

RNA Structure Genetic Code & Translation

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Type of RNA

- 1. Ribosomal RNA (rRNA)**
 - 16S (small ribosomal subunit)
 - 23S (large ribosomal subunit)
 - 5S (large ribosomal subunit)
- 2. Transfer RNA (tRNA)**
- 3. Messenger RNA (mRNA)**

Type of RNA

- 1. Small Nuclear RNA (snRNA)**
- 2. Micro RNA (miRNA)**
- 3. Small Interfering RNA (siRNA)**
- 4. Double Stranded RNA (dsRNA)**

RNA Silencing (Interferenc) family

Micro RNA = **miRNA** = Endogenous

Small Interfering RNA = **siRNA** = Exogenous

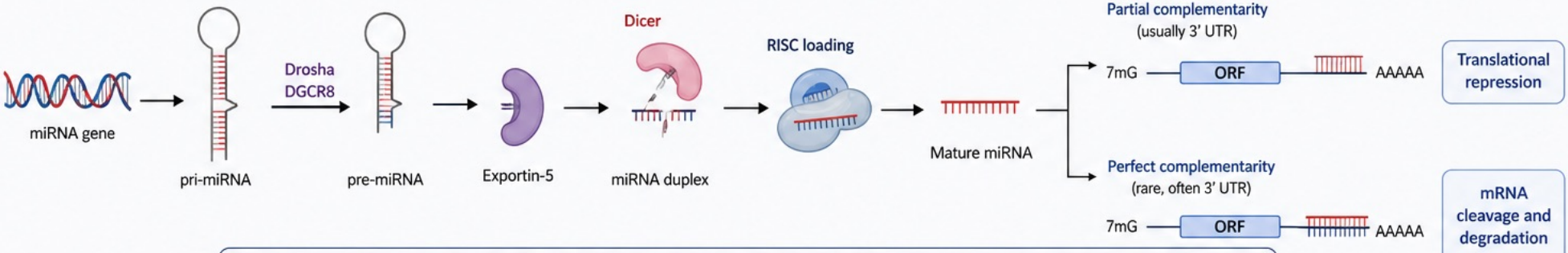
RNA Silencing (Interference) family

- **20 – 25 Nucleotide**
- **Non Coding Sequence**
 - No Protein synthesis
- **Negative Regulation of The Gene**
 - Break m-RNA
 - Repress the translation of m-RNA

Role of miRNA in Cancer

miRNA Biogenesis

Gene Silencing



miRNAs post-transcriptionally regulate gene expression by targeting oncogenes or tumor suppressor genes, thereby acting as **OncomiRs** or **Tumor Suppressor miRNAs**

OncomiRs (miRNAs acting as oncogenes)

Upregulated in cancer

Tumor suppressor genes

- Promote cell proliferation
- Inhibit apoptosis
- Enhance invasion
- Induce metastasis
- Promote angiogenesis

Tumor Suppressor miRNAs

Downregulated in cancer

Oncogenes

- Inhibit cell proliferation
- Promote apoptosis
- Reduce invasion
- Suppress metastasis
- Inhibit angiogenesis

Pathogenesis of Atherosclerosis

- Over Activity of Proteinase **Enzyme Gene**
- Activation of Proteinase Enzyme
- Increase Arterial Endothelium Damage
- Atherosclerosis

Role of mi-RNA in Disease

- mi-RNA712 - Endogenes
- mi-RNA712 >>> Bind With TIMP3 gene
- Tissue Inhibitor of Metallo-proteinases 3 (TIMP3)
- Decrease ——— TIMP3 synthesis
- Increase Proteinase Enzyme
- Increase Endothelium Damage
- Arterial Damage
- Atherosclerosis

What does increase risk of atherosclerosis ?

- A. Increase mi-RNA712 & Increase TIMP3 gene expression
- B. Decrease mi-RNA712 & Decrease TIMP3 gene expression
- C. Increase mi-RNA712 & Decrease TIMP3 gene expression
- D. Decrease mi-RNA712 & Increase TIMP3 gene expression

What does increase risk of atherosclerosis ?

- A. Increase mi-RNA712 & Increase TIMP3 gene expression
- B. Decrease mi-RNA712 & Decrease TIMP3 gene expression
- C. Increase mi-RNA712 & Decrease TIMP3 gene expression**
- D. Decrease mi-RNA712 & Increase TIMP3 gene expression

What can be early marker for detection of probability of atherosclerosis ?

- A. Mutation / Increase mi-RNA712
- B. Non-Mutated Increase mi-RNA712
- C. Mutated / Decrease mi-RNA712
- D. Non-Mutation / Decrease mi-RNA712

What can be early marker for detection of probability of atherosclerosis ?

- A. Mutation mi-RNA712 Gene and Increase mi-RNA712
- B. Non-Mutated mi-RNA712 Gene and Increase mi-RNA712**
- C. Mutated mi-RNA712 Gene / Decrease mi-RNA712
- D. Non-Mutation mi-RNA712 Gene / Decrease mi-RNA712

What can be done to decrease risk of atherosclerosis ?

- A. Anti-mi-RNA712
- B. mi-RNA712
- C. Anti-TIMP3 RNA
- D. TIMP3 RNA

What can be done to decrease risk of atherosclerosis ?

A. Anti-mi-RNA712

B. mi-RNA712

C. Anti-TIMP3 RNA

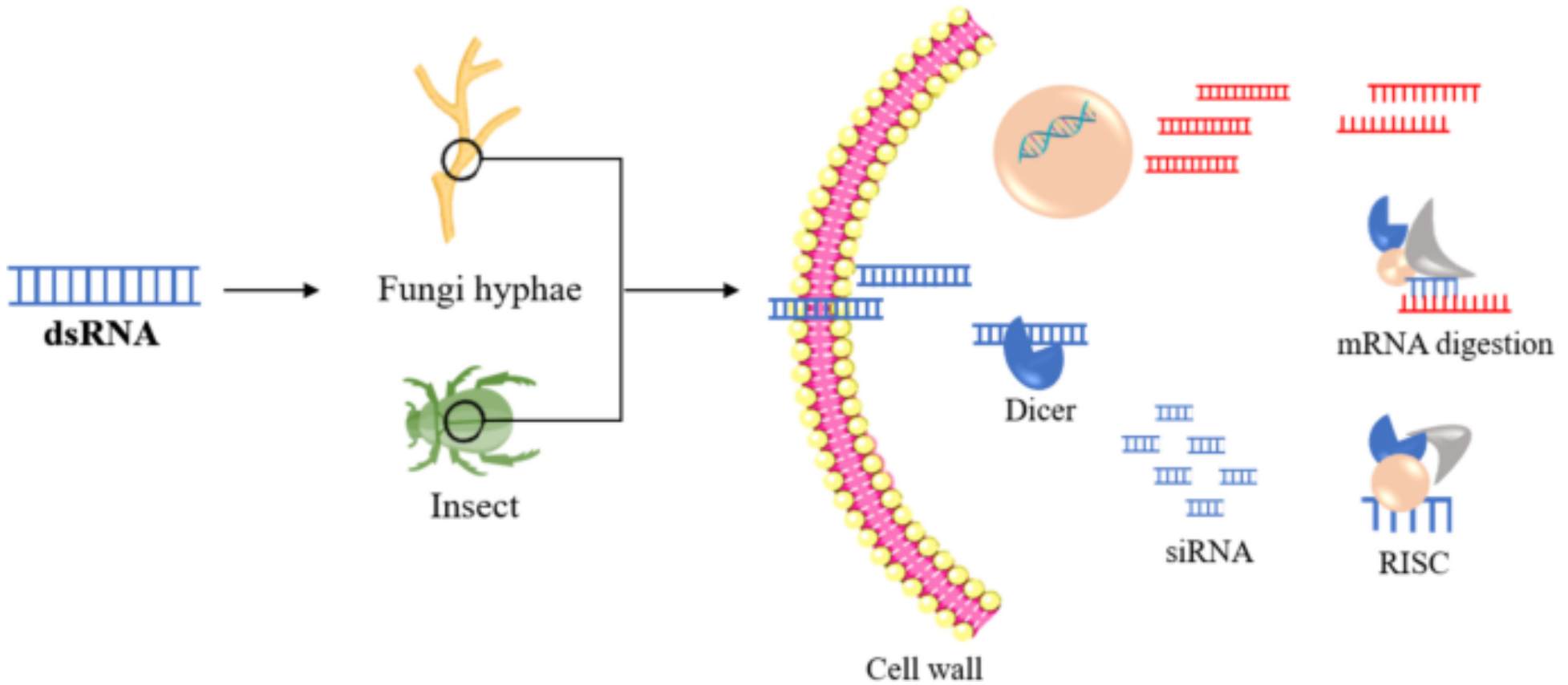
D. TIMP3 RNA

Positron Emission Tomography Scan

Use

of

FDG = Fluro-Deoxy-Glucose



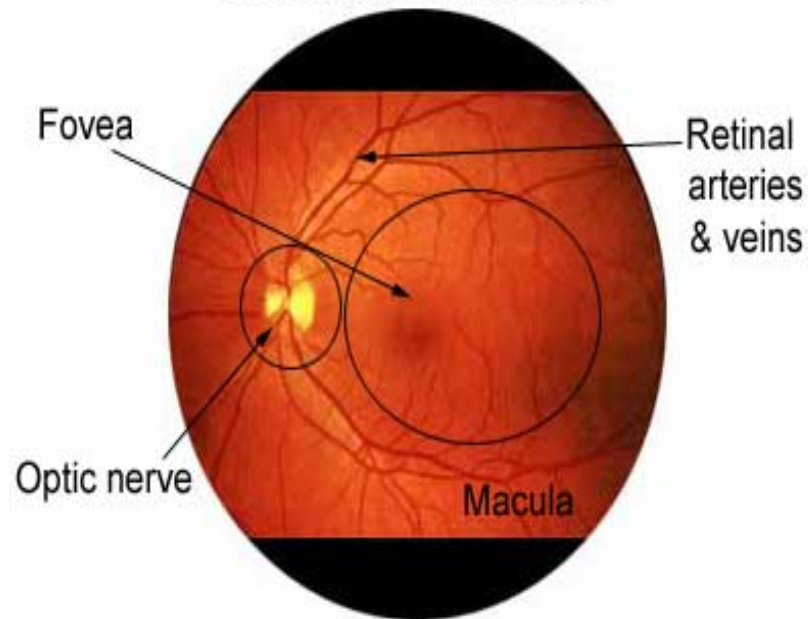
Dicer = Endogenous Enzyme – Ribonuclease
RISC = RNA Induce Silencer Complex

Age related Macular Degeration (AMD)

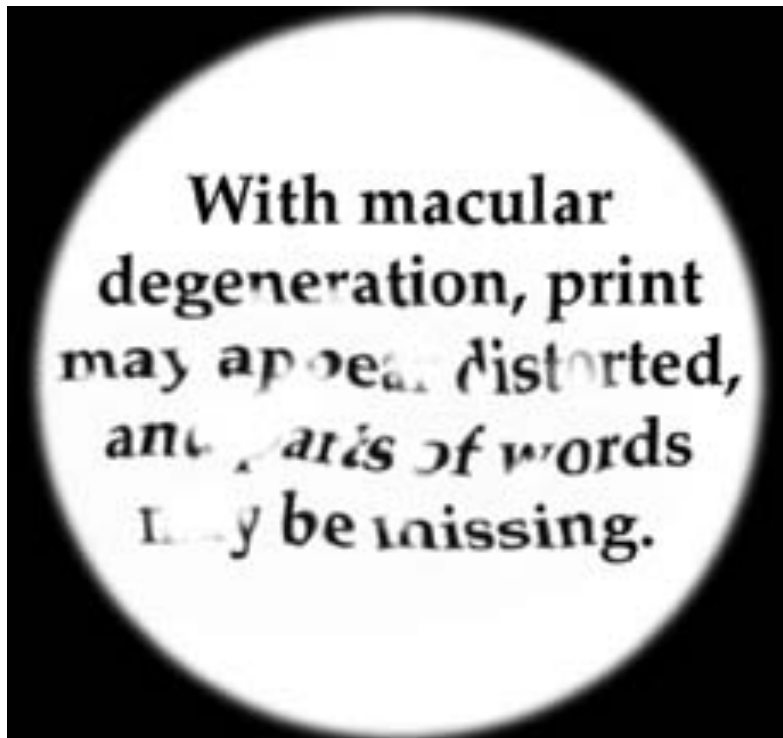


Blindness

Normal Macula



- Visual blurring



Very High --- Vascular Endothelial Growth Factor (**VEGF**)

VEGF --- **Increase blood vessel growth.**

Excess blood vessels behind the retina - **Blurring of Vision**

siRNA — dsRNA (as drug)- **injected into the eye**
specifically **targets the mRNA of VEGF.**

Destroy hundreds of **mRNA**,
So **Suppression VEGF Synthesis**

Youtube Link

- [Gene Silencing by microRNAs \(youtube.com\)](#)
- [Explained! 2024 Nobel Prize in Physiology or Medicine. What is microRNA mediated gene regulation? \(youtube.com\)](#)

snRNA vs snRNP

- Small Nuclear RNA
- U1, U2, U3, U4, U5, U6
- Small Nuclear Ribonucleoproteins

Medulloblastoma

- Brain Cancer
- Mutation >>> U1 snRNA

r-RNA (Ribosomal RNA)

Association with several proteins

Type :

- In Prokaryots = 23S, 16S, and 5S
- In Eukaryots = 28S, 18S, 5.8S, and 5S

“S” = Svedberg unit

Related to the molecular weight and shape.

Function :

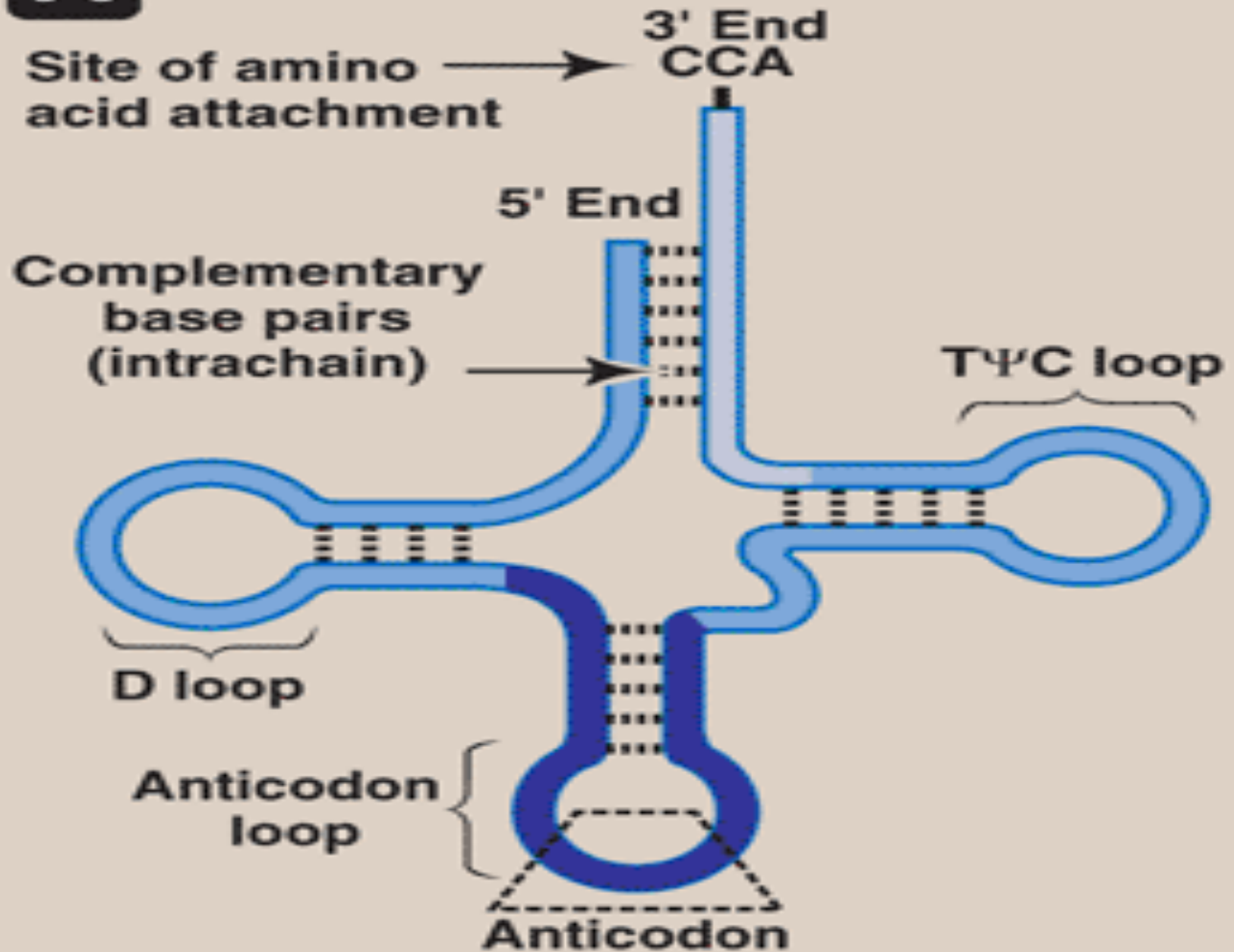
Sites for protein synthesis.

Catalysts in protein synthesis.

