
61	C	B/o Asmita	Ankush Birajdar	31-05-24	10065221	2098	Female	7353437345	31	Btech	50	30/9/23	6/7/24	Singleton	LSCS	Cephalic	NRNST	Clear	HTN	2	Yes	No	No	7	8	NA	None	None	2	4	31-05-24	34w+6d	30	1800	42	21/6/24	22	37w+6d	31	1660	43	5/7/24	37	33	1980	45	10/10/24	3m	37	4120	52	63	87.8	120	26	67
62	T	B/o Deepa	Laxmikant Kuri	10-06-24	10067390	3008	Male	6378211990	32	Btech	30	8/10/23	14/7/24	Singleton	LSCS	Cephalic	Prev LSCS, BOH, MTHFR	Clear	MTHFR mutation	2	Yes	No	No	7	8	NA	None	None	1	3	10-06-24	34w+6d	33	2440	46	16/6/24	7	35w+6d	33	2440	46	17/7/24	39	35	3020	49	4/11/24	3m+2w	41	6470	57	81	115	92.4	32	72
63	C	B/o Manjula T-1	Sanju Desai	12-06-24	10068259	2794	Male	7815034688	26	BA	35	8/10/23	14/7/24	Multiple	LSCS		Twins	Clear	UTI	2	Yes	No	No	7	8	NA	None	None	1	3	12-06-24	35w+2d	32	2010	47	23/6/24	12	37w	33	2000	47	12/7/24	31	35	2320	50	2/11/24	3m+3w	39	6000	58	73	83.5	139	29	69
64	C	B/o Manjula T-2	Sanju Desai	12-06-24	10068260	2795	Male	7815034688	26	BA	35	8/10/23	14/7/24	Multiple	LSCS		Twins	Clear	UTI	2	Yes	No	No	8	9	NA	None	None	1	3	12-06-24	35w+2d	32	1950	46	23/6/24	12	37w	33	2140	46	12/7/24	31	34	2780	48	2/11/24	3m+3w	38	5100	55	56	94.1	124	30	67
65	T	B/o Rekha 1	Nagaraj Nayak	20-06-24	10070204	3013	Male	7400401437	31	12th	40	27-7-23	2/8/24	Multiple	LSCS		Anamnios	Clear	PPROM	1	Yes	No	No	7	8	NA	None	None	1	3	20-06-24	34w	30	1640	43	1/7/24	11	35w+4d	31	1640	44	12/8/24	40	33	2180	46	22/11/24	3m+ 1w	40	5660	58	49	104	100	29	71
66	T	B/o Rekha 2	Nagaraj Nayak	20-06-24	10070205	3014	Male	7400401437	31	12th	40	27-7-23	2/8/24	Multiple	LSCS		Anamnios	Clear	PPROM	1	Yes	No	No	7	9	NA	None	None	1	3	20-06-24	34w	31	1600	43	1/7/24	11	35w+4d	32	1720	44	12/8/24	40	35	2260	47	22/11/24	3m+1w	41	5560	58	54	100	108	28	72
67	T	B/o Rekha 3	Nagaraj Nayak	20-06-24	10070206	3015	Male	7400401437	31	12th	40	27-7-23	2/8/24	Multiple	LSCS		Anamnios	Clear	PPROM	1	Yes	No	No	7			None	None	1	3	20-06-24	34w	30	1680	43	1/7/24	11	35w+4d	30	1760	44	12/8/24	40	35	2400	47	22/11/24	3m+1w	42	5800	60	50	120	102	29	71

SL No:	Study	Infant name	Fathers name	Date of birth	IP No	HRB No	Sex	Phone number	Maternal age	Maternal education	Family Income per month (k)	LMP	EDD	Singleton/multiple	Mode of delivery	Vaginal- cephalic/ breech	Caesarean indication	Liquor- clear/ meconium/ blood	Antenatal risk factor	Antenatal steroid doses	Cried at birth	Bag and mask ventilation	CPAP	APGAR-1	APGAR-5	APGAR-10	Postural deformity	Mechanical consequences of Birth Process	Breast feeding started at	Enteral supplementation started at	Date of birth	Corrected age	Birth OFC	Birth weight	Birth height	Date of assessment at discharge	Day of life at discharge	Corrected age at discahrge	Discharge OFC	Discharge weight	Discharge height	Date of assessment at 40w CGA	Day of life at 40w CGA	OFC at 40w CGA	Weight at 40w CGA	Height at 40w CGA	Date of assessment at 3 months of CGA	Day of life at 3monthCGA	OFC at 3 monthCGA	Weight at 3 months CGA	Height at 3 months CGA	Serum zinc at birth	Serum zinc at 3 months CGA	Serum copper at 3 months CGA	HNNE at 40w CGA	HINE at 3 months CGA
