

**ORAL MEDICINE AND**  
**RADIOLOGY- CRRI WORK**  
**DONE**  
**20.04.2020-25.04.2020**

**DEPARTMENT OF ORAL MEDICINE AND RADIOLOGY**  
**E-CLASSES FOR CRI BDS**

**20.4.2020:**

**SUPERVISION AND DISCUSSION BY**  
**Dr.SIVAN SATHISH, MDS, MFDS RCPS**  
**PROFESSOR AND HOD, ORAL MEDICINE**



<b><u>Work done</u></b>	<b><u>Participants</u></b>	<b><u>Timing</u></b>
Discussion on Soft skills management	CRRI	10.30-11.00 am
Clinical case presentation	CRRI, Final year	11.00-11.30 am
Discussion with PGs on <i>Neurological diseases</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRI, Final year	02 pm -03 pm
NEET Questions Discussion	CRRI, Final Year	03 pm- 04 pm

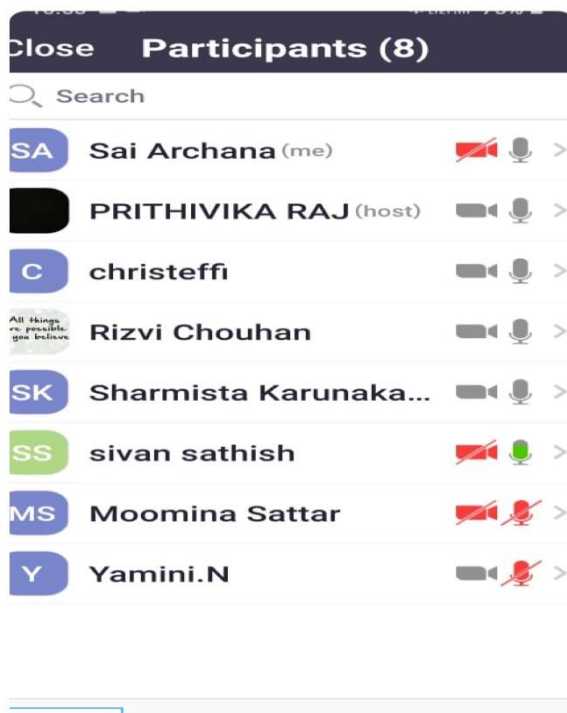
- **NAME OF STAFFS PARTICIPATED: 4**

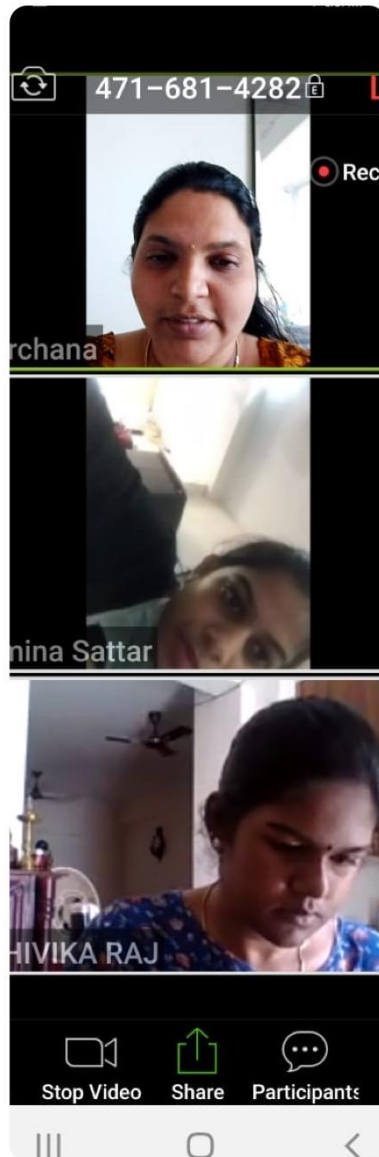
1. Dr. SIVAN SATHISH
2. Dr. CHRISTEFFI MABEL
3. Dr. SAI ARCHANA
4. Dr. MOOMINA

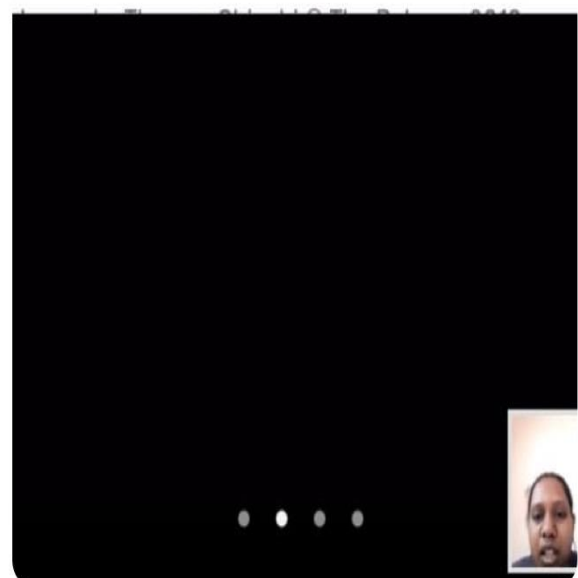
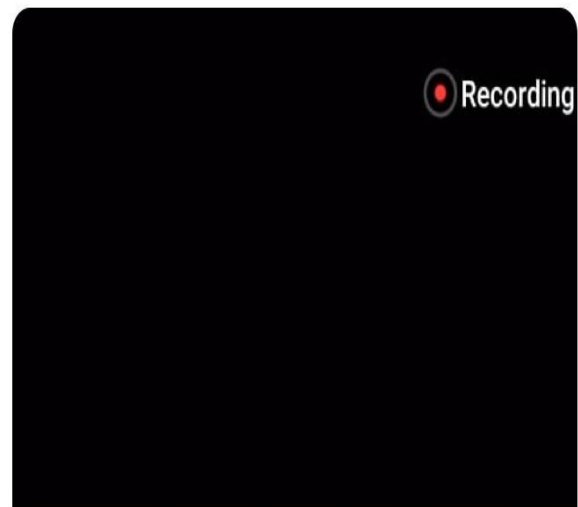
- **STUDENTS PARTICIPATED:**

