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Inside

Prevalence of iron-deficiency anaemia among young children in rural Bangladesh

Anaemia during early childhood affects immunity, growth and optimum brain development. The Child Development Unit at ICDDR,B conducted a longitudinal intervention study on iron deficient anaemic children identified from 30 villages of Monohardi sub-district, Bangladesh. Out of 1,237 children under 2 years of age, 60% were anaemic, but only half of the anaemic children were iron deficient anaemic. The iron deficient children responded well to short term iron intervention, but understanding the other causes of anaemia is important to deliver effective therapy to prevent this early childhood problem.

Anaemia is one of the major public health problems in young children, particularly in developing countries, where malnutrition and infections are prevalent. Early childhood anaemia adversely affects growth, immunity and cognitive development (1). The causes of anaemia are multi-factorial and interlinked. However, in developing countries most anaemia is thought to be nutrition related.

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Bangladesh is a country where 22% of all newborns are low birth weight, 43% of <5 children are stunted (height-for-age <-2 z scores) and 17% of them are wasted (weight-for-height <-2 z scores) (2). The majority of these children are anaemic due to coexisting micronutrient deficiencies. According to a national rural survey report, the prevalence of anaemia is as high as 78% among infants aged 6-11 months and is 64% among young children aged 12- 23 months (3). Another review of anaemia in Bangladesh also found that its prevalence ranged from 49 to 81% with a suggestion that rural children have higher levels of anaemia than urban children (4). A representative survey of 14 rural districts of Bangladesh (7,764 people of 0-60 years of age) identified an association of anaemia with large families, poor sanitation, low socioeconomic status, poor nutritional status and higher parasitic infection (5). Anaemic children are therefore exposed to several risk factors associated with poverty, and that in turn detrimentally affects their development.

Iron deficiency anaemia is considered to be the main type of anaemia among young children in Bangladesh. The main aetiologies of iron deficiency anaemia in these children are hypothesized to be due to poor dietary iron intake, especially during this period of rapid growth (4). A national survey in 1995-1996 reported that the diets of children aged 1-3 years had marked deficits in energy, protein, iron and other nutrients (6). Approximately 60% of their dietary iron source came from cereals in this survey. Usually, Bangladeshi mothers start supplementary feeding around 3 months of age, with rice-based foods that lack both iron and protein, which likely contributes to iron deficiency during early infancy and childhood. Low birth weight is another important contributor to iron deficiency anaemia as these babies are usually born with low iron stores.

Although iron deficiency anaemia is considered to be the major cause of anaemia in young children, interestingly one study in Bangladesh reported that out of 48% anaemic children 2-6 years old, only 18% had iron deficiency anaemia considering their haemoglobin level <11.0 g/dL and serum ferritin level <12 µg/L. In Bangladesh, most anaemia studies in children are only based on blood haemoglobin level. Only a few studies have specifically focused on anaemia due to iron deficiency. Therefore, there is a need to determine the actual prevalence of iron deficiency anaemia in young children and its response to iron treatment.

The current document reports the prevalence of iron deficiency anaemia among young children of rural Bangladesh. For this purpose 30 villages of Monohardi sub-district were randomly selected. All the <2 children in these villages were identified from the birth registries of the nutrition centres of the National Nutrition Programme. We invited all the mothers of apparently healthy children (without any chronic illnesses or congenital anomalies) to join the anaemia survey. Mothers of 1,237 children gave

written consent and participated in the survey-based screening. Children were assessed for haemoglobin, ferritin, C-reactive protein, and serum transferrin receptors using simple sandwich enzyme linked immunosorbant assay (7) through heel-pricked blood samples.

Using a criteria of haemoglobin <11 g/dL and serum transferrin receptors >5 mg/L (8,9), we classified 225 children (18%) as mildly to moderately anaemic from iron deficiency. Severely anaemic children with haemoglobin level <8 g/dL were excluded from the study and sent to local hospital for proper management. The study team compared the 225 iron deficient anaemic children with 209 non-anaemic children matched for age, sex and village. A total of 434 children were recruited into the longitudinal component of the study and assessed for further anthropometric and socio-demographic measures. Diagnosed iron deficient anaemic children received 30 mg ferrous sulphate syrup daily for 6 months. All the enrolled children were reassessed for their iron status 9 months later.

Of all 1,237 survey-children, the mean age was 13.0 months and the standard deviation was 5.1 months. The prevalence of anaemia was 60% (n=755) when a single parameter haemoglobin level <11.0 g/dL was used. However considering other parameters of low iron status such as ferritin (<12 µg/L) and C-reactive protein (<5 mg) along with low haemoglobin level, only 25% children had iron deficiency anaemia. When low haemoglobin and high serum transferrin receptor (>5 mg/L) was considered as an indicator of iron deficiency, only 30% of anaemic children had iron deficiency anaemia.

Out of 225 mild to moderately iron deficient anaemic children the distribution of anaemia at baseline was similar across different age groups (Figure 1). Children with iron deficiency anaemia were more likely to be stunted compared with non-anaemic children; a higher percentage of anaemic children were severely stunted children. Mothers of anaemic children scored low in an intelligence test and provided less care to their children compared to mothers of non-anaemic children. They were also from poorer economic conditions, although the parental education and father's occupation were similar in the two groups (Table 1). All the iron deficient anaemic children responded adequately to 6 months of iron treatment (Table 2).

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Comments

As in previous studies (10) we also found the prevalence of anaemia in these communities in rural Bangladesh to be as high as 60%, but only half of the anaemic children were iron deficient anaemic based on different diagnostic criteria.

