9/1/25

Baltimore (ars)

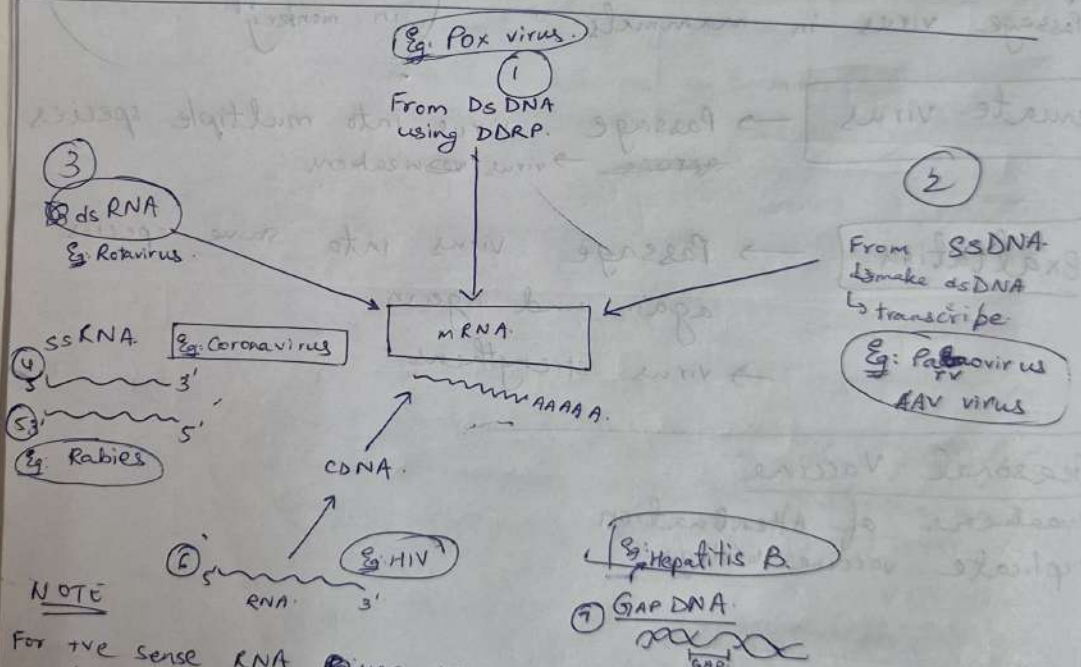
- DNA is genetic info.
- Harshey & Chase → proved DNA is genetic info.

How to prove RNA is genetic info.

- Frankel and conrat experiment
  - ↳ They mixed capsid of one TMV and mixed with the genomic info of another TMV and vice versa.
  - ↳ Read the experiment

How to prove protein is genetic information?

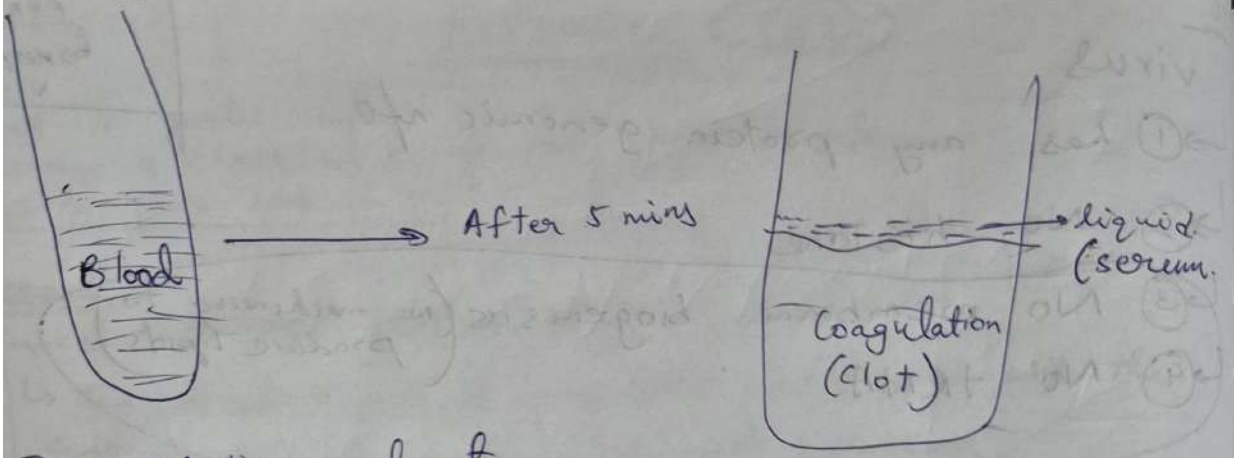
↳ Find out



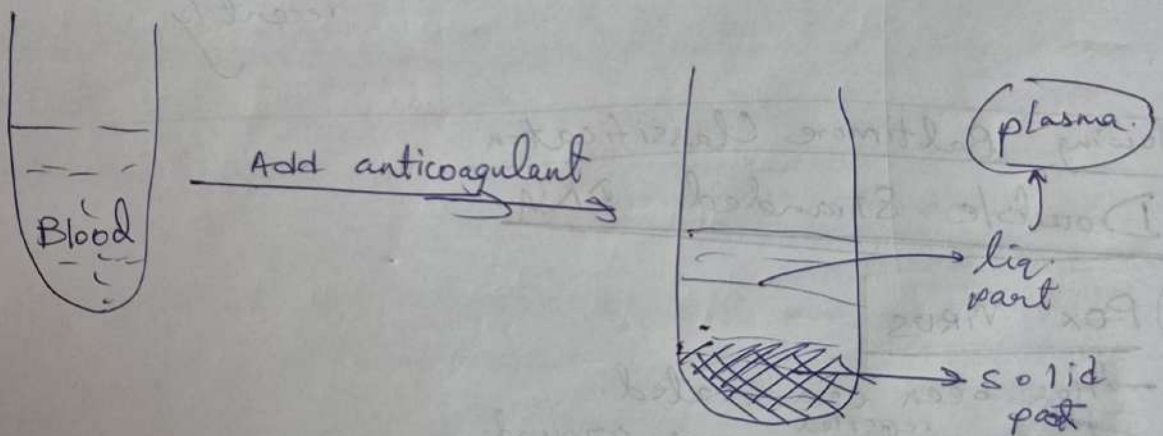
NOTE

For +ve sense RNA, use reverse primer → binds at 3' and RDRP will cause negative sense RNA to form.