1. Raj Prithvika
2. Rizvi Chauhan
3. Sumithra
4. Sharmista
5. Soundhar Rajan
6. Yamini

**PICTURES:**

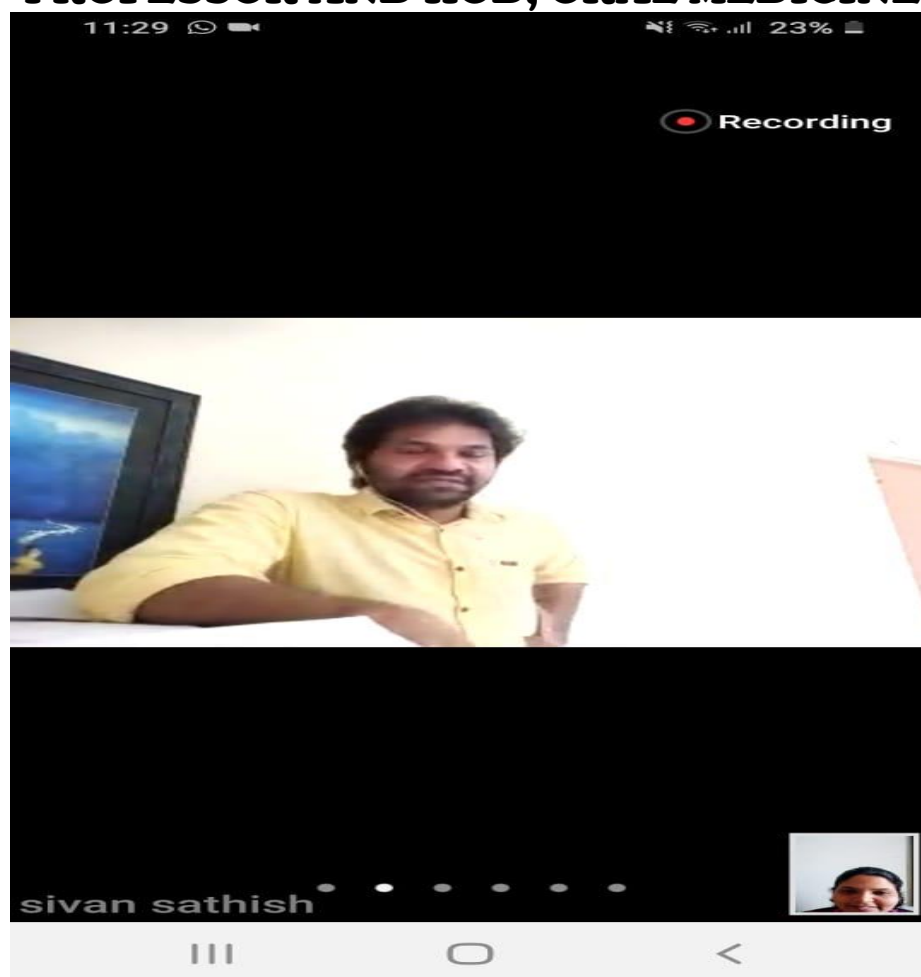






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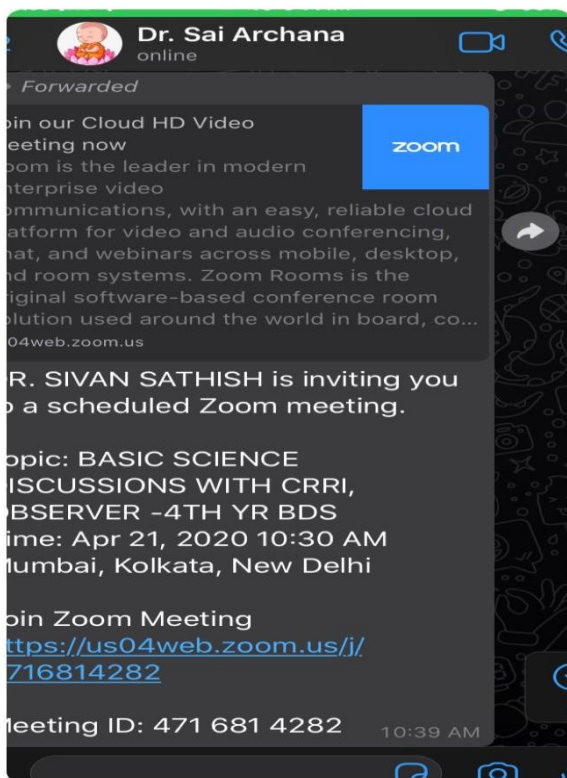
<b><u>Work done</u></b>	<b><u>Participants</u></b>	<b><u>Timing</u></b>
Discussion on NEET Questions	CRRi	10.30-11.00 am
Clinical case presentation	CRRi, Final year	11.00-11.30 am
Discussion with PGs on <i>VITAMIN B12</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRi, Final year	02 pm -03 pm
Theory class on Ideal long case	III Year, CRRi, Final Year	03 pm- 04 pm

- **NAME OF STAFFS PARTICIPATED: 4**

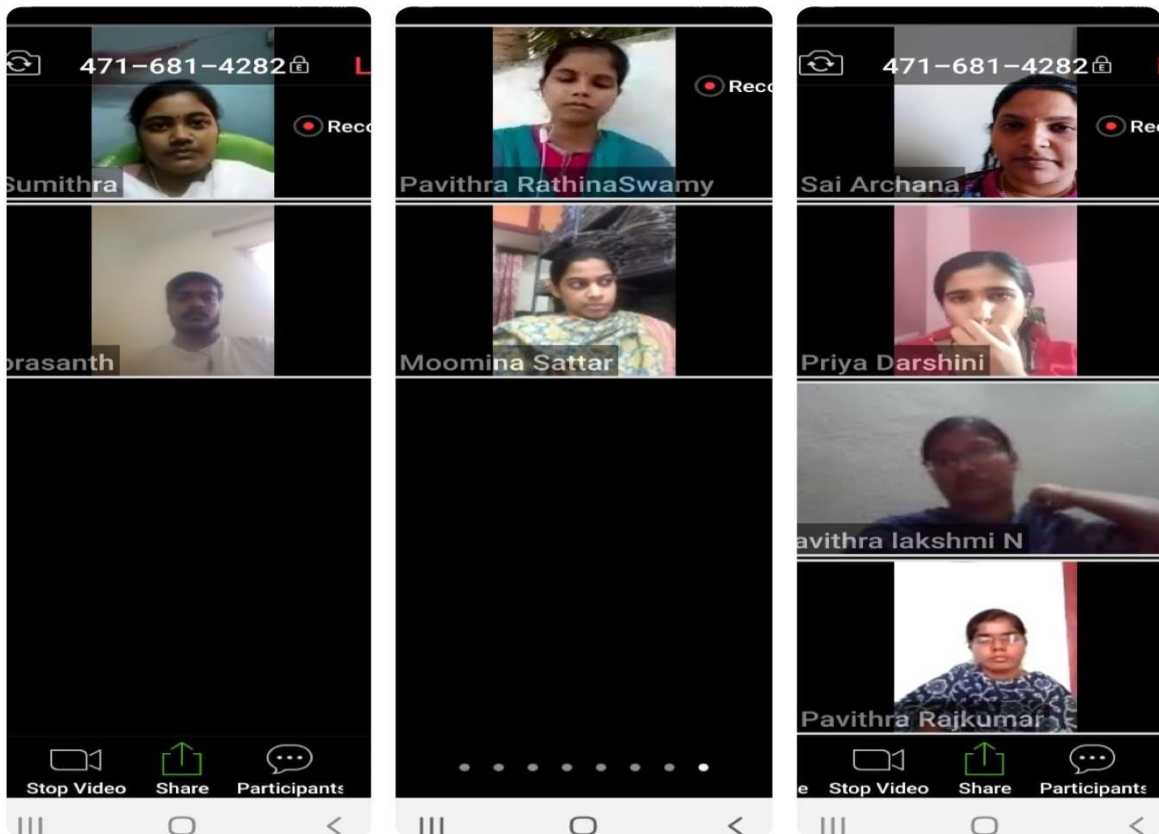
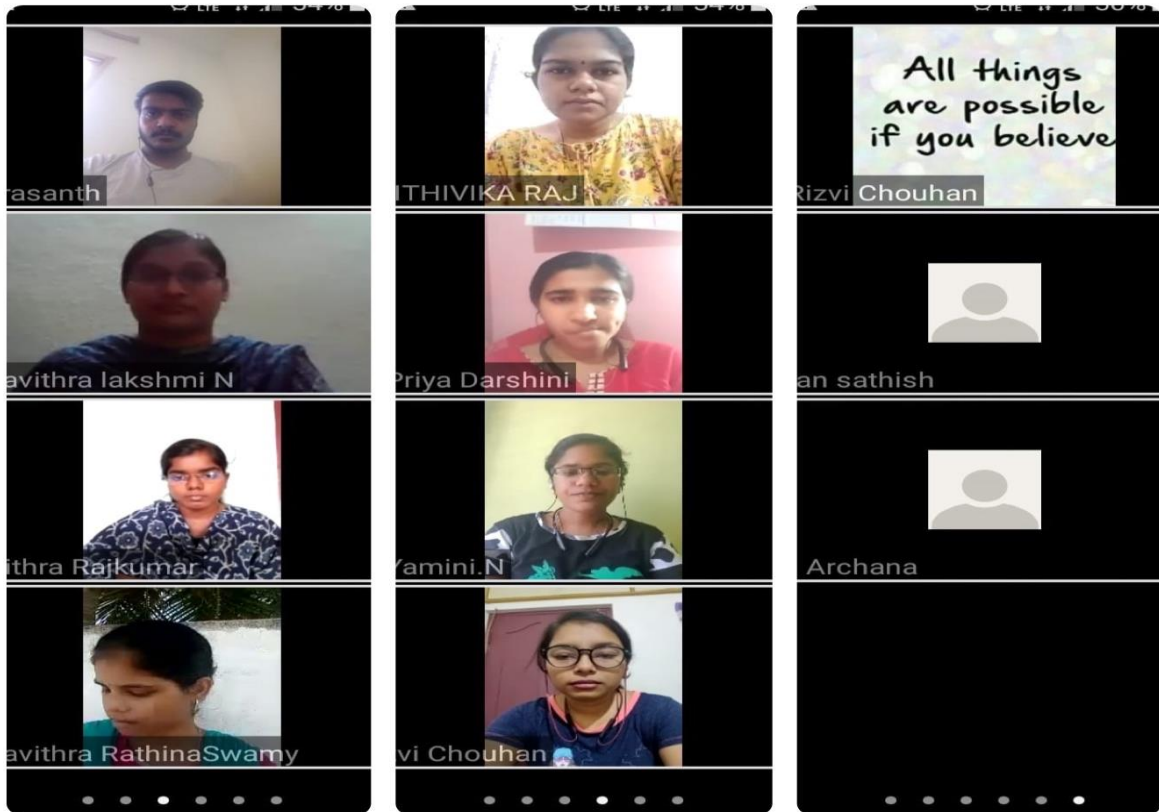
5. Dr. SIVAN SATHISH
6. Dr. CHRISTEFFI MABEL
7. Dr. SAI ARCHANA
8. Dr. MOOMINA

