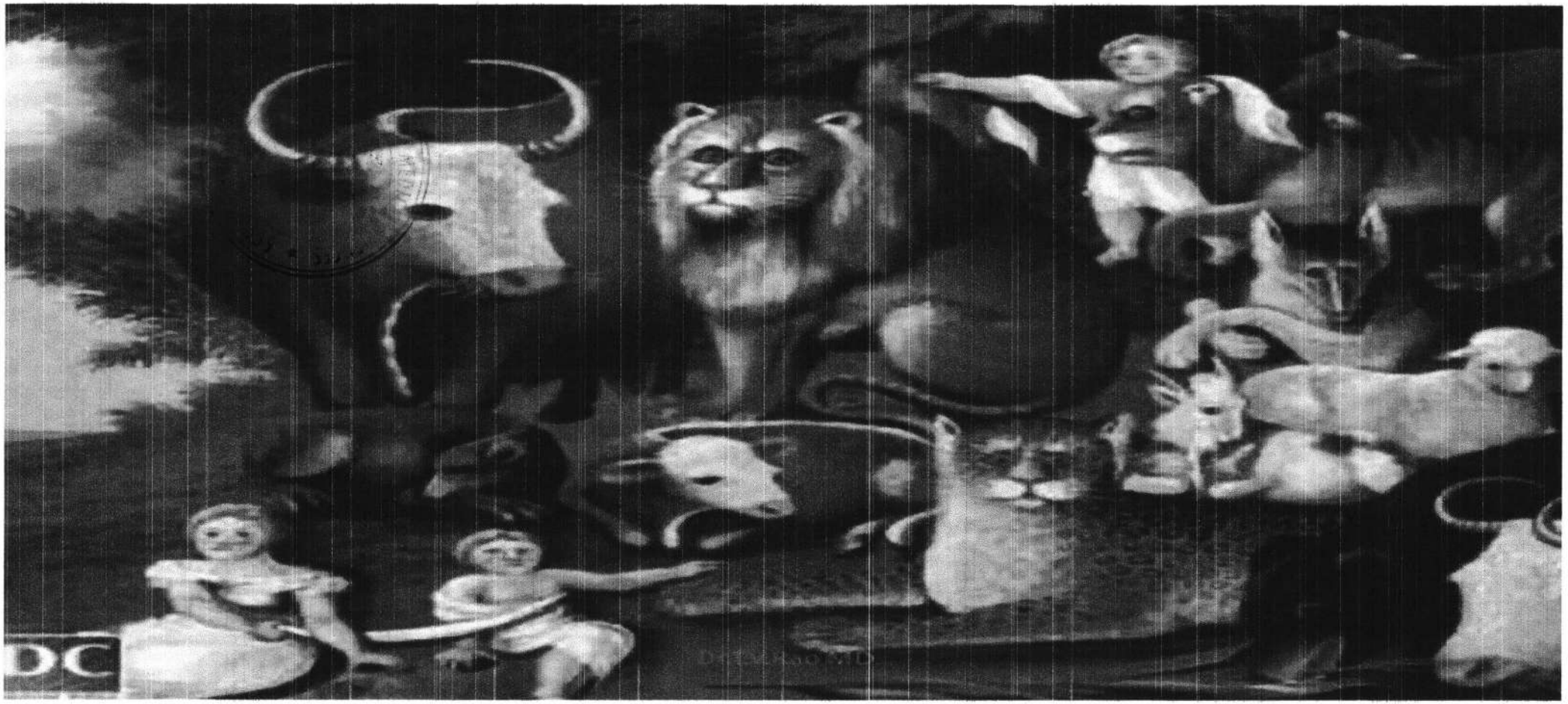
BRUCELLOSIS

Dr. Iswarya Babu P



INTRODUCTION

CLASSIFICATION

PATHOGENESIS

MODE OF TRANSMISSION

CLINICAL MANIFESTATIONS

LABORATORY DIAGNOSIS

TREATMENT

PREVENTION

NTRODUCTION

Sir David Bruce -1886 – Malta island – **MALTA FEVER**

Undulant fever

Zoonotic ds – sheep, goat, cattle

Occupational or domestic exposure

Highly contagious febrile illness - **BRUCELLOSIS**

BRUCELLA – Obligate aerobe

fastidious, **GNCB**

SYNONYMS

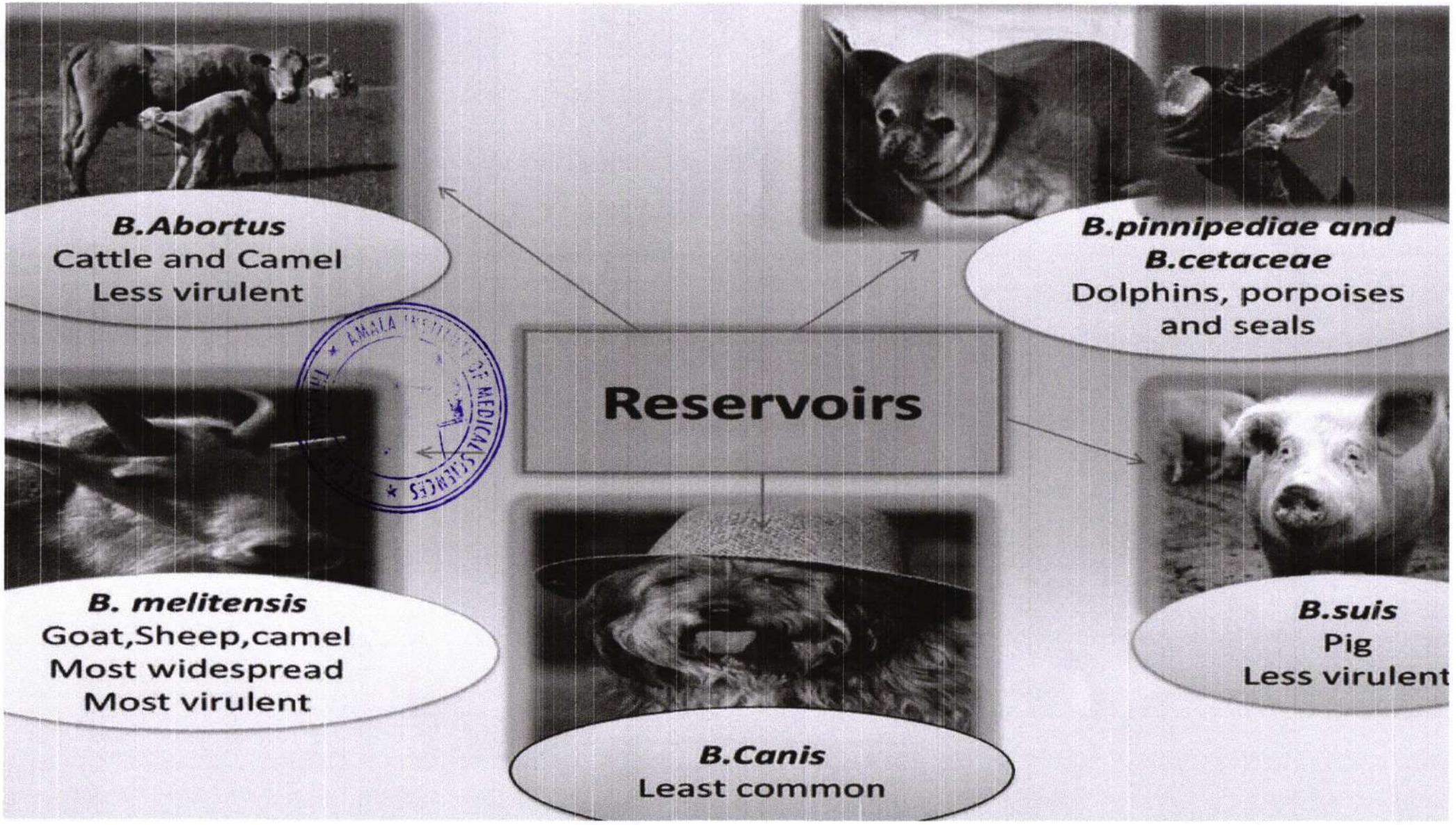
In humans

- Undulant fever
- Malta fever
- Mediterranean Fever
- Rock Fever of Gibraltar

In animals

- Epizootic Abortion
- Contagious Abortion
- Bang's Disease

CLASSIFICATION



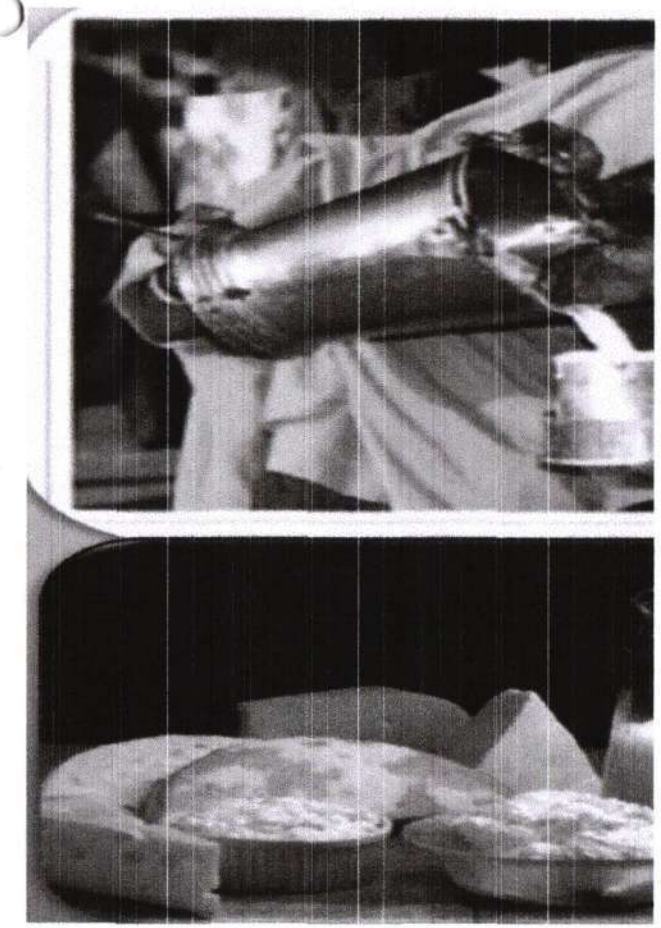
PATHOGENESIS

TRANSMISSION - Food borne

Direct contact

Air borne

Person to person



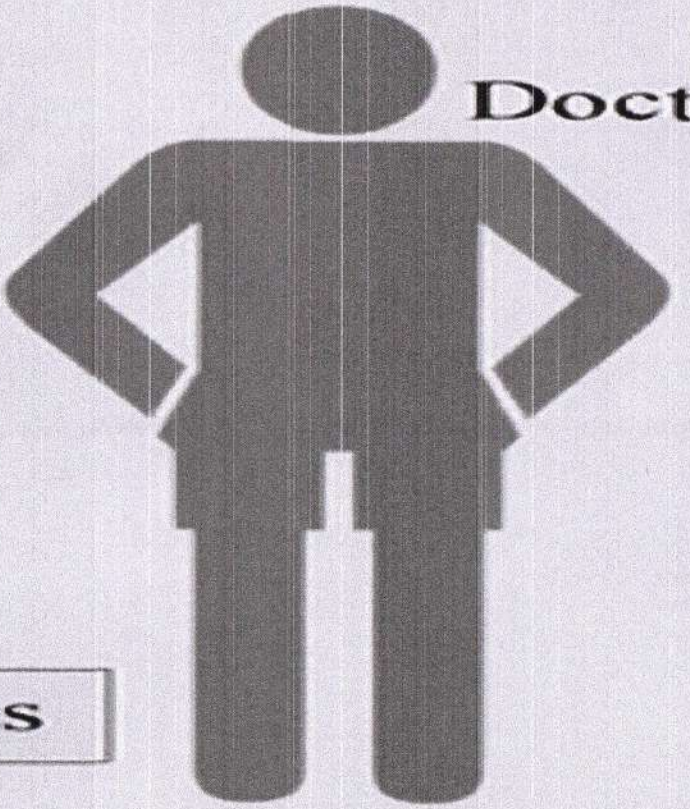
SPREAD – **Blood stream** – disseminates to various organs