Figure 1: Mean haemoglobin and serum transferrin receptor levels in different age groups of children

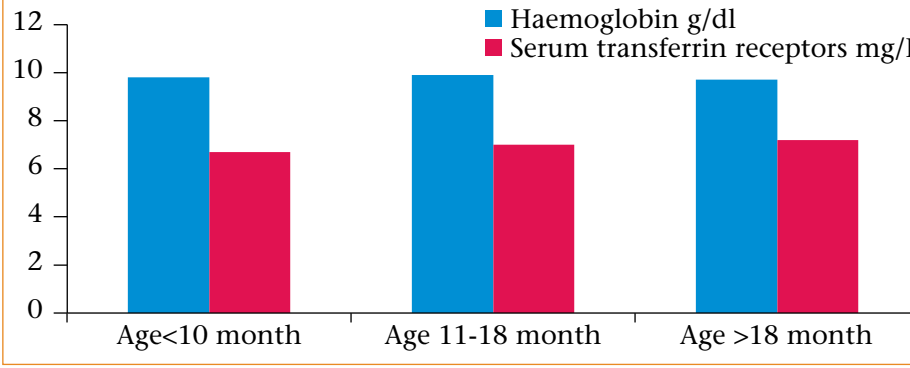

Table 1: Socio-demographic status and children's biological characteristics in iron deficient anaemic and non-anaemic children at baseline

Variable	Anaemic Mean ±SD or % (n=225)	Non-anaemic Mean ±SD or % (n=209)	P Value
Family expenditure (Tk/month)	4,781±2,485	4,209±1,218	0.005
Father occupation (Low skilled job)	66%	60%	0.10
Father's education (<grade 5)	63%	60%	0.55
Mother's education(<grade 5)	52%	46%	0.12
Mother's IQ (intelligence)	20±10	25±11	<0.001
Amount of stimulation and care available at home	5.6±3	6.6±3	<0.038
Mean age (months)	15.3±4.4	15.8±5.3	0.27
Severe wasting (<-3.0 z score)	8.9%	8.6%	0.53
Severe stunting (<-0.3 z score)	22.7%	14.4%	0.02

Table 2 Iron status of iron deficient anaemic children at baseline and 9 months later, following 6 months intervention with iron supplement after diagnosis

Iron status	Baseline measures Mean ±SD or % or Median (25^{th} -75^{th} percentile)	After 9 months Mean ±SD or % or Median (25^{th} -75^{th} percentile)
Haemoglobin g/dL	9.8±0.8	12.5±1
% of haemoglobin <11.5 g/dL	100%	4.8%
Serum tranferrin receptor mg/L	7.1±2	4.4±1.3
Serum ferritin µg/L	16 (9, 29)	43 (23, 56)
C-reactive protein mg/L	1.5 (0.5,5)	0.6 (0.1,1.6)

For early intervention to prevent anaemia, it is imperative to determine the other causes of anaemia in this age group because it is well documented that anaemia at younger age affects optimum brain development (1). Other critical micronutrients for red cell synthesis include vitamin B12, and folic acid, and trace amounts of vitamin C, riboflavin, and copper. Vitamin A deficiency is also common in this population and is reported to contribute to anaemia (10). In a national rural survey anaemia was associated with Vitamin A deficiency in mothers. In Bangladesh there is a government policy of supplementing <5 children with vitamin A every 6 months. Coverage is estimated to be 97% (2), however, the bio-availability of these supplements is uncertain due to intestinal malabsorption or helminthes infestation. Recently, it has been reported that environmental toxins, e.g. arsenic, may also contribute to anaemia (11). This requires further investigation. In addition, the prevalence of disorders that carries abnormal haemoglobin, especially haemoglobin-E is frequently identified in Southeast Asia. A report by the World Health Organization estimated that there are around 4.8 million carriers of haemoglobin E in Bangladesh (12).

To protect the next Bangladeshi generation, it is important that iron deficiency anaemia be identified and aggressively treated with available short-term, low-cost supplements. At the same time necessary interventions need to be initiated for poverty alleviation and to improve mothers' knowledge regarding childrearing practices. Further research to identify other causes of anaemia and cost-effective interventions to prevent them are an important public health priority.

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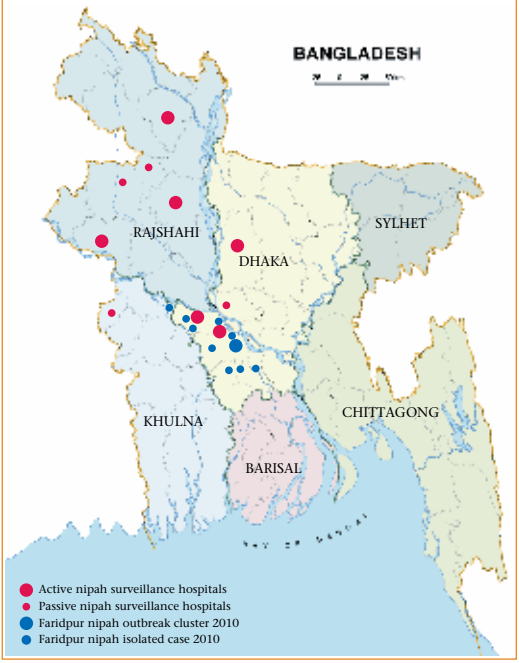
Nipah outbreak in Faridpur District, Bangladesh, 2010

Physicians at Faridpur Medical College Hospital recognized a cluster of encephalitis cases within Faridpur District in January 2010. A subsequent field investigation suggested that the initial cases acquired Nipah through drinking raw date palm sap and subsequent transmission occurred through person-to-person contact. In March 2010, one of the hospital physicians who cared for two Nipah patients died from Nipah encephalitis. To prevent further outbreaks of Nipah virus, people in the 'Nipah belt' should be warned about the risk of drinking fresh date palm sap. Practical steps to interrupt contamination of date palm sap should be introduced, and protective measures to reduce patient to caregiver transmission undertaken.