- **STUDENTS PARTICIPATED:**

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**22.4.2020:**

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<b><u>Work done</u></b>	<b><u>Participants</u></b>	<b><u>Timing</u></b>
Discussion on NEET Questions	CRRI	10.30-11.00 am
Clinical case presentation	CRRI, Final year	11.00-11.30 am
Discussion with PGs on <i>HYPERPARATHYROIDISM</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRI, Final year	02 pm -03 pm
NEET Questions discussion	CRRI, Final Year	03 pm- 04 pm

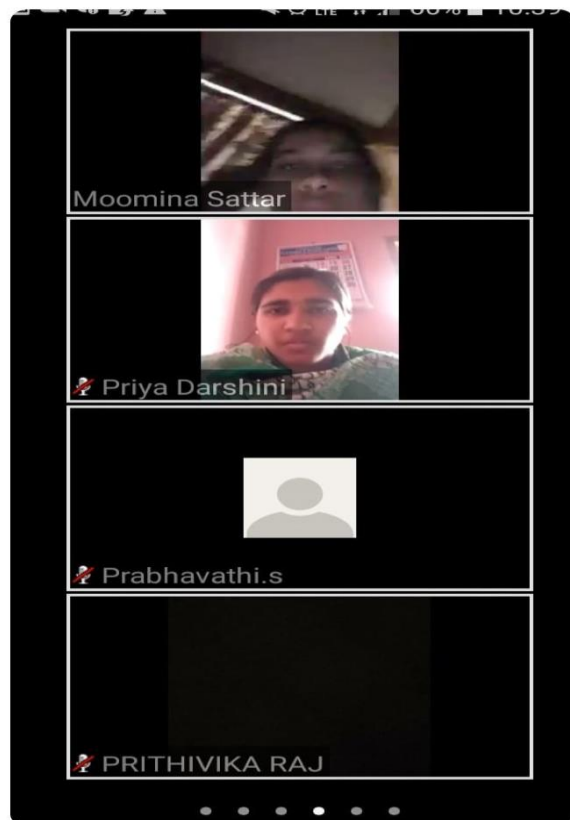
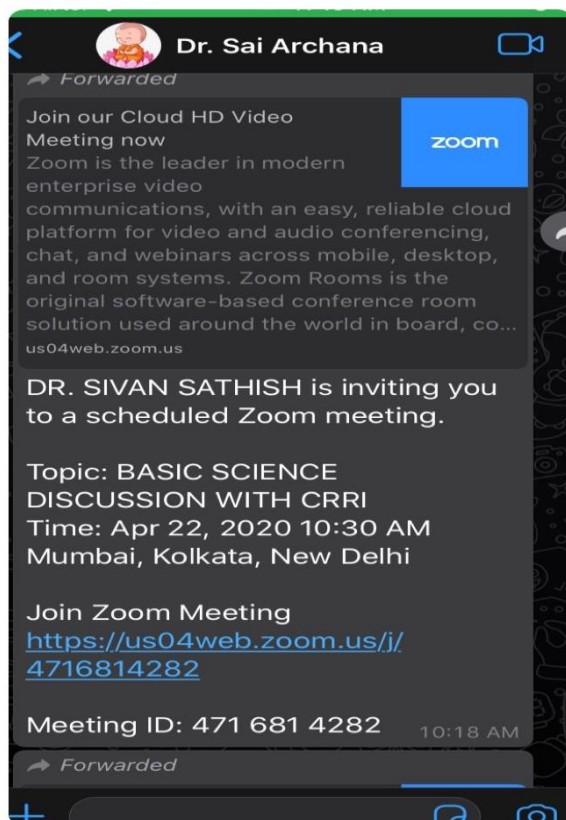
- **NAME OF STAFFS PARTICIPATED: 4**

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3. Dr. SAI ARCHANA
4. Dr. MOOMINA

- **STUDENTS PARTICIPATED:**

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2. Rizvi Chauhan
3. Sumithra
4. Sharmista
5. Soundhar Rajan
6. Yamini

**PICTURES:**



**Participants (16)**

Search

- prasanth (me)
- Pavithra lakshmi N (host)
- Sai Archana
- Pavithra Rajkumar
- Pavithra Rathinaswamy
- Prabhavathi.s
- PRITHIVIKA RAJ
- Priya Darshini
- Sharmista Karunakarar

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**Participants (16)**

Search

- P prasanth (me)
- PL Pavithra lakshmi N (host)
- SA Sai Archana
- PR Pavithra Rajkumar
- Pavithra Rathinaswamy
- P Prabhavathi.s
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- Priya Darshini
- SK Sharmista Karunakarar

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- Moomina Sattar (me)
- Pavithra lakshmi N (Host)
- sivan sathish
- Pavithra Rajkumar
- Pavithra Rathinaswamy
- Prabhavathi.s
- prasanth
- PRITHIVIKA RAJ
- Priya Darshini

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**Participants (10)**

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- SA Sai Archana (me)
- PRITHIVIKA... (host)
- Rizvi Chouhan
- sivan sathish
- Vignesh ravi
- Moomina Sattar
- Sharmista Karunaka.
- SOUNDHAR RAJAN
- Sumithra
- Yamini.N