∴ Thus, to make 100 copies of RNA from +ve RNA, use intermediate of -ve RNA which is



EDTA → Anticoagulant
↳ chelate calcium ion
↳ blood is liquid form



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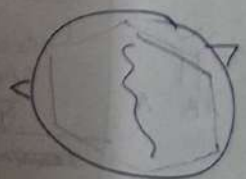
Envelope structures → glycosylated
↳ Post translationally modified proteins
↳ generate an Antibody mediated immune response.

Internal proteins → raise cell mediated immune respons

virion → single virus particle.



NOTE

No virus
- ① has any protein genomic info.
- ② No histone
- ③ No membrane biogenesis (No machinery to produce lipids)
- ④ No tRNA

→ Tupanvirus found recently

Chicken pox → Herpes virus

Following Baltimore Classification

① Double Stranded DNA

(A) Pox Virus

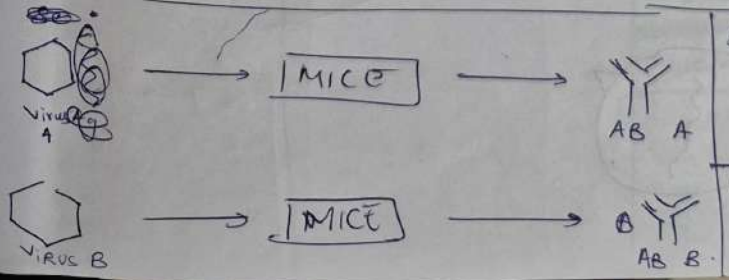
→ Has been eradicated.
- ↳ no reported ~~active~~ cases around.

→ painful
- ↳ Genome ~~strut~~ is stable
- ↳ no major mutation in the genome
- ↳ no variants.
- ↳ one type of vaccine is enough.
- ↳ polymerase is stable → thus, no missense mutation is not obtained.

→ complex symmetry.

→ single serotype.

~~→ host esv~~



Serotype
- ↳ serum specific type.

→ Linear epitope

→ complex epitope

→ Immune response is robust for complex epitope.

• Any cell of human body → produce INF-1

α

β

• INF γ → Type II INF
↳ produced by lymphocyte.

Granulocyte
↳ Neutrophil
↳ Basophil

Agranulocyte
↳ lymphocyte
↳ B
↳ T.

INF III → INF λ
↳ by fibroblast.

Bystandard phenominon

• When the cell survives generational attack of virus, few cells survive and are able to protect using INF.

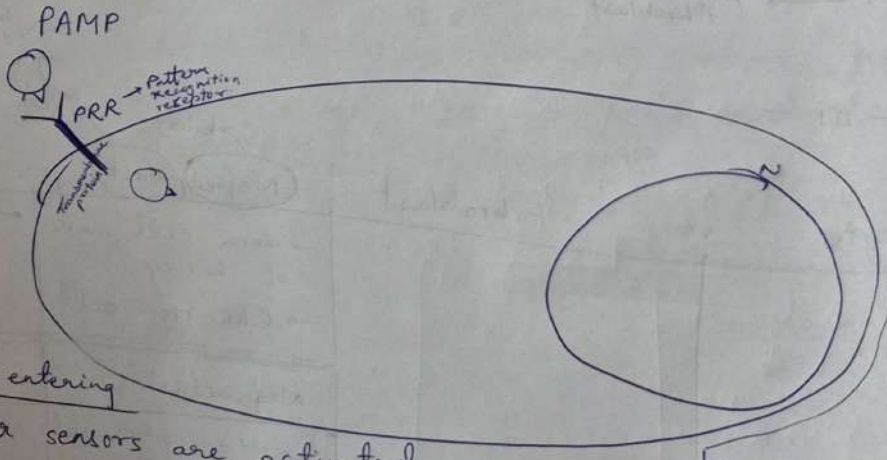
⇒ half life of INF is not very high.
↳ not long memory response
↳ cell can't remember.

How INF are produced and protect other cells

pinocytosis
cellular drinking

phagocytosis

if PAMP is liquid
↑
DAMP
Danger Associated mol. pattern



Upon virus entering

① Cellular sensors are activated.
↳ proteins, NA is out in the cytoplasm

• TLR-9 → cellular proteins
Toll like receptor ↳ recognize ds DNA of virus

TLR 9 → cpG
TLR 3 →
required very high transcription

Eg:

How many viral particles enter the body? → about thousand.

Which cell type does the virus affect?

(i) cell which is more susceptible.

(ii) The appropriate cell with the corresponding cell ~~rect~~ receptors.

∴ Cell with lesser immune response or weaker metabolome will have ~~better~~ higher chance of getting infected.

- The cell which is affected by virus produce non-specific protein molecules. ~~that are~~
  - ↳ called interferons. → INNATE IMMUNE MOLECULES → NON-SPECIFIC.
    - ↳ interfere with virus infectivity.
- Go to other cells in the region and interfere with the virus in those other cells in the region.

∘ Irrespective of ~~pox~~ virus, or corona virus, same INF are produced

- Chr 9 & chr 12 → involved in INF production.
- IFN-I → produced by fibroblast
  IFN-II
  IFN-III

- Fibrocyte vs Fibroblast.