ORGANS – RE sys, LN, Spleen, Liver, Bone marrow
musculoskeletal sys, GUT

Brucellosis Spread

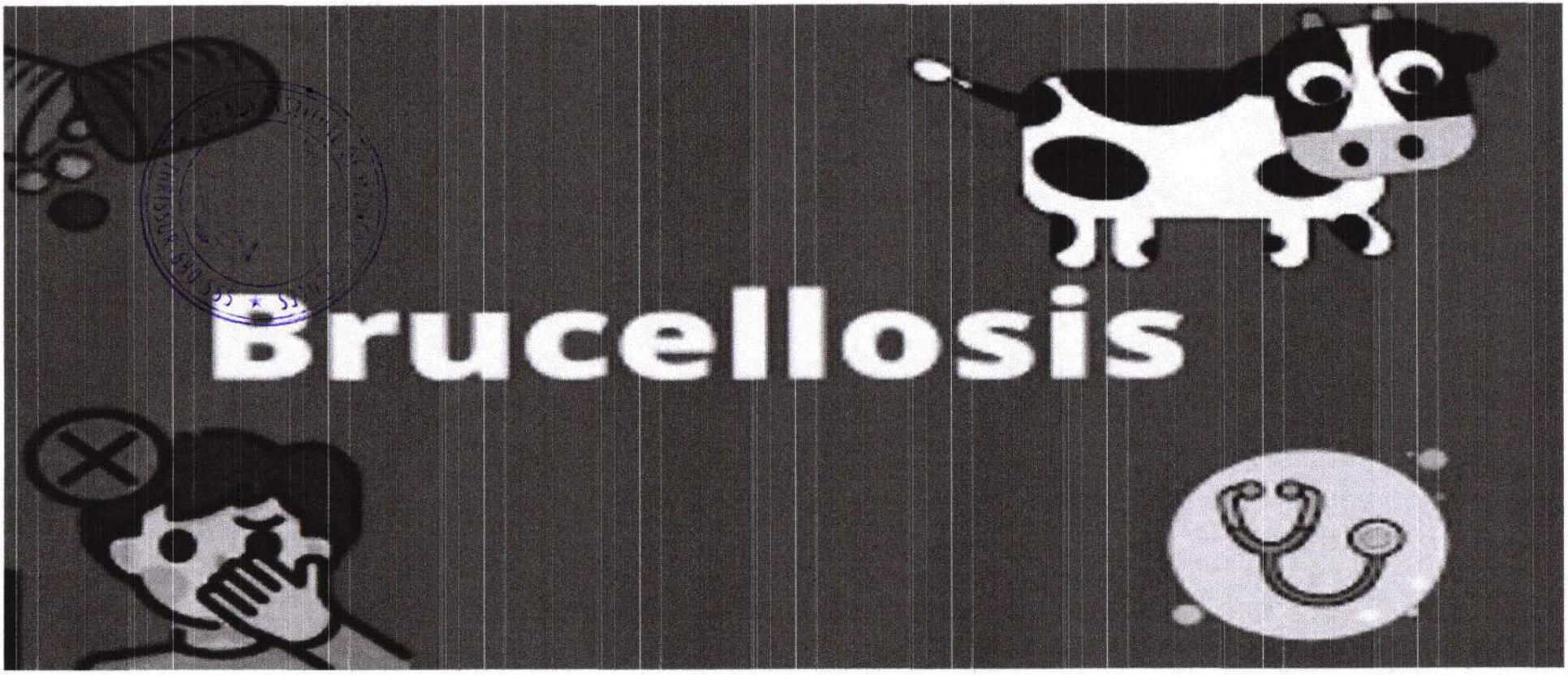
Brucellosis

Spread

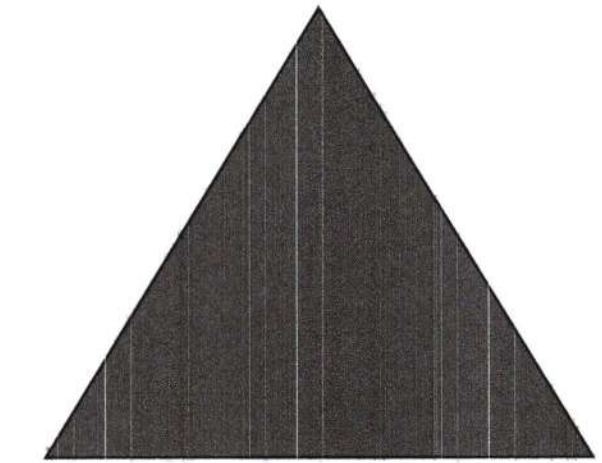


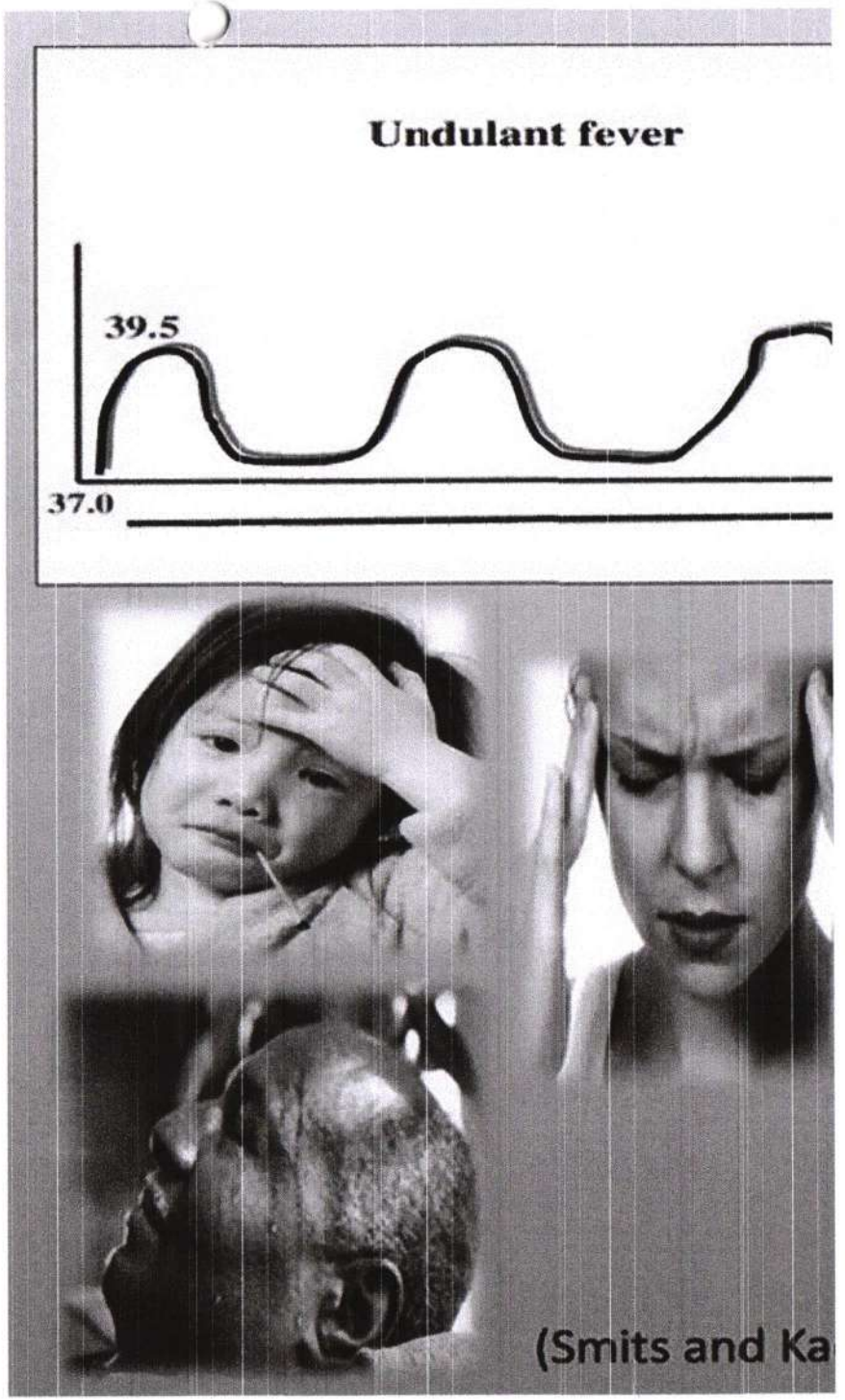
1. Milk
2. Raw Diary products
3. Direct contact (Butcher and farmers)
4. Meat-processing industry
5. Veterinary doctors

CLINICAL MANIFESTATIONS



CLASSICAL TRIAD





Typhoid like illness

CNS

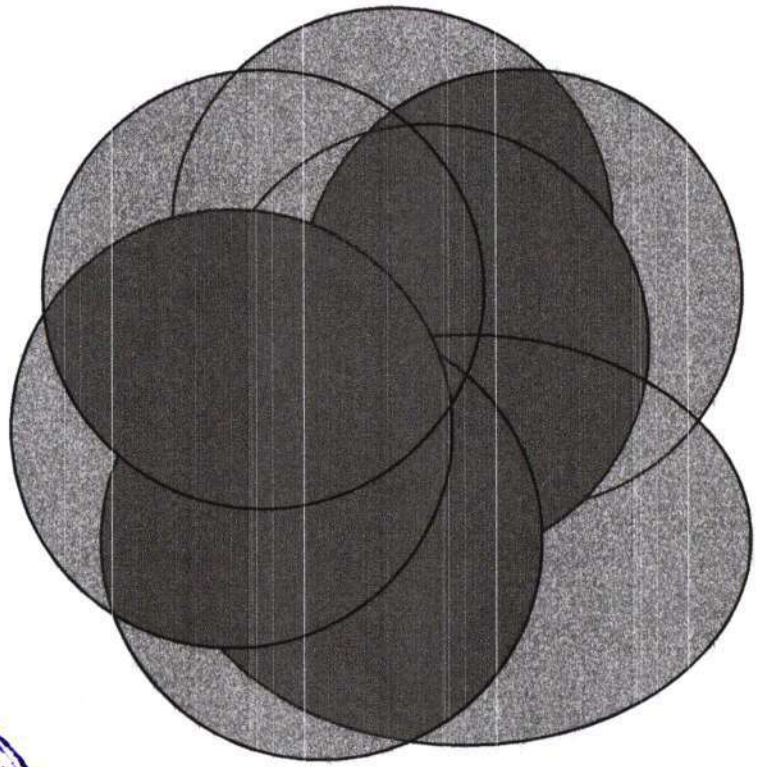
Depression & lethargy with meningitis

Non specific sym

Cough, Pharyngitis

Abdominal pain,

Headache, Diarrhea, Rash, Weakness



Undulating fever

– in between febrile period ; afebrile periods

Muskuloskeletal symptoms –

Vertebral Osteomyelitis

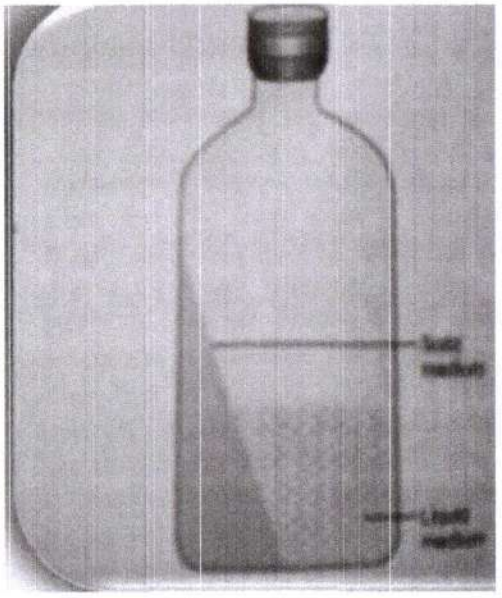
Septic Arthritis

;ABORATORY DIAGNOSIS

CULTURE

Specimen – blood, BM, CSF, joint fluid, other tissues

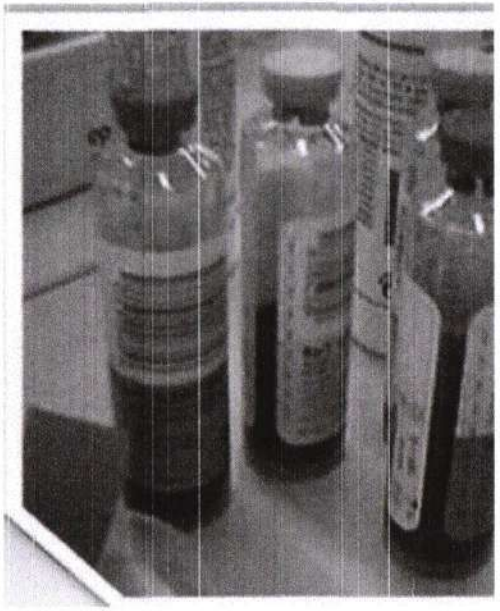
Blood – Multiple sample – 8-10ml/bottle, 2-3 times during febrile period, b4 Abs

