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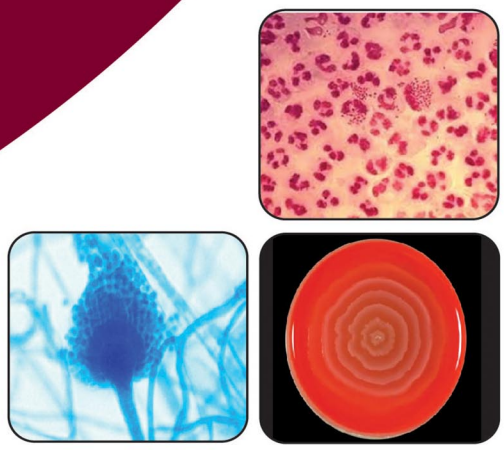
# Review of MICROBIOLOGY & IMMUNOLOGY

## *A Must-buy Book for NEET-PG, INI-CET & FMG Exams*

*Facts and concepts from latest editions of Harrison, Park, Apurba Sastry's Essentials of Medical Microbiology, Apurba Sastry's Essentials of Medical Parasitology and Apurba Sastry's Essentials of Hospital Infection Control.*

**Student's Feedback**

- Concise, accurate and written in an authentic manner, must buy
- Awesome book
- It is the best book to review Microbiology
- Must buy for PG preparation
- Wonderful book
- Best book by subject specialty
- Reading this MCQ book along with author's text book (Essentials of Medical Microbiology) is fantastic.



**Apurba S Sastry**
**Sandhya Bhat**

**9th Edition**

JAYPEE

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★★★★★ Most Important
★★★★ Very Important
★★★ Important

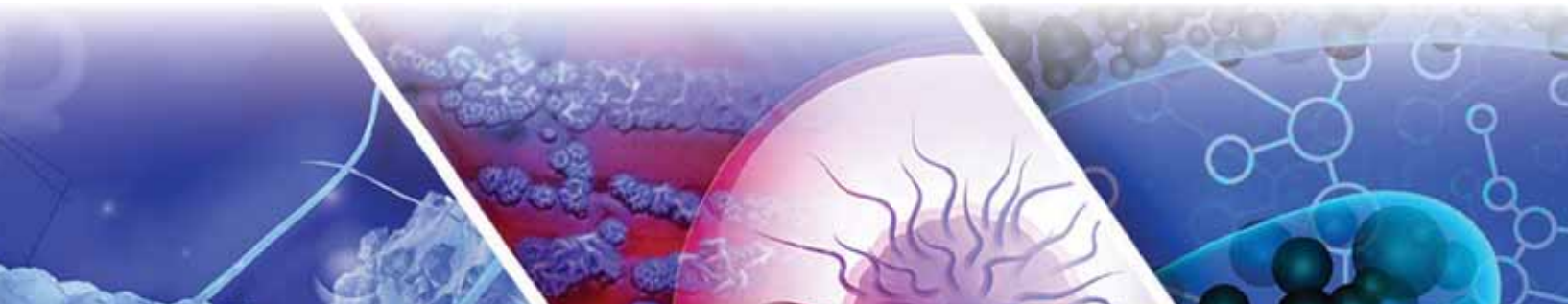
SECTION 2

# Immunology

## CHAPTER OUTLINE


2.1 Immunity
2.2 Antigen, Antibody, Antigen-Antibody Reaction, and Complement
2.3 Structure of Immune System and Immune Response
2.4 Hypersensitivity
2.5 Autoimmunity, Immunodeficiency, Transplantation, and Immunoprophylaxis



CHAPTER 2.1

# Immunity

## INNATE IMMUNITY

The term 'immunity' is defined as the resistance offered by the host against microorganism(s) or any foreign substance(s). Immunity can be broadly classified into:

1. Innate immunity: present right from the birth
2. Acquired/Adaptive immunity: acquired during the course of the life.

**Table 2.1.1: Differences between innate and acquired immunity**

| Innate immunity | Acquired/Adaptive immunity |
|---|---|
| Resistance to infection that an individual possesses from birth | Resistance to infection that an individual acquires during his lifetime |
| Immune response occurs in minutes | Immune response occurs in days |
| Prior exposure to the antigen is not required | Develops following the antigenic exposure |
| Diversity is limited, acts through a restricted set of reactions | More varied and specialized responses |
| Immunological memory responses are absent | Immunological memory responses are present |
| Respond to microbial antigens that are not specific to some microbe, rather shared by many microbes called microbes-associated molecular patterns (MAMP) | Respond to specific microbial antigens |
| Host cell receptors (pattern recognition receptors) are nonspecific, e.g. Toll-like receptor | Host cell receptors are specific, e.g. T cell receptors and B cell immunoglobulin receptors |
| **Components of innate immunity**<br>• Anatomical barriers, such as skin and mucosa<br>• Physiological barriers (e.g. body temperature)<br>• Phagocytes (neutrophils, macrophages and monocytes)<br>• Natural killer (NK) cells<br>• Other Classes of lymphocytes -γδ T cells , NK-T cells, B-1 cells and marginal-zone B cells<br>• Mast cells<br>• Dendritic cells<br>• Complement pathways: alternate and mannose binding pathways<br>• Fever and inflammatory responses<br>• Normal resident flora<br>• Cytokines- TNF, certain interleukin (IL-1, IL-6, IL-8, IL-12, IL-16, IL-18), IFN-α, β and TGF-β<br>• Acute phase reactant proteins (APRs) | **Components of acquired immunity:**<br>• T cell<br>• B cell<br>• Classical complement pathway<br>• Antigen presenting cells<br>• Cytokines (IL-2, IL-4, IL-5, IFN-γ)<br><br>**Types of acquired immunity:**<br>It can be classified into two ways:<br>• Active and passive immunity<br>• Artificial and natural immunity |

### Factors Influencing Innate Immunity

- ❑ Depends on the Species, Race, Individual (genetic influence)
- ❑ Age, Hormonal influence and Nutrition.

### Toll Like Receptors

They are so named because they are similar to Toll receptors present in the fruit fly- Drosophila, where it is the main receptor for induction of innate immunity by bind to particular MAMP molecules on microbial surfaces. They are of 13 types, out of which important ones are:

**Important Points**

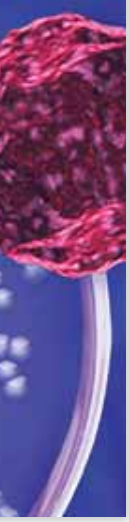
**Toll like receptors:**
They are the principle host cell receptors of innate immunity

- ❑ TLR-2 binds to bacterial peptidoglycan
- ❑ TLR-3 binds to dsRNA of viruses
- ❑ TLR-4 binds to LPS of Gram-negative bacteria
- ❑ TLR-5 binds to flagella of bacteria
- ❑ TLR-7 & 8 bind to ssRNA of viruses
- ❑ TLR-9 binds to bacterial DNA.

### Acute Phase Reactant Proteins (APRs)

They are the proteins synthesized by liver at steady concentration, but their synthesis either increases or decreases exponentially during acute inflammatory conditions.

Though liver is the primary site, APRs can also be synthesized by various other cells such as endothelial cells, fibroblasts, monocytes and adipocytes.



**Positive APRs** are the proteins whose levels increase during acute inflammation. Examples:

- Serum Amyloid A
- C- Reactive protein
- Complement proteins: Complement factors (C1–C9), factor B, D, and properdin
- Coagulation protein, e.g. fibrinogen, von Willebrand factor
- Proteinase inhibitors, e.g. α1 antitrypsin
- α1 acid glycoprotein
- Mannose binding protein
- Haptoglobin
- Metal binding proteins, e.g. ceruloplasmin

**Negative APRs:** They are the proteins whose levels are decreased during acute inflammation thus creating a negative feedback that stimulates the liver to produce positive APRs. Examples of negative APRs include: albumin, transferrin, and antithrombin.

**Role of APRs:** They have a wide range of activities that contribute to the host defense:

- APRs have various antimicrobial and anti-inflammatory activities (e.g. complement)
- Metal binding proteins can chelate various metals such as iron, copper, etc. making them unavailable for the bacteria.

### *C-Reactive Protein (CRP)*

CRP is an example of APR that rise in acute inflammatory conditions including bacterial infections. It belongs to beta globulin family.

