PROJECT REPORT

Studies on "Practice of Organic Farming" in Hampa & BuggaSangala, Guntakal mandal, Anantapur (Dist.) A.P.

Submitted by

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III B.Sc., B.Z.C.

Regd. No: 200151010.

Under the supervision of

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Lecturer in Botany

Submitted to

DEPARTMENT OF BOTANY,

SKP GOVT. DEGRE COLLEGE, GUNTAKAL,

ANANTAPURAMU – 515 803, A.P., INDIA.

SKP GOVERNMENT DEGREECOLLEGE, GUNTAKAL,
ANANTAPUR DIST.

CERTIFICATE

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Regd. No. : 20015 1010 of III B.Sc.(B.Z.C/M.B.B.C) has submitted the "Project work " entitled "Practice of Organic Farming" in Hampa & Buggasangala, Guntakal Mandal, Anantapur (Dist.) A.P.

to the Department of Botany, SKP Govt. Degree College, Guntakal as a part of Elective Paper - "Organic Farming and Sustainable Agriculture" (Paper-VII) of Botany curriculum (VI-semester), during the academic year 2021-2022. It is a record of Bonafide Project work carried-out by him/her under my supervision.

Date: Verified

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Study of "Practicing Organic Farming" in Buggasangala Village of Guntakal Mandal,Anantapramu Dist.

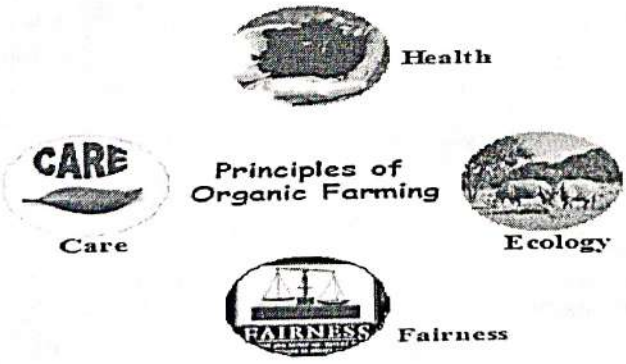
&

Hampa Village of Maddikera Mandal, Kurnool Dist.

I-INTRODUCTION:

System of farming that uses organic inputs like green manures, cow dung, etc., for cultivation is Organic farming. Organic farming, agriculture system that uses ecologically based pest Controls and biological fertilizers derived largely from animal and plant wastes and nitrogen-fixing Cover Crops Modern organic farming was developed by a response to the environmental harm caused by the use chemical pesticides and Synthetic fertilizers in conventional agriculture, and it has numerous ecological benefits.

Compared with Conventional agriculture, organic farming uses decreases fewer pesticides, reduces Soil erosion, decreases nitrate leading into ground water and surface water, and recycles animal wastes back into the farm. These benefits are counterbalanced by higher food costs for Consumers and generally lower yields. Indeed, yields of organic crops have been found to be about 25 percent lower overall than conventionally grown crops, although this can vary Considerably depending upon the type of crop. The challenge to maintain its environmental benefits increase yields, and reduce prices while meeting the challenges of climate change and an increasing world population.



Principles of organic farming includes...