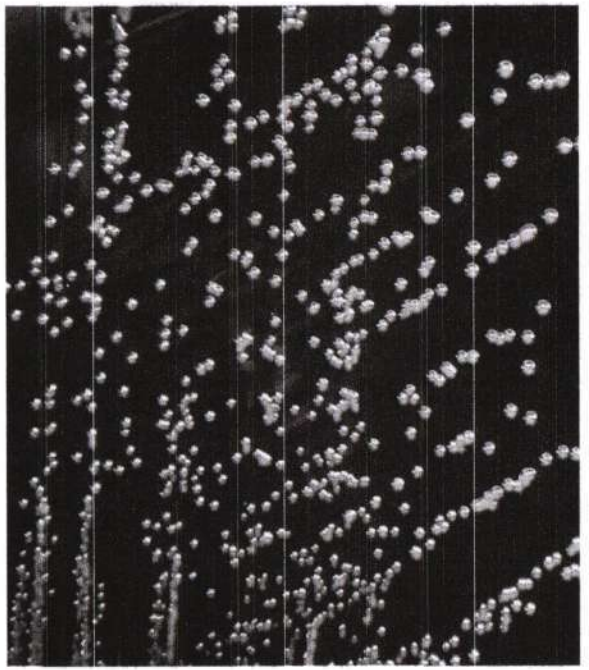


;one marrow > Blood – High yield

ıutomated blood culture – BacT/Alert

/ALDI – TOF or VITEK





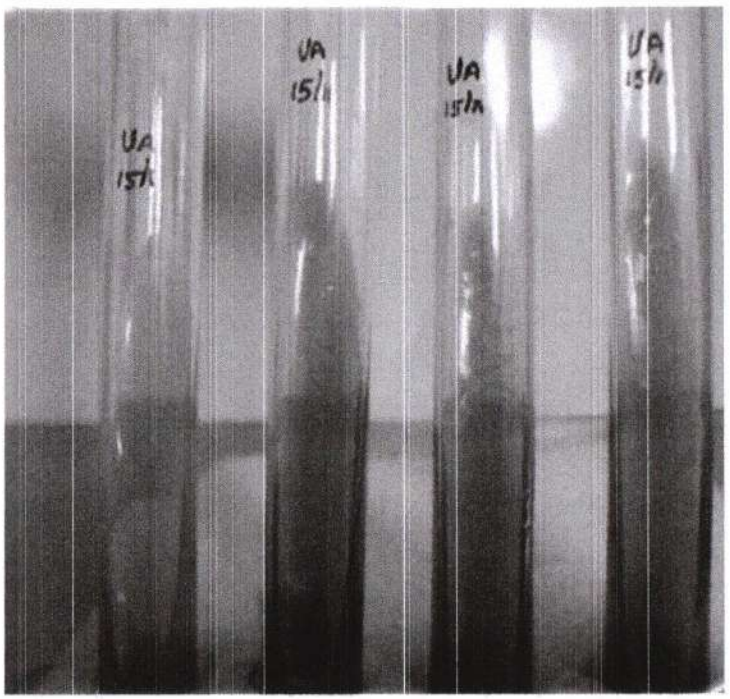
BLOOD AGAR – Small smooth transparent non hemolytic colonies

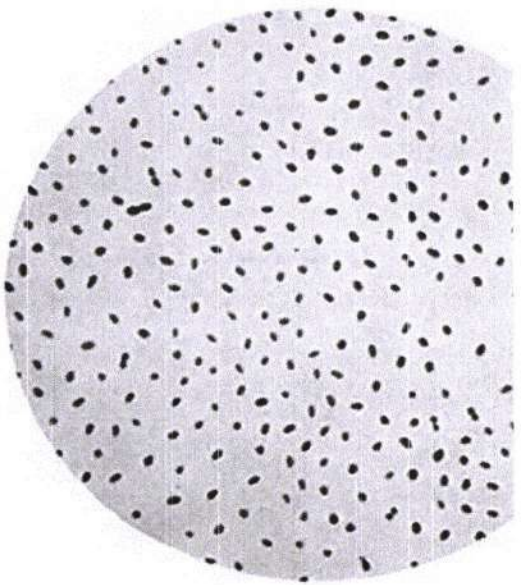
Gram stain – **GNCB**

Non motile

Catalase + ve

Rapid urease positive





SEROLOGY

SAT – GOLD STANDARD TEST

Tube agg test

Detects IgM, IgG

Titre 1:160 - non endemic area

1:320 - endemic areas

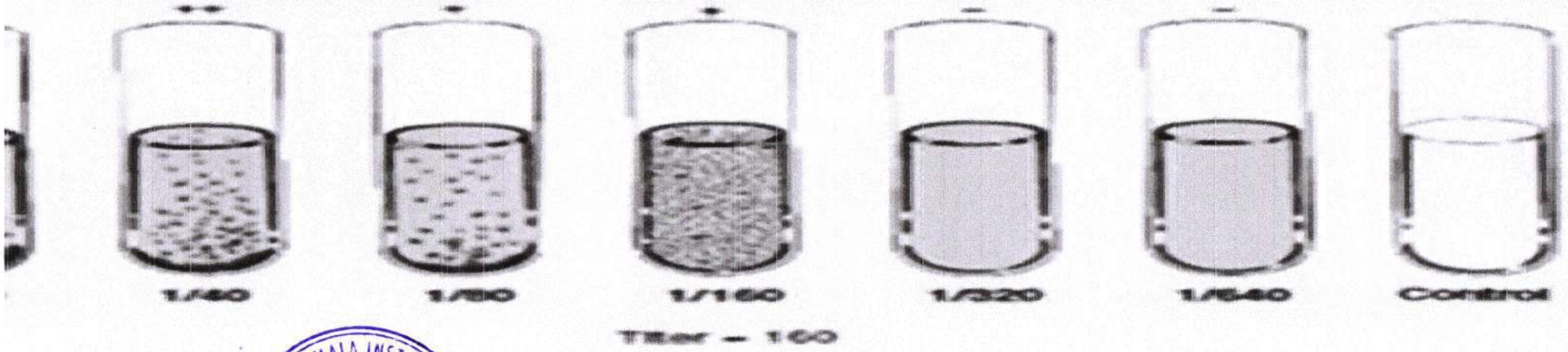
2ME SAT Test – Destroys IgM

Detects IgG – **Chronic Brucellosis**

False –ve – Prozone

Blocking Abs

False +ve - Other GNB infections



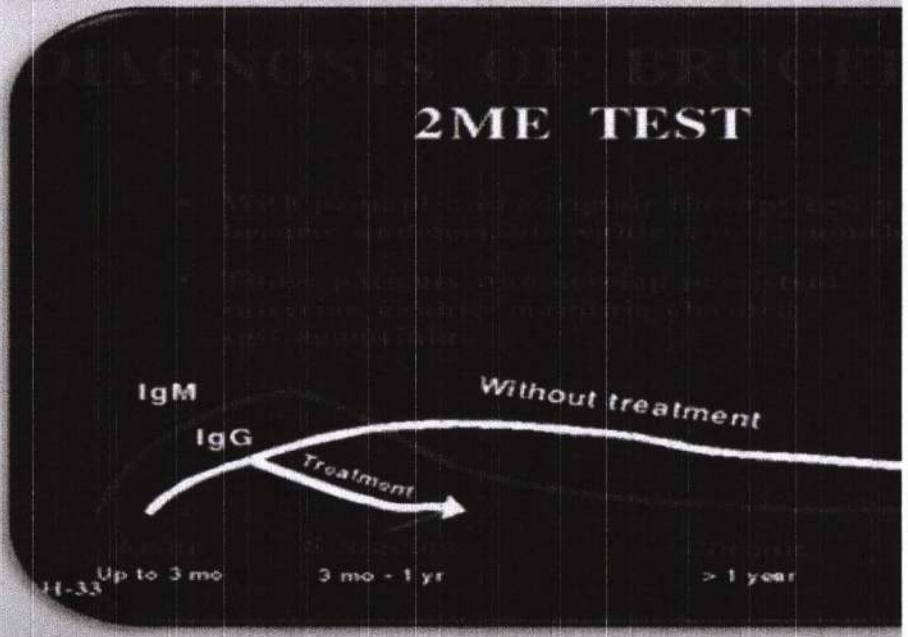
Titer – 160

- ✓ Acute brucellosis is most likely to be associated with a titer above 1:160
- ✓ A great majority of patients wi have titers of 1:160 to 1:320
- ✓ 4 fold rise titer in convalescer sera

2-Mercaptoethanol test (2ME)

lercaptoethanol disrupts -
sulfide bonds of IgM .Hence only
G is detected
seful for specific detection of IgG
nd titers higher than 1:80 are
ggestive of active infection.
gh IgG antibody titer or a titer
at is higher after treatment
ggests relapse or persistent
fecton.
ME test will be negative if STA test
really negative
ne prognosis of acute brucellosis
ay be predicted from the fall of
ME.

(Al-Anazi KA and Al-Jasser AM)



Merits:

- The test helps to confirm the stage of the disease.
- more specific
- +ve. in chronic Brucellosis

ELISA - 2nd MC Serology method

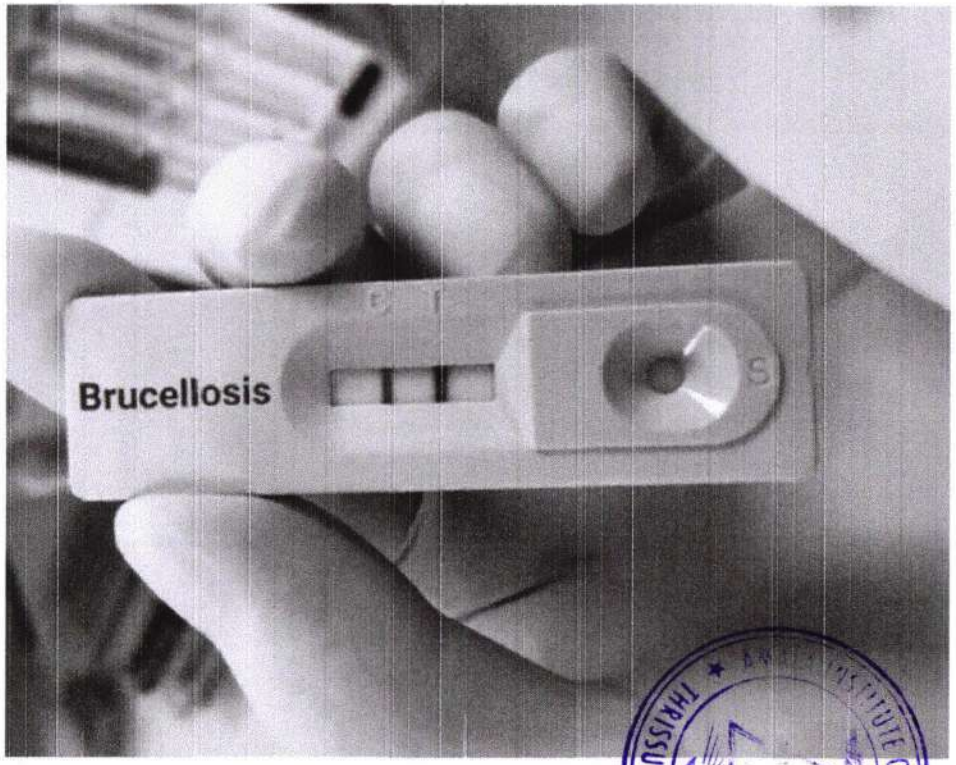
- Sensitivity 100%
- Uses **cytoplasmic protein or LPS Ag**
- Detects **IgM, IgG, IgA Abs individually**.