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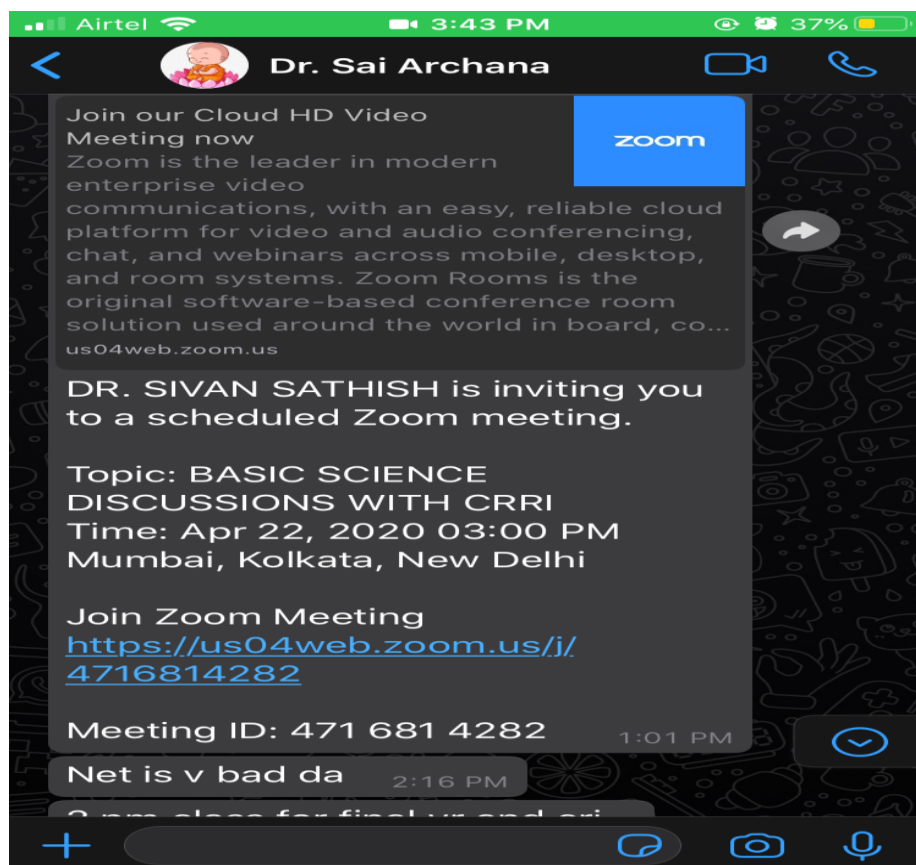
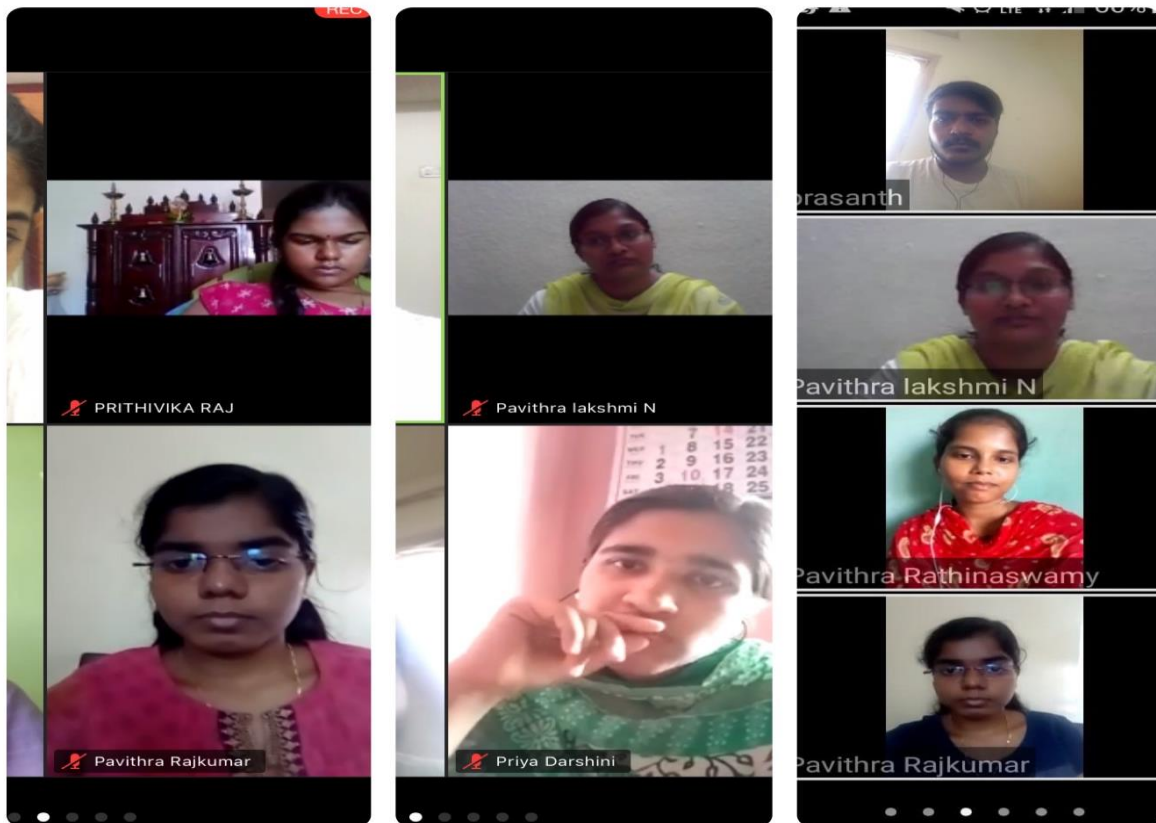
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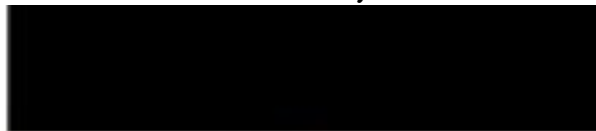
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- Vignesh ravi
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- Sharmista Karunaka.
- SOUNDHAR RAJAN
- Sumithra
- Yamini.N

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**23.4.2020:**

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<b><u>Work done</u></b>	<b><u>Participants</u></b>	<b><u>Timing</u></b>
Discussion on NEET Questions	CRRi	10.30-11.00 am
Clinical case presentation	CRRi, Final year	11.00-11.30 am
Discussion with PGs on Insulin and anti-diabetic drugs	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRi, Final year	02 pm -03 pm
NEET Questions discussion	CRRi, Final Year	03 pm- 04 pm

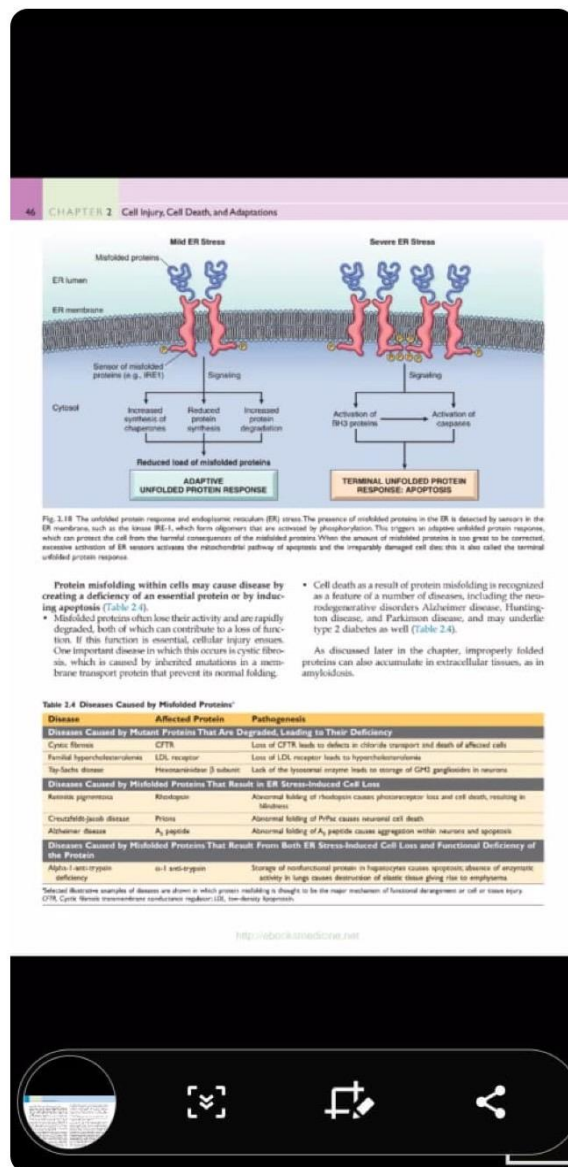
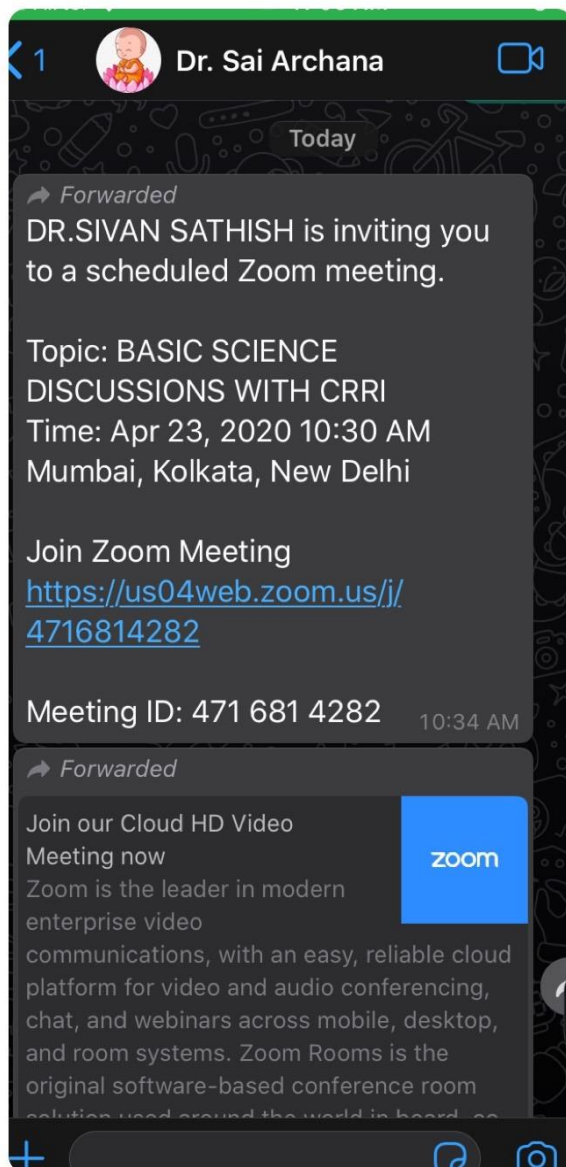