Eight outbreaks of Nipah encephalitis have been recognized in Bangladesh from 2001 to 2008 (1,2). The Institute for Epidemiology Disease Control and Research (IEDCR) of the Government of Bangladesh, in collaboration with ICDDR,B, established 10 Nipah surveillance sites beginning in February 2006 in hospitals located in the region where prior Nipah outbreaks were recognized (Figure 1). The objective of the surveillance is to identify outbreaks of encephalitis and to characterize the conditions and risk

factors for transmission of the responsible viruses. Each of the six active site submits a monthly report to IEDCR and ICDDR,B on the number of meningo-encephalitic cases, while the four passive sites only report if they suspect a high number of encephalitis cases.

Figure 1: Surveillance hospital locations, clusters and isolated cases, Faridpur, Bangladesh 2010



Senior physicians and paediatricians of corresponding active surveillance hospitals are the focal persons and each hospital has a designated Nipah surveillance study physician. The active surveillance sites maintain a registry throughout the year of patients who fulfil the enrollment criteria based on encephalitis and/or pulmonary presentation (3). The encephalitic presentation includes fever with evidence of acute brain pathology, i.e, altered mental status, new onset of seizures, or a new neurological deficit. Respiratory presentation includes acute onset <7 days of symptoms with fever, severe shortness of breath and chest radiograph with diffuse infiltrates.

To cost effectively focus scarce public health surveillance resources, active surveillance focuses on identifying clusters of patients with meningo-encephalitis. We define a cluster as two or more persons living within a 30 minute walk of each other who develop similar symptoms within 21 days of each other. Surveillance physicians keep a detailed address of each case in the registry that they match cases with using distance and time. They also ask individual patients or attendants about recent deaths in their community or any other cases with similar symptoms not yet reported. When active surveillance identifies a cluster they report it to IEDCR and ICDDR,B and we promptly conduct an epidemiological investigation in the community to search for more cases of meningo-encephalitis syndrome and to collect additional samples to try to identify the aetiology of the disease.

To identify all hospital reported cases that occur during the usual Nipah season from January to March, we collected blood and cerebrospinal fluid

samples at three active surveillance sites, Faridpur, Rajshahi and Rangpur Medical College Hospitals, which admit the largest number of acute meningo-encephalitis cases. We tested these samples for anti-Nipah IgM by ELISA at IEDCR and ICDDR,B virology laboratory in Dhaka to confirm both clusters and isolated cases.

In December 2009, an experienced surveillance officer from Rajbari Sadar Hospital reported a suspected Nipah encephalitis case and referred the patient to Faridpur Medical College Hospital, where a blood sample was collected that was found positive for IgM antibody against Nipah virus. We then initiated sample collection from each meningo-encephalitis case from Faridpur surveillance hospital. In total, from December 2009 to March 2010, we enrolled 331 cases from the six active surveillance sites. Among them 293 (89%) cases were from Faridpur, Rajshahi and Rangpur Medical College Hospitals. Fifty seven percent of cases (167) were under 15 years of age (Table 1). No Nipah cases were confirmed by laboratory testing from Rangpur and Rajshahi Medical College Hospitals. We found twelve anti-Nipah IgM positive cases at Faridpur Medical College Hospital: two of them were part of a cluster, and the remainder were isolated cases from a widespread geographic area in and around Faridpur district (Figure 1).

Table 1: Age and sex distribution of enrolled patients in three medical college hospitals with acute meningoencephalitis syndrome from December 2009 to March 2010

	Medical college hospitals			Total n (%)
	Faridpur n (%)	Rajshahi n (%)	Rangpur n (%)	
Age group				
<5 years	16 (21)	48 (37)	31 (35)	95 (32)
6 to 15	29 (39)	18 (14)	25 (28)	72 (25)
16 to 25	11 (15)	14 (11)	11 (12)	36 (12)
26 to 40	6 (8)	20 (16)	15 (17)	41 (14)
>40 years	13 (17)	29 (22)	7 (8)	49 (17)
Sex				
Male	39 (52)	81 (63)	49 (55)	169 (58)

During this same period, we investigated seven meningo-encephalitis clusters: one from Rangpur; and three each from Rajshahi and Faridpur. In Rangpur and in Rajshahi we could not find any exposure history, or epidemiological links between cases of these clusters and each case was anti-Nipah IgM negative.