Nefroblast vs Nefrocyte

kidney

mother cell

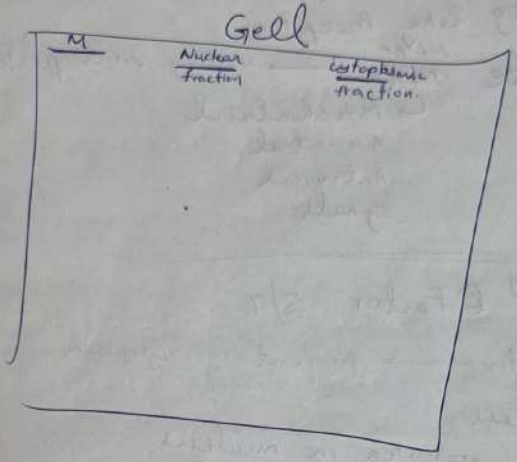
can form cyte.

cell

→ inner cell mass of kidney → like stem cell	→ daughter cell kidney
Nefroclast	
↳ phag…	
↳ kill…	

How to prove STAT is in cytoplasm ?

- Lyse the cell using
  - ↳ TRITON - X100 (detergent).
- Do mild treatment.
  - ↳ 0.1 · Triton X 100 with mild SDS
- Fractionate and load onto SDS page.



GAPDH → marker for cytoplasm fraction

Histone 3 → marker for nuclear fract

@16/1/25

DLS → Dynamic light scattering.

(1) → used to measure cell size.

Important protein (Proteins produced by host cell to stop virus molecule)

(1) IFITM → outward protein (transmembrane)

- ↳ increase diameter.
- ↳ antiviral.
- ↳ can be anchored with other proteins which we want to express on the surface.
  - ↳ Thus, can act as drug delivery system

(2) Tethrin

- ↳ inward protein.
- ↳ protein trafiking.

NOTE	
NF-kβ → strong TF	pro inflamatory → expressed before inflamation
inflammation promotion → Histamine (cytokine) ↳ modulate inflamation → IL8, IL18 ↳ pro inflamatory → Caspase 1,	anti-inflammatory → IL10. → TGF-β → Cancerous agent

↳ ssRNA → not detected by cell sensor

↳ dsRNA → detected by TLR 3.

13 TLR.

• proteins from virus → displayed on MHCI.

↳ endogeneous protein.

• RIG → protein which senses mitochondria. (Retinoic acid inducible gene)

• Mitochondria has RLR → Rig like Receptor

↳ can activate MAVS → Nullify virus protein.

~~MAVS~~

↳ Mitochondrial Associated Antiviral Signals

• IRF 3/7 → INF ~~Responsive~~ Regulatory Factor 3/7.

↳ Transcription factor → present in cytoplasm.

↳ migrate to Nucleus

↳ bind to specific promoter in nucleus.

↳ help to produce INF proteins.

↳ protect neighbouring cell

How to produce INF without virus particle?

~~(1) Use virus coat.~~

(1) Using Antigen that causes non specific INF production.

(2) Use rhetenoic acid (Vitamin A)

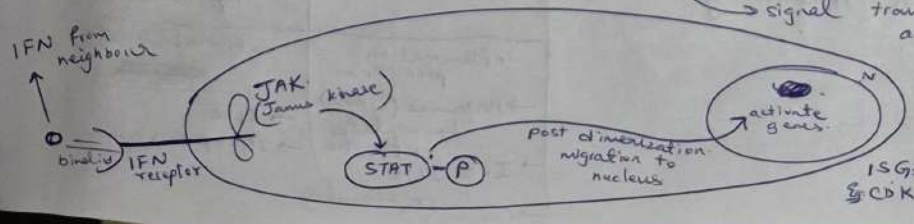
↳ vitamin A can lead to production of RA - pathw

Neighbouring cell

JAK → STAT → applicable to neighbouring ce

JAK STAT pathway

→ signal transducer and activator of transcri



③ Viperins
↳ strong affinity for Lipid molecule
↳ modulate Lipid Biogenesis
↳ can prevent budding out of virus particle by preventing virus particles to get Lipid membrane

④ CH 25 H → cholestrol 25 Hydroxylase.
↳ modulate lipid Biogenesis

INF → produce 2-5 OAs → oligo Adenylate Synthetase.
↳ protein can synthesize A.
↳ can dimerize RNAse L (Ribo Nuclease
↳ can chegw mRNA in host cel

Thus, if cell treated with INF, (PEG INF)
above process occurs
↳ dissociation of mRNA occurs

→ Also activates (PKR)
phosphorylation and deactivation
(eif-2) → purpose is to bind Ribosome and mRNA binding
↓
eukaryotic initiating factor
↓
Thus, ~~mRNA~~ transcription and translation is turned off in the cell that is treated with INF

Half life of INF → 12 hrs

Q Females are more susceptable to viral infection ?

- → FSL
- → LH
- → progesthone
- → estrogen

} Femal hormone

→ During periods, FSH : LH moves to ratio of @ 1:8

- Life of ova in tube is dependent on FSH : LH ratio.
- Hormones can play role in zika.

Zika ↓ infect only pregnant females

---

③ MHC - 1 (CTL response)

→ viral proteins → endogeneous.

↳ a 15-8 mer is formed ~~which~~ of viral protein to be displayed on MHC-I

↳ chewing of protein in proteosome

↳ displaying of protein between α_1 & α_2 in MHC groove.

↳ can activate $CD8^+$ T cell population.

- $CD8^+$ immune response → super critical for viral infection control

---

④ AB - mediated @ response

Cross presentation

↓

presenting ~~Ag~~ endogen each peptide on MHC I

↓

Can activate CD_4^+ T cell

NOTE

New term

↳ Ab mediated enhancement of viruses

② Carbohydrate Metabolism

① A person with diabetes has a less chance to get viral infection.

