University of Rajshahi **Rajshahi-6205** **Bangladesh.**

RUCL Institutional Repository **http://rulrepository.ru.ac.bd**

Institute of Biological Sciences (IBSc) PhD Thesis

2011

Effect of Breast Feeding on the Health Status of Children of Rajshahi

Haque, Md. lmdadul

University of Rajshahi

http://rulrepository.ru.ac.bd/handle/123456789/971

Copyright to the University of Rajshahi. All rights reserved. Downloaded from RUCL Institutional Repository.

EFFECT OF BREAST FEEDING ON THE HEALTH STATUS OF CHILDREN OF RAJSHAHI

THESIS SUBMITTED FOR THE DEGREE
OF
DOCTOR OF PHILOSOPHY
IN THE
INSTITUTE OF BIOLOGICAL SCIENCES
RAJSHAHI UNIVERSITY, BANGLADESH

By

Md. Imdadul Haque

MBBS, DCH,

May 2011

INSTITUTE OF BIOLOGICAL SCIENCES
RAJSHAHI UNIVERSITY, RAJSHAHI
BANGLADESH

Dedicated
To
My Parents

DECLARATION

I hereby declare that the thesis entitled **'EFFECT OF BREAST FEEDING ON THE HEALTH STATUS OF CHILDREN OF RAJSHAHI'** submitted in the Institute of Biological Sciences, University of Rajshahi for the degree of **Doctor of Philosophy** is the result of my own investigation carried out under the supervision of Professor Dr. M. Khalequzzaman, Director, Institute of Biological Sciences, University of Rajshahi and Co-Supervisor Professor A. B. Siddique, Rajshahi Medical College.

The work as a whole or in part there of has not been submitted in any form for any other degree elsewhere.

Rajshahi
May 2011

Md. Imdadul Haque
Candidate

M. Khalequzzaman
Ph D, FZSB, FRES (London)
Professor & Director

Institute of Biological Sciences
University of Rajshahi
Rajshahi 6205, Bangladesh
Tel: 0721 750928

Certificate

This is to certify that Md Imdadul Haque worked under my supervision as a Ph. D. fellow. I am pleased to forward his thesis entitled '**EFFECT OF BREAST FEEDING ON THE HEALTH STATUS OF CHILDREN OF RAJSHAHI**', which is carried out in the Institute of Biological Sciences, University of Rajshahi. He has fulfilled all the requirements of the regulations and prescribed period of research for submission of thesis for the award of the degree of **Doctor of Philosophy**.

08.05.2011

M Khalequzzaman
Professor and Director
Institute of Biological Sciences
University of Rajshahi
Rajshahi, Bangladesh

CONTENTS

	Page No.
Acknowledgement	i
Abstract	iii
Abbreviation	xii
Chapter-1: Introduction	**1-38**
1.1 Breast Feeding	1
1.2 Human Milk Components	8
1.2.1 Leucocytes	10
1.2.2 Soluble Mediators	15
1.2.3 Immunoglobulins, Lactoferrin and Oligosaccharides	16
1.2.4 Dietary Protein Antigens	19
1.3 Nutritional and non-nutritional components	20
1.4 Benefit of Breast Feeding	25
1.4.1 Benefits for Children	26
1.4.1.1 Lower Rates of Infectious Disease	27
1.4.1.2 Lower Rates of Chronic Disease	30
1.4.2 Benefits to Mother	34
1.4.3 Other Health Benefits	35
1.4.4 Benefits to the Family	36
1.4.5 Benefits to the Nation	36
1.4.6 Economic Benefits	37
1.5 Objectives of the Study	38

Chapter-2: Subjects and Methods **39-48**

2.1 Sample and Setting 39

2.2 Study Procedure 39

2.2.1 Variable selection 40

2.2.2 Chi-square test statistic 41

2.3 Definitions used in the study 41

2.4 Questionnaire used in the study 45

Chapter 3: Results **49-103**

3.1 Breast feeding pattern and different disease incidence 49

3.2 Complementary feeding pattern and different disease incidence 55

3.3 Alternate feeding pattern and different disease incidence 60

3.4 Nature of food and different disease incidence 65

3.5 Parental educational status in different disease incidence 69

3.6 Nature of housing and incidence of different diseases 73

3.7 Drinking water source and different disease incidence 77

3.8 Sanitation and different disease incidence 81

3.9 Family size and different disease incidence 85

3.10 Parental socio-economic status and different disease incidence 89

3.11 Nutritional Status and different disease incidence 94

3.12 Age of the patients and different disease incidence 98

Chapter-4: Discussion **104-115**

Chapter-5: Conclusion **116-117**

Chapter-6: Literature Cited **118-155**

List of Abbreviations

GIT	Gastrointestinal tract
DD	Diarrheal disorder
AGN	Acute glomerulonephritis
AWD	Acute watery diarrhea
ALL	Acute lymphoblastic leukemia
RMCH	Rajshahi Medical College Hospital
OPD	Out Patient Department
PCB	Polychlorinated Biphinyl
DDT	Dichloro-diphenyl-tricholoethane
CMA	Cow's milk allergy
NK	Natural killer
CMV	Cytomegalovirus
IgM	Immunoglobulin M
IgA	Immunoglobulin A
IgG	Immunoglobulin G
PPH	Postpartum hemorrhage
WHO	World Health Organisation
TGF	Transforming growth factor

List of Tables

Table No.		Page No.
1.	Breast feeding pattern and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	53
2.	Complementary feeding pattern and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	58
3.	Alternative feeding pattern and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	63
4.	Nature of food and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	67
5.	Parental education status and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	71
6.	Housing type and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	75
7.	Drinking water source and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	79
8.	Sanitary system and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	83
9.	Family size and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	87
10.	Parental education status and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	92
11.	Nutritional status and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.	96
12.	Age and different disease incidence among children of Rajshahi.	100

List of Figures

Figure No.		Page No.
1.	Relation between breastfeeding patterns and incidence of diseases among children	54
2.	Relation between complementary feeding and different disease incidence.	59
3.	Relation between alternate feeding pattern and different disease incidence.	64
4.	Relation between Nature of food and different disease incidence.	68
5.	Relation between parental education and incidence of diseases among children	72
6.	Relation between housing type and different disease incidence.	76
7.	Relation between drinking water source and different disease incidence.	80
8.	Relation between sanitary system and different disease incidence.	84
9.	Relation between Family size and different disease incidence.	88
10.	Relation between Parental socio-economic status and disease incidence.	93
11.	Relation between nutritional status and different disease incidence.	97
12.	Relation between children ages and different disease incidence.	101

List of Plates

Plate No.		Page No.
1.	Diarrheal diseased infant feeding ORS	102
2.	Pneumonic child with chest indrawing	102
3.	Pneumonic child with chest indrawing and abdominal distension	103
4.	A malnutrient child	103

Acknowledgement

I would like to express my deep sense of gratitude and debt to my respected supervisors Dr. M. Khalequzzman, Professor and Director, Institute of Biological Sciences (IBSc), University of Rajshahi for his continuous help, stimulating discussion, valuable suggestion and proper guidance during the supervision of the whole research work. My heartiest gratitude to Professor A. B. Siddiqui, Head of the department of Paediatrics, Rajshahi Medical College (RMC), for his enthusiastic encouragement and advice to me for doing this study.

I am grateful to the Chairman, Department of Zoology, University of Rajshahi, where I had started my Ph D research work and then switched to the IBSc. I am grateful to all teachers of the IBSc for their inspiration and occasional help. I am also indebted to Professor Dr. Anwar Habib, Head, Department of Pharmacology, RMC for his valuable advice in completing my study. My thanks are also due to Director, Rajshahi Medical College Hospital (RMCH), for permitting to collect data in paediatrics unit both in patients and out patients departments.

I must express my sincere gratitude to Dr. Apurba Kumar Roy, Associate Professor, Department of Genetic Engineering and Biotechnology, and his wife Dr. Bharoti Das, Lecturer, Department of Zoology, Baneswer

College, Rajshahi for their valuable support and advice regarding protocol development, suggestions and continuous co-operation during this study.

I shall ever remain indebted to all those children and mothers whose participation made this study possible. I like to thank to my senior colleagues, friends and junior doctors of paediatrics department of RMCH for their interest and support.

My grateful thanks are extending to my relatives, well-wishers and members of my family especially to my beloved son Md. Rizwanul Haque for constant source of inspiration during my work. My appreciation and thanks also go to my wife Nilufer Sultana for her constant tireless moral support and keeping patience throughout the work.

The Author

Abstract

In settings with excessive child mortality and a strong association between breastfeeding and mortality, it is important to know if and how improved breastfeeding practices can reduce infant and child mortality. Therefore this study tries to touch these issues on the effect of breast feeding on child health. The primary source of data for the present study was collected from the hospitalized children in Rajshahi Medical College Hospital The patients were chosen by taking alternate bed serial in the hospital ward and alternate registration serial in the OPD. The study population consisted of 800 children between ages 0-12 years. Antecedent histories of breast-feeding was taken to evaluate whether breast-feeding was associated with the risk of clinically severe diseases. A questionnaire was developed for self-administration after an extensive review of the literature.

It is found that pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis, neonatal jaundice, encephalitis, congenital heart diseases were significantly negatively related with breastfeeding. Enteric fever, meningitis, bronchiolitis, hemolytic anaemia, AGN, AWD and GBS were also negatively associated with breast feeding.

Breast feeding pattern

There were 209 pneumonia cases and among them 9.56% cases had no history of breast feeding. 7.18% cases were exclusive breastfeeding up to 6 months, 55.02% cases got irregularly breast milk and 28.23% cases got sufficient breast milk. The number of perinatal asphyxia children was 78. Regarding breastfeeding 71.79% did not get any breast milk (most of them were getting

parental i/v nutrition as they were very much sick). Irregular breastfeeding cases were 21.79% and 6.42% patients were getting sufficient breast milk up to certain period. Regarding meningitis out of 47 cases, 10.64% did not get breastfeeding, 21.28% got breastfeeding up to 6 months. 42.55% got irregular and 25.53% got sufficient breastfeeding. Encephalitis cases were 20, of them 10% had no history of breastfeeding. 40% got breastfeeding up to 6 months of age, 10% got irregular breast milk and 40% got sufficient breast milk. Out of 46 bronchiolitis patients, 17.39% children never got breast milk, 32.61% of children got breastfeeding up to 6 months of age, another 32.61% of children got irregular and 17.39% of children got sufficient breastfeeding. Viral hepatitis patients were 12 and regarding their breastfeeding status 16.66% of cases did not get any breast feeding, 25% of cases got breast milk up to 6 months of ages. 50% of cases got breastfeeding irregular and 8.33% of patients got sufficient breastfeeding. Bronchial asthma cases were 15. Regarding their breastfeeding status 6.67% of cases did not get any breast feeding. 20% of cases got breast milk up to 6 months of ages. 46.66% of cases got breastfeeding irregularly and 26.67% of patients got sufficient breastfeeding Among 6 cases of GBS 33.33% got breastfeeding up to 6 months of ages. 66.66% of cases got irregular breastfeeding. Among seven cases of pleural effusion 14.28% had no history of breastfeeding 42.86% of cases got breastfeeding up to 6 months of ages and another 42.86% of cases got breastfeeding irregular.

Complementary feeding

Among pneumonia patients, regarding complementary feeding, 9.56% did not get any complementary food; 7.18% got regularly complementary food; 47.85% cases got early complementary food and 35.41% got complementary food late in infancy that is after 6 months of age. Regarding 47 meningitis cases 10.64% did not get any complementary feeding, 21.28% patients got

regularly complementary feeding, 38.29% got early and 29.79% got complementary feeding in late stage. Bronchiolitis patients were 46 out of which 17.39% did not get any complementary feeding, 32.61% got regular complementary feeding, 43.48% got early and 6.52% got late complementary feeding. There were 33 AWD cases, out of which 30.30% of cases did not get any complementary feeding. 27.27% of patients got regular complementary feeding, 36.36% of patients got early and 6.06% of patients got late complementary feeding. Viral hepatitis cases were 12, out of them 16.66% did not get complementary feeding, 25% got regular complementary feeding, 16.66% of patients got early and 41.67% of patients got late complementary feeding. Among 15 cases of bronchial asthma, 6.67% did not get, 20% got regular complementary feeding, 60% of patients got early and 13.33% of patients got late complementary feeding.

Alternate feeding pattern

Of 209 pneumonia cases 7.18% patients did not get any alternative feedings, 41.15% got infant formula as alternative feedings and 51.67% of patients got cow milk as alternative feedings. Among 27 neonatal jaundice patients. 7.41% did not get any alternative feed, 44.44% patients got infant formula and 48.15% patients got cow milk as alternative feedings. Regarding alternative feedings of 40 cases of enteric fever, 30% patients did not get any alternative feedings. 40% got infant formula and 30% patients got cow's milk as alternative feedings. Among 47 meningitis patients, 21.28% of patients did not get any alternative feedings, infant formula was given in 42.55% patients and cow milk was fed to 36.17% of patients. Encephalitic patients were 20, of which 40% patients did not get any alternative feedings, 50% got infant formula and only 10% got cow milk as alternative feedings. Among alternative feedings of 46 bronchiolitis patients, 32.61% of children did not get any

complementary feedings. 39.13% got infant formula and 28.26% got cow milk as alternative feedings. Regarding alternative feedings among the 12 viral hepatitis patients, 25% of patients did not get alternate feeding, 58.33% got infant formula and 16.66% got cow milk as alternate feeding. Regarding alternate feeding of 15 bronchial asthma patients, 20% did not get any alternate feeding. 13.33% got infant formula and 66.66% got cow milk.

Nature of food

Regarding nature of food out of 122 pneumonia patients, 12.29% cases got family diet. 56.56% got diluted milk and 31.15% fed on carbohydrate only. Among 16 perinatal asphyxia eight neonatal sepsis and 15 neonatal jaundice cases all patients were offered diluted milk. Among four enteric fever cases, 70% got family diet and 30% got diluted milk. Meningitis patients were 33, among them family diet was offered to 33.33% patients, 45.45% got diluted milk and only carbohydrate was given to 21.21% of children. Viral hepatitis cases were 12 among them 83.33% got family diet and 16.66% got diluted milk. Regarding rheumatic fever 90% patients got family diet and 10% had history of getting diluted milk among total 10 patients. All patients got family diet among 11 cases of aplastic anaemia. Among 15 bronchial asthma cases 80% got family diet and 20% got diluted milk febrile convulsion patients were 15 among which 66.66% got family diet. 26.67% got diluted milk and 6.66% cases got carbohydrate only.

Parental educational status

Among pneumonia patients 15.31% were primary, 4.30% secondary, 8.61% higher and 71.77% were illiterate. In the same category of 78 perinatal asphyxia cases, the percentage of primary. secondary, higher and illiterate parents were 35.9. 12.82. 32.05 and 19.23 respectively. Among 27 neonatal jaundice cases. their parents were 37.03, 7.40 and 55.56% were primary, secondary and illiterate respectively. There were 40 enteric fever cases of them,

educational status of the parents were 30% primary, 2.50% secondary, 5% higher and 62.50% illiterate. Among 47 meningitis cases, 36.17% parents were educated up to primary, 12.77% were secondary, 12.77% had higher qualification and 38.29% were illiterate. Bronchiolites cases were 46, their parental educational status were 32.61% primary, 13.04% secondary and 54.24% illiterate. Viral hepatitis cases were 12, parental educational status were 50% primary, 16.67% secondary, 8.33% higher and 25% illiterate. Rheumatic fever cases were ten and their parental educational status were 50% primary, 10% secondary and remaining 40% illiterate. There were 15 bronchial asthma cases in this study and their parental educational status were 26.67% primary, 33.33% secondary and 40% illiterate.

Nature of housing

The housing status of pneumonia patients were 36.36% brick building, 54.06% bamboo thatched and remaining 9.57% were other types. Perinatal asphyxia cases were 78 of them 38.46, 32.05 and 29.48% were brick building, bamboo thatched and others types respectively. There were 27 neonatal jaundice cases and their housing status were 44.44 % brick building, 22.22% bamboo thatched and 33.33% others types. Among 40 enteric fever cases housing status were 45.00% brick building, 12.50% bamboo thatched and 42.50% other types. The meningitis cases were 47 among them housing status were 42.55% brick building, 25.53% bamboo thatched and 31.91% others. Housing status was 43.48% brick building, 50% bamboo thatched and 6.52% others among 46 bronchiolitis cases. Among 12 viral hepatitis cases 66.67% were from brick building housing and 33.33% from bamboo thatched housing. Among 10 Rheumatic fever cases 50% from brick building and 50% from bamboo thatched housing. Bronchial asthma cases were 15, their housing status were 46.67% brick building and 53.33% bamboo thatched. Among seven pleural effusion cases, 71.42% housing was brick building and 28.57% was bamboo thatched.

Drinking water source

Among pneumonia patients regarding their drinking water sources were 55.02% tube well water, 36.84% supply water and 8.13% from other sources. Regarding drinking water among 40 enteric fever cases 20% from tube well water, 37.50% from supply water source and 42.50% from other source. Meningitis cases were 47, of them 59.57% from tube well water and 40.42% from supply water got their drinking water. Among 46 bronchiolitis cases, 43.48% got drinking water from tube well water and 56.42% got from supply water. Out of 33 AWD cases 15.15% got drinking water from tube well water, 30.30% got from supply water and 54.54% got from other source. There were 12 cases of viral hepatitis in this study their drinking water were supplied from 16.67% tube well water 66.66% supply water and 16.67% from other source. There were fifteen cases of bronchial asthma in this study their drinking water were supplied from 66.66% tube well water 26.67% supply water and 6.66% from other source. There were seven cases of pleural effusion in this study, their drinking water were supplied from 71.43% tube well water and 28.57% from supply water.

Sanitation

Among pneumonia cases in the category of sanitation status 90.91% sanitary, 7.71% service type and 1.91% open air. Among 27 neonatal jaundice cases, sanitation status were 88.88% sanitary, 11.11% service type. Regarding sanitation among 40 enteric fever cases 37.5% were sanitary, 50% service type and 12.50% open air. Meningitis cases were 47, of them 59.57% sanitary, 34.04% service type and 6.38% open air. Among 46 bronchiolitis cases 56.52% were sanitary and 43.47% were service type. Out of 33 AWD cases, sanitation status were 12.12% sanitary, 54.54% service type and 33.33% open air. Among 12 cases of viral hepatitis in this study 66.66% sanitary, 16.67% service type and 16.67% in open air. Out of 10 rheumatic fever, 60% sanitary and 40% were

service type. There were 15 cases of bronchial asthma and the sanitation status was 66.66% sanitary, 26.67% service type and 6.66% open air.

Family size

There were 209 cases of pneumonia in this study, of them 87.08% from large family and 12.91% from small family. Among 38 neonatal sepsis cases in this study 71.05% belonged to large family and 28.95% belonged to small family. Neonatal jaundice cases were 27, among them 44.44% belonged to large and 55.56% to small family. Out of 40 enteric fever cases 67.50% from large family and 32.50% from small family. Meningitis cases were 47, among them 74.47% belonged to large family and 25.53% belonged to small family. Bronchiolitis cases were 46, of them 76.09% from large and 23.91% from small family. Among 33 AWD cases 75.75% and 24.25% from large and small family respectively. Viral hepatitis cases were 12, of them 66.67% were from large and 33.33% from small family. Out of 10 rheumatic fever cases 80% from large and 20% from small family. There were 15 bronchial asthma cases in this study, among them 46.67% were coming from large family and 53.33% from small family. GBS cases were six, of them 50% were from large and another 50% from small family.

Parental socio-economic status

Among pneumonia patients 9.57, 5.26, 46.89 and 38.27% were from rich, medium, poor and very poor family respectively. Preterm and LBW cases were 45, their parental socio-economic status were 11.11, 4.44, 28.88, and 55.56% as rich, medium, poor and very poor respectively. Neonatal jaundice cases were 27, of them 11.11% were from medium status family, 44.44% from poor class family and another 44.44% from very poor class family. Among 40 enteric

fever cases in this study 7.50% medium, 55% poor and 37.50% from very poor family. Meningitis cases were 47 and their parental socio-economic status were 25.53% rich, 10.64% medium, 31.91% poor and another 31.91% from very poor family. Broncholitis cases were 46 of them 4.35% from rich family, 8.70% from medium, 36.95% from poor and remaining 50% from very poor family. Total AWD cases were 33 out of them 9.09% were coming from rich family, 9.09% from medium, 45.45% poor and 36.36% were coming from very poor family. Viral hepatitis cases were 12, of them 8.33% medium, 33.33% poor and 58.33% from very poor class family. 10%, from medium, 50% from poor class and 40% from very class family among 10 rheumatic fever cases in this study. Among 15 bronchial asthma patients, 13.33% from medium, 40% from poor and 46.67% from very poor family status. Among seven pleural effusion cases in this study 42.86% were poor and 57.14% from very poor family status.

Nutritional Status

The incidence of nutritional status and pneumonia shows that 40.19% were normal, 37.32% had mild malnutrition, 15.31% had moderate and 7.17% had severe malnutrition. Among 40 enteric fever cases in this study, 35, 40, 17.50 and 7.50% were normal, mild, moderate and severe malnutrition respectively. Out of 47 meningitis cases 34.04% had normal nutrition status, 29.78% had mild, 21.28% had moderate and 14.89% had severe malnutrition. Among 46 bronchiolitis cases, 43.48% were normal, 32.60% had mild, and 23.91 had moderate malnutrition, no cases of bronchiolitis had suffered from severe malnutrition. AWD were 33 out of which 18.18% were normal, 42.42% mild, 24.24% moderate and 15.15% severely malnourished. There were 12 viral hepatitis cases of them 83.33% were normal, and 16.66% had mild

malnutrition. Bronchial asthma cases were 15, among them 46.67% were normal, 26.66% were mild, 20% moderate and remaining 6.66% were severely malnourished. Out of six GBS cases, 50% were normal, 33.33% mild, and 16.66% were moderately malnourished. Pleural effusion cases were seven among them 42.85% were within normal nutritional status, 14.28% were mild, 28.57% were moderate and 14.28% were severely malnourished.

Age of the patients

Among 209 cases of pneumonia in this study 35.88% were within 0-2 months, 34.93% were in the age group 3-6 months, 22.00% in the 7-12 months age group, and only 7.17% cases were in the age group of 1-2 years. All of the 78 perinatal asphyxia, 45 preterm and LBW, 38 neonatal sepsis and 27 neonatal jaundice cases were within the age group of 0-2 months indication that this disease occur only at this age group. Enteric fever cases were 40 among them 37.5% in 3-5 years and 62.5% were in the age group of 6-12 years. Among 47 meningitis cases 10.63% were in 0-2 months, 21.27% were 3-6 months, 36.17% were in 7-12 months, 8.51% in 1-2 years, 12.76% in 3-5 years and 10.64% were in the age group of 6-12 years. Broncholitis cases were 46 among them 65.22% were in 3-6 months, 21.74% in 7-12 months and 13.04% in 1-2 years. Out of 33 AWD cases 18.18% were in 0-2 months, 30.30% were in 3-6 months, 27.27% in 7-12 months, 15.15% in 1-2 years and 9.09% were in 3-5 years age group. All the ten rheumatic fever and 11 aplastic anaemia cases were in the age group of 6-12 years. Bronchial asthma cases were 15 among them 53.33% were in 3-5 years and 46.67% were in the age group of 6-12 years.

Abbreviation

GIT	Gastrointestinal tract
DD	iarrheal disorder
AGN	Acute glomerulonephritis
AWD	Acute watery diarrhea
ALL	Acute lymphoblastic leukemia
RMCH	Rajshahi Medical College Hospital
OPD	Out Patient Department
PCB	Polychlorinated Biphinyl
DDT	Dichloro-diphenyl-tricholoethane
CMA	Cow's milk allergy
NK	Natural killer
CMV	Cytomegalovirus
IgM	Immunoglobulin M
IgA	Immunoglobulin A
IgG	Immunoglobulin G
PPH	Postpartum hemorrhage
WHO	World Health Organisation
TGF	Transforming growth factor

Chapter 1

INTRODUCTION

Chapter 1

INTRODUCTION

1.1 Breast Feeding

Breastfeeding is recognized as the preferred form of infant nutrition. Infants who are breastfed experience nutritional and developmental advantages that enhance their health throughout their lives. The choice to breastfeed conveys health benefits to the mother as well. Breastfeeding also imparts economic benefits for families as well as savings for our society (Clark and Bungum 2003). For survival of the children breast feeding has been identified as a major component. Breast feeding among mammals can be considered to be a gift of God. For lack of proper breast feeding all animals including human beings can not achieve full physical and mental growth. The single most cost effective intervention to reduce infant mortality in developing countries would be the promotion of exclusive breastfeeding. The estimated reduction of infant mortality by promoting exclusive breastfeeding is 13% (Jones *et al.* 2003, Matthew *et al.* 2009). Non-exclusive breastfeeding rather than exclusive breastfeeding can increase the risk of dying due to diarrhea and pneumonia among 0–5 month old infants by more than two-fold (Arifeen *et al.* 2001, Mihrshahi *et al.* 2008). Benefits of exclusive breastfeeding up to six months duration have been studied all over the world and there are enormous amount of evidence to

support this (WHO 2002a). The World Health Organization recommended exclusive breastfeeding for six months (WHO 2002b) and most of the international community has followed these guidelines (NHMRC 2003, AAP 2005).

The best first food for babies is breast milk and is the fundamental right of child (Kulkarni *et al.* 2004). Nutrients in the breast milk are the nature's recipe for excellent growth and development. Breast milk is safe, hygienic, inexpensive, readily available to the infant at right temperature and with ideal nutritional value. It reduces childhood infective diseases especially diarrhea and atopic illnessess (eczema, asthma). Mothers who do not breast feed, their babies will have lower IQ, abnormal development and increased infant mortality rate. Breast feeding prevents mother from PPH, it also helps to promote natural family planning and protection against pregnancy and may cause amenorrhea which prevents breast cancer, ovarian cancer and type 2 diabetes in mothers (Campbell and Mongar 2006, Agampodi *et al.* 2007). Despite the well known maternal and child benefits, the breast feeding trend has declined. World wide global data shows that less than 40% of babies below 6 months of age are exclusively breast fed. In our society this trend could be due to western influence, urbanization and increased economic power combined with the increased availability of commercial milk substitutes (Morisky *et al.* 2002).

The World Health Organization recommends exclusive breastfeeding for the first 6 months of life and continuation of breastfeeding for 2 years

(WHO 2002a). The WHO and the United Nations International Children's Emergency Fund have articulated a global strategy for infant and young child feeding (WHO 2002b) and recommendations in the form of guiding principles for complementary feeding of the breastfed child (PAHO 2001) to focus attention on the effect of feeding practices on health and growth of infants and young children. Although these feeding recommendations were based on the evidence available in the published literature, the effects of following these recommended infant feeding practices (IFPs) on growth during infancy and early childhood have not been evaluated.

Breastfeeding has many health and developmental advantages for infants and mothers and is the preferred way of feeding infants to promote optimal infant health and reduced morbidity later in life (Jones *et al.* 2003, Binns and Davidson 2003, WHO 2000, Qiu *et al.* 2009). In Asian cultures, and perhaps more generally, breastfeeding also protects against early *Helicobacter pylori* infection (Okuda *et al.* 2001, 2007, Pearce *et al.* 2005, Horta *et al.* 2007). A recent cohort study from Shanghai suggests that breastfeeding may offer a mother some protection against developing Type II diabetes (Villegas *et al.* 2008). Factors that are important in the initiation of breastfeeding include a favourable paternal attitude toward breastfeeding, as perceived by the mother (Scott *et al.* 2006), whether the mother had an operative delivery, giving prelacteal feeds and ethnicity (Xu *et al.* 2007). The time that the decision to breastfeed is made, maternal age and education and smoking patterns are also important in some societies (Scott and Binns 1999, Gottschang 2007).

The promotion of breastfeeding is a key component of child survival strategies. International policy places emphasis on exclusive breastfeeding during the first 6 months of life, with some groups promoting early initiation of breastfeeding within 1 hour of birth (WHO 1991, UNCF 2005). Although there is an extensive scientific basis for the impact of breastfeeding on postneonatal mortality (Victoria *et al.* 1987, Darmstadt *et al.* 2005), evidence is sparse for its impact on neonatal mortality (Lawn *et al.* 2005) and, to our knowledge, nonexistent for the contribution of the timing of initiation to any mortality impact. Maternal colostrum, produced during the first days after delivery, has long been thought to confer additional protection because of its immune and nonimmune properties (Lawrence and Lawrence 2005). However, epidemiologic data indicate that a high proportion of neonatal deaths are a result of obstetric complications (Kusiako *et al.* 2000), and these are unlikely to be affected by colostrum, transitional breast milk, or mature breast milk. Elucidating the role of timing of initiation of breastfeeding is particularly relevant for sub-Saharan Africa, where neonatal and infant mortality rates are high but most women already exclusively or predominantly breastfeed their infants (Jones *et al.* 2003).

Infants are born with immature immune systems; they are therefore far more susceptible than adults to a variety of diseases, notably infections of the gastro-intestinal tract. Evolution has therefore provided several protective mechanisms by which infants receive passive immunity from their mothers during this critical period. Passive immunity is generally accomplished through the transference of products of an adaptive immune

response, such as T cells or antibodies (Goldsby *et al.* 2003). The receiving individual (in this case the infant) is protected, but does not produce his or her own immune response. A passive transfer of immunity from mother to infant can happen in two ways: either through IgG crossing the placenta before birth, or via IgA passing through the breast milk and into the intestine of the infant after birth. Both of these transfers are essential for protecting the infant during the critical periods before birth and during immune development.

IgG: IgG is the most common antibody in the serum (Goldsby *et al.* 2003). It protects the blood from pathogens, and is important in preventing systemic infections. IgG is the only class of antibody that can cross through the placenta, and thus is the only class of antibody transfered from the mother to the fetus before birth. IgG concentrations in the fetus gradually increase during pregnancy as neonatal Fc receptors concentrate IgG inside the placenta. These Fc receptors have a higher affinity for IgG than the receptors found in the mother, allowing IgG levels in the placenta to be eventually higher than in the mother (Saji *et al.* 1999, Hemming 2001). Children born to hypogammaglobulinemic mothers (who lack normal immunoglobulin levels) are particularly vulnerable to infection, as the mothers do not have antibodies to pass to the infant. Although children born to such females may be born healthy, they are far more likely to succumb to septicemia (Williams *et al.* 1999). Gamma globulin is often administered to the mother throughout pregnancy in order to protect the child, and the child is often successfully treated with antibodies for six months after birth to

prevent severe illness. It has been suggested that intravenous IgG treatments administered to the mother during pregnancy may also help the fetus mature normally, possibly by preventing constant infection in the mother that might hinder fetal development (Williams *et al.* 1999).

Even in children born to normal mothers protection through maternal IgG is temporary. Passive immunity derived from maternal IgG wanes within the first six months of an infant's life as the antibodies are gradually degraded (Hemming 2001). It is thus critical that passive immunity be transferred to the infant during the time between parturition and the development of the infant's own memory responses to pathogens in his or her environment.

IgA: Because the mucosal tissues line the respiratory, gastrointestinal and genitourinary tracts, they are the point of entry for many pathogens. It is therefore essential that these tissues have specific defenses to prevent infection (Kaetzel 2005). Without immune protection in these areas the body would be less able to combat the large range of diseases entering through the mucosol layer. This is demonstrated by the fact that individuals with IgA deficiency, the most common immunodeficiency among people of European descent (Huang *et al.* 2003), experience more sinus and pulmonary infections than normal individuals. Such individuals are surprisingly not more susceptible to gastro-intestinal (GI) tract infections; this may be due to a compensatory mechanism that increases production of both IgG and IgM in the GI tract (O'Neal *et al.* 1999).

Most classes of antibody are unable to cross through the epithelial layer into mucosal tissues. Only IgA and IgM multimers, which have J chains that allow formation of multimeric complexes, can bind to the poly-Ig receptor (pIgR). Binding of the J chain to pIgR allows the antibody/receptor complex to be endocytosed and transported across the cell. Upon release, part of the pIgR is cleaved, and becomes a secretory complex attached to the IgA dimer. (Kaetzel 2005). This component is a glycoprotein, and allows the secretory IgA (sIgA) or IgM (sIgM) to pass through harsh environments, such as the stomach, without being degraded. Many signals in the body can up-regulate pIgR expression. Among these are several hormones and inflammatory chemokines. The ability of inflammatory chemokines to increase expression of pIgR during infection is crucial, as it aids transport of sIgA and sIgM into the mucosal layer when they are most likely to be needed for protection.

Although some IgM can cross the epithelial wall, IgA is the primary antibody isotype in muscosal secretions and in breast milk in normal individuals. IgA mediated passive immunity is passed to infants during nursing; this immunity is critical in protecting the neonates from intestinal pathogens (Hanson *et al.* 1979). IgA is present in the breast milk in concentrations between 0.5-1.5 g/l (Hanson *et al.* 2003). Although there is more IgG in the blood, in normal individuals IgA is by far the most prevalent antibody in the body. Infants who are not breast-fed, and thus do not receive IgA from their mothers, are also far more susceptible to disease. In the developing world the death rate among non-breast fed infants is

significantly higher than among those infants who are breast-fed. It is estimated that if all infants were breast-fed for the first year of life, the number of infant deaths globally would decrease by one million each year (Morrow and Rangel 2004).

The antibodies passed from mother to child are specific to antigens found in the mother's (and, presumably, the child's) environment. Because infants do not have the memory responses typical of adult immunity, it has been suggested that immunizing the mother against childhood infections, such as pertussis, might be an effective method of preventing infection in the neonate. In animal studies this has been shown to be effective, as piglets who suckle mothers that have received pertussis vaccinations are far less susceptible to serious infection (Elahi *et al.* 2006). The IgA in breast milk plays a crucial role in this defense, although other components of breast milk also protect the infant (Elahi *et al.* 2006).

1.2 Human Milk Components

Human milk is assumed to be the ideal food for the infant at least up to the age of 5 or 6 months, ensuring optimal growth and development. In many respects human milk, the most natural food available is unique. The nutritional composition of human milk varies from mother to mother, from day to day, during the day and even during a feed, and is generally suited to the individual needs of the infant. There is little doubt that human milk serves a role in infant physiology greater than being a supply of energy and nutrients. For instance, the immunological properties of human milk

(immunoglobulins, bacteriostatic proteins, living cells, antiviral lipids) are well documented. In developing countries these established beneficial properties can be translated into demonstrable advantages to the breast fed over the bottle fed infant, in terms of reduced morbidity and mortality (Järvinen *et al.* 1984).

Human milk originates in lactating mammary tissue. The basic structural unit is the alveolus, which consists of lactating cells that secrete milk into an adjoining lumen (Patton and Keenan 1975). The lumen connects to a duct system that drains the collected milk to outlets at the skin's surface. Individual arteriovenous capillary systems provide each alveolus with the individual nutrients needed for producing milk. Milk lipid, lactose, and the majority of milk proteins are produced in the lactating cells (Patton and Keenan 1975, McPherson and Kitchen 1983). Human milk contains cells, soluble mediators, immunoglobulins, lactoferrin, oligosaccharides, enzymes, peroxidases, lysozyme, secretory component, bifidus factor, growth factors, hormones, and foreign food antigens. Occasional bacteria and several viruses (rubella, CMV, hepatitis B, vaccinia) have been observed in milk, either passing from the maternal circulation or entering the milk by reflux from the infant during suckling (Ogra and Ogra 1979). Maternal histocompatibility antigens are also present in the cells of milk (Beer *et al.* 1974). The following will focus purely on immunologic components.