Dipstick Assays – anti Brucella IgM

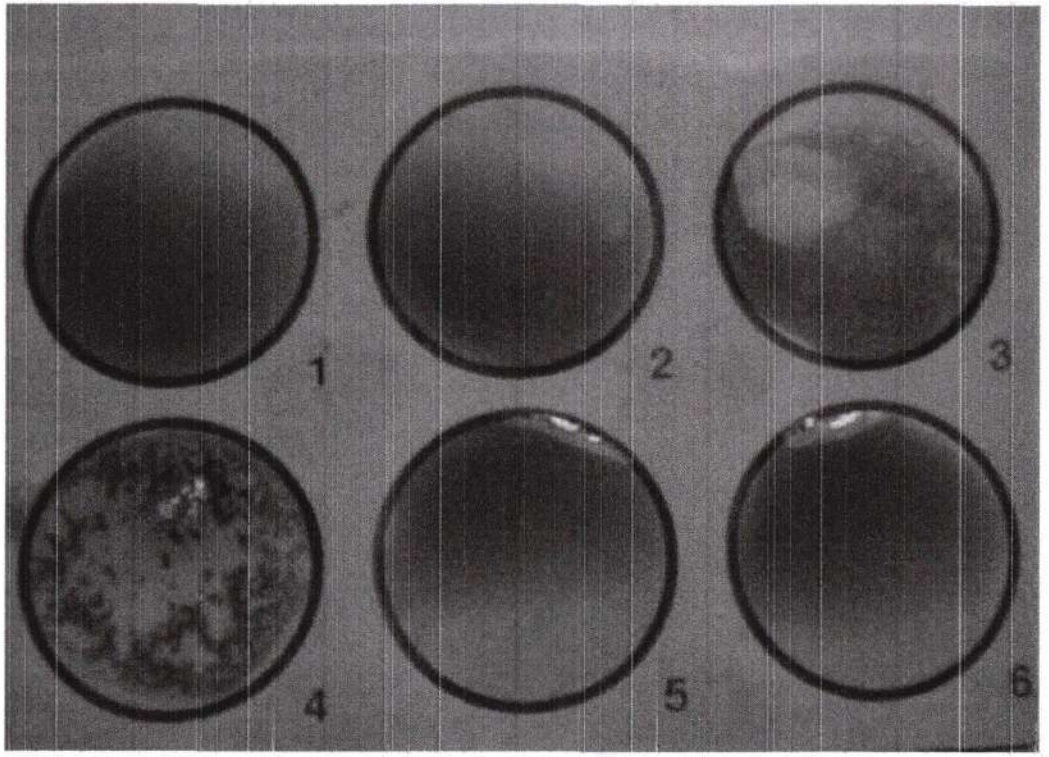
PCR Assay – rapid, sensitive & specific

species identification

RAPID CARD TEST



ROSE BENGAL CARD TEST IN MILK



TREATMENT

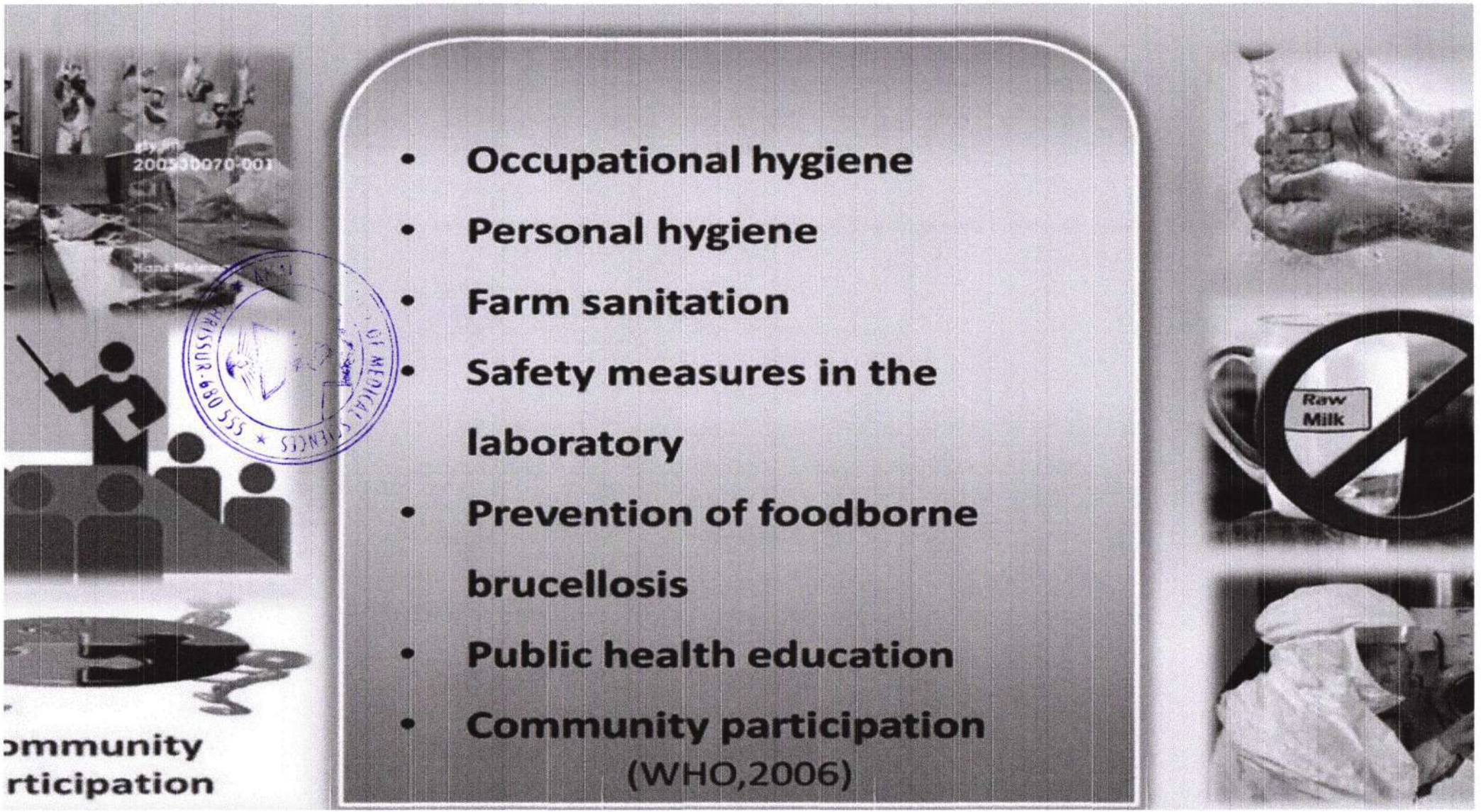
Standard Regimen - [redacted]

Alternative to Gen - **Streptomycin**

WHO Regimen - [redacted]

[redacted]

REVENTION


Occupational hygiene
Personal hygiene
Farm sanitation
Safety measures in the laboratory
Prevention of foodborne brucellosis
Public health education
Community participation
(WHO,2006)
Raw Milk
ommunity
rticipation

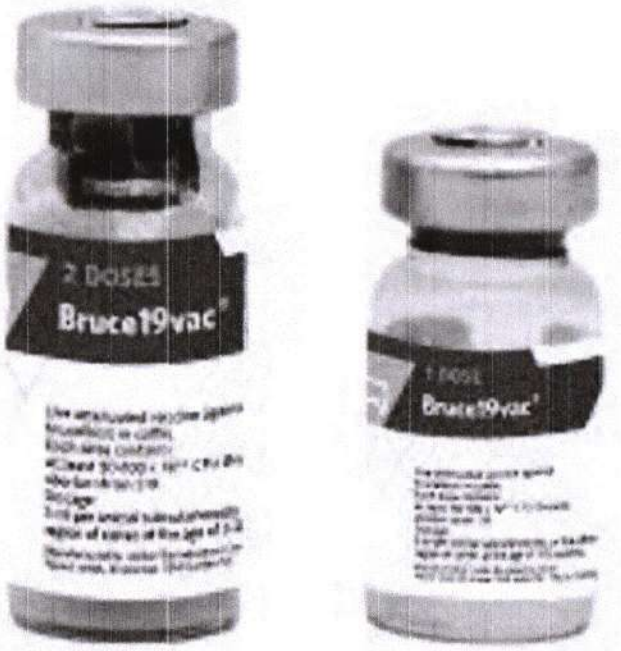
'ACCINE

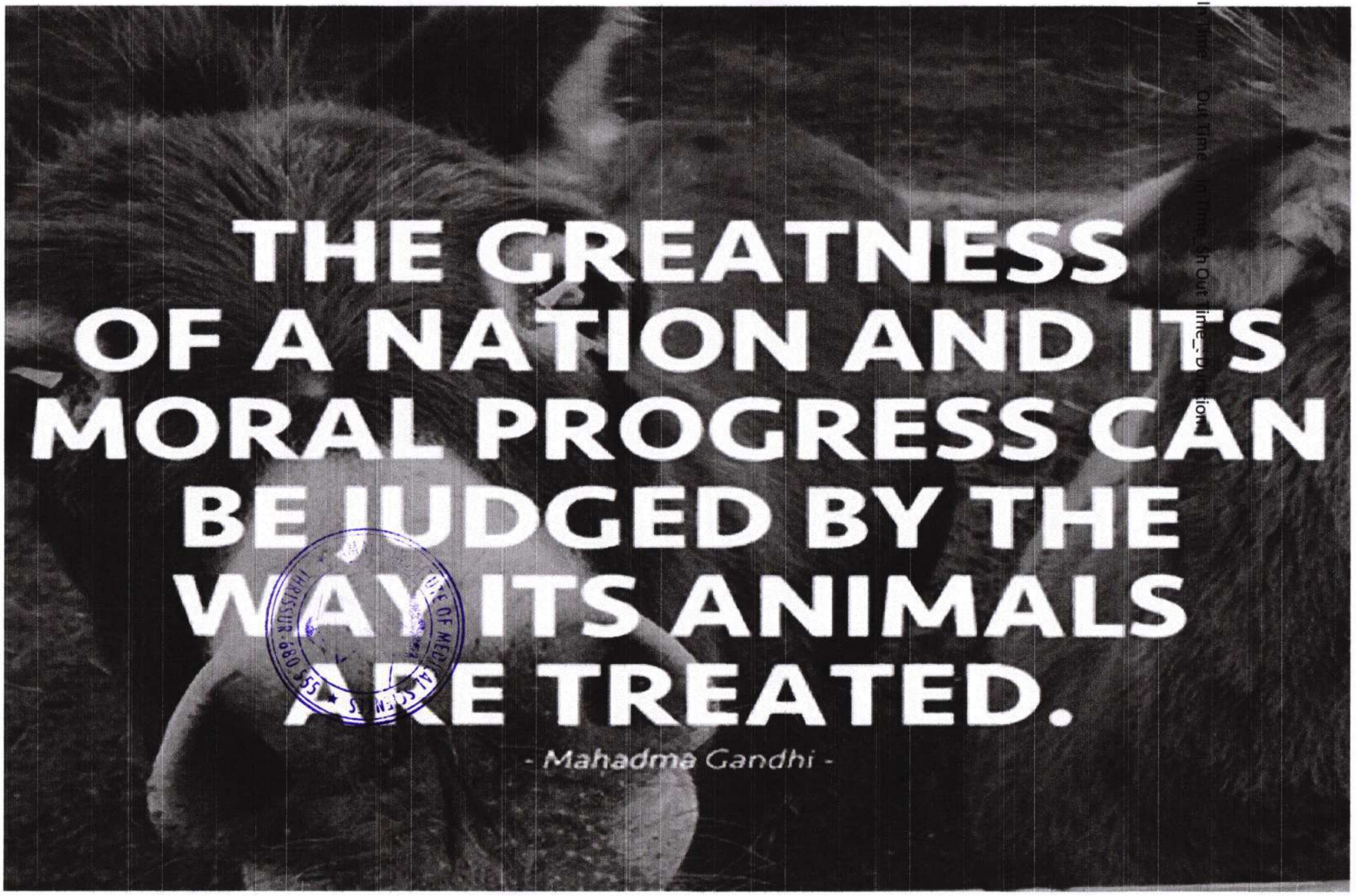
\V using **B.abortus 19 strain -** cattle

melitensis rev -1 strain – sheep & goat

)-BA

LAV using B.abortus - Human




THE GREATNESS OF A NATION AND ITS MORAL PROGRESS CAN BE JUDGED BY THE WAY ITS ANIMALS ARE TREATED.
- Mahadma Gandhi -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Dank U
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Grazie
Thank you
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Takk
Gracias
Dziękuję
Děkuju
danke
Kiitos

Histopathology Techniques

Dr. Dennis John
Senior Resident
Dept. Of Pathology

Introduction

- Histological technique deals with the preparation of tissue for microscopic examination.
- The aim of good histological technique to preserve microscopic anatomy of tissue.
- Make them hard so that very thin section (4 to 5 micron) can be made.

Introduction

- Good staining should be possible.
- After staining, the section should represent the anatomy of the tissue as close to as possible to their structure in life.
- This is achieved by passing the total as selected part of the tissue through a series of process.

Techniques

- **These techniques are:**

1. Fixation
2. Dehydration
3. Cleaning
4. Embedding
5. Cutting
6. Staining

Fixation

- This is the process by which the constituents of cells and tissue are fixed in a physical and chemical state so that they will withstand subsequent treatment with various reagents with minimum loss of architecture .This is achieved by exposing the tissue to chemical compounds, call fixatives.

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Mechanism of action of fixatives

- Most fixatives act by denaturing or precipitating proteins which then form a sponge or meshwork, tending to hold the other constituents.

Contin.....

- Good fixative is most important factors in the production of satisfactory results in histopathology.
- Following factors are important:

- Fresh tissue
- Proper penetration of tissue by fixatives
- Correct choice of fixatives

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Contin....

- No fixative will penetrate a piece of tissue thicker than 1 cm.
- For dealing with specimen thicker than this, following methods are recommended:

1. Solid organ:

Cut slices as necessary as but not thicker than 5 mm.

.

Continu....

2. Hollow organ:

Either open or fill with fixative or pack lightly with wool soaked in fixative.

3. Large specimen:

It requires dissection, Inject fixative along the vessels or bronchi as in case of lung so that it reaches all parts of the organs.

Properties of an Ideal Fixative

- Prevents autolysis and bacterial decomposition.
- Preserves tissue in their natural state and fix all components.
- Make the cellular components insoluble to reagent used in tissue processing.
- Preserves tissue volume.

Properties of an Ideal Fixative

- Avoid excessive hardness of tissue.
- Allows enhanced staining of tissue.
- Should be non-toxic and non-allergic for user.
- Should not be very expensive.

Temperature

- The fixation can be carried out at room temperature.
- Tissue should not be frozen once it has been placed in the fixative solution, for a peculiar ice crystals distortion will result.

Speed of fixation

- The speed of fixation of most fixative is almost 1 mm/hour.
- Therefore, a fixation time of several hours is needed for most specimens.

Amount of fixative fluid

- This should be approximately 10-20 times the volume of the specimen.
- Fixative should surround the specimen on all sides.

Factor affecting fixation

- Size and thickness of piece of tissue.
- Tissue covered by large amount of mucous fix slowly.
- Tissue covered by blood or organ containing very large amount of blood also fix slowly.
- Fatty and lipomatous tissue fix slowly.
- Fixation is accelerated by agitation.
- Fixation is accelerated by maintaining temperature around 60oc.

Classification of Fixatives

- Classified into three categories.

1. Tissue fixatives
2. Cytological fixatives
3. Histochemical fixatives

Tissue fixatives

- There are many tissue fixatives i.e
  - Buffered formalin
  - Buffered gluteraldehyde
  - Zenker's formal saline
  - Bowen's fluid

Cytological fixatives

- Cytological fixatives are

- Ethanol
- Methanol
- Ether

Histochemical fixatives

- These are

- Formal saline
- Cold acetone
- Absolute alcohol

Tissue Processing

- In order to cut thin sections of the tissues, it should have suitable hardness and consistency when presented to the knife edge. These properties can be imparted by infiltrating and surrounding the tissue with paraffin wax, colliding or low viscosity nitrocellulose, various types of resins or by freezing. This process is called tissue processing

Continu....

- It requires 24hours and done in many stages.
- It can be subdivided into

a) dehydration
b) clearing
c) impregnating
d) embedding.

Note:

It is important that all specimens are properly labeled before processing is started.

Types of tissue processing

- There are two types

1. Manual Tissue Processing:

2. Mechanical Tissue Processing:

Manual Tissue Processing

- In this process the tissue is changed from one container of reagent to another by hand.