② Woman are more succeptible than men?

Glut → glucose transporters.
→ channel proteins
→ used for entering glucose in cell
→ produced in the cell only.

• Thus,

→ Glut glycosylated to surface → more and more
→ enhance trafiking to surface

Thus,

→ If more glucose in cell, whole glycolysis process is evolved
⇒ Pyruvate formed.
⇒ go to mitochondria → TCA cycle → more ATP
⇒ Complete glycolysis will increase.
⇒ Cell becomes ATP factory
⇒ Virus increasing carbohydrat metabolism.

No. of proteins in mitochondria = 1000.

Warber effect:

→ metabolizing glucose in alternate way

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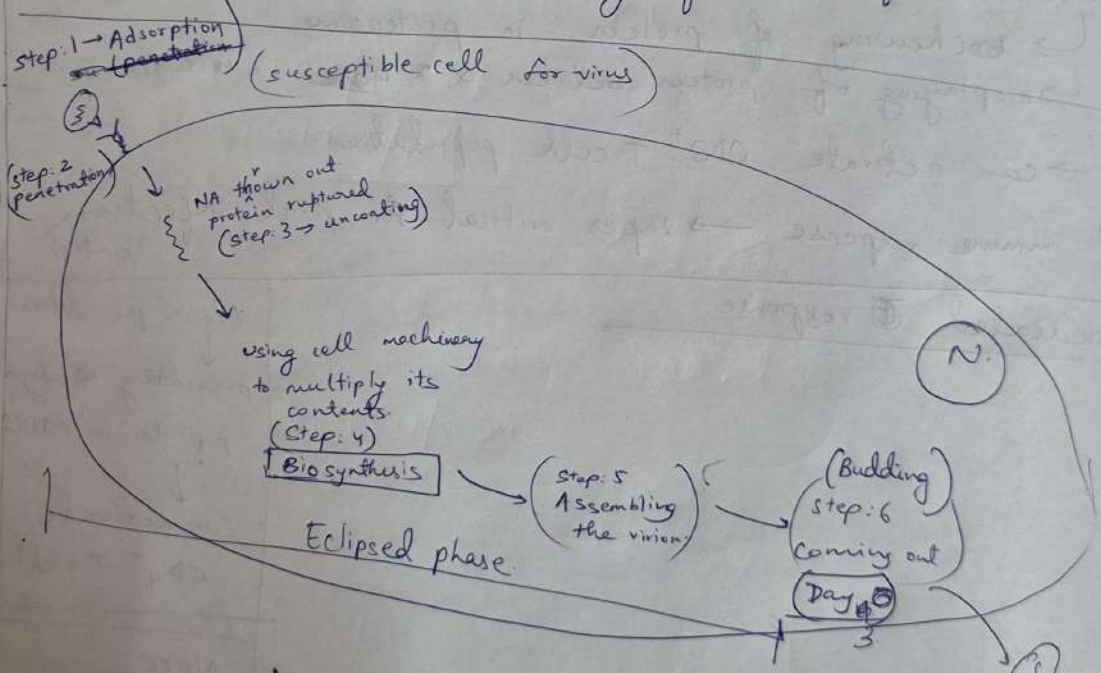
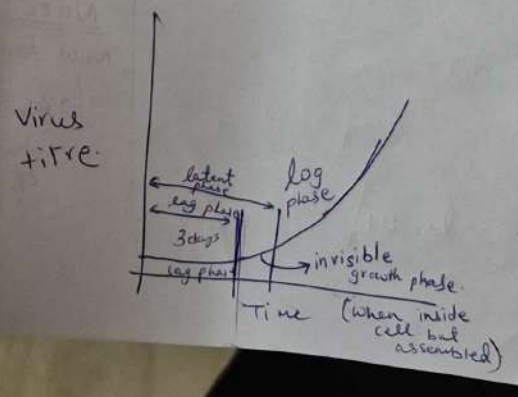
(Missed 15 mins)

Cytopathic effect → changes seen in cell after virus infection.

- (i) Convention of spindle shaped to round cells.
- (ii) Detachment of cells from other cells.
- (iii) Reduced nuclear size/content → Pyknosys.
- (iv) Vacule formation. (vaculation)
- (v) ~~Sonation~~ Syncytia.
  - ↳ cells fuse to form joint cell with multinucleated form.
- (vi) Plaque formation
  - ↳ non staining of part of cell.





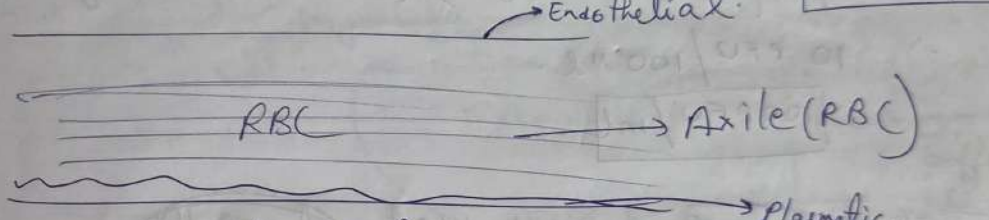
- Intracellular virion. (inside cell assembled virus particle)
- Extracellular virion

No viral particle observed in ~~eclipse~~ eclipsed phase.

Animal System

Eg: Got virus infection in nostril on Day 0.
↳ Talking about hepatitis virus
↳ virus goes to nearest lymph node. (Tonsil).
↳ acquire necessary info at lymph node.
↳ Via lymphatics, virus particle goes to liver
→ Untill here is primary infection.

↳ multiplication in liver
↳ next comes out of liver and enters in blood vessels.
↻.



Blood vessel.

→ virus in blood → viremia.
→ When virus enters blood, blood flow converts from streamline to turbulent.