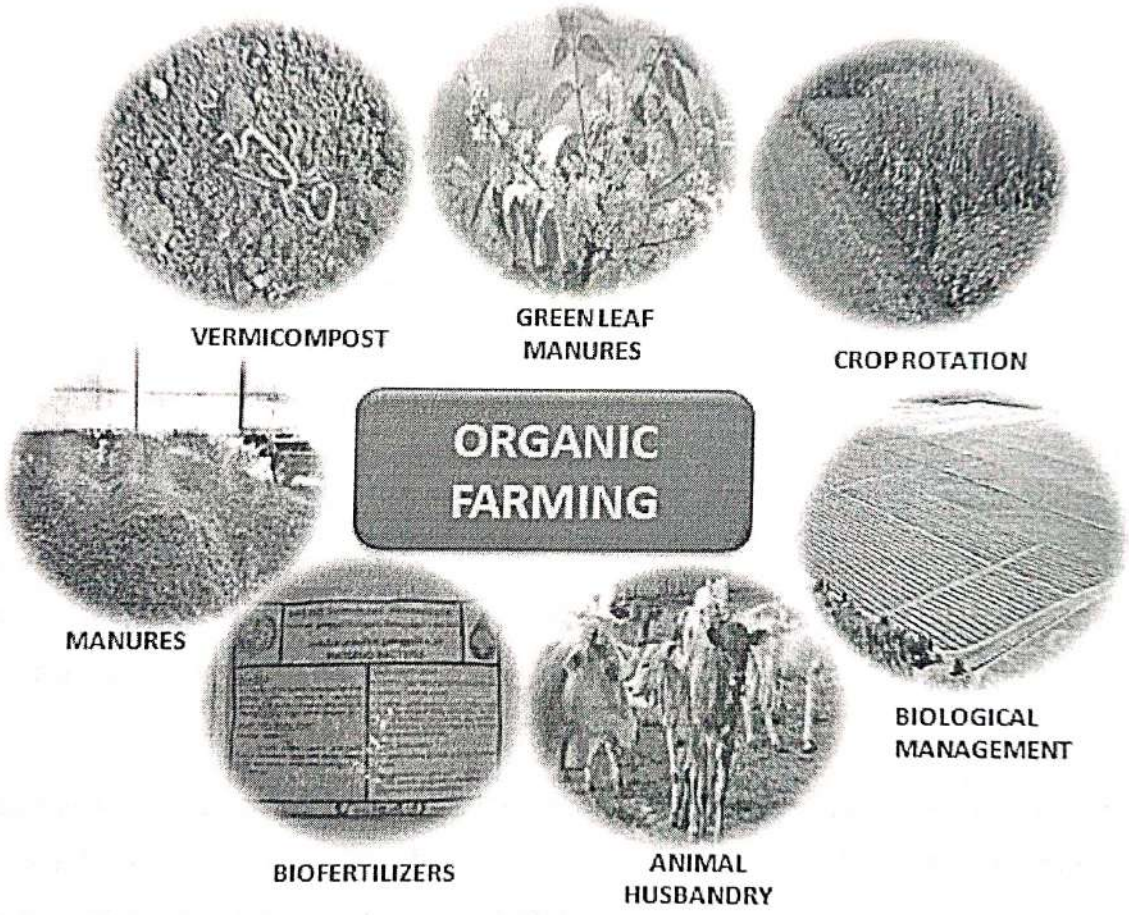
*The principle of health:- Organic farming should sustain and enhance the health of soil, plant, animal, human and planet as one and indivisible.

*The principle of ecology:- Organic farming should be based on living ecological systems and cycles, work with them, emulate them and help sustain them.

P.T.O

*The principle of fairness:-Organic farming should build on relationships that ensure fairness with regard to the common environment and life opportunities.

*The principle of care:-Organic farming should be managed in a precautionary and responsible manner to protect the health and well being of current and future generations and the environment.



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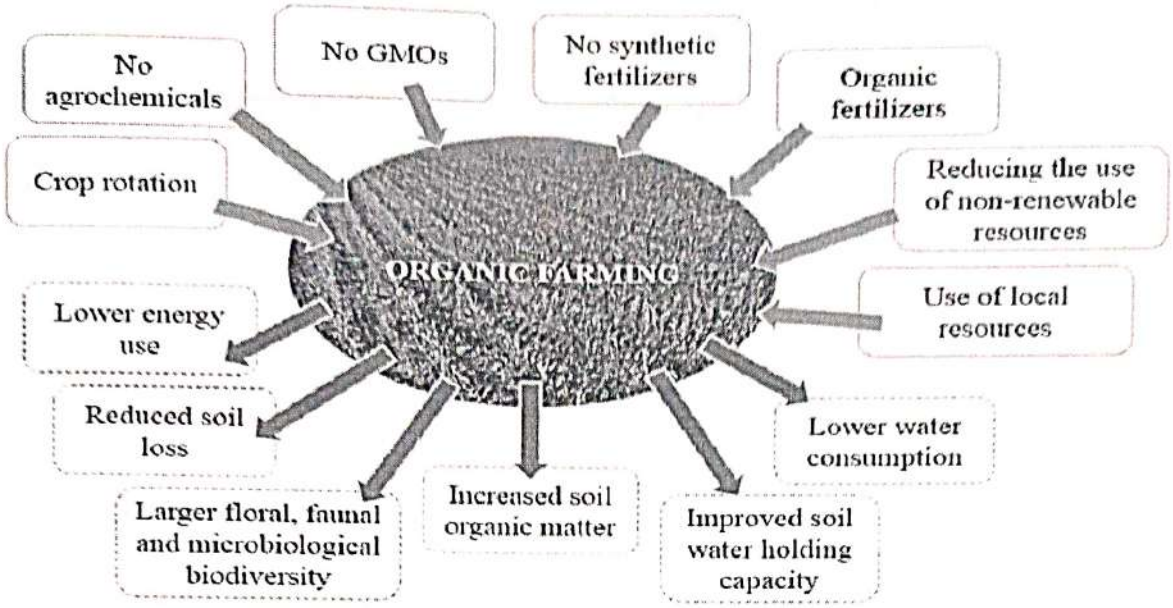