1.2.1 Leucocytes

Total number and origin of milk leucocytes: Human colostrum contains 2 to 4 x 10^6 cells/ml, the number of which decays rapidly in four days post partum and decreases more gradually thereafter (Ogra and Ogra 1978, Goldman *et al.* 1982). It is estimated that on average 2 billion each of polymorphonuclear leucocytes and mononuclear cells are ingested by the breast-fed baby during its first four days (Murphey and Buescher 1993). The mean total cell count in preterm colostrum has been found to be significantly higher than in full-term colostrum (Jain *et al.* 1991). This contrasts with the finding of Rodriguez *et al.* (1989) who detected a slightly larger number of leucocytes in milk of mothers delivering preterm, but the difference was not statistically significant (0.55 x 10^6 vs. 0.42 x 10^6 cells/ml). Alcohol consumption has been associated with an increase in number of leucocytes in human milk (Na *et al.* 1997). It is likely that human milk leucocytes originate from blood (Goldman and Goldblum 1996). No leucocytes, other than a few macrophages, appear in the mammary gland until late pregnancy and throughout lactatation. The vast majority of B cells that home to the mammary gland transform into plasma cells that remain sessile in the mammary gland (Goldman and Goldblum 1996). In contrast, other leucocytes attracted to the site from the maternal circulation, probably due to the presence of chemoattractant factors (Michie *et al.* 1998, Böttcher *et al.* 2000b), traverse the mammary epithelium and become part of the milk secretions.

Rajshahi University Library
Documentation Section
Document No. D-3485
Date...9/10/13.....

Mononuclear phagocytic cells: In the milk of healthy women delivered full-term, the predominant cellular component (60 to 90% of milk cells) is the macrophage (Smith and Goldman 1968, Ho *et al.* 1979, Eglinton *et al.* 1994), with a morphology resembling that of tissue macrophages (Pitt 1979). Despite expressing the monocyte markers Leu-M3 and Leu-M5, they also appear phenotypically more similar to tissue macrophages (Xanthou 1997). Additionally, occasional monocytes are found (Smith and Goldman 1968, Ho *et al.* 1979). The structural and functional characteristics of breast milk macrophages are not completely defined. They display unusual morphology, including many lipid-filled vacuoles, milk fat globules, and casein micelles (Smith and Goldman 1968, Smith *et al.* 1971, Crago *et al.* 1979, Ho *et al.* 1979, France *et al.* 1980, Baldus *et al.* 1995). Studies of mothers that have delivered preterm and at full term have shown that milk macrophages are a fully mature tissue macrophage population (Rodriguez *et al.* 1989). They adhere to glass, although less than do their counterparts in peripheral blood (Miler *et al.* 1990). They are activated, as indicated by their high motility (Özgaragoz *et al.* 1988), but their migratory activity and chemotaxis have also been shown to be significantly less than those of less mature blood monocytes (Clemente *et al.* 1986, Thorpe *et al.* 1986, Rodriguez *et al.* 1989). They have been demonstrated to mount a respiratory burst after in vitro stimulation (Tsuda *et al.* 1984, Cummings *et al.* 1985, Speer *et al.* 1985; 1986).

Activation, as indicated by induction of the oxidative burst and prostaglandin production, has been suggested to occur through the IgA

receptors they contain (Robinson *et al.* 1991). Moreover, they show high phagocytic activity (Smith and Goldman 1968, Goldman and Smith 1973, Rodriguez *et al.* 1989), but the number of particles engulfed per cell has been reported to be markedly lower than for blood leucocytes (Miler *et al.* 1990). They have also been demonstrated to kill ingested *Candida albicans* (Cummings *et al.* 1985). That they exhibit strongly carbohydrate antigens in addition to peptide ones may be the result of cytokine-mediated stimulation or increased phagocytic activity (Baldus *et al.* 1995). They also possess the capability of producing toxic oxygen radicals for intracellular killing of microorganisms (Tsuda *et al.* 1984). Some authors suggest that, as elsewhere in the body, human milk macrophages may provide the first line of defense against pathogens (Waksman 1979).

Neutrophils: According to the literature, neutrophils are rare in human milk (8-28%) in breast-feeding mothers (Smith and Goldman 1968, Eglinton *et al.* 1994). However, some authors report as high as 40-60% of neutrophils (Ho *et al.* 1979, Crago *et al.* 1979). Human milk polymorphonuclear cells are functionally exudate cells with less locomotive, adherence, microbicidal, and stimulated respiratory burst capabilities than those of their counterparts in blood (Ho and Lawton 1978, Kohl *et al.* 1980, Weaver *et al.* 1984, Thorpe *et al.* 1986, Buescher and McIlheran 1993, Grazioso and Buescher 1996). Although the interpretation was initially that such lower adherence, polarity, and motility are due to inhibitors in human milk (Thorpe *et al.* 1986), further investigations suggest that they are typical for activated neutrophils, as evidenced by their high expression of the

activation marker CD11b and decreased expression of L-Selectin (Keeney *et al.* 1993). In an older study, the phagocytic ability of human neutrophils was demonstrated, however, to be comparable to that of peripheral blood (Ho *et al.* 1979). Inhibition of neutrophil function in vitro in colostrum and mature milk has been associated with antioxidant activities (Grazioso and Buescher 1996).

T cells: Only 3 to 9% of human milk leucocytes are lymphocytes (Smith and Goldman 1968, Crago *et al.* 1979), with T cells accounting for 74 to 83% of them (Bertotto *et al.* 1990, Jain *et al.* 1991, Wirt *et al.* 1992). They display predominantly the phenotype and functional characteristics of memory T cells (Bertotto *et al.* 1990). The great majority of T cells express antigens involved in intercellular adhesion (LFA-1, ICAM-1) and T-cell activation (CDw29, HLA-DR) (Bertotto *et al.* 1990, Gibson *et al.* 1991, Wirt *et al.* 1992). Milk T cells also exhibit good responsiveness to a variety of bacterial and viral antigens (Smith and Goldman 1968, Parmely *et al.* 1976, Ogra and Ogra 1978), and produce significant amounts of interferon (IFN)-γ (Bertotto *et al.* 1990). These characteristics suggest that milk T cells may be antigenpulsed T cells capable of mounting a secondary immune response.

B cells: B cells comprise 4 to 26% of total milk lymphocytes (Bertotto *et al.* 1990, Jain *et al.* 1991, Wirt *et al.* 1992). They have been found to produce IgA, as first demonstrated by Murillo and Goldman (1970). A high proportion of colostral B lymphocytes show production of the antibodies directed against *Escheria coli* antigen following oral

immunisation (Ahlstedt *et al.* 1975), representing evidence that B cells migrate from GALT to the mammary gland. Evidence from labelling and receptor studies further supports this hypothesis (Roux *et al.* 1977, Bush and Beer 1979). Primed B cells stimulated to blast-transformation and bearing specific membrane IgA migrate in large numbers from the Peyer's patches to draining mesenteric lymph nodes. Further, they travel to the body by way of efferent lymph and the bloodstream to the lamina propria of mucous membranes throughout the body, including the mammary glands, where they evolve into IgA-secreting plasma cells (Roux *et al.* 1977). The migration to the mammary gland becomes a major pathway only during late pregnancy and lactation (Roux *et al.* 1977). Lymphocyte migration appears to be directed by cell-surface molecules termed "homingreceptors", which are leucocyte-endothelial adhesion molecules that interact selectively at areas of specialized endothelium on postcapillary venules to capture the lymphocytes in particular lymphoid organs (Slade and Schwatz 1987). These high endothelial venules express specific surface proteins that have been designated as vascular addressins (Carlos and Harlan 1994).

Natural killer (NK) cells: NK cells represent a small proportion of colostral cells and display low cytotoxic activity (Moro *et al.* 1985, Wirt *et al.* 1992). In contrast to peripheral blood, the majority of colostral NK cells exhibited a degenerated appearance with many vacuoles and no electron-dense granules (Moro *et al.* 1985).

Eosinophils: Eosinophils account for about 2% of milk cells (Vassella *et al.* 1992, Eglinton *et al.* 1994). Vassella *et al.* (1992) has reported that the number of eosinophils in human milk is positively correlated with their number in peripheral blood, suggesting migration of eosinophils from peripheral blood to mammary gland as described for lymphocytes. In that study, eosinophil count was significantly higher in the milk of atopic women (4%) than in that of nonatopic mothers. Their function in human milk is unknown.

Basophils: Basophils are seldom found in the milk (0.1% of cells) of atopic mothers (Vassella *et al.* 1992). Their number is positively correlated with the number of eosinophils in peripheral blood, with a tendency towards higher basophil counts in the milk of atopic women. The authors suggest that the capacity of basophils to release histamine in the gut of the infant might increase the permeability of the gut mucosa and probably the risk for sensitisation to food allergens.

Other cells: Occasional epithelial cells appear in human milk (Crago *et al.* 1979, Ho *et al.* 1979).

1.2.2 Soluble Mediators

Cytokines are glycoproteins, secreted predominantly by activated T cells, monocytes, and macrophages that have effects on a variety of cells of the immune system and also on numerous other cells and systems throughout the body. Human milk contains cytokines such as interleukin (IL)-1 (Söder 1987), IL-4 (Eglington *et al.* 1994), IL-5 (Böttcher *et al.*

2000a), IL-6 (Saito *et al.* 1991), IL-8 (Basolo *et al.* 1993), IL-10 (Garofalo *et al.* 1995), IL-12 (Bryan *et al.* 1999), IL-13 (Böttcher *et al.* 2000a), IL-16 (Böttcher *et al.* 2000b), IFN-γ (Basolo *et al.* 1993), tumour necrosis factor (TNF)-α (Rudloff *et al.* 1992), and TGF-β (Noda *et al.* 1984). In addition, human milk mononuclear cells have also been demonstrated to show a potential, upon stimulation, for production of lymphokines such as IL-2 and IL-3 (Skansén-Saphir *et al.* 1993). Furthermore, maternal cells in human milk have been found to contain mRNA for various cytokines, revealing a unique cytokine profile for human milk (Srivastava *et al.* 1996).

1.2.3 Immunoglobulins, Lactoferrin and Oligosaccharides

Immunoglobulins: The immunoglobulins are glycoproteins with oligosaccharide sequences attached to their heavy and infrequently to their light chains. Human milk contains appreciable amounts of the IgA, IgG, and IgM (Xanthou *et al.* 1995). Small amounts of IgD and IgE classes have also been detected in milk (Xanthou *et al.* 1995, Duchén and Björkstén 1996). IgG, IgM, and IgD in milk seem to mediate antibody-dependent cytotoxicity and opsonic activity against bacteria (Xanthou *et al.* 1995).

IgE combines with antigens in the gut lumen and releases chemical mediators that cause increased vascular permeability (Xanthou *et al.* 1995) IgA makes up about 90% of human milk immunoglobulins. In common with other secretions such as nasal fluid and saliva, the chief immunoglobulin of human milk is of the secretory type of IgA (sIgA). It is present at its highest concentrations in the first few days postpartum (in the colostrum) (Hanson *et*

al. 1975), and then falls away progressively to a basal level of 0.2 to 0.3 g/L (Savilahti *et al.* 1991, Machtinger and Moss 1986). A number of sIgA antibodies to the common bacteria, viruses, and fungi to which the mother has been exposed have been described in human milk (Ogra *et al.* 1983). Studies have also established the presence of IgA antibodies in human milk to food proteins such as black beans and soybean (Cruz *et al.* 1981), cow's milk proteins (McClelland and McDonald 1976, Hanson *et al.* 1977, Machtinger and Moss 1986, Savilahti *et al.* 1991), and gliadin (Mascart-Lemone et al. 1991). Breastmilk IgA to casein or whole milk has been detected in 84% of milk donors (Machtinger and Moss 1986).

Human milk sIgA is produced locally in the lactating mammary gland, being elaborated by B cells situated proximal to the ductal epithelium. These local plasmacytes are derived from the gut-associated lymphoid tissue where they have been exposed to specific enteric antigens such as microbes and foods, and thus migrate to various mucosal sites and exocrine glands like the breast (Hanson *et al.* 1979, Slade and Schwatz 1987). The antibodies bind to the polymeric Ig-receptor, or secretory component, on the basal portion of the glandular epithelial cells, are transported through them, and appear on the mucosal membrane (Hanson 1998). There is evidence that also those lymphocytes shed into the colostrum are capable of producing immunoglobulin (Slade and Schwartz 1987, Murillo and Goldman 1970). Levels of the milk IgA antibodies to food antigens have not been shown to be influenced by the antigenic load in the mother's diet (Mascart-Lemone *et al.* 1991).

IgA antibodies in colostrum and human milk appear to be particularly important during the first few days of life, when the infant's mucosal IgA production is deficient (Hanson *et al.* 1977, Perkkiö and Savilahti 1980). After ingestion, maternal milk IgA antibodies have been suggested passively to protect the infant by reducing or preventing antigen entry across the immature gastrointestinal epithelium (Walker 1979). In an experimental model, Walker *et al.* (1975) have shown that intestinal antibodies can prevent resorption of native proteins and instead increase the uptake of degraded material, probably by binding the antigens and exposing them to intestinal enzymes. IgA antibodies may also play a role in excluding potential food allergens from human milk by forming immune complexes that can be phagocytosed by human milk macrophages (Walker 1979).

Lactoferrin: Lactoferrin is a 80-kDa single-chain glycoprotein produced by epithelial cells, neutrophils, and mononuclear phagocytes. It is the principal whey protein in human milk, and more than 80% of the protein is in apo form. Its concentration in human milk ranges from about 7 g/l in colostrum to about 1 g/l in mature milk (Masson and Heremans 1971). Because apolactoferrin binds ferric ions (Masson and Heremans 1966), it is able to compete with the iron-binding properties of bacteria. As a result, microorganisms cease to multiply (Stephens *et al.* 1980), contributing to the anti-infective properties of human milk.

Oligosaccharides: Human milk, compared with milk from other species, is unique, because of its high concentration of complex

oligosaccharides (Kunz and Rudloff 1993). Their antiadhesive qualities very effectively reduce bacterial and viral adherence in the upper respiratory and gastrointestinal tracts (Zopf and Roth 1996). In addition, by facilitating receptor glycosylation, they may facilitate attachment to intestinal epithelium or entry into the circulation of bioactive factors such as TGF-β (Pabst 1997).

1.2.4 Dietary Protein Antigens

It is widely accepted that potentially allergenic macromolecules are absorbed by the normal adult gut and transmitted in human milk (Kilshaw and Cant 1984, Stuart *et al.* 1984, Chandra *et al.* 1986, Machtinger and Moss 1986, Sorva and Mäkinen-Kiljunen 1994). Ovalbumin has been found in human milk at maximal levels 4 or 6 h after ingestion, and is of normal molecular size and indistinguishable from native ovalbumin by the radioimmunoassay (Kilshaw and Cant 1984). The same study also detected egg-derived ovomucoid in milk.

β-lactoglobulin (BLG) has been regarded as one of the most important proteins causing symptoms of CMA, and has been shown by enzyme-linked immunosorbent assay (ELISA) to be present in human milk in concentrations up to 16 μg/l (Kilshaw and Cant 1984, Stuart *et al.* 1984, Chandra *et al.* 1986, Sorva and Mäkinen-Kiljunen 1994).With a highly sensitive method having a detection limit of 0.002 μg/l, BLG was found in the milk of 75% of mothers consuming cow's milk (Sorva and Mäkinen-Kiljunen 1994). In earlier studies the maximal BLG level was detected after

8 to 12 hours of milk intake and demonstrated to vary inter- and intraindividually (Axelsson *et al.* 1986, Høst *et al.* 1990). In work by Sorva and Mäkinen-Kiljunen (1994) BLG was found in 1 or 2 hours or at both times after an oral cow's milk load in half of the samples. Casein has also been detected in human milk from half of the mothers tested on a cow's milk-containing diet by a sensitive ELISA (Stuart *et al.* 1984, Chandra *et al.* 1986). It has been demonstrated for BLG that human milk may contain intact protein, not only immunologically active peptides (Kilshaw and Cant 1984).

Several groups report the disappearance of symptoms in infants already sensitized upon abstinence of the mother from certain foods and the subsequent reappearance of symptoms in the infants upon their reintroduction to the mother's diet (Gerrard 1979, Sorva and Mäkinen-Kiljunen 1994). Several studies suggested that cow's milk protein in human milk can cause infantile colic (Jakobsson and Lindberg 1978, Iacono *et al.* 1991, Lucassen *et al.* 1998). These reports indicate such small amounts of food proteins or their split products can bring out the symptoms of food allergy in the suckling infant. Gerrard (1979) reported that as little as 5 ml of cow's milk ingested by the mother caused appearance of symptoms of CMA in the breast-fed.

1.3 Nutritional and non-nutritional components

Breastmilk contains all of the nutrients needed by the newborn baby during the first weeks of life. These include the metabolic fuels (fat, protein,

carbohydrate), water, and the raw materials for tissue growth and development, such as fatty acids, amino acids, minerals, vitamins, and trace elements.

More than 98% of the fat in breastmilk is in the form of triglycerides, constructed within the mammary epithelial cell from medium- and long-chain fatty acids derived either from the maternal circulation (carbon chain lengths ≤ 16) or manufactured locally (carbon chain lengths ≥ 16) (Jensen 1989). Short-chain fatty acids (carbon chain length ≤ 8) are only present in trace amounts. Oleic acid and palmitic acid are the most abundant fatty acids in breastmilk triglycerides, with comparatively high proportions of the essential fatty acids, linoleic acid and linolenic acid. Comparatively high proportions of other long-chain polyunsaturated fatty acids, such as arachidonic acid and docosahexaenoic acid, are also present (Jensen 1989). These long-chain fatty acids are constituents of brain and neural tissue and are needed in early life for mental and visual development (Ballabriga 1994). At least half of the triglyceride molecules in breastmilk contain palmitic acid attached to the central carbon of the glycerol component, a property that increases digestibility, absorption, and mineral balance (Jensen 1989, Carnielli *et al.* 1995). The lipid component of breastmilk is the transport vehicle for fat-soluble micronutrients such as prostaglandins and vitamins A, D, E, and K.

Proteins account for approximately 75 % of the nitrogen-containing compounds in breastmilk. Non-protein nitrogen substances include urea,

nucleotides, peptides, free amino acids, and DNA. The proteins of breastmilk can be divided into two categories: micellar caseins and aqueous whey proteins, present in the ratio of about 40:60 (Lonnerdal 1985). The predominant casein of human milk is β-casein, which forms micelles of relatively small volume and produces a soft, flocculent curd in the infant's stomach. The major whey proteins are α-lactalbumin, lactoferrin, secretory IgA, and serum albumin (Lonnerdal 1985), with a large number of other proteins present in smaller amounts. Secretory IgA is the principal immunoglobulin of breastmilk. It is synthesized in the mammary epithelial cell by the coupling of two IgA molecules, produced locally by lymphocytes resident in the breast tissue, with two proteins, J-chain and secretory component (Lonnerdal 1985). The specificity of breastmilk secretory IgA antibodies reflects the mother's exposure to mucosal infection and is independent of the specificity profile of blood-borne IgA (Mata 1986). Many of the proteins in breastmilk have a multitude of potential functions. Lactoferrin, for example, transports and promotes the absorption of iron, is bacteriostatic to a range of organisms, and acts as a nutritional protein, producing amino acids for absorption on digestion (Lonnerdal 1985, Prentice *et al.* 1987).

The principal carbohydrate of human milk is lactose, a β-disaccharide manufactured in the mammary epithelial cell from glucose by a reaction involving α-lactalbumin (Mepham 1987). In addition, breastmilk contains significant quantities of oligosaccharides, predominantly lactose-N-tetraose and its monofucosylated derivatives, representing about 10% of total milk

carbohydrate. The oligosaccharide composition reflects the Lewis blood group and secretor status of the mother (Kunz and Rudloff 1994).

In addition to the nutritional components, breastmilk contains a wealth of bioactive components that may have beneficial non-nutritional functions (Lonnerdal 1985, Mata 1986, Koldovsky 1994, Goldman and Goldblum 1995). These include a wide range of specific and non-specific antimicrobial factors; cytokines and anti-inflammatory substances; and hormones, growth modulators, and digestive enzymes, many of which have multiple activities. These components may be of particular importance for young infants because of the immaturity of the host defence and digestive systems early in life. The physiological significance of many of these substances has yet to be determined, and some may be present merely as "spillover" or excretory products from metabolic processes occurring within the mammary epithelial cell. For those with established significance, the site of action may be within the mother's breast, within the infant's alimentary canal, or, after absorption, within the infant's body. Some antimicrobial components, for example, are active both within the breast, minimizing the risk of breast infection and mastitis (Prentice *et al.* 1985), and within the baby's gastrointestinal and respiratory tracts, protecting the mucosal surfaces from infection by bacteria, viruses, and parasites (Mata 1986). By contrast, the site of action of the peptide feedback inhibitor of lacation (FIL) is within the breast, its function being the autocrine regulation of milk production (Wilde *et al.* 1995). On the other hand, casomorphins, opioid-like substances that may affect infant behaviour and mood in addition to a range of other

functions, are produced in the baby's intestines by the degradation of breastmilk casein (Schusdziarra 1992). Many bioactive substances are also valuable nutrient sources and ultimately are digested and absorbed in the normal way. Protease inhibitors in breastmilk may afford a degree of protection from digestion for some breastmilk components (Lonnerdal 1985). A sufficient proportion of antimicrobial proteins, for example, escape digestion and emerge in the faeces, suggesting that antimicrobial activity continues throughout the length of the infant's gastrointestinal tract (Prentice *et al.* 1987).

Breastmilk has also been shown to be an excretory route for a range of substances that might be harmful to the baby. These include viruses, such as human immunodeficiency virus (HIV) (Stiehm 1992); environmental and occupational pollutants, such as DDT, PCBs, and dioxins (Astrup-Jensen 1988); components of the mother's diet that might be toxic or allergenic, such as trans-fatty acids, aflatoxins, and cow's milk protein (Chappell *et al.* 1985, Host *et al.* 1988, Zarba and Groopman 1992); commonly used stimulants, such as nicotine, caffeine, and theobromine (Berlin 1981, Dahlstrom *et al.* 1990); and various drugs and radioactive compounds (DiLallo *et al.* 1987, Bennett 1988, Lazarus and Edwards 1988). Where exposure to xenobiotics jeopardizes infant health, difficult and often controversial decisions have to be made about whether the risks outweigh the benefits of breastfeeding (Bennett 1988, Cutting 1993).

1.4 Benefit of Breast Feeding

Benefits of breastfeeding to both the infant and the mother are greater when breastfeeding continues for longer amounts of time and remains exclusive for the first sixmonths. Breastfeeding is important due to the many health benefits to both the mother and child. The benefits for infants are believed to lie in four general areas: optimal growth and nutrition, defense against infections, the enhancement of maternal-infant bonding, and avoidance of allergic diseases (Cunningham 1979). Human milk provides the newborn with nutrients, growth factors, and anti-infectious substances important for host defense against infections (Goldman *et al.* 1982). During past decades, breastfeeding has been suggested to account for the decrease in number and severity of infants' intestinal and respiratory infections (Cunningham 1979), as well as of septicemia and meningitis (Winberg and Wessner 1971). The protective activity of breastfeeding against infections is explained by antimicrobial factors such as the immunoglobulins, leucocytes, lysozyme, lactoferrin, and bifidus factor present in human milk and a lessened risk of contamination with pathogenic microorganisms, particularly when hygiene is poor. Although knowledge is still lacking, breastfeeding may impart specific immune advantages to the neonate through enhancement or induction of the still-developing neonatal immune system (Slade and Schwartz 1987).

Breastfeeding and human milk afford to the infant a number of benefits. Prolonged breastfeeding has been recommended to prevent or delay

the development of atopic disease (Businco *et al.* 1983, Chandra *et al.* 1985, Businco *et al.* 1987, Høst *et al.* 1988, Zeiger *et al.* 1989, Saarinen and Kajosaari 1995, Oddy *et al.* 1999).

Colostrum and milk contain numerous potentially immunologically active components such as leucocytes, cytokines, and immunoglobulins (Slade and Schwartz 1987, Xanthou *et al.* 1995, Wagner *et al.* 1996, Goldman *et al.* 1997, Bernt and Walker 1999, Hanson 2000). A breast-fed infant ingests an average of 10^8 leucocytes per day with breastfeeding, often continuing for several weeks. Maternal cells and cytokines may reside biologically intact in the gut of the breast-fed infant due to special characteristics of human milk cells and of the newborns gastrointestinal tract. Human milk is believed to impart specific immune advantages to the neonate through enhancement or induction of the still-developing neonatal immune system (Slade and Schwartz 1987, Xanthou *et al.* 1995, Wagner *et al.* 1996, Cummins and Thompson 1997, Bernt and Walker 1999, Goldman 2000, Hanson 2000).

1.4.1 Benefits for Children

Parents who breastfeed their children are rewarded with a lengthy list of benefits. Breastfed children exhibit greater resistance to infectious disease and stronger immune systems than their formula fed peers. They also experience lower rates of chronic diseases. The ideal composition of human milk provides nutritional, growth, and developmental advantages to the child (USDHHS 2000).

1.4.1.1 Lower Rates of Infectious Disease

Research has repeatedly demonstrated lower rates of infectious diseases among breastfed babies. The explanation for this enhanced resistance to disease can be found in the biology of human milk. When a lactating mother is exposed to an infectious agent, her mature immune system begins to produce secretory immunoglobulin A [S-IgA], a compound that is the primary disease fighter in the human immune system. This substance is secreted into her breastmilk and consumed by her nursing baby. The child's own immune system may also be producing S-IgA, but children under the age of two have immature immune responses (USDHHS, 2000) that are sometimes incapable of preventing disease. The consumption of the mother's S-IgA not only provides active resistance to disease, it also stimulates the production of additional S-IgA in the infant, resulting in stronger immune responses among breastfed infants than in their formula fed peers (Cunningham, 1995).

Gastroenteritis, or the family of digestive diseases whose primary symptom is diarrhea, occurs less often among breastfed children and is less severe when it does occur. Howie *et al.* (1990) studied rates of gastrointestinal illness in infants in Dundee, U.K. The mothers were recruited while pregnant and were visited at home by a nurse at 2 weeks postpartum and again at 1, 2, 3, 4, 6, 9, 12, 15, 18, 21 and 24 months. Health and illness data were reported based on the mother's recollection of her child's health as well as information on the method of infant feeding. The

prevalence of gastrointestinal disease among fully breastfed infants (defined as infants who received no supplemental nutrition until at least 13 weeks of age) was 2.1%, whereas 19.5% of the formula fed babies suffered diarrhea. This trend continued throughout the first year even if the child was weaned at 13 weeks (Howie *et al.* 1990).

Howie *et al.* (1990) also analyzed data on respiratory tract infections in the same study. Respiratory tract infections were observed in 23% of the breastfed and 38.9% of bottlefed infants. This finding suggests that breastfeeding is beneficial in preventing respiratory tract infections.

Another common childhood infection, otitis media or ear infection, was also found to occur less often among breastfed infants in an Arizona-based cohort study. Data were gathered from the health records of a health maintenance organization. Infant feeding method was determined from health records or from questionnaires sent to parents. Infants were divided into five groups: no breastfeeding, breastfeeding less than 4 months, breastfeeding plus supplemental formula begun before 4 months, breastfeeding plus supplements begun between 4 and 6 months, and exclusive breastfeeding until at least 6 months. After controlling for potential confounding variables, such as family history of allergy, use of day care and maternal smoking, the researchers found a clear trend toward fewer episodes of otitis media in breastfed infants. The data illustrated a dose-response relationship with a decreasing risk for infants who were exclusively breastfed for longer periods, peaking with a 61% reduction in risk for those

infants breastfed exclusively for at least 6 months. The researchers proposed two possible explanations for this protective effect. The mechanics of breastfeeding are significantly different than those of bottlefeeding and result in better drainage of fluids. Also, the general protection from infection offered by breastfeeding may aid in the prevention of otitis media (Duncan *et al.* 1993).

A study of risk factors for antibiotic-resistant pneumonia revealed that breastfeeding also protects children from this infectious disease. This case-control study analyzed the rates of antibiotic-resistant pneumonia in children ages 2 – 59 months in North America. Parents were interviewed by telephone regarding various risk factors including the diet of the child. Interviews were based on a standard questionnaire and were conducted by experienced surveillance personnel. Current breastfeeding proved to be a strong protective factor against invasive pneumococcal disease among 2- to 11-month olds, reducing their risk for this disease by 73%. The study did not analyze the impact of past infant feeding choices on current health, nor did it propose an explanation for reduced risk among children currently being breastfed (Levine *et al.* 1999).

The protective effect of breastfeeding against infection also extends to the urinary tract according to a study of infants up to 6 months of age. Researchers conducted a case-control study with participants recruited from the hospital of the Medical School of Naples, in Italy. Information on feeding method was collected from clinical charts. Infants were divided into

groups based on the extent and exclusivity of breastfeeding. The study found that ever being breastfed reduced an infant's risk of contracting a urinary tract infection (UTI) by 62%. Current breastfeeding exhibited a stronger protective effect with an 82% risk reduction. The researchers concluded that "breastfeeding seems to protect against UTI during the first 6 months of life" (Pisacane *et al.* 1992).

1.4.1.2 Lower Rates of Chronic Disease

The preceding studies demonstrate the protective effect breastfeeding offers from infectious disease. Recent research also indicates that breastfed infants suffer lower rates of chronic disease. Since human milk is ideally composed for the infant, certain metabolic diseases are less likely to occur in breastfed infants (Cunningham 1995, Pettitt *et al.* 1997). Chronic digestive and respiratory diseases are also less common (Cunningham, 1995; Oddy *et al.*, 1999), and studies show that breastfeeding reduces risk for childhood cancer (Shu *et al.* 1999).

Type 1 diabetes (previously known as juvenile diabetes) has become more prevalent as rates of breastfeeding have decreased. Twenty-five percent of Type 1 diabetes cases are directly attributable to a lack of breastfeeding. Research shows that a primary trigger for the development of type 1 diabetes in susceptible children is exposure to cow's milk protein. A particular protein fragment which is found in cow's milk and cow's milk-based formula stimulates an immune response, but the structure of this protein is similar enough to that of the human system that antibodies

produced to fight the bovine protein end up destroying human pancreatic beta cells as well. This autoimmune response worsens over time until clinical diabetes is developed (Cunningham 1995).

Type 2 diabetes (formerly known as adult onset diabetes) is also less likely to occur when a history of breastfeeding exists. A study of Pima Indians, a population with very high rates of type 2 diabetes, uncovered an association between breastfeeding and reduced risk for the disease. Trained interviewers questioned the mothers of study participants regarding infant feeding choices from the subjects' childhoods. Participants were classified as exclusively breastfed, partially breastfed or exclusively bottlefed. The study analyzed rates by age and weight. In all age and weight ranges, participants who were exclusively breastfed until at least four months of age showed lower rates of type 2 diabetes. Even some breastfeeding demonstrated a protective effect as the highest rates of disease were found when a history of exclusive bottlefeeding existed. The researchers noted that subjects with a history of breastfeeding showed lower rates of obesity and suggested that this factor could explain the protective effect of breastfeeding against type 2 diabetes (Pettitt *et al.* 1997).

As with type 1 diabetes, several chronic digestive diseases have been linked to early exposure to cow's milk proteins. Inflammatory bowel disease, Crohn's disease and celiac disease are intestinal conditions that stem from immunological issues. Signs of these conditions have been detected as early as 2 weeks of age in bottlefed infants. The occurrence of these diseases has

been strongly associated with lack of breastfeeding or with its early termination. Research does not conclusively indicate whether these diseases stem from early exposure to foreign antigens, from the lack of protections found in human milk, or from some combination of both factors, but breastfeeding was once again observed to be a protective factor (Cunningham 1995).

Another example of the dose-response relationship between breastfeeding and chronic disease has been reported for childhood leukemia. Researchers using a case-control design analyzed a large number of childhood leukemia cases for risk factors in an effort to determine causes of this disease. Infant feeding method was determined through a structured telephone interview conducted with the subjects' mothers. Participants were classified as not primarily breastfed, breastfed 1-6 months and breastfed longer than six months. A 21% risk reduction was seen in children who were ever primarily breastfed, but this protection increased as breastfeeding duration increased. The risk of acute leukemia was reduced by 43% if a child was primarily breastfed for longer than 6 months. The authors theorized that the stronger immune systems of breastfed children might account for their reduced risk (Shu *et al.* 1999).

Stronger immune systems may also help protect children from chronic respiratory conditions. Asthma and allergies, chronic respiratory diseases typically diagnosed during childhood, are observed less often in children with a history of breastfeeding. In one study over 2000 Australian children were

followed from birth to six years to determine incidence of these diseases. Infant feeding method was determined by a questionnaire that was completed by parents when their children were one year old. History of breastfeeding was categorized by both duration of any breastfeeding and duration of exclusive breastfeeding. Children who were exposed to cow's milk- or soy-based formulas before four months of age were 25% more likely to have diagnosed asthma, 40% more likely to have wheezed three times or more in the past year, and 30% more likely to have had a positive skin test for allergies by the age of six years. Duration of exclusive breastfeeding proved to be a stronger protective factor than the duration of breastfeeding. The researchers concluded that interventions promoting an increased duration of exclusive breastfeeding may help to reduce the morbidity and prevalence of childhood asthma (Oddy *et al.* 1999).

In addition to offering protection from a wide range of infectious and chronic diseases, breastfeeding has the potential to improve a child's future health by reducing the risk of being overweight or obese. A cross sectional study conducted in Germany assessed height and weight measurements of over 100,000 children. Infant feeding method was further investigated on a subset of nearly 10,000. Breastfeeding exposure was classified as none, less than two months, three to five months, six to twelve months or greater than twelve months. After adjusting for confounding factors including social class and parental education, a dose-response relationship between exclusive breastfeeding and protection from being overweight or obese emerged. For the longest period of exclusive breastfeeding studied, 12 months or greater,

risk of overweight was reduced by 57% and risk of obesity was reduced by 72%. The researchers suggested that the respect for infants' satiety signals normally developed during breastfeeding helped parents to offer appropriate portions to their children, and that the lower intake of protein among breastfed infants also contributed to the decreased risk (von Kries *et al.* 1999).

1.4.2 Benefits to Mother

The mother also benefits when she breastfeeds her baby. She is less likely to develop pre-menopausal breast, uterine, and ovarian cancers. She will lose weight faster following delivery and have a lower chance of developing diabetes, arthritis and osteoporosis (Huggins 1999, Riordan and Auerbach 1999, Riordan 2005, LLLI 2004, AAP 2005). Hormones released during breastfeeding, such as prolactin, may promote maternal behaviors and reduce vulnerability to stress (Else-Quest *et al.* 2003, Britton *et al.* 2006, Ekstrom and Nissen 2006, Gribble, 2006).