E.g. **“Ribozyme”**.

t-RNA

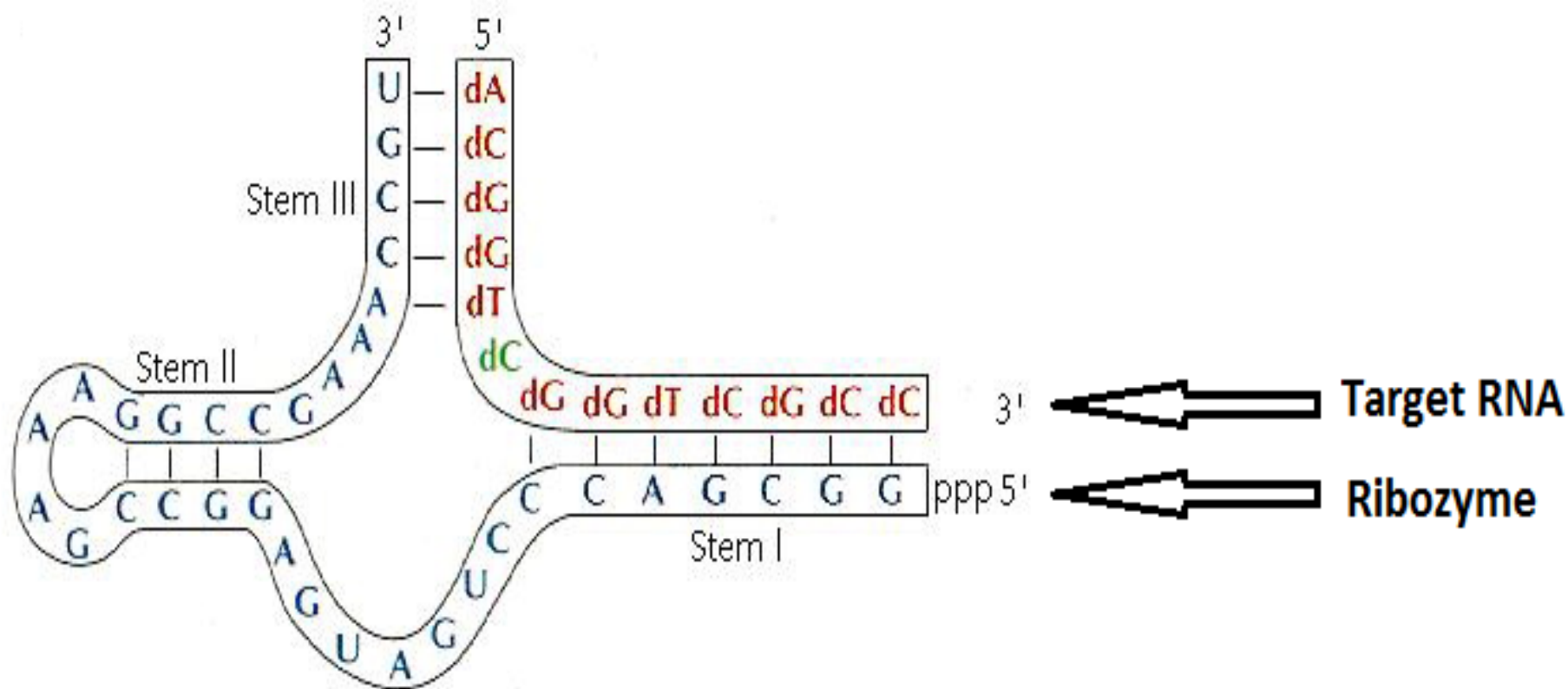
A

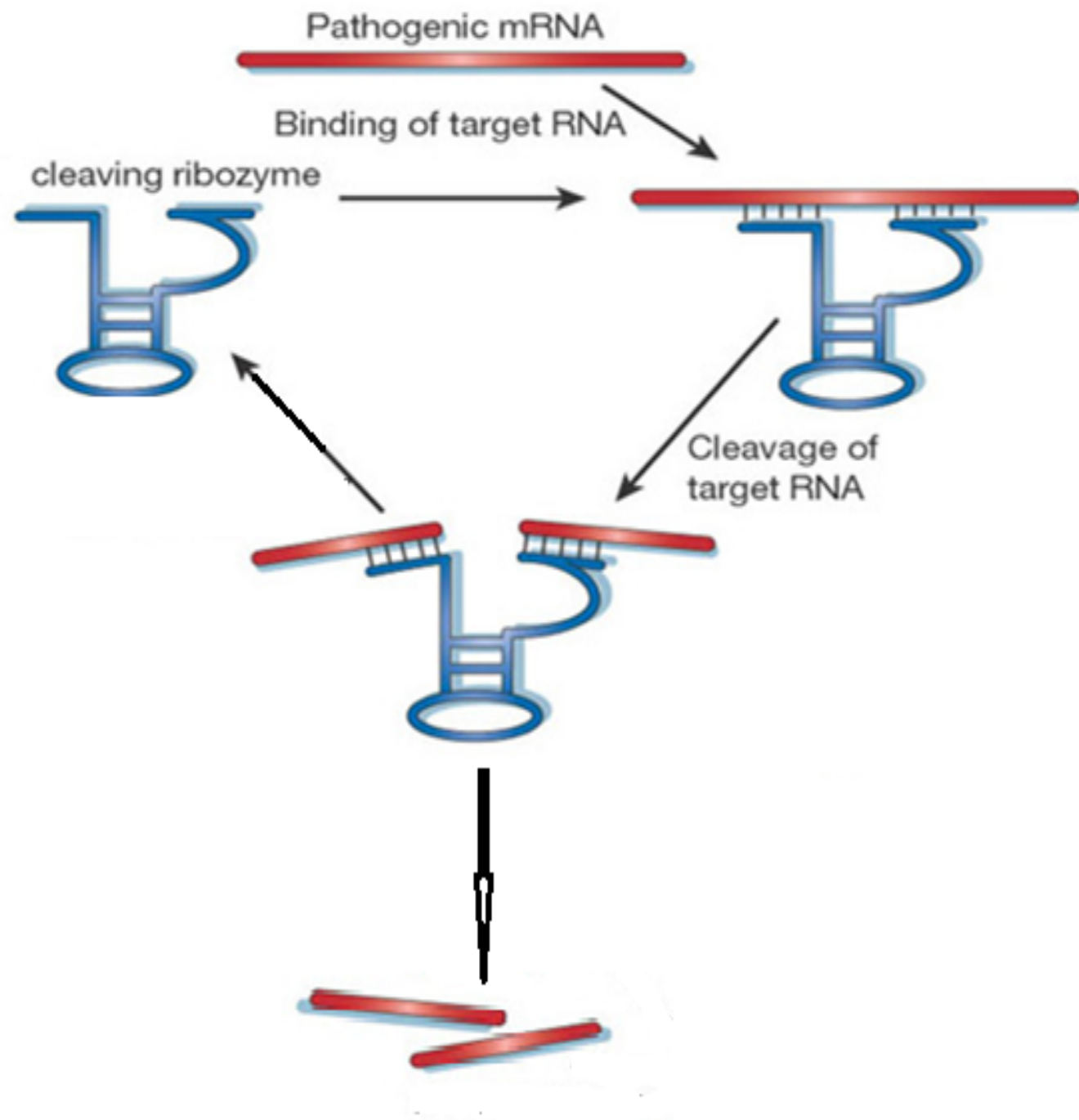


t- RNA (transfer RNA)

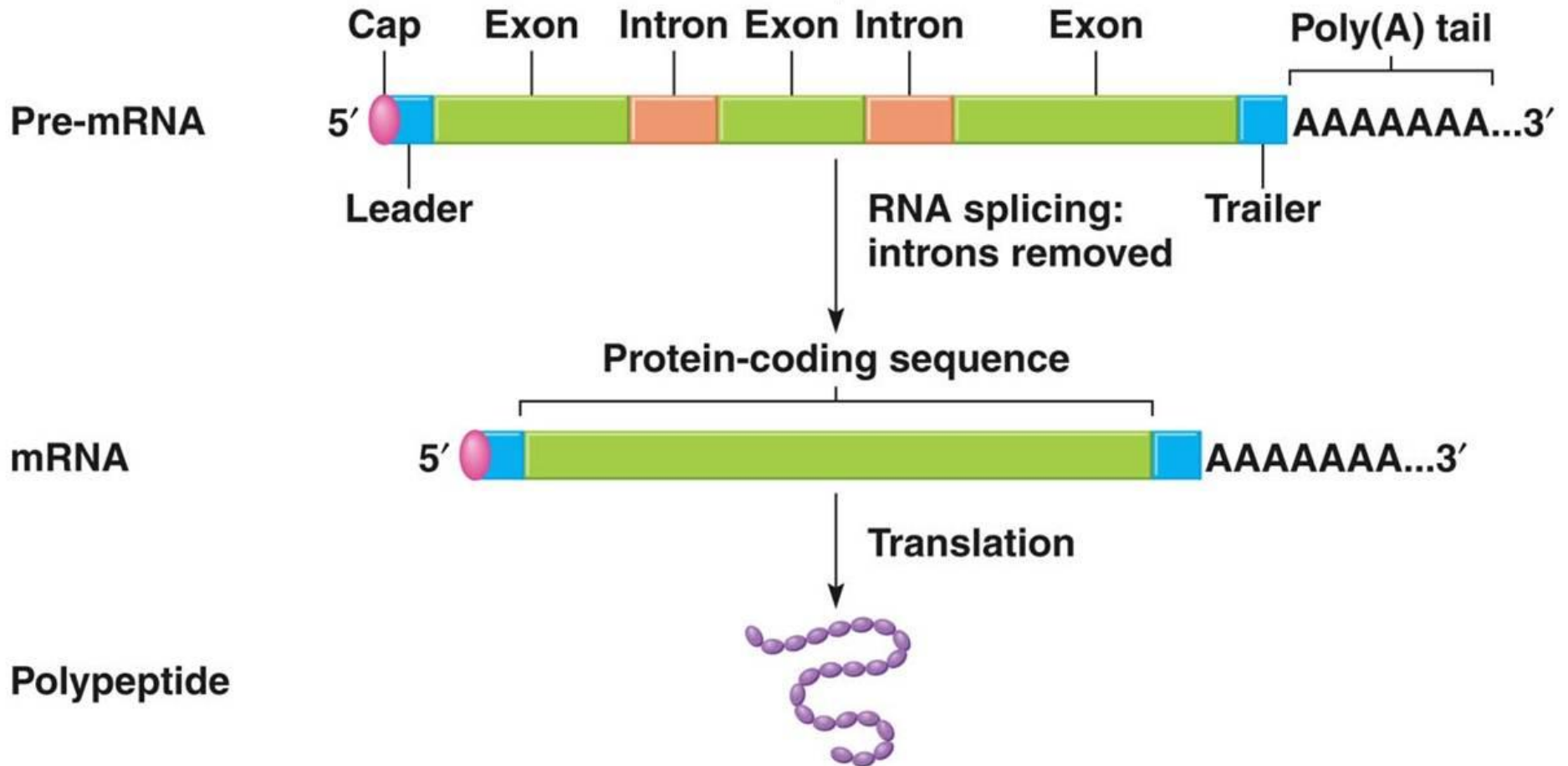
- Smallest (73 – 93 ns)
- Easily soluble = s-RNA
- Specific tRNA for All 20 amino acids.
- Clover leaf like structure.
- Unusual bases (for example, dihydrouracil,)
- Intrachain base-pairing = Looks secondary & tertiary structure.
- Serves as an “adaptor” molecule

- **Dihydrouacil arm** = Recognition Enzyme to add amino acid
- **Pseudouridine arm** = Binding t-RNA to ribosome
- **Anticodon arm** = Recognize triplet codon on m-RNA
- **Acceptor arm** = carries amino acid





hn-RNA (Pre-mRNA) & m-RNA



m-RNA

Mature Messenger RNA

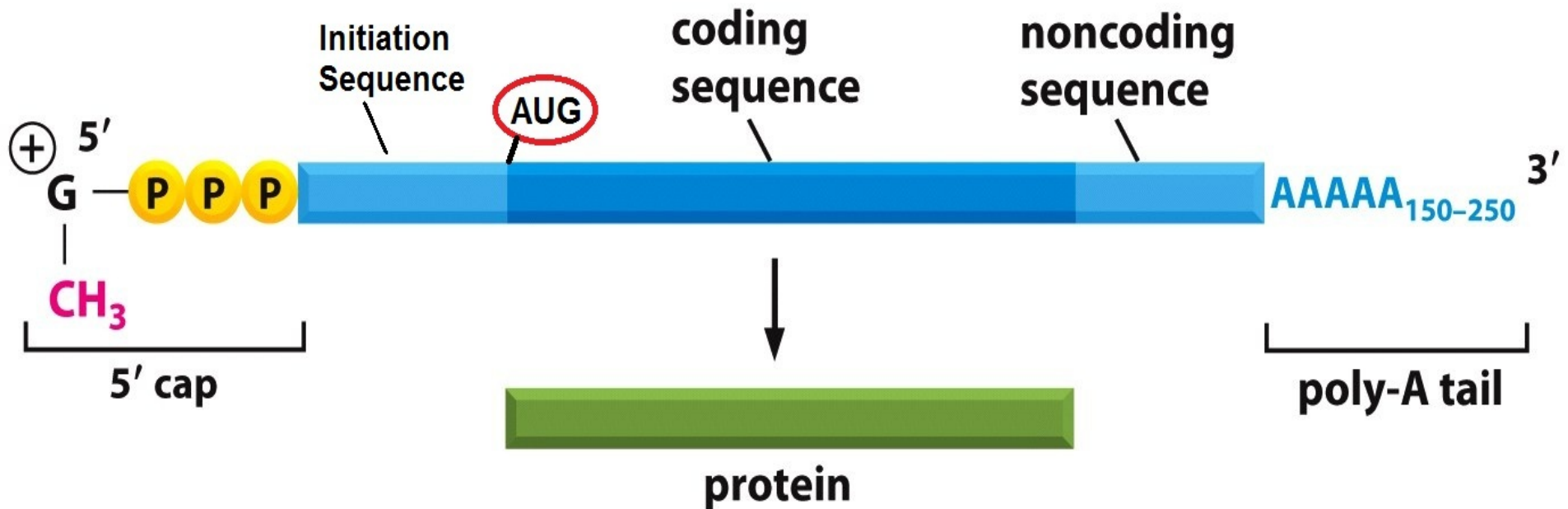
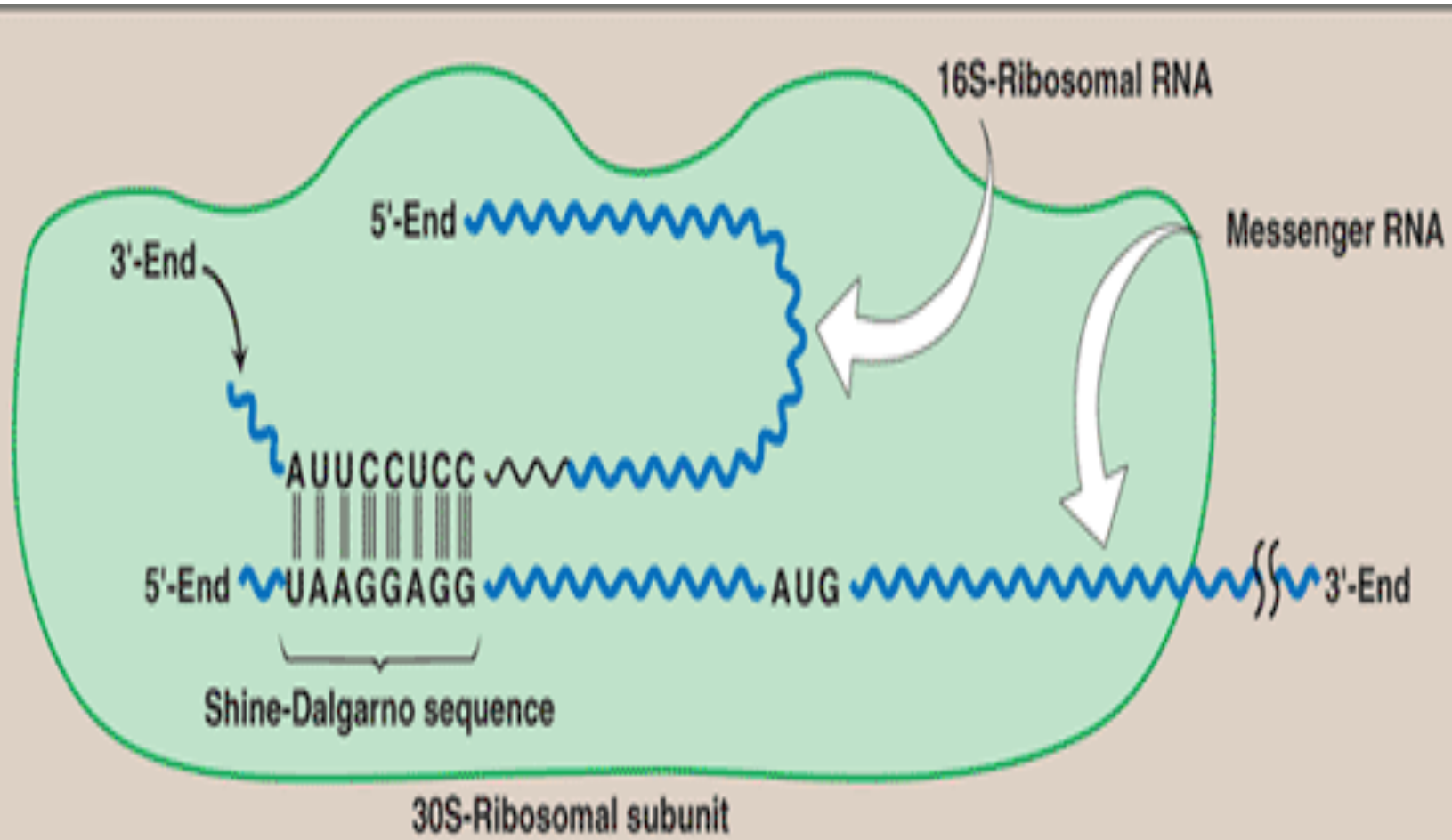