History

The concepts of organic agriculture were developed in the early 1900s by Sir Albert Howard, F.H. King, Rudolf Steiner, and others who believed that the use of animal manures (often made into compost), cover crops, crop rotation, and biologically based pest controls resulted in a better farming system. Howard, having worked in India as an agricultural researcher, gained much inspiration from the traditional and sustainable farming practices he encountered there and advocated for their adoption in the West. Such practices were further promoted by various advocates such as J.I. Rodale and his son Robert, in the

1940s and onward, who published Organic Gardening and Farming magazine and a number of texts on organic farming. The demand for organic food was stimulated in the 1960s by the publication of Silent Spring, the 1960s by the publication of Silent Spring, the 1960s by the publication of Silent Spring, the 1960s by the publication of Silent Spring, by Rachel Carson, which documented the extent of environmental damage caused by insecticides.

NEED OF ORGANIC FARMING:

- Excessive use of chemical fertilizers reduced the fertility of soil.
- Excessive use of chemicals has led to soil, water and air pollution.

P.T.O

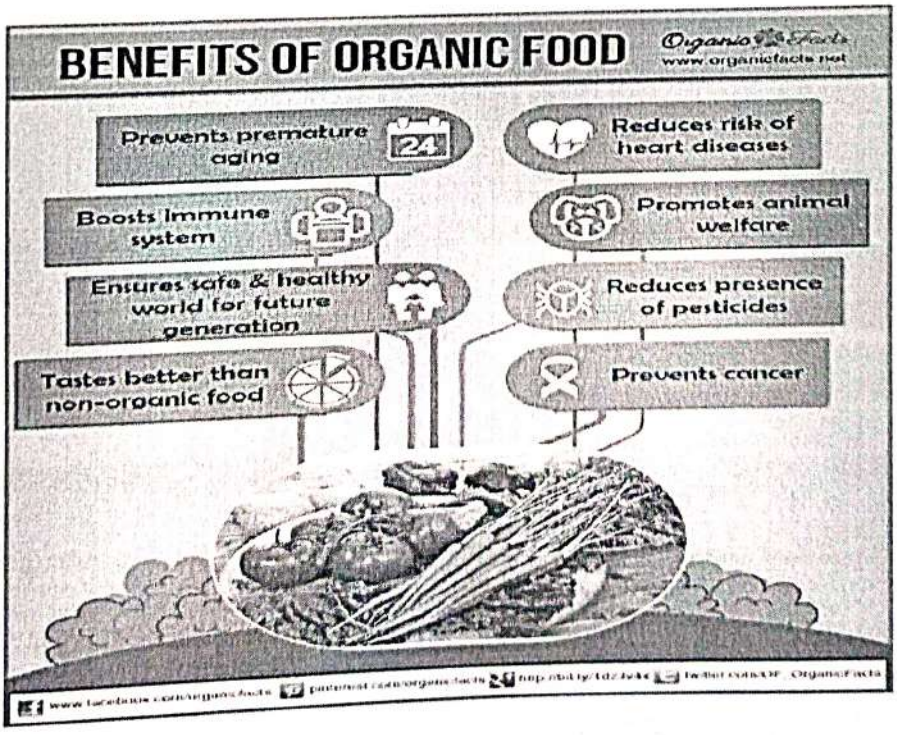
- To conserve ecosystem.
- To promote sustainable development.
- Inexpensive farming.
- Increased demand of organic products due to safety of food.

BENEFITS OF ORGANIC FARMING:

- Environment-friendly.
- Promotes sustainable development.
- Healthy and tasty food Inexpensive process.
- It uses organic inputs.
- Generates income.
- Generates income through exports .

Source of employment .

Organic farming is more labor intensive. Hence, it generates more employment.