In Faridpur, the first cluster consisted of two cases, both of which had fever, with features of brain pathology, but both of whom tested anti-Nipah IgM negative. The second Faridpur cluster involved two cases who also had

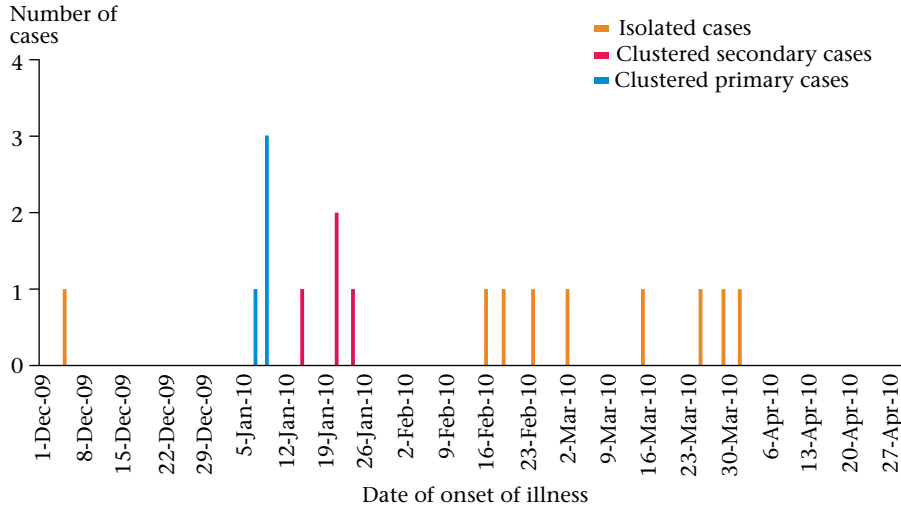
fever with features of brain pathology, but with no epidemiological link between them. One Nipah antibody negative case survived, but a Nipah antibody positive case, who collected date palm sap and had a history of drinking raw date palm sap, died. The third Faridpur cluster was reported to IEDCR and ICDDR,B on 14 January 2010, and consisted of eight cases. We defined probable cases as people with fever and altered mental status or new onset of seizures or respiratory distress. Confirmed Nipah cases were serum anti-Nipah IgM antibody positive. The index case was a 45 year old male from Bhanga sub-district who died after being admitted to the hospital. Another 40 year old female case, who was a neighbour of the index case, presented with vomiting, headache, convulsion, altered mental status, and loss of consciousness, and died at home. Both of them were probable cases. Two other cases from the 40-year old female's family, 10 and 11 year old girls, who were admitted in hospital with a clinical diagnosis of encephalitis, died. One girl was found anti-Nipah IgM positive. The other girl was a probable Nipah case.

After one week, the study physician from Fardipur Medical College Hospital reported another upsurge of cases from the same sub-district. The investigation team identified two more probable and two more confirmed Nipah encephalitis cases. These four cases were all between 32 and 60 years of age; three of the four died.

We observed two generations of Nipah transmission in this large cluster. The male index case had a history of drinking raw date palm sap with other family members and his house was under a bat roost. He had no physical contact with subsequent cases of the first generation. The subsequent three cases had onset of illness on a same day (Figure 2). All three cases were from a single family, living about 250 meters away from a bat roost, who drank raw date palm sap together during the previous week. After another seven days, three other persons who had a history of close physical contact with the index case became sick. All of them died. None of them had a history of drinking raw date palm sap within the last one month. The last case of the cluster, the wife of the index case, was the only survivor. She had reported drinking raw date palm sap and had contact with the index case.

As part of Nipah surveillance, we also investigated all isolated laboratory confirmed Nipah positive cases. Among the nine cases identified between December 2009 to April 2010, one case was an intern doctor, who had very recently joined the paediatric department of Fardipur Medical College Hospital. He performed several physical examinations, without any personal protective equipment, on two young girls, aged 5 and 7 years old, who had been admitted to the hospital with encephalitis. One of these two children died, and the doctor died after six days. All three were Nipah antibody positive. The overall case fatality rate of Nipah encephalitis cases was 88% (15/17).

Figure 2: Faridpur Nipah cases by date of illness onset, 2010 (N=17)



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Comments

This large Nipah outbreak, across a wide geographic area in Faridpur district in 2010, suggests repeated introduction of the virus from its wildlife reservoir in *Pteropus* bats into people. Two of the infected persons subsequently transmitted the virus to caregivers, including a physician. Previous outbreak investigations in Bangladesh provide compelling evidence of person-to-person transmission of Nipah infection (4). However, the infection and death of a physician who cared for Nipah patients was the first reported nosocomial transmission of Nipah virus in Bangladesh. Repeated episodes of person-to-person transmission of Nipah virus emphasize the importance of interrupting viral transmission through body fluids, both at household level and at healthcare facilities.

Several protective interventions to prevent person-to-person transmission should be prioritized through the development of appropriate, culturally

sensitive health messages for the caregiver focusing on frequent hand washing, avoiding sharing of food and bed, and maintaining a three feet distance while caring for patients (two hands length). Also prompt isolation of patients with meningo-encephalitis syndrome during the Nipah virus season and implementation of basic infection control measures can reduce the risk to hospital staff and patients (5). As the laboratory diagnosis for Nipah virus is not available during initial evaluation of patients with meningo-encephalitis syndrome, health care workers should routinely practice basic infection control measures with every patient. Novice physicians should receive specific training on personal protective measures before beginning hospital duties.

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Clusters of severe respiratory infections identified through hospital-based influenza surveillance, Bangladesh, 2009-2010

Acute respiratory infections are the leading cause of death in Bangladesh. During May 2009-March 2010 we conducted cluster investigations embedded within the national hospital based influenza surveillance to identify any outbreak in the community and to characterize new viral pathogens causing severe respiratory infections. Out of 186 case-patients (85 clusters), 24 (13%) tested positive for influenza and 76 (41%) for other respiratory viruses. Respiratory syncytial virus accounted for 32% severe respiratory infections among children aged less than 1 year.