Mechanical Tissue Processing

- In this the tissue is moved from one jar to another by mechanical device.
- Timings are controlled by a timer which can be adjusted in respect to hours and minutes.
- Temperature is maintained around 60oC.

Note:

The processing, whether manually or mechanically, involves the same steps and reagents in same sequence.

Sequence of tissue processing

- **Dehydration:**
- Tissues are dehydrated by using increasing strength of alcohol; e.g. 50%, 70%, 90% and 100%.
- The duration for which tissues are kept in each strength of alcohol depends upon the size of tissue, fixative used and type of tissue.
- Delicate tissue will get high degree of shrinkage by two great concentration of alcohol.
- The volume of alcohol should be 50-100 times that of tissue.

Clearing

- During dehydration water in tissue has been replaced by alcohol.
- The next step alcohol should be replaced by paraffin wax.
- As paraffin wax is not alcohol soluble, we replace alcohol with a substance in which wax is soluble. This step is call clearing.

Continu....

- Clearing of tissue is achieved by any of the following reagents:

- Xylene
- Chloroform
- Benzene
- Carbon tetrachloride
- Toluene

Note:

- Xylene is commonly used. Small piece of tissue are cleaned in 0.5 – 1 hour; whereas larger (5cm or more thick) are cleaned in 2-4 hours.

Impregnation with Wax

- This is allowed to occur at melting point temperature of paraffin wax, which is 54-60oC. Volume of wax should be about 25-30 times the volume of tissues.
- The duration of impregnation depends on size and types of tissues and the clearing agents employed.
- Longer periods are required for larger pieces and also for harder tissue like bones and skin as compared to liver kidney, spleen,lung etc.

Continu....

- Total duration of 4 hours is sufficient for routine impregnation.
- **Types of Wax employed for Impregnation**:

1. Paraffin wax
2. Water soluble wax
3. Other material, like colloidin, gelatin, paraplast etc.

- Paraffin wax is used routinely. It has hard consistency, so section of 3-4 micron thickness can be cut.

Embedding

- Impregnated tissues are placed in a mould with their labels and then fresh melted wax is poured in it and allowed to settle and solidify.
- Once the block has cooled sufficiently to form a surface skin it should be immersed in cold water to cool it rapidly.
- After the block has completely cooled it is cut into individual blocks and each is trimmed.
- Labels are made to adhere on the surface of the block by melting the wax with a metal strips sufficiently wormed.

Microtomy

- For *light microscopy*, a glass knife mounted in a microtome is used to cut 4-6 um-thick tissue sections which are mounted on a glass microscope slide.
- For transmission *electron microscopy*, a diamond knife mounted in an ultramicrotome is used to cut 50-nm-thick tissue sections which are mounted on a 3-mm-diameter copper grid. Then the mounted sections are treated with the appropriate *stain.*
- Frozen tissue embedded in a freezing medium is cut on a microtome in a cooled machine called a *cryostat.*

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Staining

- Staining is a process by which we give colour to a section.
- There are hundreds of stainsn available.

- ***Classification of Stains:***

- Acid stains
- Basic stains
- Neutral stains

Acid Dyes

- In an acid dye the basic component is colored and the acid component is colorless.
- Acid dyes stain basic components e.g. eosin stains cytoplasm.
- The color imparted is shade of red.

Basic Dyes

- In a basic dye the acid component is colored and the basic component is colorless.
- Basic dyes stain acidic components e.g. basic fuchsin stains nucleus.
- The color imparted is shade of blue.

Neutral Dyes

- When an acid dye is combined with a basic dye a neutral dye is formed.
- As it contains both colored radicals, it gives different colors to cytoplasm and nucleus simultaneously.
- This is the basis of Leishman stain.

Special stains

- When a specific components of tissue e.g. fibrous tissue, elastic tissue, nuclear material is to be stained, certain special stains are used which specifically stain that component tissue.

Procedure of staining:

- There are two types of staining,
- Manual Staining
- Automatic staining

Manual Staining

- In a small laboratory when a few slides are stained daily, this is the method of choice.
- Although it is time consuming it is economical.
- Different reagent containers are placed in a special sequence and the slides are removed from one container to another manually.

Automatic staining

- In this procedure an automatic stainer is required.
- It has a timer, which controls the time.
- It has a mechanical device which shifts the slides from one container to next after the specified time.
- Advantages of automated stainer are:
- It reduces the man power
- It controls the timing of staining accurately
- Large number of slides can be stained simultaneously
- Less reagents are used
- **Note:**

  Slides stained either manually or by automatic stainer, pass through same sequences.

How it works?

- Hematoxylin
  - **has a deep blue-purple color** and
  - stains nucleic acids by a complex, incompletely understood reaction.
  - nuclei are stained blue,

- Eosin
  - **is pink and stains proteins** nonspecifically.
  - cytoplasm and extracellular matrix have varying degrees of pink staining.
  - **if abundant polyribosomes** are present, the cytoplasm will have a distinct blue cast.

- Well-fixed cells show considerable intranuclear detail. Nuclei show varying cell-type- and cancer-type-specific patterns of condensation of heterochromatin (hematoxylin staining) that are diagnostically very important.

Betsy

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Introduction to Endocrine Pancreas

- **Major cells**

❑ ***β cells*** :- Insulin → regulates glucose utilization in tissues and reduces blood glucose levels

❑ ***α cells*** :- Glucagon → stimulates glycogenolysis in the liver & increases blood sugar

❑ ***δ cells*** :- Somatostatin → suppresses both insulin and glucagon release

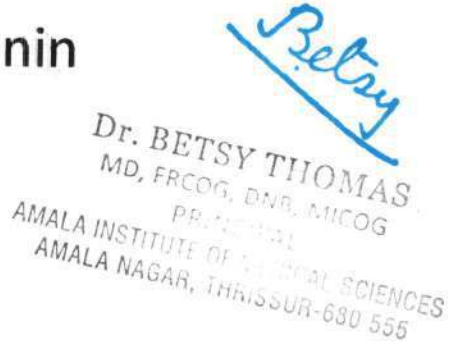
❑ ***Pancreatic Polypeptide cells (PP)*** :- Pancreatic Polypeptide → stimulation of secretion of gastric and intestinal enzymes and inhibition of intestinal motility

Introduction to Endocrine Pancreas

- **Minor cells**

❑ **D1 cells:-** Vasoactive Intestinal Polypeptide → induces glycogenolysis and hyperglycemia, also stimulates gastrointestinal fluid secretion and causes secretory diarrhea

❑ ***Enterochromaffin cells :-*** Serotonin



Diabetes Mellitus

Definition

" Diabetes mellitus is a group of metabolic disorders sharing the common feature of hyperglycemia "

Robbins

Epidemiology

- According to WHO → 346 million people suffer from diabetes worldwide

- India and China → largest contributors

- Middle & low income nations → 80% - diabetes related deaths

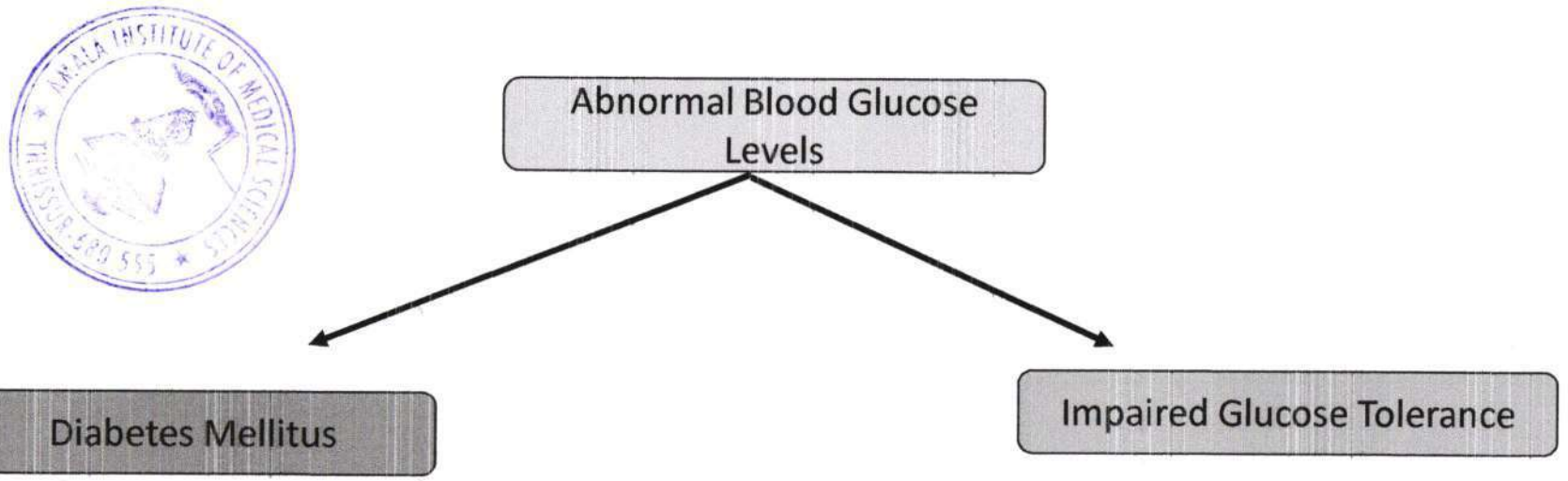
Etiology

- Autoimmune causes
- Sedentary lifestyle
- Poor eating habits

Diabetes & Obesity → Diabesity Epidemic

Diagnosis

- Normal Blood sugar level → 70 – 120 mg/dl (fasting)



Diagnostic Criteria for DM

- According to ADA & WHO:-

✓ *A fasting plasma glucose ≥ 126 mg/dL*

✓ *A random plasma glucose ≥ 200 mg/dL (in a patient with classic hyperglycemic signs)*

✓ *2-hour plasma glucose ≥ 200 mg/dL during an oral glucose tolerance test (OGTT) with a loading dose of 75 gm*

✓ *A glycated hemoglobin (HbA1C) level ≥ 6.5%*

Diagnostic Criteria for IGT

- ✓A fasting plasma glucose between 100 and 125 mg/dL ("impaired fasting glucose")
- ✓ 2-hour plasma glucose between 140 and 199 mg/dL following a 75-gm glucose OGTT
- ✓A glycated hemoglobin (HbA1C) level between 5.7% and 6.4%

Diagnosis

- All tests except Random Blood Sugar repeated & confirmed on separate day

- If discordance in values in two assays → test with greater abnormality considered as 'readout'

Classification

Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)

Immune-mediated
Idiopathic

Type 2 diabetes (combination of insulin resistance and β-cell dysfunction)

Genetic defects of β-cell function

Maturity-onset diabetes of the young (MODY), caused by mutations in:
- Hepatocyte nuclear factor 4α (*HNF4A*), MODY1
- Glucokinase (*GCK*), MODY2

Hepatocyte nuclear factor 1α (*HNF1A*), MODY3
- Pancreatic and duodenal homeobox 1 (*PDX1*), MODY4
- Hepatocyte nuclear factor 1β (*HNF1B*), MODY5
- Neurogenic differentiation factor 1 (*NEUROD1*), MODY6

Neonatal diabetes (activating mutations in *KCNJ11* and *ABCC8*, encoding Kir6.2 and SUR1, respectively)
Maternally inherited diabetes and deafness (MIDD) due to mitochondrial DNA mutations (m.3243A→G)
Defects in proinsulin conversion
Insulin gene mutations

Genetic defects in insulin action

Type A insulin resistance
Lipoatrophic diabetes

Exocrine pancreatic defects

Chronic pancreatitis
Pancreatectomy/trauma
Neoplasia
Cystic fibrosis
Hemochromatosis
Fibrocalculous pancreatopathy

Endocrinopathies

Acromegaly
Cushing syndrome
Hyperthyroidism
Pheochromocytoma
Glucagonoma

Infections

Cytomegalovirus
Coxsackie B virus
Congenital rubella

Drugs

Glucocorticoids
Thyroid hormone
Interferon-α
Protease inhibitors
β-adrenergic agonists
Thiazides
Nicotinic acid
Phenytoin (Dilantin)
Vacor

Genetic syndromes associated with diabetes

Down syndrome
Klinefelter syndrome
Turner syndrome
Prader-Willi syndrome

Gestational diabetes mellitus

Classification

- ***Type 1 DM :-*** autoimmune disease characterized by pancreatic β cell destruction and an absolute deficiency of insulin

- ***Type 2 DM:-*** combination of peripheral resistance to insulin action and an inadequate secretory response by the pancreatic β cells ("relative insulin deficiency")

➢ Classification

Table 24-7 Type 1 Versus Type 2 Diabetes Mellitus

Type 1 Diabetes Mellitus	Type 2 Diabetes Mellitus
Clinical	
Onset: usually childhood and adolescence	Onset: usually adult; increasing incidence in childhood and adolescence
Normal weight or weight loss preceding diagnosis	Vast majority are obese (80%)
Progressive decrease in insulin levels	Increased blood insulin (early); normal or moderate decrease in insulin (late)
Circulating islet autoantibodies (anti-insulin, anti-GAD, anti-ICA512)	No islet autoantibodies
Diabetic ketoacidosis in absence of insulin therapy	Nonketotic hyperosmolar coma more common
Genetics	
Major linkage to MHC class II genes; also linked to polymorphisms in *CTLA4* and *PTPN22*, and insulin gene VNTRs	No HLA linkage; linkage to candidate diabetogenic and obesity-related genes (*TCF7L2*, *PPARG*, *FTO*, etc.)
Pathogenesis	
Dysfunction in T cell selection and regulation leading to breakdown in self-tolerance to islet autoantigens	Insulin resistance in peripheral tissues, failure of compensation by β-cells
	Multiple obesity-associated factors (circulating nonesterified fatty acids, inflammatory mediators, adipocytokines) linked to pathogenesis of insulin resistance
Pathology	
Insulitis (inflammatory infiltrate of T cells and macrophages)	No insulitis; amyloid deposition in islets
β-cell depletion, islet atrophy	Mild β-cell depletion