• **NAME OF STAFFS PARTICIPATED: 4**

1. Dr. SIVAN SATHISH
2. Dr. CHRISTEFFI MABEL
3. Dr. SAI ARCHANA
4. Dr. MOOMINA

• **STUDENTS PARTICIPATED:**

1. Raj Prithvika
2. Rizvi Chauhan
3. Sumithra
4. Sharmista
5. Soundhar Rajan
6. Yamini






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Search

A Sai Archana (me)

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Rizvi Chouhan

S sivan sathish

R SOUNDHAR RAJAN

S Moomina Sattar

R Pavithra Rajkumar


Pavithra RathinaSw...

P Prabhavathi.s

P prasanth

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III

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se Participants (16)

Pavithra Rajkumar

Pavithra RathinaSw...

Prabhavathi.s

prasanth

PRITHIVIKA RAJ

Priya Darshini

Sharmista Karunaka...

Sumithra

Vignesh ravi


Yamini.N


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Prabhavathi.s


Sumithra


Vignesh ravi


PRITHIVIKA RAJ

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Pavithra lakshmi N

Priya Darshini

Prasanth

Pavithra Rajkumar

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Pavithra RathinaSwamy

Sharmista Karunakaran

Yamini.N

SOUNDHAR RAJAN

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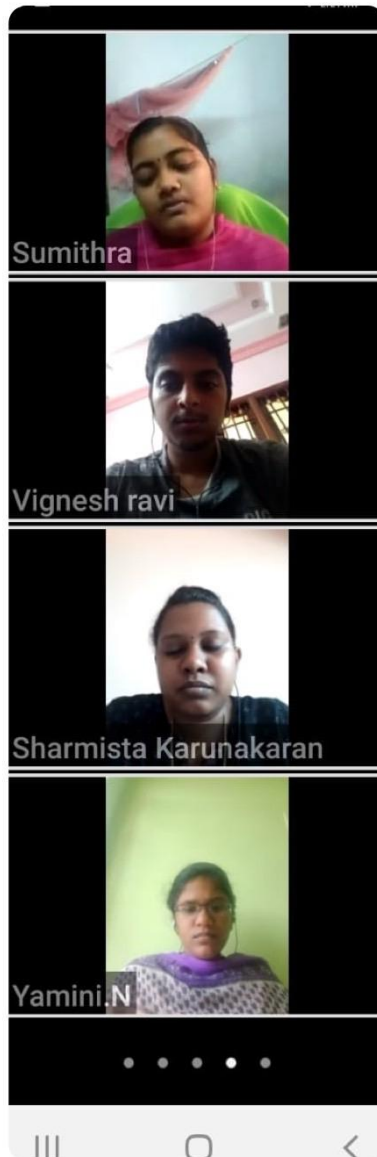
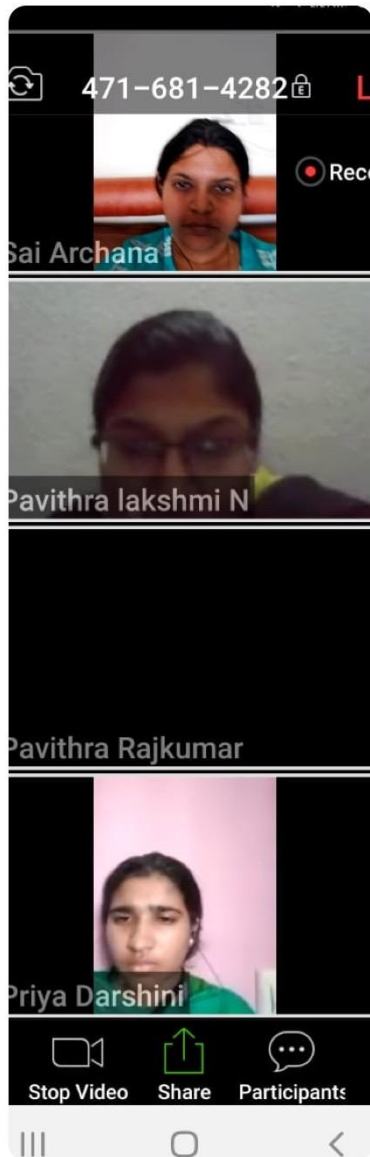
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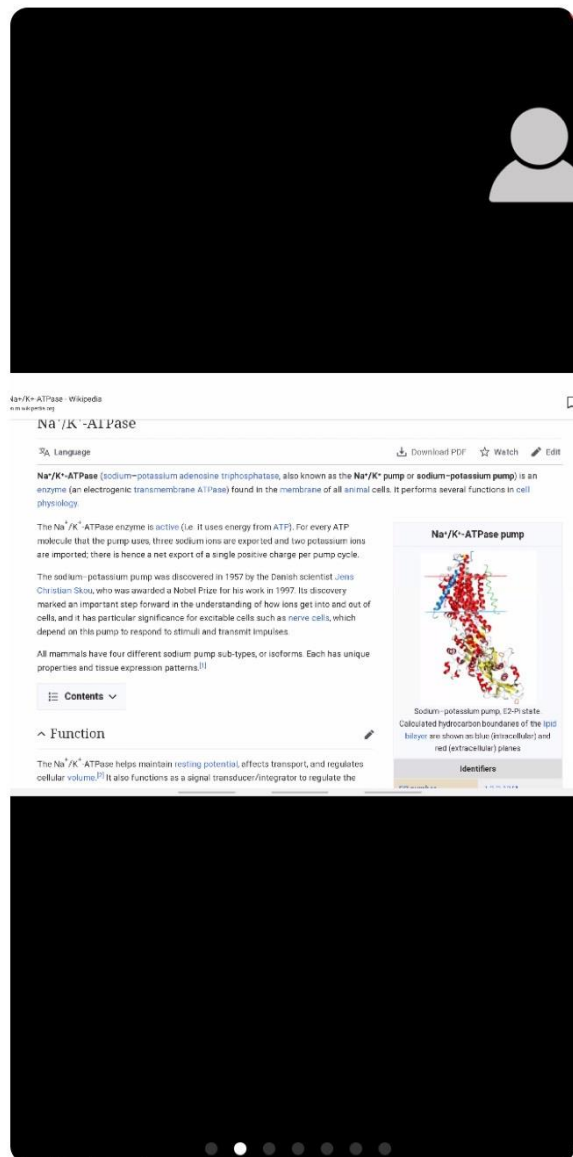
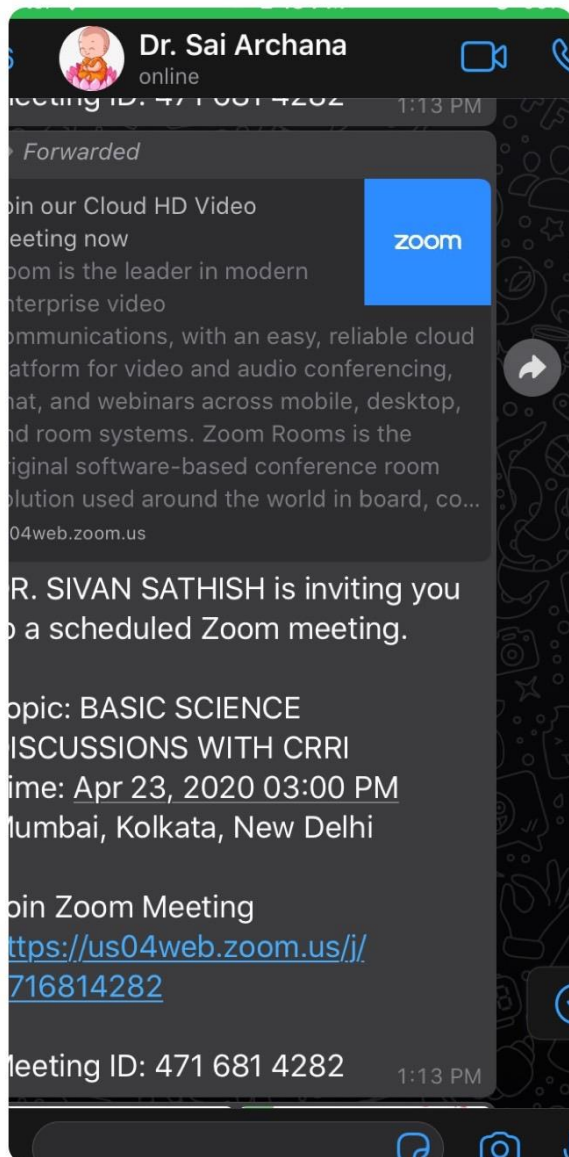
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**24.4.2020:**