Acute respiratory infection causes significant morbidity and mortality in low-income countries, including Bangladesh, and disproportionately affects children (1). The rate of upper respiratory tract infections among children <5 from a population-based surveillance in Dhaka was 1.5 events per child per year (2). In Bangladesh, two-thirds of deaths among children <5 are attributed to acute respiratory infections (3). These infections exact both a health and an economic burden. The average direct expenditure for hospitalization due to respiratory tract infection in Dhaka is US$98 (4), a sum that can be catastrophic when 50% of the population's per capita earnings is below the international poverty line of US$1.25 per day (5).

Several pathogens, including viruses, contribute to the morbidity and mortality among children <5 due to respiratory tract infections. A study conducted in Thailand among hospitalized pneumonia patients identified viral agents in the nasopharyngeal swabs among 746 (43%) of 1,730 episodes of radiologically confirmed pneumonia (6). Findings from population-based surveillance in Kamalapur, Bangladesh, suggests that human metapneumovirus are responsible for 33% of respiratory infection among children aged <13 (7). Influenza was associated with 10% of all childhood pneumonia among children <5 (2). A birth cohort study of children aged <24 months suggests that respiratory syncytial virus were the predominant aetiologic agent among 81% of diagnosed pneumonia cases (8).

Although Bangladesh is increasingly generating data on the aetiology of respiratory illnesses, clinicians and public health authorities do not know which pathogens cause clusters of severe respiratory illnesses that occur among children and adults. Each cluster represents a group of persons who are epidemiologically linked by their proximity in time and place and may be the first detectable manifestation of a novel virus that could place the

broader population at risk. As part of the national hospital based influenza surveillance, we conduct cluster investigations to identify respiratory viruses causing severe respiratory infections outbreaks in the community. In this article we present the findings of our cluster investigations conducted during May 2009 to March 2010.

During April 2007, the Institute of Epidemiology Disease Control and Research (IEDCR) of the Government of Bangladesh and ICDDR,B established an influenza surveillance network in 12 hospitals across the country to identify clusters of people with severe influenza infection (Figure 1). This surveillance was not designed to sample a large portion of the population, but rather concentrated on sampling rare events, i.e. epidemiologically linked clusters of respiratory illness of potential public health significance.

In each participating hospital, the surveillance physicians screened patients >5 years with severe acute respiratory illness (SARI) and children <5 years with severe pneumonia (Figure 1). Each month in each hospital, surveillance physicians collected samples from these selected patients and also collected demographic and clinical information using a handheld computer. Surveillance physicians collected throat and nasal swab specimens from the identified severe acute respiratory illness and severe pneumonia case-patients who met the case definitions and stored them in viral transport media (Figure 1). The field assistants transported the swabs to the ICDDR,B virology laboratory to test for influenza by real time reverse transcriptase polymerase chain reaction.

Figure 1: Surveillance case definitions

Severe acute respiratory illness (SARI):

Hospitalized patient with
– History of fever within 21 days and
– Cough or sore throat

Severe pneumonia:

Hospitalized patient with cough or difficult breathing and
– Chest indrawing or
– Stridor in calm child or
– History of convulsions or
– Not able to drink or
– Lethargic or unconscious or
– Vomits everything

Influenza like illness (ILI):

Outpatient from medicine and paediatric department with
– Fever and cough or sore throat

Cluster:

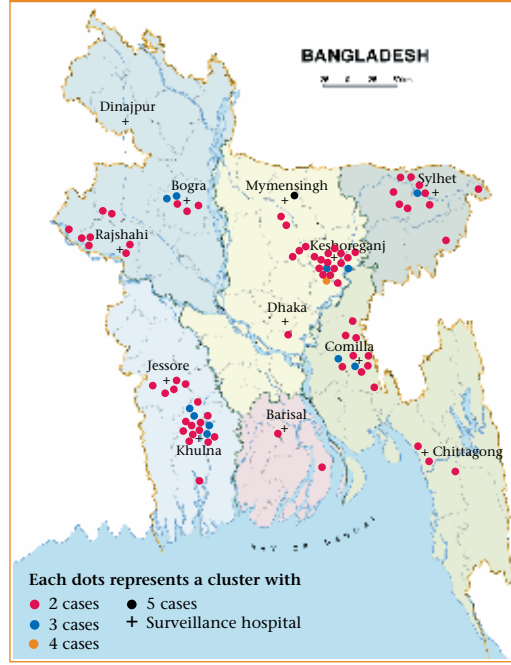
Two or more severe acute respiratory illness or severe pneumonia case-patients:
– Who live within a 30 minutes' walk or within 3 kilometre radius and
– Developed symptoms within 7 days of each other

Surveillance physicians tallied the case-patients who met the case definitions of severe acute respiratory illness and severe pneumonia to identify whether two or more of these case-patients lived within 30 minutes walk

or within 3 kilometre radius, and who had developed symptoms within 7 days of each other (Figure 1). When surveillance physicians identified such case-patients, all of the case-patients were considered as a single cluster. After identifying clusters, the surveillance physicians collected samples and notified IEDCR and ICDDR,B investigators. Field assistants enquired about potential epidemiological links, contact history, family history, travel history, community outbreaks and poultry or animal deaths using a cluster investigation form. All the cluster samples were tested at ICDDR,B laboratory by real time reverse transcriptase polymerase chain reaction for a panel of respiratory pathogens including influenza, parainfluenza type 1, type 2 and type 3, respiratory syncytial virus, adenovirus and human metapneumovirus.