Glucose Homeostasis

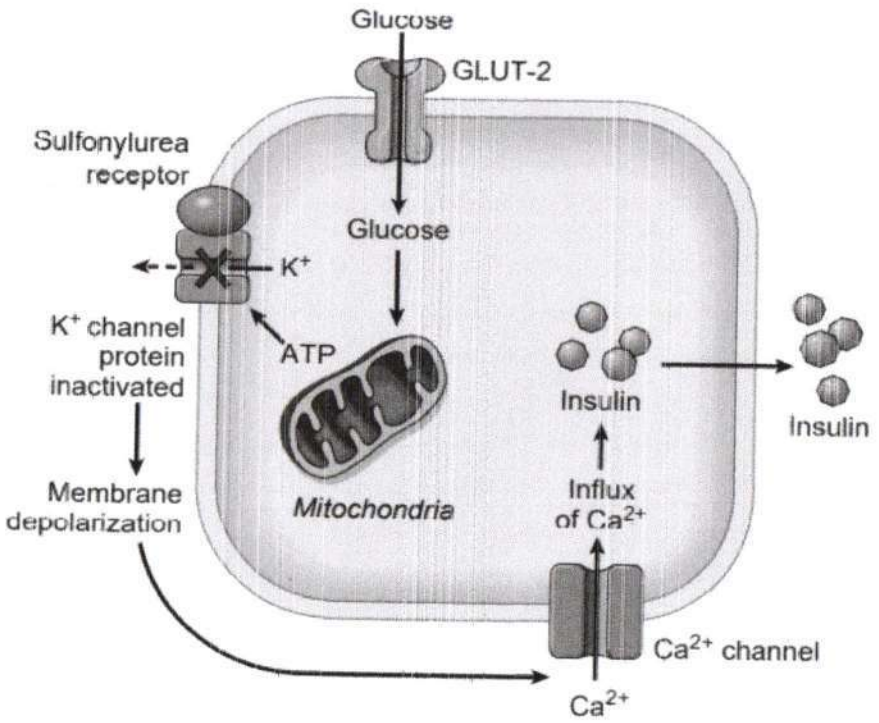


Figure 24-28 Insulin synthesis and secretion. The influx of glucose into β cells through the GLUT-2 receptors initiates a cascade of signaling events that culminates in Ca^{2+}-induced release of stored insulin (see text for details).

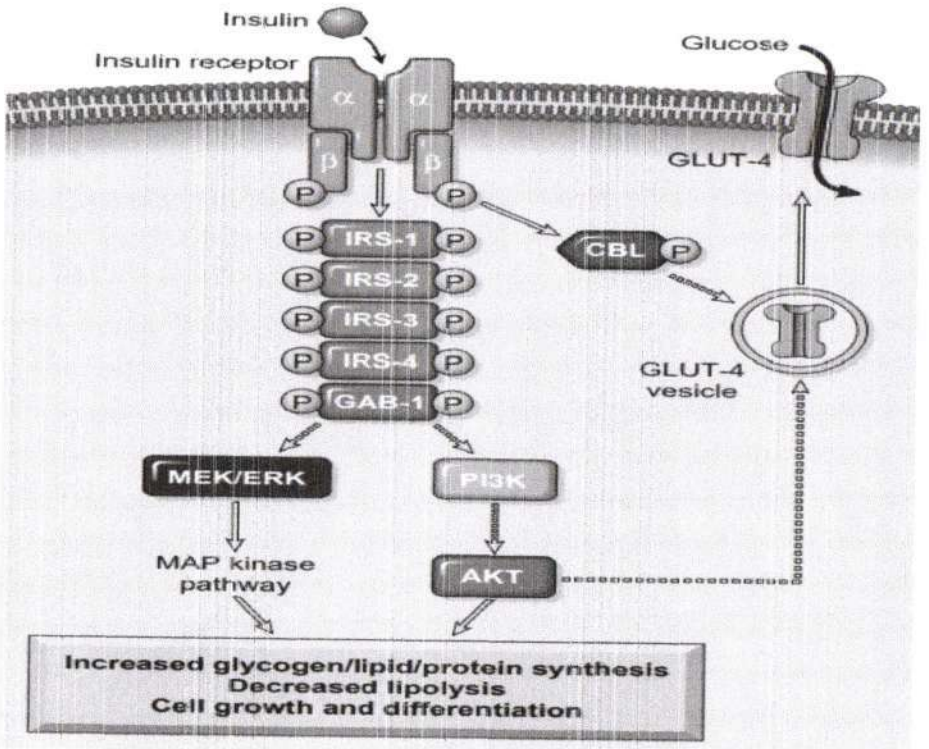
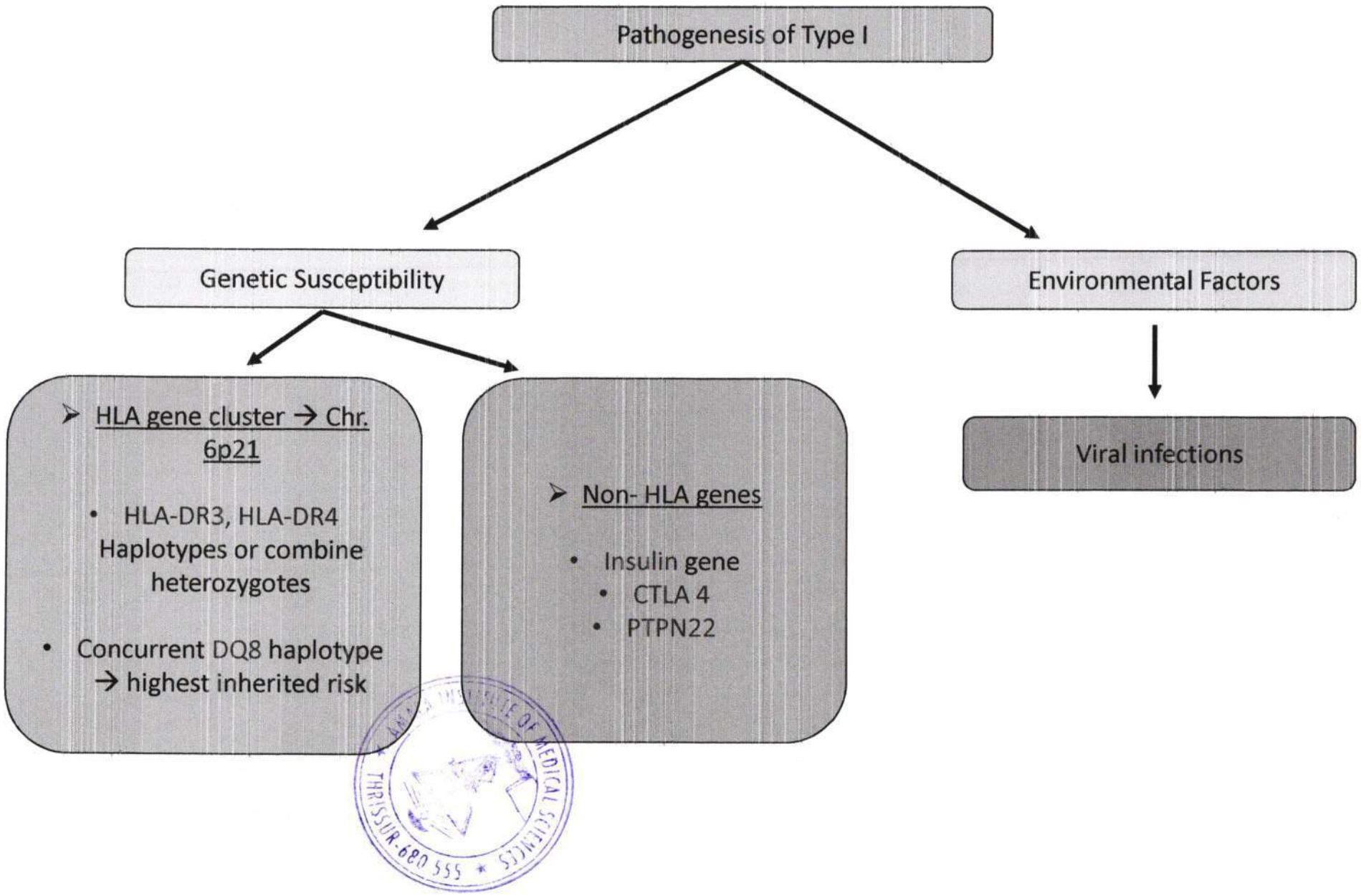


Figure 24-30 Insulin action on a target cell. The metabolic actions of insulin include promoting glycogen synthesis by activating glycogen synthase, and enhancing protein synthesis and lipogenesis while inhibiting lipolysis (see text). Dashed arrows represent intermediate proteins and binding partners that are not shown in this overview diagram.

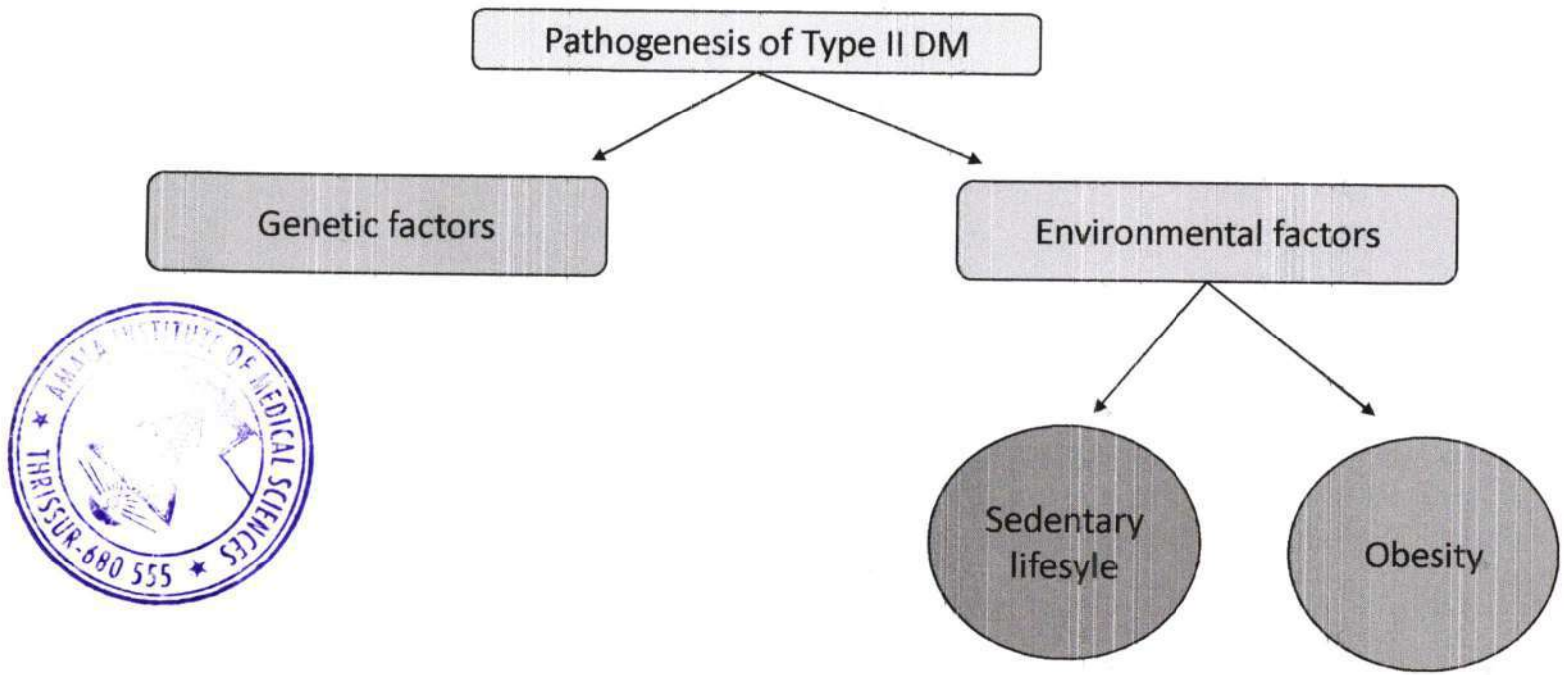
Pathogenesis Of Type 1 DM

- Auto immune disease
- Immune effector cells against endogenous β-cell antigens
- Destruction of islets
- Develops in childhood → manifest at puberty → progresses with age
- Inaccurate terms → *'Juvenile diabetes, Insulin – dependent Diabetes'*