- CRP is so named because it precipitates with C-carbohydrate (polysaccharide) antigen of Pneumococcus. However, it is not an antibody against the C-carbohydrate antigen of Pneumococcus; it is nonspecific, can be raised in any inflammatory conditions.
- Commonest marker of acute inflammation, used in most diagnostic laboratories.

**Important Points**

**CRP Level**

- Normal level <0.2 mg/dL. It increases by several folds in acute inflammatory conditions.
- Detection limit of latex agglutination test– 0.6 mg/dL
  - Insignificant increase (< 1 mg/dl), e.g. in heavy exercise, common cold, and pregnancy
  - Moderate increase (1–10 mg/dl), e.g. bronchitis, cystitis, malignancies, pancreatitis, myocardial infarction.
  - Marked increase (>10 mg/dl), e.g. acute bacterial infections, major trauma and systemic vasculitis.

**CRP can be detected by**

- Precipitation method using C carbohydrate antigen (obsolete, not in use now)
- Latex (passive) agglutination test using latex particles coated with anti-CRP antibodies.
  - It is the most widely used method employed worldwide.
  - Detection limit of CRP by latex agglutination test 0.6 mg/dL.
- **Highly sensitive CRP** (hs-CRP) test: Minute quantities of CRP can be detected by various methods (e.g. nephelometry, enzyme immunoassays). This is useful in assessing the risk to cardiovascular diseases.

## ACQUIRED OR ADAPTIVE IMMUNITY

Acquired immunity is defined as the resistance against the infecting foreign substance that an individual acquires or adapts during the course of his life.

- It is of two types: Active immunity and passive immunity.
- Active immunity can again be divided into:
  - Primary immune response (which develops after first microbial exposure)
  - Secondary immune response (which develops after subsequent microbial exposure).

**Table 2.1.2: Differences between active and passive immunity**

| Active immunity | Passive immunity |
|---|---|
| Produced actively by host immune system | Immunoglobulins received passively |
| Induced by:<br>• Infection (*natural*)<br>• Vaccination (*artificial*) | Acquired by:<br>• Mother to fetus IgG transfer (*natural*)<br>• Readymade antibody transfer (*artificial*) |
| Long lasting | Lasts for short time |
| Lag period present | No Lag period |
| Memory present | No memory |
| Booster doses are useful | Subsequent doses are less effective |
| Negative phase may occur | No negative phase |
| In immunodeficiency individuals not useful | Useful in immunodeficient individuals |

**Table 2.1.3: Differences between primary and secondary immune response**

| Primary immune response | Secondary immune response |
|---|---|
| Immune response against primary antigenic challenge | Immune response against subsequent antigenic challenge |
| Slow, sluggish (appear late) and short lived | Prompt, powerful and prolonged (long lasting) |
| Lag period is longer (4–7 days) | Lag period is absent or short (1–3 days) |

*Contd...*



*Contd...*

| Primary immune response | Secondary immune response |
|---|---|
| No negative phase | Negative phase may occur |
| Antibody produced in low titer and is of IgM type.<br>Antibodies are more specific but less avid | Antibody produced in high titer and is of IgG type<br>Antibodies are less specific but more avid |
| Antibody producing cells: Naive B cells | Antibody producing cells: Memory B cells |
| Both T dependent and T independent antigens are processed. | Only T dependent antigens are processed. |

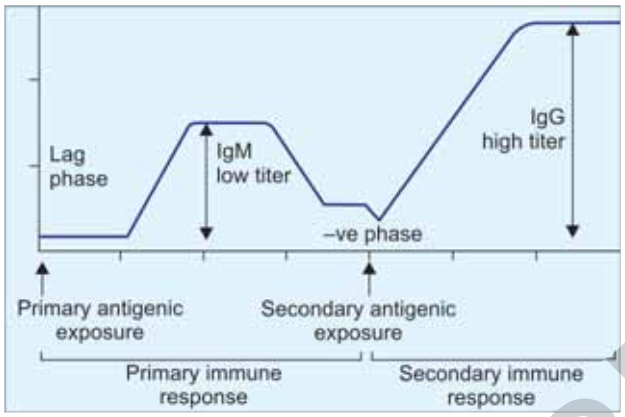



**Fig. 2.1.1:** Primary and secondary immune responses

**Important Points**

**Primary immune response**
Immune response against primary antigenic challenge
- Slow, sluggish (appear late) and short lived
- Lag period is longer (4–7 days)
- No negative phase
- Antibody produced in low titer and is of IgM type.
- Antibodies are more specific but less avid
- Antibody producing cells- Naive B cells
- Both T depd and T indepd Ag are processed.