NOTE
↳ virus contain bladikyrin.
↳ causes diapedesis.
↳ WBC go to liver → causes inflamation in liver.

⇒ Signs and symtomps in liver is seen on Day 3.
⇒ Until Signs seen, it is primary infection.
→ After signs observed, it is secondary infection.

⇒ Day 0 to Day 3 → Incubation period. (Eclipsed phase)

⇒ After secondary symptoms, virus can come out of system on Day 4 via nose. (Latent phase completed)

NOTE
Pox virus.
↳ Virus completely inside cell
↳ doesn't come out of cell
↳ to see virion, need to rupture cell

spleen
↳ largest lymphatic organ

Outer lining of Blood vessel
↓
endothelial cell.

influenza
↳ lungs
HIV
↳ lymphocyte

HERPES VIRUS

- Herpetology → study of snakes.
- genome like snake → 150 kbp
- Tegument like structure
- Ds. DNA virus.
- No vaccine.
- Latency observed.
- microRNA → can control expression of its own gene
- nuclear virus → replication in nucleus
- miR-32 / miR-35 → microRNA.
  - present in every cell.

Pox virus → everything cytoplasm

5. Once person affected with Herpes, cannot be treated.

6. HHV – I, HHV – II → Herpes simplex virus → cause human genital disease.
   HHV – III → Varicella Zoster virus → chickenpox.
   - lesions are small

   HHV – VIII

7. Virus replicates in ~~neuron~~ neurons and B-cells.

Neurons → lack centrioles → No replication

- Lifelong infection.
- Ganglia → collection of nerve ending
- Herpes → attaches to ganglia
  - when stress → virus flares up.
- Corticosteroid treatment → immune suppressant.
  - → causes Shingles disease

Reactivation of ~~chicken pox~~ Varicella ~~Zoster~~ Zoster Virus in Ganglea.

3/2/25

HHV-5 → Cytomegalo virus (CMV) → cells infected give very large nucleu
(neurotropic virus · human)

↳ carries very strong promoter.
↳ CAG → hybrid promoter
↳ Chicken β-actin & CMV hybrid promoter
↳ for enchanced gene expression.

• Kozak sequence
↳ identified in CMV promoter.
↳ 4th position → Guanosine
↳ -3rd position → Purine residue (A or G)
} gives maximum expressi of ge
only In eukaryotic genome.

NOTE

Drug in market → Guanosine analog. → Gancyclo

Viral specific
Thymidine kinase → can phosphorylate guanos
↳ can incorporate A C TG in replicatio

• Gancyclovir → Guanosine analog. → phosphorylo Guanosin
Citovine
↳ can stop repl

---

which AA get phosphorylated?
↳ serine
↳ Threonine

β) HHV VI

β) HHV VII

HHV IV → Epstein Barr virus (EBV)

HHV VIII → Kaposis Sarcoma HV

EBV → mononucleosis

EBV → replicates in B cells.

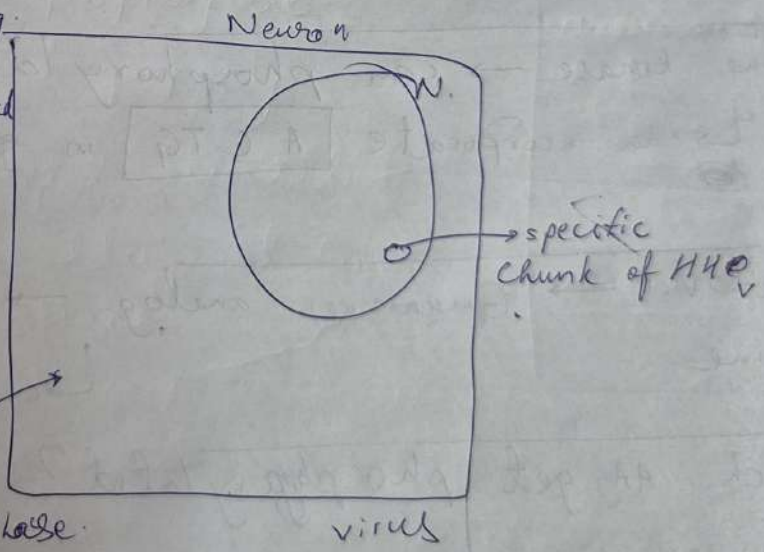
KSHV → Sarcoma

Carsinoma → Epithelial origin

Sarcoma → Mesoderm origin

Latency

- Virus is very long.
- latency associated transcript.
- Only one transcript.
- LANS
- No latent phase.
- lysogenic phase



- sequested in nucleus
- Corticosteroid → immune supressive drug.
- Corticosteroids
  ↳ steroids → Alter the CNS function
- cortical → part of organ
- Black fungus → corticosteroid.
- When stress, LAT produces lots of protein.
  ↳ apoptosis occours.

Lysogenic ⟷ cell infected with virus
  ↳ but cell is intact

lytic → LAT produced

Read Latency, lysogenic and lytic cycle

"Lytic stage → Influenza.

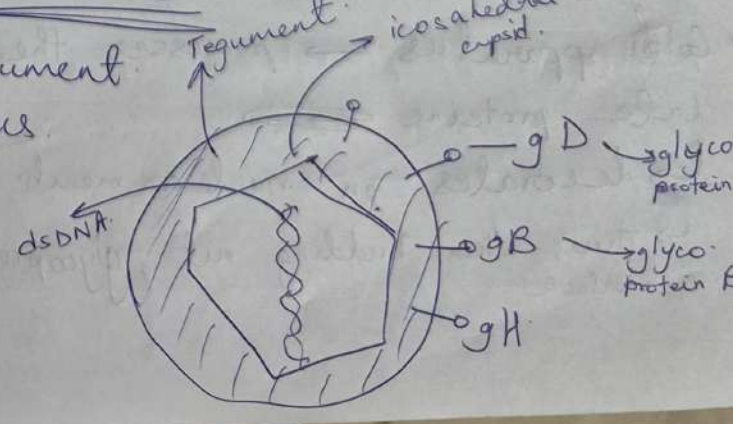
lysogenic virus → HIV."