P.T.O

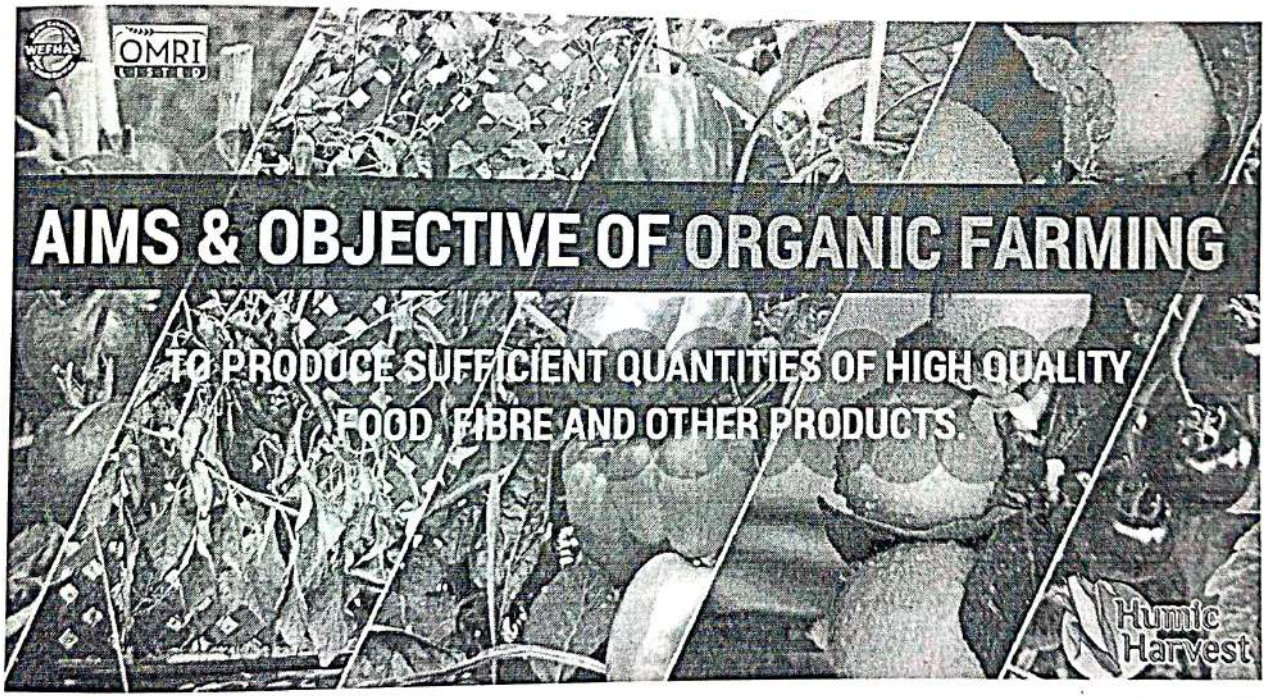
LIMITATION OF ORGANIC FARMING

- Less output. Higher price.
- The lack of awareness.
- Organic products generally demand a higher price due to a higher demand.
- Shorter shelf life. Organic products have a shorter shelf life due to the absence of artificial preservatives.

RELEVANCE OF ORGANIC FARMING IN INDIA

High nutritional value.

- Maximum profit.
- › Employment opportunity.



AIMS OF ORGANIC FARMING

1.To produce high quality of food in sufficient quality.

2.To interact in a constructive & life enhancing way with natural systems and cycles.

P.T.O

3.To encourage and entrance biological cycles within the farming system involving micro, soil, flora& fauna.

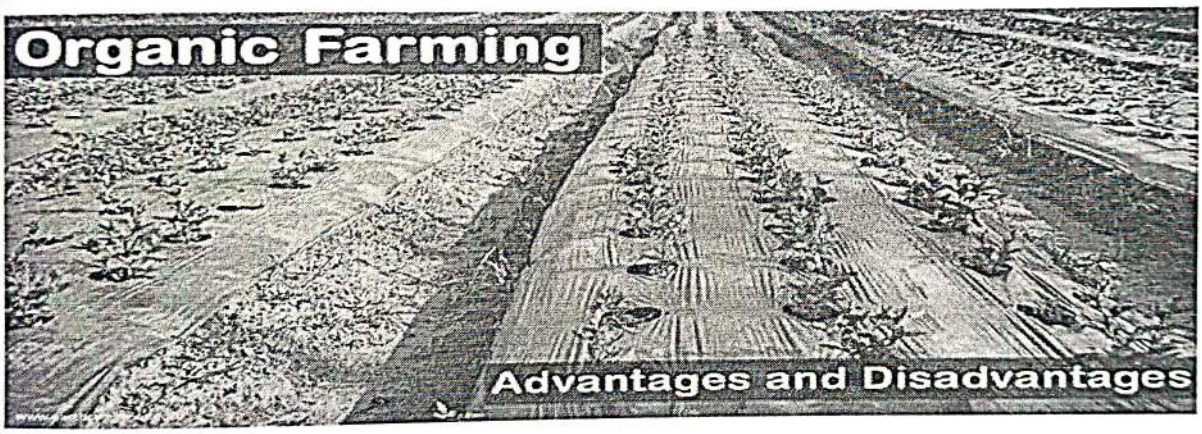
4.To maintain & increase long-term flexibility of soils.