## OTHER TYPES OF IMMUNITY

### Local (or mucosal) Immunity

Local or mucosal immunity is the immune response that is active at the mucosal surfaces such as intestinal or respiratory or genitourinary mucosa:

- It is usually mediated by a type of IgA antibody called secretory IgA.
- Local immunity can only be induced by natural infection or by live vaccination, e.g. after OPV (but not by killed vaccines).

**Important Points**

**Local or mucosal immunity**
Immune response that is active at the mucosal surfaces
- Mediated by secretory IgA.
- Induced by natural infection or by live vaccination

### Herd Immunity

Herd immunity is defined as the overall immunity of a community (or herd) towards a pathogen:

- Herd immunity plays a vital role in preventing epidemic diseases. If the herd immunity is good, that means large population of the community are immune towards a pathogen. Hence, epidemics are less likely to occur and eradication of the disease may be possible.
- Elements that contribute to create a strong herd immunity are:
  - Occurrence of clinical and subclinical cases in the herd
  - Ongoing immunisation programme
  - Herd structure, i.e. type of population involved
  - Type of pathogen: Herd immunity may not be strong in a community against all the pathogens.

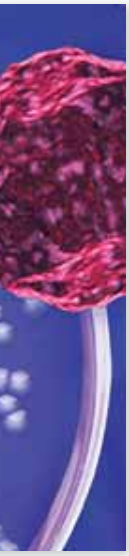
**Important Points**

**Herd immunity develops following vaccination against:**
- Diphtheria and Pertussis vaccine
- Measles, Mumps and Rubella (MMR) vaccine
- Polio (Oral polio vaccine)
- Smallpox vaccine

### Adoptive Immunity

It is the process of transfer of CMI from one individual to other.

- It occurs following injection of immunologically competent T-lymphocytes known as Transfer factor.
- It is useful for treatment when the CMI is low, e.g. in lepromatous leprosy.





## Multiple Choice Questions

1. **Toll like receptors: correct statement is:** *(Recent Question 2015)*
   a. Antigen specific
   b. Acts by cytokine release
   c. Part of adaptive immunity
   d. Facilitates microbial invasion
2. **Not true about innate immunity:** *(NEET Pattern Based)*
   a. Not influenced by hormones
   b. Dependent on genetic constitution
   c. Identical twins have same degree of resistance
   d. Not influenced by exposure to antigen
3. **All are true about innate immunity except:**
   a. Acts as first line of defense *(NEET Pattern Based)*
   b. Complements are examples
   c. Nonspecific
   d. Not effected by genetic influences
4. **Transfer factor is an example of:** *(NEET Pattern Based)*
   a. Artificial active immunity
   b. Natural active immunity
   c. Adoptive immunity
   d. Artificial passive immunity
5. **Components of innate immunity:** *(PGI Nov 2010)*
   a. T lymphocyte
   b. B lymphocyte
   c. Complements
   d. NK cells
   e. Integrins
6. **Innate immunity is stimulated by which part of bacteria?** *(DNB Dec 2011)*
   a. Carbohydrate sequence in the cell wall
   b. Flagella
   c. Bacterial cell membrane
   d. Nucleus
7. **Innate immunity active against viral cells:** *(AI 2007)*
   a. NK cells
   b. Cytotoxic T cells
   c. B cells
   d. Memory B cell
8. **All of the following are a part of the innate immunity except:** *(AIIMS May 2005)*
   a. Complement
   b. NK cells
   c. Macrophages
   d. T cells
9. **Active immunity can be induced by:** *(PGI May 2013)*
   a. Toxoids
   b. Subclinical infection
   c. Antitoxin
   d. Immunoglobulins
   e. Antigen exposure
10. **True about passive immunity:** *(PGI May 2013)*
    a. Cannot be given with active immunity
    b. Last for 4–5 days only
    c. It can be given before disease occurrence
    d. Can be transferred by antibodies from another Host
    e. Takes longer time to develop
11. **Superantigen is produced by:** *(Recent Question 2013)*
    a. *Staphylococcus aureus*
    b. *Streptococcus pneumoniae*
    c. Pseudomonas
    d. Clostridium