During May 2009-March 2010, we identified 85 clusters of severe respiratory illness comprised of 85 individuals >5 years with severe acute respiratory illness and 101 children <5 years with severe pneumonia. Among these 85 clusters, 72 (85%) were comprised of 2 case-patients, 11 (13%) of 3 case-patients, 1 (1%) of 4 case-patients and 1 (1%) of 5 case-patients (Figure 2). Severe acute respiratory illness case-patients ranged in age between 5 and 100 years with a mean age of 35 years (±21.5). For severe pneumonia the mean age was 4.6 months. Of 101 severe pneumonia case-patients, 73 (72%) were male, and among the 85 severe acute respiratory illness case-patients, 51 (60%) were male. A total of 8 clusters in different households were identified among twins and all of these case-patients were male. Out of 156 case-patients who provided information, 105 (67%) live in homes where poultry were raised.

Figure 2: Location of identified clusters by IEDCR/ICDDR,B hospital-based influenza surveillance during May 2009-March 2010



Among the 85 severe acute respiratory illness case-patients 17 (20%) had a history of smoking and 7 (8%) had pre-existing lung disease (Table 1).

Out of the 186 case-patients, 100 (54%) tested positive for respiratory viruses. Among these 100 positive case-patients, 32

Table 1: Clinical presentations of the case-patients identified by IEDCR/ ICDDR,B hospital-based influenza surveillance during May 2009-March 2010

History and clinical presentations	Case patients	
	Severe Acute Respiratory Illness n=85 N (%)	Severe Pneumonia n=101 N (%)
Cough	85 (100)	100 (99.0)
Difficulty breathing	45 (53.0)	97 (96.0)
Runny nose	36 (42.0)	58 (57.0)
Sore throat	27 (32.0)	0 (0.0)
Headache	46 (54.0)	0 (0.0)
History of smoking	17 (20.0)	0 (0.0)
Pre-existing lung disease	7 (8.0)	6 (6.0)
Chest indrawing	0 (0.0)	90 (89.0)
Rhonchi	0 (0.0)	45 (45.0)
Crepitation	0 (0.0)	65 (64.0)

were infected with respiratory syncytial virus, 24 (24%) with influenza and 20 (20%) with human metapneumovirus (Table 2). All 32 (32%) case-patients infected with respiratory syncytial virus were severe pneumonia case-patients (Table 2). Among all case-patients the median interval between symptom onset and date of admission was 3 days and median interval between date of admission and availability of laboratory results was 7 days. The total median lag between symptom onset and laboratory confirmation of its aetiology was 10 days. The age group of 0-5 years was most infected with the respiratory viruses (Figure 3). Among the 64 chest x-ray findings available from the case-patients, 32 (50%) had a normal chest x-ray, 13 (20%) lobar consolidation, 5 (8%) alveolar infiltration, and 5 (8%) had interstitial infiltration.

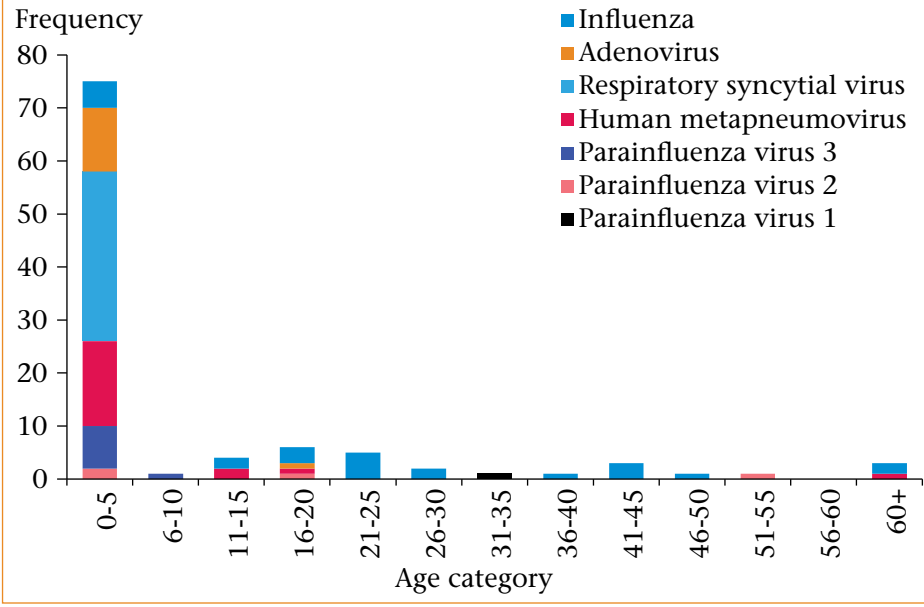
The median interval between symptom onset and date of admission of all the cluster case-patients was 3 days (interquartile range = 2-4 days). Information about the outcome of hospitalization was available for 176 (95%) of 186 case patients. Among these 176 case-patients, 1 patient died (case fatality proportion 0.5), 3 (2%) were transferred to another hospital, 119 (68%) fully recovered, 53(30%) recovered partially, i.e. either were discharged from the hospital while patient was improving, or discharged on request by the patient or patient's guardian.