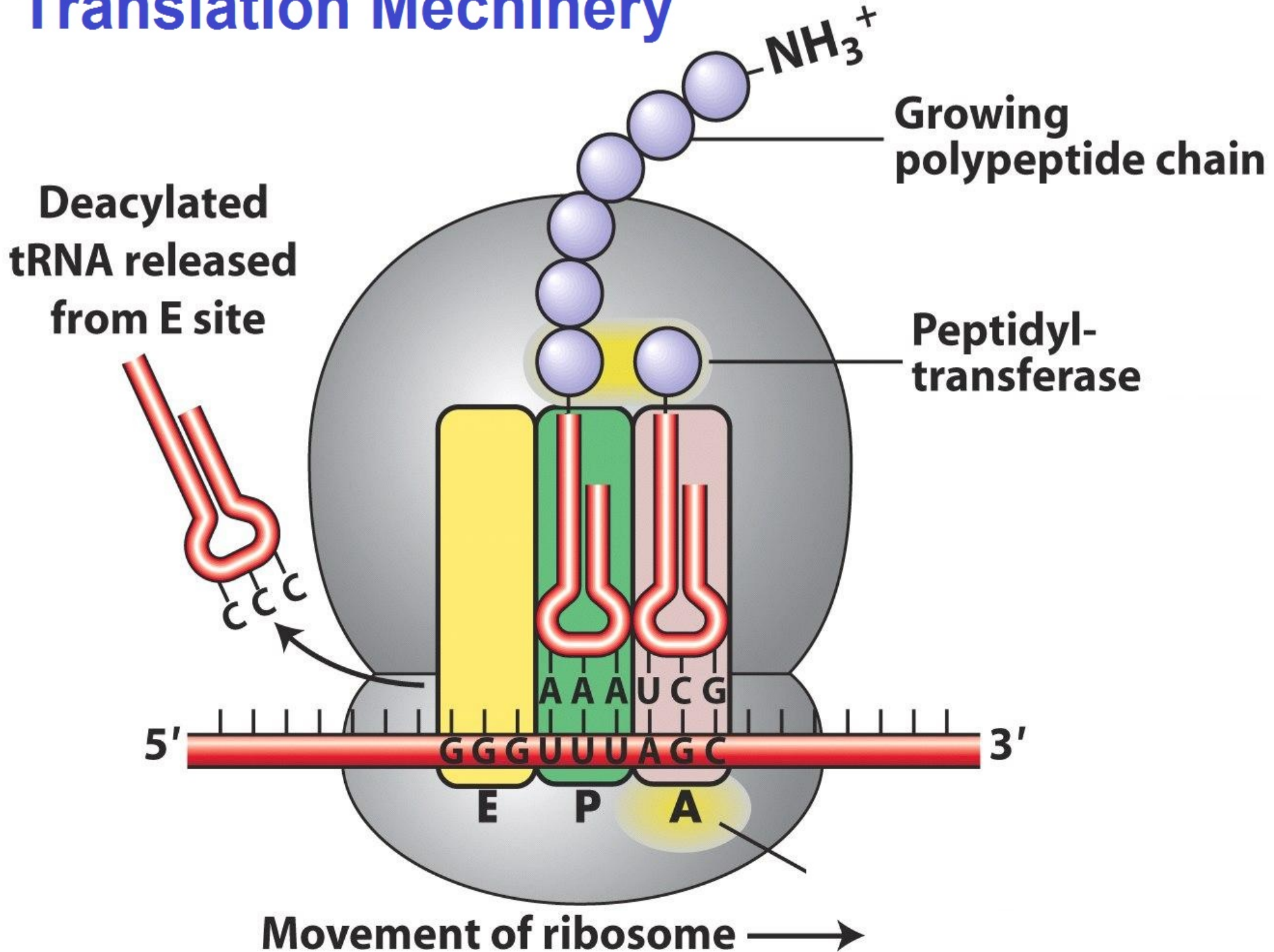
Figure 7-16a Essential Cell Biology 3/e (© Garland Science 2010)

Shine – Dalgarno Sequence

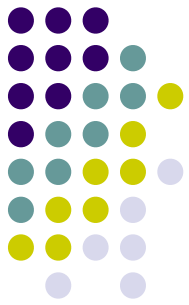
“Facilitate Initiation of Translation”



Translation Machinery



Genetic Code



- Nucleotides read in triplet “codons”
 - 5' → 3'
- Each codon translates to an amino acid
- 64 possible codons
 - 3 positions and 4 possibilities (AGCU) makes 4^3 or 64 possibilities
 - Degeneracy or redundancy of code
 - Only 20 amino acids
 - Implications for mutations

Genetic Code



		second position											
		U		C		A		G					
U	U	UUU	Phe	(F)	UCU	Ser	(S)	UAU	Tyr	(Y)	UGU	Cys	(C)
	U	UUC	Phe	(F)	UCC	Ser	(S)	UAC	Tyr	(Y)	UGC	Cys	(C)
	U	UUA	Leu	(L)	UCA	Ser	(S)	UAA	STOP		UGA	STOP	
	U	UUG	Leu	(L)	UCG	Ser	(S)	UAG	STOP		UGG	Trp	(W)
C	C	CUU	Leu	(L)	CCU	Pro	(P)	CAU	His	(H)	CGU	Arg	(R)
	C	CUC	Leu	(L)	CCC	Pro	(P)	CAC	His	(H)	CGC	Arg	(R)
	C	CUA	Leu	(L)	CCA	Pro	(P)	CAA	Gln	(Q)	CGA	Arg	(R)
	C	CUG	Leu	(L)	CCG	Pro	(P)	CAG	Gln	(Q)	CGG	Arg	(R)
A	A	AUU	Ile	(I)	ACU	Thr	(T)	AAU	Asn	(N)	AGU	Ser	(S)
	A	AUC	Ile	(I)	ACC	Thr	(T)	AAC	Asn	(N)	AGC	Ser	(S)
	A	AUA	Ile	(I)	ACA	Thr	(T)	AAA	Lys	(K)	AGA	Arg	(R)
	A	AUG	Met	(M)	ACG	Thr	(T)	AAG	Lys	(K)	AGG	Arg	(R)
G	G	GUU	Val	(V)	GCU	Ala	(A)	GAU	Asp	(D)	GGU	Gly	(G)
	G	GUC	Val	(V)	GCC	Ala	(A)	GAC	Asp	(D)	GGC	Gly	(G)
	G	GUA	Val	(V)	GCA	Ala	(A)	GAA	Glu	(E)	GGA	Gly	(G)
	G	GUG	Val	(V)	GCG	Ala	(A)	GAG	Glu	(E)	GGG	Gly	(G)

5' - BASE	MIDDLE BASE			3' - BASE	
	U	C	A		G
U	Phe	Ser	Tyr	Cys	U
	Phe	Ser	Tyr	Cys	C
	Leu	Ser	Stop	Stop	A
	Leu	Ser	Stop	Trp	G
C	Leu	Pro	His	Arg	U
	Leu	Pro	His	Arg	C
	Leu	Pro	Gln	Arg	A
	Leu	Pro	Gln	Arg	G
A	Ile	Thr	Asn	Ser	U
	Ile	Thr	Asn	Ser	C
	Ile	Thr	Lys	Arg	A
	Met	Thr	Lys	Arg	G
G	Val	Ala	Asp	Gly	U
	Val	Ala	Asp	Gly	C
	Val	Ala	Glu	Gly	A
	Val	Ala	Glu	Gly	G

1 These four rows show sixteen amino acids whose codons begin (5') with A.

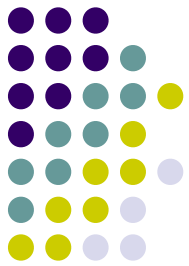
2 This column shows sixteen amino acids whose codons have the middle base U.

4 The codon 5'-AUG-3' designates methionine (Met).

3 These four, separated rows show sixteen amino acids whose codons end (3') with G.

Use of the genetic code table to translate the codon AUG

Genetic Code



1. Triplet Codons :

Each codon is a consecutive sequence of three bases

2. Non-overlapping :

Codes are always read one after another.

3. Non-punctuated :

Codes are always continues.

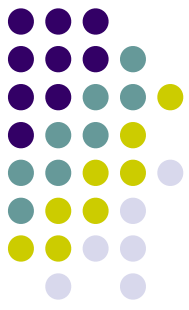
4. Specificity (unambiguous) :

One codon always codes for specific amino acid

5. Universal :

Codons represent same amino acid in all species.

Except in Mitochondria



6. **Degenerate :**

Each codon corresponds to a single A.A.

But A.A. may have more than one triplet codon.

7. **Initiator codon :**

AUG is start codon

8. **Termination codons**

Nuclear DNA = UAA, UGA, UAG,

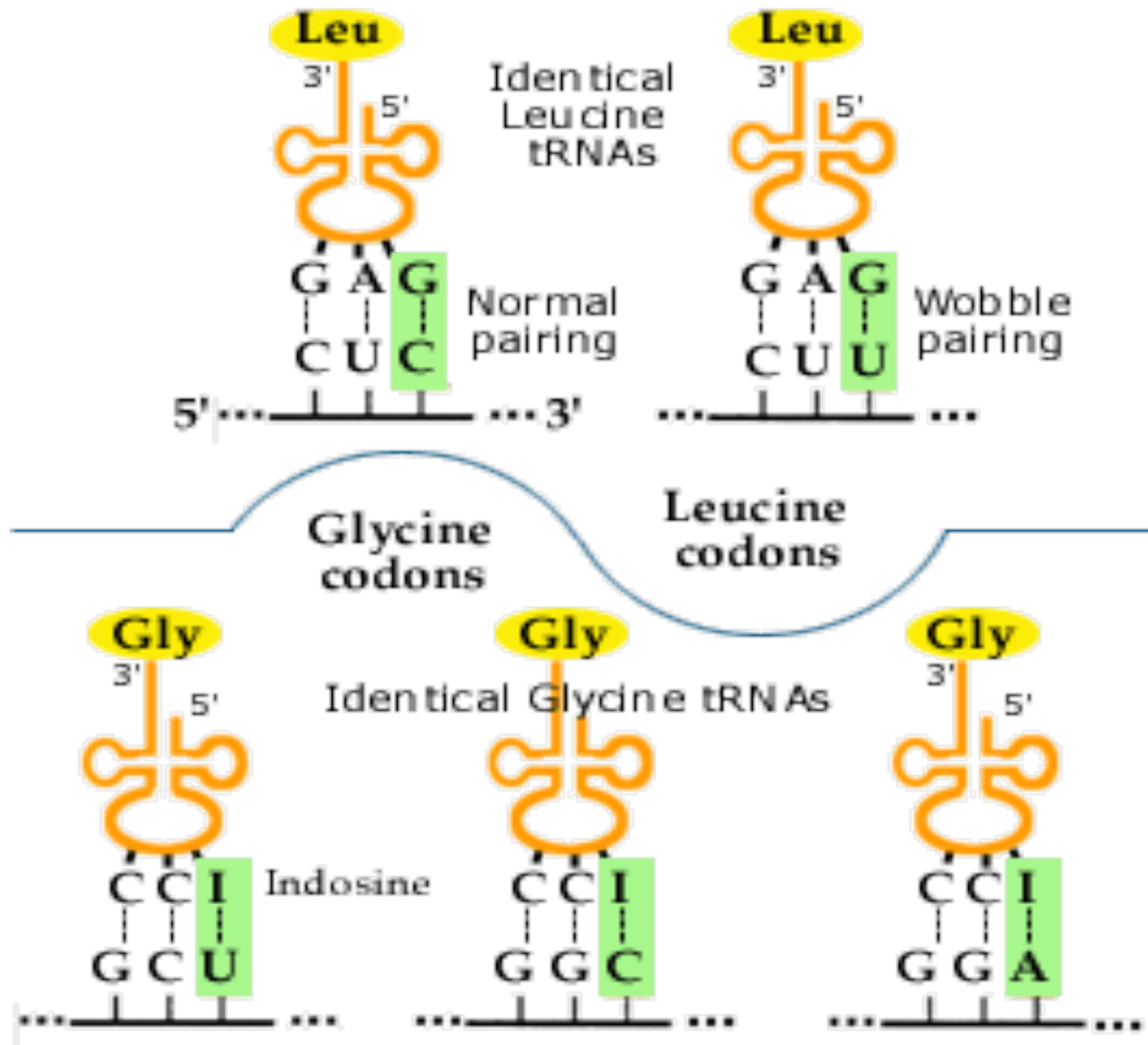
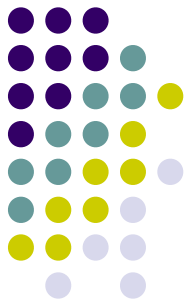
Mitochondrial DNA = AGG, AGA,

9. **Wobbling Phenomenon :**

The reduced stringency between the third base of the codon and the complementary nucleotide in the anticodon is called wobble.

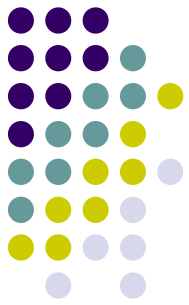
It reduce effect of mutation.

Wobbling Phenomenon





Mutation



Mutation

Vs

Damage

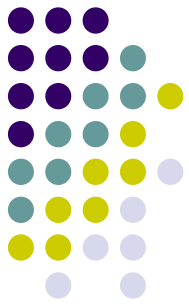
Vs

Polymorphism

Vs

Substitution

Evolution



>>>Before 10,000 Year Ago

.....Hunting was source of Food

>>>Than Human invented

.....Agriculture

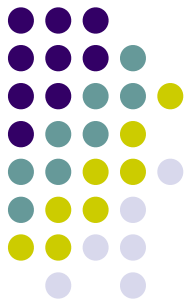
>>>Until Human unable to digest milk,

.....**mutated lactose enzyme gene**

Very High Number of Lactose Intolerance

Than We evolved.....

Same you can think what capacity do you like you
generated for 1000 stream down sons or daughters



**Find Prevalence of Tuberculosis
in Last 100 years**

Vs

**Find Prevalence of HIV
in Last 100 years**

Philosophy of Genetical Evolution

Life exists entirely
in the margin of error
between damage and perfect repair.

Evaluation occurs simply,
If Population survive with the tolerable mistake

Philosophy of Genetical Evolution

Matching Perfectly With Life

Life exists entirely

in the margin of error

between damage (**Multiple GF/BF**) and
perfect repair (**Breakup and Find New**).

Evaluation occurs simply,

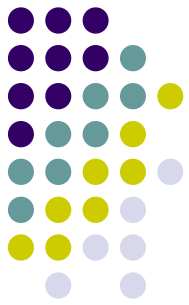
If Population survive with the tolerable mistake
(marriage)

Mutation - Tumour Suppressor Gene



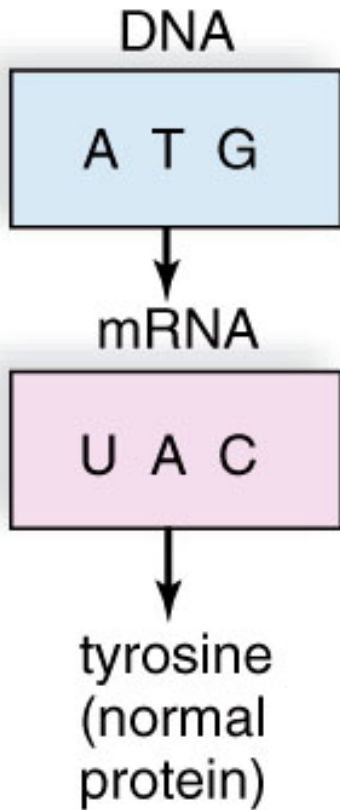
- » p53 Gene = p53 protein = 7 domain
- » One of Domain Work as
 - » DNA Binding Protein (Rich In Arginine)
- » p53 = 37% chance of Mutation in Cancer
 - » Colon
 - » Breast
 - » Lungs
- » No Synthesis of p53 Proteins
- » No Binding with DNA for it's regulation
- » Over Production of NPM1 (Nucleophosmin) gene
- » Leukemia