68	T	B/o Anuradha	Pavan Kundgol	23-06-24	10070807	3004	Female	9980463366	23	BCom	37	26/9/23	20/7/24	Singleton	LSCS	Breech	Oligo with FGR	Clear	None	2	Yes	No	No	8	9	NA	None	None	1	3	23-06-24	36w	30	1800	44	27/6/24	4	36w+4d	30	1720	44	25/7/24	38d	32	2420	46	8/8/24	3m	39	5830	54	72	113	96.7	26	71
69	C	B/o Vaishnavi T-1	Chethan Hebsur	04-07-24	10073724	3020	Male	9972476771	26	BA	100	28/10/24	3/8/24	Multiple	LSCS		Twins	Clear	Twins	1	Yes	No	No	7	8	NA	None	None	1	3	04-07-24	34w+4d	31	2200	47	14/7/24	11	36w+1d	32	2160	48	17/8/24	31	35	4070	53	16/11/24	3m+1w	39	6010	63	72	88.4	140	30	69
70	C	B/o Vaishnavi T-2	Chethan Hebsur	04-07-24	10073725	3021	Male	9972476771	26	BA	100	28/10/24	3/8/24	Multiple	LSCS		Twins	Clear	Twins	1	Yes	No	No	7	8	NA	None	None	1	3	04-07-24	34w+4d	30	2200	47	14/7/24	11	36w+1d	31	2210	48	17/8/24	31	34	2740	49	16/11/24	3m+1w	40	5060	60	68	91.4	135	29	68
71	C	B/o Renuka	Vinod Jadhav	15-07-24	10076184	3010	Male	7897897633	27	10th	30	10/11/23	10/8/24	Singleton	LSCS	Cephalic	Oligo	Clear	HTN	2	Yes	No	No	7	9	NA	None	None	1	3	15-07-24	35w+2d	32	2250	44	22/7/24	8	36w+3d	33	2100	45	19/8/24	36	35	2570	47	22/11/24	3m	38	5010	58	67	92.2	138	26	68
72	C	B/o Pooja T-1	Nivruti Koli	10-08-24	10008331	3084	Male	8867814056	24	BA	60	2/12/23	7/9/24	Multiple	LSCS		Twins	Clear	DCDA Twin	2	Yes	No	1	7	9	NA	None	None	2	4	10-08-24	36w	32	2000	44	17/8/24	4	36w+4d	32	1760	44	14/9/24	28	34	2200	46	2//1/25	3m+2w	38	6010	58	68	98.8	131	30	70
73	C	B/o Pooja T-2	Nivruti Koli	10-08-24	10008332	3085	Female	8867814056	24	BA	60	2/12/23	7/9/24	Multiple	LSCS		Twins	Clear	DCDA Twin	2	Yes	No	1	7	9	NA	None	None	2	4	10-08-24	36w	30	1700	42	17/8/24	4	36w+4d	30	1620	42	14/9/24	28	31	2320	44	2/1/25	3m+2w	42	5210	54	66	88.4	134	30	68
74	T	B/o Malavva T-1	Sanju Bagewadi	12-08-24	10083113	2890	Male	7844689923	22	12th	30	28/11/23	3/9/24	Multiple	LSCS		Twins	Clear	DCDA Twins	2	Yes	No	No	7	9	NA	None	None	1	3	12-08-24	36w+6d	32	1900	46	19/8/24	7	37w+6d	32	1830	46	6/9/24	21	34	2620	48	19/12/24	3m+2w	39	5140	53	64	109	129	33	71
75	T	B/o Malavva T-2	Sanju Bagewadi	12-08-24	10083113	2891	Male	7844689923	22	12th	30	28/11/23	3/9/24	Multiple	LSCS		Twins	Clear	DCDA Twins	2	Yes	No	No	7	9	NA	None	None	1	3	12-08-24	36w+6d	32	1800	43	19/8/24	7	37w+6d	33	1760	44	6/9/24	21	34	2420	46	19/12/24	3m+2w	40	5980	57	63	107	142	33	72