Lifecycle of Herpes virus

Herpes → contains Tegument
  ↳ envelope virus.

Vp → viral protein.
  ↳ internal protein.

gp → glycoprotein.
  ↳ outer protein.



NOTE
In enveloped virus, gP is outer

- Infects a neuron.
  - ↳ receptor → Nectin. (HVR)
    - ↓
    - binding of HV to cell surface
  - High nectin in neurons.
- Goes directly to Nuclear pore complex.
- Virus has signal for nucleus.
- Capsid fuses out.
- proteins released
  - ↳ Early, intermediate, late proteins
- completely in nucleus
- only 1 transcript produced → LAT released.
- VTk → can phosphorylate
  - ↳ understand properly.
- No pH mediated degradation of capsid in cytoplasm.
- there is budding out from nuclear membrane
- Golgi apparatus → processes the late proteins
  - ↳ decorates on nuclear membrane
  - ↳ Thus, when budding out, glycoproteins occur on surface.

Neurons don't divide ↳ Because absence of centrion
Neuroma

ADENO VIRUS

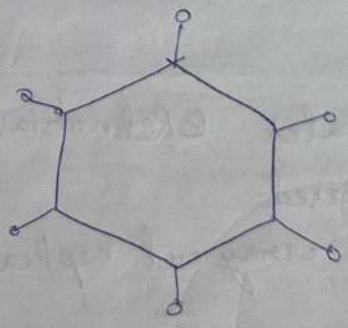
- ds DNA.
- Adenitis → ~~inflammation~~ Glandular related.
- These virus isolated from glands.
- Covieshield → on adenovirus.
- Adeno → pathogen.
- Can cause flu-like symptoms in young ones.
- seasonal infection.
- observed when fluctuation between hot and cold.
- Flu like symptoms.
- Indians → resistant.
  - ↳ affected in childhood.
  - ↳ Ab present against AdS.
- ⇒ Covishield → not affective for Indians.
  - ↳ good for european countries.
- prime-boost strategy
  - ↳ prime with Antigen A.
  - ↳ boost with other Antigen

4/2/25 Adenovirus

How to stain & see virus particle?

→ 1% PTA

→ 1% PUS



→ Easily dissociate.
  - ↳ fragile virus

- No envelope.
- Naked capsid.
- Pentone → where fibre comes out
- Hexone.
- Protease inside virion.
- Ds DNA spread across virus.

Proteins in Adeno

- E1A → immediate early protein
- E2F → binds to DNA promoter sequence
  → causes replication.
  → cell undergoes mitosis due to multiple copies of DNA.
  → oncoprotein.
  → In bound form with retinoblastoma protein
  → tightly regulated.
  → Thus, retinoblastoma → tumor suppressor.

NOTE

In cell cycle, Phosphorylation of Retinoblastoma protein by CDK will cause E2F to free which can carry out replication.

- Thus

In virus infection,

→ E1A → phosphorylates Retinoblastoma.
  ↳ E2F released
  ⇒ Go and carry out replication.

→ Thus,
Adeno → oncovirus.

2nd class in Baltimore Classification

SSDNA

- genome size is very small → 2-6 kbp
- other than capsid, everything is relied on host.
- replicate in ~~not~~ nucleus.
- very efficiently replicate in dividing cells (mitotic cells)
- ~~b form, z form, a form of Di~~
- SSDNA → lack secondary structures.
- dsDNA → has secondary str of a form, b form, z form.
- huge mutation rate
- cell carries (Uracil-N-glycosylase)

↓

prevent C → U.
Since after converting to U, it converts back to T instead of C. Thus, mutation occurs.

isolation of RNA
↓
form loop (secondary str)

NOTE
C → U very common

NOTE
~~Abobec~~
APOBEC-3G
↓
- host produces the protein
- Allows C → U conversion
- In HIV infection, this protein causes deamination of HIV Cytosine, thus making it ineffective

- viral genome will be multiple in number due to sos mechanism # missed the point

- Sometimes, these virus require other virus to ~~infect~~ replicate
  ↳ called dependovirus.

① Gyrovirus

- Chicken Anemia Virus ~~in bir.~~
  ↳ model to study HIV in chicken.

- Virus carries multiple proteins.
  ↳ VP3 → Apoptin

② Circovirus

- 2500 bp genome size.
- circular genome.

③ Geminivirus

- in plants.
- plant pathogen.

④ Parvovirus

- Parvovirus (parvoenteritis) in dogs
  ↳ diarrhea in dogs.
  ↳ bleeding in intestine
- 5th disease → slapped cheek disease } → in humans
  ↓
  Redness disease.

NOTE

- CRESS → Circular Replicase Encoding Single Stranded DNA.
- genome of ss DNA → majorly 2 protein.

DNA

Rep → Replicase

Cap → make viral capsid

8. GGAAGGACGGGGG → can form quadruplex

9. ~~How to prove protein binding to quadro~~

9. How RNA - protein ~~react~~ interaction can be studied?

i) Reassortment

ii) Primer → PCR → RNA protein dehybridize

iii)

13/2/25

+ve SS RNA

E/NE

- Arto virus.
- Corona virus.

POLIO VIRUS

- ~~Understanding~~

NOTE

Understanding translation:

- 40s unit attaches to promoter.
- scans further for start codon.
- When AUG achieved, 60s subunit comes.
- tRNA called.

