WEEKLY EPIDEMIOLOGICAL REPORT

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Ministry of Health

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The immune system (Part II)

This is the second in a series of two articles on the immune system

Phagocytes - Mononuclear phagocytes, Neutrophils etc. engulf foreign organisms or particles. They form a link between the specific and the non specific arms of the immune system by presenting foreign fragments on their surface to T-cells and B-cells..

Auxiliary cells control inflammation and soluble mediators to the site of infection. – Basophils and Mast-cells have granules in them that produce inflammation in surrounding tissues. They also release a number of mediators that control the development of immune reactions. Platelets also release inflammatory mediators.

Dendritic cells (DC)

These are immune cells and present at a low frequency in those tissues which are in contact with the environment: in the skin (where they are often called Langerhans cells) and the lining of nose, lungs, stomach and intestines. Especially in immature state, they can also be found in blood. Once activated, they migrate to the lymphoid tissues where they interact with T-cells and B-cells to initiate and shape the immune response. In certain stages they have long spiky arms, called dendrites, hence the name.

Soluble mediators: A wide variety of molecules are involved in the development of immune responses. These include antibodies, cytokines and compliment (normally present in serum). Although there is a vast array of these molecules with many functions, it is important to be aware of a few as these are important in vaccination.

Antibody

An antibody is a protein used by the immune system to identify and neutralise foreign objects like bacteria and viruses. Each antibody recognises a specific antigen unique to its target. Production of antibodies is referred to as the humoral immune system. The terms antibody and immunoglobulin are often used interchangeably. They are found in the blood and tissue fluids, as well as many secretions. They are synthesised and secreted by plasma cells which are derived from the B-cells of the immune system. B-cells are activated upon binding to their specific antigen and differentiate into plasma cells.

There are five classes of antibody – IgG, IgA, IgM, IgD and IgE. These are all structurally slightly different and have a range of functions. Each B-cell can produce only one specific antibody to an antigen, each antibody is highly specific and will bind to only one antigen.

Cytokines

Cytokines are small protein molecules that regulate communication among immune system cells and between immune cells and those of other tissue types. Immune cells, as well as other cell types in response to external stimuli actively secrete these chemicals. Cytokines that are produced by immune cells form a subset known as lymphokines.

The actions of cytokines are complex - the same cytokine can have different effects on a cell depending on the state of the cell. For instance, there are several known cytokines that have both stimulating and suppressing action on lymphocyte cells and immune response. There are three classes of cytokines. Hundreds of cytokines have been discovered and more and more are being discovered.

Complement

The complement system is derived from many small plasma proteins that form the complex biochemical cascade of the immune system, leading to cell destruction, attraction of immune cells, opsonisation and inflammation; it can mark pathogens for phagocytosis. It consists of more than 35 proteins, 12 which are directly, involved in the complement pathways, while the rest have regulatory functions. There are three biochemical pathways, which activate the complement system: the classical complement pathway, the alternate complement pathway and the mannan-binding lectin pathway.

Opsonisation – antibody and complement

An opsonin is any molecule that acts as a binding enhancer for the process of phagocytosis. During

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the process of opsonisation, antigens are bound by antibody and/or complement molecules. Phagocytic cells express receptors that bind opsonin molecules. With the antigen coated in these molecules, binding of the antigen to the phagocyte is greatly enhanced. Examples of opsonin molecules include the IgG antibody and the C3b, C4b and iC3b components of the complement system.

Antigen presenting cells

There are a number of cells that are able to take up foreign antigen and "present" it on their surface for the rest of the immune system to see – in particular, T-cells and B-cells.
Cells that are particularly good at presenting antigen are: Dentritic cells, Macrophages and B-cells.

Humoral and cellular immune responses – making memory

Immune memory is retained by B-cells and T-cells. Responses by B-cells are humoral, responses by T-cells are called cellular.

Humoral immunity is mediated by secreted antibodies, produced in the cells of the B lymphocyte lineage (B-cell). Secreted antibodies bind to antigens on the surfaces of invading microbes, which flags them for destruction. Humoral immunity refers to antibody production, and all the accessory processes that accompany it.