Breastfeeding also helps mother in various ways. Suckling soon after birth helps in early expulsion of placenta through oxytocin reflex. This decreases postpartum haemorrhage. Continuing breastteeding helps early involution of the uterus. Breastfeeding helps in birth spacing by its contraceptive effects. A study in rural Bangladesh have shown that the pattern of infant feeding affect the length of postpartum amenorrhoaa (Ford and Huffman 1988). It has been claimed that breastfeeding may prevent more pregnancies than all family planning programmes in the world (Hanson 1998). Breastfeeding protect mother from breast and ovarian cancer.

In Bangladesh, mean lactational period was shown to be 25 months, lactational amenorrhoea 19 months and total birth interval 36 months (Chowdhury *et al.* 1977). However, it should be remembered that 6.68, 7.00, 2.50, and 2.26% of lactating mothers in Bangladesh, India, Philippines and USA respectively, may become pregnant before menstruation is resumed (Ginneken 1977). These mothers may not be exclusively breastfeeding, however. Lactational amenorrhoea of the mother permit her to recover her iron store, correct anaemia and enhances her immune and nutritional status. breastfeeding prevents obesity in mothers. Lactation is an anti diabetic factor. Some lactating mothers enjoy remission of the diabetic state. A feeling of wellbeing is a more common phenomenon in diabetic mother during lactation (Lawrence 1989).

1.4.3 Other Health Benefits

Choosing to breastfeed can also have a significant impact on cancer rates. First, it has been demonstrated that a female infant who is breastfed can expect a 25% reduction in risk for breast cancer later in life (USDHHS 2000). Also, a large case-control study of breast cancer patients in several U.S. states shows a reduction in risk when a woman has a history of lactating for 24 cumulative months over the course of her childbearing years. More than 5800 cases and 8200 controls were interviewed by telephone regarding their lactation and reproductive history. After adjusting for confounding factors, breastfeeding for at least 24 months showed a risk reduction of 28% for premenopausal breast cancer. Early initiation of breastfeeding strengthened this protection with an odds ratio of 0.54 for women whose first period of lactation started before 20 years of age. The

researchers suggest that lactation "may reduce the risk of breast cancer simply by interrupting ovulation or by modifying pituitary and ovarian hormone secretion" (Newcomb *et al.* 1994).

1.4.4 Benefits to the Family

Breastfeeding also helps the family economically. Because of improper breastfeeding practices in Bangladesh, the total loss has been quantified at approximately US$1 billion per year, which is about 2 percent of national produce (USBC 2002). It has been estimated that exclusive artificial feeding will cost about Taka 1600-2100 per month. If the cost of cleaning, bottle, nipple sterilization and time spent in doing these are considered, this will increase the cost further. Thus, the cost will be about the monthly pay of a low-paid staff or a regular paid labourer which means it unaffordable.

1.4.5 Benefits to the Nation

Breastfeeding offers important economic advantages to national budgets, family planning programmes, hospital expenditures. The average monthly cost of formula in developing countries for a 6-month-old infant comes to US$22, an amount equal to a greater than the average monthly per capita income in many countries (Berg and Brem 1989). At a hospital in the Philippines, promotion of breastfeeding resulted in an annual saving of more than US$100,000 (Gonzales 1990). In the New York, the total cost of hospital treatment of bottle-fed babies during their first four months is 15 times the cost of treating breastfed babies (Berg and Brem 1989).

Import of breast milk substitute in Bangladesh from abroad drain the valuable currency equivalent to 3200 crore taka per year that could have been saved by exclusive breastfeeding. Contraceptive effect also helps national economy if it is used optimally. In Indonesia, contraceptive effect of breast feeding has been estimated to be equivalent to entire national family planning programming without breast feeding, an additional 80 million US$ would have to be spent to achieve the same contraceptive effect.

1.4.6 Economic Benefits

Clearly the choice to provide breastmilk to an infant provides a substantial health benefit to the child, but that choice will also result in an economic benefit for the family. Based on infant formula prices, the cost of feeding a child artificial baby milk (ABM) will total $1200 - $2700 per year, depending on the preparation purchased (Baumslag and Michels 1995). When these numbers are extended to include large numbers of children, such as those serviced by Women, Infants and Children (WIC) clinics, the savings become impressive. WIC spent $661.9 million in its 2001 fiscal year on ABM, whereas breastmilk is available for the slight additional cost of food to meet the mother's increased nutritional needs, less than $10 per month for WIC clients in 2001 (USDA 2003). When the USDA estimated the cost savings of increasing breastfeeding rates to the goals set in HP2010, they considered the resulting reduced rates of only three diseases – otitis

media, gastroenteritis, and necrotizing enterocolitis – and estimated a yearly cost savings of $3.6 billion (USDA 2001).

1.5 Objectives of the Study

In settings with excessive child mortality and a strong association between breastfeeding and mortality, it is important to know if and how improved breastfeeding practices can reduce infant and child mortality. Therefore this study tries to touch these issues on the effect of breast feeding on child health.

The main objective of this research is to

1. Determine the effect of breastfeeding on child health.
2. Relationship between feeding patterns and child disease incidence.
3. Selected suitable complementary feeding and alternative feeding for child.
4. Relationship between some socioeconomic status and living status and child disease incidence.

Chapter 2

SUBJECTS AND METHODS

Chapter 2

SUBJECTS AND METHODS

2.1 Sample and Setting

The primary source of data for the present study was collected from the hospitalized children in Rajshahi Medical College Hospital (RMCH) during 2007 and 2008.

The data was collected from Pediatric Wards 10, 24, 27 and outpatients department (OPD) of RMCH. During selection of the patient random selection were followed taking two samples weekly having 15 subjects in each sample. The patients were chosen by taking alternate bed serial in the hospital ward and alternate registration serial in the OPD. The study population consisted of 800 children between ages 0-12 years. Eligible fathers and mothers of those children were interviewed and informations were collected. The food, feeding pattern, pathological, imaging test and their socio-economic characteristics were recorded. Antecedent histories of breast-feeding was taken to evaluate whether breast-feeding was associated with the risk of clinically severe diseases.

2.2 Study Procedure

Raw data were collected by survey method. A questionnaire was developed for self-administration after an extensive review of the

literature. The 12-item questionnaire were made among which ten questions were general about socio-economic, housing and educational status of parents; one question about different breast feeding patterns; and one question having two sub-questions comprising of 13 and 10 respectively, about the nature and pattern of different diseases.

Each question was scored individually according to the type of response. For example, about 'the frequency of breastfeeding' women were asked to write the number of times they breastfeed their baby in a 24 h period. Appropriate working procedure was followed for survey. Interviews were made from the interviewee and fill the questionnaire face-to-face method.

2.2.1 Variable selection

To perform the research work ten parameters were selected, each of them has some variables. The variables are coded and started from 0 or 1 and expended up to necessary number. In this research selected variables were:

a) Breastfeeding

b) Complementary feeding

c) Alternative feeding

d) Nature of food.

e) Fathers education

f) Mothers education

g) Economic status

h) Types of houses

i) Source of drinking water

j) Types of sanitation

2.2.2 Chi-square test statistic

The χ^2 test statistic which is used to examine the association between various factors which is as follows

$$\chi^2 = \sum_{i=1}^{r} \sum_{j=1}^{c} \frac{O_{ij}}{E_{ij}}^2 - N$$

Which follows χ^2 distributions with (r-1)(c-1) degrees of freedom. Where, r is the no. of rows, c is the no. of columns, O_{ij} be the observed number of observations, E_{ij} be the expected number of observations and N = total number of observations.

2.3 Definitions used in the study

AGN (Acute Glomerlo Nephritis): It is the classic example of acute nephritic syndrome, and was defined by sudden onset of gross hematuria, edema, hypertension and renal insufficiency.

ALL (Acute Lymphoblastic Luekaemia): Acute lymphoblastic luekaemia was defined as excessive and uncontrolled proliferation of the white blood cells especially, lymphocytes, results in anemia, bleeding manifestations, and infection. It is also called blood cancer.

Aplastic Anaemia: Aplastic anaemia was defined as pancytopenia with acellular or hypocellar bone marrow. This disease is characterized by anaemia, bleeding menifestation and infection.

AWD (Acute Wateny Diarrhoea): Acute wateny diarrhoea was defined as passage of three or more loose watery stool with or without vomiting per 24 hours is called diarrhoea.

BF up to 6 months: Breast feeding up to 6 months was the infant received breast milk only and no other solids or liquids with the exception of vitamins, minerals, medicines or oral rehydration solution It is also called exclusive breast feeding.

Bronchial asthma: Bronchial asthma was defined as a chronic inflamatory disorder causing hyper responsiveness of airways to certain stimuli resulting in variable airflow limitation at least partly reversible, presenting as wheeze, breathlessness, cough and chest tightness.

Bronchiolitis: Bronchiolitis was defined an acute respiratory illness due to inflamation of the bronchioles and is caused by different viruses. Occurs between 2 months to 2 years of life.

Complementary feeding: Complementary feeding included milk, infant formula, gruel or semi-solid foods given in addition to breast milk, normally started at 6 months of life.

Encephalitis: Encephalitis was defined as the inflamation of the brain caused by virus.

Enteric fever: Enteric fever was defined as an infectious disease also known as typhoid fever caused by *Salmonella typhi*.

Febrile convulsion: Febrile convulsion was defined as a result of rise of temperature due to extra cranial infection during the age of 6 months to 6 years. In this disease there may be positive family history.

GBS (Guillain Barre Syndrome): Guillain Barre Syndrome was defined as an acute post infections autoimmune polyradiculoneuropathy, that causes demyelination and or axonal degeneration in motor but sometimes in sensory and autonomic nerves, leading to weakness and occasionally sensory and autonomic disturbance.

Hemolytic Anaemia: Hemolytic anaemiea was defined as a result of hemolysis of blood. In this study hemolytic anemia means congenital.

Irregular BF: Irregular breast feeding was the baby got breast milk along with other drinks or foods since birth that is infant formula, cow's milk, sujii, shaboo etc.

Kala azar: Kala azar was defined as a chronic infectious disease caused by parasite *Leishmania donavani* characterized by fever for long days more than two weeks, pallor, gradual weight loss and organomegaly.

LBW: Low birth weight was defined as the birth weight of a newborn below 2.5 kg.

Meningitis: Meningitis was defined as inflammation of the meninges coverings the brain which is caused by bacteria, virus, fungus etc.

Neonatal sepsis: Neonatal sepsis was defined as systemic response to infection during neonatal period.

Nephrotic syndrome: Nephrotic syndrome was defined as the syndrome characterized by generalized swelling of the whole body, along with massive proteinuria, hypoalbuminemia and hypercholesterolemia.

No BF: No breast feeding was when the infant received no breast milk since birth to two years of age.

Pleural Effusion: Partial effusion was defined as the accumulation of excess quantity of fluid in the pleural space caused by bacterial pneumonia, malignancy, tuberculosis and viral infection.

Pneumonia: Pneumonia was defined as the constellation of signs and symptoms of an acute respiratory illness caused by bacteria and viruses and is characterised by fast breathing, chest indrawing and other sings of respiratory distress.

Preinatal asphyxia: Preinatal asphyxia was defined as an insult to the fetus or newborn infant due to lack of oxygen and or lack of perfusion to various organs which will manifest as difficulty in establishing spontaneous respiration evident by delayed cry after birth.

Preterm: Preterm or premature neonates were those who delivered before the thirty-seventh (37th) week from the first day of the last menstrual period.

Regular complementary feeding: Regular complementary feeding was defined a complementary feeding started at 6 months of life.

Rheumatic fever: Rhenmatic fever was defined as an inflamatory disease predominently of childhood (5-15 years of age), resulting from a systemic response which is a sequelae to group A, B hemolytic streptococci infection of pharynx, principally affecting heart, joints, central nervous system, skin and sub-cutuneous tissue.

Sufficient BF: Sufficient breast feeding was the infant got breast milk less than 6 months, and during that period no other milk or drinks was given.

Viral Hepatitis: Viral Hepatitis was defined as an acute inflamation of liver caused by different types of hepatotrophic viruses, of them hepatitis A, B, C, D, E are common. This disease is characterized by jaundice, abdominal pain, loss of appetite, vomiting and weakness.

2.4 Questionnaire used in the study

1. Name of the
   Child:.....
2. Address:.....
   ☐ Rural ☐ Urban ☐ Periurban
3. Age: ☐ (0-2 m) ☐ (3-6 m) ☐ (6m-1y) ☐ (1-2 y) ☐ (2-5y) ☐ (5-12 y)
4. a) Height.....cm b) Weight.....kg
5. Parental status:
   Father: A. Education level: ☐ Primary ☐ Secondary ☐ Higher ☐Others
   B. Socio-economic status:☐ Rich ☐ Medium ☐Poor ☐Very poor

Mother: A. Education level: ☐ Primary ☐ Secondary ☐ Higher ☐ Others No

B. Socio-economic status : ☐ Rich ☐ Medium ☐Poor ☐Very poor

6. Mother health: a)Age:..... b) Height.....cm c) Weight.....kg d) Lactation priod..... e) Next conception.....

7. Family type: a)Number of family member..... b)Number of child.....

8. Type of House: ☐ Pacca ☐ Kacha c) ☐ Other

9. Source of drinking water: a) ☐ Tube well b) ☐ Supply c) d) ☐ Other

10. Sanitation type: a) ☐ Sanitary b) ☐ Kacha c) ☐ Other

11. Food and Feeding pattern

Breast feeding: ☐ No breast feed ☐ Up to six month ☐ Irregular ☐ Sufficient

Complementary feeding: ☐ Regular ☐ Early started ☐ Late started

Alternative feeding: Doctors choice milk: ☐ Lactogen ☐ Myboy ☐ Biomeal ☐ Mother choice ☐ Mother smile ☐ cows milk

Nature of feed: ☐ Family diet ☐ Diluted milk ☐ Carbohydrate only ☐ Other

CURRENT DISEASE INVESTIGATION

12.A. General:

1. Level of Conscious semiconscious	:	☐ Conscious ☐ Unconscious ☐
2. Appearance	:	☐ Ill looking ☐ Normal looking
3. Pallor	:	☐ Mild ☐ moderate ☐ severe ☐ absent
4. Jaundice	:	☐ mild ☐ moderate ☐ severe ☐ absent
5. Edema	:	☐ absent ☐ present
6. Clubbing	:	☐ absent ☐ present
7. Koilonychias	:	☐ absent ☐ present
8. Cyanosis	:	☐ absent ☐ present
9. Skin pigmentation	:	☐ absent ☐ present
10. Lymphadenopathy	:	☐ absent ☐ present
11. Vital signs	:	i. Pulse:..... ii. B.P.:..... iii. Respiration rate:..... iv. Temperature:.....

12. Dehydration : ☐ no ☐ some ☐ severe
13. Any other remarkable sign :.....

12.B. Systemic

1. Organ (Palpable) : ☐ liver ☐ spleen ☐ Right kidney ☐ Left kidney ☐Urinary bladder
2. Abdomen : Distention: ☐ Present ☐ Absent
3. Genatalia & hernia : ☐ harnia ☐ hydrocle ☐ abnormal genetalia
4. Bowel sound : ☐ present ☐ absent
5. Cardiovascular system : i. Apex beat: Location ☐ normal ☐ deviated Types ☐ normal ☐ heaving ☐ tapping
   ii. Heart sounds ☐ normal ☐ audible ☐ not audible
   iii. Cardiac murmur: ☐ present ☐ absent
6. Respiratory system : i. Chest movement:☐ normal ☐ symmetrical ☐ asymmetrical
   ii. Chest expansibility: ☐ normal ☐ reduced
   iii. Vocal resonance: ☐ normal ☐ increased
   ☐ decreased
   iv. Breath sound: ☐ present ☐ absent
   v . Added sound: Ronchi: ☐ present ☐ absent
   Crepitation: ☐ present ☐ absent
7. Nervous system: Cerebral function:
   i. Appearance: ☐ Ill looking ☐ Normal looking

ii. Emotional state: ☐ Normal: ☐ abnormal

iii. General intelligence: ☐ Average ☐ below average

iv. Orientation: ☐ Oriented ☐ disoriented

8. Motor function

i. Bulk to muscle: ☐ Normal ☐ reduced ☐

ii. Tone of muscle: ☐ Normal ☐ reduced ☐increased

iii. Power: ☐Normal ☐reduced ☐increased

iv. Reflex: Ankle☐Normal☐reduced ☐exaggerated Knees:☐Normal ☐reduced☐exaggerated: Planter: ☐flexion ☐extension

v. Gait: ☐Abnormal ☐hemiplegics ☐ataxic

vi. Involuntary movement: ☐present ☐ absent

9. Sensory function

i. Tactile function: ☐Intact ☐absent

ii. Sense of position: ☐Intact ☐absent

iii. Sens of vibration: ☐Intact ☐absent

iv. Pain sensation: ☐Intact ☐absent

v. Temperature sensation: ☐Intact ☐absent

10. Locomotors system

i.Bulk to muscle: ☐Normal ☐reduced ☐hypertrophy

ii. Tone of muscle:☐Normal☐reduced ☐increased

iii. Power: ☐Normal ☐reduced ☐increased

iv. Reflex: Ankle☐Normal☐reduced ☐exaggerated: Knees:☐Normal ☐reduced☐exaggerated: Planter: ☐flexion ☐extension

v. Gait: ☐Abnormal ☐hemiplegic ☐ataxic

vi. Involuntary movement: ☐present ☐ absent

vii. Joint: Swelling ☐Tenderness☐ temperature

viii. Range of movement: ☐ Normal ☐ reduced

ix. Joint deformity : ☐present ☐ absent

11. Reported Genetical Diseases ☐ Hemolytic anemia (HA) ☐Myopathy ☐Diabetic ☐Hypertension☐Cardiac ☐Bronchial ☐Hemophilia

Chapter 3

RESULTS

Chapter 3

RESULTS

The results on the incidence of different diseases on the breast feeding patterns have been shown in Tables 1-12. Among 800 subjects no breast feeding patients were 197 (24.63%), breastfeeding up to six months were 139 (17.37%), irregular breastfeeding were 334 (41.75%) and sufficient breast feeding were 130 (16.25%). The different aspects of the findings are described in details below.

3.1 Breast feeding pattern and different disease incidence

The incidence of different diseases with relation of breast feeding pattern has been presented in Table 1. There were 209 pneumonia cases and among them 9.56% cases had no history of breast feeding, 7.18% cases were exclusive breastfeeding up to 6 months, 55.02% cases got irregularly breast milk and 28.23% cases got sufficient breast milk. The number of perinatal asphyxia children was 78. Regarding breastfeeding 71.79% did not get any breast milk (most of them were getting i/v nutrition as they were very much sick). Irregular breastfeeding cases were 21.79% and 6.42% patients were getting sufficient breast milk up to certain period. Here no cases were found who had exclusive breast feeding up to 6 months. The cases of preterm and low birth weight (LBW), neonatal sepsis and neonatal jaundice were 45, 38 and 27 babies. For these three diseases no case was recorded in exclusive breast feeding and sufficient breast feeding (Fig 1A).

In this study there were 40 patients of enteric fever (typhoid fever) and among them 30% did not get breast feeding, 30% got exclusive breastfeeding for 6 months, and 40% got irregular breastfeeding. Regarding meningitis out of 47 cases, 10.64% did not get breastfeeding. 21.28% got breastfeeding up to 6 months, 42.55% got irregular and 25.53% got sufficient breastfeeding. Encephalitis cases were 20, of them 10% had no history of breastfeeding, 40% got breastfeeding up to 6 months of age, 10% got irregular breast milk and 40% got sufficient breast milk (Fig. 1B).

Out of 46 bronchiolitis patients. 17.39% children never got breast milk, 32.61% of children got breastfeeding up to 6 months of age. another 32.61% of children got irregular and 17.39% of children got sufficient breastfeeding. There were 31 cases of nephrotic syndrome of which 6.45% did not get any breast milk. 29.03% got breastfeeding up to 6 months, 48.38% got irregular breastfeeding and 16.13% got sufficient breastfeeding. In this study there were 34 haemolytic anemia patients, regarding their breastfeeding status 2.94% did not get breast milk, 29.41% of cases got breast milk up to 6 months of age, and 44.11% got irregular breastfeeding and 23.53% cases got sufficient breastfeeding. Fifteen congenital heart diseases were found in this study and 6.66% of patients did not get breast feeding, 66.67% of cases got breast milk up to 6 months of life and 26.67% of cases got irregular breastfeeding (Fig. 1C).

ALL cases were 29, out of which 27.59% cases did not get breast feeding, 31.03% of cases got breast milk up to 6 months of ages. 34.48% of cases got breastfeeding irregular and 6.90% got sufficient breastfeeding.

Among 33 cases of AWD, 30.30% of patients did not get breast feeding. 27.27% of patients got breastfeeding up to 6 months, 30.30% of children got irregular breastfeeding and 12.12% of children got sufficient breastfeeding. Total kala-azar patients were 22, among them 9.09% of cases did not get breast feeding, 36.36% of cases got breast milk up to 6 months of ages and another 36.36% of cases got breastfeeding irregular and 18.18% of patients got sufficient breastfeeding. Ten cases of AGN patients were found in this study of them 40% got breastfeeding for 6 months, 30% got irregular and 30% got sufficient breastfeeding (Fig. 1D).

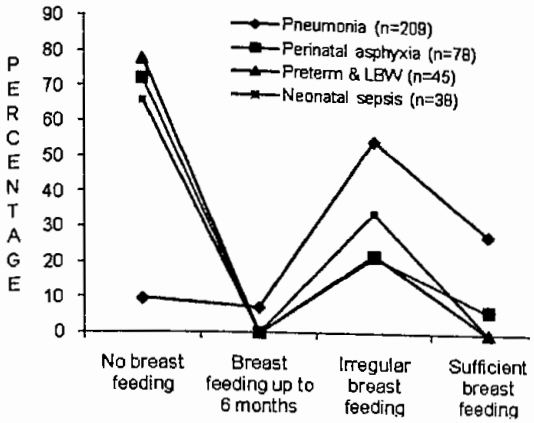
Viral hepatitis patients were 12 and regarding their breastfeeding status 16.66% of cases did not get any breast feeding, 25% of cases got breast milk up to 6 months of ages. 50% of cases got breastfeeding irregular and 8.33% of patients got sufficient breastfeeding. Again 10 cases of rheumatic fever was recorded, out of which 20% of patients got no breast milk, 30% of cases got breast feed up to 6 months of ages. 40% of cases got irregular and 10% of patients got sufficient breastfeeding. There were 11 cases of aplastic anaemia. Regarding their breastfeeding status 9.09% of cases did not get any breast feeding, 27.27% of cases got breast milk up to 6 months of ages. 45.45% of cases got breastfeeding irregularly and 18.18% of patients got sufficient breastfeeding. Bronchial asthma cases were 15. Regarding their breastfeeding status 6.67% of cases did not get any breast feeding, 20% of cases got breast milk up to 6 months of ages. 46.66% of cases got breastfeeding irregularly and 26.67% of patients got sufficient breastfeeding (Fig. 1E).

Fifteen cases of febrile convulsion were recorded of them 6.67% of cases did not get any breast feeding, 20% of cases got breast milk up to 6 months of age. 46.66% of cases got breastfeeding irregular and 26.67% of patients got sufficient breastfeeding. Among 6 cases of GBS 33.33% got breastfeeding up to 6 months of ages. 66.66% of cases got irregular breastfeeding. Among seven cases of pleural effusion 14.28% had no history of breastfeeding 42.86% of cases got breastfeeding up to 6 months of ages and another 42.86% of cases got breastfeeding irregular (Fig. 1F).

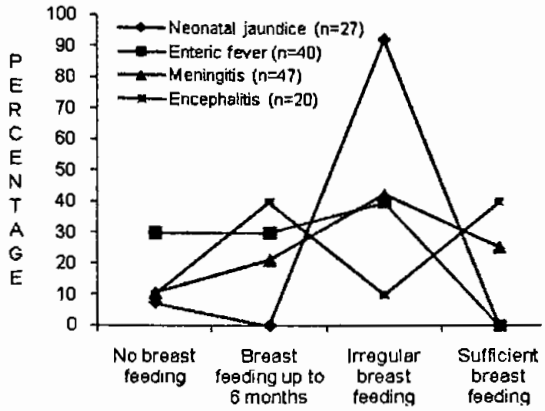
Form the chi-square value it is found that pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis, neonatal jaundice, encephalitis, congenital heart diseases were significantly ($p<0.001$) related with breastfeeding. Though perinatal asphyxia, preterm and LBW, neonatal sepsis and neonatal jaundice are neonatal diseases found within 4 weeks of life, so they were not found within breast feeding upto six months group. Due to absence of the patients in that group the calculation of chi-square value showed significant difference though the said diseases are not related with the breastfeeding. Enteric fever was significant at 1% level ($p<0.01$), whereas meningitis, bronchiolitis, hemolytic anaemia, AGN, AWD and GBS were significant at 5% level ($p<0.05$). Neohrotic syndrome, ALL, kala azar, viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma, febrile convulsion and pleural effusion were found not related with breastfeeding as those chi square vaules were found insignificant.

Table 1. Breast feeding pattern and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

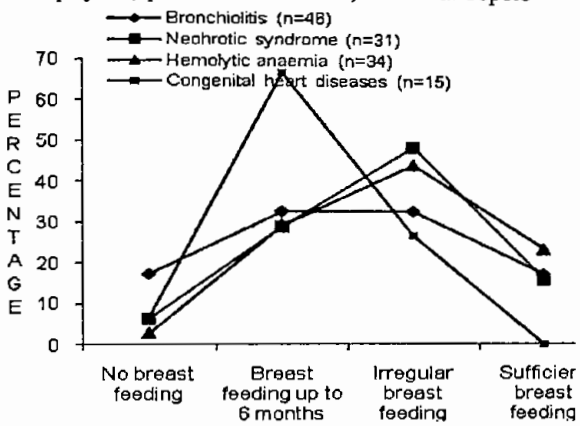
Diseases	No breast feeding	Breast feeding up to 6 months	Irregular breast feeding	Sufficient breast feeding	χ^2 (df)
Pneumonia (n=209)	20 (9.56)	15 (7.18)	115 (55.02)	59 (28.23)	79.90 (3)***
Perinatal asphyxia (n=78)	56 (71.79)	-	17 (21.79)	5 (6.42)	108.48 (2)***
Preterm & LBW (n=45)	35 (77.78)	-	10 (22.22)	-	75.09 (1)***
Neonatal sepsis (n=38)	25 (65.79)	-	13 (34.21)	-	41.41 (1)***
Neonatal jaundice (n=27)	2 (7.41)	-	25 (92.59)	-	30.20 (1)***
Enteric fever (n=40)	12 (30.00)	12 (30.00)	16 (40.00)	-	11.23 (2)**
Meningitis (n=47)	5 (10.64)	10 (21.28)	20 (42.55)	12 (25.53)	8.64 (3)*
Encephalitis (n=20)	2 (10.00)	8 (40.00)	2 (10.00)	8 (40.00)	19.90 (3)***
Bronchiolitis (n=46)	8 (17.39)	15 (32.61)	15 (32.61)	8 (17.39)	8.54 (3)*
Nephrotic syndrome n=31)	2 (6.45)	9 (29.03)	15 (48.38)	5 (16.13)	7.19 (3)ns
Hemolytic anaemia (n=34)	1 (2.94)	10 (29.41)	15 (44.11)	8 (23.53)	10.95 (3)*
Congenital heart diseases (n=15)	1 (6.66)	10 (66.67)	4 (26.67)	-	26.70 (2)***
ALL (n=29)	8 (27.59)	9 (31.03)	10 (34.48)	2 (6.90)	4.15 (3)ns
AWD (n=33)	10 (30.30)	9 (27.27)	10 (30.30)	4 (12.12)	8.83 (3)*
Kala azar (n=22)	2 (9.09)	8 (36.36)	8 (36.36)	4 (18.18)	7.12 (3)ns
AGN (n=10)	-	4 (40.00)	3 (30.30)	3 (30.30)	6.79 (2)*
Viral hepatitis (n=12)	2 (16.66)	3 (25.00)	6 (50.00)	1 (8.33)	1.39 (3)ns
Rheumatic fever (n=10)	2 (20.00)	3 (30.00)	4 (40.00)	1 (10.00)	1.27 (3)ns
Aplastic anaemia (n=11)	1 (9.09)	3 (27.27)	5 (45.45)	2 (18.18)	1.78 (3)ns
Bronchial asthma (n=15)	1 (6.67)	3 (20)	7 (46.66)	4 (26.67)	2.17 (3)ns
Febrile convulsion (n=15)	1 (6.67)	3 (20)	7 (46.66)	4 (26.67)	3.17 (3)ns
GBS (n=06)	-	2 (33.33)	4 (66.66)	-	4.25 (1)*
Pleural effusion (n=07)	1 (14.28)	3 (42.86)	3 (42.86)	-	4.10 (2)ns



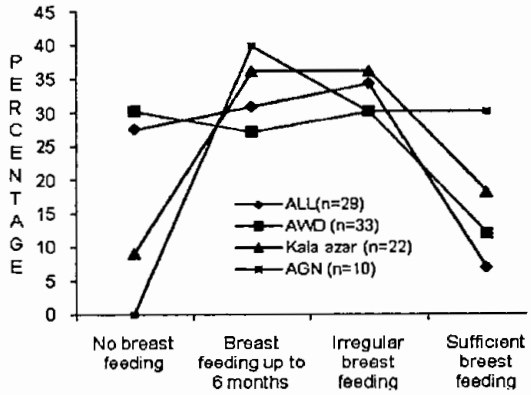
A. Breastfeeding and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis



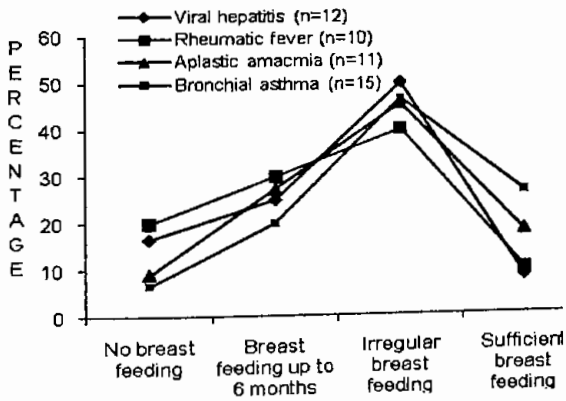
B. Breastfeeding and neonatal jaundice, enteric fever, meningitis, encephalitis



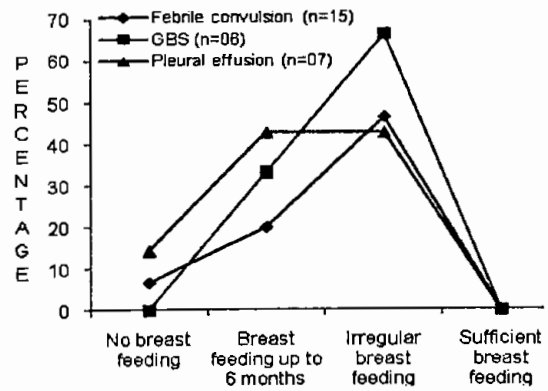
C. Breastfeeding and bronchiolitis, ncohrotic syndrome, haemolytic anaemia, congenital heart disease.



D. Breastfeeding and ALL, AWD, Kala azar and AGN



D. Breastfeeding and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma



E. Breastfeeding and febrile convulsion, GBS, pleural effusion

Fig. 1. Relation between breastfeeding patterns and incidence of diseases among children

3.2 Complementary feeding pattern and different disease incidence

The incidence of different diseases with relation of complementary feeding pattern has been presented in Table 2. Among 209 children of pneumonia in this study, regarding complementary feeding, 9.56% did not get any complementary food; 7.18% got regularly complementary food; 47.85% cases got early complementary food and 35.41% got complementary food late in infancy that is after 6 months of age. No complementary feeding was given in either of the patients of perinatal asphyxia, preterm and LBW, neonatal sepsis and neonatal jaundice because all of them were neonate that below six months of age. In this study there were 40 cases of enteric fever of them 30% did not get any complementary food, 30% got complementary food as regular basis, 25% started complementary feedings early period that is before 6 months of age and in 15% patients complementary feeding started at later period that is after 6 months. Regarding 47 meningitis cases 10.64% did not get any complementary feeding, 21.28% patients got regularly complementary feeding, 38.29% got early and 29.79% got complementary feeding in late stage. Among 20 encephalitis cases, 10% did not get any complementary food, 40% got regularly and 50% got early complementary feeding (Fig 2A).

Bronchiolitis patients were 46 out of which 17.39% did not get any complementary feeding, 32.61% got regular complementary feeding, 43.48% got early and 6.52% got late complementary feeding. Regarding

complementary feeding of 31 nephrotic syndrome patients 6.45% did not get any complementary feeding. 29.03% got regular complementary feeding, 38.71% got early complementary feedings, and 25.80% of cases got late complementary feeding. Among 34 hemolytic anaemia patients 2.94% did not get any complementary feeding, 29.41% of cases got regular complementary feeding, another 29.41% of cases got early complementary feeding, and 38.24% of patients got in late stage. There were 15 congenital heart disease patients, of them 6.66% did not get complementary feeding, 66.67% got regular complementary feeding. 20% got early and 6.66% of cases got late complementary feeding (Fig. 2B).

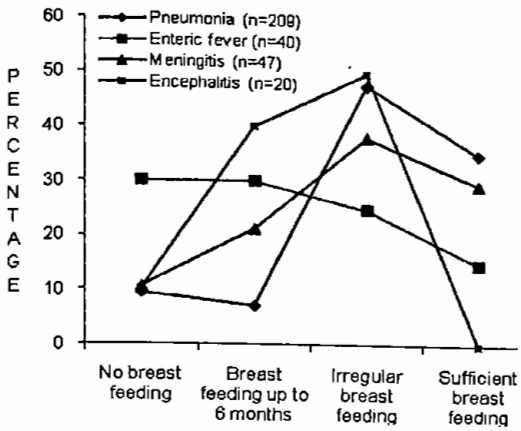
Among 29 ALL cases 27.59% did not get any complementary feeding, 31.03% of cases got regular complementary feeding, 27.59% got early and 13.79% got late complementary feeding. There were 33 AWD cases, out of which 30.30% of cases did not get any complementary feeding, 27.27% of patients got regular complementary feeding, 36.36% of patients got early and 6.06% of patients got late complementary feeding. Kala azar cases were recorded for 22 patients, of whom 9.09% did not get any complementary feeding, 36.36% of patients got regular complementary feeding, 27.27% of patients got early and another 27.27% of patients got late complementary feeding. Ten AGN cases were recorded, among them 40% got regular complementary feeding, 40% of patients got early and 20% of patients got late complementary feeding (Fig. 2C).