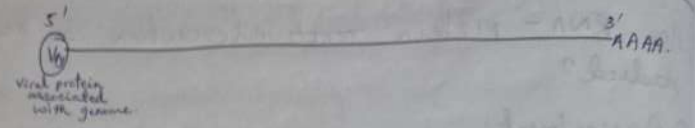
- Thus, if any secondary structure added just above AUG, protein still obtained.
- But, secondary structure added near ribosome binding site causes **ribosome shunting**

Protein machinery in a eukaryotic cell?

i) Ribosome
ii) tRNA
iii) mRNA
iv) Initiation factor
v) Elongation factor
vi) Termination factor

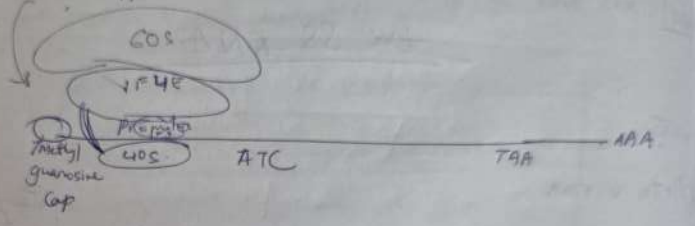
polio viral genome

① poly A binding protein
② poly A polymerase



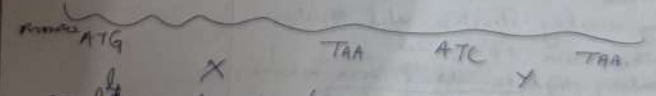
- 5' doesn't have cap
- VPg → 22 aa protein.
- Cap independent translation.

- In a capped mRNA,



~~- In polio.~~
- Polio virus infection causes protease activity on N terminal of eIF4E, thus resulting no 60S activity.
- IF cleaved by virus specific protein.
- No protein formation from host side.
- Thus, no cap dependent translation.

~~poly~~ cistronic mRNA.

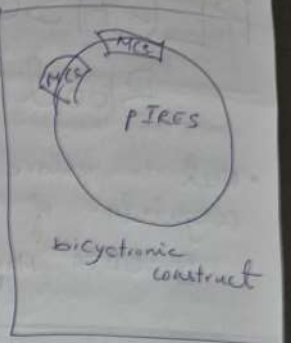


Really only X. (Ribosome falls off After First TAA)
But if VPg present between X & Y,
then even Y is produced ~~without~~ in equal conc with X

⇒ IRES → internal ribocome entry site.

NOTE

- Polio virus binds to host cell receptor CD155 or PVR which is ~~present~~ expressed only upto age of 5-6 yrs. Thus, no infection can happen after 6 years.
- The virus replicates exclusively in neurons.
- Virus can replicated in myelinated neurons.



bicystronic construct

NOTE

All neurons are not myelinated.

Purpose of myelin
↓
for empulse transmission
↓
for protection

Picornavirus → Foot and mouth disease.

17/2/25

Quiz paper discussion

Hershey & chase → DNA as a genetic material
↳ phosphorus/ sulphur

Pox virus
↳ dumbell shaped nucleus.

Rabies
↳ bullet shaped virus

7-methyl guanosine tri phosphate capping → help to protect side chain of DNA.

PLEASE READ

Blood vs plasma vs serum

- Need to remove calcium from blood to prevent coagulation of blood
  - Eg: EDTA, Heparin, Na citrate

Goitre → swolen ~~kna~~ neck
- → deficiency of iodine.
- → problem in Thyroid.

- All metabolism depends on T_3 & T_4.
- ~~More BMR = more~~

- BMR good ⇒ Food conversion rate is high

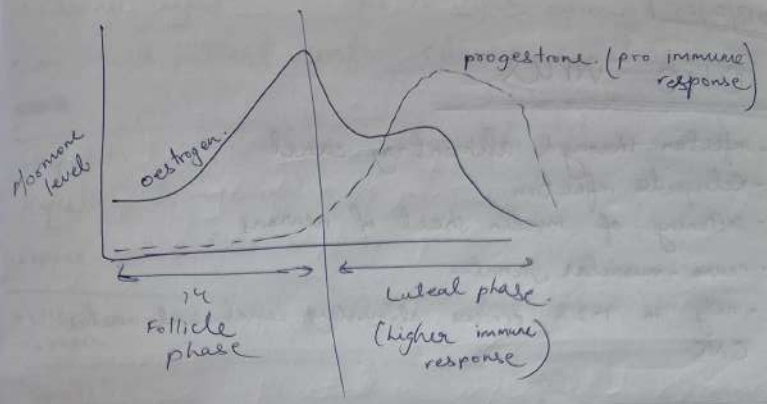
Lower BMR ⇒ high GAG

OR

Lower iodine = high GAG.

OR

High TSH = High gag



LH is high, progestrone high

non infectious

- False impression of only envelope → DI particle
  ↳ Degenerate

Pulse Chase experiment.
↳ tag with $[S^{35}]$ and continue to track protein

- Actinomysin D
  Cyclohexamide } → completely shut down protein production.

Lysogenic phase → genome integrated in cell.

18/2/25

Polio virus

- infection through alimentary canal.
- Retrograde infection
- softening of myelin sheet of neurons
- neuro-muscular junction
- only in 1-2%, crosses alimentary canal and reaches CNS

Hepatitis C virus

- +ve ~~stran~~ sense SS RNA virus.
- Causes Jaundice
  ↳ inflammation of liver.
- Hepatitis like symptoms → if liver metabolism altered
- affect liver epithelium.
- vaccine present for all hepatitis except C.
- Hepatitis D → contaminant
  ↳ ~~co~~ reported with hepatitis B
  ↳ helper virus
- Hepa A, E → contaminated food/water.
- Hepa B, C, D → blood spread.
- Hepa B → not RNA.
- Hepa A, C, D, E → SS RNA virus

- Chronic hepatitis → leads to liver cancer.
- Can treat patient with chronic hepatitis

- Hepatitis A → spread from contaminated stool.