Cell-mediated immunity is an immune response that does not involve antibodies but rather involves the activation of macrophages and natural killer cells, the production of antigen-specific cytotoxic T-lymphocytes, and the release of various cytokines in response to an antigen. Cellular immunity protects the body by:

- activating antigen-specific cytotoxic T-lymphocytes that are able to destroy body cells displaying epitopes (fragments) of foreign antigen on their surface, such as virus-infected cells, cells with intracellular bacteria, and cancer cells displaying tumor antigens;
- activating macrophages and natural killer cells, enabling them to destroy intracellular pathogens; and stimulating cells to secrete a variety of cytokines that influence the function of other cells involved in adaptive immune responses and innate immune responses. Cell-mediated immunity is directed primarily at microbes that survive in phagocytes and microbes that infect non-phagocytic cells. It is most effective in removing virus-infected cells, but also participates in defending against fungi, protozoans, cancers, and intracellular bacteria.

Primary and secondary responses

When the body is first exposed to an antigen, several days pass before the adaptive immune response becomes active. Immune activity then rises, levels off, and falls. Following exposures to the same antigen, the immune system responds much more quickly and reaches higher levels. Because the first, or primary, immune response is slow, it cannot prevent disease, although it may help in recovery. In contrast, subsequent, or secondary, immune responses usually can prevent disease because the pathogen is detected, attacked, and destroyed before symptoms appear.

The infant immune system

The infants' immune system is intact but immature at birth. Some vaccines such as BCG and Hepatitis B work well when they are administered at birth whereas others do not generate as strong a response.

The main problem with babies' immunity is that it is very naïve. At the time of birth babies have not been exposed to any pathogens. This means that babies have to generate a full immune response to every pathogen they encounter. Each immune response takes about 10 days to generate. This is where maternal antibody can be important when present: It will help to protect infants if they are exposed to a pathogen in those first 10 days. Unlike other animals (such as ruminants) which rely mainly on passive transfer of maternal antibodies in breast milk, humans receive most of their maternal antibodies through placental transfer of IgG. However, there will still be some antibodies transferred in breast milk, but the levels are much lower. In addition human babies don't have a porous stomach (like calves do) in order to absorb antibodies. Therefore, most of the antibodies in breast milk will work in protecting pathogens crossing the oral cavity.

Developing immune system before and after birth

Maternal

The immune system is designed to recognise 'self' versus 'non self'. This means our own immune system can recognise our own cells as being safe and anything else as being a threat. Obviously this has implications in pregnancy, where a developing foetus will be expressing antigens from the father. Therefore during pregnancy modifications occur in the maternal immune system at many levels. These changes are necessary to ensure a successful pregnancy. In the absence of such changes the mother's immune system would recognise the foetus as foreign (like a pathogen) and reject it. Potentially dangerous T-cell responses are down regulated (reduced) and some aspects of the non-specific immune system are activated. As previously mentioned, at this time specific IgG antibody passes from the mother through the placenta to the developing fetus providing it with temporary protection against some of the infections that the mother has been exposed to or vaccinated against. This gives opportunities to provide newborns with transient protection against some diseases.

Infant

The infant's immune system is relatively complete at birth. It is clear that the IgG antibodies received from mother are important for the protection of the infant during the first few months of life while the infant is starting to develop its own repertoire. Passive transient protection by IgA against many common illnesses is also provided to the infant in breastmilk. Mother's milk provides IgA against a wide range of microbes that the mother has had in her gut. Breast milk has also been shown to assist in the development of the infant's own immune system. There is some, although weak, evidence to show that breastfed infants respond better to some vaccines. The major impetus however for the expansion of lymphocytes (B and T cells) is the exposure to microbes which colonise the gut during birth.

Premature and low birth weight infants are at increased risk of experiencing complications of vaccine preventable diseases and although the immunogenicity of some vaccines may be decreased in the smallest preterm infants, the antibody concentrations achieved are usually protective.