Viral hepatitis cases were 12, out of them 16.66% did not get complementary feeding, 25% got regular complementary feeding, 16.66% of patients got early and 41.67% of patients got late complementary feeding. In the present study there were ten rheumatic fever cases, regarding their complementary feeding 20% of patients did not get any complementary feeding, 30% got regular complementary feeding, 30% of patients got early and 20% of patients got late complementary feeding. Aplastic anaemia cases were 11, of them 9.09% did not get complementary feeding, 27.27% got regular, 36.36% of patients got early and 27.27% of patients got late complementary feeding. Among 15 cases of bronchial asthma, 6.67% did not get, 20% got regular complementary feeding, 60% of patients got early and 13.33% of patients got late complementary feeding (Fig. 2D).

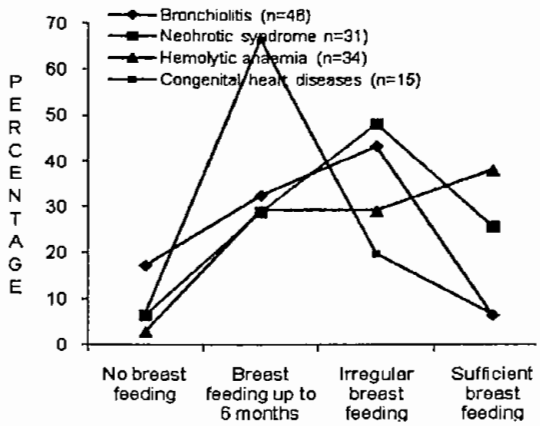
Out of 15 cases of febrile convulsion patients, 6.67% did not get complementary feeding, 20% got regular complementary feeding, 53.33% of patients got early and 20% of patients got late complementary feeding. Among 6 cases of GBS, 33.33% got regular complementary feeding, 50% of patients got early and 16.67% of patients got late complementary feeding. Pleural effusion patients were 7, of which 14.28% did not get complementary feeding, 42.86% got regular and another 42.86% got early complementary feeding (Fig. 2E).

Table 2. Complementary feeding pattern and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

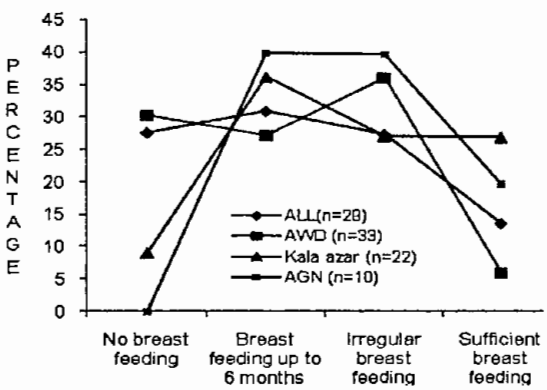
Diseases	No complementary feeding	Regular complementary feeding	Early complementary feeding	Late complementary feeding
Pneumonia (n=209)	20 (9.56)	15 (7.18)	100 (47.85)	74 (35.41)
Enteric fever (n=40)	12 (30.00)	12 (30.00)	10 (25.00)	6 (15)
Meningitis (n=47)	5 (10.64)	10 (21.28)	18 (38.29)	14 (29.79)
Encephalitis (n=20)	2 (10.00)	8 (40.00)	10 (50.00)	-
Bronchiolitis (n=46)	8 (17.39)	15 (32.61)	20 (43.48)	3 (6.52)
Nephrotic syndrome n=31)	2 (6.45)	9 (29.03)	12 (38.71)	8 (25.80)
Hemolytic anaemia (n=34)	1 (2.94)	10 (29.41)	10 (29.41)	13 (38.24)
Congenital heart diseases (n=15)	1 (6.66)	10 (66.67)	3 (20.00)	1 (6.66)
ALL(n=29)	8 (27.59)	9 (31.03)	8 (27.59)	4 (13.79)
AWD (n=33)	10 (30.30)	9 (27.27)	12 (36.36)	2 (6.06)
Kala azar (n=22)	2 (9.09)	8 (36.36)	6 (27.27)	6 (27.27)
AGN (n=10)	-	4 (40.00)	4 (40.00)	2 (20.00)
Viral hepatitis (n=12)	2 (16.66)	3 (25.00)	2 (16.66)	5 (41.67)
Rheumatic fever (n=10)	2 (20.00)	3 (30.00)	3 (30.00)	2 (20.00)
Aplastic anaemia (n=11)	1 (9.09)	3 (27.27)	4 (36.36)	3 (27.27)
Bronchial asthma (n=15)	1 (6.67)	3 (20)	9 (60)	2 (13.33)
Febrile convulsion (n=15)	1 (6.67)	3 (20)	8 (53.33)	3 (20)
GBS (n=06)	-	2 (33.33)	3 (50)	1 (16.67)
Pleural effusion (n=07)	1 (14.28)	3 (42.86)	3 (42.86)	-



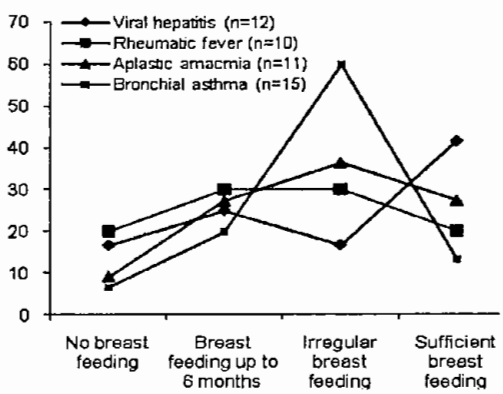
A. Complementary feeding and pneumonia, enteric fever, meningitis and encephalitis.



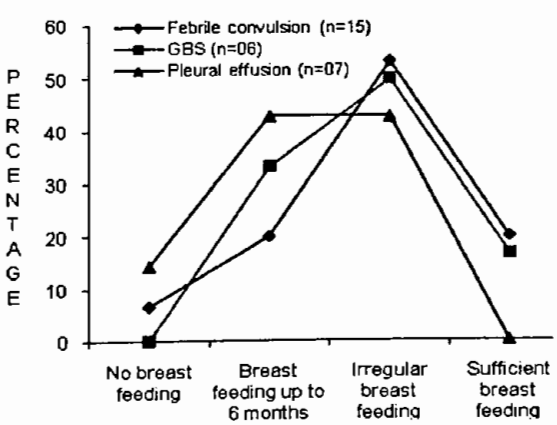
B. Complementary feeding and bronchiolitis, nephrotic syndrome, hemolytic anaemia, Congenital heart disease.



C. Complementary feeding and ALL, AWD, kala azar, AGN.



D. Complementary feeding and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma



E. Complementary feeding and febrile convulsion, GBS, pleural effusion.

Fig. 2. Relation between complementary feeding and different disease incidence.

3.3 Alternate feeding pattern and different disease incidence

The incidence of different diseases with relation of alternate feeding pattern has been presented in Table 3. Of 209 pneumonia cases 7.18% patients did not get any alternative feedings, 41.15% got infant formula as alternative feedings and 51.67% of patients got cow's milk as alternative feedings. Perinatal asphyxia cases were 78, of which 15.38% patients were given infant formula, 12.82% were given cow's milk and 71.79% cases did not get alternative feed. Regarding alternative feedings of 45 preterm and LBW patients, 15.55% patients were getting infant formula, 6.66% patients fed on cow's milk as alternative food and no alternative given in 77.78% cases. Neonatal sepsis patients were 38, among them 65.79% did not get any alternative feeding, infant formula was given in 23.68% patients and 10.52% cases were fed on cow's milk (Fig 3A).

Among 27 neonatal jaundice patients, 7.41% did not get any alternative feed, 44.44% patients got infant formula and 48.15% patients got cow's milk as alternative feedings. Regarding alternative feedings of 40 cases of enteric fever, 30% patients did not get any alternative feedings. 40% got infant formula and 30% patients got cow's milk as alternative feedings. Among 47 meningitis patients, 21.28% of patients did not get any alternative feedings, infant formula was given in 42.55% patients and cow's milk was fed to 36.17% of patients. Encephalitic patients were 20, of which 40% patients did not get any alternative feedings, 50% got infant formula and only 10% got cow's milk as alternative feedings (Fig. 3B).

Among alternative feedings of 46 bronchiolitis patients, 32.61% of children did not get any complementary feedings, 39.13% got infant formula and 28.26% got cow's milk as alternative feedings. There were 31 nephrotic syndrome patients, among them 29.03% of cases did not receive any alternative feedings. 32.25% of cases got infant formula and 38.71% got cow's milk. Hemolytic anaemia patients were 34, among which 29.41% of cases did not get alternate feeding, 41.18% of cases got infant formula and 29.41% got cow's milk. Among 15 congenital heart disease patients, 66.66% did not get any alternative feedings, 20% cases got infant formula and 13.33% got cow's milk (Fig. 3C).

Among 29 of ALL cases, 31.03% did not get any alternative feedings, 41.38% got infant formula and 27.59% got cow's milk as alternate feeding. AWD patients were 33, of them 27.27% did not get alternate feedings, 42.42% of cases got infant formula and 30.30% got cow's milk as alternate feeding. Out of 22 kala azar patients, 36.36% did not get any alternative feeding, 45.45% of cases got infant formula and 18.18% got cow's milk as alternate feeding. Among ten AGN patients, 40% did not get alternate feeding, 30% got infant formula and 30% got cow's milk as alternate feeding (Fig. 3D).

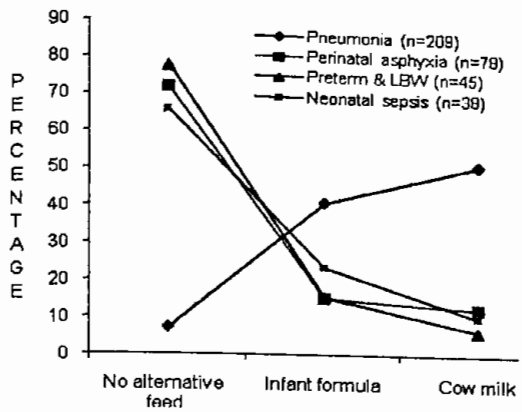
Regarding alternative feedings among the 12 viral hepatitis patients, 25% of patients did not get alternate feeding, 58.33% got infant formula and 16.66% got cow's milk as alternate feeding. Rheumatic fever cases were ten, of which 30% did not get any alternate feeding, 40% got infant formula and

30% got cow's milk as alternate feeding. Among 11 aplastic anaemia patients, 27.27% of patients did not get any alternative feed, 45.45% of cases got infant formula and 27.27% of patients got cow's milk as alternate feeding. Regarding alternate feeding of 15 bronchial asthma patients, 20% did not get any alternate feeding, 13.33% got infant formula and 66.66% got cow's milk (Fig. 3E).

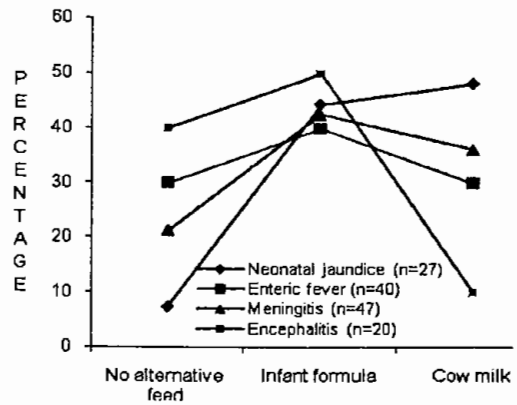
Among 15 febrile convulsion patients 20% did not get alternate feeding, another 20% got infant formula and 60% got cow's milk. GBS patients were 6 and 33.33% of them did not get alternate feeding and 66.66% infant formula. Out of seven pleural effusion patients, 42.86% did not get alternate feeding, 28.57% got infant formula and 28.57% got cow's milk as alternative feeding (Fig. 3F).

Table 3. Alternative feeding pattern and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

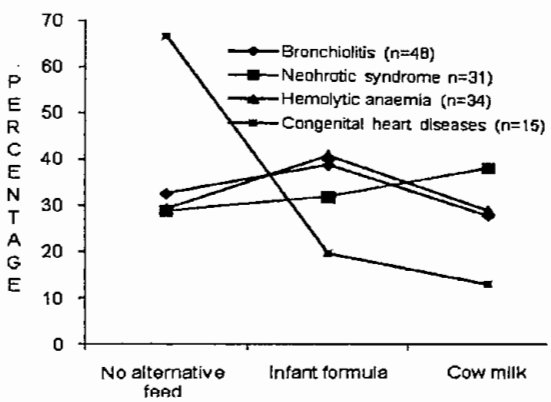
Diseases	No alternative feed	Infant formula	Cow milk
Pneumonia (n=209)	15 (7.18)	86 (41.15)	108 (51.67)
Perinatal asphyxia (n=78)	56 (71.79)	12 (15.38)	10 (12.82)
Preterm & LBW (n=45)	35 (77.78)	7 (15.55)	3 (6.66)
Neonatal sepsis (n=38)	25 (65.79)	9 (23.68)	4 (10.52)
Neonatal jaundice (n=27)	2 (7.41)	12 (44.44)	13 (48.15)
Enteric fever (n=40)	12 (30.00)	16 (40.00)	12 (30.00)
Meningitis (n=47)	10 (21.28)	20 (42.55)	17 (36.17)
Encephalitis (n=20)	8 (40.00)	10 (50.00)	2 (10.00)
Bronchiolitis (n=46)	15 (32.61)	18 (39.13)	13 (28.26)
Nephrotic syndrome n=31)	9 (29.03)	10 (32.25)	12 (38.71)
Hemolytic anaemia (n=34)	10 (29.41)	14 (41.18)	10 (29.41)
Congenital heart diseases (n=15)	10 (66.66)	3 (20.00)	2 (13.33)
ALL(n=29)	9 (31.03)	12 (41.38)	8 (27.59)
AWD (n=33)	9 (27.27)	14 (42.42)	10 (30.30)
Kala azar (n=22)	8 (36.36)	10 (45.45)	4 (18.18)
AGN (n=10)	4 (40.00)	3 (30.00)	3 (30.00)
Viral hepatitis (n=12)	3 (25.00)	7 (58.33)	2 (16.66)
Rheumatic fever (n=10)	3 (30.00)	4 (40.00)	3 (30.00)
Aplastic anaemia (n=11)	3 (27.27)	5 (45.45)	3 (27.27)
Bronchial asthma (n=15)	3 (20.00)	2 (13.33)	10 (66.66)
Febrile convulsion (n=15)	3 (20.00)	3 (20.00)	9 (60.00)
GBS (n=06)	2 (33.33)	4 (66.66)	-
Pleural effusion (n=07)	3 (42.86)	2 (28.57)	2 (28.57)



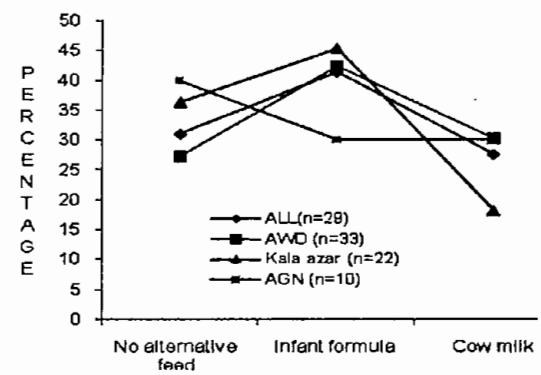
A. Alternate feeding and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis



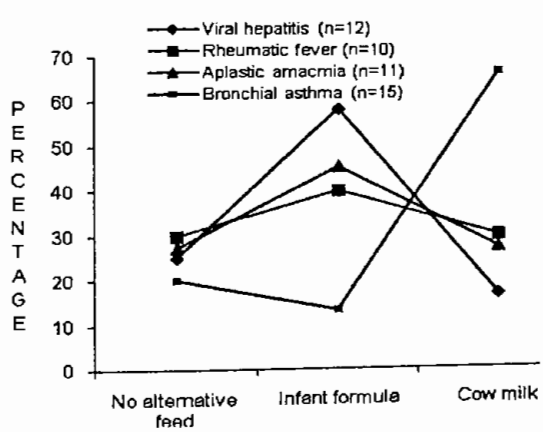
B. Alternate feeding and neonatal jaundice, enteric fever, meningitis, encephalitis.



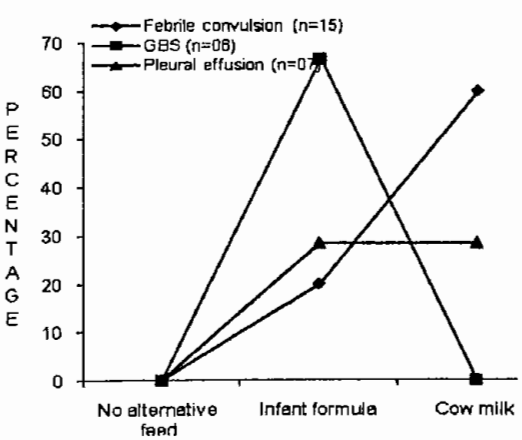
C. Alternate feeding and bronchiltis, nephrotic syndrome, hemolytic anaemia, Congenital heart disease.



D. Alternate feeding and ALL, AWD, Kala azar, AGN.



E. Alternate feeding and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



F. Alternate feeding and febrile convulsion, GBS and pleural efffusion.

Fig. 3. Relation between alternate feeding pattern and different disease incidence.

3.4 Nature of food and different disease incidence

The incidence of different diseases with relation of nature of food has been presented in Table 4. Regarding nature of food out of 122 pneumonia patients, 12.29% cases got family diet, 56.56% got diluted milk and 31.15% fed on carbohydrate only. Among 16 perinatal asphyxia eight neonatal sepsis and 15 neonatal jaundice cases all patients were offered diluted milk (Fig 4A).

Among 40 enteric fever cases, 70% got family diet and 30% got diluted milk. Meningitis patients were 33, among them family diet was offered to 33.33% patients, 45.45% got diluted milk and only carbohydrate was given to 21.21% of children. Among 20 cases of encephalitis 85% got family diet and 15% fed on diluted milk. Bronchiolitis patients were 16, 37.50% rot family diet and 62.50% of children got diluted milk (Fig. 4B).

Thirty one nephrotic syndrome patients were recorded with 80.64% got family diet, 16.13% of cases got diluted milk and 3.22% of patients got only carbohydrate diet. Among 34 hemolytic anaemia patients 82.35% of cases got family diet and 17.65% of cases got diluted milk only. Out of nine congenital heart disease patients 66.66% got family diet and 33.33% got diluted milk. Regarding 29 ALL cases, 79.31% got family diet, 13.79% got diluted milk and 6.90% of cases got carbohydrate only (Fig. 4C).

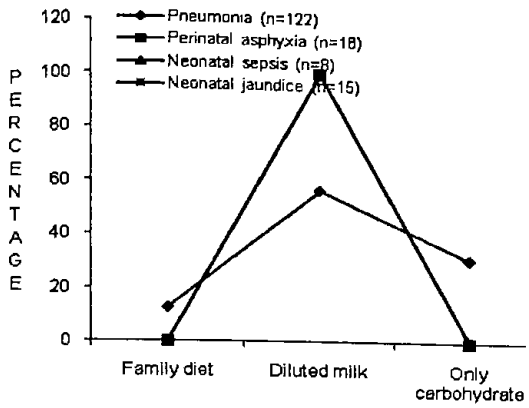
Among 15 cases recorded for AWD, 20% got family diet, 46.66% got diluted milk and 33.33% of patients got carbohydrate only. Regarding 22

kala azar patients 90.91% got family diet, 4.54% got diluted milk and 4.54% of patients got carbohydrate only. All patients received family diet among 10 AGN cases. Viral hepatitis cases were 12 among them 83.33% got family diet and 16.66% got diluted milk (Fig. 4D).

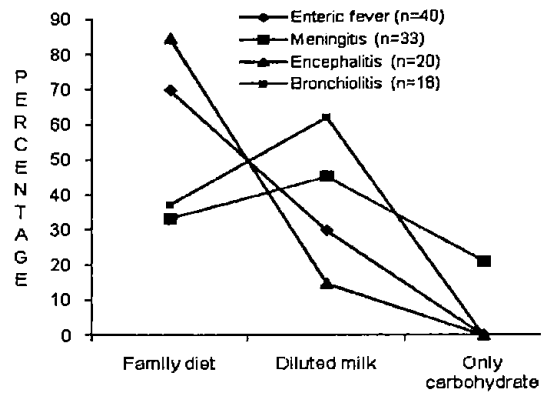
Regarding rheumatic fever 90% patients got family diet and 10% had history of getting diluted milk among total 10 patients. All patients got family diet among 11 cases of aplastic anaemia. Among 15 bronchial asthma cases 80% got family diet and 20% got diluted milk febrile convulsion patients were 15 among which 66.66% got family diet, 26.67% got diluted milk and 6.66% cases got carbohydrate only (Fig. 4E). All patients got family diet among 6 GBS and 7 of pleural effusion cases (Fig. 4F).

Table 4. Nature of food and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

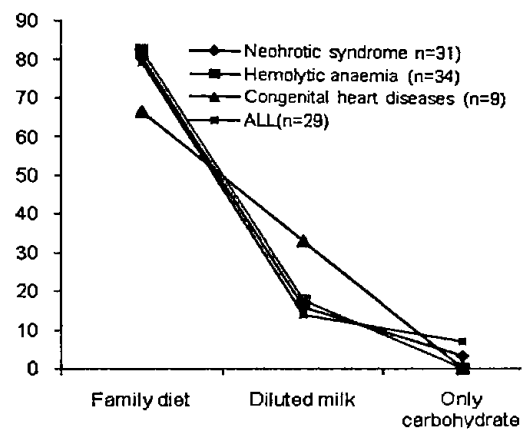
Diseases	Family diet	Diluted milk	Only carbohydrate
Pneumonia (n=122)	15 (12.29)	69 (56.56)	38 (31.15)
Perinatal asphyxia (n=16)	-	16 (100.00)	-
Neonatal sepsis (n=8)	-	8 (100.00)	-
Neonatal jaundice (n=15)	-	15 (100.00)	-
Enteric fever (n=40)	28 (70.00)	12 (30.00)	-
Meningitis (n=33)	11 (33.33)	15 (45.45)	7 (21.21)
Encephalitis (n=20)	17 (85.00)	3 (15.00)	-
Bronchiolitis (n=16)	6 (37.50)	10 (62.50)	-
Nephrotic syndrome n=31)	25 (80.64)	5 (16.13)	1 (3.22)
Hemolytic anaemia (n=34)	28 (82.35)	6 (17.65)	-
Congenital heart diseases (n=9)	6 (66.66)	3 (33.33)	-
ALL(n=29)	23 (79.31)	4 (13.79)	2 (6.90)
AWD (n=15)	3 (20.00)	7 (46.66)	5 (33.33)
Kala azar (n=22)	20 (90.91)	1 (4.54)	1 (4.54)
AGN (n=10)	10 (100.00)	-	-
Viral hepatitis (n=12)	10 (83.33)	2 (16.66)	-
Rheumatic fever (n=10)	9 (90.00)	1 (10.00)	-
Aplastic anaemia (n=11)	11 (100.00)	-	-
Bronchial asthma (n=15)	12 (80.00)	3 (20.00)	-
Febrile convulsion (n=15)	10 (66.66)	4 (26.67)	1 (6.66)
GBS (n=06)	6 (100.00)	-	-
Pleural effusion (n=07)	7 (100.00)	-	-



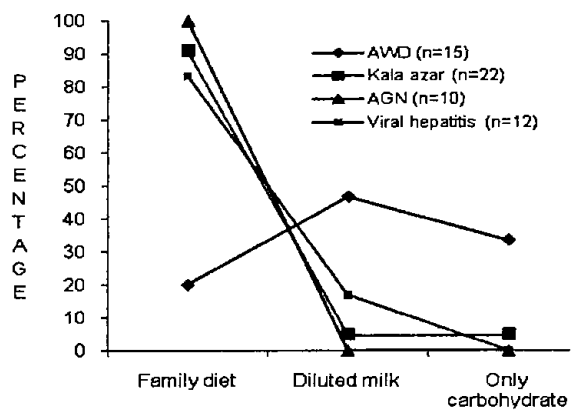
A. Nature of food and pneumonia, perinatal asphyxia, neonatal sepsis, neonatal jaundice



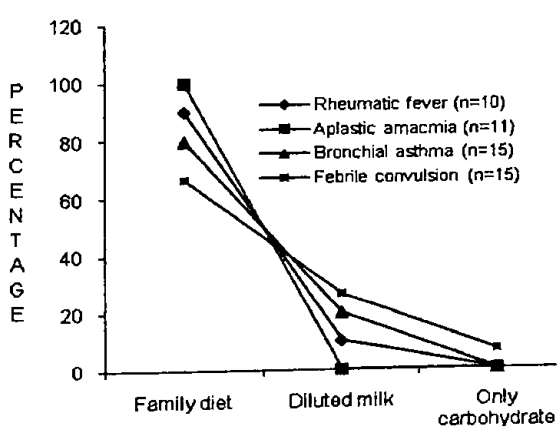
B. Nature of food and enteric fever, meningitis, encephalitis and bronchiolitis.



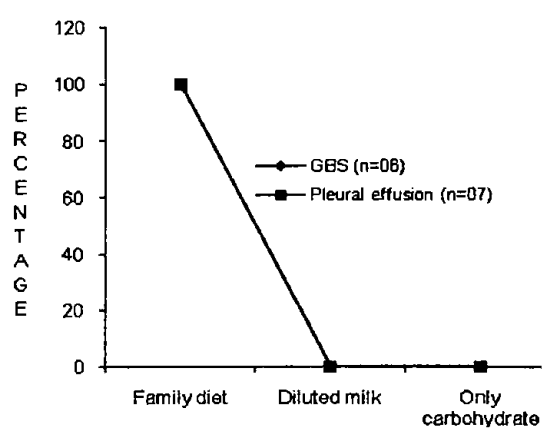
C. Nature of food and nephrotic syndrome, hemolytic anaemia, congenital heart disease, ALL



D. Nature of food and AWD, Kala azar, AGN, viral hepatitis.



E. Nature of food and rheumatic fever, aplastic anaemia, bronchial asthma, Febrile convulsion.



F. Nature of food and GBS and Pleural effusion.

Fig. 4. Relation between Nature of food and different disease incidence.

3.5 Parental educational status in different disease incidence

Incidences of different diseases in relation to parental educational status have been presented in the Table 5. There were 209 cases of pneumonia in this study, of which 15.31% were primary, 4.30% secondary, 8.61% higher and 71.77% were illiterate. In the same category of 78 perinatal asphyxia cases, the percentage of primary, secondary, higher and illiterate parents were 35.9, 12.82, 32.05 and 19.23 respectively. Preterm and LBW cases were 45 and educational status of parent was 22.22% primary, 11.11% secondary and 66.66% were illiterate. Among 38 neonatal sepsis cases, their parent were 31.58% primary, 18.42% secondary and 50% were illiterate (Fig 5A).

Among 27 neonatal jaundice cases, their parents were 37.03, 7.40 and 55.56% were primary, secondary and illiterate respectively. There were 40 enteric fever cases of them, educational status of the parents were 30% primary, 2.50% secondary, 5% higher and 62.50% illiterate. Among 47 meningitis cases, 36.17% parents were educated up to primary, 12.77% were secondary, 12.77% had higher qualification and 38.29% were illiterate. Encephalitis cases were 20, among them parental educational status was 25, and 50% were primary, secondary and illiterate respectively (Fig. 5B).

Bronchiolites cases were 46, their parental educational status were 32.61% primary, 13.04% secondary and 54.35% illiterate. There were 31 nephrotic syndrome cases in this study and their parental educational status were 48.39% primary, 38.70% secondary, 3.22% higher and 9.68% illiterate. Among 34 hemolytic anaemia children their parental educational status was

47.07% primary, 14.70% higher and 38.23% illiterate. Among 15 congenital heart disease patients their parents were 20, 40 and 40% primary, secondary and illiterate respectively (Fig. 5C).

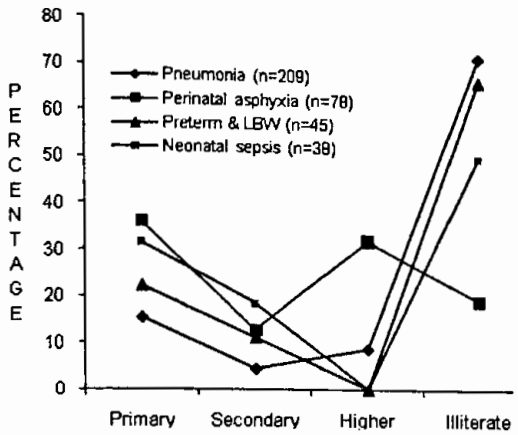
ALL cases were 29, out of them 34.48% parents were primarily educated, 27.58% were secondarily educated, 13.79% had higher qualification and remaining 24.14% were illiterate. AWD cases were 33, parental educational status were 45.45% primary, 6.06% secondary, 3.03% higher and 45.45% illiterate. Among 22 kala azar cases parental educational status were 31.82% primary, 13.63% secondary and 54.55% illiterate. Among ten AGN cases, educational status of the parents was 50% primary and 50% illiterate (Fig. 5D).

Viral hepatitis cases were 12, parental educational status were 50% primary, 16.67% secondary, 8.33% higher and 25% illiterate. Rheumatic fever cases were ten and their parental educational status were 50% primary, 10% secondary and remaining 40% illiterate. Aplastic anaemia cases were 11, their parental educational status were 27.27% primary, 27.27% secondary and 45.45% illiterate. There were 15 bronchial asthma cases in this study and their parental educational status were 26.67% primary, 33.33% secondary and 40% illiterate (Fig. 5E).

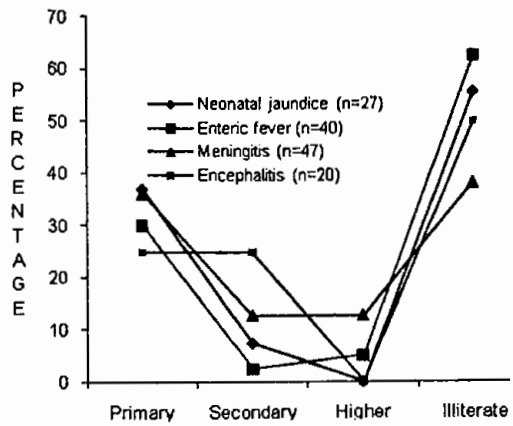
Febrile convulsion cases were 15 and their parents were 40% primary, 40% secondary and 20% illiterate. Among six GBS cases, parental educational status was 66.66% primary and 33.33% illiterate. Pleural effusion cases were seven in this study, their parental educational status was 42.86 % primary and 57.14 % illiterate (Fig. 5F).

Table 5. Parental education status and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

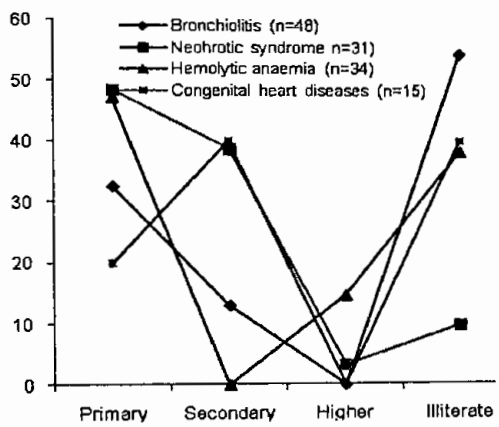
Diseases	Primary	Secondary	Higher	Illiterate
Pneumonia (n=209)	32 (15.31)	9 (4.30)	18 (8.61)	150 (71.77)
Perinatal asphyxia (n=78)	28 (35.90)	10 (12.82)	25 (32.05)	15 (19.23)
Preterm & LBW (n=45)	10 (22.22)	5 (11.11)	-	30 (66.66)
Neonatal sepsis (n=38)	12 (31.58)	7 (18.42)	-	19 (50.00)
Neonatal jaundice (n=27)	10 (37.03)	2 (7.40)	-	15 (55.56)
Enteric fever (n=40)	12 (30.00)	1 (2.50)	2 (5.00)	25 (62.50)
Meningitis (n=47)	17 (36.17)	6 (12.77)	6 (12.77)	8 (38.29)
Encephalitis (n=20)	5 (25.00)	5 (25.00)	-	10 (50.00)
Bronchiolitis (n=46)	15 (32.61)	6 (13.04)	-	25 (54.35)
Nephrotic syndrome n=31)	15 (48.39)	12 (38.70)	1 (3.22)	3 (09.68)
Hemolytic anaemia (n=34)	16 (47.07)	-	5 (14.70)	13 (38.23)
Congenital heart diseases (n=15)	3 (20.00)	6 (40.00)	-	6 (40.00)
ALL(n=29)	10 (34.48)	8 (27.58)	4 (13.79)	7 (24.14)
AWD (n=33)	15 (45.45)	2 (6.06)	1 (3.03)	15 (45.45)
Kala azar (n=22)	7 (31.82)	3 (13.63)	-	12 (54.55)
AGN (n=10)	5 (50.00)	-	-	5 (50.00)
Viral hepatitis (n=12)	6 (50.00)	2 (16.67)	1 (8.33)	3 (25.00)
Rheumatic fever (n=10)	5 (50.00)	1 (10.00)	-	4 (40.00)
Aplastic anaemia (n=11)	3 (27.27)	3 (27.27)	-	5 (45.45)
Bronchial asthma (n=15)	4 (26.67)	5 (33.33)	-	6 (40.00)
Febrile convulsion (n=15)	6 (40.00)	6 (40.00)	-	3 (20.00)
GBS (n=06)	4 (66.66)	-	-	2 (33.33)
Pleural effusion (n=07)	3 (42.86)	-	-	4 (57.14)



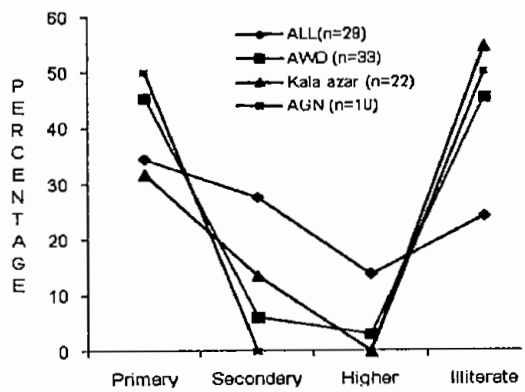
A. Parental education and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis.



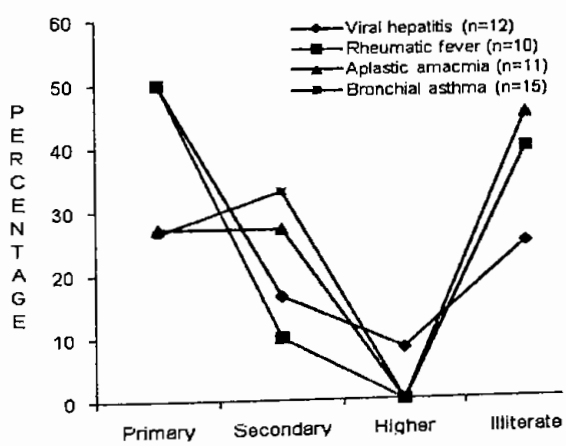
B. Parental education and neonatal jaundice, enteric fever, meningitis, encephalitis.



