

## COMMUNITY MEDICINE

# Sensitivity vs specificity

Start with the denominator: who is already known to have disease?

| Feature            | Sensitivity                                  | Specificity                                       |
|--------------------|----------------------------------------------|---------------------------------------------------|
| Starting group     | People with disease                          | People without disease                            |
| Correct result     | True positive (TP)                           | True negative (TN)                                |
| Formula            | $TP / (TP + FN)$                             | $TN / (TN + FP)$                                  |
| Higher value means | Fewer false negatives                        | Fewer false positives                             |
| Example            | 90 of 100 diseased people test positive: 90% | 180 of 200 disease-free people test negative: 90% |

**MEMORY CUE**

Sensitivity starts with the **sick**. Specificity starts with those **without disease**.

**COMMON TRAP**

A positive result asks a different question:  $TP / (TP + FP)$ , the positive predictive value. It depends on prevalence. Sensitivity is not the probability of disease after a positive test.

**RECALL**

Of 80 people with disease, 20 test negative. What is the sensitivity?

## IMMUNOLOGY

# Active vs passive immunity

Did the person make the response, or receive ready-made antibodies?

| Feature     | Active immunity                   | Passive immunity                           |
|-------------|-----------------------------------|--------------------------------------------|
| Source      | Own immune response to an antigen | Antibodies transferred from another source |
| Onset       | Takes time to develop             | Immediate antibody availability            |
| Persistence | Usually longer lasting; varies    | Temporary as antibodies decline            |
| Memory      | Can generate immune memory        | Does not create recipient memory by itself |
| Examples    | Infection; vaccination            | Placental IgG; immune globulin             |

**MEMORY CUE**

Active = **your immune system acts**. Passive = **protection is passed to you**.

**COMMON TRAP**

An injection is not automatically a vaccine. A vaccine presents antigen; immune globulin provides antibodies. Both may be used in some exposure protocols for different purposes.

**RECALL**

Maternal IgG reaches a fetus through the placenta. Is this active or passive immunity?

## MICROBIOLOGY

# Exotoxin vs endotoxin

The key difference is composition, not simply whether toxin is outside a cell.

| Feature          | Exotoxin                                | Endotoxin                                  |
|------------------|-----------------------------------------|--------------------------------------------|
| Composition      | Protein toxin                           | LPS / LOS; lipid A is the toxic component  |
| Bacterial source | Gram-positive or Gram-negative bacteria | Gram-negative outer membrane               |
| Release          | Produced and released by bacteria       | Shed during growth and released with lysis |
| Heat             | Many are heat-labile; exceptions exist  | Relatively heat-stable                     |
| Toxoid use       | Selected toxins: tetanus, diphtheria    | Not used as a conventional toxoid          |

**MEMORY CUE**

Exotoxin = **protein product**. Endotoxin = **outer-membrane lipid A**.

**COMMON TRAP**

“All exotoxins are heat-labile” is false. Some are heat-stable. Endotoxin release also does not require bacterial death.

**RECALL**

Which part of lipopolysaccharide is responsible for its endotoxic activity?

## PATHOLOGY

# Iron deficiency vs beta-thalassemia trait

Both can cause microcytosis. The cause and supporting investigations differ.

| Feature         | Iron deficiency                              | Beta-thalassemia trait                       |
|-----------------|----------------------------------------------|----------------------------------------------|
| Core defect     | Insufficient iron for hemoglobin production  | Reduced beta-globin synthesis                |
| Ferritin        | Usually low; inflammation can mask depletion | Often normal unless iron deficiency coexists |
| RBC count       | Often reduced or not elevated                | Often relatively high despite microcytosis   |
| Blood film      | Microcytosis with variable cell size         | Marked microcytosis; target cells may occur  |
| Supporting test | Iron studies                                 | Hb analysis: HbA2 usually elevated           |

## MEMORY CUE

Small cells with **low iron stores**: think iron deficiency. Small cells with a **relatively high RBC count**: consider thalassemia trait.

## COMMON TRAP

The CBC pattern is a clue, not confirmation. Iron deficiency and thalassemia can coexist. Alpha-thalassemia trait may have normal HbA2.

## RECALL

Microcytosis, a relatively high RBC count and raised HbA2 suggest which condition?

Sources [6, 7] | Answers and references: page 7

## BIOCHEMISTRY + MEDICINE

# B12 vs folate deficiency

Both impair DNA synthesis and can cause megaloblastic anemia.

| Feature            | Vitamin B12 deficiency                               | Folate deficiency                           |
|--------------------|------------------------------------------------------|---------------------------------------------|
| Blood morphology   | Macro-ovalocytes; hypersegmented neutrophils         | Similar megaloblastic pattern               |
| Homocysteine       | Can be elevated                                      | Can be elevated                             |
| Methylmalonic acid | Usually elevated                                     | Usually normal                              |
| Neurological clue  | Neuropathy or posterior-column dysfunction may occur | Typical B12 neurological syndrome is absent |
| Key distinction    | Neurological disease can occur without anemia        | Blood findings alone may look similar       |

**MEMORY CUE**

**MMA points toward B12**; homocysteine can rise in either deficiency.

**COMMON TRAP**

MMA can rise with renal impairment, so it is not interpreted alone. Folate can improve blood counts while unrecognized B12-related neurological injury continues.

**RECALL**

Macrocytosis with raised homocysteine and normal MMA favors which deficiency?

## CLOSE THE NOTES

# Five clues. Five answers.

Original practice questions. These are not claimed as previous exam questions.

**01 / COMMUNITY MEDICINE**

Of 80 people with disease, 20 test negative. Calculate sensitivity.

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**02 / IMMUNOLOGY**

Maternal IgG crosses the placenta. Name the type of immunity.

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**03 / MICROBIOLOGY**

Identify the toxic component of bacterial lipopolysaccharide.

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**04 / PATHOLOGY**

Microcytosis + relatively high RBC count + elevated HbA2: what is the likely diagnosis?

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**05 / BIOCHEMISTRY**

Macrocytosis + elevated homocysteine + normal MMA: which deficiency is favored?

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**REVISION ROUTINE**

Answer without looking. Check page 7. Revisit only the missed comparisons, then test yourself again tomorrow.

## CHECK YOUR RECALL

# Answers + source notes

A free five-pair sample from the planned Medryva comparison series.

- 01** 75%. There are 60 true positives among 80 people with disease.
- 02** Natural passive immunity: transferred maternal antibodies.
- 03** Lipid A, the membrane-anchoring component of LPS.
- 04** Beta-thalassemia trait; confirm with appropriate hemoglobin analysis.
- 05** Folate deficiency is favored by this pattern; interpret with clinical context.

## REFERENCES

1. CDC: Diagnostic Sensitivity and Specificity
2. Monaghan et al.: Diagnostic test measures (2021)
3. CDC Pink Book: Principles of Vaccination
4. Medical Microbiology: Bacterial Pathogenesis
5. StatPearls: Biochemistry, Lipopolysaccharide
6. Merck Manual: Characteristics of Common Anemias
7. Merck Manual: Thalassemias
8. Merck Manual: Megaloblastic Macrocytic Anemias

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**More comparisons and corrections: @medryva**