Hepatitis B → Blood bank.

- Non-A - non-B virus.

- Use serum Antibodies of patient on 70000 blood samples
  ↳ paper

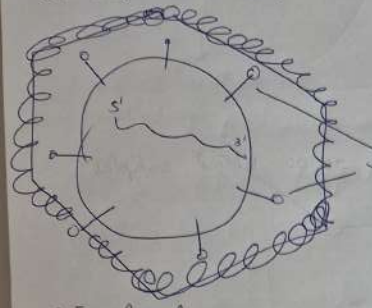
Hepatitis C

- Heavily poly U/UC.
- only 1 C → U mutation can lead to recovery of virus at 5' end.
- C at 5' doesn't recover virus
- U at 5' recovers virus
- IFN + Ribavirin → very strong potential hepatitis C treatment.
- Protease inhibitor → if protein not cut into 7 parts, budding of virus cannot happen.

- PEG → condensing reagent
  → nanoparticle agent
- virus → nanoparticle
- No multiple reading frame

CORONA VIRUS

- Respiratory pathogen.
- Corona → crown



Spike protein.

- Enveloped virus
- SARS → 2005
  - ↳ severe acute respiratory syndrome
  - ↳ ~~Virus~~ ~~Covid~~ CoV.
  - ↳ virus restricted to hong kong
- ~~Zoonotic~~ Zoonitic virus → spread from animal to human
- Anthropo zoonitic → human to animal.
- MERS → Middle east Respiratory syndrome
  - ↳ in Saudi
  - ↳ spread from camel.
- 2019.
  - ↳ Covid 19.
  - ↳ ~~SARS~~ SARS -19.
  - ↳ same group.
  - ↳ China

• Cause of death in India

~~comorbidity~~

↳ majorly not because of virus.

↳ mostly due to panic

~~death~~

↳ symptomatic treatment

Chloroquine

• Spike protein.

↳ 3-6 kb in size.

↳ very hard to clone gene

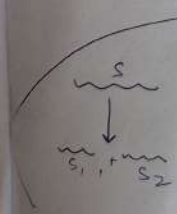
↳ huge

↳ if expressed in bacteria, the function is nill

↳ thus, have to be expressed in ~~cell~~ eukaryotic model, not even yeast.

↳ same for SARS, MERS CoV.

Biology of spike



only when cleavage of S, then 2 protein separated out.

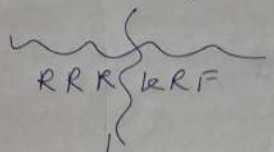
⇒ needed for normal functioning

ORIGINAL seq → RRQ KRRF

cleavage

RRQ + KRRF

• But due to single mutation,



glycosylation
↓
glucose addition

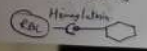
Since mutation,
not any protease can cleave
→ only upper respiratory tract has the protease.

→ further mutation, caused ~~ete~~ glycosylation around spike protein

→ prevented attack of protease on breaking of S1 & S2

20/2/25

Corona virus

→ one of the largest virus of ss RNA group
→ heavily glycosylated protein.
→ RNA covered with nucleoprotein.
→ Corona ⟶ enveloped → derived from host cell.
→ Hemaglutinin – esterases dimer
↳ Hemagglutinin → 

Esterases helps break the hemagglutinin binding with host cell receptor (acts after attach

- Thus Esterases → used for elution of viral protein
- Larger genome ⇒ higher chance of mutation.
- SARS CoV-1 → highly mutated virus
- ~~multiple copies~~

Sars-CoV 2

- multiple ORF.
- spike protein → large protein (S).
- Ribosome frameshifting
- Leaky Scanning } → common for RNA virus

- Leaky scanning → creating secondary structures in DNA before start codon which causes different types of expression.

eg: AAAATT ~ GGGG CCCC
↳ not a good primer
↳ primer overlap

→ Thus, before start codon, if secondary str.

Ribosome frame shift

- can be a -1 or +1 frameshifting.
- -1 frame shift → +3 translocation.
- ~~different kinds~~
- binding in terms of 3 nt.
- for any ORF, we can have 3 types of protein.

- difference between expression vector & clonning vector is RBS
- Expression vector have RBS
- In eukaryotic system, GCCACC is RBS
- If RBS added before GOI, we can get your product

RBS of GCCACC will cause very firm binding of 40S ribosomal subunit. But, if RBS is not GCCACC, then the protein may not be produced. Instead, RBS scans further and may start forming protein from ORF2

ACE

↳ Angiotensin converting enzyme

↳ convert Angiotensin I ⟶ Angiotensin II

↳ reduce diameter of blood vessel

↳ BP high

⇓

Body synthesizes ACE2

↓

to reduce BP

- Thus, if ACE2 is used by sars-cov, then BP of patient continuously increases.

Renin
↳ enzyme from kidney
Rennin
↳ Chymosin
↳ Cheese
made from this

Angio → blood vessel

NOTE

- Docking of virus done by 2 time cleavage of spike protein.
- S1/S2 domain & S2' domain ⟶ 2 cleavage sites.
- Serine proteases ⟶ TMPRSS — protease inhibitor

↓

treatment for prostate cancer.

- TMPRSS2 ⟶ coreceptor
  ↳ help virus entering in host cell.

#watch surfactant molecule · ibiology video.

Pyroptosis → cell death due to caspase 1
- Create tunnel in cell.
- Gasdermin
- proinflammatory cytokine.

Netosis → Neutrophyll specific apoptosis

Apoptosis → caspase 3 & 7

Eosinophill specific apoptosis.

Eetosis → Eosinophill specific apoptosis.

Network of neutrophill.

• Pantoposis

• single ORF of COV → cause multiple types of frameshift mutation.
→ cause different proteins
→ different immune response started

• NS4 of COV → diabetes

• Alloxan → target β-cell