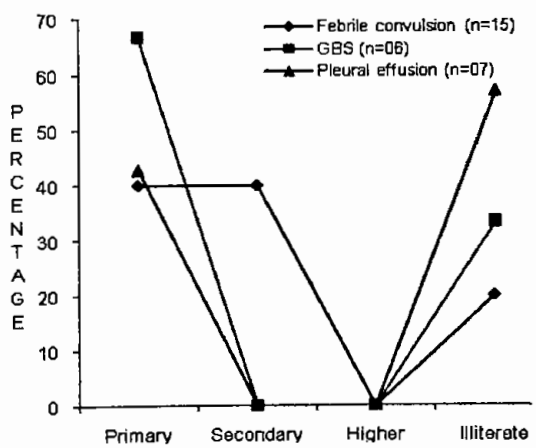
C. Parental education and bronchiolitis, nephrotic syndrme, hemolytic anaemia, congenital heart disease.



D. Parental education and ALL, AWD, kala azar, AGN.



E. Parental education and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



F. Parental education and febrile convulsion, GBS, pleural effusion.

Fig. 5. Relation between parental education and incidence of diseases among children

3.6 Nature of housing and incidence of different diseases

The incidence of different diseases in relation to the nature of parental housing has been presented in the Table 6. There were 209 cases of pneumonia in this study, their housing status were 36.36% brick building, 54.06% bamboo thatched and remaining 9.57% were other types. Perinatal asphyxia cases were 78 of them 38.46, 32.05 and 29.48% were brick building, bamboo thatched and others types respectively. Preterm and LBW cases were 45, among them housing status were 44.44% brick building, 48.88% bamboo thatched and 6.68% others types. Neonatal sepsis cases were 38, of them 34.21% brick building and 65.79% were bamboo thatched (Fig 6A).

There were 27 neonatal jaundice cases and their housing status were 44.44 % brick building, 22.22% bamboo thatched and 33.33% others types. Among 40 enteric fever cases housing status were 45.00% brick building, 12.50% bamboo thatched and 42.50% other types. The meningitis cases were 47 among them housing status were 42.55% brick building, 25.53% bamboo thatched and 31.91% others. Encephalitis patients were 20 and housing status were 75% brick building and remaining 25% were bamboo thatched (Fig. 6B).

Housing status was 43.48% brick building, 50% bamboo thatched and 6.52% others among 46 bronchiolitis cases. There were 31 nephrotic syndrome cases and their housing status was 48.39% brick building, 48.39% bamboo thatched and remaining 3.22% others. Hemolytic anaemia cases were 34, their housing status were 38.23% brick building, 38.23% bamboo thatched and 23.53% others. Housing status was 46.66% brick building, 20% bamboo

thatched and 33.33% others among 15 congenital heart disease patients (Fig. 6C).

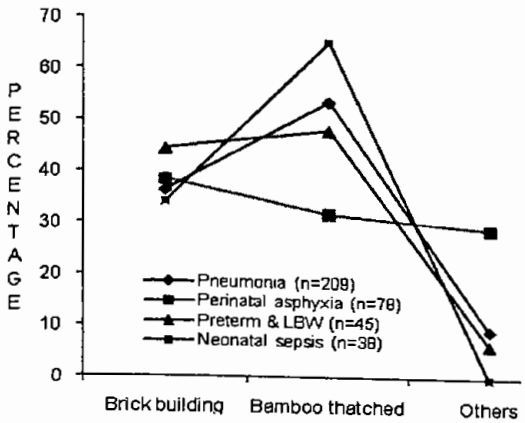
There were 29 ALL cases in this study, their housing status were 34.48% brick building, 37.93% bamboo thatched and 27.59% others. AWD cases were 33 and their housing status was 42.42% brick building, 48.48% bamboo thatched and 9.09% others. Among 22 kala Azar cases the housing status was 31.82% brick building and 68.18% bamboo thatched. There were ten AGN cases and 50% children were from brick building and 50% from bamboo thatched houses (Fig. 6D).

Among 12 viral hepatitis cases 66.67% were from brick building housing and 33.33% from bamboo thatched housing. Among 10 Rheumatic fever cases 50% from brick building and 50% from bamboo thatched housing. Aplastic amaemia cases were 11 among them 45.45% from brick building and 54.55% from bamboo thatched housing. Bronchial asthma cases were 15, their housing status were 46.67% brick building and 53.33% bamboo thatched (Fig. 6E).

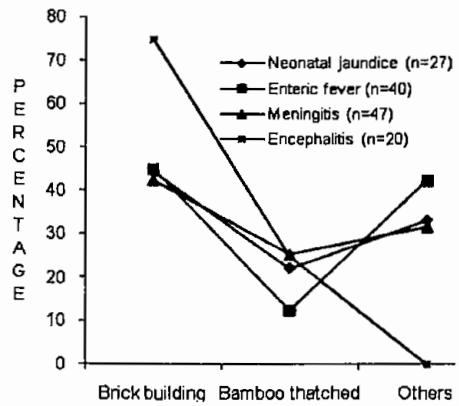
There were 15 febrile convulsion cases in this study and their housing status were 40% brick building, 40% bamboo thatched and 20% others. GBS cases were six, among them 66.67% were from brick building housing and 33.33% from bamboo thatched housing. Among seven pleural effusion cases, 71.42% housing was brick building and 28.57% was bamboo thatched (Fig. 1F).

Table 6. Housing type and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

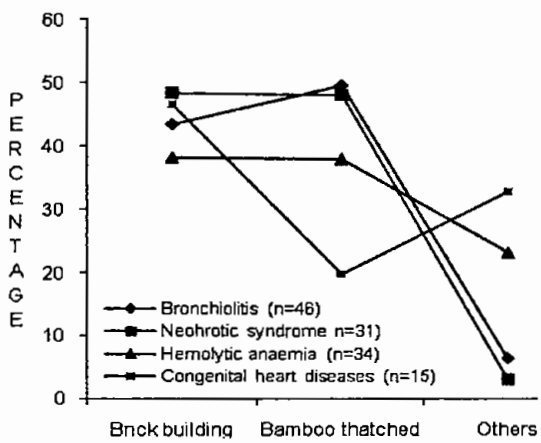
Diseases	Brick building	Bamboo thatched	Others
Pneumonia (n=209)	76 (36.36)	113 (54.06)	20 (9.57)
Perinatal asphyxia (n=78)	30 (38.46)	25 (32.05)	23 (29.48)
Preterm & LBW (n=45)	20 (44.44)	22 (48.88)	3 (6.68)
Neonatal sepsis (n=38)	13 (34.21)	25 (65.79)	-
Neonatal jaundice (n=27)	12 (44.44)	06 (22.22)	9 (33.33)
Enteric fever (n=40)	18 (45.00)	5 (12.50)	17 (42.50)
Meningitis (n=47)	20 (42.55)	12 (25.53)	15 (31.91)
Encephalitis (n=20)	15 (75.00)	5 (25.00)	-
Bronchiolitis (n=46)	20 (43.48)	23 (50.00)	3 (6.52)
Nephrotic syndrome n=31)	15 (48.39)	15 (48.39)	1 (3.22)
Hemolytic anaemia (n=34)	13 (38.23)	13 (38.23)	8 (23.53)
Congenital heart diseases (n=15)	7 (46.66)	3 (20.00)	5 (33.33)
ALL(n=29)	10 (34.48)	11 (37.93)	8 (27.59)
AWD (n=33)	14 (42.42)	16 (48.48)	3 (9.09)
Kala azar (n=22)	7 (31.82)	15 (68.18)	-
AGN (n=10)	5 (50.00)	5 (50.00)	-
Viral hepatitis (n=12)	8 (66.67)	4 (33.33)	-
Rheumatic fever (n=10)	5 (50.00)	5 (50.00)	-
Aplastic anaemia (n=11)	5 (45.45)	6 (54.55)	-
Bronchial asthma (n=15)	7 (46.67)	8 (53.33)	-
Febrile convulsion (n=15)	6 (40.00)	6 (40.00)	3 (20.00)
GBS (n=06)	4 (66.67)	2 (33.33)	-
Pleural effusion (n=07)	5 (71.42)	2 (28.57)	-



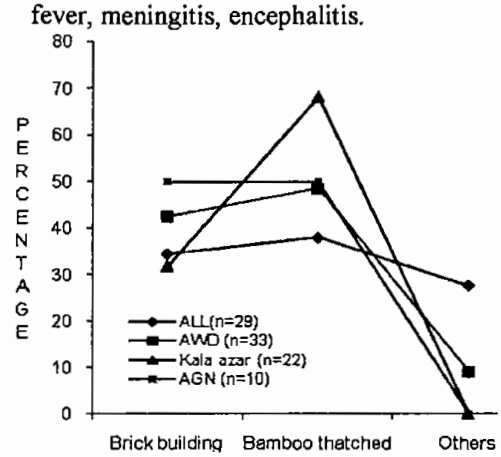
A. Housing type and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis



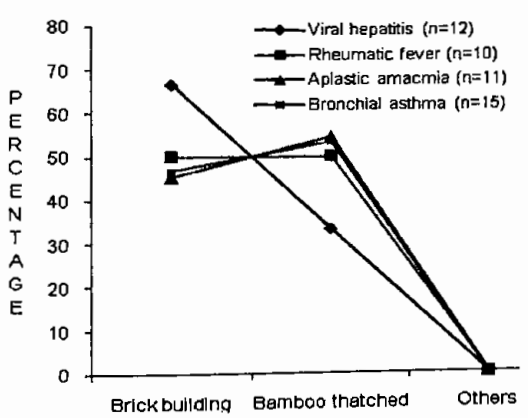
B. Housing type and neonatal jaundice, enteric fever, meningitis, encephalitis.



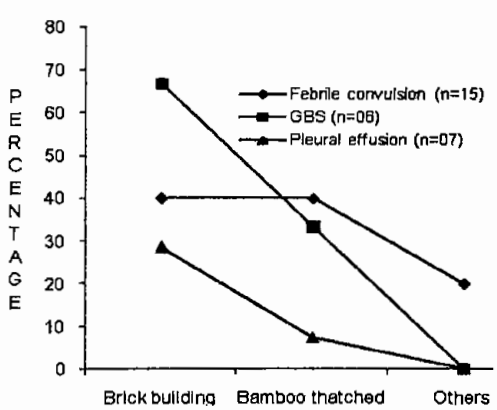
C. Housing type and bronchiolitis, nephrotic syndrome, hemolytic anaemia, congenital heart disease.



D. Housing type and ALL, AWD, kala azar, AGN.



E. Housing type and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



F. Housing type and febrile convulsion, GBS pleural effusion.

Fig. 6. Relation between housing type and different disease incidence.

3.7 Drinking water source and different disease incidence

The incidence of different diseases in relation to drinking water source has been presented in Table 7. There were 209 cases of pneumonia in this study and their drinking water sources were 55.02% tube well water, 36.84% supply water and 8.13% from other sources. Among 78 of perinatal asphyxia cases, 70.51% got tube well water, 25.64% supply water and 3.84% from other sources. Out of 45 preterm and LBW cases, 51.11 and 48.88%; of 38 cases of neonatal sepsis 55.26 and 44.73%; and of 27 neonatal jaundice 51.85 and 48.14% patients the source of drinking water was tube well and supply water respectively (Fig 7A).

Regarding drinking water among 40 enteric fever cases 20% from tube well water, 37.50% from supply water source and 42.50% from other source. Meningitis cases were 47, of them 59.57% from tube well water and 40.42% from supply water got their drinking water. There were 20 cases of encephalitis in this study, their drinking water were supplied from 75% tube well water and 25% supply water (Fig. 7B).

Among 46 Bronchiolitis cases, 43.48% got drinking water from tube well water and 56.42% got from supply water. Nephrotic syndrome cases were 31, of them 45.16% got from tube well water, 54.83% from supply water. Regarding drinking water among 34 of hemolytic anaemia cases 76.47% from tube well water, 23.52% from supply water source. Out of 15 Congenital heart disease cases 66.66% got drinking water from tube well water and 33.33% got from supply water (Fig. 7C).

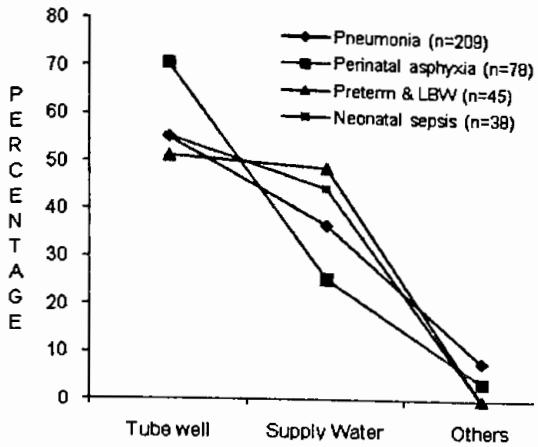
ALL cases were 29, of them 68.96% from tube well water. 31.03% from supply water source got their drinking water. Out of 33 AWD cases 15.15% got drinking water from tube well water, 30.30% got from supply water and 54.54% got from other source. Regarding drinking water among 22 of kala azar cases, 72.72% from tube well water and 27.27% from supply water source. Among 10 AGN cases 60% got drinking water from tube well water and 40% got from supply water (Fig. 7D).

There were 12 cases of viral hepatitis in this study their drinking water were supplied from 16.67% tube well water 66.66% supply water and 16.67% from other source. Rheumatic fever cases were 10, of them 50% from tube well water and 50% from supply water got their drinking water. Among 11 aplastic amaemia cases of them 54.54% got drinking water from tube well water and 45.45% got from supply water. There were fifteen cases of bronchial asthma in this study their drinking water were supplied from 66.66% tube well water 26.67% supply water and 6.66% from other source (Fig. 7E).

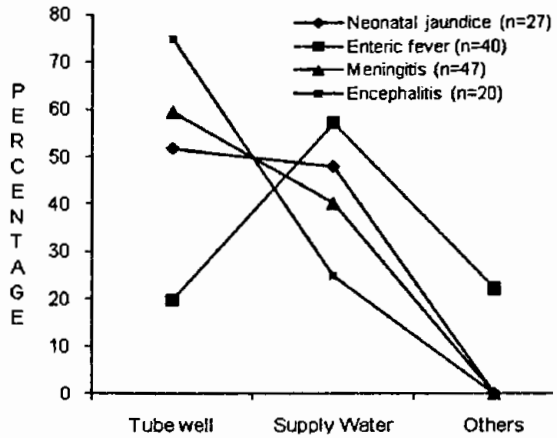
Regarding drinking water among 15 febrile convulsion cases 60.00% from tube well water, 40% from supply water source. GBS cases were six. of them 66.67% from tube well water and 33.33% from supply water got their drinking water. There were seven cases of pleural effusion in this study, their drinking water were supplied from 71.43% tube well water and 28.57% from supply water (Fig. 7F).

Table 7. Drinking water source and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

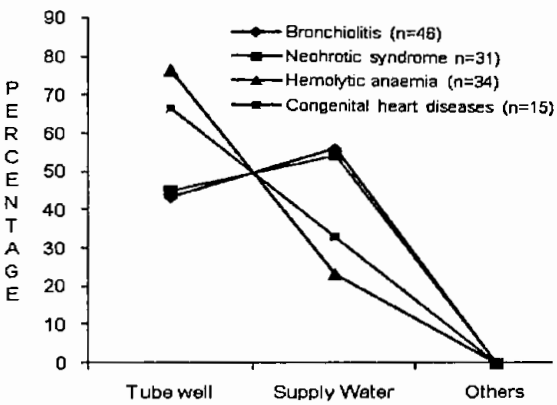
Diseases	Tube well water	Supply water	Others
Pneumonia (n=209)	115 (55.02)	77 (36.84)	17 (8.13)
Perinatal asphyxia (n=78)	55 (70.51)	20 (25.64)	3 (3.84)
Preterm & LBW (n=45)	23 (51.11)	22 (48.88)	-
Neonatal sepsis (n=38)	21 (55.26)	17 (44.73)	-
Neonatal jaundice (n=27)	14 (51.85)	13 (48.14)	-
Enteric fever (n=40)	8 (20.00)	15 (37.50)	17 (42.50)
Meningitis (n=47)	28 (59.57)	19 (40.42)	-
Encephalitis (n=20)	15 (75.00)	5 (25.00)	-
Bronchiolitis (n=46)	20 (43.48)	26 (56.52)	-
Nephrotic syndrome n=31)	14 (45.16)	17 (54.83)	-
Hemolytic anaemia (n=34)	26 (76.47)	8 (23.52)	-
Congenital heart diseases (n=15)	10 (66.66)	5 (33.33)	-
ALL(n=29)	20 (68.96)	9 (31.02)	-
AWD (n=33)	5 (15.15)	10 (30.30)	18 (54.54)
Kala azar (n=22)	16 (72.72)	6 (27.27)	-
AGN (n=10)	6 (60.00)	4 (40.00)	-
Viral hepatitis (n=12)	2 (16.67)	8 (66.66)	2 (16.67)
Rheumatic fever (n=10)	5 (50.00)	5 (50.00)	-
Aplastic anaemia (n=11)	6 (54.54)	5 (45.45)	-
Bronchial asthma (n=15)	10 (66.66)	4 (26.67)	1 (6.66)
Febrile convulsion (n=15)	9 (60.00)	6 (40.00)	-
GBS (n=06)	4 (66.67)	2 (33.33)	-
Pleural effusion (n=07)	5 (71.43)	2 (28.57)	-



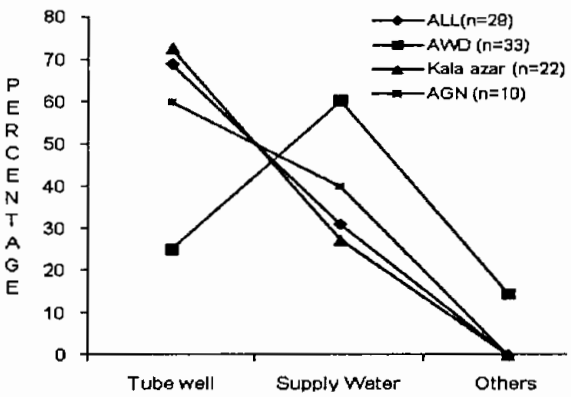
A. Drinking water and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis



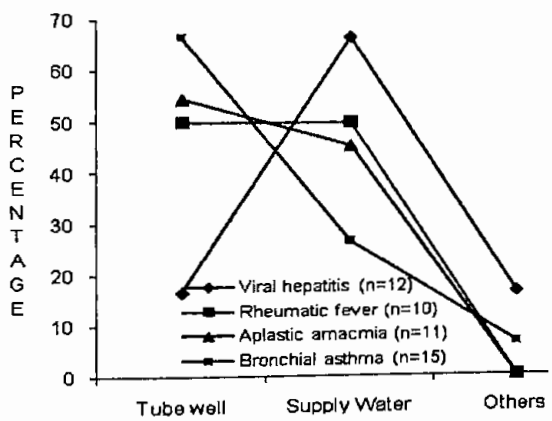
B. Drinking water and neonatal jaundice, enteric fever, meningitis, encephalitis.



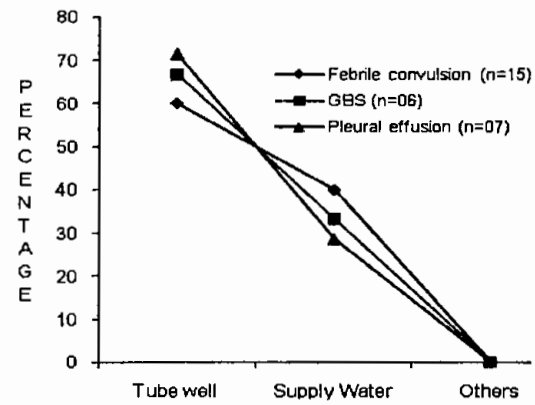
C. Drinking water and bronchilitis, nephrotic syndrome, hemolytic anaemia, congenital heart disease.



D. Drinking water and ALL, AWD, kala azar, AGN.



E. Drinking water and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



F. Drinking water and febrile convulsion, GBS, pleural effusion.

Fig. 7. Relation between drinking water source and different disease incidence.

3.8 Sanitation and different disease incidence

The incidence of different disease in relation to sanitation has been presented in Table 8. There were 209 pneumonia cases in this study, their sanitation status were 90.91% sanitary, 7.17% service type and 1.91% open air. Among 78 perinatal asphyxia cases, 70.51% were sanitary and 29.48% were service type. Preterm & LBW cases were 45, of them 84.44% sanitary 11.11% service type and 4.44 open air. There were 38 cases of neonatal sepsis in this study, their sanitation status were 89.47% sanitary and 10.52% service type (Fig 8A).

Among 27 neonatal jaundice cases, sanitation status were 88.88% sanitary, 11.11% service type. Regarding sanitation among 40 enteric fever cases 37.5% were sanitary, 50% service type and 12.50% open air. Meningitis cases were 47, of them 59.57% sanitary, 34.04% service type and 6.38% open air. Twenty cases of encephalitis in this study, their sanitation status were 75% sanitary, 25% service type (Fig. 8B).

Among 46 bronchiolitis cases 56.52% were sanitary and 43.47% were service type. Nephrotic syndrome cases were 31, of them 70.96% were sanitary and 28.13% were service type. Regarding sanitation among 34 hemolytic anaemia cases 76.47% were sanitary and 23.52% service type. Out of 15 congenital heart diseases cases 73.33% sanitary and 26.66% service type (Fig. 8C).

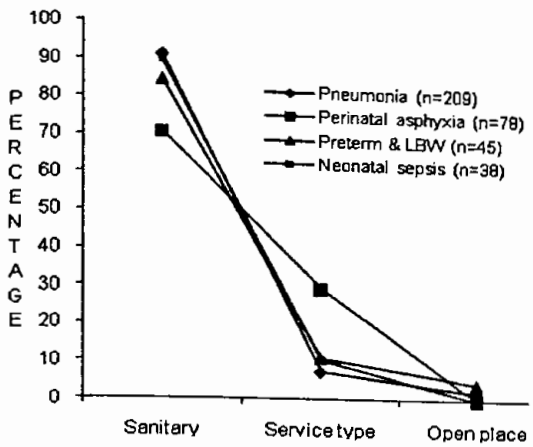
There were 29 cases of ALL in this study, their sanitation status is 68.96% sanitary, 24.14% service type and 6.89% open air. Out of 33 AWD cases, sanitation status were 12.12% sanitary, 54.54% service type and 33.33% open air. Regarding sanitation status among 22 kala Azar cases 72.72% sanitary, 18.18% service type and 9.09% open air. Among 10 AGN cases 50% sanitary and 50% service type (Fig. 8D).

Among 12 cases of viral hepatitis in this study 66.66% sanitary, 16.67% service type and 16.67% in open air. Out of 10 rheumatic fever, 60% sanitary and 40% were service type. Among 11 aplastic amaemia cases sanitation status were 63.63% sanitary, 36.36% service type. There were 15 cases of bronchial asthma and the sanitation status was 66.66% sanitary, 26.67% service type and 6.66% open air (Fig. 8E).

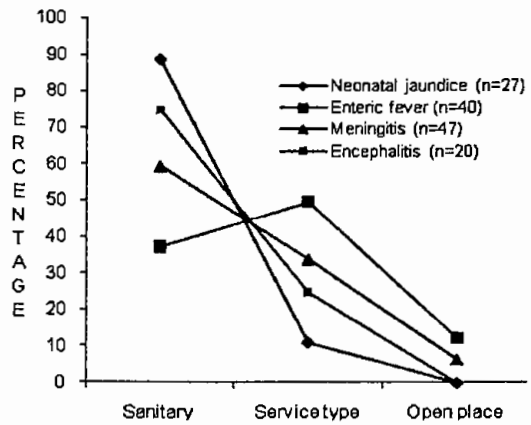
Regarding sanitation status among 15 febrile convulsions 66.66% was sanitary and 33.33% was service type. GBS cases were six, their sanitation status were 83.33% sanitary and 16.67% service type. Sanitation status was 71.43% sanitary and 28.57% service type among seven pleural effusion cases in this study (Fig. 8F).

Table 8. Sanitary system and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

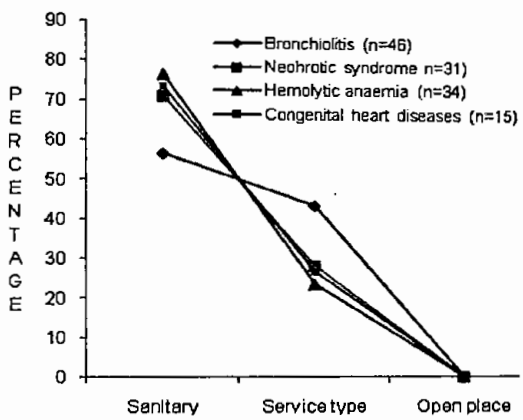
Diseases	Sanitary	Service type	Open place
Pneumonia (n=209)	190 (90.91))	15 (7.17)	4 (1.91)
Perinatal asphyxia (n=78)	55 (70.51)	23 (29.48)	-
Preterm & LBW (n=45)	38 (84.44)	5 (11.11)	2 (4.44)
Neonatal sepsis (n=38)	34 (89.47)	4 (10.51)	-
Neonatal jaundice (n=27)	24 (88.88)	3 (11.11)	-
Enteric fever (n=40)	15 (37.50)	20 (50.00)	5 (12.50)
Meningitis (n=47)	28 (59.57)	16 (34.04)	3 (6.38)
Encephalitis (n=20)	15 (75.00)	5 (25.00)	-
Bronchiolitis (n=46)	26 (56.52)	20 (43.47)	-
Nephrotic syndrome n=31)	22 (70.96)	9 (28.13)	-
Hemolytic anaemia (n=34)	26 (76.47)	8 (23.52)	-
Congenital heart diseases (n=15)	11 (73.33)	4 (26.66)	-
ALL(n=29)	20 (68.96)	7 (24.14)	2 (6.89)
AWD (n=33)	4 (12.12)	18 (54.54)	11 (33..33)
Kala azar (n=22)	16 (72.72)	4 (18.18)	2 (9.09)
AGN (n=10)	5 (50.00)	5 (50.00)	-
Viral hepatitis (n=12)	8 (66.66)	2 (16.67)	2 (16.67)
Rheumatic fever (n=10)	6 (60.00)	4 (40.00)	-
Aplastic anaemia (n=11)	7 (63.63)	4 (36.36)	-
Bronchial asthma (n=15)	10 (66.66)	4 (26.67)	1 (6.66)
Febrile convulsion (n=15)	10 (66.66)	5 (33.33)	-
GBS (n=06)	5 (83.33)	1 (16.67)	-
Pleural effusion (n=07)	5 (71.43)	2 (28.57)	-



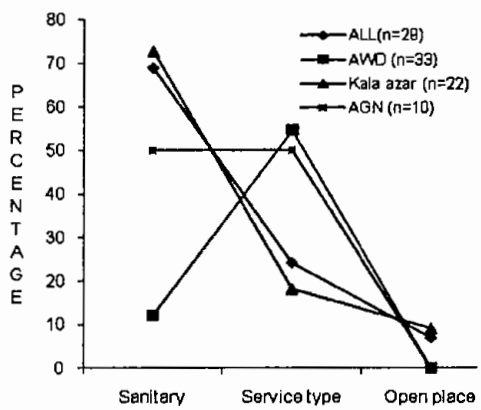
A. Sanitation system and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis



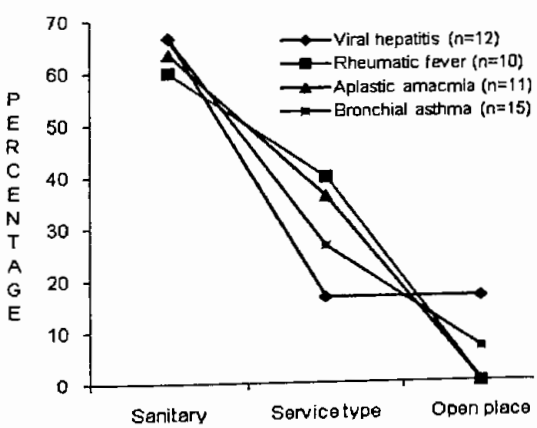
B. Sanitation system and neonatal jaundice, enteric fever, meningitis, encephalitis.



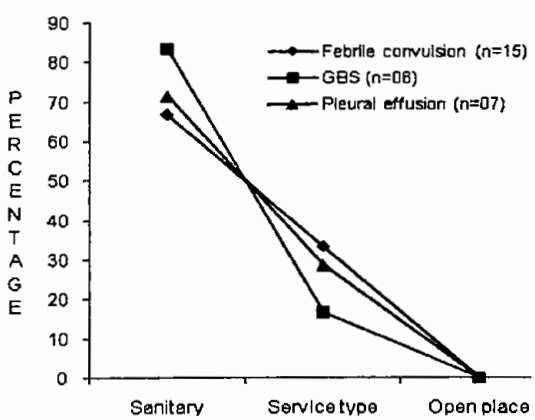
C. Sanitation and broncholitis, nephritic syndrome, haemolytic anaemia, congenital heart disease.



D. Sanitation system and ALL, AWD, kala azar, AGN.



E. Sanitary system and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma



F. Sanitary system and febrile convulsion, GBS, Pleural effusion.

Fig. 8. Relation between sanitary system and different disease incidence.

3.9 Family size and different disease incidence

Incidence of different diseases in relation with family size has been presented in Table 9. There were 209 cases of pneumonia in this study, of them 87.08% from large family and 12.91% from small family. Out of 78 cases of perinatal asphyxia 74.35% and 25.64% from large and small family respectively. Preterm and LBW cases were 45, of them 71.11% and 28.89% from large and small family respectively. Among 38 neonatal sepsis cases in this study 71.05% belonged to large family and 28.95% belonged to small family (Fig 9A).

Neonatal jaundice cases were 27, among them 44.44% belonged to large and 55.56% to small family. Out of 40 enteric fever cases 67.50% from large family and 32.50% from small family. Meningitis cases were 47, among them 74.47% belonged to large family and 25.53% belonged to small family. Among 20 encephalitis cases 60% and 40% from large and small family respectively (Fig. 9B).

Bronchiolitis cases were 46, of them 76.09% from large and 23.91% from small family. Nephrotic syndrome cases were 31, of them 61.29% belonged to large and 38.71% belonged to small family. Out of 34 hemolytic anaemia cases 73.53% were from large and 26.47% from small family. Among 15 congenital heart disease patients 66.67% were from large and 33.33% from small family (Fig. 9C).

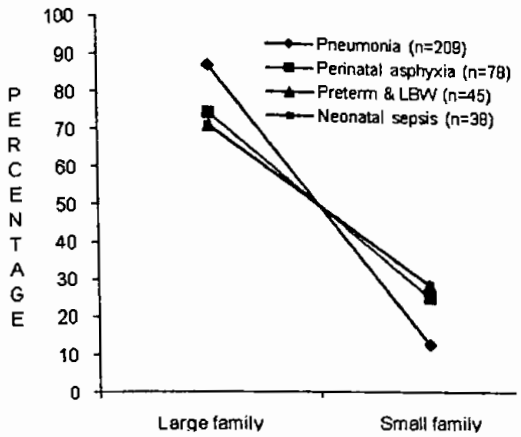
Out of 29 ALL cases, 48.28% belonged to large and 51.72% from small family. Among 33 AWD cases 75.75% and 24.25% from large and small family respectively. Kala azar cases were 22 among them 54.55% and 45.45% were large and small family respectively. All the 10 AGN cases were coming from large family (Fig. 9D).

Viral hepatitis cases were 12, of them 66.67% were from large and 33.33% from small family. Out of 10 rheumatic fever cases 80% from large and 20% from small family. Aplastic anaemia cases were 11, of them 63.64% and 36.36% from large and small family respectively. There were 15 bronchial asthma cases in this study, among them 46.67% were coming from large family and 53.33% from small family (Fig. 9E).

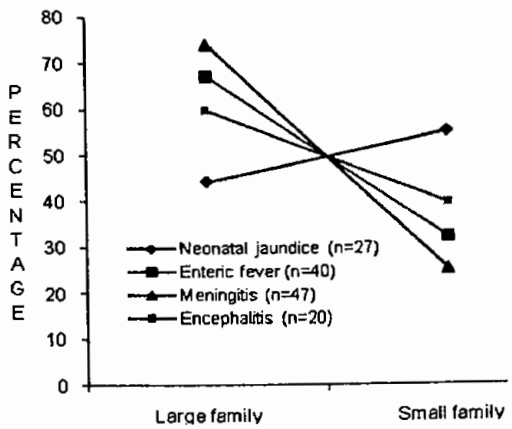
Febrile convulsion cases were 15 of them 46.67% from large family and 53.33% from small family. GBS cases were six, of them 50% were from large and another 50% from small family. Among seven pleural effusion cases 71.43% were from large family and 28.57% were from small family in this study (Fig. 9F).

Table 9. Family size and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

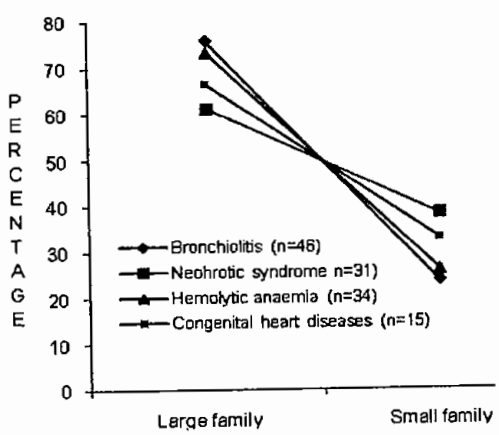
Diseases	Large family	Small family
Pneumonia (n=209)	182 (87.08)	27 (12.91)
Perinatal asphyxia (n=78)	58 (74.35)	20 (25.64)
Preterm & LBW (n=45)	32 (71.11)	13 (28.89)
Neonatal sepsis (n=38)	27 (71.05)	11 (28.95)
Neonatal jaundice (n=27)	12 (44.44)	15 (55.56)
Enteric fever (n=40)	27 (67.50)	13 (32.50)
Meningitis (n=47)	35 (74.47)	12 (25.53)
Encephalitis (n=20)	12 (60.00)	8 (40.00)
Bronchiolitis (n=46)	35 (76.09)	11 (23.91)
Nephrotic syndrome n=31)	19 (61.29)	12 (38.71)
Hemolytic anaemia (n=34)	25 (73.53)	9 (26.47)
Congenital heart diseases (n=15)	10 (66.67)	5 (33.33)
ALL(n=29)	14 (48.28)	15 (51.72)
AWD (n=33)	25 (75.75)	8 (24.25)
Kala azar (n=22)	12 (54.55)	10 (45.45)
AGN (n=10)	10 (100.00)	-
Viral hepatitis (n=12)	8 (66.67)	4 (33.33)
Rheumatic fever (n=10)	8 (80.00)	2 (20.00)
Aplastic anaemia (n=11)	7 (63.64)	4 (36.36)
Bronchial asthma (n=15)	7 (46.67)	8 (53.33)
Febrile convulsion (n=15)	7 (46.67)	8 (53.33)
GBS (n=06)	3 (50.00)	3 (50.00)
Pleural effusion (n=07)	5 (71.43)	2 (28.57)



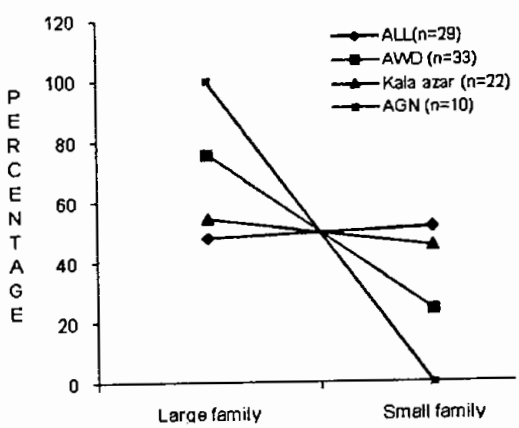
A. Family size and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis



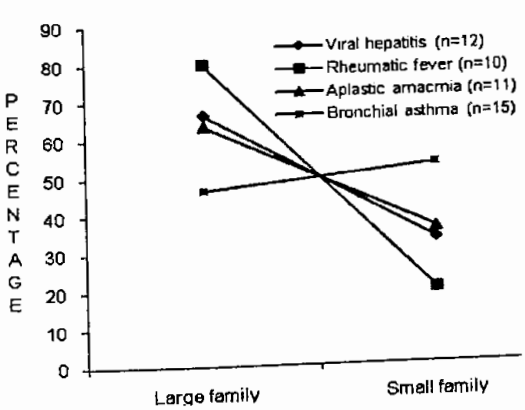
B. Family size and neonatal jaundice, enteric fever, meningitis, encephalitis.