Point Mutation

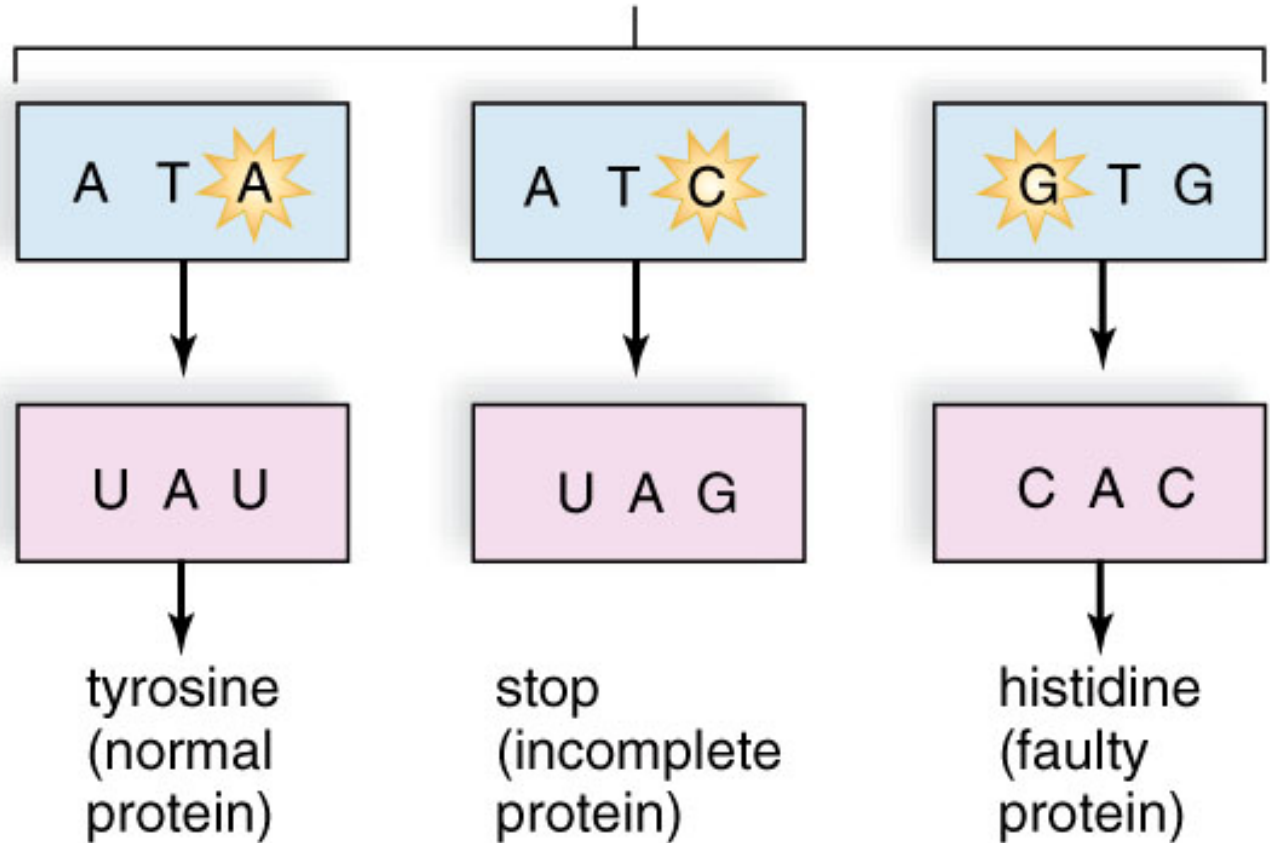


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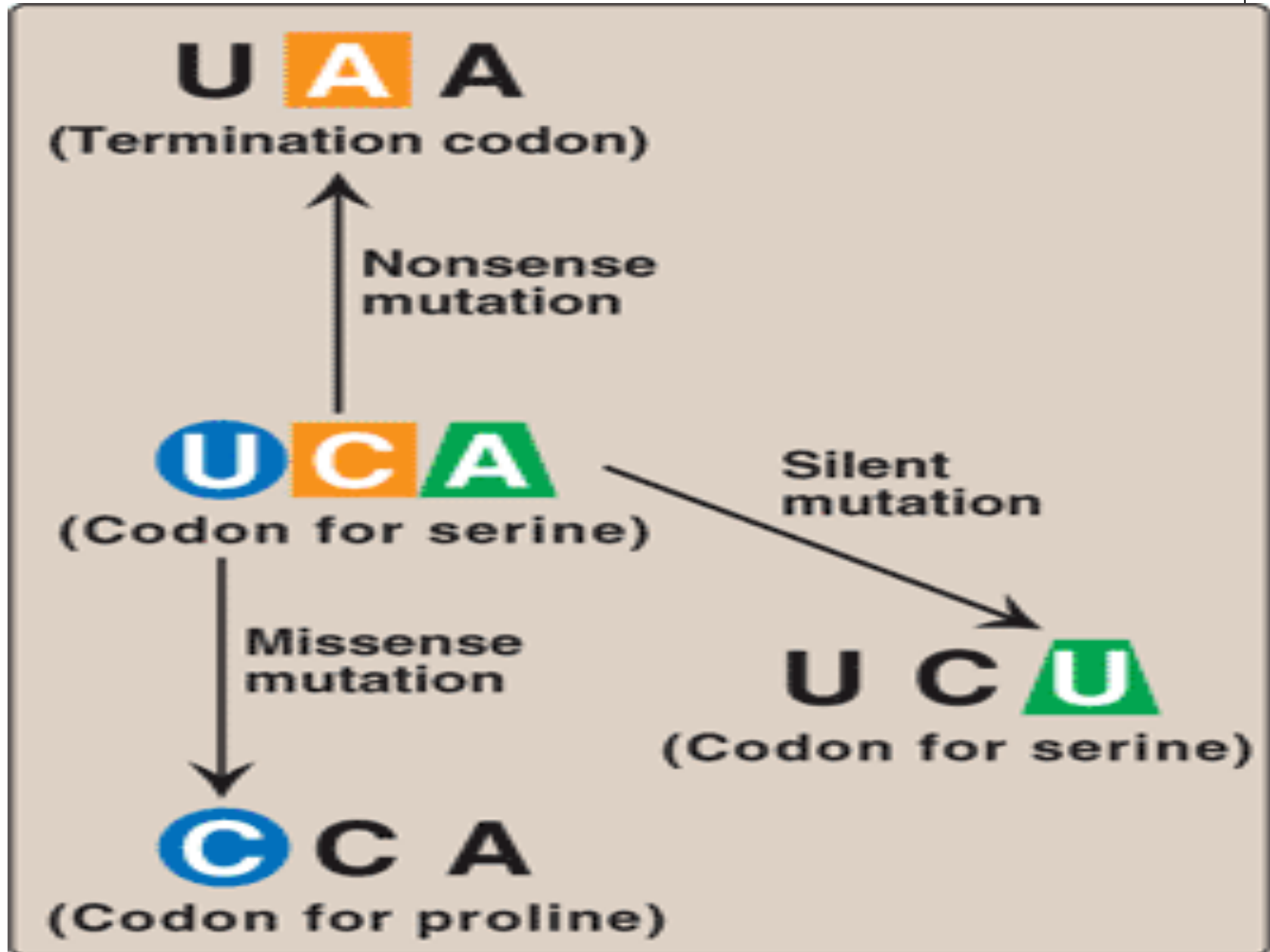
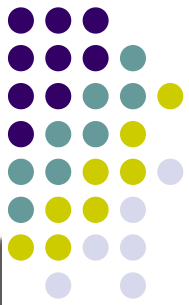
No mutation



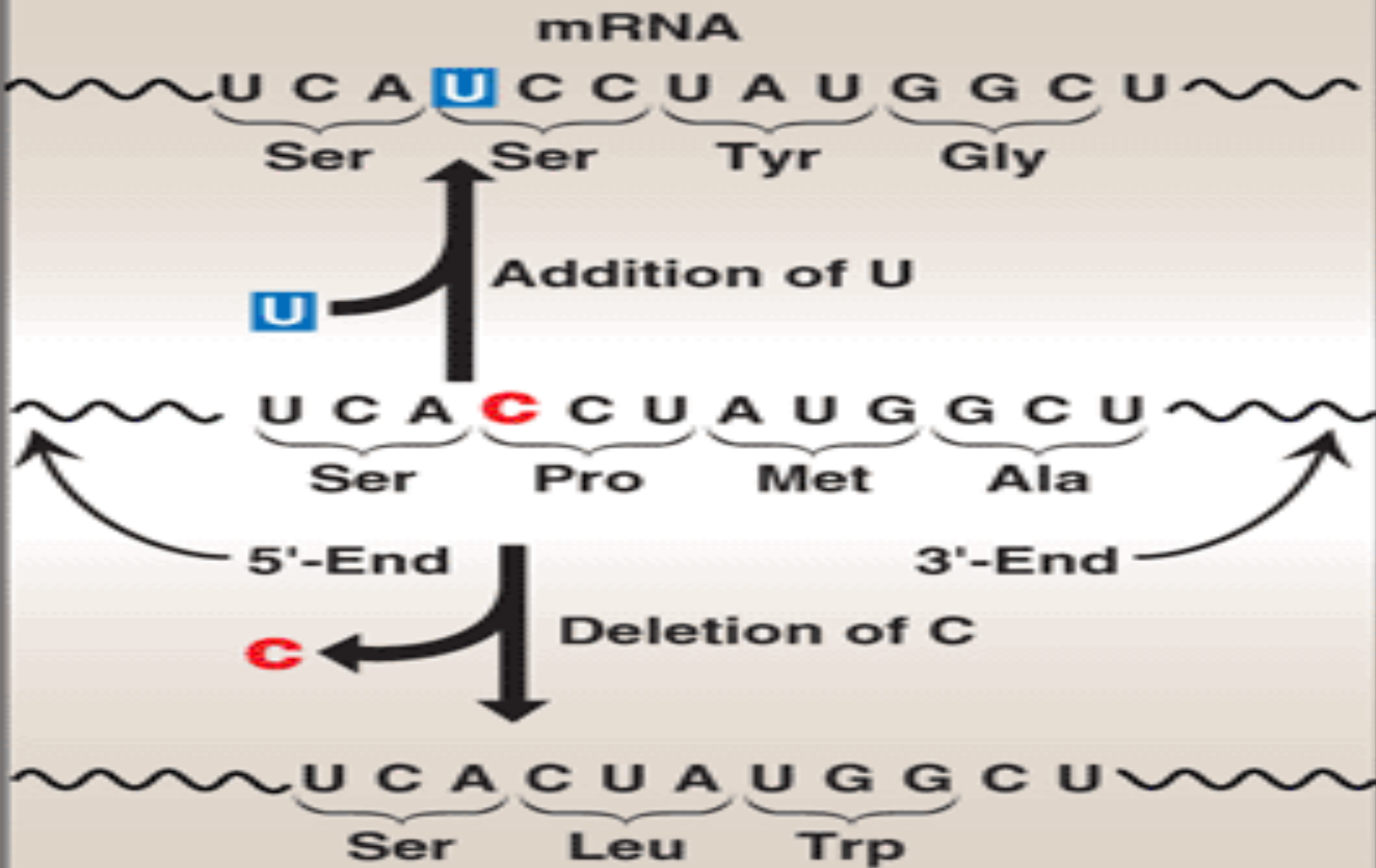
Point mutations



Effect Of Mutation

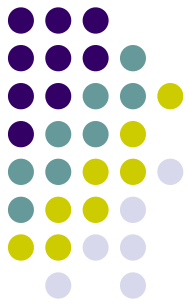


Addition of base



Deletion of base

Insertion Type of Mutation



DNA Sequence

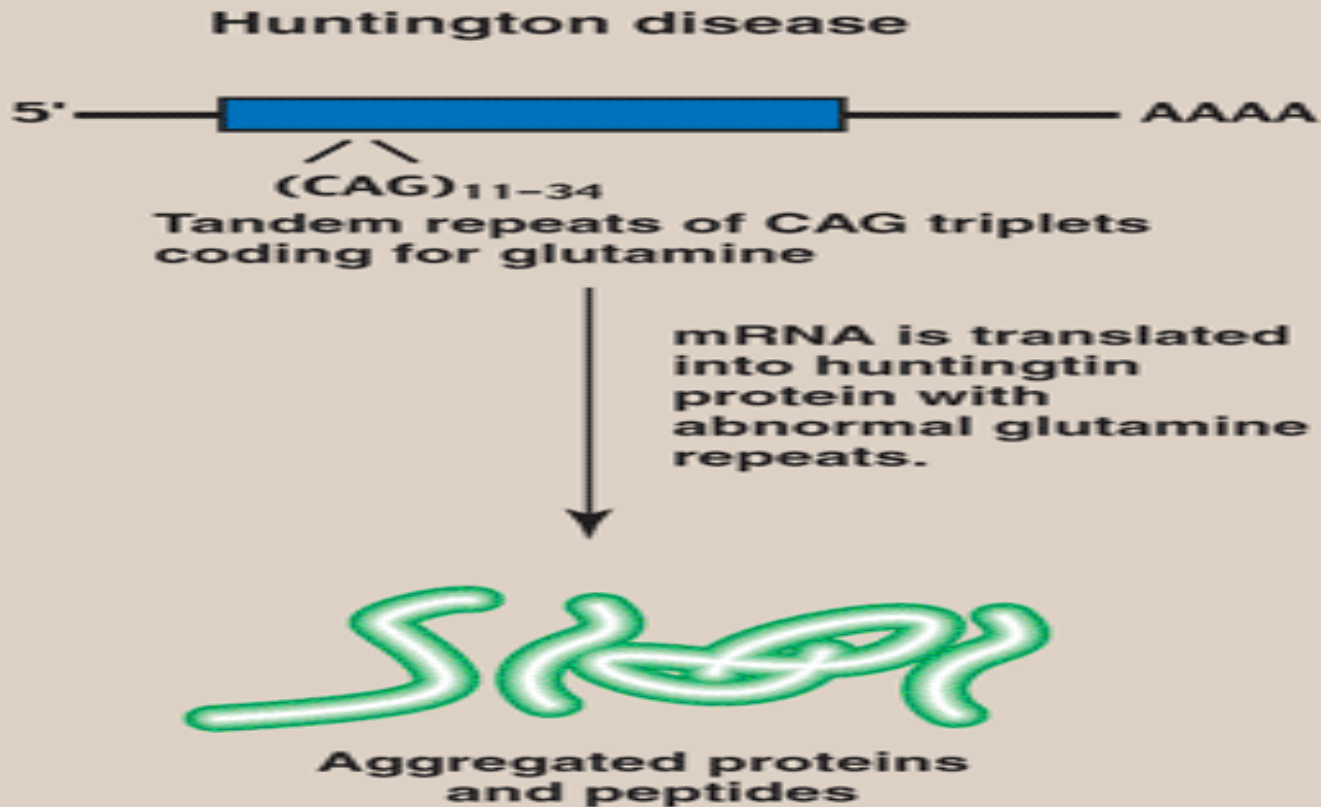
Amino Acid Sequence

Normal: CAG CCC ACT → Gln - Pro - Thr
Codon 1 Codon 2 Codon 3

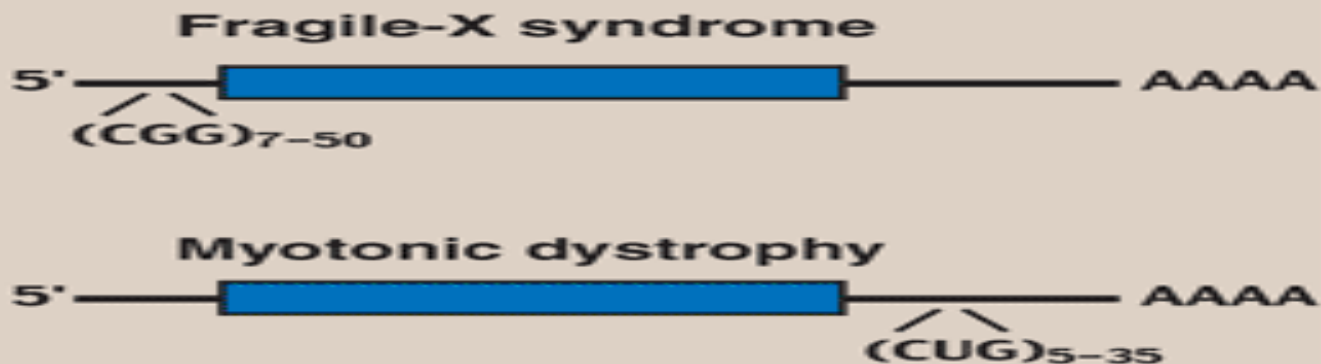
Insertion Mutation (Frameshift): CAG TCC CAC T → Gln - Ser - His ?
Codon 1 Codon 2 Codon 3 Codon 4

Insertion Mutation (Non-frameshift): CAG TTT CCC ACT → Gln - Phe - Pro - Thr
Codon 1 Codon 2 Codon 3 Codon 4