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<b><u>Work done</u></b>	<b><u>Participants</u></b>	<b><u>Timing</u></b>
Combined theory class on Computed tomography	Final years, CRRi	8.30-09.30 am
NEET Questions Discussion	CRRi	10.30-11.00 am
Clinical case presentation	CRRi, Final year	11.00-11.30 am
Discussion with PGs on Amelogenesis	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRi, Final year	02 pm -03 pm
NEET Questions discussion	CRRi, Final Year	03 pm- 04 pm



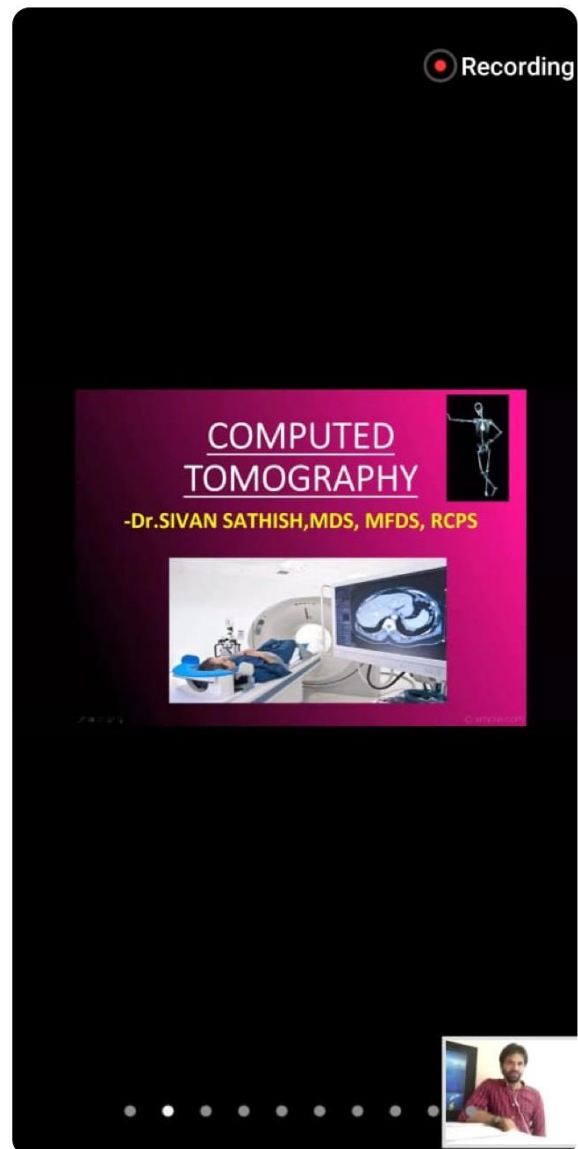
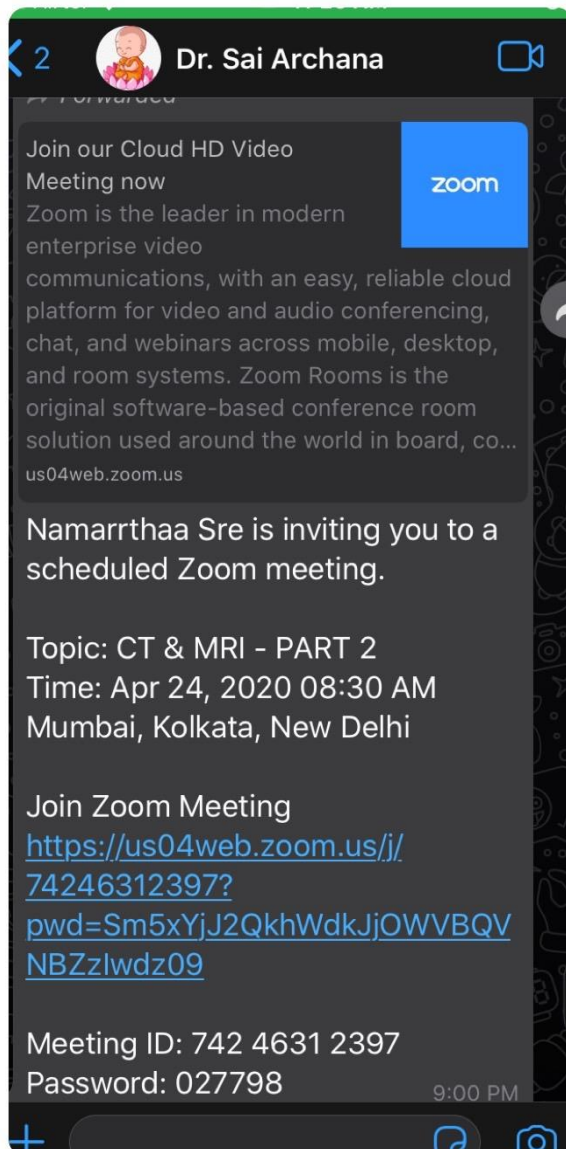
- **NAME OF STAFFS PARTICIPATED: 4**

1. Dr. SIVAN SATHISH
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4. Dr. MOOMINA

- **STUDENTS PARTICIPATED:**

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3. Sumithra
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**PICTURES:**





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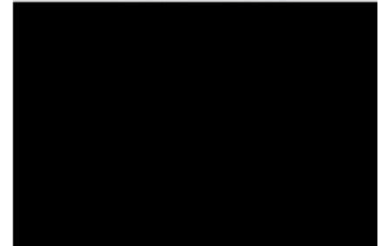
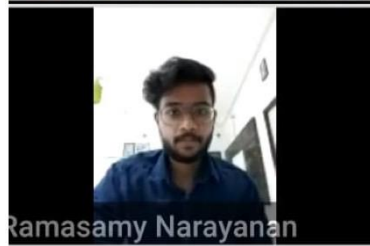
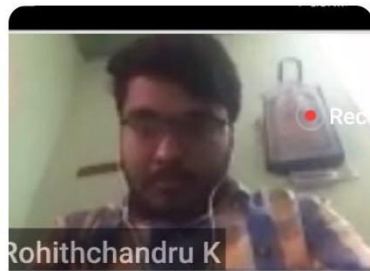
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- PL Pavithra laksh... (host)
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- P prasanth
- PRITHIVIKA RA I