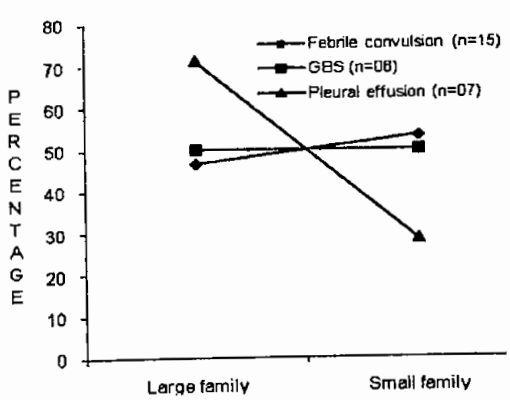
C. Family size and bronchial asthma, nephritic syndrome, haemolytic anaemia, congenital heart disease.



D. Family size and ALL, AWD, kala azar, AGN.



E. Family size and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



F. Family size and febrile convulsion, GBS, pleural effusion.

Fig. 9. Relation between Family size and different disease incidence.

3.10 Parental socio-economic status and different disease incidence

The incidence of different diseases in relation with parental socioeconomic status has been presented in Table 10. There were 209 cases of pneumonia in this study of them 9.57, 5.26, 46.89 and 38.27% were from rich, medium, poor and very poor family respectively. Among 78 perinatal asphyxia cases in this study, 6.41% were from rich family, 5.13% from medium, 38.46% from poor and remaining 50% from very poor family. Preterm and LBW cases were 45, their parental socio-economic status were 11.11, 4.44, 28.88, and 55.56% as rich, medium, poor and very poor respectively. Neonatal sepsis cases were 38 and their parental socio-economic status were 26.31, 39.47 and 34.21% as medium, poor and very poor respectively (Fig 10A).

Neonatal jaundice cases were 27, of them 11.11% were from medium status family, 44.44% from poor class family and another 44.44% from very poor class family. Among 40 enteric fever cases in this study 7.50% medium, 55% poor and 37.50% from very poor family. Meningitis cases were 47 and their parental socio-economic status were 25.53% rich, 10.64% medium, 31.91% poor and another 31.91% from very poor family. Among 20% encephalitis patients, 25% were coming from medium status family, 45% from poor family, and remaining 30% from very poor family (Fig. 10B).

Broncholitis cases were 46 of them 4.35% from rich family, 8.70% from medium, 36.95% from poor and remaining 50% from very poor family. There were 31 nephrotic syndrome cases, 12.90, 41.93 and 45.16% were from medium, poor and very poor status of family respectively. Hemolytic anaemia cases were 34, among them 5.88% from rich, 8.82% from medium, 50% from poor and 35.29% from very poor family. Among 15 congenital heart disease patients 26.66% from medium, 40% from poor and remaining 33.33% from very poor family (Fig. 10C).

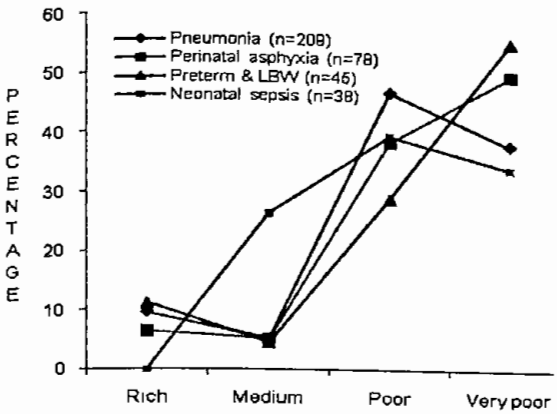
ALL cases were 29, of them 10.34% rich, 31.03% medium, 24.14%, poor and 34.48% from very poor family status. Total AWD cases were 33 out of them 9.09% were coming from rich family, 9.09% from medium, 45.45% poor and 36.36% were coming from very poor family. Among 22 kala azar patients, 13.63% medium, 40.91% poor and 45.45% from very poor family. Out of 10 AGN cases, 20%, medium, 40% poor and another 40% very poor family status (Fig. 10D).

Viral hepatitis cases were 12, of them 8.33% medium, 33.33% poor and 58.33% from very poor class family. 10%, from medium, 50% from poor class and 40% from very poor class family among 10 rheumatic fever cases in this study. Aplastic anaemia cases were 11, of them 18.18% from medium, 27.27% from poor and 54.55% from very poor class family. Among 15 bronchial asthma patients, 13.33% from medium, 40% from poor and 46.67% from very poor family status (Fig. 10E).

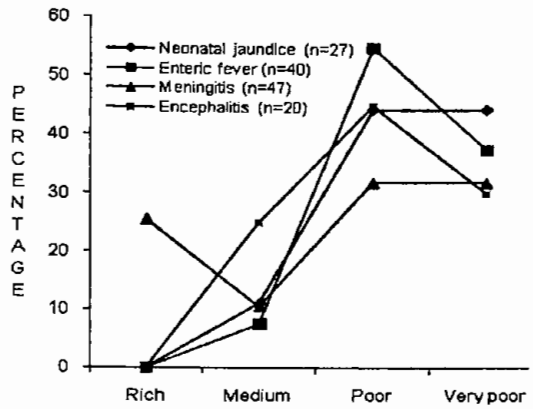
Febrile convulsion cases were 15 of them 13.33% rich, 40% medium, 20% poor and remaining 26.67% were from very poor family status. Out of six GBS cases 16.67% medium, 66.66% poor, and 16.67% cases from very poor family status. Among seven pleural effusion cases in this study 42.86% were poor and 57.14% from very poor family status (Fig. 10F).

Table 10. Parental education status and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

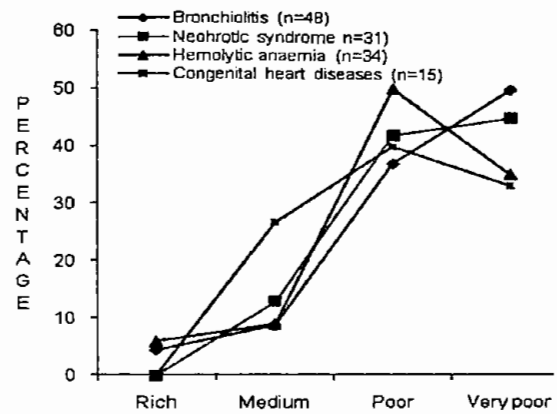
Diseases	Rich	Medium	Poor	Very poor
Pneumonia (n=209)	20 (9.57)	11 (5.26)	98 (46.89)	80 (38.27)
Perinatal asphyxia (n=78)	5 (6.41)	4 (5.13)	30 (38.46)	39 (50.00)
Preterm & LBW (n=45)	5 (11.11)	2 (4.44)	13 (28.88)	25 (55.56)
Neonatal sepsis (n=38)	-	10 (26.31)	15 (39.47)	13 (34.21)
Neonatal jaundice (n=27)	-	3 (11.11)	12 (44.44)	12(44.44)
Enteric fever (n=40)	-	3 (7.50)	22 (55.00)	15 (37.50)
Meningitis (n=47)	12 (25.53)	5 (10.64)	15 (31.91)	15(31.91)
Encephalitis (n=20)	-	5 (25.00)	9 (45.00)	6 (30.00)
Bronchiolitis (n=46)	2 (4.35)	4 (8.70)	17 (36.95)	23(50.00)
Nephrotic syndrome n=31)	-	4 (12.90)	13 (41.93)	14 (45.16)
Hemolytic anaemia (n=34)	2 (5.88)	3 (8.82)	17 (50.00)	12 (35.29)
Congenital heart diseases (n=15)	-	4 (26.66)	6 (40.00)	5 (33.33)
ALL (n=29)	3 (10.34)	9 (31.03)	7 (24.14)	10 (34.48)
AWD (n=33)	3 (9.09)	3 (9.09)	15 (45.45)	12 (36.36)
Kala azar (n=22)	-	3 (13.63)	9 (40.91)	10 (45.45)
AGN (n=10)	-	2 (20.00)	4 (40.40	4 (40.40)
Viral hepatitis (n=12)	-	1 (8.33)	4 (33.33)	7 (58.33)
Rheumatic fever (n=10)	-	1 (10.00)	5 (50.00)	4 (40.00)
Aplastic anaemia (n=11)	-	2 (18.18)	3 (27.27)	6 (54.55)
Bronchial asthma (n=15)	-	2 (13.33)	6 (40.00)	7 (46.67)
Febrile convulsion (n=15)	2 (13.33)	6 (40.00)	3(20.00)	4 (26.67)
GBS (n=06)	-	1 (16.67)	4 (66.66)	1 (16.67)
Pleural effusion (n=07)	-	-	3 (42.86)	4 (57.14)



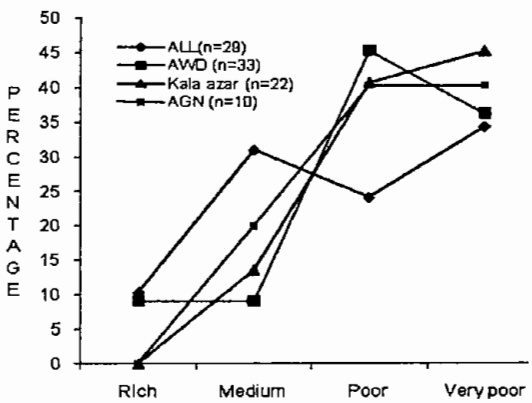
A. Parental socio-economic status and pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis



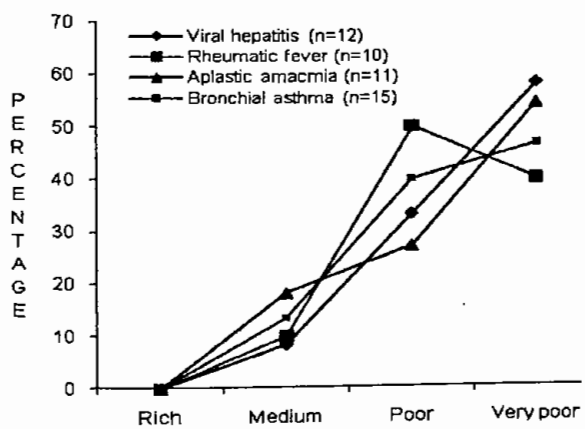
B. Parental socio-economic status and neonatal jaundice, enteric fever, meningitis, encephalitis.



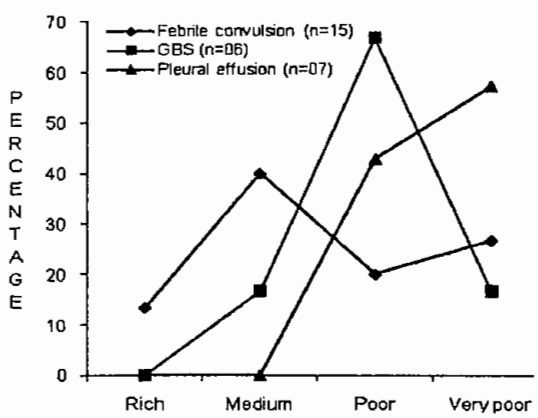
C. Parental socio-economic status and broncholitis, nephritic syndrome, haemolytic anaemia, congenital heart disease.



D. Parental socio-economic status and ALL, AWD, kala azar and AGN.



E. Parental socio-economic status and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



F. Parental socio-economic status and febrile convulsion, GBS, pleural effusion.

Fig. 10. Relation between Parental socio-economic status and disease incidence.

3.11 Nutritional Status and different disease incidence

The incidence of nutritional status in different diseases has been presented in the Table 11. There were 209 cases of pneumonia, of them 40.19% were normal, 37.32% had mild malnutrition, 15.31% had moderate and 7.17% had severe malnutrition. Among 40 enteric fever cases in this study, 35, 40, 17.50 and 7.50% were normal, mild, moderate and severe malnutrition respectively. Out of 47 meningitis cases 34.04% had normal nutrition status, 29.78% had mild, 21.28% had moderate and 14.89% had severe malnutrition. Encephalitis patients were 20, out of them 40% were normal, 35% had mild malnutrition, 20% moderate and remaining 5% were severely malnutrition (Fig 11A).

Among 46 bronchiolitis cases, 43.48% were normal, 32.60% had mild, and 23.91 had moderate malnutrition, no cases of bronchiolitis had suffered from severe malnutrition. Out of 31 nephrotic syndrome cases 51.56% were normal, 25.80% mildly and 22.58% were moderately malnourished. Total 34 hemolytic anaemia cases were considered in this study and among them 23.52% were normal, 44.11, 23.52 and 8.82% were mild nutrient, moderate nutrient, and severely malnourished respectively. Congenital heart disease cases were 15, among them 13.33% were normal, 40% were mild, 33.33% were moderately nutrient and 13.33% were severely malnourished (Fig. 11B).

There were 29 cases of ALL, out of them 44.83% were normal, 34.48% mild, 17.24% moderate, and 3.45% were severe malnourished.

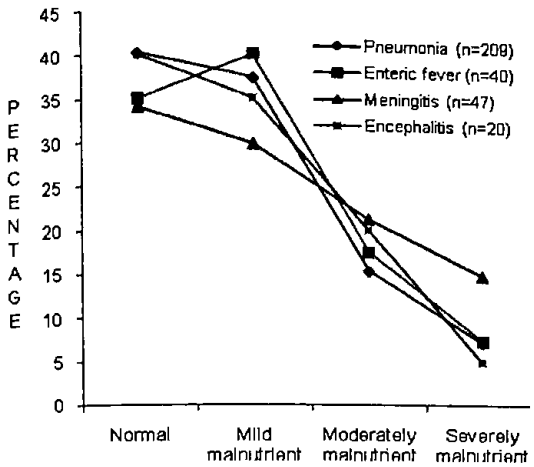
AWD were 33 out of which 18.18% were normal, 42.42% mild, 24.24% moderate and 15.15% severely malnourished. Among 22 kala azar cases in this study 22.72% were normal, 31.82% had mild malnutrition, another 31.82% moderate and 13.64% had severe malnutrition. Out of 10 AGN cases, 60% were normal, 30% had mild and 10% had moderate malnutrition (Fig. 11C).

There were 12 viral hepatitis cases of them 83.33% were normal, and 16.66% had mild malnutrition. Out of 10 rheumatic fever cases 70% were normal and 30% had mild malnutrition. Among 11 aplastic anaemia cases 36.36% had nutritional status within normal limit, 45.45% had mild malnutrition, and 18.18% moderately malnourished. Bronchial asthma cases were 15, among them 46.67% were normal, 26.66% were mild, 20% moderate and remaining 6.66% were severely malnourished (Fig. 11D).

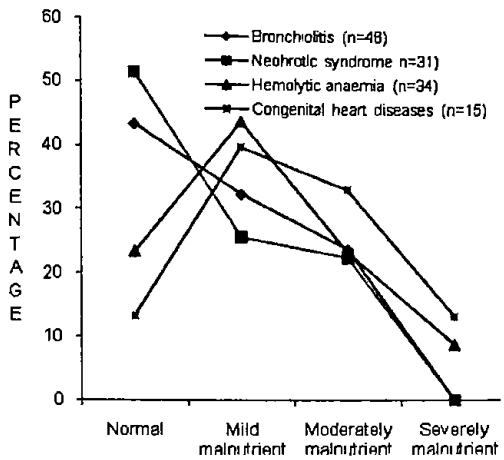
Among 15 febrile convulsion cases 66.66% were normal and 33.33% were mildly malnourished. Out of six GBS cases, 50% were normal, 33.33% mild, and 16.66% were moderately malnourished. Pleural effusion cases were seven among them 42.85% were within normal nutritional status, 14.28% were mild, 28.57% were moderate and 14.28% were severely malnourished (Fig. 1E).

Table 11. Nutritional status and different disease incidence (number of children and percentage within parenthesis) among children of Rajshahi.

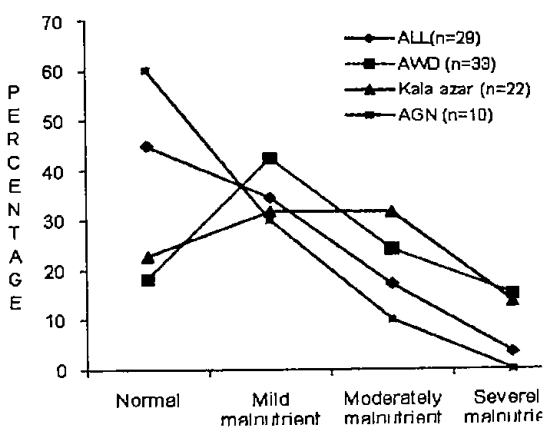
Diseases	Normal	Mild malnutrient	Moderately malnutrient	Severely malnutrient
Pneumonia (n=209)	84 (40.19))	78 (37.32)	32 (15.31)	15 (7.17)
Enteric fever (n=40)	14 (35.00)	16 (40.00)	7 (17.50)	3 (7.50)
Meningitis (n=47)	16 (34.04)	14 (29.78)	10 (21.28)	7 (14.89)
Encephalitis (n=20)	8 (40.00)	7 (35.00)	4 (20.00)	1 (5.00)
Bronchiolitis (n=46)	20 (43.48)	15 (32.60)	11 (23.91)	-
Nephrotic syndrome n=31)	16 (51.56)	8 (25.80)	7 (22.58)	-
Hemolytic anaemia (n=34)	8 (23.52)	15 (44.11)	8 (23.52)	3 (8.82)
Congenital heart diseases (n=15)	2 (13.33)	6 (40.00)	5 (33.33)	2 (13.33)
ALL(n=29)	13 (44.83)	10 (34.48)	5 (17.24)	1 (3.45)
AWD (n=33)	6 (18.18)	14 (42.42)	8 (24.24)	5 (15.15)
Kala azar (n=22)	5 (22.72)	7 (31.82)	7 (31.82)	3 (13.64)
AGN (n=10)	6 (60.00)	3 (30.00)	1 (10.00)	-
Viral hepatitis (n=12)	10 (83.33)	2 (16.66)	-	-
Rheumatic fever (n=10)	7 (70.00)	3 (30.00)	-	-
Aplastic anaemia (n=11)	4 (36.36)	5 (45.45)	2 (18.18)	-
Bronchial asthma (n=15)	7 (46.67)	4 (26.66)	3 (20.00)	1 (6.66)
Febrile convulsion (n=15)	10 (66.66)	5 (33.33)	-	-
GBS (n=06)	3 (50.00)	2 (33.33)	1 (16.66)	-
Pleural effusion (n=07)	3 (42.85)	1 (14.28)	2 (28.57)	1 (14.28)



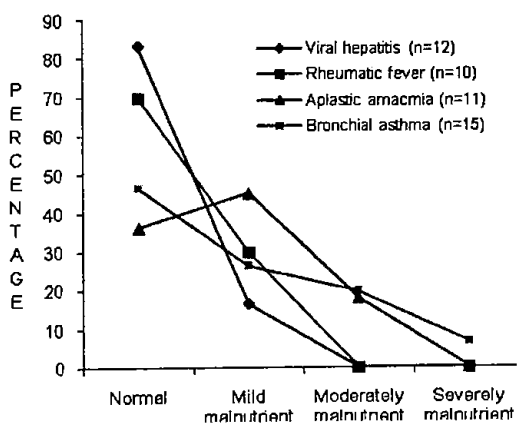
A. Nutritional status and pneumonia, enteric fever, meningitis, encephalitis.



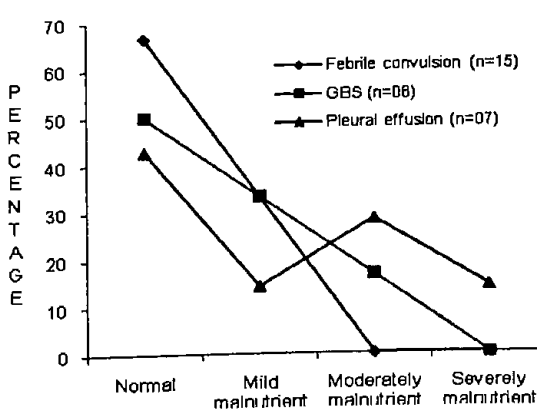
B. Nutritional status and bronchiolitis, nephritic syndrome, haemolytic anaemia, congenital heart disease.



C. Nutritional status and ALL, AWD, kala azar, AGN.



D. Nutritional status and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



E. Nutritional status and febrile convulsion, GBS, pleural effusion.

Fig. 11. Relation between nutritional status and different disease incidence.

3.12 Age of the patients and different disease incidence

The incidence of diseases in different age groups has been presented in Table 12. Among 209 cases of pneumonia in this study 35.88% were within 0-2 months, 34.93% were in the age group 3-6 months, 22.00% in the 7-12 months age group, and only 7.17% cases were in the age group of 1-2 years. All of the 78 perinatal asphyxia, 45 preterm and LBW, 38 neonatal sepsis cases were within the age group of 0-2 months indicates that this diseases occur only at this age group(Fig-12A).

27 neonatal jaundice cases were within the age group of 0-2 months, and it occurs during this period only. Enteric fever cases were 40 among them 37.5% in 3-5 years and 62.5% were in the age group of 6-12 years. Among 47 meningitis cases 10.63% were in 0-2 months, 21.27% were 3-6 months, 36.17% were in 7-12 months, 8.51% in 1-2 years, 12.76% in 3-5 years and 10.64% were in the age group of 6-12 years. Encephalitis cases were 20 among them 40.00% were in 3-5 years group, and 60.00% were in the age group of 6-12 years(Fig-12B).

Broncholitis cases were 46 among them 65.22% were in 3-6 months, 21.74% in 7-12 months and 13.04% in 1-2 years. Out of 31 nephroptic syndrome 83.87% in 3-5 years and 16.13% in the age group of 6-12 years. Hemolytic anaemia cases were 34 among them 14.70% were in 1-2 years, 47.06% in 3-5 years and 38.23% were in the age group of 6-12 years. Congenital heart diseases were 15 and among them 33.33% were in 3-6

months, 26.66% in 7-12 months, 20% in 1-2 years and another 20% in the age group of 3-5 years(Fig-12C).

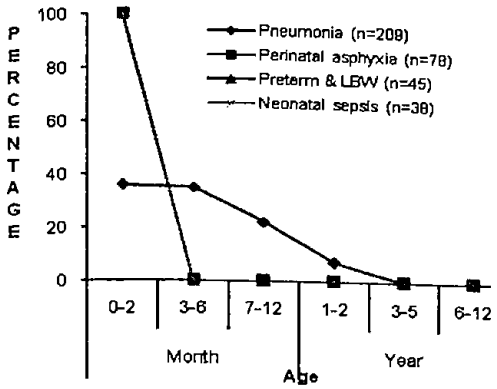
6.89% were in 1-2 years age group, 68.96% in 3-5 years age and 24.14% were in 6-12 years age group of 29 ALL cases in this study. Out of 33 AWD cases 18.18% were in 0-2 months, 30.30% were in 3-6 months, 27.27% in 7-12 months, 15.15% in 1-2 years and 9.09% were in 3-5 years age group. Kala azar cases were 22 among them 54.54% were in 3-5 years and 45.46% were in 6-12 years age group. AGN cases were ten and all were in the age group of 6-12 years(Fig-12D).

Out of 12 viral hepatitis cases 50% were in 3-5 years and another 50% in the age group of 6-12 years. All the ten rheumatic fever and 11 aplastic anaemia cases were in the age group of 6-12 years. Bronchial asthma cases were 15 among them 53.33% were in 3-5 years and 46.67% were in the age group of 6-12 years(Fig-12E).

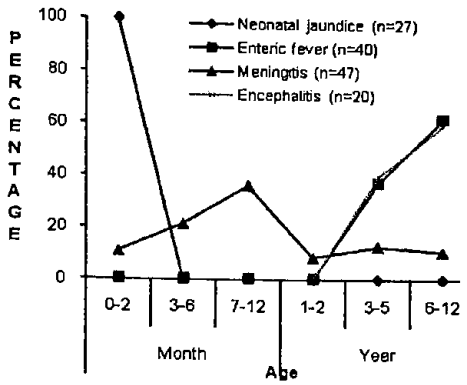
Out of 15 febrile convulsion cases 53.33% were in 1-2 years and 46.67% were in 3-5 years age group. GBS patients were 06 and of them 50% from 3-5 years and another 50% from 6-12 years. There were seven pleural effusion cases in this study and all of them were in the age group of 6-12 years(Fig-12F).

Table 12. Age and different disease incidence among children of Rajshahi.

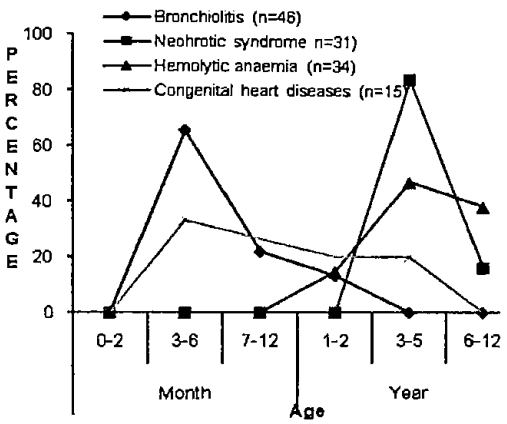
Name of the Disease	Month			Year		
	0-2	3-6	7-12	1-2	3-5	6-12
Pneumonia n=209	75(35.88)	73(34.93)	46(22.00)	15(7.17)	-	-
Perinatal asphyxia n=78	78(100)	-	-	-	-	-
Preterm LBW n=45	45(100)	-	-	-	-	-
Neonatal sepsis n=38	38(100)	-	-	-	-	-
Neonatal jaundice n=27	27(100)	-	-	-	-	-
Enteric fever n=40	-	-	-	-	15(37.5)	25(62.5)
Meningitis n=47	5(10.63)	10(21.27)	17(36.17)	4(8.51)	6(12.76)	5(10.64)
Encephalitis n=20	-	-	-	-	8(40.00)	12(60.0)
Bronchiolitis n=46	-	30(65.22)	10(21.74)	6(13.04)	-	-
Nephrotic syndrome n=31	-	-	-	-	26(83.87)	5(16.13)
Hemolytic anemia n=34	-	-	-	5(14.70)	16(47.06)	13(38.23)
Congenital heart disease n=15	-	5(33.33)	4(26.66)	3(20.00)	3(20.00)	-
ALL n=29	-	-	-	2(6.89)	20(68.96)	7(24.14)
AWD n=33	6(18.18)	10(30.30)	9(27.27)	5(15.15)	3(9.09)	-
Kala-Azar n=22	-	-	-	-	12(54.54)	10(45.46)
AGN n=10	-	-	-	-	-	10(100)
Viral hepatitis n=12	-	-	-	-	6(50.00)	6(50.00)
Rhumatic Fever n=10	-	-	-	-	-	10(100)
Aplastic anaemia n=11	-	-	-	-	-	11(100)
Bronchial Asthma n=15	-	-	-	-	8(53.33)	7(46.67)
Febrile convulsion n=15	-	-	-	8(53.33)	7(46.67)	-
GBS n=06	-	-	-	-	3(50.00)	3(50.00)
Plural effusion n=07	-	-	-	-	-	7(100)



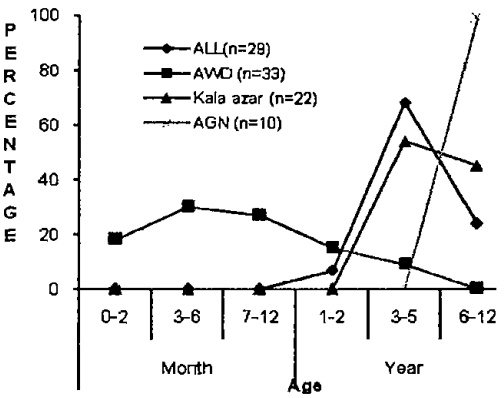
A. Children age and pneumonia, prenatal asphyxia, preterm & LBW, neonatal sepsis.



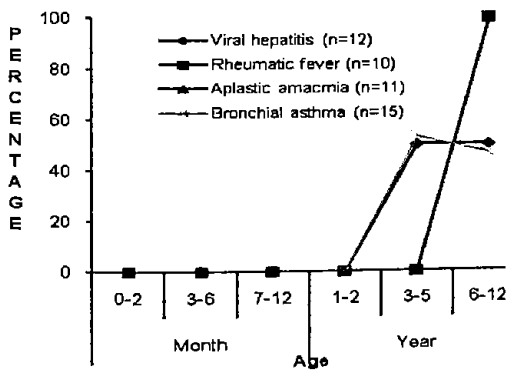
B. Children age and neonatal jaundice, enteric fever, meningitis, encephalitis.



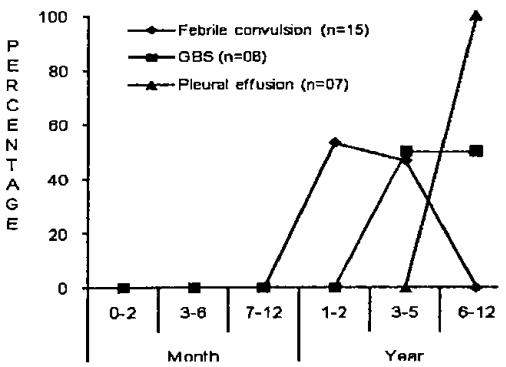
C. Children age and bronchiolitis, neohrotic syndrome, haemolytic anaemis, congenital heart disease.



D. Children age and ALL, AWD, kala azar, AGN.



E. Children age and viral hepatitis, rheumatic fever, aplastic anaemia, bronchial asthma.



F. Children age and febrile convulsion, GBS, pleural effusion.

Fig. 12. Relation between children ages and different disease incidence.

Plate 1. Di rrheal diseased infant feeding ORS

Plate 2. Pneumonic child with chest indrawing.

Plate 3. Pneumonic child with chest indrawing and abdominal distension.

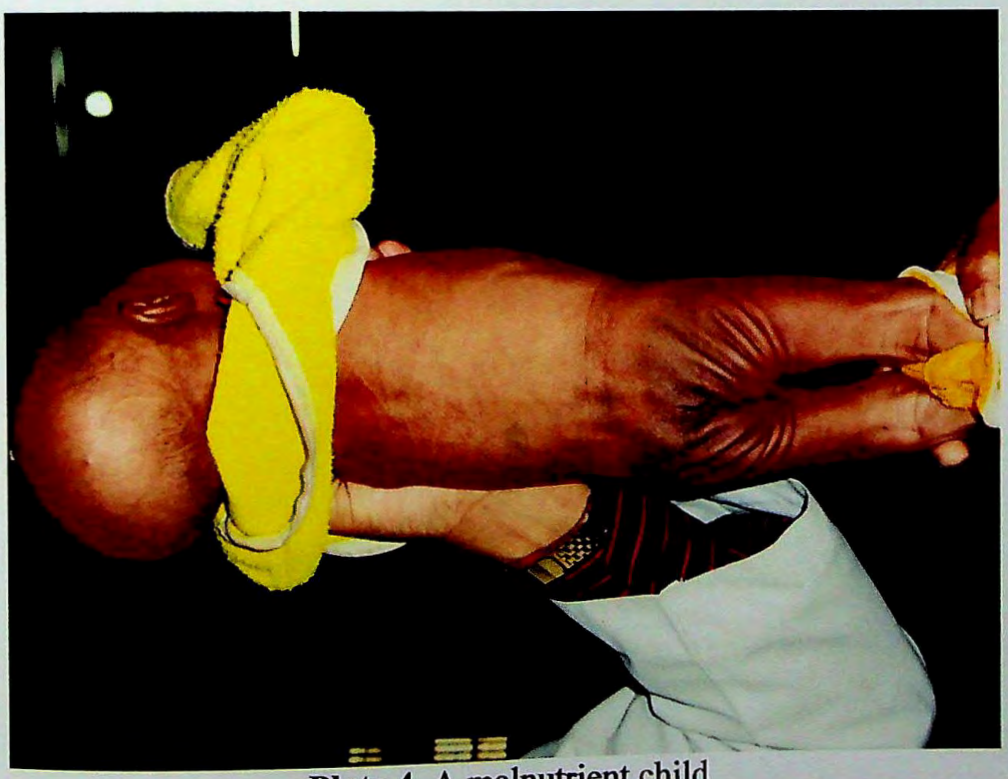


Plate 4. A malnutrient child

Chapter 4

DISCUSSION

Chapter 4

DISCUSSION

Breastfeeding has been suggested as a modifiable influencing factor. When given exclusively, breastfeeding reduces the risk of infectious diseases in infants in developing countries (WHO 2000, Bahl *et al.* 2005). In industrialized countries, exclusive breastfeeding during the first 6 months seems to decrease the risk of gastrointestinal tract infections, compared with exclusive breastfeeding during only the first 3 to 4 months (Kramer *et al.* 2003). On the basis of these and other reports, the WHO (2001) recommended that all children be exclusively breastfeed for 6 months instead of 4 months. However, the organization also called for more research regarding the benefits of 6 vs 4 months of exclusive breastfeeding (WHO 2001). Thus far, several studies in industrialized countries revealed that a shorter duration of breastfeeding increases the risk of common infectious diseases, such as respiratory and gastrointestinal tract infections (Howie *et al.* 1990, Duncan *et al.* 1993, Bachrach *et al.* 2003, Kramer *et al.* 2003, Oddy *et al.* 2003, Pettigrew *et al.* 2003, Pardo-Crespo *et al.* 2004, Chantry *et al.* 2006, Paricio *et al.* 2006, Quigley *et al.* 2007, Qiu *et al.* 2010).

Breastfeeding might have a prolonged protective effect by influencing the severity, including hospital admission and frequency, of common infectious diseases (Quigley *et al.* 2006, 2007). However,

immunologic evidence of a prolonged protective effect of increased dose and duration of breastfeeding has not been well established. Short-term protective effects are caused by several factors in human breast milk. Epidermal growth factor helps to induce maturation of the intestinal epithelium, immunoglobulin A and oligosaccharides prevent attachment of pathogens, and lactoferrin has broad antimicrobial properties including disruption of the bacterial outer membrane (Hanson *et al.* 2002, Lawrence and Pane 2007).