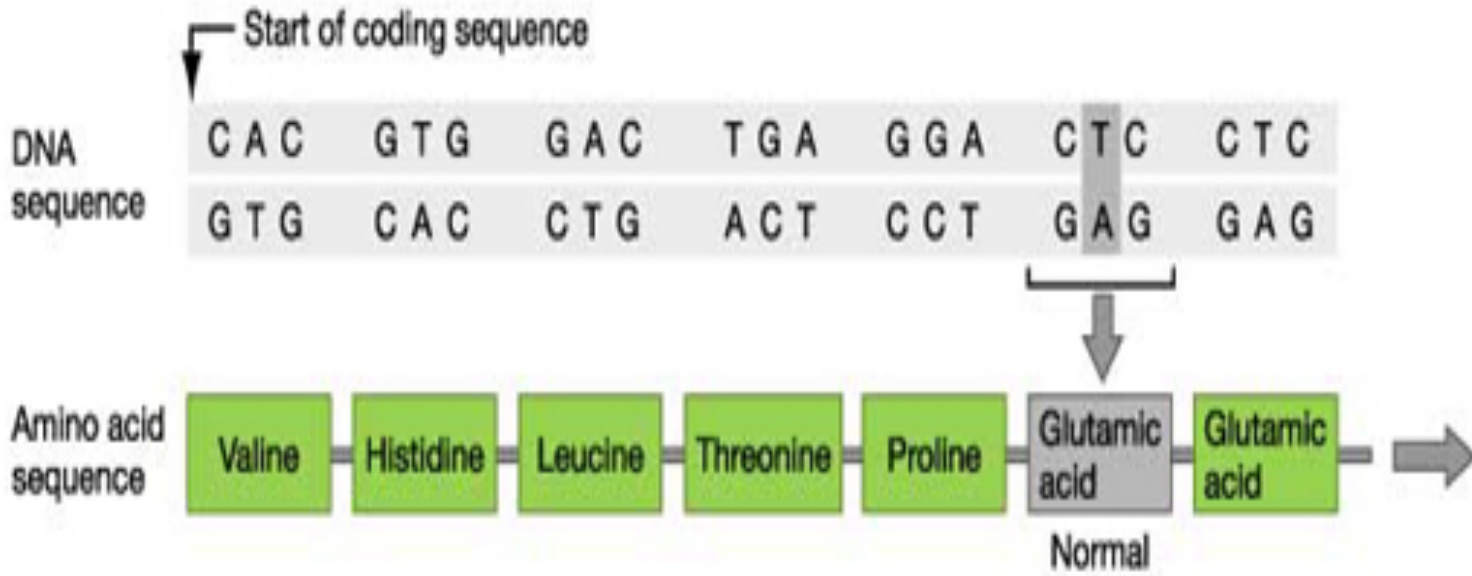
Huntington disease



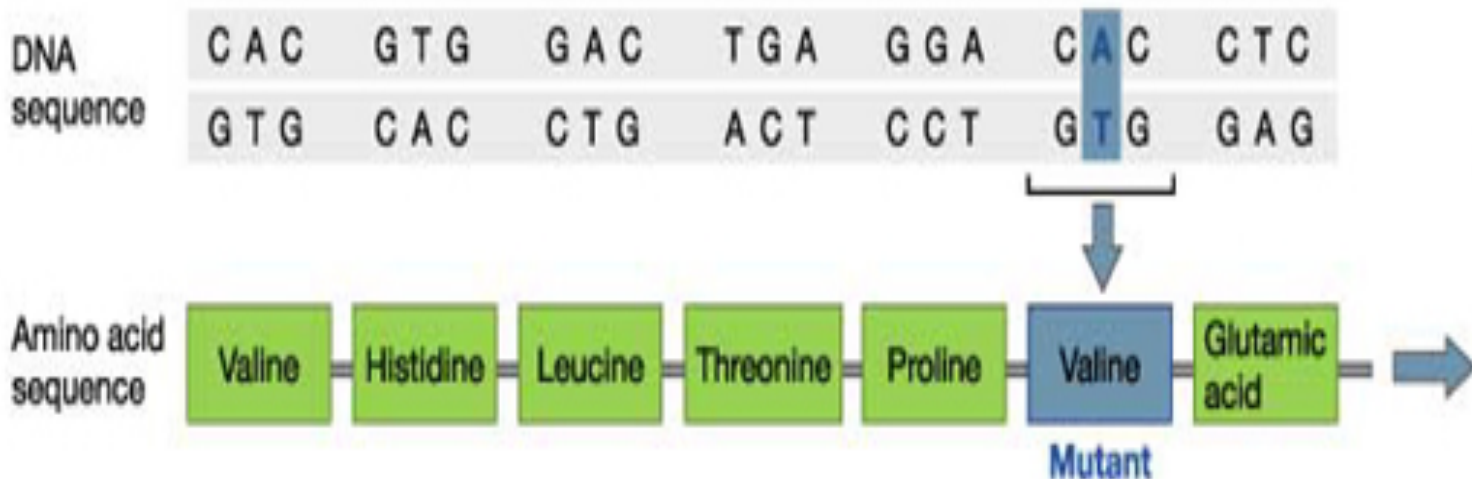
Other triplet expansion diseases



Point Mutation - Mis-Sense



Normal red blood cells



Sickled red blood cells

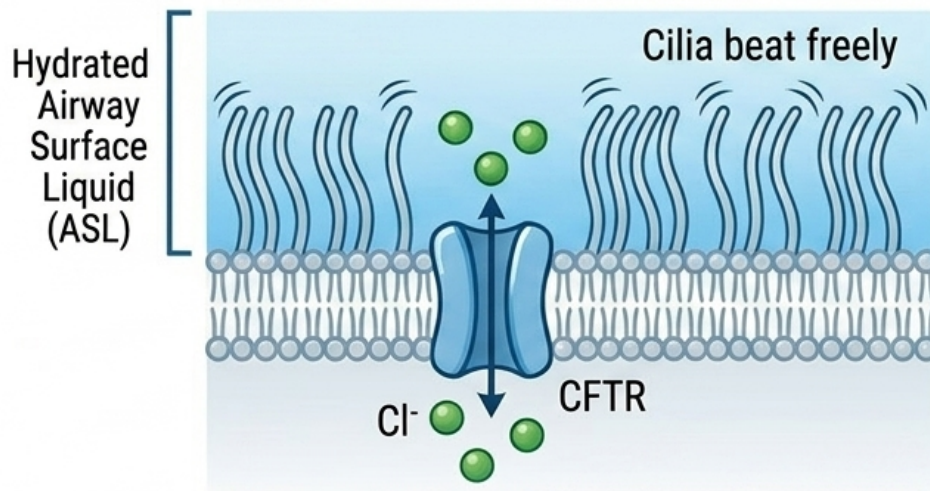
Deletion Type of Mutation

Deletion of “CTT” code from CFTR Gene

CFTR = Cystic Fibrosis Transmembrane Conductance Regulator

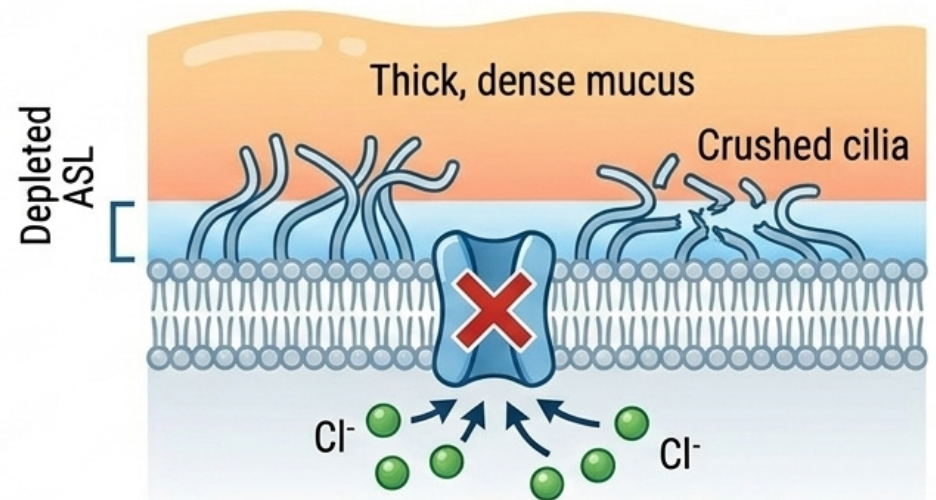
The Mechanics of the CFTR Channel

Functional CFTR



- Cl⁻ moves freely
- Hydrated Airway Surface Liquid (ASL)
- Cilia beat freely

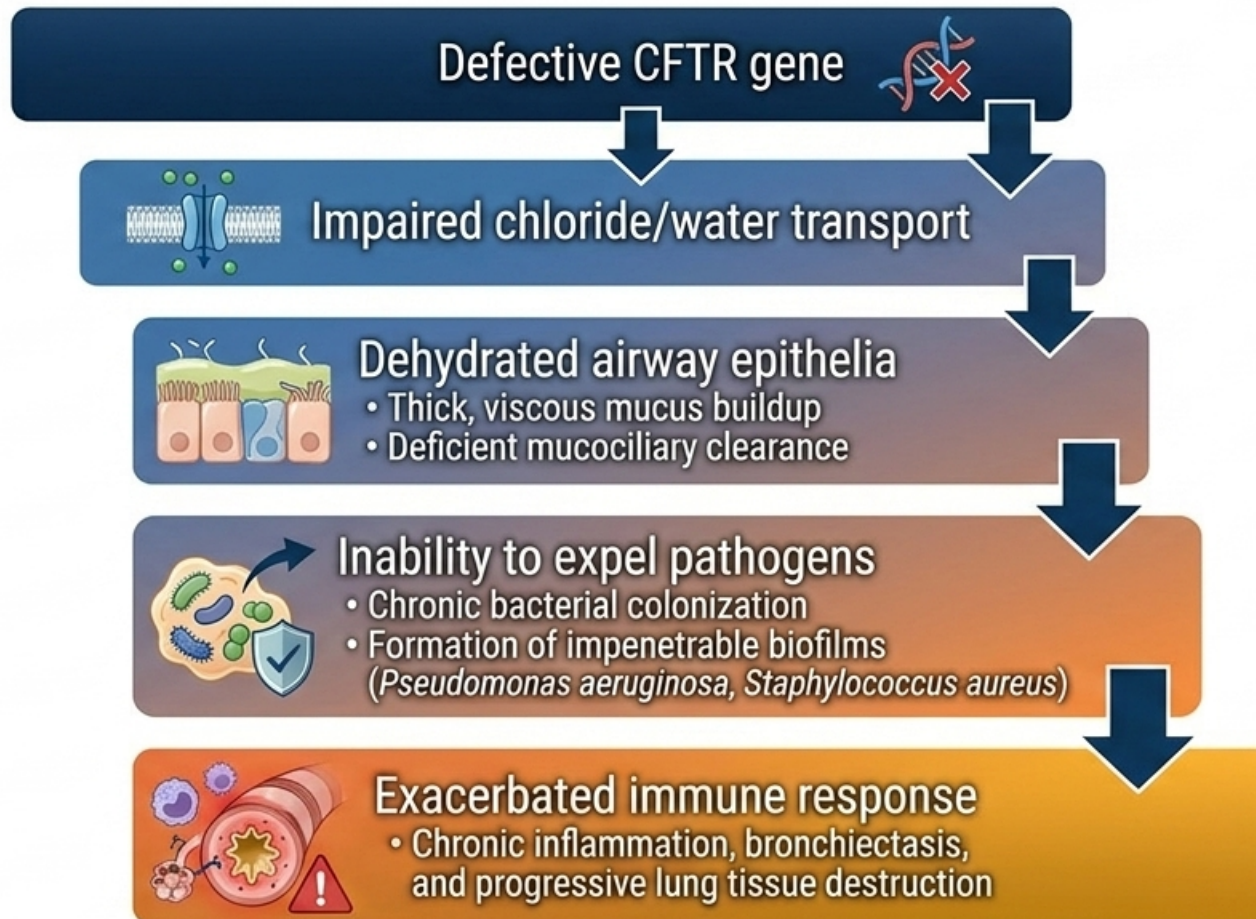
Blocked/Absent CFTR



- Cl⁻ trapped inside
- Depleted ASL
- Thick, dense mucus crushes cilia

Cystic Fibrosis

The Pathophysiology Cascade



Clinical Feature in Cystic Fibrosis

- GIT
- Respiratory
- Fallopian Tube
- Seminal Vesicles
- Pancreatic duct
- Billiary System

Mutation



Point Mutation

Effect of Mutation

Substitution Mutation

Deletion Mutation

Insertion Mutation

Transition Mutation
Purine to Purine

Transversion Mutation
Purine to Pyrimidine

Gene Deletion
Thalassemia

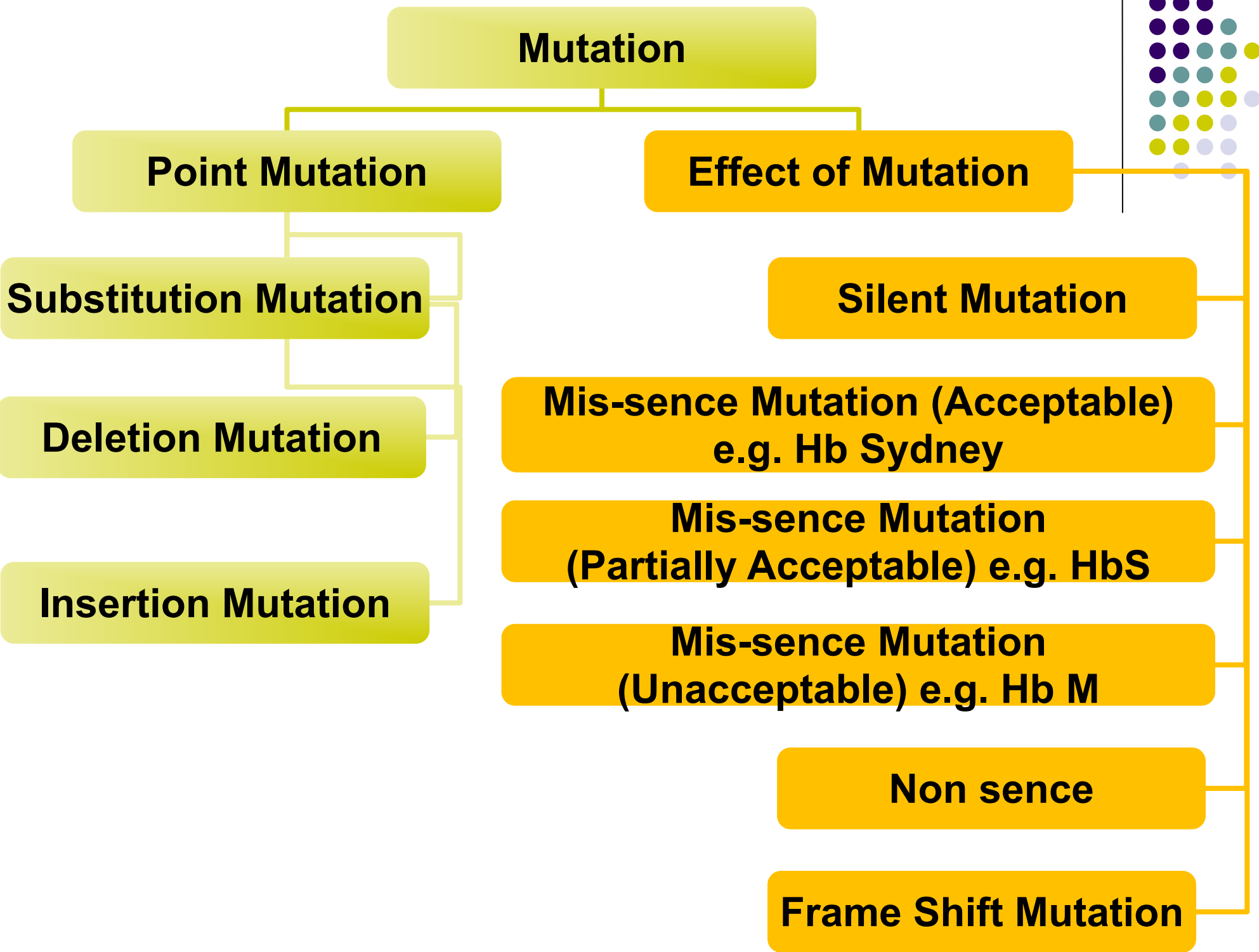
Codon Deletion
Cystic fibrosis

Base deletion
Frame Shift Mutation

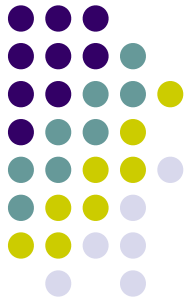
Gene Insertion
Duchenne Muscular Dystrophy

Codon Insertion
Huntington's chorea

Base deletion
Frame Shift Mutation



Substitution Mutation

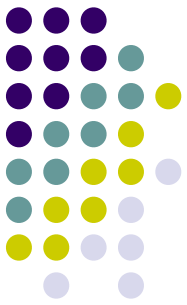


- **Transition**

- Purine replace by Purine
- Pyrimidine replace by Pyrimidine

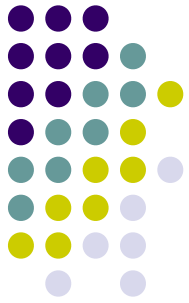
- **Transversion**

- Purine replace by Pyrimidine
- Pyrimidine replace by Purine
- E.g. Sickle cell anaemia
- GAG (glutamic acid)= GUG (valine)



Translation (Protein Synthesis)

Translation



I Steps:

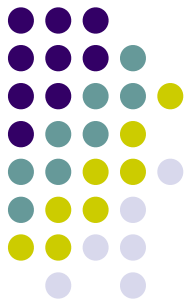
I Initiation

I Elongation

I Termination

I Post - Translation

Translation Process



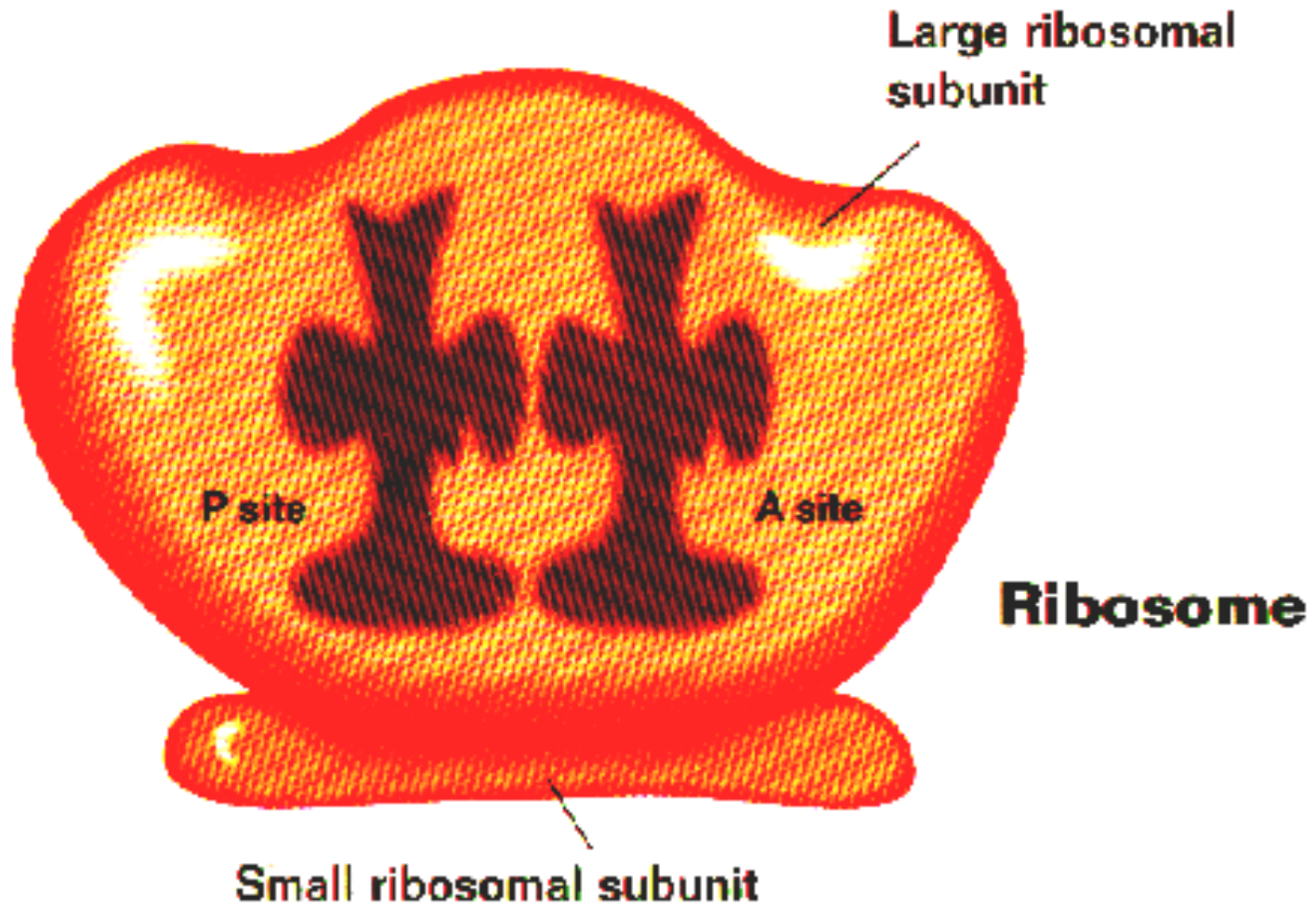
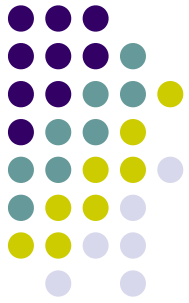
I Requires

- I Ribosomes (50s + 30s)
- I r-RNA
- I t-RNA
- I m-RNA
- I Amino acid

I Ribosome

- I Made of protein and r-RNA (Nucleo-protein)
- I Has internal sites for 2 t-RNA molecules.
- I Two subunit
 - § Prokaryotic 50S + 30S subunits = form a 70S.
 - § Eukaryotic 60S + 40S subunits = form an 80S.

Ribosome



Large ribosomal subunit

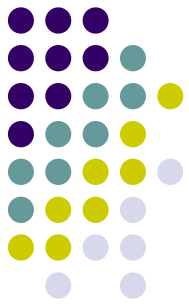
P site

A site

Ribosome

Small ribosomal subunit

Translation



I Initiation

- I Ribosomal subunits **assemble** on mRNA
- I r-RNA aids in binding of mRNA

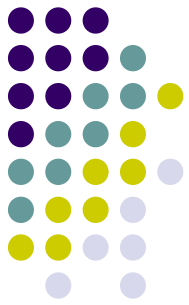
I Elongation

- I **t-RNAs** with appropriate anticodon loops **bind to complex**
- I have **amino acid attached** (done by other enzymes)
- I **Amino acids transfer** from t-RNA 2 to t-RNA 1
- I Process repeats

I Termination

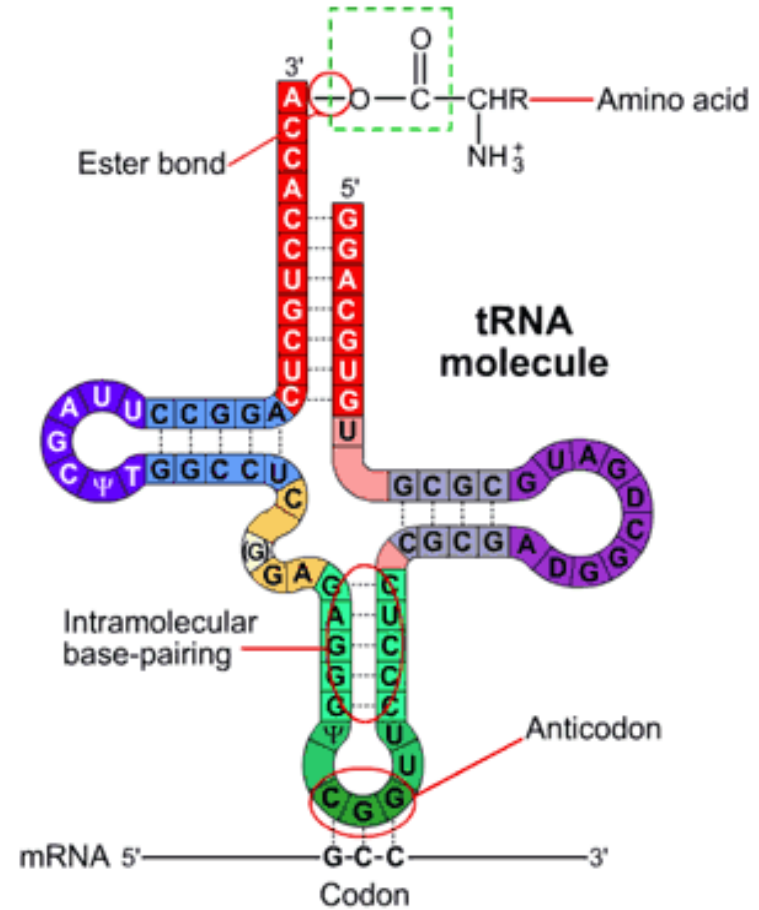
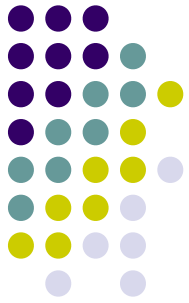
- I t-RNA with **stop codon** binds into ribosome
- I No amino acid attached to t-RNA
- I Complex falls apart

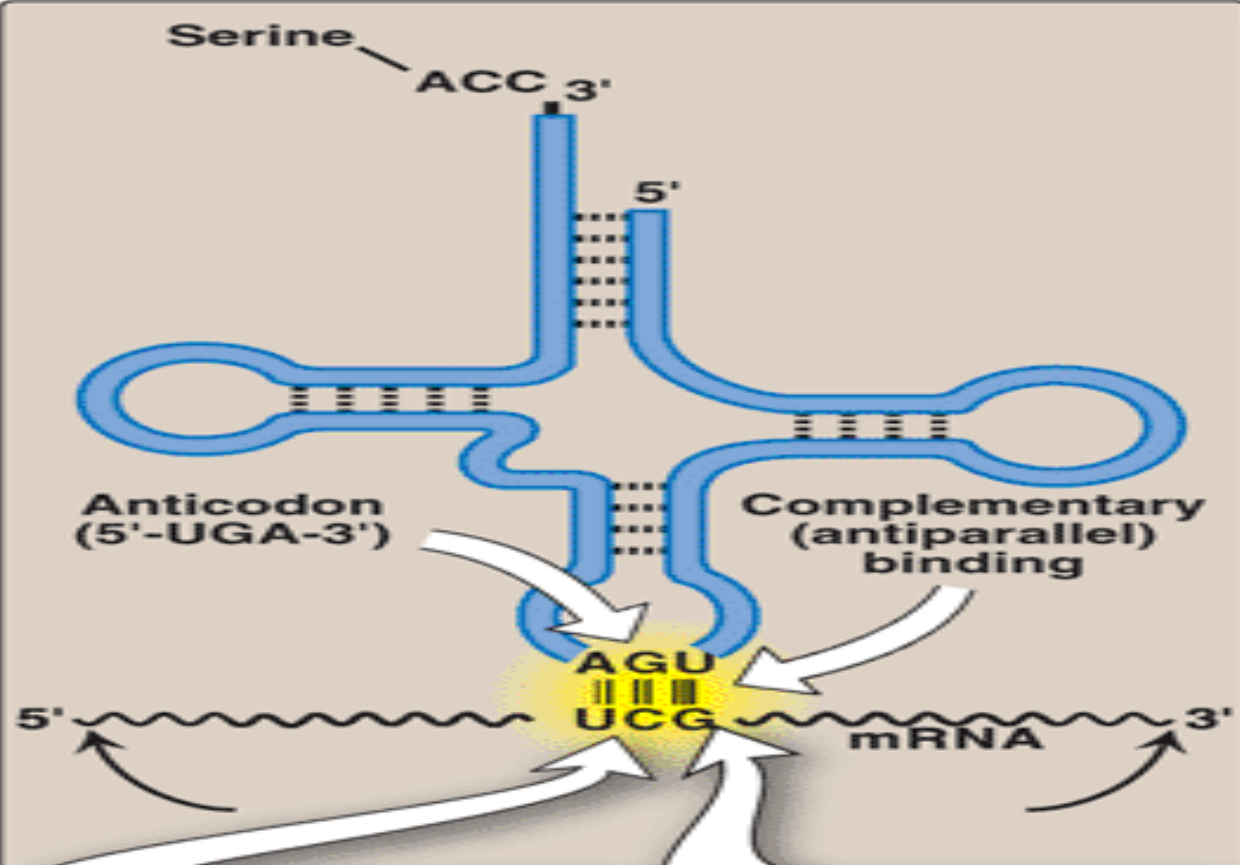
A – P site on ribosome



- | Ribosome has two binding sites for t-RNA
 - **P & A** sites —
- | Together, they cover two neighboring codons.
- | **P-site** binds
 - | codon is occupied by **Peptidyl t-RNA**.
 - | This t-RNA carries the **chain of amino acids** that has already been synthesized.
- | **A site** binds
 - | incoming **Aminoacyl-tRNA** as directed by the codon.
 - | This codon **specifies the next amino acid** to be added to the growing peptide chain.

Transfer RNA





Traditional base-pairing observed in first and second positions of codon:

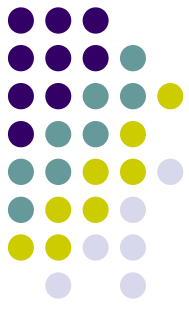
tRNA	mRNA
A	U
G	C
U	A
C	G

Nontraditional base-pairing is possible between the third (3') position of the codon and the first (5') position of the anticodon:

tRNA	mRNA
A	U
G	C
U	A
C	G
H	U
	C
	A

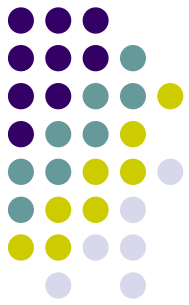


Aminoacyl-tRNA synthetases

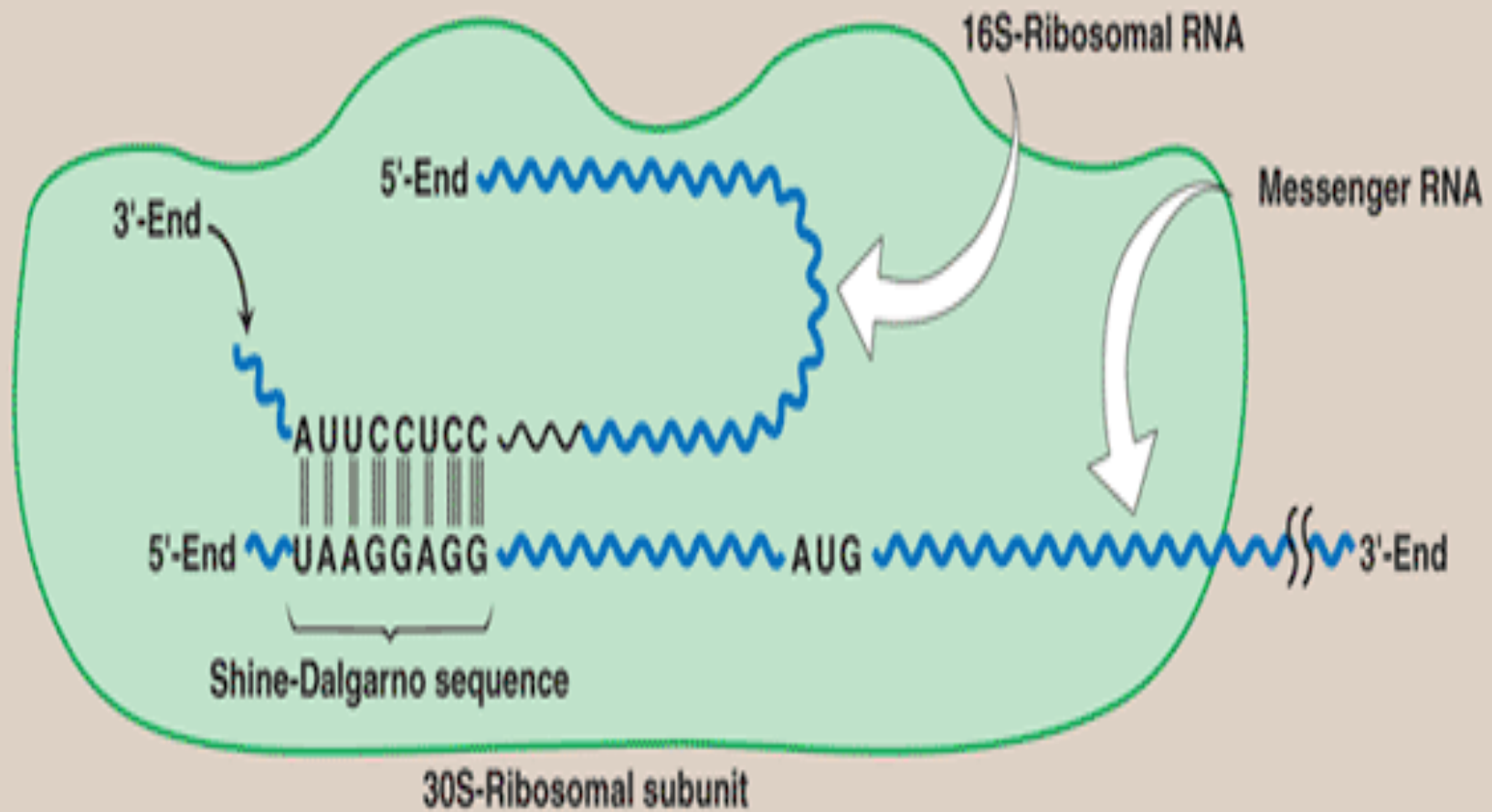


- I Required for attachment of amino acids to their corresponding t-RNA.
- I Carboxyl group of an amino acid to the 3'-end of t-RNA.
- I Require ATP.
- I Extreme specificity
- I High fidelity of translation of the genetic message.
- I Enzyme have “proofreading” or “editing” activity

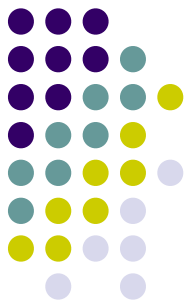
Initiation



- | Involves the assembly of the components
- | Components include
 - | Two ribosomal subunits.
 - | m-RNA to be translated.
 - | Aminoacyl t-RNA specified by the first codon
 - | GTP
 - | Initiation factors
 - | In Prokaryotes, Initiation factors (**IF-1, IF-2, IF-3**)
 - | In eukaryotes, more than 10 (designated eIF).



Complementary binding between prokaryotic mRNA Shine-Dalgarno sequence and 16S rRNA.



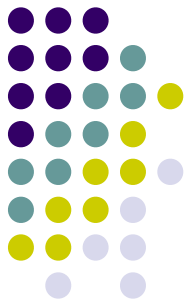
I Shine - Dalgarno (SD) sequence

- I Purine rich sequence (e.g. 5'-UAAGGAGG-3')
- I 6 - 10 bases upstream of the initiating AUG codon
- I Near m-RNA 5'-end.
- I 16S r-RNA of the 30S ribosomal subunit has complementary base pair at 3'-end.
- I Facilitating the binding of the 30S ribosomal subunit on the mRNA

I In eukaryotes

- I the 40S ribosomal subunit binds to the cap structure at the 5'-end of the mRNA
- I By eIF-4 and moves down the mRNA until it encounters the initiator AUG.
- I This “scanning” process requires ATP.