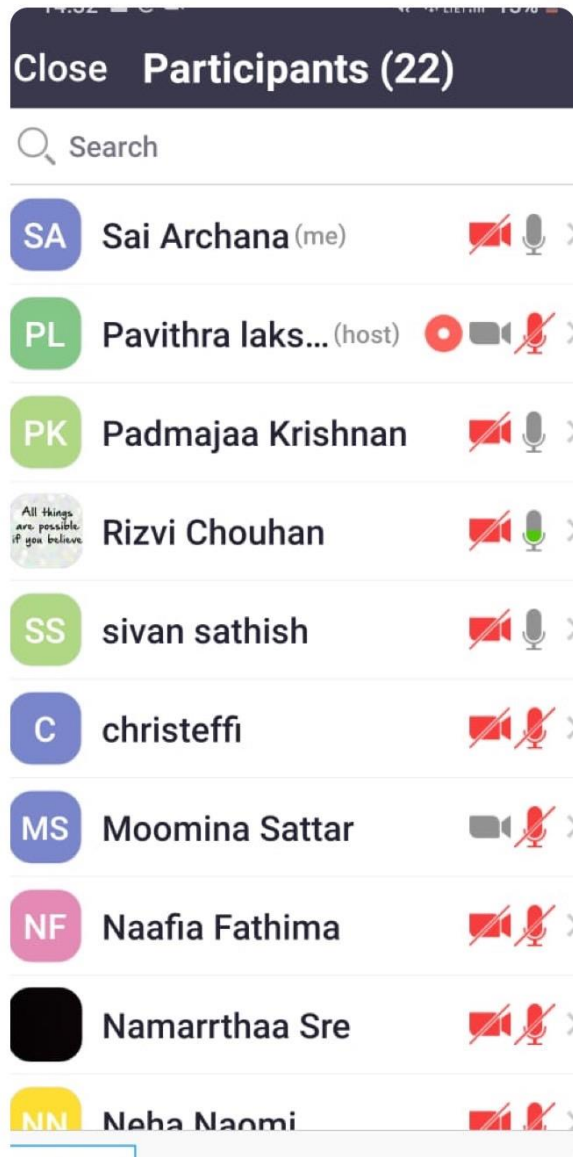
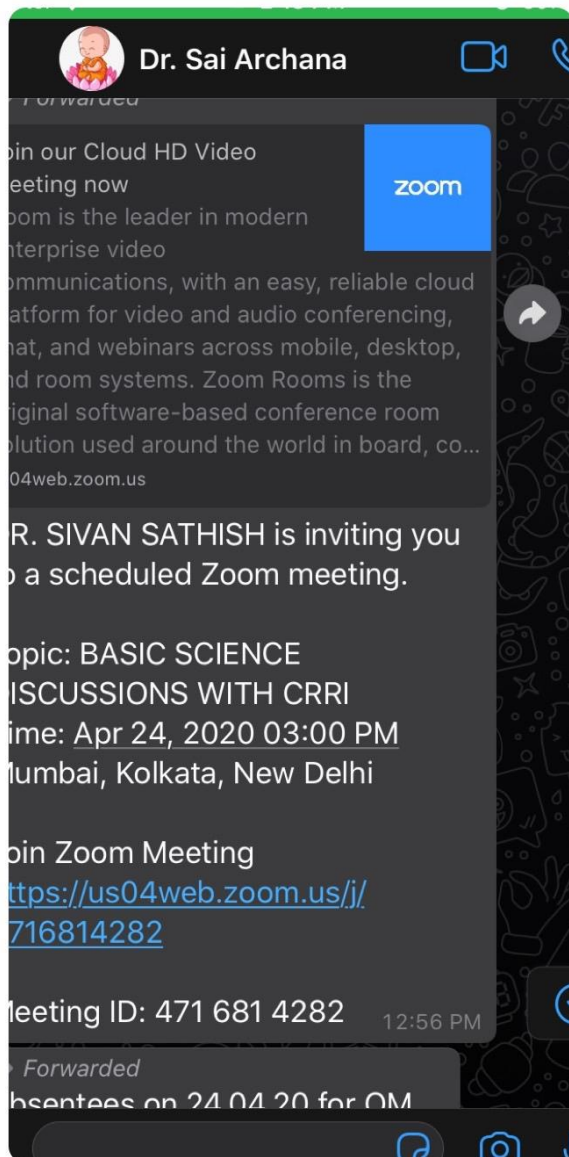
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- PR Pavithra Rajkumar
- Pavithra RathinaSw...
- P Prabhavathi.s
- P prasanth
- PRITHIVIKA RAJ
- Priya Darshini
- Rizvi Chouhan
- SK Sharmista Karunaka...
- SR SOUNDHAR RAJAN

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## Sharmista Karunakaran left

**Aging.** Cellular senescence results in a diminished ability of cells to respond to stress and, eventually, the death of cells and of the organism. The mechanisms underlying cellular aging are discussed at the end of this chapter.

With this introduction, we proceed to a discussion of the progression and morphologic manifestations of cell injury, and then to the biochemical mechanisms in injury caused by different noxious stimuli.

### SEQUENCE OF EVENTS IN CELL INJURY AND CELL DEATH

Although various injurious stimuli damage cells through diverse biochemical mechanisms, all tend to induce a stereotypic sequence of morphologic and structural alterations in most types of cells.

#### Reversible Cell Injury

**Reversible injury is the stage of cell injury at which the deranged function and morphology of the injured cells can return to normal if the damaging stimulus is removed (Fig. 2.3).** In reversible injury, cells and intracellular organelles typically become swollen because they take in water as a result of the failure of energy-dependent ion pumps in the plasma membrane, leading to an inability to maintain ionic and fluid homeostasis. In some forms of injury, degenerated organelles and lipids may accumulate inside the injured cells.

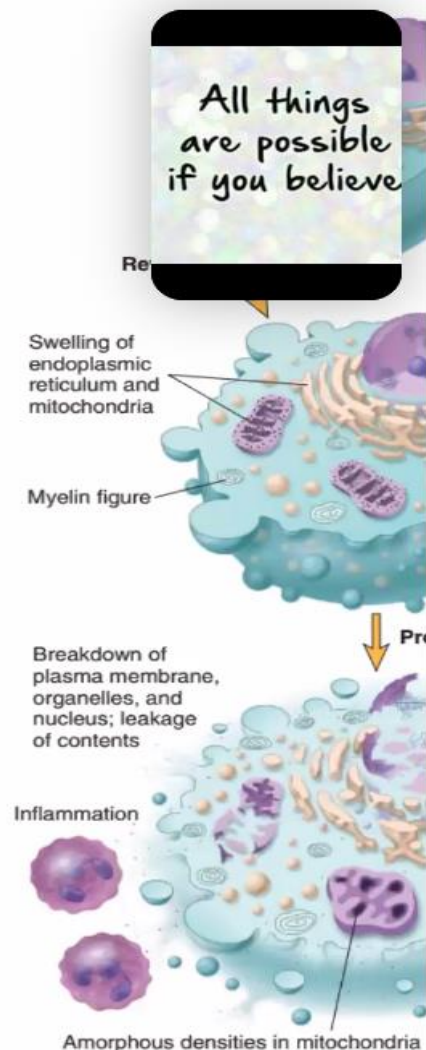
#### MORPHOLOGY

The two main morphologic correlates of reversible cell injury are cellular swelling and fatty change.

- **Cellular swelling** (Fig. 2.4B) is commonly seen in cell injury associated with increased permeability of the plasma membrane. It may be difficult to appreciate with the light microscope, but it is often apparent at the level of the whole organ. When it affects many cells in an organ, it causes pallor (as a result of compression of capillaries), increased turgor, and an increase in organ weight. Microscopic examination may show small, clear vacuoles within the cytoplasm; these represent distended and pinched-off segments of the endoplasmic reticulum (ER). This pattern of nonlethal injury is sometimes called hydropic change or vacuolar degeneration.
- **Fatty change** is manifested by the appearance of triglyceride-containing lipid vacuoles in the cytoplasm. It is principally encountered in organs that are involved in lipid metabolism, such as the liver, and hence it is discussed in Chapter 16.