Both mother and child are affected by infant feeding. The duration, frequency and the amount of feeding affect the child's nutritional status, which influences the infant's health (Howie 2002). An adequate supply of human breast milk is known to satisfy virtually all the nutritional needs of an infant on a minimum of 6 months. Breast milk is easily digestible and facilitates skin-to-skin contact and physical warmth between mother and child, strengthening the emotional bond between them (Dermer 1998). Breast milk and especially colostrum, in the long term, prevents arteriosclerosis, hypertension and obesity. It also prevents allergy to non-specific proteins and develops immunity (van Odijk *et al.* 2003, Scholtens *et al.* 2010). Similarly, studies also reported health benefits to mothers who breastfeed their infants. These include protective effects against breast cancer in premenopausal women, ovarian cancer and osteoporosis (Dermer 1998).

Optimal infant- and young child-feeding practices are crucial for nutritional status, growth, development, health, and ultimately the survival of infants and young children (Black *et al.* 2003, Bhutta *et al.* 2008, Saha *et al.* 2008). Worldwide, suboptimal breastfeeding still accounts for deaths of 1.4 million children aged less than five years (under-five mortality). The timely introduction of complementary feeding can prevent almost 6% of under-five mortality (Jones *et al.* 2003). It was estimated that, if 90% of infants are covered with a package of intervention to protect, promote, and support the optimal young child-feeding practices, almost one-fifth of overall under-five mortality can be averted (Jones *et al.* 2003). The poor complementary feeding practices mean that many children continue to be vulnerable to irreversible outcomes of stunting, poor cognitive development, and significantly increased risk of infectious diseases, such as diarrhoea and acute respiratory infection (Saha *et al.* 2008, WHO 1998, Hop *et al.* 2000).

Infants who were breastfed for 4 months or 4 to 6 months did not have lower risks of upper and lower respiratory or gastrointestinal tract infections in the first 6 months compared with never-breastfed infants (all $P>0.05$) (Duijts *et al.* 2010). Compared with never breastfed infants, those who were breastfed for 6 months or longer had lower risks of upper respiratory tract infections, lower respiratory tract infections and gastrointestinal tract infections. The Cebu, Philippines, case study reaffirms the general finding of a large body of research that breast-feeding protects against diarrheal morbidity (Feachem and Koblinsky 1984, Popkin *et al.*

1986). Popkin *et al.* (1990) observed that adding either nonnutritive liquids or nutritive foods or liquids to the breast-milk diet is associated with a large increase in the occurrence of diarrhea and that the protective effect of breast-feeding decreases with age.

Exclusive breast feeding decreases diarrhoeal mortality seven fold. It is associated with a 40% reduction in diarrhoeal disease in infancy (Kramer *et al.* 2001), with even greater protection against hospitalisation or persistent diarrhoea. There is evidence of a dose-response relationship with 6 months of exclusive breast feeding giving the best protection. The mechanisms by which breast feeding protects against DD are multiple and include the contents of breast milk, the better nutritional status of the child, the low cost and the promotion of the mother-child relationship. (Huttly *et al.* 1997).

The main findings of this population based prospective cohort study were that breastfeeding for 6 months seems to have protective effects for development of respiratory and gastrointestinal tract infections during the first 6 months. Several studies have revealed that a shorter period of breastfeeding increases the risks of physician visits for illness, lower respiratory tract infections, and gastrointestinal symptoms (Howie *et al.* 1990, Pettigrew *et al.* 2003, Quigley *et al.* 2007). Studies that were able to take the exclusiveness of breastfeeding into account revealed that exclusive breastfeeding, followed by partial breastfeeding, or predominant breastfeeding during 6 months or more was associated with a lower risk of

gastrointestinal tract infection compared with breastfeeding for less than 3 months (Kramer *et al.* 2003). Infants who were breastfed for less than 4 months had a higher risk of hospitalization for infectious diseases compared with those who were breastfed for more than 4 months. In addition, infants who were breastfed for 4 to 6 months showed higher risks of both pneumonia and recurrent otitis media compared with those who were breastfed 6 months or longer (Chantry *et al.* 2006, Paricio *et al.* 2006).

The present results are difficult to compare with these studies, because different breastfeeding categories and various definitions of the breastfeeding categories (predominant or exclusive) and the outcomes (self-reported or doctor-diagnosed infections) were used. We observed protective effects of breastfeeding on infectious diseases mainly in the first 6 months of life. Most studies have revealed protective effects of breastfeeding on common infections in the first 8 to12 months of life (Howie *et al.* 1990, Chantry *et al.* 2006, Paricio *et al.* 2006, Quigley *et al.* 2007). One study, which distinguished between infectious diseases until and from the age of 6 months, revealed results similar to those from our study (Kramer *et al.* 2003). Although the authors used exclusive breastfeeding for 3 months as the reference group, exclusive breastfeeding for 6 months reduced the risk of gastrointestinal tract infections between the ages of 3 and 6 months but not between the ages of 6 and 12 months (Kramer *et al.* 2003).

Several studies have revealed that a shorter period of breastfeeding increases the risks of physician visits for illness, lower respiratory tract infections, and gastrointestinal symptoms (Howie *et al.* 1990, Pettigrew *et al.* 2003, Quigley *et al.* 2007). Studies that were able to take the exclusiveness of breastfeeding into account revealed that exclusive breastfeeding, followed by partial breastfeeding, or predominant breastfeeding during 6 months or more was associated with a lower risk of gastrointestinal tract infection compared with breastfeeding for less than 3 months (Kramer *et al.* 2003). Infants who were breastfed for less than 4 months had a higher risk of hospitalization for infectious diseases compared with those who were breastfed for more than 4 months (Duijts *et al.* 2010). In addition, infants who were breastfed for 4 to 6 months showed higher risks of both pneumonia and recurrent otitis media compared with those who were breastfed 6 months or longer (Chantry *et al.* 2006, Paricio *et al.* 2006).

A study in Ghana reported that 22% of all neonatal deaths could be prevented if all women could initiate breastfeeding within one hour of delivery (Edmond *et al.* 2006, 2007). An epidemiological evidence of a causal association between early initiation of breastfeeding and infection-specific neonatal mortality has also been documented (Edmond *et al.* 2007). The use of prelacteal feeding was 8% in rural Bangladesh (Saha *et al.* 2008) and 71% in urban Bangladesh (Hassan *et al.* 2006). Although this practice was prevalent across the cultures, there was an international consensus that

providing other liquids in addition to breastmilk in the first six months of life was unnecessary and harmful (Martines *et al.* 1992).

Exclusive breastfeeding under six months (17.38%) in the present study was far short from all-India average of 46.4% (IIPS 2007). This difference in breastfeeding is due to because the present study was undertaken on the hospitalized children only but the IIPS (2007) study was done on cohort basis on a population. Similar findings were observed by Saha *et al.* (2008) in Bangladesh and Hop *et al.* (2000) in Viet Nam in longitudinal studies. Several studies showed that partial breastfeeding was associated with increased risk of child morbidity and mortality (Black *et al.* 2003, Jones *et al.* 2003, Bhutta *et al.* 2008). Even introduction of plain water was reported to interfere with breastfeeding (Sachdev *et al.* 1991). If the practice of giving plain water could be avoided, almost 15% increase in exclusive breastfeeding rate could be achieved (Sinababu *et al.* 2010). About one-fourth of study children who received liquids and solids, along with breastfeeding at 0-6 months of age, remained at risk for infectious diseases and undernutrition (Black *et al.* 2003, Jones *et al.* 2003). Because of associated exposure to pathogens and interference with successful breastfeeding, current recommendations strongly discouraged bottle-feeding (PAHO 2003). Cousens *et al.* (1993) found that, when prolonged breastfeeding was accompanied with complementary solid foods, there was a reduction in clinical malnutrition (Cousens *et al.* 1993).

The current recommendations advocated the introduction of complementary food after six months of exclusive breastfeeding (WHO 2003, PAHO 2003). A positive association was observed between the intake of complementary food and the nutritional status in Yemen (Jumaan *et al.* 1989). A study in Bangladesh documented that the frequency, amount, energy-density, and diversity of food remained important issues in complementary feeding (Kimmons *et al.* 2005). Factors, such as characteristics of diet or child's appetite, are known to influence the frequency of complementary feeding (Wamani *et al.* 2005, Dewey and Brown, 2003). Although these were not measured in this study, it is unlikely that such factors could solely explain the observed deviance from recommendations. Traditional beliefs and practices, besides lack of knowledge regarding current feeding recommendations, might also play a part (Dobe 2002).

Results of studies on cessation of breastfeeding of children suggest that mothers who have lower education stop breastfeeding earlier than those with higher education (Morisky *et al.* 2002, Lande *et al.* 2003, Aryal 2007). Other factors that also relate to the duration of breastfeeding are present age of mothers and socioeconomic status (Michaelsen *et al.* 1994, Nolan and Goel 1995, Killersreiter *et al.* 2001, Lande *et al.* 2003, Giashuddin and Kabir 2003, 2004). Younger mothers are most likely to terminate breastfeeding early compared to older counterparts (Akin *et al.* 1981, Jain and Bongaarts 1981, Islam *et al.* 2006).

Education and occupation have been considered to measure socio-economic status of a woman (Yadava and Jain 1998). Since in Bangladesh, more than 75 percent of the mothers were found illiterate with no earning sources. So, husband's education has also been included for the analysis. The education showed an inverse relationship with the duration of BF, which may be due to the fact that literate mothers probably start giving food supplements to their children earlier and so a shorter period of lactation. Similar, findings have also been obtained by other researchers based on data of developing countries (Jain and Bongaarts 1981, Ahamed 1986).

A strong association was found between delayed initiation of breastfeeding and increased neonatal mortality in a large observational study in rural Ghana (Edmond *et al.* 2006). Other studies have also suggested that breast milk may have its greatest effects in the neonatal period (Habicht *et al.* 1986, Victora *et al.* 1987). However, there is little evidence from randomized controlled trials, and inferring causation from observational studies is fraught with difficulties. Edmond *et al.* (2006) reported a marked dose-response relation in our previous analysis; with neonatal mortality increasing significantly as delay in initiation of breastfeeding increased. However, evidence about biological plausibility and effect on cause-specific mortality in neonates is sparse.

Early initiation of breastfeeding may reduce neonatal mortality by decreasing the ingestion of infectious pathogens (Clemens *et al.* 1999). Early

breast milk also provides many immunocompetent factors, including immunoglobulins and lymphocytes that may stimulate humoral or cell-mediated immune systems (Goldman *et al.* 1982, Goldman 1993, Brandtzaeg 2003), and it may also prime the gastrointestinal tract (GIT) and decrease intestinal permeability and translocation of infectious pathogens, including HIV (Goldman 2000, Rollins *et al.* 2001). Close skin-to-skin contact between the maternal-infant dyad may also stimulate the mucosa-associated lymphoid tissue system (Brandtzaeg 2002, Anderson *et al.* 2003). In contrast, prelacteal feeding and predominant and partial breastfeeding may result in the ingestion of infectious pathogens and may also act on the early GIT to increase permeability (Badruddin *et al.* 1991, Goldman 2000). Metabolic pathways (especially glucose or sodium homeostasis) may also be significantly disrupted (Hawdon *et al.* 1992, De Rooy and Hawdon 2002, Avery and Fletcher 2005).

A large study from Bangladesh was designed to detect associations between type of neonatal breastfeeding (exclusive, predominant, partial) and cause-specific neonatal mortality, but effects on all-cause neonatal mortality were only reported (Arifeen *et al.* 2001). A study from Pakistan reported a 3-fold reduction in risk of early neonatal sepsis in exclusively breastfed compared with partially breastfed hospitalized neonates (Bhutta and Ysuf 1997). Case reports were also published that describe hypernatremia, acidosis, and hypoglycemia in neonates provided with prelacteal feeds (Akre 1989, de L Costello *et al.* 2000, Oppe and Redstone 1968). Overall, these

results indicate that early breast milk is significantly associated with reduced infection-specific neonatal mortality in young infants (Edmond *et al.* 2007).

It is generally believed that breast feeding directly promotes overall health of the child and results in decreased childhood morbidity and mortality (Morisky *et al.* 2002). In this study breast feeding was practiced by 97.54% of women similar results 97.0% were reported by Benakappa *et al.* (2007) in study of Srilanka (Agampodi *et al.* 2007) they observed 100% initiation of breast feeding but exclusive breast feeding up to 4 months was seen in 61.6% of study women. In our study, ratio of exclusive breast feeding was 68.70% which is slightly higher than above results.

Small number 2.4% of women did not breast feed their babies, the reasons were maternal serious illness, figure consciousness, insufficient milk and occupation problem, almost same reasons were reported in the study of India and mini survey study of Pakistan (Morisky *et al.* 2002, Benakappa *et al.* 2007, Kalra *et al.* 1982). We divided age in two groups among them most of the women 63.15% were in 31-40 years age group. When educational level was assessed, the ratio of primary educated women was 35.37% and uneducated was 25.94% and also breast feeding practices were seen more in this group of study population. Similarly Singh and Bhalwar (2007) reported that most mothers 22.8% and 28.6% were primary and secondary school educated, small number 4.6% were illiterate, in contrast we found large number of uneducated women in our study, the reason may

be that our study women mostly were from nearby rural areas of the hospital (Singh and Bhalwar 2007, Victoria *et al.* 2005).

As far as the duration of breast feeding is concerned, majority 36.49% of women breast feed their babies for 2 years and even 1.75% continued it more than 2 years, with little variation to our results in one survey of Pakistan, reported that 17.6% of mothers breast feed up to 5 months, 35.6% up to 1 year and 29.2% beyond 1 year also in one study they observed, that duration of breast feeding varied according to mothers educational level and socioeconomic status of family and longer duration was seen in women of rural areas (Morisky *et al.* 2002, Kalra *et al.* 1982).

In the present the findings indicate that most of the diseases studies had association with the breast feeding. Though some of the diseases are not statistically related with the breast feeding but due to immunity development as a result of breast feeding the major killing diseases could be prevented.

Chapter 5

CONCLUSION

Chapter 5

CONCLUSION

In summary, this study conducted in Rajshahi Medical College Hospital, Bangladesh has shown that infants who are exclusively breastfed from birth to six months of age have a significantly lower prevalence of infection than those infants who are not exclusively breastfed. It is found that pneumonia, perinatal asphyxia, preterm and LBW, neonatal sepsis, neonatal jaundice, encephalitis, congenital heart diseases were significantly negatively related with breastfeeding. Enteric fever, meningitis, bronchiolitis, hemolytic anaemia, AGN, AWD and GBS were also negatively associated with breast feeding.

Undoubtedly breastfeeding is invaluable in the developing world, particularly amongst the lower socioeconomic and disadvantaged groups. But the cultural practice of food avoidance of many nutritious foods and restrictive diet could affect the overall health and well-being of both the mother and her infant. The practice of withholding the breast after birth, discarding valuable colostrum, and giving prelacteal feeds to the newborn needs to be urgently addressed through programs and breastfeeding interventions that infiltrate to the rural areas and urban slums across the country.

The practice of introducing early supplementary food is another major concern in terms of infant health. Breastfeeding promotion and intervention activities in Bangladesh should take into account the cultural and traditional practices that impact on postpartum women's health and the belief that breast milk is insufficient for an infant until six months.

Chapter 6

LITERATURE CITED

Chapter 6

LITERATURE CITED

AAP (American Academy of Pediatrics). 2005. Policy statement: Breastfeeding and the use of human milk. *Pediatrics*, 115: 496-506.

Agampodi SB, Agampodi TC, Piyaseeli UKD. 2007. Breastfeeding practices in a public health field practice area in Sri Lanka: a survival analysis *Int. Breastfeed. J.* 2:13 doi:10.1186/1746-4358-2-13

Ahamed MM. 1986. Breastfeeding in Bangladesh, *J. Biosocial Sci.*, 18(4): 425-34.

Ahlstedt S, Carlsson B, Hanson LÅ, Goldblum RM. 1975. Antibody production of human colostral cells. I. Immunoglobulin class, specificity, and quantity. *Scand. J. Immunol.*, 4: 535-539.

Akin JS, Bilsborrow R, Guilkey DK, Popkin BM, Benoit D, Cantrelle P, Garenne M, Levi P. 1981. The determinants of breast-feeding in Sri Lanka. *Demography*, 18: 287-307.

Akre J. 1989. Infant feeding: the physiological basis. *Bull WHO*, 67(suppl): 1–108.

Anderson GC, Moore E, Hepworth J, Bergman N. 2003. Early skin-to-skin contact for mothers and their healthy newborn infants. *Cochrane Database Syst. Rev.*, 2: CD003519.

Arifeen S, Black RE, Antelman G, Bacqui A, Caulfield L, Becker S. 2001. Exclusive breastfeeding reduces acute respiratory infection and diarrhea deaths among infants in Dhaka slums. *Pediatrics,* 108: e67.

Aryal TR. 2007. Breastfeeding in Nepal: patterns and determinants. *J. Nepal Med. Assoc.*, 46: 13-19.

Astrup-Jensen A. 1988. Environmental and occupational chemicals. In: Bennett PN, ed. *Drugs and human lactation.* Amsterdam: Elsevier, 551-573.

Avery GB, Fletcher MA. 2005. Neonatology: pathophysiology and management of the newborn. London, United Kingdom: Lippincott, Williams& Wilkins.

Axelsson I, Jakobsson I, Lindberg T, Benediktsson B. 1986. Bovine beta-lactoglobulin in breast milk. *Acta Paediatr. Scand.*, 75: 702-707.

Bachrach VR, Schwarz E, Bachrach LR. 2003. Breastfeeding and the risk of hospitalization for respiratory disease in infancy: a meta-analysis. *Arch. Pediatr. Adolesc. Med.* 157(3): 237-243.

Badruddin S, Islam A, Hendricks K. 1991. Dietary risk factors associated with acute and persistent diarrhea in children in Karachi, Pakistan. *Am. J. Clin. Nutr.*, 54: 745–749.

Bahl R, Frost C, Kirkwood BR, Edmond K, Martines J, Bhandari N, Arthur P. 2005. Infant feeding patterns and risks of death and hospitalization in the first half of infancy: multicentre cohort study. *Bull WHO,* 83(6): 418-426.

Baldus SE, Thiele J, Park Y-O, Charles A, Mross C, Hanisch F-G, Zirbes TK, Wickenhauser C, Fischer R. 1995. Carbohydrate and peptide antigens in macrophage populations derived from human bone marrow and milk: an immunomorphological and immunochemical analysis. *Histochem. J.*, 27: 630-638.

Ballabriga A. 1994. Essential fatty acids and human tissue composition. An overview. *Acta. Paediatr.*, S402: 63-68.

Bamslaug N, Michels DL. 1995. Milk, money and madness: *The culture and politics of breastfeeding*. Westport, CT: Bergin and Garvey.

Basolo F, Conaldi PG, Fiore L, Calvo S, Toniolo A. 1993. Normal breast epithelial cells produce interleukins 6 and 8 together with tumor-necrosis factor: defective IL-6 expression in mammary carcinoma. *Int. J. Cancer*, 55: 926-930.

Beer AE, Billingham RE, Head J. 1974. The immunologic significance of the mammary gland. *J. Invest. Dermatol.* 63: 65-74.

Benakappa DG, Raju MS, Benakappa AD. 2007. Breast feeding practices in rural Karnataka (India) with special reference to lactation failure. *J. Japan Pediatr. Int.*, 31; 391-398.

Bennett PN. 1988. *Drugs and human lactation.* Amsterdam: Elsevier.

Berg A and Brem S. 1989. A case for promoting breastfeeding in projects tolimit fertility. World Bank technical paper no. 102. Washington.

Berlin CM. 1981. Excretion of methylxanthines in human milk. *Semin. Perinatol.* 5: 389-394.

Bernt KM, Walker WA. 1999. Human milk as a carrier of biochemical messages. *Acta Paediatr.*, 430(Suppl): 27-41.

Bertotto A, Gerli R, Fabietti G, Crupi S, Arcangeli C, Scalise F, Vaccaro R. 1990. Human breast milk T lymphocytes display the phenotype and functional characteristics of memory T cells. *Eur. J. Immunol.*, 20:1877-1880.

Bhutta ZA, Ysuf K. 1997. Early onset neonatal sepsis in Pakistan: a case control study of risk factors in a birth cohort. *Am. J. Perinatol.,* 14: 577–581.

Bhutta ZA, Ahmed T, Black RE, Cousens S, Dewey K, Giugliani E, Haider BA, Kirkwood B, Morris SS, Sachdev HPS, Shekar M. 2008. What works? Interventions for maternal and child undernutrition and survival. *Lancet*, 371: 417-440.

Binns C, Davidson G. 2003. Infant Feeding Guidelines for Health Workers. In Dietary Guidelines for Children in Australia Canberra: National Health and Medical Research Council.

Black RE, Morris SS, Bryce J. 2003. Where and why are 10 million children dying every year? *Lancet*, 361(9376): 2226-2234.

Böttcher MF, Jenmalm MC, Garofalo RP, Björkstén B. 2000a. Cytokines in breast milk from allergic and nonallergic mothers. *Pediatr. Res.*, 47: 157-162.

Böttcher MF, Jenmalm MC, Björkstén B, Garofalo RP. 2000b. Chemoattractant factors in breast milk from allergic and nonallergic mothers. *Pediatr. Res.*, 47: 592-597.

Brandtzaeg P. 2002. Current understanding of gastrointestinal immunoregulation and its relation to food allergy. *Ann. N. Y. Acad. Sci.*, 964: 13–45.

Britton JR, Britton HL and Gronwaldt V. 2006. Breastfeeding, Sensitivity and Attachment. *Pediatrics*, 118(5): 1436

Bryan D-L, Hawkes JS, Gibson RA. 1999. Interleukin-12 in human milk. *Pediatr. Res.*, 45: 858-859.

Buescher ES, McIlheran SM. 1993. Polymorphonuclear leukocytes and human colostrum: Effects of *in vivo* and *in vitro* exposure. *J. Pediatr.*, 17: 424-433.

Bush JF, Beer AE. 1979. Analysis of complement receptors on B-lymphocytes in human milk. *Am. J. Obstet. Gynecol.*, 133: 708-712.

Businco L, Cantani A, Meglio P, Bruno G. 1987. Prevention of atopy: results of a long-term (7 months to 8 years) follow-up. *Ann. Allergy*, 59: 183-186.

Businco L, Marchetti F, Pelligrini G, Cantani A, Perlinin R. 1983. Prevention of atopy disease in at risk newborns by prolonged breast feeding. *Ann. Allergy*, 51: 296-299.

Campbell S, Mongar A. 2006. In: *Ten Teachers Obstetrics* (pp 286-299). 18th (Edn) London UK, British Publishers.

Carlos TM, Harlan JM. 1994. Leukocyte-endothelial adhesion molecules. *Blood*, 84: 2068-2101.

Carnielli VP, Luijendijk IHT, van Goudoever JB, Sulkers EJ, Boerlage AA, Degenhart HJ, Sauer PJJ. 1995. Feeding premature newborn infants palmitic acid in amounts and stereoisomeric position similar to that of human milk: effects on fat and mineral balance. *Am. J. Clin. Nutr.*, 61: 1037-1042.

Chandra RK, Puri S, Cheema PS. 1985. Predictive value of cord blood IgE in the development of atopic disease and role of breast feeding in its prevention. *Clin. Allergy*, 15: 517-522.

Chandra RK, Puri S, Suraya C, Cheema PS. 1986. Influence of maternal food antigen avoidance during pregnancy and lactation on incidence of atopic eczema in infants. *Clin. Allergy*, 16: 563-539.

Chantry CJ, Howard CR, Auinger P. 2006. Full breastfeeding duration and associated decrease in respiratory tract infection in USchildren. *Pediatrics*, 117(2): 425– 432.

Chappell JE, Clandinin MT, Kearney-Volpe C. 1985. Trans fatty acids in human milk lipids: influence of maternal diet and weight loss. *Am. J. Clin. Nutr.* 42: 49-56.

Chowdhury AKMA, Huffman SL and Curlin GT. 1977. Amalnutrition Menarche and Marriage in Rural Bangladesh. Dhaka: Choler Research laboratory.

Clark SGJ, Bungum TJ. 2003. The Benefits of Breastfeeding: An Introduction for Health Educators. *Californian J. Health Promot.* 1(3): 158-163.

Clemens J, Elyazeed RA, Rao M, Savarino S, Morsy BZ, Kim Y, Wierzba T, Naficy A, Lee YJ. 1999. Early initiation of breastfeeding and the risk of infant diarrhea in rural Egypt. *Pediatrics,* 104: E3.

Clemente J, Clerici N, Espinosa MA, Leyva-Cobián F. 1986. Defective response of human alveolar and colostral macrophages. *Immunol, Lett,* 12: 271-276.

Cousens S, Nacro B, Curtis V, Kanki B, Tall F, Traore E, Chen JA. 1993. Prolonged breast-feeding: no association with increased risk of clinical malnutrition in young children in Burkina Faso. *Bull WHO.* 71:713-22.

Crago SS, Prince SJ, Pretlow TG, McGhee JR, Mestecky J. 1979. Human colostral cells. 1. Separation and characterization. *Clin. Exp. Immunol.*, 38: 585-597.

Cruz JR, Garcia B, Urrutia JJ, Carlsson B, Hanson L. 1981. Food antibodies in milk from Guatemalan women. *J. Pediatr.*, 99: 600-602.

Cummings NP, Neifert MR, Pabst MJ, Johnston RB Jr. 1985. Oxidative metabolic response and microbicidal activity of human milk macrophages: effect of lipopolysaccharide and muramyl dipeptide. *Infect. Immun.*, 435-439.

Cummins AG, Thompson FM. 1997. Postnatal changes in mucosal immune response: A physiological perspective of breast feeding and weaning. *Immunol. Cell Biol.*, 75: 419-429.

Cunningham AS. 1979. Morbidity in breast-fed and artificially-fed infants. II. J Pediatr 95: 685-689.

Cunningham AS. 1995. Breastfeeding: Adaptive behavior for child health and longevity. In Stuart-Macadam P, Dettwyler KA (Eds.), *Breastfeeding: Biocultural perspectives* (pp. 243-264). New York: Walter de Gruyter.

Cutting WAM. 1993. Breastfeeding and HIV infection: advice depends on the circumstances. *Pediatr. AIDS HIV Infect.*, 4:1-2.

Dahlstrom A, Lundell B. Curvall M, Thapper L. 1990. Nicotine and cotinine concentrations in the nursing mother and her infant. *Acta. Paediatr. Scand.*, 79: 142-147.

Darmstadt GL, Bhutta ZA, Cousens S, et al. Adam T, Walker N, de Bernis L. 2005. Evidence-based, cost-effective interventions: how many newborn babies can we save? *Lancet*, 365: 977–988.

de L Costello AM, Pal DK, Manandhar DS, Rajbhandari S, Land JM, Patel N. 2000. Neonatal hypoglycaemia in Nepal 2: availability of alternative fuels. *Arch. Dis. Child Fetal Neonatal Ed.*, 82: F52– 58.

De Rooy L, Hawdon J. 2002. Nutritional factors that affect the postnatal metabolic adaptation of full-term small- and large-for-gestational age infants. *Pediatrics*, 109: e42.

Dermer A. 1998. Breastfeeding and women's health. *J. Women's Health*, 7: 427–433.

Dewey KG, Brown KH. 2003. Update on technical issues concerning complementary feeding of young children in developing countries and implications for intervention programs. *Food Nutr. Bull.*,24: 5-28.

DiLallo D, Bertollini R. CamposVenuti G. Risica S. Perucci CA, Simula S. 1987. Radioactivity in breast milk in Central Italy in the aftermath of Chernobyl. *Acta. Paediatr. Scand.*, 76: 530-531.

Dobe M. 2002. Optimal infant feeding in rural areas—the missing agenda of communication needs. *Indian J. Public Health*, 46: 145-150.

Duchén K, Björkstén B. 1996. Total IgE levels in human colostrum. Pediatr. *Allergy Immunol.*, 7: 44-47.

Duijts L, Jaddoe VWV, Hofman A, Moll HA 2010. Prolonged and Exclusive Breastfeeding Reduces the Risk of Infectious Diseases in Infancy. Pediatrics, 126: e18-e25. Available at: http://www.pediatrics.org/ cgi/content/full/126/1/e18.

Duncan B, Ey J, Holberg CJ, Wright AL, Martinez FD, Taussig LM. 1993. Exclusive breastfeeding for at least 4 months protects against otitis media. *Pediatrics*, 91(5): 867– 872.

Edmond KM, Zandoh C, Quigley MA, Amenga-Etego S, Owusu-Agyei S, Kirkwood BR. 2006. Delayed breastfeeding initiation increases risk of neonatal mortality. *Pediatrics*, 117: 380–386.

Edmond KM, Kirkwood BR, Amenga-Eteg S, Owusu-Agyei S, Hurt LS. 2007. Effect of early infant feeding practices on infection-specific neonatal mortality: an investigation of the causal links with observational data from rural Ghanal *Am. J. Clin. Nutr.* 86: 1126 – 1131.

Eglinton BA, Roberton DM, Cummins AG. 1994. Phenotype of T cells, their soluble receptor levels, and cytokine profile of human breast milk. *Immunol. Cell Biol.,* 72: 306-313.

Ekstrom A, Nissen E. 2006. A mother's feelings for her infant are strengthened by excellent breastfeeding counseling and continuity of care. *Pediatrics*, 118: 309-314.

Elahi S, Buchanan RM, Babiuk LA, Gerdts V. 2006.Maternal immunity provides protection against pertussis in newborn piglets. *Infect. Immu.*, 74(5): 2619–2627.

Else-Quest NM, Hyde JS, Clark R. 2003. Breastfeeding, bonding and the mother-infant relationship. *Merrill-Palmer Qtly.*, 49(4): 495-517.

Feachem RG, Koblinsky MA. 1984. Interventions for the control of diarrhoeal diseases among young children: promotion of breast-feeding. *Bull. WHO*, 62: 271-291.

Ford K and Huffman S. 1988. Nutrition, infant feeding and postpartum amenorrhoea in rural Bangladesh. *J. Biosoc. Sci.*, 20: 461-469.

France GL, Marmer DJ, Steele RW. Breast-feeding and Salmonella infection. *Am. J. Dis. Child*, 1980. 134:147-152.

Garofalo R, Chheda S, Mei F, Palkowetz KH, Rudloff HE, Schmalstieg FC, Rassin DK, Goldman AS. 1995. Interleukin-10 in human milk. *Pediatr. Res.*, 37: 444-449.

Gerrard JW. 1979. Allergies in breast-fed babies to ingredients in breast milk. *Ann. Allergy*, 42: 69-72.

Giashuddin MS, Kabir M. 2003. Breastfeeding duration in Bangladesh and factors associated with it. *Indian J. Commun. Med.*, 28: 34-38.

Giashuddin MS, Kabir M. 2004. Duration of breastfeeding in Bangladesh. *Indian J. Med. Res.*, 119: 267-272.

Gibson CE, Eglinton BA, Penttilä IA, Cummins AG. 1991. Phenotype and activation of milk-derived and peripheral blood lymphocytes from normal and coeliac subjects. *Immunol. Cell Biol.*, 69: 387-393.

Ginneken JV. 1977. Fetility regulation during human lactation. The choice of contraception during lactation. .*J Biosoc. Sci.(Suppl)*, 4: 102.

Goldman AS. 1993. The immune system of human milk: antimicrobial, anti-inflammatory and immunomodulating properties. *Pediatr. Infect. Dis. J.*, 12: 664 –671.

Goldman AS. 2000. Modulation of the gastrointestinal tract of infants by human milk. Interfaces and interactions. An evolutionary perspective in symposium: bioactivity in milk and bacterial interactions in the developing immature intestine. *J. Nutr.*, 130(suppl): 426S–31S.

Goldman AS, Smith CW. 1973. Host resistance factors in human milk. *J. Pediatr.*, 82: 1082-1090.

Goldman AS, Goldblum RM. 1995. Defense agents in human milk. In: Jensens RG, ed. *Handbook of milk composition.* New York: Academic Press,:727-745.

Goldman AS, Goldblum RM. 1996. Transfer of maternal leukocytes to the infant by human milk. In: Olding L (ed) *Reproductive Immunology/Current Topics in Microbiology and Immunology*. pp. 205-213. Springer-Verlag, Heidelberg.

Goldman AS, Garza C, Nichols BL, Goldblum RM. 1982. Immunologic factors in human milk during the first year of lactation. *J. Pediatr.* 100: 563-567.

Goldman AS, Chheda S, Garofalo R. 1997. Spectrum of immunomodulationg agents in human milk. *Int. J. Pediatr., Hematol. Oncol.*, 4: 491-497.

Goldsby RA, Kindt TJ, Osborne BA, Kuby J. 2003. *Immunology*. Freeman, 5th addition edition.

Gonzales RB. 1990. A large scale rooming in programme in a developing country: the Dr. Jose Fabella Memorial Hospital experience. *Int J Gynaecol Obstet; (Suppl.)*, 3(1): 31-34.

Gottschang SZ. 2007. Maternal bodies, breast-feeding, and consumer desire in urban China. *Med. Anthropol. Q.* 21(1): 64-80.

Grazioso CF, Buescher ES. 1996. Inhibition of neutrophil function by human milk. *Cell. Immunol.* 168: 125-132.

Gribble KD. 2006. Mental health, attachment and breastfeeding: Implications for adopted children and their mothers. *Int. Breastfeed. J.*, 1 (5). http://www.internationalbreastfeedingjournal.com/content /1/1/5.

Habicht JP, DaVanzo J, Butz WP. 1986. Does breastfeeding really save lives, or are apparent benefits due to biases? *Am. J. Epidemiol.* 123: 279–90.

Hanson L. 1998. Breast-feeding provides possible and long-lasting active immunity. *Ann. Allergy Asthma,* 81: 523-533.

Hanson L , 2000. The mother-offspring dyad and the immune system. *Rev. Acta Paediatr.*, 89: 252-258.

Hanson LÅ, Ahlstedt S, Carlsson B, Fallstrom SP. 1977. Secretory IgA antibodies against cow's milk protein in human milk and their possible effect in mixed feeding. *Int. Arch. Allergy Appl. Immunol.*, 54: 457-462.

Hanson LÅ, Carlsson B, Ahlstedt S, Svanborg C, Kajser B. 1975. Immune defence factors in human milk. *Mod. Prob. Pediatr.,* 15: 63-72.

Hanson LA, Carlsson B, Dahlgren U, Mellander L, Eden CS. 1979. The secretory IgA system in the neonatal period. *Ciba Found Symp*, 77: 187–204.

Hanson LA, Korotkova M, Haversen L, et al. 2002. Breast-feeding, a complex support system for the offspring. *Pediatr. Int.*, 44(4): 347–352.

Hanson LA, Korotkova M, Lundin S, Haversen L, Silfverdal SA, Mattsby-Baltzer I, Strandvik B, Telemo E. 2003. The transfer of immunity from mother to child. *Ann. NY Acad. Sci.*, 987: 199–206.