### ACUTE PHASE REACTANTS AND CRP

12. **Span of C reactive protein half-life:** *(TNPG 2014)*
    a. 18 hrs
    b. 2 hrs
    c. 12 hrs
    d. 15 hrs



## Explanations

1. **Ans. (b) (Acts by .....)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p98, 2/e p104*

   TLR is receptor of innate immunity, antigen nonspecific. These are signals generated following binding of TLRs to MAMPs activate transcription factors that stimulate expression of genes encoding cytokines and enzymes, which are involved in several antimicrobial activities of cells of innate immunity.

2. **Ans. (a) (Not influenced by hormones)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p97, 2/e p104, Ananthanarayan 9/e p78, 10/e p81, Kuby's Immunology 6/e p53*
   - ❑ Innate immunity is influenced by hormones: Endocrine disorders like diabetes are associated with enhanced susceptibility to infection due to altered innate immunity.
   - ❑ Innate immunity is dependent on genetic constitution of the individual. Homogenous identical twins exhibit similar degree of innate immunity.
   - ❑ Innate immunity is nonspecific, it is not influenced by exposure to antigen.

3. **Ans. (d) (Not effected by genetic influences)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p97, 2/e p104, Ananthanarayan 9/e p78, 10/e p81, Kuby's Immunology 6/e p53*
   - ❑ Innate immunity refers to the resistance to infection that an individual possess from birth by its genetic or constitutional make up.
   - ❑ Other options are correct- for explanation, refer text.

4. **Ans. (c) (Adoptive...)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p103, 2/e p110, Ananthanarayan 9/e p81, 10/e p86*

   **Adoptive immunity**
   - ❑ Acquired by injection of immunologically competent T-lymphocytes known as Transfer factor
   - ❑ Used for treatment when the CMI is low, e.g. Lepromatous leprosy.

5. **Ans. (c) (d) (e) (Complements, NK cells, Integrins)** *Apurba Sastry's Essentials of Medical Microbiology 1/e p97, 2/e p104, Ref: Ananthanarayan 9/e p79, 10/e p82-83, Kuby's Immunology 6/e p53, Harrison 19/e p2106, 20/e p2454*
   - ❑ Complement pathways (alternate and mannose binding , NK cells, and Pattern recognition receptors like Integrins are components of Innate immunity
   - ❑ For the detail list of components of Innate immunity- Refer text.

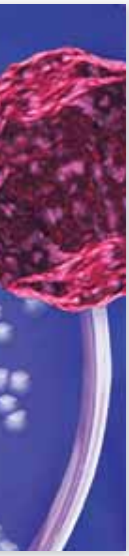
6. **Ans. (a) (Carbohydrate sequence in cell wall)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p97, 2/e p104, Harrison 19/e p2107, 20/e p2452-54,55*
   - ❑ Pattern recognition receptors are important component of innate immunity
   - ❑ Toll-like receptors are, e.g. of PRR Protein Family which bind to carbohydrate antigens on bacterial and viral surfaces.

   Examples of microbial ligands that bind to Pattern recognition receptors on host cells include:
   - ❑ Bacterial and viral carbohydrates residues on cell wall
   - ❑ Terminal mannose and Carbohydrate on HLA molecules
   - ❑ Lipopolysaccharide (LPS)
   - ❑ Viral DNA Bacterial muramyl dipeptide

7. **Ans. (a) (NK cells)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p97, 2/e p172, Ananthanarayan 9/e p137, 10/e p139*
   - ❑ Natural killer cells are components of innate immunity *Kuby's Immunology 6/e p53*
   - ❑ Natural killer cells possess spontaneous cytotoxicity towards virus infected cells and malignant cells and their cytotoxicity is not antibody dependent nor MHC restricted
   - ❑ About Other options: Cytotoxic T cells, B cells and Memory B cell are components of adaptive/acquire immunity *Kuby's Immunology 6/e p53*
   - ❑ Components of Innate immunity- refer text.

8. **Ans. (d) (T cells)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p97, 2/e p104, Kuby's Immunology 6/e p53*
   - ❑ Alternate pathways of Complement , NK cells and Macrophages (as phagocytes) are the components of innate immunity
   - ❑ B cell, T cell, Classical complement and Antigen presenting cells are components of adaptive/acquired immunity.