OBJECTIVES OF ORGANIC FARMING

1.To develop a sustainable agriculture system for guaranteed adequate food productive with forceable future.

2.To develop self-sufficient agriculture system which would as much as possible upon resources from within its own resources.

3.To develop an alternative strategy over chemical farming which would be a guideline for the working of biological processes in natural ecosystem.



Advantages of Organic Farming:-

Economical: In organic farming, no expensive fertilizers , pesticides, or HYV seeds are required for the plantation of crops. Therefore, there is no extra expense.

Good return on Investment: With the usage of cheaper and local inputs, a farmer can make a good return on investment.

High demand: There is a huge demand for organic products in India and across the globe, which generates more income through export.

Nutritional: As compared to chemical and fertilizer-utilized products, organic products are more nutritional, tasty, and good for health.

Environment-friendly: The farming of organic products is free of chemicals and fertilizers , so it does not harm the environment.

Disadvantages of Organic Farming:-

Incompetent: The major issue of organic farming is the lack of inadequate infrastructure and marketing of the product.

Less production: The products obtained through organic farming are less in the initial years as compared to that in chemical products. So, farmers find it difficult to accommodate large-scale production.

Shorter shelf life: Organic products have more flaws and a shorter shelf life than that of chemical products.

Limited production: off-season crops are limited and have fewer options in organic farming.

II-MATERIALS AND METHODS:

A. MATIRIALS /EQUIPMENT USED IN ORGANIC FARMING:

*Water supply

*Tractor

*Cultivator

*.Plow

*Tiller

*Harrow

*.Broadcast or air seeder

*Seed drill, air seeder or precision gun

*Transplanter

*Harvester or combine

*Equipment for transportation or moving earth, such as a backhoe, front-end loader or motorized cart.

B. METHODS USED IN ORGANIC FARMING:

1. Soil management:

After cultivation of crops the soil looses it's nutrients quality depletes organic agriculture initiates the use of natural ways to increase the fertility of the soil. Hence it focuses on the use of bacteria that is present in animal waste which helps in making the soil nutrients more productive & fertile.

Jeevamrutham preparation

Materials required for Ghana jeevamrutham Preparation:-

* Desi cow dung : 100 kg

*Jaggery. : 1 kg

* Besan : 1 kg

* Polythene Sheet

PREPARATION:-

* Take 100 kg of DESI COW DUNG and add 1 kg JAGGERY and 1 kg BESAN.

*Furthermore , mix the mixture well and store it as a heep for 48 hrs in a shade.

*If the temperature drops below 12 degree the heap should be covered with rug sack to maintain a constant temperature for conductive climatic conditions for microorganisms.

P.T.O

Ghana Jeevamrutham Preparation by farmers in BUGGA SANGALA

* After 48 hrs spread the mixture on a clean surface and Sunday it flip it upside down during the day so that all particles get exposed to sunlight and quickly dries up.

.* Once it is completely dried up break the lumps into powder from with the wooden bat and fill them into sacks and store it in a cool and dry place.

Application of Ghana Jeevamrutham:-

* Therefore, The prepared powder form of Ghana jeevamrutham is good enough for the 1 ACRE are before planting.

Dosage of Ghana Jeevamrutham :- 5 quintal / Acre.