Table 2: Demographics and proportion of laboratory confirmed viral pathogens of the case-patients identified by IEDCR/ICDDR,B hospital-based influenza surveillance during May 2009-March 2010

Respiratory viruses	Age of the case-patients	Proportion of respiratory virus positive cases	Duration of hospitalization
	Median (IQR*)	N (%)	Median (IQR*)
Respiratory syncytial virus	3 months (2-7 months)	32 (32%)	5 (3-7 days)
Influenza	23 years (15-40 years)	24 (24%)	4 (3-6 days)
Human metapneumovirus	8 months (4 months-5 years)	20 (20%)	5 (3-7 days)
Adenovirus	1.5 years (3 months-2.5 yrs.)	13 (13%)	4 (2-6 days)
Parainfluenza 3	1 year (5 months-9 yrs.)	9 (9%)	4 (2-6 days)
Parainfluenza 2		4 (4%)	5 (3-7 days)
Parainfluenza 1		1 (1%)	4 (2-7 days)

*Interquartile range

Figure 3: Age groups infected with different respiratory viruses, May 2009-March 2010



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Comments

Respiratory viral pathogens caused a substantial proportion of severe respiratory illness case-patients during May 2009-March 2010. While the respiratory syncytial virus and human metapneumovirus affected children <5, the median age of case-patients infected with influenza virus was 22 years (interquartile range = 15-40 years). Respiratory viruses other than influenza disproportionately affected children <5.

Our data suggest that there is a time lag ranging from 3-10 days from symptom onset to the investigation of clusters and laboratory confirmation of its aetiology. This time lag represents approximately 2-3 incubation periods for a respiratory virus like influenza, which has an incubation period of 1.4 days (9). Such delays suggests that potential public health events of international concern are unlikely to be identified before their 2nd or 3rd generation (9). Early recognition provides an opportunity for early intervention. Clinicians are urged to remain alert and report these clusters to public health authorities as soon as they are identified.

The transmission of respiratory infections can be interrupted through non-pharmaceutical interventions, for example by improving respiratory hygiene and washing hands with soap (10,11) and pharmaceutical measures, including antiviral medications and vaccines. Handwashing and maintaining respiratory hygiene by covering the mouth and nose during coughing or sneezing can significantly reduce the risk of respiratory infections (10,11). Unlike pharmaceutical interventions these non-pharmaceutical interventions have the benefit of preventing the transmission of multiple pathogens.

Our surveillance suggests that respiratory viruses play a significant role in causing clusters of hospitalized severe respiratory illnesses. Clinicians, the government, and health organizations can work to reduce the burden of these diseases by encouraging people to maintain hand hygiene, such as hand washing, and respiratory hygiene, such as covering mouth and nose during coughing or sneezing. In addition, vaccine trials among young children for influenza and, when available, for respiratory syncytial virus, may demonstrate additional useful interventions.

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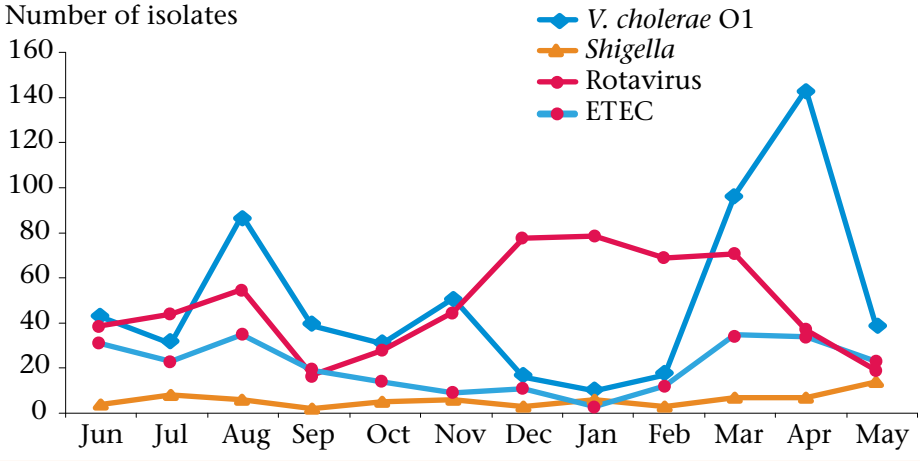
Surveillance updates

With each issue of HSB, updates of surveillance data described in earlier issues are provided. These updated tables and figures represent the most recent observation period available at the time of publication. We hope these updates will be helpful to health professionals who are interested in current patterns of disease and drug resistance in Bangladesh.

Proportion of diarrhoeal pathogens susceptible to antimicrobial drugs: June 2009-May 2010

Antimicrobial agents	*Shigella* (n=83)	*V. cholerae* O1 (n=619)
Nalidixic acid	25.0	Not tested
Mecillinam	62.2	Not tested
Ampicillin	50.6	Not tested
TMP-SMX	36.1	1.3
Ciprofloxacin	65.1	99.8
Tetracycline	Not tested	53.0
Erythromycin	Not tested	0.2
Furazolidine	Not tested	Not tested

Monthly isolation of* V. cholerae *O1*, Shigella, *Rotavirus and ETEC June 2009-May 2010



Antimicrobial resistance patterns of 112 M. tuberculosis *isolates: June 2009-May 2010*

Drugs	Resistance type		Total n=112 (%)
	Primary n=98 (%)	Acquired* n=14 (%)	
Streptomycin	11 (11.2)	0 (0.0)	11 (9.8)
Isoniazid (INH)	5 (5.1)	2 (14.3)	7 (6.3)
Ethambutal	0 (0.0)	0 (0.0)	0 (0.0)
Rifampicin	0 (0.0)	0 (0.0)	0 (0.0)
MDR (INH+Rifampicin)	0 (0.0)	0 (0.0)	0 (0.0)
Any drugs	13 (13.3)	2 (14.3)	15 (13.4)

() column percentage *Antituberculous drugs received for 1 month or more

Antimicrobial susceptibility pattern of S. pneumoniae *among children <5 years during April-June 2010*