Initiation codon

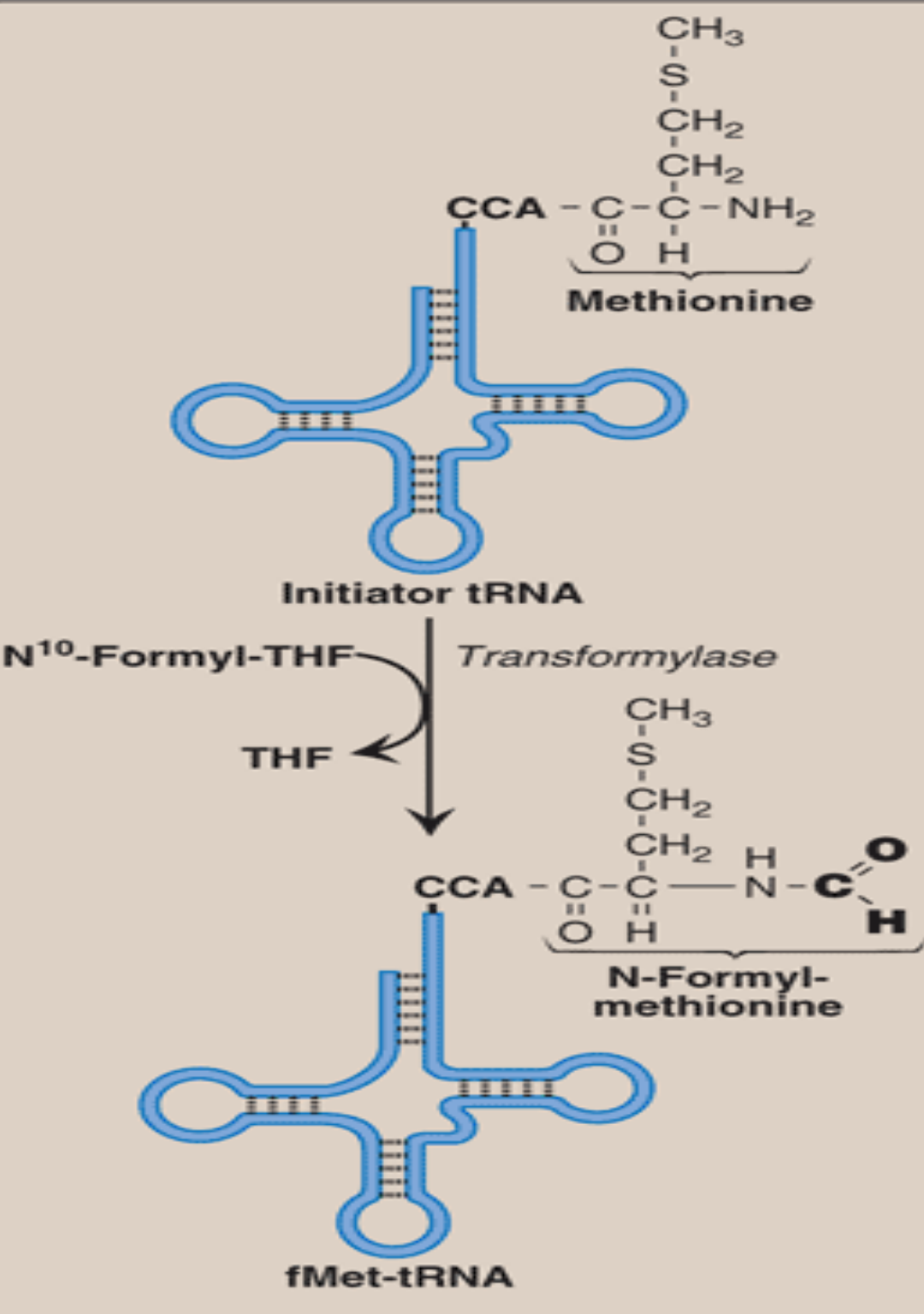
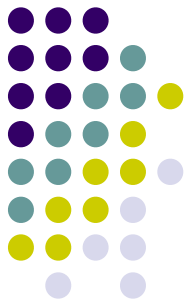


- | **AUG** is recognized by initiator t-RNA.
- | **Recognition** is facilitated by **IF-2** (bound to **GTP**).

- | Initiator **t-RNA** enters the ribosomal **P** site
- | **GTP** is hydrolyzed to **GDP**.

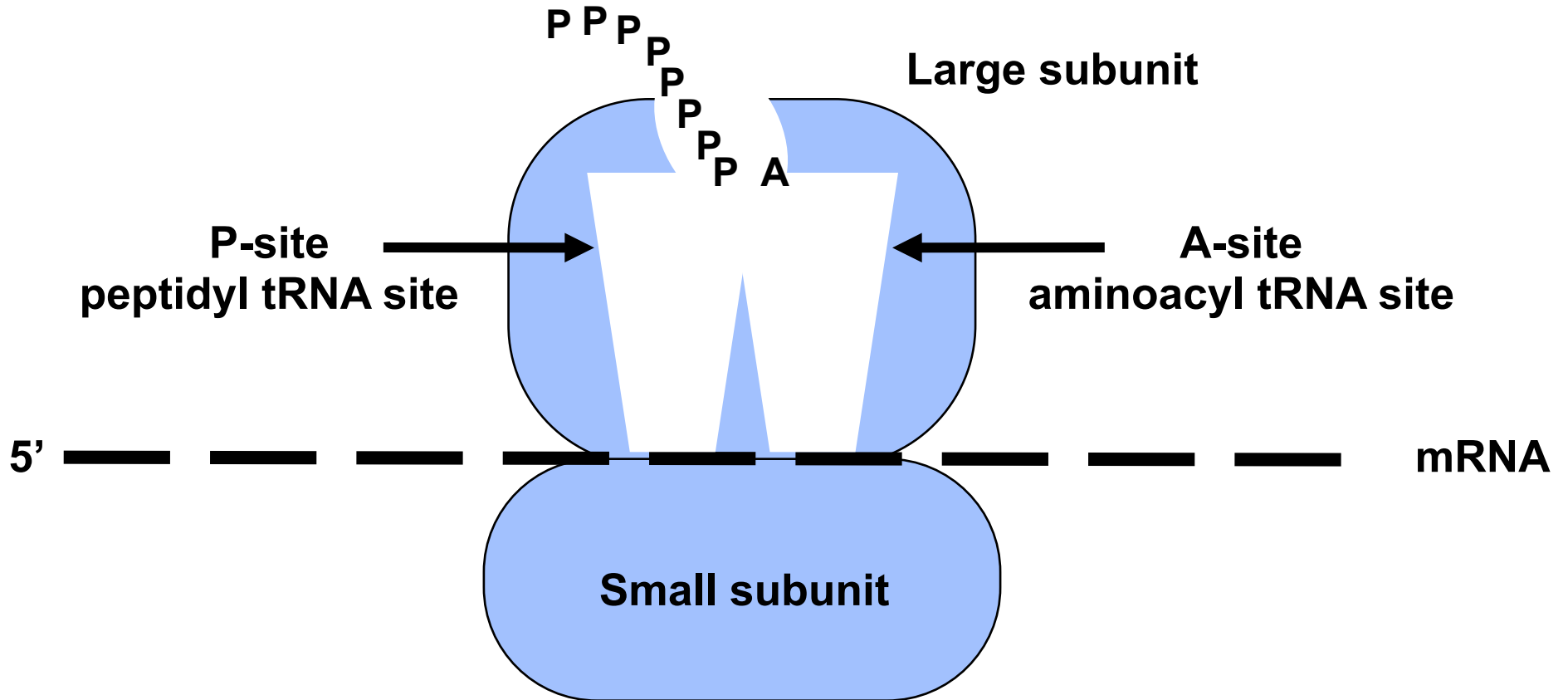
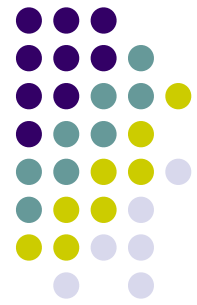
- | Initiator t-RNA carries **N-formylated methionine**.
- | N10-formyl tetrahydro**folate** as the carbon donor.

- | In eukaryotes, the initiator tRNA carries a methionine that is not formylated.

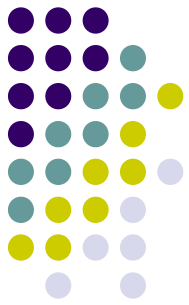


Generation of the initiator
N-formylmethionyl-tRNA
(fMet-tRNA)

Ribosome structure



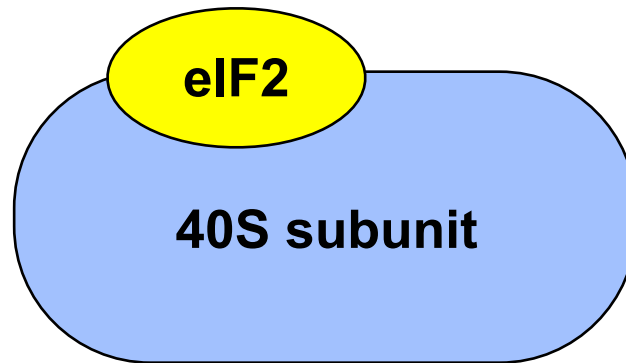
Ribosome with bound tRNAs and mRNA



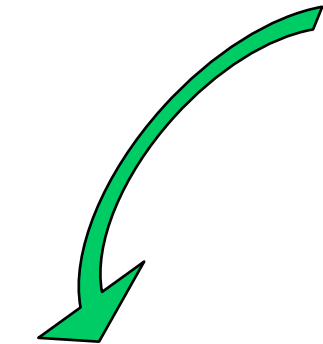
Initiation of protein synthesis: mRNA binding

M

Initiator tRNA bound to the small ribosomal subunit with the eukaryotic initiation factor-2 (eIF2)

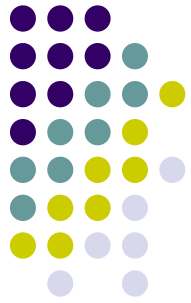
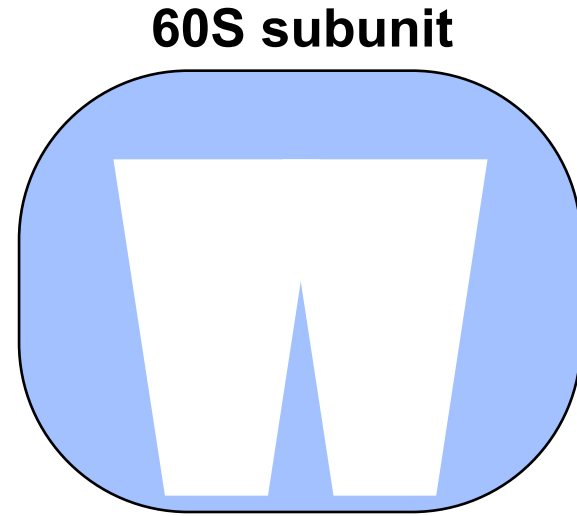


The small subunit finds the 5' cap and scans down the mRNA to the first AUG codon

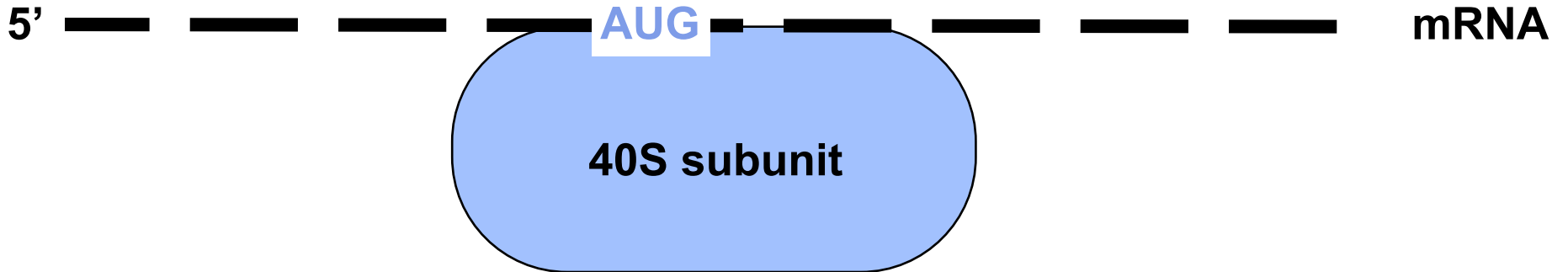


5' cap ■■■■■ ■■■■■ ■■■■■ ■■■■■ ■■■■■ ■■■■■ **AUG** ■■■■■ ■■■■■ **mRNA**

- the initiation codon is recognized
- eIF2 dissociates from the complex
- the large ribosomal subunit binds



M

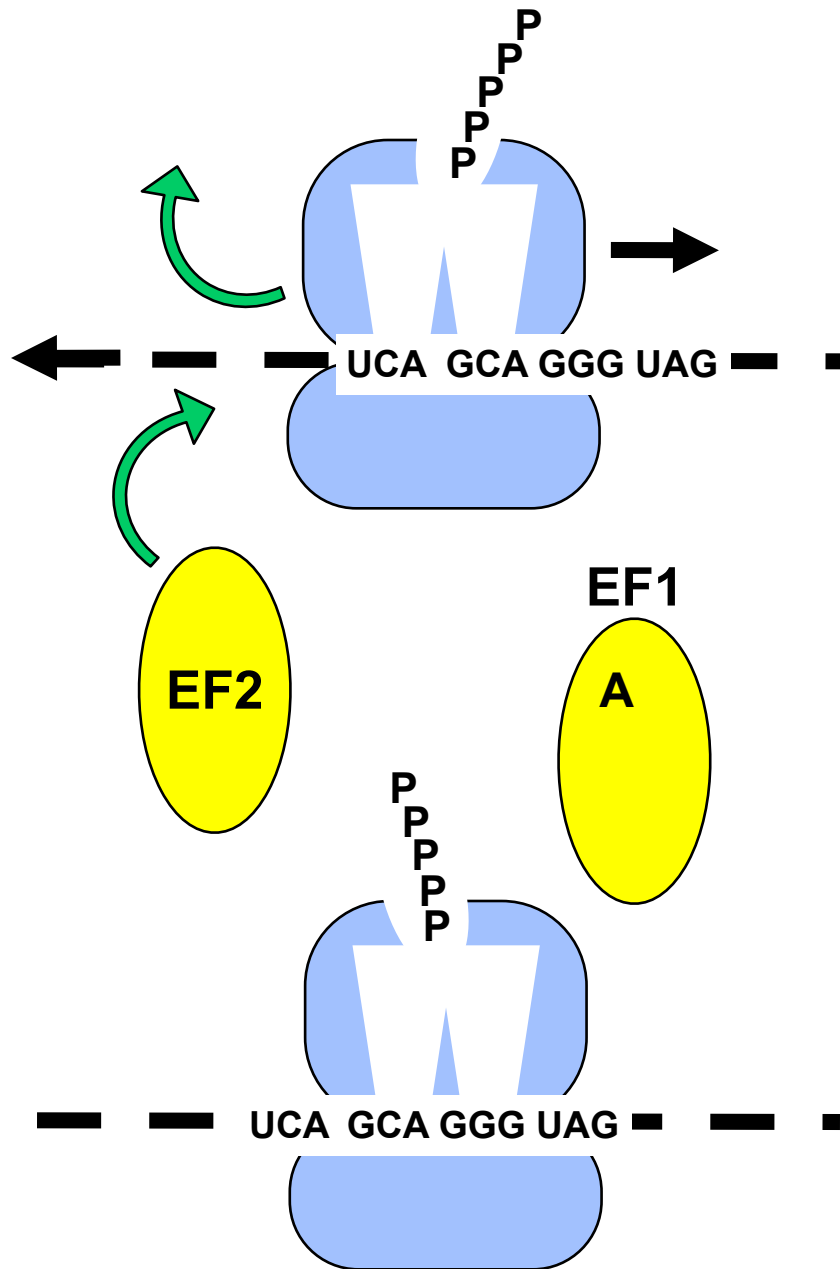


Elongation

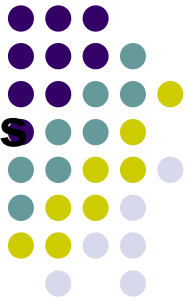


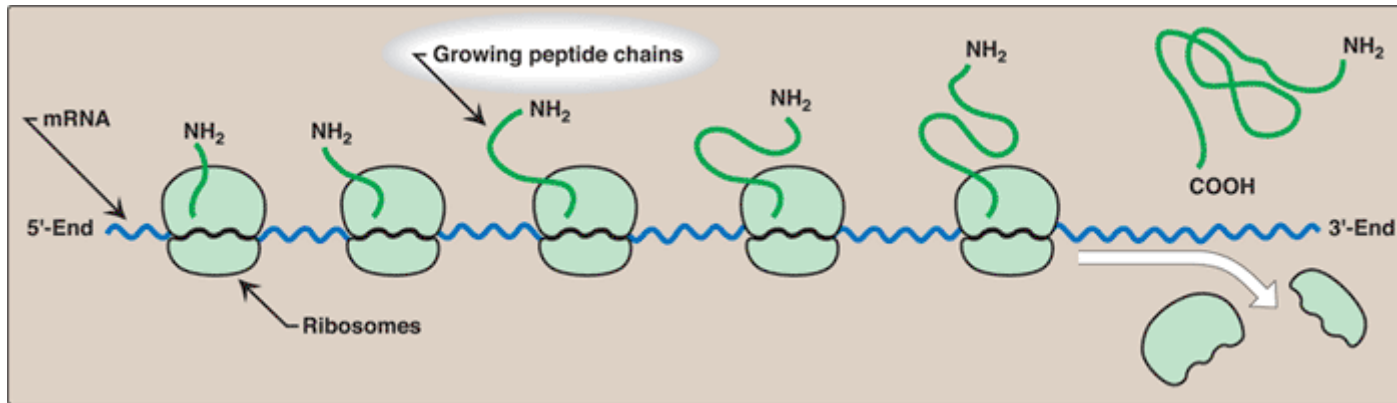
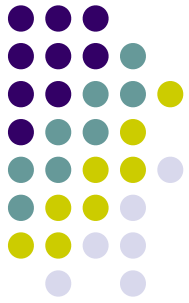
- I Addition of amino acids to the carboxyl end of the growing chain.
- I Ribosome moves from the 5'-end to the 3'-end of mRNA
- I New aminoacyl-tRNA for next triplet codon appears in ribosomal A site
- I Facilitated by EF-Tu, EF-Ts & EF-G & requires GTP.
- I After the peptide bond has been formed, the ribosome moves to next triplet codon toward the 3'-end of the mRNA. = Translocation
- I This causes movement of the uncharged tRNA into the ribosomal E site and movement of the peptidyl-tRNA into the P site.