Materials required for Drava Jeevamrutham:-

*Cow dung - 10 kg

*Cow urine 10 lt

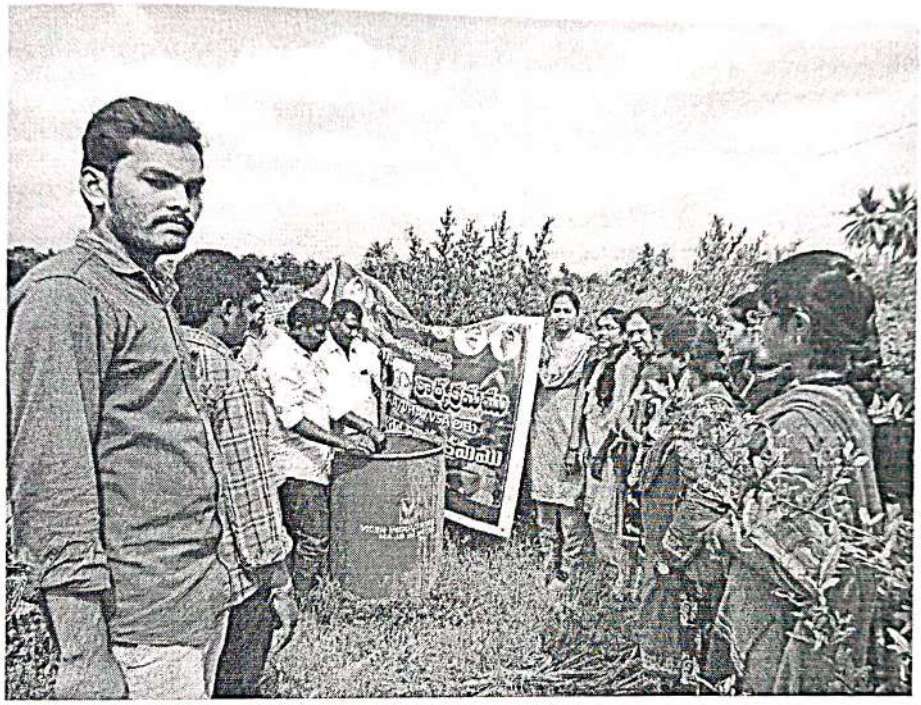
P.T.O

*Clean water - 200 lt*Jaggery-2 kg

*Pulse flour-2kg

*Barrel/ Drum - 1 no. (200 lt)

Preparation of Drava Jeevamrutham:-



Drava Jeevamrutham Preparation by farmers in BUGGA SANGALA

*Take 200 liters of water into a barrel.*Then, add 10 kg fresh local cow dung and 5-10 liters of aged cow urine.

*Furthermore, add 2 kg of Jaggery and 2 kg of pulse flour and handful of soil from the bund of field.

*Stir the solution at regular intervals well and let it ferment for 48 hours in the shade.

*Thus, Jeevamrutham is ready for application on the plants or to the field.

After the 48-hour fermentation process, the aerobic and anaerobic bacteria present in the cow dung and cow urine multiply as they eat up organic ingredients.

P.T.O

Application of Drava Jeevamrutham:-

*Apply the Jeevamrutham on the crops twice a month with irrigation water or as a 10% foliar spray.

*It provides nutrients, and importantly, acts as a catalytic agent that promotes the activity of microorganisms in the soil, as well as increases the activity of earthworms living in the soil.

*In addition, it also helps to prevent the disease-causing microbes like fungal and bacterial plant pathogens.

*However, Jeevamrutham is only needed for the first 3 years of the organic farming system, after which the soil system becomes fertile and self sustaining.

Dosage of Drava Jeevamrutham:-

200 liters of Drava Jeevamrutham is enough to apply one acre land.

2. **Weed management**

Weed is the unwanted plant that grown in agriculture fields. Organic agriculture focuses on lowering weed rather than removing it completely. The two most widely used weed management techniques.

3. **Mulching:**

A process where we use plastic filmor plant residues on the surface of the soil to block the growth of weed.

4. **Crop diversity**

Monoculture in the practice used in the agriculture field where we harvest & cultivate only one type of crop in a particular location. Recently polyculture has come of crops to meet the increasing crop demand & produce the required.

P.T.O

III-RESULTS:

Result is mentioned in the tabular form.

TABLE.1: Details of the crops and yields of various farmers in Hampa village & Buggasangala village.

	NAME OF THE FARMER	LAND AREA (ACRES)	CROP	ORGANIC FARMING YIELD (TONNES)	CONVENTIONAL FARMING YIELD (TONNES)
1	NARAYANASWAMY	1	PADDY	8	15
2	SRI RAMULU	9	CORIANDER	25	30
3	TIRUMALESH	9	TOMATO	11	25

TABLE:-2.Details of the crops:- Profit/Loss of various farmers in Hampa village & Buggasangala village

	NAME OF THE FARMER	LANDED AREA(ACRES)	CROP	PROFIT/LOSS	PROFIT/LOSS
1	NARAYANA SWAMY	1	PADDY	50,000	20,000
2	SRI RAMULU	9	CORIANDER	1,80,000	1,00000
3	TIRUMALESH	9	TOMATO	2,00,000	50,000

P.T.O

PROJECT WORK ON ORGANIC FARMING

Visited "BUGGASANGALA" Village. Farmer & Narayana Swamy.

Dhrava Jeevamrutham

Field visit in farmer, Narayana swami's field at BUGGA SANGALA

1.NARAYANA SWAMY

He has cultivated paddy crop in both conventional and organic farming methods in the consecutive years 2020-21 and 2021-22. He got more profit (during, he got 8 quintals when organic farmed and when conventional farming he got 10 quintals) when he practiced conventional farming based on the above data mentioned in the result table.