Antimicrobial agents	Total tested (n)	Susceptible n (%)	Reduced susceptibility n (%)	Resistant n (%)
Ampicilin	0	0 (0.0)	0 (0.0)	0 (0.0)
Cotrimoxazole	1	0 (0.0)	0 (0.0)	1 (100.0)
Chloramphenicol	1	1 (100.0)	0 (0.0)	0 (0.0)
Ceftriaxone	1	1 (100.0)	0 (0.0)	0 (0.0)
Ciprofloxacin	0	0 (0.0)	0 (0.0)	0 (0.0)
Gentamicin	1	1 (100.0)	0 (0.0)	0 (0.0)
Oxacillin	1	1 (100.0)	0 (0.0)	0 (0.0)

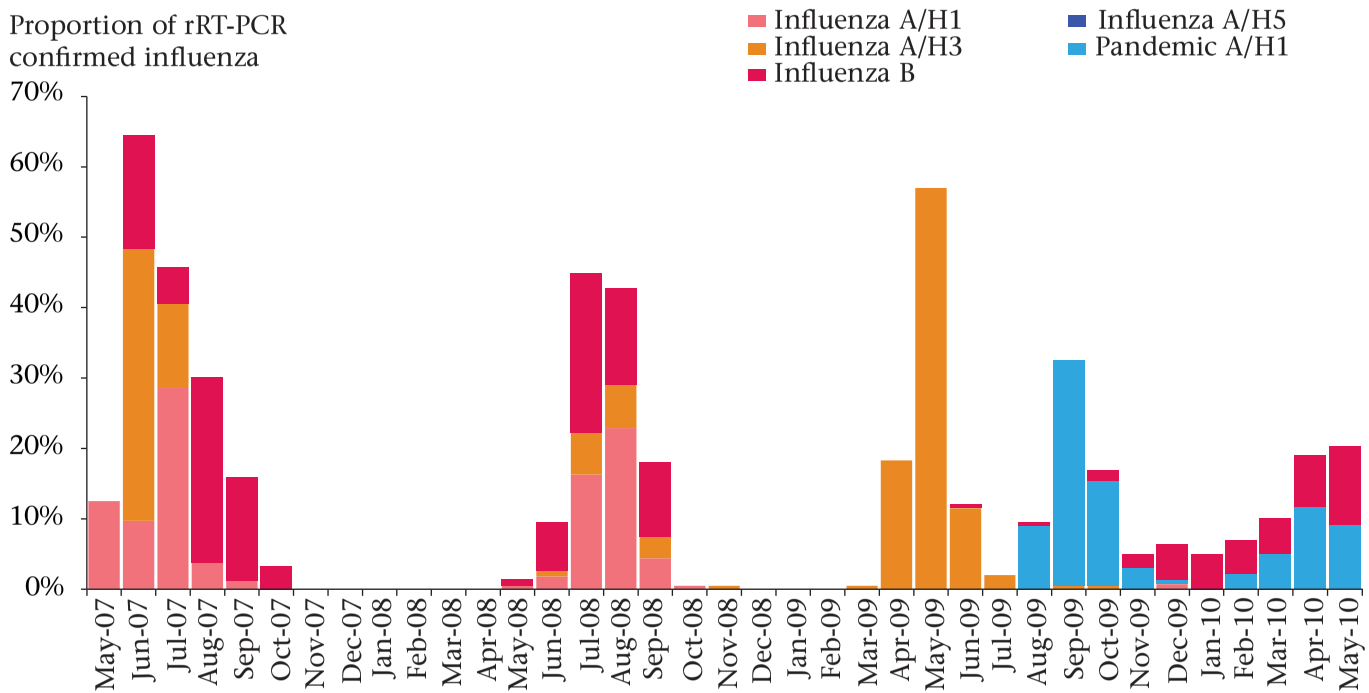
Source: ICDDR,B's urban surveillance in Kamalapur (Dhaka).

Antimicrobial susceptibility pattern of S. typhi *among children <5 years during April-June 2010*

Antimicrobial agents	Total tested (n)	Susceptible n (%)	Reduced susceptibility n (%)	Resistant n (%)
Ampicilin	29	13 (45.0)	0 (0.0)	16 (55.0)
Cotrimoxazole	29	13 (45.0)	0 (0.0)	16 (55.0)
Chloramphenicol	29	12 (41.0)	0 (0.0)	17 (59.0)
Ceftriaxone	29	29 (100.0)	0 (0.0)	0 (0.0)
Ciprofloxacin	29	0 (0.0)	29 (100.0)	0 (0.0)
Nalidixic Acid	29	0 (0.0)	0 (0.0)	29 (100.0)

Source: ICDDR,B's urban surveillance in Kamalapur (Dhaka).

Proportion of laboratory confirmed influenza among hospitalized severe acute respiratory illness (SARI) and outpatient influenza like illness (ILI) cases between May 2007 and May 2010



Source: Patients participating in hospital-based influenza surveillance in Dhaka National Medical College Hospital, Community-based Medical College Hospital (Mymensingh), Jahurul Islam Medical College Hospital (Kishoregonj), Rajshahi Medical College Hospital, Shaheed Ziaur Rahman Medical College Hospital (Bogra), LAMB Hospital (Dinajpur), Bangabandhu Memorial Hospital (Chittagong), Comilla Medical College Hospital, Khulna Medical College Hospital, Jessore General Hospital, Jalalabad Ragib-Rabeya Medical College Hospital (Sylhet) and Sher-e-Bangla Medical College Hospital (Barisal)

A bat roost at a village in Faridpur District.

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