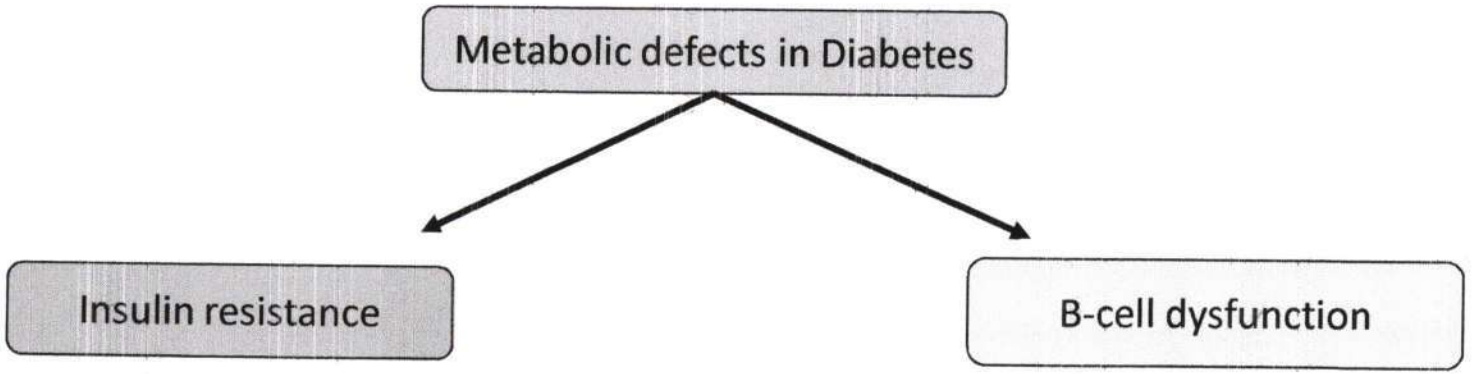

Pathogenesis of Type I
Genetic Susceptibility
Environmental Factors
HLA gene cluster → Chr. 6p21
HLA-DR3, HLA-DR4 Haplotypes or combine heterozygotes
Concurrent DQ8 haplotype → highest inherited risk
Non- HLA genes
Insulin gene
CTLA 4
PTPN22
Viral infections

Pathogenesis of Type II DM

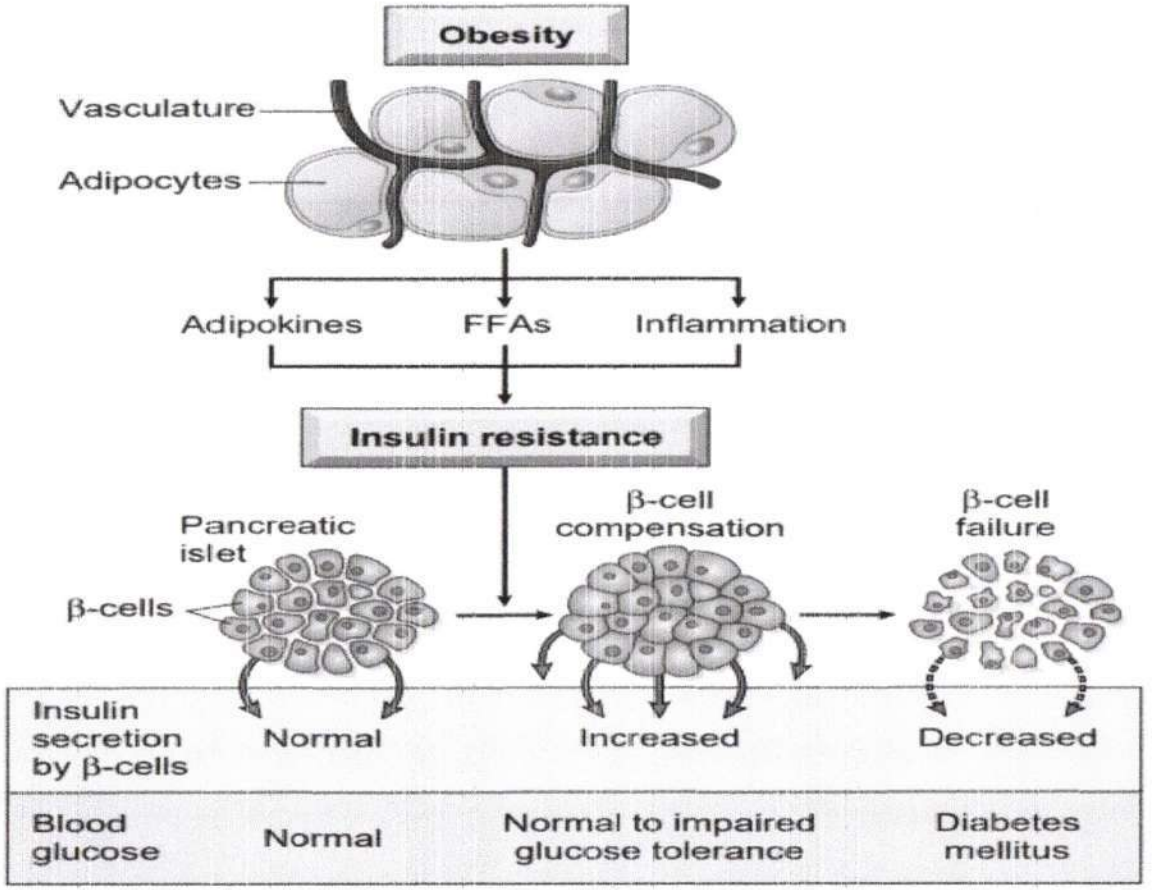
- Complex disease → genetic and environmental factors and a proinflammatory state



Pathogenesis of Type II DM



Obesity & Insulin Resistance



B – cell Dysfunction

- ***Lipotoxicity*** → excess FFA compromise β-cell function
- ***Glucotoxicity*** → chronic hyperglycemia
- ***Incretin effect*** → reduced secretion of GIP and GLP-1, hormones that promote insulin release
- ***Amyloid deposition***

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Clinical Features – Type 1 DM

- Triad → polyuria, polydipsia, polyphagia

- <u>Diabetic ketoacidosis</u>

 - fatigue, nausea and vomiting, severe abdominal pain, a characteristic fruity odor, and deep, labored breathing (also known as Kussmaul breathing)

Clinical Features – Type 2 DM

Apart from previous,

- ***Hyperosmolar Hyperosmotic Syndrome (HHS)***- severe dehydration resulting from sustained osmotic diuresis

- Hypoglycemia

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LEPTOSPIROSIS

HOW TO DIAGNOSE ?

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CASE CLASSIFICATION

Leptospirosis is difficult to distinguish from a number of other diseases on clinical grounds alone. History of possible exposure is paramount to aid clinical diagnosis.

CLINICAL CASE
A case that is compatible with the following clinical description: • Acute febrile illness with history of exposure to water and/or environment possibly contaminated with infected animal urine with ANY of the following symptoms: Headache • Myalgia particularly associated with the calf muscles and lumbar region • Arthralgia • Conjunctival suffusion • Meningeal irritation • Anuria or oliguria and/or proteinuria • Jaundice • Hemorrhages (from the intestines and lungs) • Cardiac arrhythmia or failure • Skin rash • Gastrointestinal symptoms such as nausea, vomiting, abdominal pain, diarrhoea

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CASE CLASSIFICATION

PROBABLE CASE
A clinical case AND positive ELISA/other Rapid tests.
CONFIRMED CASE
A confirmed case of leptospirosis is a suspected OR probable case with any one of the following laboratory tests: ▪ **Microscopic Agglutination Test (MAT),** For single serum specimen - titre ≥1:400 For paired sera - four fold or greater rise in titre ▪ **Positive PCR** (samples should be taken within 10 days of disease onset) ▪ **Positive culture for pathogenic leptospires** (blood samples should be taken within 7 days of onset and urine sample after the 10 th day) ▪ Demonstration of leptospires in tissues using **immunohistochemical staining** (e.g. in post mortem cases) ▪ In places where the laboratory capacity is not well established, a case can be considered as **confirmed if the result is positive by 2 different rapid diagnostic tests.** ▪ Cases that require confirmation are: i. Hospitalized cases ii. All suspected leptospirosis death cases

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Summary of organs affected in Icteric Leptospirosis

Organ	Clinical features	Investigations reveal
Kidney	Decrease in urine output, features of uremia	Increase in Serum Creatinine, Increase in Blood Urea
Liver	Jaundice, hepatomegaly	Increase in Serum Bilirubin with normal or mildly elevated SGPT and SGOT and increased CPK
Lungs	Cough, haemoptysis, dyspnoea with increase in respiration rate and basal creps	X ray chest shows lower and mid zone opacities.
Heart	Hypotension, irregular pulse	ECG reveals the type of arrhythmia
Blood	Bleeding tendencies	Decrease in platelet count
Brain	Altered consciousness with neck rigidity	CSF shows increase in cells, increase in protein, normal sugar

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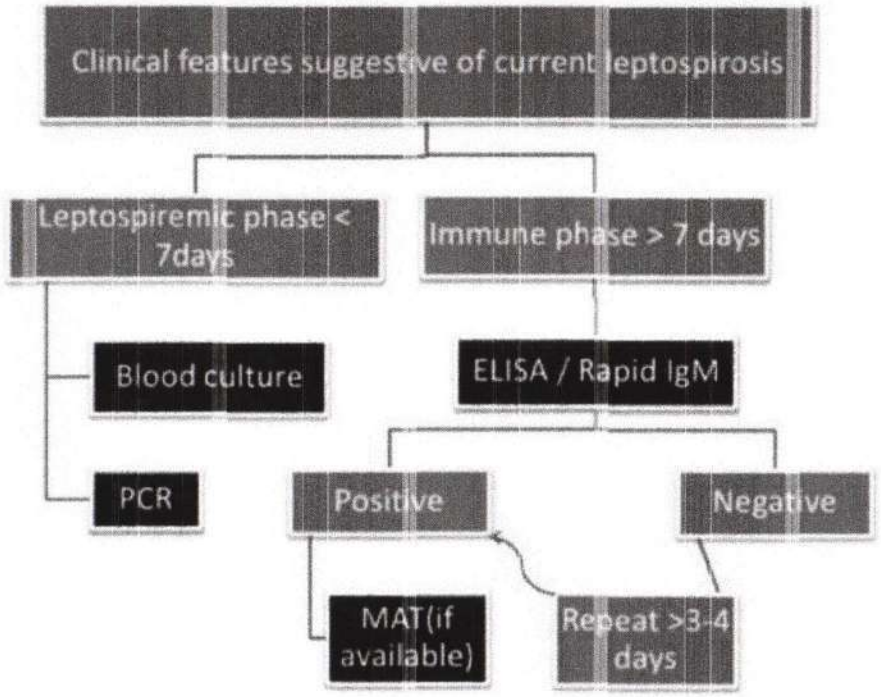
Approach to Diagnosis



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TREATMENT

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- Severe cases are usually treated with high doses of **IV C-penicillin** (2 M units 6 hourly for 5-7 days).
- Less severe cases treated orally with antibiotics such as **doxycycline** (2 mg/kg up to 100 mg 12-hourly for 5-7 days), **ampicillin** or **amoxicillin.**
- Third generation cephalosporin, such as **ceftriaxone** and **cefotaxime**, and **quinolone** antibiotics may also be effective.
- Monitoring and supportive care as appropriate, e.g. dialysis, mechanical ventilation.

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Inhalational (Pulmonary) anthrax

- also known as Woolsorter's disease
- results from breathing anthrax spores into the lungs.
- Earliest symptoms resemble those of a respiratory infection such as **mild fever** and **sore throat**.
- After one to three days of acute phase, increasing **fever**, **dyspnea**, **stridor**, **hypoxia**, and **hypertension** occur usually leading to death within 24 hours.

Gastrointestinal Anthrax

- results from ingestion of inadequately-cooked meat from animals with anthrax.
- symptoms include fever, nausea, and vomiting, loss of appetite, abdominal pain, bloody diarrhea, and sometimes rapidly developing ascitis.
- After the bacterium invades the bowel system, it spreads through the bloodstream throughout the body, making even more toxins on the way.
- This form of anthrax is the rarest .

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Incubation period:

- Cutaneous anthrax occurs **1 to 7 days (usually 2 to 5 days)** after spores enter the body through breaks in the skin.
- Inhalational anthrax occurs **2 to 7 days (but sometimes up to 2 months)** after inhaling large amounts of anthrax spores
- Gastrointestinal anthrax occurs **2 to 5 days** after swallowing spores

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Treatment:

- **Cutaneous/gastrointestinal anthrax**

  - **Ciprofloxacin**, **penicillin** or **doxycycline** are the drugs of choice, usually given for 7–10 days. The duration of therapy for gastrointestinal anthrax is not well defined.

  - If the case is associated with a bio-terrorist attack involving aerosolised anthrax where the risk is high, **ciprofloxacin or doxycycline are recommended and should be given for at least 60 days.**

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• Inhalational anthrax

- Recommended initial treatment of pulmonary anthrax is an **intravenous multi-drug regimen of either ciprofloxacin or doxycycline** along with one or more agents to which the organism is typically sensitive.

- Ciprofloxacin has been recommended on the basis of in vivo (animal) findings. It should be used in preference to doxycycline in cases where meningitis is suspected because of the lack of adequate central nervous system penetration by the latter.

- After susceptibility testing and clinical improvement, the empiric regimen may be altered. A **penicillin-based antibiotic, such as amoxycillin or amoxycillin/ clavulanic acid** may then be used to complete the course.