9. **Ans. (a) (b) (e) (Toxoids, Subclinical infection, Antigen exposure)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p102, 2/e p107, Ananthnarayan 9/e p81-82, 10/e p83*





- Active immunity can be induced by any substance that actively stimulates the immune system to produce antibody. Vaccines, toxoids, infection or antigen exposure can induce active immunity
- Antitoxin and immunoglobulins are, e.g. of Passive immunity.

**10. Ans. (c, d) (It can be given before disease occurrence, Can be transferred by antibodies from another Host)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p103, 2/e p109, Ananthanarayan 9/e p82-83, 10/e p84*

- Immunoglobulins can be given along with vaccine in postexposure prophylaxis, e.g. Rabies immunoglobulins
- Passive immunity last for days to months
- Immunoglobulins should be given immediately after the exposure but before the disease occurrence
- Passive immunity can be transferred between individuals by Serum therapy (antibodies)
- Passive immunity works immediately

**11. Ans. (a) (*Staphylococcus aureus*)** *Ref: Stite's Medical Immunology 10/e p152, Ref: Apurba Sastry's Essentials of Medical Microbiology 2/e p114, Ananthnarayan 9/e p93*

## ACUTE PHASE REACTANTS AND CRP

**12. Ans. (a) (18 hrs)** *Ref: Apurba Sastry's Essentials of Medical Microbiology 1/e p101, 2/e p107, C-reactive protein: a critical update, J Clin Invest. 2003.*

The plasma half-life of CRP is about 19 hours and is constant under all conditions of health and disease, so that the sole determinant of circulating CRP concentration is the synthesis rate.

# Review of MICROBIOLOGY & IMMUNOLOGY

***Salient Features***

- All the chapters have been updated with the latest epidemiology, laboratory diagnosis and treatment.
- Newer sterilization indicators, CSSD, newer automations (MALDITOF, VITEK, BacT/ALERT VIRTUO), newer molecular methods (LAMP, Real-time PCR, Biofire FilmArray, etc.) included.
- MRSA, VRSA, VRE, Pneumococcal vaccines, DPT vaccine, epidemiology of meningococcal meningitis, tuberculosis (leprosy), melioidosis, scrub typhus, yaws eradication program have been thoroughly updated.
- Laboratory diagnosis, treatment and PEP of HBV and HCV, HIV updated thoroughly.
- Nipah virus (including the Kerala outbreak, 2018), poliomyelitis (update in vaccine schedule, epidemiology and end game strategy) included.
- Hospital Infection Control chapter has been thoroughly updated with topics such as biomedical waste (2016 guideline, with 2018–2019 amendment), needle stick injury, antimicrobial stewardship and environmental surveillance.
- Recent questions included from PGI–2019 (Nov and May), AIIMS–2019 (Nov and May), JIPMER–2019 (Nov and May) and NEET 2020 Pattern Questions, DNB 2019 Pattern Questions, latest MCQs from other national and state entrances.
- Image-based question bank is strengthened with new images (more than 300 images are included). Images are incorporated in respective chapters.
- Updated with latest references from Harrison 19th and 20th ed., Apurba Sastry's Essentials of Medical Microbiology 2nd ed., Apurba Sastry's Essentials of Medical Parasitology 2nd ed., Apurba Sastry's Essentials of Hospital Infection Control, Park 24th ed., Jawetz 27th ed. and Anathanarayan 10th ed.
- Only MCQ book on Microbiology written by the subject specialist and textbook authors.
- **Each chapter contains:**
  - o Chapterwise concise complete text in a new layout for enabling the students to study antegrade manner and important points given in separate boxes.
  - o Chapterwise MCQs with detailed explanations given from previous year exams of National and State exams including DNB and NEET Pattern Questions.
- Hundred percent authentic and correct answers with the updated references.
- More emphasis is given to the sections like Immunology, Parasitology and Mycology.
- The book contains lot of tables, flowcharts, mnemonics which help the students for better recall.
- This book is useful for Interns, students preparing for entrance exams and for second professional students.
- For any queries, suggestions and feedback kindly join us at Facebook page jaypeeexamzone.

**Author's online support**

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