Elongation

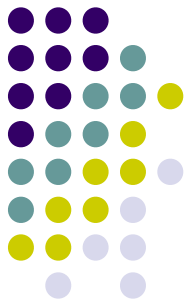


- the uncharged tRNA dissociates from the P-site
- **translocation** = the ribosome shifts one codon along mRNA, moving peptidyl tRNA from the A-site to the P-site. **requires EF2**
- **next aminoacyl tRNA** then binds within the **A-site**; tRNA binding requires EF1
- **energy for elongation is provided by the hydrolysis of two GTPs:**
 - one for translocation
 - one for aminoacyl tRNA binding



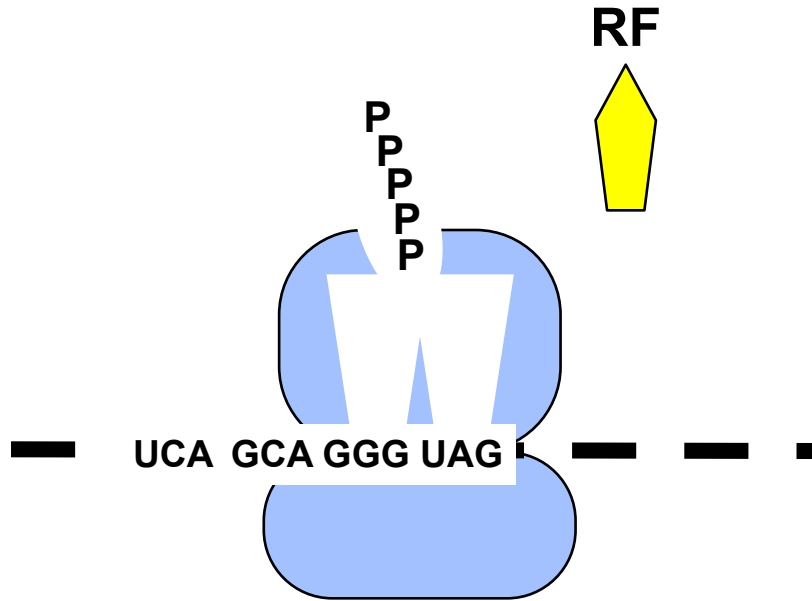


Termination

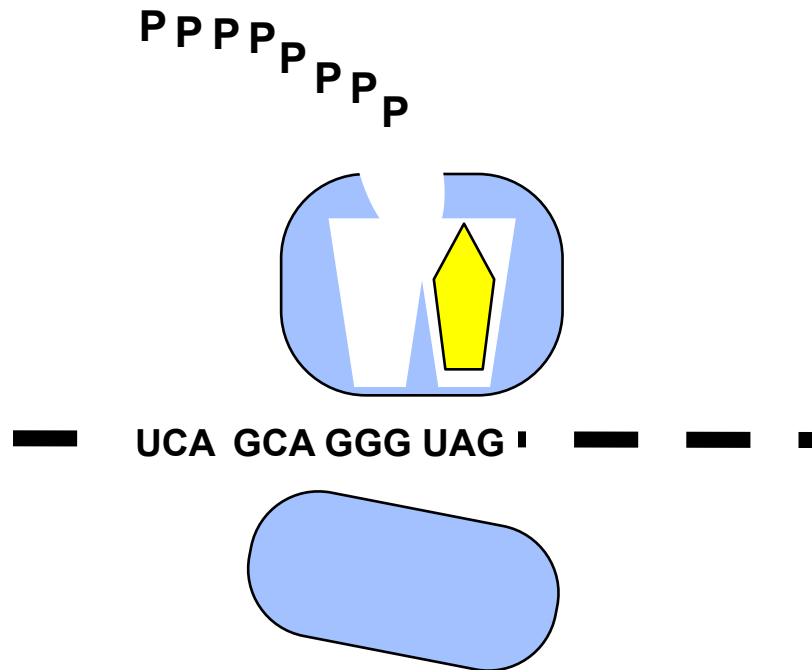


- | Termination occurs when one of the three termination codons moves into the A site.
- | Termination codons are **recognized** in by
 - | **RF-1 = UAA and UAG**
 - | **RF-2 = UAA and UGA**
- | RF induces peptidyltransferase to **hydrolyze the bond** linking the **peptide to the tRNA** at the A site, causing the **nascent protein to be released** from the ribosome.
- | **RF-3** (bound to GTP) cause **release of RF-1 or RF-2** as GTP is hydrolyzed.

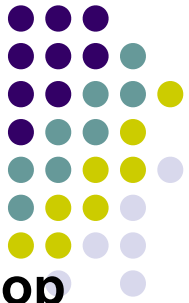
Termination



- when translation reaches the stop codon, **RF binds within the A-site**, recognizing the stop codon

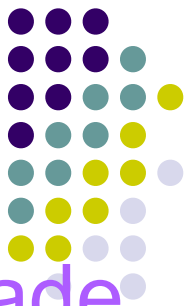


- RF catalyzes the **hydrolysis** of the completed polypeptide from the peptidyl tRNA, and the **entire complex dissociates**



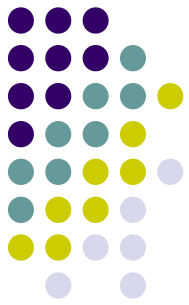
Post-translation modification

Trimming



- I Many proteins are initially made as **initially made as large**, precursor molecules that are **not functionally active**.
- I Precursor proteins are **cleaved in the endoplasmic reticulum or the Golgi apparatus**, others are cleaved in developing secretory vesicles.
- I **Zymogens** are inactive secreted enzymes (including the proteases required for digestion).
- I They **become activated through cleavage** when they reach their proper sites of action.
- I E.g. Pancreatic zymogen, trypsinogen, becomes activated to in the small intestine.

Post-translation modification



Covalent Alterations

1. Phosphorylation :

- | On hydroxyl groups of serine, threonine, tyrosine.
- | increase or decrease the functional activity of the protein.

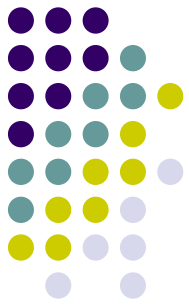
2. Glycosylation :

- | Proteins of a plasma membrane.
- | Carbohydrate attached to serine or threonine hydroxyl groups (O-linked) or the amide nitrogen of asparagine (N-linked).

3. Hydroxylation :

- | Proline and lysine residues = collagen

Post-translation modification

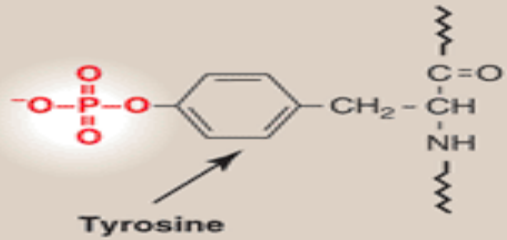
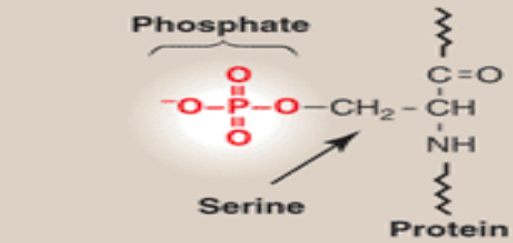


Covalent Alterations

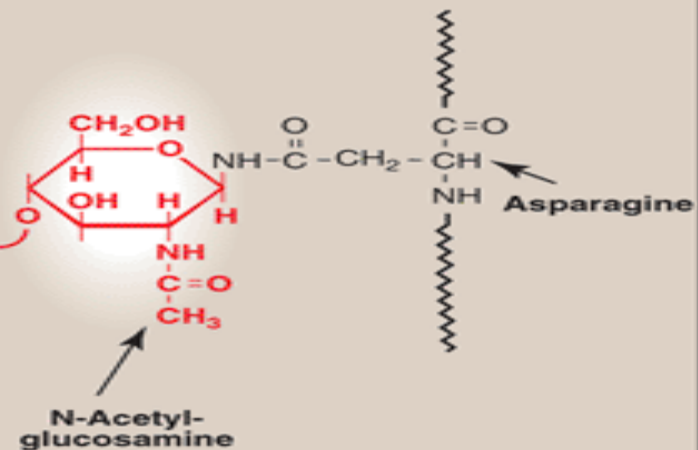
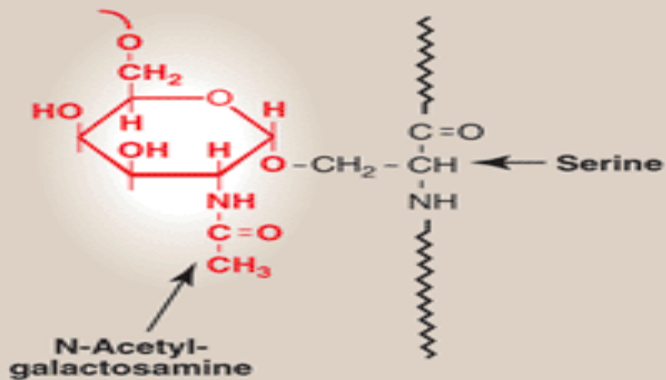
1. Other covalent modification :

- I **Vitamin K–dependent carboxylation** of Glutamate residues of **clotting factors**.
- I **Biotin** = ϵ -amino groups of lysine residues of biotin-dependent enzymes
= carboxylation reactions.
e.g. **pyruvate carboxylation**
- I Attachment of lipids, such as farnesyl groups, can help anchor proteins in membranes.
- I Acetylated

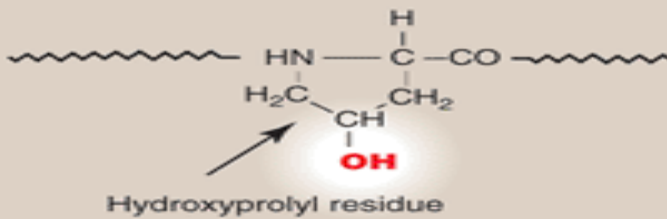
Phosphorylation



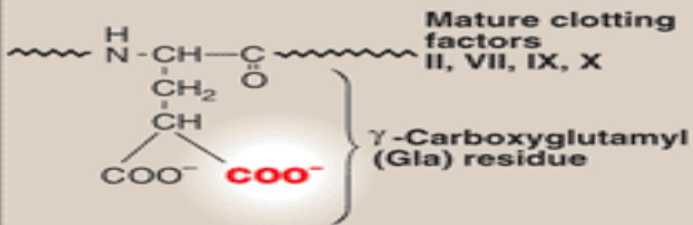
Glycosylation



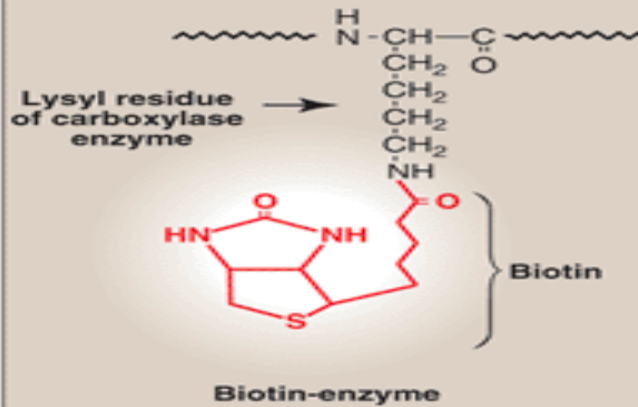
Hydroxylation



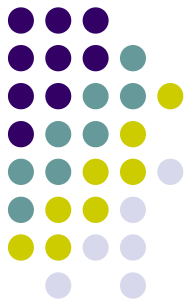
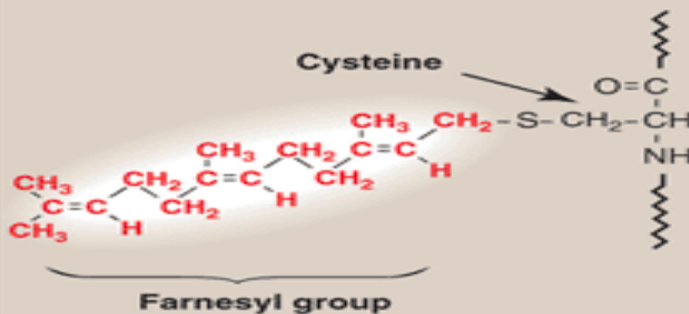
Carboxylation



Biotinylated enzyme



Farnesylated protein



Post-translation modification



Protein degradation

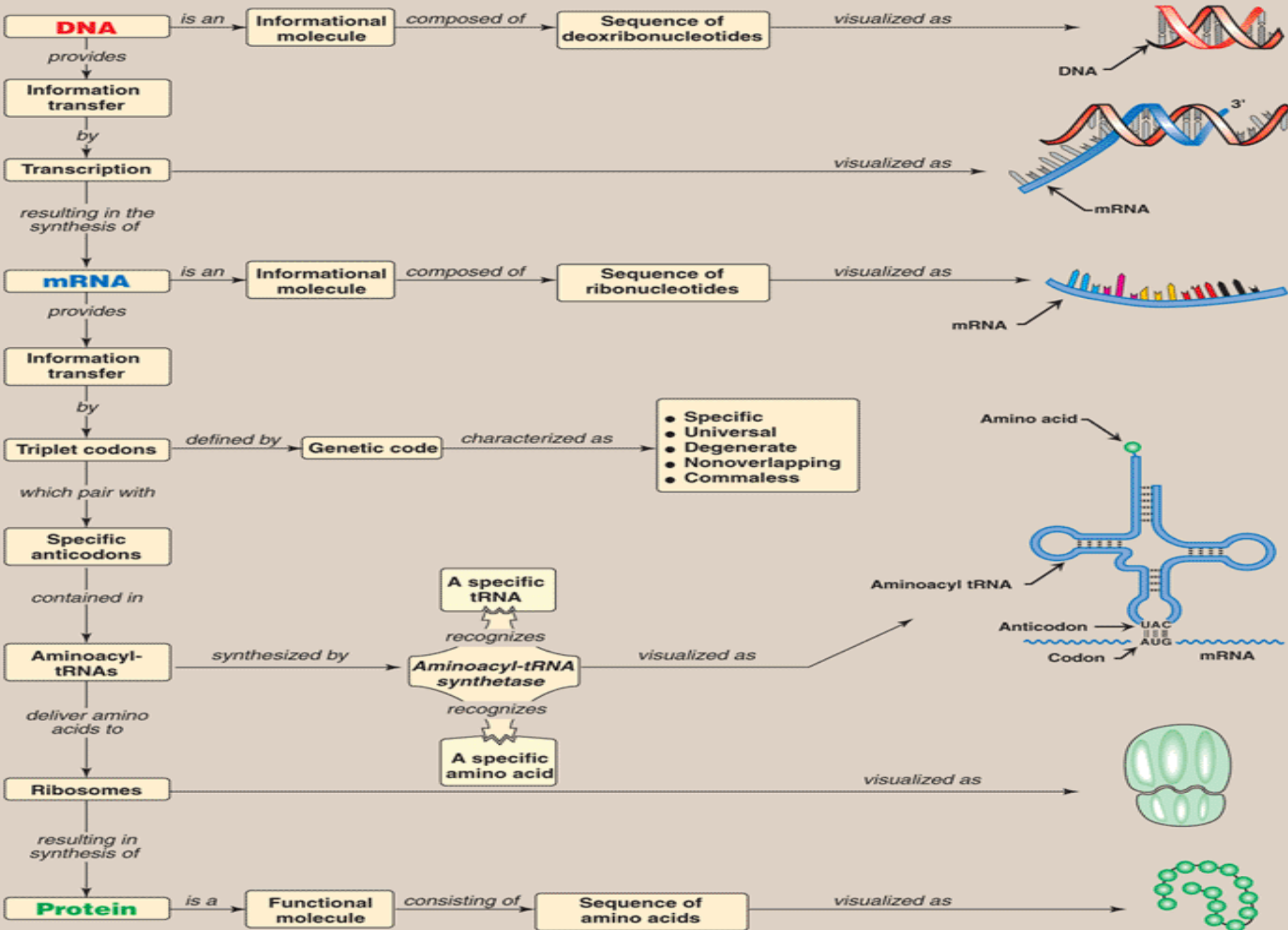
- I **Defective Protein**, for rapid turnover are often marked for destruction by **ubiquitination**
- I The attachment of a small, highly conserved protein, called **ubiquitin**.
- I Proteins **marked** in this way are rapidly degraded by a cellular component known as the “**proteasome**”.

Inhibitor of Translation



<u>Inhibitor (Antibiotic)</u>	
<i>Erythromycin</i>	<i>50 s ribosomal subunit</i>
<i>Clindamycin</i>	<i>50 s ribosomal subunit</i>
<i>Tetracycline</i>	<i>30 s ribosomal subunit</i>
Puramycin	Amino-acyl t-RNA
Chloramphenicol	Peptidyl Transferase
<i>Diphtheria Toxin</i>	<i>eEF-2</i>

Flow of genetic information



Thanks