Source-The immune system and vaccination-available form *http://www.immune.org.nz/immune-system-and-vaccination*

Compiled by Dr. Madhava Gunasekera of the Epidemiology Unit

Table 4: Selected notifiable diseases reported by Medical Officers of Health 27th July – 02nd August (31st Week)

RDHS	Dengue Fever		Dysentery		Encephaliti		E Fever		F Poisoning		Leptospiros		T Fever		V Hepatitis		H Rabies		Chickenpox		Meningitis		Leishmaniasis		WRCD %	
	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	T*	C**
Colombo	154	5903	8	130	0	13	2	90	0	35	1	142	0	5	2	57	0	0	2	272	1	32	0	0	**69**	**31**
Gampaha	83	2331	13	121	0	11	4	32	1	24	6	233	1	12	4	135	0	0	1	112	4	67	0	5	**80**	**20**
Kalutara	36	1112	8	104	0	16	1	50	0	13	4	259	0	2	0	14	0	0	2	14	0	49	0	0	**77**	**23**
Kandy	34	1187	2	94	0	6	0	15	0	7	0	51	0	80	0	61	0	0	0	89	0	10	0	2	**65**	**35**
Matale	13	305	2	65	0	3	0	16	0	3	2	49	0	2	1	32	0	0	0	33	1	26	0	6	**38**	**62**
NuwaraEliya	12	169	1	110	0	2	0	6	0	3	0	21	2	54	1	18	0	0	2	65	0	9	0	0	**69**	**31**
Galle	9	567	5	71	0	12	0	3	0	79	0	145	1	33	0	9	0	1	7	213	1	39	0	0	**68**	**32**
Hambantota	7	215	0	34	0	3	0	10	0	20	0	147	3	50	0	71	0	0	0	74	0	23	9	209	**58**	**42**
Matara	11	348	4	60	0	9	0	19	0	27	1	115	2	47	2	124	0	2	5	188	4	53	1	58	**88**	**12**
Jaffna	11	512	6	151	0	5	5	277	0	83	0	7	0	325	0	14	0	1	0	119	1	43	0	0	**100**	**0**
Kilinochchi	0	34	0	14	0	0	0	9	0	5	0	9	0	16	0	0	0	0	0	2	0	7	0	6	**25**	**75**
Mannar	0	57	0	34	0	1	0	54	0	14	0	11	0	17	0	2	0	0	0	11	0	4	0	1	**80**	**20**
Vavuniya	1	56	4	35	0	11	1	8	0	13	0	48	0	2	1	2	0	2	0	19	0	26	0	7	**100**	**0**
Mullaitivu	1	93	0	10	0	1	0	6	0	34	0	31	0	6	0	0	0	2	0	7	0	4	1	10	**80**	**20**
Batticaloa	8	453	3	198	0	4	0	3	0	14	0	28	0	2	0	9	1	3	3	29	1	7	0	0	**64**	**36**
Ampara	5	111	4	77	0	0	0	4	0	6	0	24	0	1	0	2	0	0	0	57	0	11	0	1	**43**	**57**
Trincomalee	3	167	1	48	0	3	0	4	0	1	1	56	0	7	0	3	0	1	0	32	0	4	0	26	**67**	**33**
Kurunegala	17	2196	0	119	0	26	0	29	0	15	0	203	0	24	0	37	0	1	1	252	0	84	0	34	**33**	**67**
Puttalam	3	664	0	47	0	4	0	15	0	35	1	24	0	12	0	4	0	0	1	60	0	20	0	7	**15**	**85**
Anuradhapura	6	384	3	64	0	13	0	3	1	19	1	285	0	17	0	15	00	1	3	120	2	80	6	256	**58**	**42**
Polonnaruwa	11	263	1	49	0	1	1	13	0	53	1	142	0	3	1	24	0	1	2	106	0	16	7	99	**86**	**14**
Badulla	8	344	1	121	0	3	0	12	0	8	2	38	0	56	1	36	0	0	0	88	1	50	0	4	**71**	**29**
Monaragala	2	168	3	81	0	3	2	16	1	20	0	182	4	35	7	67	0	1	0	39	3	18	0	9	**73**	**27**
Ratnapura	20	1338	4	263	0	80	0	34	0	16	2	245	1	35	3	210	0	1	1	110	1	59	0	8	**61**	**39**
Kegalle	22	759	2	86	0	11	2	17	0	8	2	135	1	60	5	161	0	0	7	220	2	82	0	0	**100**	**0**
Kalmune	0	480	0	107	0	1	0	3	0	73	0	5	0	2	0	4	0	0	3	62	0	8	0	1	**46**	**04**
SRI LANKA	**477**	**20216**	**75**	**2293**	**00**	**242**	**18**	**748**	**03**	**628**	**24**	**2635**	**15**	**905**	**28**	**1111**	**01**	**17**	**40**	**2563**	**22**	**831**	**24**	**749**	**64**	**36**

Source: Weekly Returns of Communicable Diseases (WRCD).