-Treatment should be **continued for 60 days in all cases of inhalational anthrax.**

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Endocrine Pancreas –

Diabetes Mellitus

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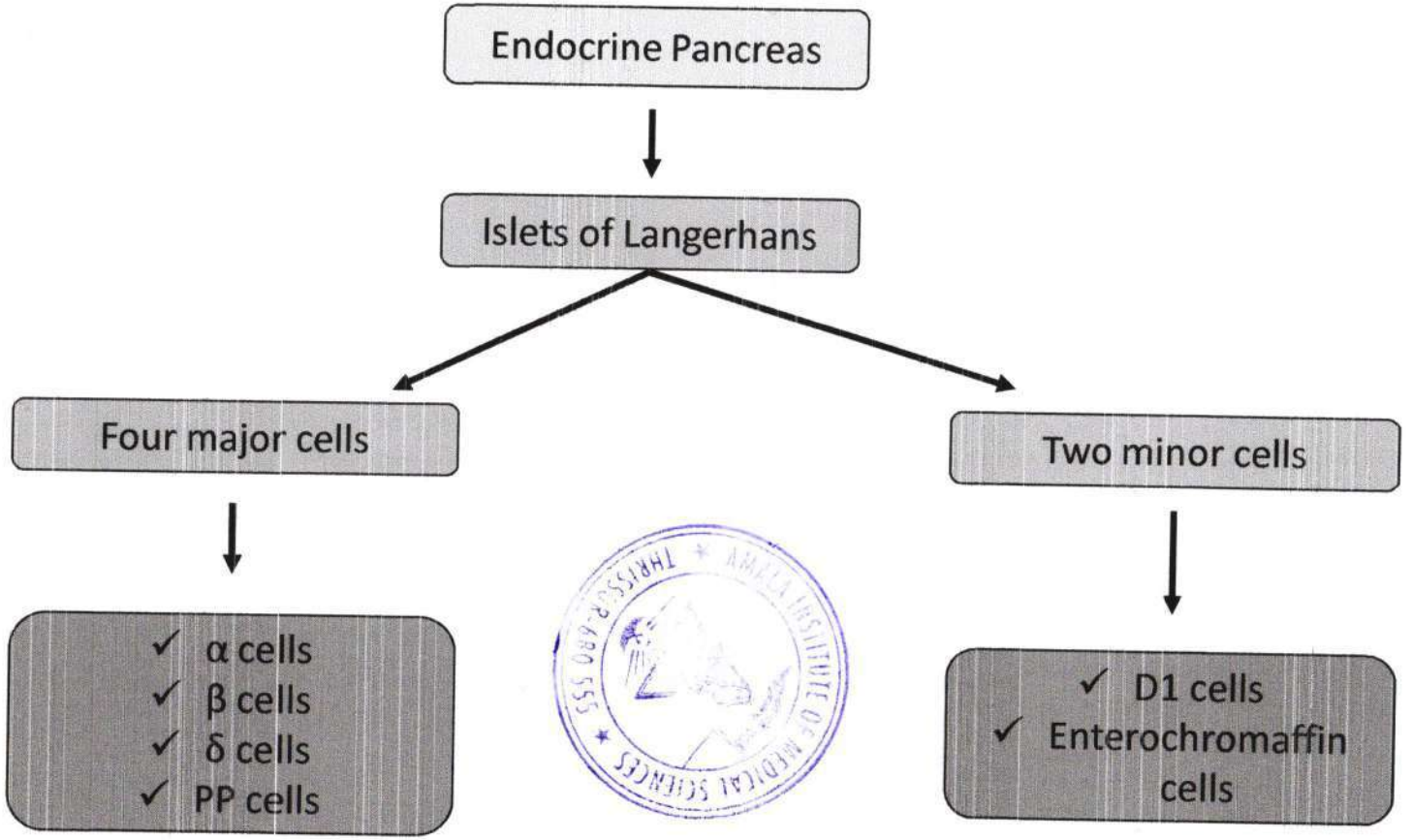
Synopsis

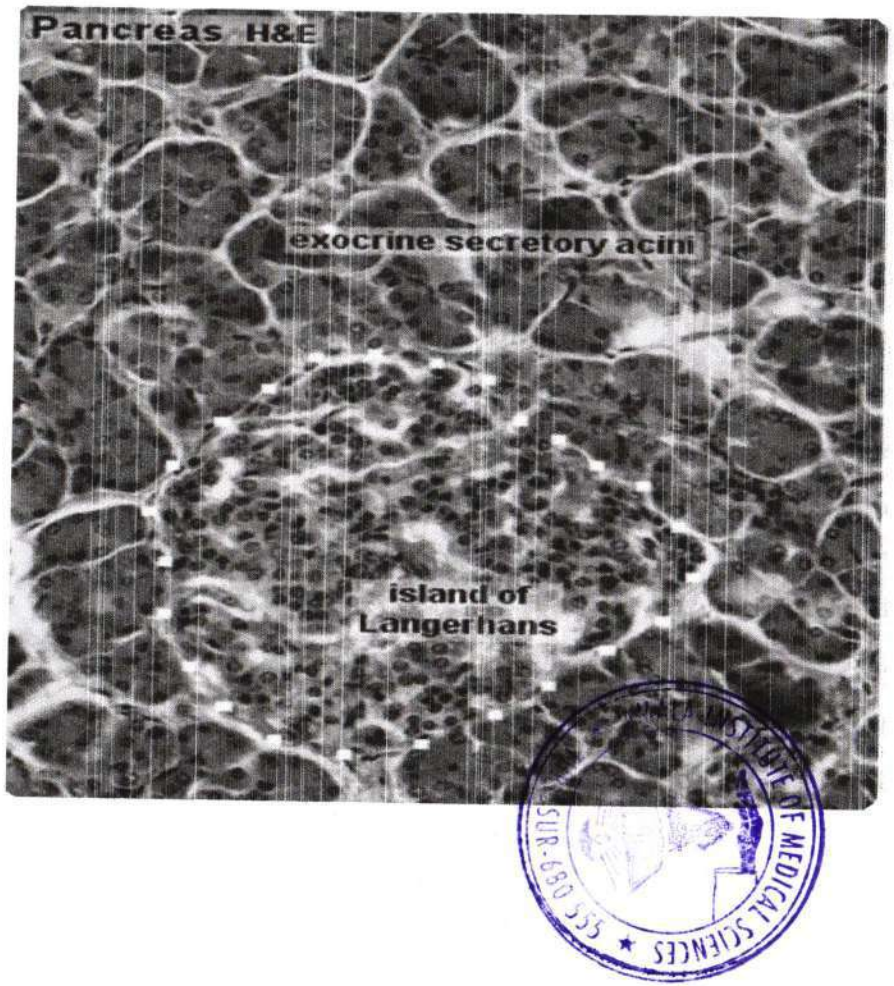
- Introduction to Endocrine Pancreas
- DM – Definition
    - Epidemiology
    - Etiology
    - Diagnosis
    - Classification
    - Pathogenesis
    - Clinical Features
    - Complications

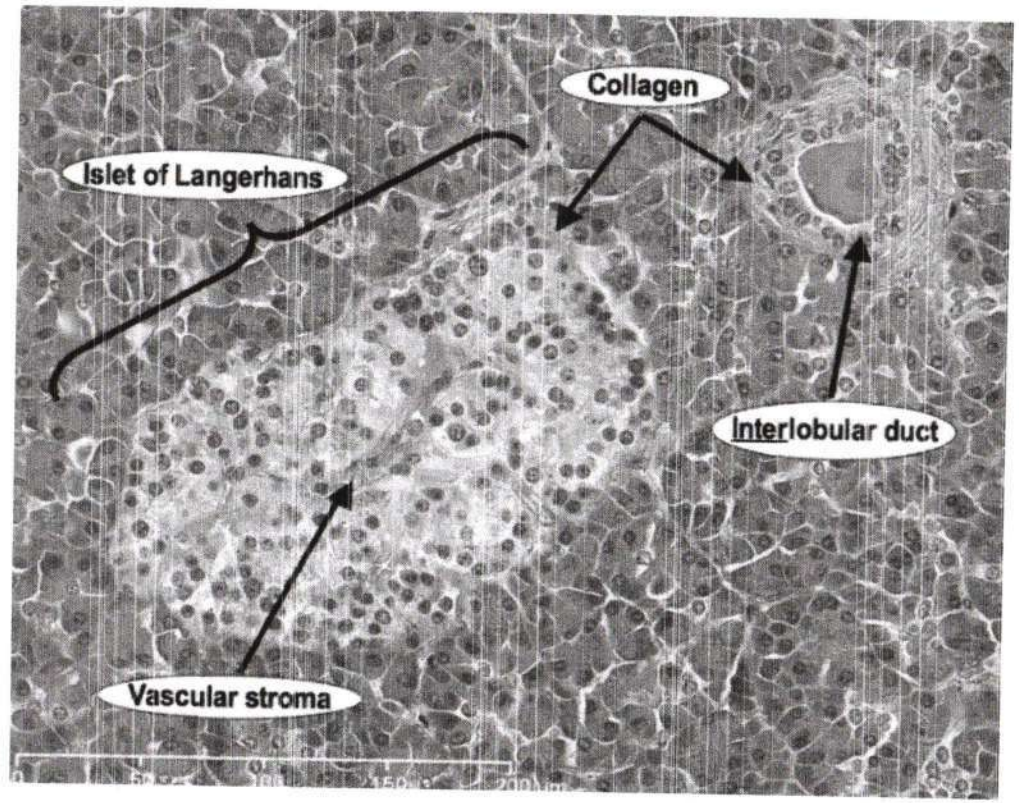
Endocrine Pancreas

Introduction on Endocrine Pancreas



Introduction to Endocrine Pancreas





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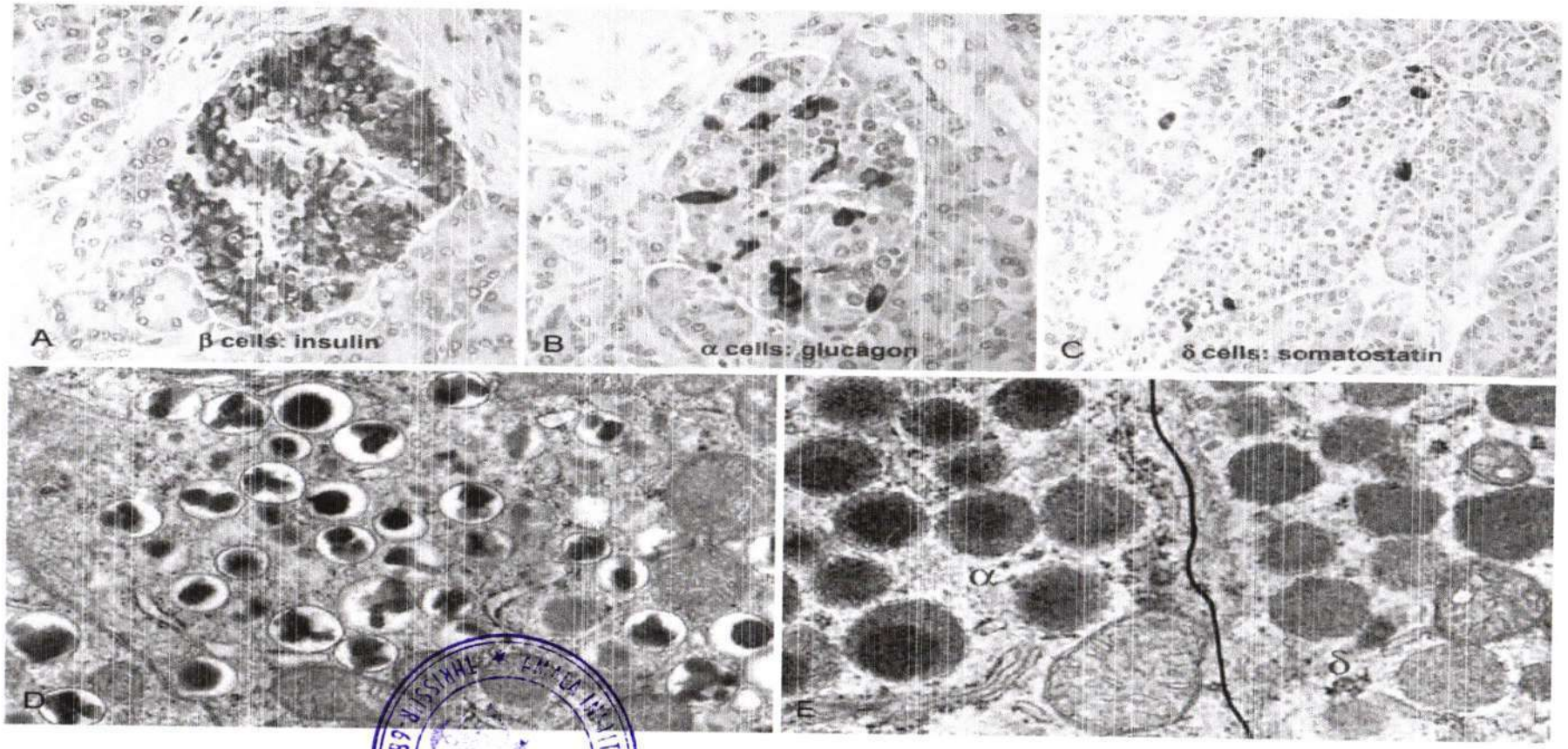

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