P.T.O

2.SRI RAMULU

He has cultivated coriander crop in both conventional and organic farming. He used sprinkler system for water management. He cultivated in the consecutive years 2020-21 and 2021-22 has got more profit during organic farming he got 25 quintals and during conventional farming he got 22 quintals which results less in profit. When he practiced organic farming. He got profit based on the above mentioned in the result table.

3.TIRUMALESH

He also cultivated Tomato in 9 acres. So this was cultivated in both farming in the consecutive years 2020-21 and 2021-22 in Hampa village. He got profit during conventional farming he got 25 quintals. He got profit in only conventional farming compared to organic farming.

IV-DISCUSSION

By using Drava Jeevamrutham & Ghana Jeevamrutham and some organic insecticides like brahmastra & agnihastra, the farmer Sriramulu got more profit and natural crop when compared to the other farmers Narayanaswamy & Tirumalesh. If they also use the organic practices like Sriramulu, they will be also get the more profit and natural and healthy crop.

V-CONCLUSION:

In my research, by observing i conclude that organic farming is super natural gift from our ancient people.

BY practicing organic farming, we can learn a lot of things, as follow....

*Human beings and animals grow healthy when organic gardening is practiced, in our house premises.

*The food obtained from organic farming is safe and healthy for our human consumption.

So, this organic farming is the best way for our future generations.

GROW ORGANICALLY AND ENJOY THE NATURAL HEALTH

P.T.O

VI- REFERENCES:

1. Principles of organic farming by P.L.Maliwal.

2. Organic agriculture for sustainability by Dr.K.K.Krishna Murthy.

ORGANIC AGRICULTURE IS SOCIETY'S BRIGHTEST HOPE FOR POSITIVE LIFE

VII- AKNOWLEDGEMENTS:

1. The Principal & Dept. of Botany, S.K.P Govt. College, Guntakal
2. Narayana Swamy, Sriramulu, Thirumalesh and other farmers of Buggasangala village Guntakal Mandal, Anantapuramu District & Hampa village, Maddikera Mandal, Kurnool District.
3. Shajiya, Agricultural Assistant, Buggasangala & other Agricultural Officers of Guntakal Mandal.
4. Dr. A.S.Vijaya Kumar, Lecturer in Botany, Department of Botany. S.K.P. G.D.C, Guntakal.
5. Peer group of III B.Sc-BZC Students, who accompanied me, during this project work.

Nice presentation

P.T.O

PROJECT REPORT

Studies on "practice of Terrace farming" in Guntakal and neighbouring mandals of Anantapur (Dist.), A.P.

Submitted by

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20015/1258

Introduction :-

Terrace Farming may said to be Method of growing crops on sides of hill or Mountains by planting on graduated terraces buit into the slope.

The Method has been employed effectively to maximize arable land area in variable terrains and to reduce soil erosion and water loss

Terrace Farming main perpose to increase the Food production due to increase of population In Terrace Farming may crop can be cultivated by hilly areas whe the cultivated land is less consumares may more in number.

There are three types of Tarrac Farming:-

A) Bench terraced Fram land

B) sloping terraced Farm land

c) combinatination level terraced Farmlands.

A) Bench terraced Farm land

Bench terraces are a soil and water conservation measure used on sloping land with relatively deep soil to retain water and control erosion. They are normally constructed by cutting and filling to produce a series of level steps or benches. This allows water to infiltrate slowly in to the soil. Bench terraces are reinfored by retaining banks of soil or stones on the forward edges. This practice is typical for rice-based cropping systems.

B) sloping terraced Fram land.

A terrace consists of a flat or gently gemorphic surface, called a tread, that is typically bounded on one side by a steeper ascending slope, which is called a "riser" or "scarp". The tread and the stepper descending slope (riser or scarp) together constitute the terrace.

c) Combination Level Terraced Farmlands

It refers to the flattening of farmlands so that rain or irrigation water is evenly distributed over the field. and water runoff is minimized

Garden Farming :-

Gardening is the practice of growing and cultivating plants as part of horticulture in gardens ornamental plants are after grown For their flowers, Foliage. or overall appearance useful plants, such as root vegetables, leafy-vegetables, Fruits, and herbs, are grown for consumption, For use as dyes, or For medicinal or cosmetic use.

In Gardeing farming may use to growth of Sesonial plants like berry's. apple's or other kind of fruits may use to grown in gravden farming when they are adopted in a particular region also.