***T=**Timeliness refers to returns received on or before 02nd August , 2013 Total number of reporting units 339. Number of reporting units data provided for the current week:216 C** Completeness

A = Cases reported during the current week. **B** = Cumulative cases for the year.**H Rabies***= Human Rabies, **E Fever***=Enteric Fever, **F Poison*** =Food Poisoning, **T Fever***=Typhus Fever, **V Hepatitis***=Viral Hepatitis

Table 1: Vaccine-Preventable Diseases & AFP

27th July – 02nd August (31st Week)

Disease	No. of Cases by Province									Number of cases during current week in 2013	Number of cases during same week in 2012	Total number of cases to date in 2013	Total number of cases to date in 2012	Difference between the number of cases to date in 2013 & 2012
	W	C	S	N	E	NW	NC	U	Sab					
AFP*	00	02	00	01	00	00	00	00	00	03	02	52	49	04.1 %
Diphtheria	00	00	00	00	00	00	00	00	00	-	-	-	-	-
Mumps	06	15	04	01	00	02	02	03	01	34	53	989	2717	- 63.6 %
Measles	45	11	19	00	01	02	05	05	38	126	00	1790	92	+ 1845.6 %
Rubella	00	00	00	00	00	00	00	00	00	00	-	21	-	-
CRS**	00	00	00	00	00	00	00	00	00	00	-	06	-	-
Tetanus	00	00	00	00	00	00	00	00	00	00	00	12	13	- 07.7 %
Neonatal Tetanus	00	00	00	00	00	00	00	00	00	00	-	00		-
Japanese Encephalitis	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Whooping Cough	00	00	00	00	00	00	00	00	01	01	01	56	24	+ 133.3 %
Tuberculosis	01	41	26	13	04	68	04	13	01	171	177	5206	5475	- 04.9 %

Key to Table 1 & 2

Provinces: **W**: Western, **C**: Central, **S**: Southern, **N**: North, **E**: East, **NC**: North Central, **NW**: North Western, **U**: Uva, **Sab**: Sabaragamuwa.

RDHS Divisions: **CB**: Colombo, **GM**: Gampaha, **KL**: Kalutara, **KD**: Kandy, **ML**: Matale, **NE**: Nuwara Eliya, **GL**: Galle, **HB**: Hambantota, **MT**: Matara, **JF**: Jaffna, **KN**: Killinochchi, **MN**: Mannar, **VA**: Vavuniya, **MU**: Mullaitivu, **BT**: Batticaloa, **AM**: Ampara, **TR**: Trincomalee, **KM**: Kalmunai, **KR**: Kurunegala, **PU**: Puttalam, **AP**: Anuradhapura, **PO**: Polonnaruwa, **BD**: Badulla, **MO**: Moneragala, **RP**: Ratnapura, **KG**: Kegalle.

Data Sources:

Weekly Return of Communicable Diseases: Diphtheria, Measles, Tetanus, Neonatal Tetanus, Whooping Cough, Chickenpox, Meningitis, Mumps., Rubella, CRS,

Special Surveillance: AFP* (Acute Flaccid Paralysis), Japanese Encephalitis

CRS =**Congenital Rubella Syndrome

AFP and all clinically confirmed Vaccine Preventable Diseases except Tuberculosis and Mumps should be investigated by the MOH

Dengue Prevention and Control Health Messages

Reduce, Reuse or Recycle the plastic and polythene collected in your home and help to minimize dengue mosquito breeding.

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Comments and contributions for publication in the WER Sri Lanka are welcome. However, the editor reserves the right to accept or reject items for publication. All correspondence should be mailed to The Editor, WER Sri Lanka, Epidemiological Unit, P.O. Box 1567, Colombo or sent by E-mail to **chepid@sltnet.lk. Prior approval should be obtained from the Epidemiology Unit before publishing data in this publication**

ON STATE SERVICE

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