Hassan MQ, Hannan A, Kabir ARML, Barua PC, Rahman AKMF, Rahman A. 2006. Infant and young child feeding practices in urban areas of Bangladesh (abstract). In: Khan MSI, Rahim MA, Ahmed T (eds.) Combating malnutrition and intestinal diseases in children: are we doing enough?: abstracts book [of the] 8th Commonwealth Congress on Diarrhoea and Malnutrition, 6-8 February, ICDDR,B, Dhaka. Dhaka: International Centre for Diarrhoeal Disease Research, Bangladesh, 2006:20.

Hawdon JM, Ward Platt MP, Aynsley Green A. 1992. Patterns of metabolic adaptation for preterm and term infants in the first neonatal week. *Arch. Dis. Child.*, 67: 357– 365.

Hemming VG. 2001. Use of intravenous immunoglobulins for prophylaxis or treatment of infectious diseases. *Clin. Diagn. Lab. Immunol.* 8(5): 859–863.

Ho FCS, Wong RLC, Lawton JWM. 1979. Human colostral and breast milk cells. A light and electron microscopic study. *Acta Paediatr. Scand.*, 68: 389-396.

Ho PC, Lawton JWM. 1978. Human colostral cells: phagocytosis and killing of *E. coli* and *C. albicans*. *J. Pediatr.*, 93: 910-915.

Hop LT, Gross R, Giay T, Sastroamidjojo S, Schultink W, Lang NT. 2000. Premature complementary feeding is associated with poorer growth of Vietnamese children. *J Nutr.* 130: 2683–2690.

Horta BL, Bahl R, Martines JC, Victora CG. 2007. Evidence on the long-term effects of breastfeeding: systematic review and meta-analyses Geneva:WHO.

Høst A, Husby S, Osterballe O. 1988. A prospective study of cow's milk allergy in exclusively breast fed infants: incidence, pathogenetic role of early inadvertent exposure to cow's milk formula, and characterization of bovine milk protein in human milk. *Acta Paediatr. Scand.,* 77: 663-670.

Høst A, Husby S, Hansen LG, Osterballe O. 1990. Bovine beta-lactoglobulin in breast milk from atopic and nonatopic mothers. Relationship to maternal intake of homogenized and unhomogenized milk. *Clin. Exp. Allergy,* 20:383-387.

Howie P, Forsyth J, Ogston S, Clark A, du Florey CV. 1990. Protective effect of breast feeding against infection. *Br. Med. J.* 300: 11-16.

Howie PW. 2002. Protective effect of breastfeeding against infection in the first and second 6 months of life. *Adv. Expt. Med. Biol.*, 503: 141–147.

Huang JB, Yang WC, Hu CC, Yang AH, Lin CC. 2003. IgA deficiency with membranous glomerulonephritis: A case report and review. *J. Nephrol.*, 16: 154–158.

Huttly SR, Morris SS, Pisani V. 1997. Prevention of diarrhoea in young children in developing countries. *Bull WHO*, 75: 163-174.

Iacono G, Carroccio A, Montalto G, Cavataio F, Bragion E, Lorello D, Balsamo V, Notarbartolo A. 1991. Severe infantile colic and food intolerance: a long-term prospective study. *J. Pediatr. Gastroenterol. Nutr.* 12: 332-335.

IIPS (International Institute for Population Sciences). 2007. National family health survey (NFHS 3), 2005-06: India. V. I. Mumbai: International Institute for Population Sciences, 540p.

Islam S, Yadava KNS, Alam MA. 2006. Differentials and determinants of the duration of breastfeeding in Bangladesh: a multivariate analysis. *Proc. Pakistan Acad. Sci.*, 43: 1-14.

Jain AK, Bongaarts J. 1981. Breastfeeding patterns, correlates and fertility effects. *Stud. Fam. Plann.*,12: 79-99.

Jain N, Mathur NB, Sharma VK, Dwarkadas AM. 1991. Cellular composition including lymphocyte subsets in preterm and full term human colostrum and milk. *Acta. Paediatr. Scand.* 80: 395-399.

Jakobsson I, Lindberg T. 1978. Cow's milk as a cause of infantile colic in breast-fed infants. *Lancet*, 2: 437-439.

Järvinen K-M, Mäkinen-Kiljunen S, Suomalainen H. 1999. Cow's milk challenge via human milk evokes immune responses in suckling infants with cow's milk allergy. *J. Pediatr.* 135: 506-512.

Jensen RG. 1989. Lipids in human milk-composition and fatsoluble vitamins. In: Lebenthals E, ed. *Textbook of gastroenterology and nutrition in infancy*. New York: Raven Press, 157-208.

Jones G, Steketee RW, Black RE, Bhutta ZA, Morris SS. 2003. Bellagio Child Survival Study Group. How many child deaths can we prevent this year? *Lancet*, 362: 65-71.

Jumaan AO, Serdula MK, Williamson DF, Dibley MJ, Binkin NJ, Boring JJ. 1989. Feeding practices and growth in Yemeni children. *J. Trop. Pediatr.*, 35: 82-86.

Kaetzel CS. 2005. The polymeric immunoglobulin receptor: bridging innate and adaptive immune responses at mucosal surfaces. *Immunol. Rev.*, 206(1): 83–89.

Kalra A, Kalra K, Dayal RS. 1982. Breast feeding practices in different residential, economic and educational groups. *J. Indian Practitioner*, 19: 419-426.

Keeney SE, Schmalstieg FC, Palkowetz KH, Rudloff HE, Le B-M, Goldman AS. 1993. Activated neutrophils and neutrophil activators in human milk: increased expression of CD11b and decreased expression of L-selectin. *J. Leukocyte Biol.* 54: 97-104.

Killersreiter B, Grimmer I, Bührer C, Dudenhausen JW, Obladen M. 2001. Early cessation of breast milk feeding in very low birthweight infants. *Early Hum. Dev.*, 60: 193-205.

Kilshaw PJ, Cant AJ. 1984. The passage of maternal dietary proteins into human breast milk. *Int. Arch. Allergy Appl. Immunol.*, 75: 8-15.

Kimmons JE, Dewey KG, Haque E, Chakraborty J, Osendarp SJ, Brown KH. 2005. Low nutrient intakes among infants in rural Bangladesh are attributable to low intake and micronutrient density of complementary foods. *J. Nutr.* 135: 449-451.

Kohl S, Pickering LK, Cleary TG, Steinmetz KD, Loo LS. 1980. Human colostral cytotoxicity. II. Relative defects in colostral leukocyte cytotoxicity and inhibition of peripheral blood leukocyte cytotoxicity by colostrum. *J. Infect. Dis.* 142: 884-891.

Koldovsky O. 1994. Hormonally active peptides in human milk. *Acta Pediatr* (Suppl), 402: 89-93.

Kramer MS, Chalmers B, Hodnett ED, Sevkovskaya Z, Dzikovich I, Shapiro S, Collet JP, Vanilovich I, Mezen I, Ducruet T, Shishko G, Zubovich V, Mknuik D, Gluchanina E, Dombrovskiy V, Ustinovitch A, Kot T, Bogdanovich N, Ovchinikova L, Helsing E. 2001. Promotion of Breast feeding Intervention Trial (PROBIT). *JAMA,* 285: 413-420.

Kramer MS, Guo T, Platt RW, Sevkovskaya Z, Dzikovich I, Collet JP, Shapiro S, Kramer MS, Kakuma R. 2003. Optimal duration of exclusive breastfeeding. *Cochrane Database Syst Rev.*, 1: CD003517.

Kulkarni RN, Ajenaya S, Gujar R. 2004. Breast Feeding Practices in an urban community of Kalamboli, Navi Mumbai. *Indian J. Commu. Med.* 29(4): 179

Kunz C, Rudloff S. 1993. Biological functions of oligosaccharides in human milk. *Acta Paediatr.,* 82: 903-912.

Kunz C, Rudloff S. 1994. Biological functions of oligosaccharides in human milk. *Acta Paediatr.* S402: 903-912.

Kusiako T, Ronsmans C, Van der Paal L. 2000. Perinatal mortality attributable to complications of childbirth in Matlab, Bangladesh. *Bull WHO,* 78: 621–627.

Lande B, Andersen LF, Baerug A, Trygg KU, Lund-Larsen K, Veierød MB, Bjørneboe GE. 2003. Infant feeding practices and associated factors in the first six months of life: the Norwegian infant nutrition survey. *Acta Paediatr.*, 92: 152-161.

Lawn JE, Cousens S, Zupan J. 2005. Four million neonatal deaths: When? Where? Why? *Lancet*, 365: 891–900.

Lawrence RA. 1989. *Breastfeeding: A guide for the medical profession*, 3rd ed. St. Louis: The C.V. Mosby Company.

Lawrence RA, Lawrence RM. 2005. *Breastfeeding: A Guide for the Medical Profession.* 6th ed. St Louis, MO: Mosby.

Lawrence RM, Pane CA. 2007. Human breast milk: current concepts of immunology and infectious diseases. *Curr. Probl. Pediatr. Adolesc. Health Care,* 37(1): 7–36

Lazarus C, Edwards E. 1988. Radiopharmaceuticals. In: Bennett PN, ed. *Drugs and human lactation.* Amsterdam: Elsevier, 495-549.

Levine OS, Farley M, Harrison LH, Lefkowitz L, McGeer A, Schwartz B. 1999. Risk factor for invasive pneumococcal disease in children: A population-based case-control study in North America. *Pediatrics,* 103(3): e28.

LLLI (Le Leche League International). 2004. *The womanly art of breastfeeding (7th ed.).* Schaumburg, IL: Le Leche League International. 480pp.

Lonnerdal B. 1985. Biochemistry and physiological functions of human milk proteins. *Am. J. Clin. Nutr.,* 42: 1299-1317.

Lucassen PLBJ, Assendelft WJJ, Gubbels JW, van Eijk JTM, van Geldrop WJ, Neven A, Knuistingh A. 1998. Effectiveness of treatments for infantile colic: systematic review. *BMJ,* 316: 1563-1569.

Machtinger S, Moss R. 1986. Cow's milk allergy in breast-fed infants: The role of allergen and maternal secretory IgA antibody. *J. Allergy Clin. Immunol.* 77: 341-348.

Martines JC, Rea M, De Zoysa I. 1992. Breast feeding in the first six months: no need for extra fluids. *BMJ,* 304:1068-1069.

Mascart-Lemone F, Donnen P, Paluku B, Brasseur D, Van den Broeck J, Vaerman J-P, Hennart P, Duchateau J. 1991. Serum and breast milk antibodies to food antigens in African mothers and relation to their diet. In: Mestecky J, Blair, Ogra PL (eds) *Immunology of milk and the neonate*. Plenum Publishing Corporation, New York, pp. 201-206.

Masson PL, Heremans JF. 1966. Studies on lactoferrin, the iron-binding protein of secretions. In: Peeters H (ed) Protides of the Biological fluids. Elsevier North-Holland, New York. pp.115-124.

Masson PL, Heremans JF. 1971. Lactoferrin in milk from different species. *Comp. Biochem. Biophys.*, 39B: 119-129.

Mata L. 1986. Breastfeeding and host defense. *Front Gastrointest Res.*,13: 119-133.

Matthew AK, Amodu AD, Sani I, Solomon SD. 2009. Infant feeding practices and nutritional status of children in north western Nigeria. *Asian J. Clin. Nutr.*, 1: 12-22.

McClelland DDC, McDonald TT. 1976. Antibodies to cow's milk proteins in human colostrum. *Lancet,* ii: 1251-1252.

McPherson AV, Kitchen BJ. 1983. Reviews of the progress of dairy science: The bovine milk fat globule membrane- Its formation, composition, structure and behaviour in milk and dairy products. *J. Dairy. Res.* 50:107-133.

Michaelsen KF, Larsen PS, Thomsen BL, Samuelson G. 1994. The Copenhagen cohort study on infant nutrition and growth: duration of breastfeeding and influencing factor. *Acta Paediatr.*, 83: 565-571.

Michie CA, Tantscher E, Schall T, Rot A. 1998. Physiological secretion of chemokines in human breast milk. *Eur. Cytokine Netw.* 9:123-129.

Mihrshahi S, Oddy WH, Peat JK, Kabir I. 2008. Association between infant feeding patterns and diarrhoeal and respiratory illness: A cohort study in Chittagong, Bangladesh. *Int. Breastfeeding J.* 3:28 doi:10.1186/1746-4358-3-28.

Miler I, Borte M, Vondracek J. 1990. Phagocytosis of cadmium microcrystals by human milk macrophages in vitro. *Allergie und Immunologie*, 36: 157-162.

Morisky DE, Kar SB, Chaudary AS, Chen KR, Shaheen M. 2002. Breast feeding Practices in Pakistan. *Pakistan J. Nutr.* 1 :137-142.

Morrow AL, Rangel JM. 2004. Human milk protection against infectious diarrhea: implications for prevention and clinical care. *Semin. Ped. Infect Dis.*, 15(4): 221–228.

Murillo GJ, Goldman AS. 1970. The cells of human colostrum II: synthesis of IgA and beta-1-c. *Pediatr. Res.* 4: 71-75.

Murphey DK, Buescher ES. 1993. Human colostrum has antiinflammatory activity in a rat subcutaneous air pouch model of inflammation. *Pediatr. Res.* 34: 208-212.

Na HR, Daniels LC, Seelig LL Jr. 1997. Preliminary study of how alcohol consumptin during pregnancy affects immune components in breast milk and blood of postpartum women. *Alcohol Alcohol.* 32:581-589.

Newcomb PA, Storer BE, Longnecker MP, Mittendorf R, Greenberg ER, Clapp RW, Burke KP, Willett WC, MacMahon B. 1994. Lactation and a reduced risk of premenopausal breast cancer. *The New England J. Med.* 330(2): 81-86.

NHMRC (National Health and Medical Research Council). 2003. Dietary Guidelines for Children and Adolescents in Australia incorporating the Infant Feeding Guidelines for Health Workers Canberra, Australia: Commonwealth Department of Health and Ageing; 2003.

Noda K, Umeda M, Ono T. 1984. Transforming growth factor activity in human colostrum. *Gann.,* 75: 109-112.

Nolan L, Goel V. 1995. Sociodemographic factors related to breastfeeding in Ontario: results from the Ontario health survey. *Can. J. Public Health*, 86: 309-312.

O'Neal CM, Harriman GR, Conner ME. 1999. Protection of the Villus Epithelial Cells of the Small Intestine from Rotavirus Infection Does Not Require Immunoglobulin A. *J. Virol.*, 251(2): 343–360.

Oddy WH, Holt PG, Sly PD, Read AW, Landau LI, Stanley FJ, Kendall GE, Burton PR. 1999. Association between breast feeding and asthma in 6 year old children: findings of a prospective birth cohort study. *Br. Med. J.* 319: 815-819.

Oddy WH, Sly PD, de Klerk NH, Landau L, Kendall G, Holt P, Stanley F. 2003. Breast feeding and respiratory morbidity in infancy: a birth cohort study. *Arch. Dis. Child,* 88(3): 224 –228.

Ogra SS, Ogra PL. 1978. Immunologic aspects of human colostrum and milk. II. Characteristics of lymphocyte reactivity and distribution of E-rosette forming cells at different times after the onset of lactation. *J. Pediatr.* 92: 550-555.

Ogra SS, Ogra PL. 1979. Components of immunolgic reactivity in human colostrum and milk. In: Ogra PL and Dayton D (eds) *Immunology of breast milk.* pp. 185-195. Raven Press, New York.

Ogra PL, Losonsky GA, Fishaut M. 1983. Colostrum-derived immunity and maternal-neonatal interaction. *Ann. NY. Acad. Sci.,* 409: 82-95.

Okuda M, Miyashiro E, Koike M, Okuda S, Minami K, Yoshikawa N. 2001. Breast-feeding prevents *Helicobacter pylori* infection in early childhood. *Pediatr. InT.*, 43:714-715.

Okuda M, Miyashiro E, Booka M, Tsuji T, Nakazawa T. 2007. *Helicobacter pylori* colonization in the first 3 years of life in Japanese children. *Helicobacter*, 12(4): 324-327.

Oppe TE, Redstone D. 1968. Calcium and phosphorous levels in healthy newborn infants given various types of milk. *Lancet*, 1: 1045–1048.

Özkaragöz F, Rudloff HB, Rajaraman S, Mushtaha AA, Schmalstieg FC, Goldman AS. 1988. The motility of human milk macrophages in gollagen gels. *Pediatr. Res.*, 23: 4494-4452.

Pabst HF. 1997. Immunomodulation by breast-feeding. *Pediatr. Infact. Dis. J.*, 16: 991-995.

PAHO (Pan American Health Organization). 2001. Guiding principles for complementary feeding of the breastfed child. Washington, DC: Pan American Health Organization, World Health Organization, Division of Health Promotion and Protection, Food and Nutrition Program, 2001.

PAHO (Pan American Health Organization). 2003. Guiding principles for complementary feeding of the breastfed child. Washington, DC: Pan American Health Organization, 37p.

Pardo-Crespo R, Perez-Iglesias R, Llorca J, et al. 2004. Breast-feeding and risk of hospitalization for all causes and fever of unknown origin. *Eur. J. Public Health*, 14(3): 230 –234.

Paricio Talayero JM, Lizan-Garcia M, Otero Puime A, Benlloch Muncharaz MJ, Beseler Soto B, Sánchez-Palomares M, Santos Serrano L, Rivera LL 2006. Full breastfeeding and hospitalization as a result of infections in the first year of life. *Pediatrics.* 118(1). Available at: www.pediatrics.org/cgi/ content/full/118/1/e92.

Parmely MJ, Beer AE, Billingham RE. 1976. *In vitro* studies of the T-lymphocyte population of human milk. *J. Exp. Med.*, 144: 358-370.

Patton S, Keenan TW. 1975. The milk fat globule membrane. *Biochem. Biophys. Acta.* 415: 273-309.

Pearce MS, Thomas JE, Campbell DI, Parker L. 2005. Does increased duration of exclusive breastfeeding protect against *Helicobacter pylori* infection? The Newcastle thousand families cohort study at age 49–51 years. *J. Pediatr. Gastroenterol. Nutr.* 41(5): 617-620.

Perkkiö M, Savilahti E. 1980. Time of appearance of immunoglobulin-containing cells in the mucosa of the neonatal intestine. *Pediatr. Res.*, 14: 953-955.

Pettigrew MM, Khodaee M, Gillespie B, Schwartz K, Bobo JK, Foxman B. 2003. Duration of breastfeeding, daycare, and physician visits among infants 6 months and younger. *Ann. Epidemiol.*, 13(6): 431– 435.

Pettitt DJ, Forman MR, Hanson RL, Knowler WC, Bennett PH. 1997. Breastfeeding and the incidence of non-insulin-dependent diabetes mellitus in Pima Indians. *Lancet*, 350(9072): 166–168.

Pisacane A, Graziano L, Mazzarella G, Scarpellino B, Zona G. 1992. Breast-feeding and urinary tract infection. *J. Pediatr.* 120(1): 87-89.

Pitt J. 1979. The milk mononuclear phagocyte. *Pediatrics*, 64 (Suppl):745-749.

Popkin BM, Lasky T, Litvin J, Spicer D, Yamamoto M. 1986. *The Infant Feeding Triad: Infant, Mother, and Household.* New York, NY: Gordon and Breach.

Popkin BM, Adair L, Akin JS, Black R, Briscoe J, Flieger W. 1990. Breast-feeding and Diarrheal Morbidity. *Pediatrics,* 86: 874-882.

Prentice A, Prentice AM, Lamb WH. 1985. Mastitis in rural Gambian mothers and the protection of the breast by milk antimicrobial factors. *Trans. R. Soc. Trop. Med. Hyg.*, 79: 90-95.

Prentice A, Ewing G. Roberts SB, Lucas A, MacCarthy A, Jarjou LMA, Whitehead RG. 1987. The nutritional role of breastmilk IgA and lactoferrin. *Acta Paediatr. Scand.*, 76: 592-598.

Qiu L, Zhao Y, Binns CW, Lee AH, Xie X. 2009. Initiation of breastfeeding and prevalence of exclusive breastfeeding at hospital discharge in urban, suburban and rural areas of Zhejiang China. *Int. Breastfeed. J.* 4:1 doi:10.1186/1746-4358-4-1. http://www.internationalbreastfeedingjournal.com/content/4/1/1.

Qiu L, Binns CW, Zhao Y, Lee AH, Xie X. 2010. Breastfeeding practice in Zhejiang Province, PR China, in the context of melamine-contaminated formula milk. *J. Health Popul. Nutr.*, 28(2):189-198

Quigley MA, Cumberland P, Cowden JM, Rodrigues LC. 2006. How protective is breast feeding against diarrhoeal disease in infants in 1990s England? A case-control study. *Arch. Dis. Child,* 91(3):245–250.

Quigley MA, Kelly YJ, Sacker A. 2007. Breastfeeding and hospitalization for diarrheal and respiratory infection in the United Kingdom Millennium Cohort Study. *Pediatrics*. 119(4). Available at: www.pediatrics.org/ cgi/content/full/119/4/e837.

Riordan J. 2005. Anatomy and physiology of lactation. In: Riordan J, ed. *Breastfeeding and human lactation.* 3rd ed. pp.67-92, Jones and Bartlett, Boston, MA.

Riordan J, Auerbach KG. 1999. Breastfeeding and human lactation (2nd ed.). Sudbury, MA: Jones and Bartlett Publishers.

Robinson G, Volovitz B, Passwell JH. 1991. Identification of a secretory IgA receptor on breast-milk macrophages: evidence for specific activation via these receptors. *Pediatr. Res.*, 29: 429-434.

Rodriguez C, Subiza JL, Mateos P, Casado de Frias E, Moro M, De la Concha EG. 1989. Comparative functional study of colostral macrophages from mothers delivering preterm and at term. *Acta. Paediatr. Scand.* 78: 337-341.

Rollins NC, Filteau SM, Coutsoudis A, Tomkins AM. 2001. Feeding mode, intestinal permeability, and neopterin excretion: a longitudinal study in infants of HIV-infected South African women. *J. Acquir. Immune. Defic. Syndr.*, 28:132–139.

Roux ME, McWilliams M, Phillips-Quagliata JM, Weisz-Carrington P, Lamm ME. 1977. Origin of IgA-secreting plasma cells in the mammary gland. *J. Exp. Med.* 146: 1311-1322.

Rudloff HE, Schmalstieg FC, Mushtaha AA, Palkowetz K, Liu SK, Goldman AS. 1992. Tumor necrosis factor-alpha in human milk. *Pediatr. Res.*, 31: 29-33.

Saarinen UM, Kajosarri M. 1995. Breastfeeding as prophylaxis against atopic disease: Prospective follow-up study until 17 years old. *Lancet*, 346: 1065–1069

Sachdev HP, Krishna J, Puri RK, Satyanarayana L, Kumar S. 1991. Water supplementation in exclusively breastfed infants during summer in the tropics. *Lancet*, 337: 929-933.

Saha KK, Frongillo EA, Alam DS, Ariffen SE, Persson LA, Rasmussen KM. 2008. Appropriate infant feeding practices result in better growth of infants and young children in rural Bangladesh. *Am. J. Clin. Nutr.*, 87: 1852-1859.

Saito S, Maruyama M, Kato Y, Moriyama I, Ichijo M. 1991. Detection of IL-6 in human milk and its involvement in production. *J. Reprod. Immunol.* 20: 267-276.

Saji F, Samejima Y, Kamiura S, and Koyama M. 1999. Dynamics of immunoglobulins at the feto–maternal interface. *Rev. Reprod.* 4: 81–89.

Savilahti E, Tainio VM, Salmenperä L, Arjonmaa P, Kallio M, Perheentupa J, Siimes MA. 1991. Low colostral IgA associated with cow's milk allergy. *Acta Paediatr. Scand.*, 80: 1207-1213.

Scholtens S, Wijga AH, Brunekreef B, Kerkhof M, Hoekstra MO, Gerritsen J, Aalberse R, de Jongste JC, Smit H A. 2010. Breast feeding, parental allergy and asthma in children followed for 8 years. *The PIAMA birth* cohort study. *Thorax*, 64: 604–609.

Schusdziarra V. 1992. Physiological role of beta-casomorphins. In: Picciano MF, Lonnerdals B. eds. *Mechanisms regulating lactation and infant nutrient utilization.* New York: Wiley Liss, 337-348.

Scott JA, Binns CW. 1999. Factors associated with the initiation and duration of breastfeeding: a review of the literature. *Breastfeed. Rev.* 7(1): 5-16.

Scott JA, Binns CW, Graham KI, Oddy WH. 2006. Temporal changes in the determinants of breastfeeding initiation. *Birth*, 33(1):37-45.

Shu XO, Linet MS, Steinbuch M, Wen WQ, Buckley JD, Neglia JP, Potter JD, Reaman GH, Robinson LL . 1999. Breastfeeding and the risk of childhood acute leukemia. *J. Nat. Cancer Inst.* 91(20): 1765-1772.

Sinahababu A, Mukhopadhyay DK, Panja TK, Saren AB, Mandal NK, Biswas AB. 2010. Infant- and Young Child-feeding Practices in Bankura District, West Bengal, India. *J. Health Popul. Nutr.*, 28(3): 294-299.

Singh PMP, Bhalwar R. 2007. Breast feeding practices among families of armed forces personnel in a large cantonment. *MJAFI*, 63: 134-136.

Skansén-Saphir U, Lindfors A, Andersson U. 1993. Cytokine production in mononuclear cells of human milk studied at the single-cell level. *Pediatr. Res.*, 32: 213-216.

Slade HB, Schwartz SA. 1987. Mucosal immunity: The immunology of breast milk. *J. Allergy Clin. Immunol.* 80: 346-356.

Smith CW, Goldman AS, Yates RD. 1971. Interactions of lymphocytes and macrophages from human colostrum. *Exp. Cell Res.*, 69: 409-415.

Smith CW, Goldman AS. 1968. The cells of human colostrum. I. In vitro studies of morphology and functions. *Pediatr. Res.*, 2: 103-109.

Söder O. 1987. Isolation of interleukin-1 from human milk. *Int. Archs. Allergy Appl. Immunol.*, 83: 19-23.

Sorva R, Mäkinen-Kiljunen S. 1994. Beta-lactoglobulin secretion in human milk varies widely after cow's milk ingestion in mothers of infants with cow's milk allergy. *J. Allergy Clin. Immunol.*, 93: 787-792.

Speer CP, Schatz R, Gahr M. 1985. Function of breast milk macrophages. *Monatssch Kinderheilkd*, 133: 913-917.

Speer CP, Gahn M, Pabst MJ. 1986. Phagocytosis-associated oxidative metabolism in human milk macrophages. *Acta Paediatr. Scand.*, 75: 444-451.

Srivastava MD, Srivastava A, Brouhard B, Saneto R, Groh-Wargo S, Kubit J. 1996. Cytokines in human milk. *Res. Commun. Mol. Patho. Pharmacol.*, 93: 263-287.

Stephens S, Dolby JM, Mantreuil J, Spik G. 1980. Differences in inhibition of the growth of commensal and enteropathogenic strains Escherichia coli by lactoferin and secretory immunoglobulin A isolated from human milk. *Immunology*, 41: 597-603.

Stiehm RE. 1992. HIV transmission by breastfeeding: report of ten cases and review. In: Picciano MF, Lonnerdals B. eds. *Mechanisms regulating lactation and infant nutrient utilization.* New York: Wiley-Liss, 179-188.

Stuart CA, Twiselton R, Nicholas MK, Hide DW. 1984. Passage of cow's milk protein in breast milk. *Clin. Allergy,* 14: 533-535.

Thorpe LW, Rudloff HE, Powell LC, Goldman AS. 1986. Decreased response of human milk leukocytes to chemoattractant peptides. *Pediatr. Res.,* 20: 373-377.

Tsuda H, Takeshige K, Shibata Y, Minakami S. 1984. Oxygen metabolism of human colostral macrophages. J. Biochem., 95: 1237-1245.

UNCF (United Nations Children's Fund/World Health Organization) 2005. Baby friendly hospital initiative. Geneva, Switzerland: United Nations Children's Fund/World Health Organization; 2005. Available at: www.unicef.org/nutrition/index_24806. html. Accessed January 27, 2005

USBC (United States Breastfeeding Committee). 2002. Economic benefits of breastfeeding [issue paper]. Raleigh, NC: United States Breastfeeding Committee. http://www.usbreastfeeding.org/Issue-Papers/Economics.pdf

USDA (US Department of Agriculture). 2001. The Economic benefits of breastfeeding: A review and analysis (Food assistance and nutrition research report no. 13). Washington, DC: Jon Weimer.

USDA (US Department of Agriculture). 2003. Summary: Fiscal year 2001 WIC food package costs. Retrieved June 17, 2003, from http://www.fns.usda.gov/oane/ MENU/WICFoodCosts/FY01Report.pdf

USDHHS (US Department of Health and Human Services). 2000. HHS blueprint for action on breastfeeding. Washington, DC: U.S. Department of Health and Human Services, Office on Women's Health.

van Odijk J, Kull I, Borres MP, Brandtzaeg P, Edberg U, Hanson LÅ, Høst A, Kuitunen M, Olsen SF, Skerfving S, Sundell J, Wille S. 2003. Breastfeeding and allergic disease: a multidisciplinary review of the literature (1966–2001) on the mode of early feeding in infancy and its impact on later atopic manifestations. *Allergy*, 58: 833–843.

Vassella CC, Hjälle L, Björkstén B. 1992. Basophils and eosinophils in human milk in relation to maternal allergy. *Pediatr. Allergy Immunol.*, 3: 28-32.

Victora CG, Vaughan JP, Lombarda C, Fuchs SMC, Gigante LP, Smith PG, Nobre LC, Teixeira AMB, Moreira LB, Barros FC. 1987. Evidence for protection by breast-feeding against infant deaths from infectious diseases in Brazil. *Lancet,* 330(8554): 319-322.

Victoria J, Jimenez MS, Hainsworth MD. 2005. Improving breast feeding practices on a broad scale at community level: success stories from Africa and Latin America. *J. Hum. Lact.*, 21: 345-354.

Villegas R, Gao YT, Yang G, Li HL, Elasy T, Zheng W, Shu XO. 2008. Duration of breast-feeding and the incidence of type 2 diabetes mellitus in the Shanghai Women's Health Study. *Diabetologia,* 51(2):258-266.

von Kries R, Koletzko B, Sauerwald T, von Mutius E, Barnert D, Grunert V, von Voss H. 1999. Breast feeding and obesity: cross sectional study. *Br. Med. J.* 319: 147-150.

Wagner CL, Anderson DM, Pittard WB III. 1996. Special properties of human milk. *Clin. Pediatr.,* 35: 283-293.

Waksman BH. 1979. Summary. In: Ogra PL, Dayton D (eds) Immunology of breast milk. Raven Press pp.257-272.

Walker WA. 1979. Antigen penetration across the immature gut: effect of immunologic and maturational factors in colostrum. In: P.L. Ogra, Dayton D (eds) Immunology of breast milk. New York: Raven Press, pp. 227-234.

Walker WA, Wu M, Isselbach KJ, Bloch KJ. 1975. Intestinal uptake of macromolecules: III: Studies on the mechanism by which immunization interferes with antigen uptake. *J. Immunol.,* 115: 854-861.

Wamani H, Astrøm AN, Peterson S, Tylleskär T, Tumwine JK. 2005. Infant and young child feeding in western Uganda: knowledge, practices and socioeconomic correlates. *J. Trop. Pediatr.*,51: 356-361.

Weaver EA, Rudloff HE, Goldblum RM, Davis CP, Goldman AS. 1984. Secretion of immunoglobulin A by surface membrane stimuli. *J. Immunol.,* 132: 684-689.

WHO (World Health Organization) 1991. Indicators for Assessing Breastfeeding Practice [reprinted report of an informal meeting 11–12 June, 1991]. Geneva, Switzerland.

WHO (World Health Organization). 1998. Complementary feeding of young children in developing countries: a review of current scientific knowledge. Geneva: World Health Organization, 237p. (WHO/NUT/98.1).

WHO (World Health Organization). 2000. Effect of breastfeeding on infant and child mortality due to infectious diseases in less developed countries: a pooled analysis. Collaborative Study Team on the Role of Breastfeeding on the Prevention of Infant Mortality. *Lancet,* 355(9202): 451–455.

WHO (World Health Organization). 2001. The Optimal Duration of Exclusive Breastfeeding: Report of an Expert Consultation. Geneva, Switzerland: Department of Nutrition for Health and Development, World Health Organization.

WHO (World Health Organization). 2002a. Report of the expert consultation on the optimal duration of exclusive breastfeeding 2002a [http://www.who.int/ child adolescent-health/publications /NUTRITION/WHO_FCH_CAH_01.23.htm]. Geneva, Switzerland

WHO (World Health Organization). 2002b. Global Strategy on Infant and Young Child Feeding. 55th World Health Assembly [http:// webitpreview.who.int/entity/ nutrition/publications/ gs_infant_ feeding_text_eng.pdf]. Geneva, Switzerland.

WHO (World Health Organization). 2003. Global strategy for infant and young child feeding. Geneva: World Health Organization, 41p.

Wilde CJ, Prentice A, Peaker M. 1995. Breastfeeding: matching supply with demand in human lactation. *Proc. Nutr. Soc.*, 54: 401-406.

Williams PE, Leen CL, Heppleston AD, Yap PL. 1999. IgG replacement therapy for primary hypogammaglobulinaemia during pregnancy: report of 9 pregnancies in 4 patients. *Blut.*, 60(3): 198–201.

Winberg J, Wessner G. 1971. Does breast milk protect against septicemia in the newborns? *Lancet*, i: 1091-1094.

Wirt DP, Adkins LT, Palkowetz KH, Schmalstieg FC, Goldman AS. 1992. Activated and memory T lymphocytes in human milk. *Cytometry*, 13: 282-290.

Xanthou M, Bines J, Walker WA. 1995. Human milk and intestinal host defence in newborns: an update. *Adv. Pediatr.* 42: 171-208.

Xanthou M. 1997. Human milk cells. Invited commentary. *Acta Paediatr.*, 86: 1288-1290.

Xu F, Binns C, Yu P, Bai Y. 2007. Determinants of breastfeeding initiation in Xinjiang, PR China, 2003–2004. *Acta Paediatr.* 96(2): 257-260.

Yadava KNS, Jain SK. 1998. Post-partum amenorrhoea in rural Eastern Utter Pradesh India. *J. Biosocial Sci.*, 30: 227-43.

Zarba A, Groopman JD. 1992. Biomarkers of aflatoxin exposure in humans: aflatoxin M1 in milk. In: Picciano MF, Lonnerdals B. eds. *Mechanisms regulating lactation and infant nutrient utilization.* New York: Wiley-Liss, 451-455.

Zeiger RS, Heller S, Mellon MH, Forsythe AB, O'Connor RD, Hamburger RN, Schatz M. 1989. Effect of combined maternal and infant food-allergen avoidance on development of atopy in early infancy: A randomized study. *J. Allergy Clin. Immunol.*, 84: 72-89.

Zopf D, Roth S. 1996. Oligosaccharides anti-infective agents. *Lancet*, 347:1017-1021.

Rajshahi University Library
Documentation Section
Document No... D-3485
Date... 27/10/13