The cytoplasm of injured cells also may become redder (eosinophilic), a change that becomes much more pronounced with progression to necrosis (described later). Other intracellular changes associated with cell injury (Fig. 2.3) include (1) plasma membrane alterations such as blebbing, blunting, or distortion of microvilli, and loosening of intercellular attachments; (2) mitochondrial changes such as swelling and the appearance of phospholipid-rich amorphous densities; (3) dilation of the ER

#### Sequence of Events in Cell



**Fig. 2.3** Reversible cell injury and necrosis. The changes that characterize reversible cell injury and necrosis. If the damaging stimulus is removed, reversible injury is considered to culminate in recovery; if the stimulus is not removed,

the cell progresses to necrosis, which is characterized by (1) detachment of ribosomes and (2) nuclear alterations, such as chromatin condensation and (3) nuclear fragmentation. The cytoplasm may contain so-called "myelin figures," which are accumulations of phospholipids resembling myelin sheaths derived from damaged cellular membranes.

In some situations, potentially reversible injury can progress to specific alterations in cellular organelles. The smooth ER is involved in the metabolism of many chemicals, and cells exposed to these chemicals may show hypertrophy of the ER as an adaptive response. Many drugs, including barbiturates, which have been commonly used as sedatives in the past, are metabolized in the liver by the cytochrome P-450 metabolic system found in the smooth ER.

**25.4.2020:**

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<b><u>Work done</u></b>	<b><u>Participants</u></b>	<b><u>Timing</u></b>
NEET Questions Discussion	CRRi	10.30-11.00 am
Clinical case presentation	CRRi, Final year	11.00-11.30 am
Discussion with PGs on <i>Oral submucous fibrosis with squamous cell carcinoma</i>	PGs, CRRIs, Final year, Third year	11.30-01.00 pm
Clinical case presentation	CRRi, Final year	02 pm -03 pm
NEET Questions discussion	CRRi, Final Year	03 pm- 04 pm

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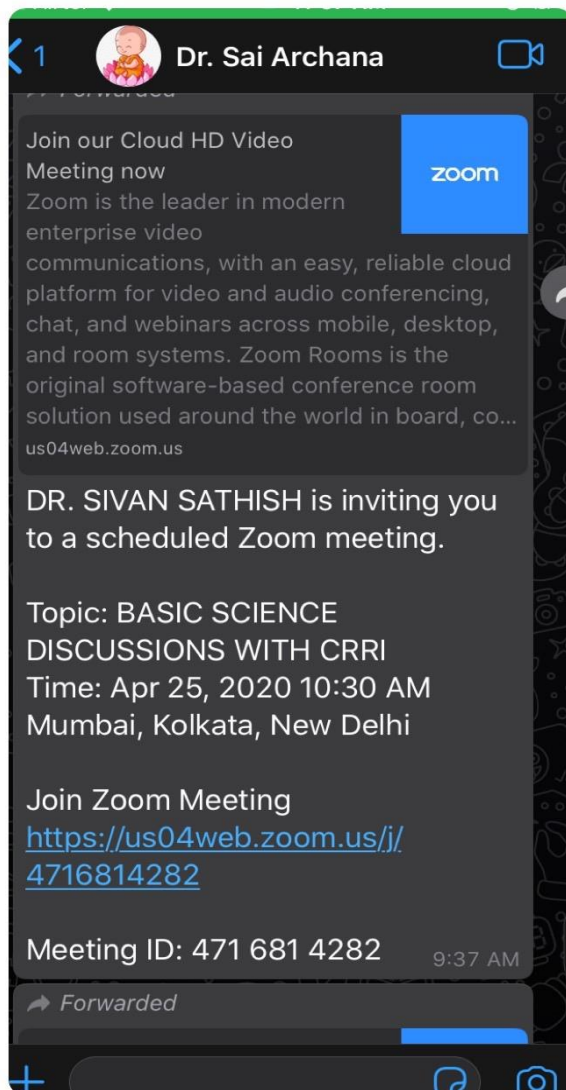
1. Dr. SIVAN SATHISH
2. Dr. SAI ARCHANA
3. Dr. MOOMINA

ABSENTEE: Dr. CHRISTEFFI MABEL

• **STUDENTS PARTICIPATED:**

1. Raj Prithvika
2. Rizvi Chauhan
3. Sumithra
4. Sharmista
5. Soundhar Rajan
6. Yamini

**Pictures:**







**Participants (10)**

- Sai Archana (me)
- PRITHIVIKA RAJ (host)
- Rizvi Chouhan
- sivan sathish
- SOUNDHAR RAJAN
- Moomina Sattar
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Extracellular Matrix

Fig. 1.17 Cell cycle checkpoints. The figure shows the cell cycle phases (G<sub>0</sub>, G<sub>1</sub>, S, G<sub>2</sub>, and M), the location of the G<sub>1</sub> restriction point, and the G<sub>1</sub>/S and G<sub>2</sub>/M cell cycle checkpoints. G<sub>1</sub> restriction point refers to the place in G<sub>1</sub> at which the cell gets committed to the cell cycle without further need of the growth factor that induced cell division. Cells from birds, mammals, and the germline of most other eukaryotic cells, such as hepatocytes, are quiescent but can enter the cell cycle; permanent cells such as neurons and cardiac myocytes have lost the capacity to proliferate. (Modified from Alberts et al., Molecular Biology of the Cell, Garland Science, 2002, Figure 15-17.)

necessary factors (see later); need of fidelity of DNA replication or factor deficiency results in arrest at the various transition points.

The cell cycle is regulated by numerous activators and inhibitors. Cell-cycle progression is driven by proteins called cyclins—named for the cyclic nature of their production and degradation—and cyclin-associated enzymes called cyclin-dependent kinases (CDKs) (Fig. 1.18). CDKs acquire the ability to phosphorylate protein substrates (i.e., kinase activity) by forming complexes with the relevant cyclins. Transiently increased synthesis of a particular cyclin leads to increased kinase activity of the appropriate CDK; binding partners as the CDK complex is degraded and the CDK activity abates. Thus, as cyclin levels rise and fall, the activity of associated CDKs likewise waxes and wanes.

More than 15 cyclins have been identified; cyclins D, E, A, and B appear sequentially during the cell cycle and bind to one or more CDKs. The cell cycle thus resembles a relay race in which each leg is regulated by a distinct set of cyclins; as one collection of cyclins leaves the track, the next set takes over.

Embedded in the cell cycle are surveillance mechanisms primed to sense DNA or chromosomal damage. These quality-control checkpoints ensure that cells with genetic imperfections do not complete replication. Thus, the G<sub>1</sub>/S checkpoint monitors the integrity of DNA before irreversibly committing cellular resources to DNA replication. Later in the cell cycle, the G<sub>2</sub>/M checkpoint ensures that there has been accurate DNA replication before the cell actually divides. When cells do detect DNA irregularities, checkpoint activation delays cell-cycle progression and triggers DNA repair mechanisms. If the genetic damage is too severe to be repaired, the cells either undergo apoptosis or enter a nonreplicative state called senescence—primarily through p53-dependent mechanisms (see later).

Enforcing the cell-cycle checkpoints is the job of CDK inhibitors (CDKIs); they accomplish this by modulating CDK-cyclin complex activity. There are several distinct CDKIs:

- One family of CDKIs—composed of three proteins called p21 (CDKN1A), p27 (CDKN1B), and p57 (CDKN1C)—broadly inhibits multiple CDKs.
- Another family of CDKIs has selective effects on cyclin CDK4 and cyclin CDK6; these proteins are called p15 (CDKN2B), p16 (CDKN2A), p18 (CDKN2C), and p19 (CDKN2D).
- Defective CDK checkpoint proteins allow cells with damaged DNA to divide, resulting in mutated daughter cells at risk for malignant transformation.

An equally important aspect of cell growth and division is the biosynthesis of other cellular components needed to make two daughter cells, such as membranes and organelles. Thus when growth factor receptor signaling stimulates cell-cycle progression, it also activates events that promote changes in cellular metabolism that support growth. Chief among these is the Warburg effect, mentioned earlier, marked by increased cellular uptake of glucose and glutamine, increased glycolysis, and (counterintuitively) decreased oxidative phosphorylation. These changes are major elements of cancer-cell growth and are discussed in greater detail in Chapter 6.

**Stem Cells**

Not all stem cells are created equal. During development, multipotent stem cells can give rise to all types of

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CHAPTER 1 The Cell as a Unit of Health and Disease

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