HISTORICAL BACK GROUND:-

Terrace Farming:-

Terrace Farming was invented by the "Inca" People who lived in the South American Mountains. This farming Method has made cultivation of crops in hilly or Mountainous regions possible. It is commonly used in Asia by rice-growing countries, such as Vietnam, Philippines, and Indonesia. In fact, the terraces of rice found in philippine's cordilleras have been acknowledged as a UNESCO World Heritage site. Apart from rice cultivation, terraces are also used to grow rice, potatoes, and Maize. Terrace farming is also commonly used in island such as the "canary Islands," because they have hilly terrains

Garden Farming:-

In 16th century the garden Farming started. In Europe garden design in the Renaissance was dominated by the "Italian garden" in the Renaissance, which developed in the French formal garden, dominating the "Baroque Period," both were formal styles, attempting to impose architectural principle's on the garden In the "18th" century. The English landscape garden developed.

Importance of Terrace Farming :-

The major benefit, of course, is the conservation of soil and water. Terraces reduce both the amount and velocity of water moving across the soil surface, which greatly reduces soil erosion. Terracing thus permits more intensive cropping than would other-wise be possible.

Indiana farmers have long recognized the importance of controlling soil erosion and the benefits of applying conservation measures. For years, the basic motivation behind their conservation efforts was to maintain the soils resource in a highly productive state

Importance of garden farming :-

Gardens are important to the plant because, despite being human-made, they represent a natural enviroment. plants and trees grow there taking in carbon and releasing oxygen. The roots of these plant's stabilze the soil and filter water.

Aims & objectives :-

* Increases farmability and land productivity of sloped fields
* contributes to water conservation slows down and reduces water runoffs, improves rainwater harvesting.
* Prevents soil erosion by decreasing rill formations.
* Boosts soil conservation
* Reduces sedimentation and water pollution. water stays long enough for havy particles to settle down and prevent downstream sedimentation and pollution of water bodies, but short enough not to harm crops.
* Increases food production by adjusting hilly land for farming.

Material Method :-

The th. Material we had collected from the internet and our teachers may tells about the Terrace farming and I had go to outcut's of our town and to collect information about the terrace farming by the help of farmer's in an hilly areas and we refered some organic farming of terrace.

Method's :-

For Terracing farming we need some Agricultural Tools hilly areas and water supply and also seeds

For Terrace Garding we need water, shade net, Poles, Garden Tools, containess

water :-

Terrace must have Access to water for watering and cleaning Having a water tap is essential than carrying water, arrange a source of Accessing water.

Shade net :-

Summer days in Tropical and Sub-Tropical Region can be very hot. Too much sunshine can with the plants and affect the growth protect the Garden with a covering such as old mosquito net or, Shade net from excessive heat and heavy Rains

Garden Tools :-

Some essental tools will be Required

Ex :- Spades, Wedges, Shovels etc

Trelis or Poles :-

Some vegetables in a garden may need support to grow especilly creepes and climbers when plating vegetables that are climbers They may need Trelis, Pre-pue pipes can poles for plant support.

Result :- Terrace farming

Table -1 : Details of the crops and yeal of various farmers in Pathakotha cherruvu village ground nut crop.

Name of the Farmer	landed area (Acres)	Crops	Yield (quintals of tonles) organic farming	conventional farming
Poliki . Dasarth	4	Ground nut	24	27
Naggappa	2	Groundnut	18	20
V. pecka Naik	1.5	Ground nut	12	15
Nagarasu	4	Ground nut	26	30
G. prasad	3	Paddy castor seeds	17	19.

Table.2 Details of the crops : profit / loss of various farmers in Patha kotha cherruvu village. Ground crops.

Name of the Farmer	landed area	Crop	Profit	Loss
Poliki Dasarth	4	Ground nut	3,00,000/-	2,00,000/-
Nagappa	2	Ground nut	1,90,000/-	1,60,000/-
V pecka Naik	1.5	Groundnut	1,00,000/-	80,000/-
Nagarasu	4	Ground nut	3,50,000/-	2,00,000/-
G. prased	3	Paddy castor seed's	30,000/-	10,000/-

Good data collection.

Discussion :-

Polik? Dasarth

The above farmer cultivated groundnut crop in both conventional and organic farming method as Terraces in the consecutive year 2021- 2022 about May to July it is an three month's crop he got more profit [During he got 24 quintals when organic farming and when conventional farming he got 27 quintals

Jagappa

He has cultivated ground nut crop in both conventional and organic farming as Terrous He used sprinklers system for water management He cultivated in the consective years 2021- 2022 He got More profit [during organic farming of Terrace he got 18 quintals and during conventional farming he got 20 quintals . which may result to getting of high profit.

References :-

1. https://en.wikipedia.org/wiki/Terrace
2. https://www.worldatlas.com/articles/
3. https://www.almanac.com

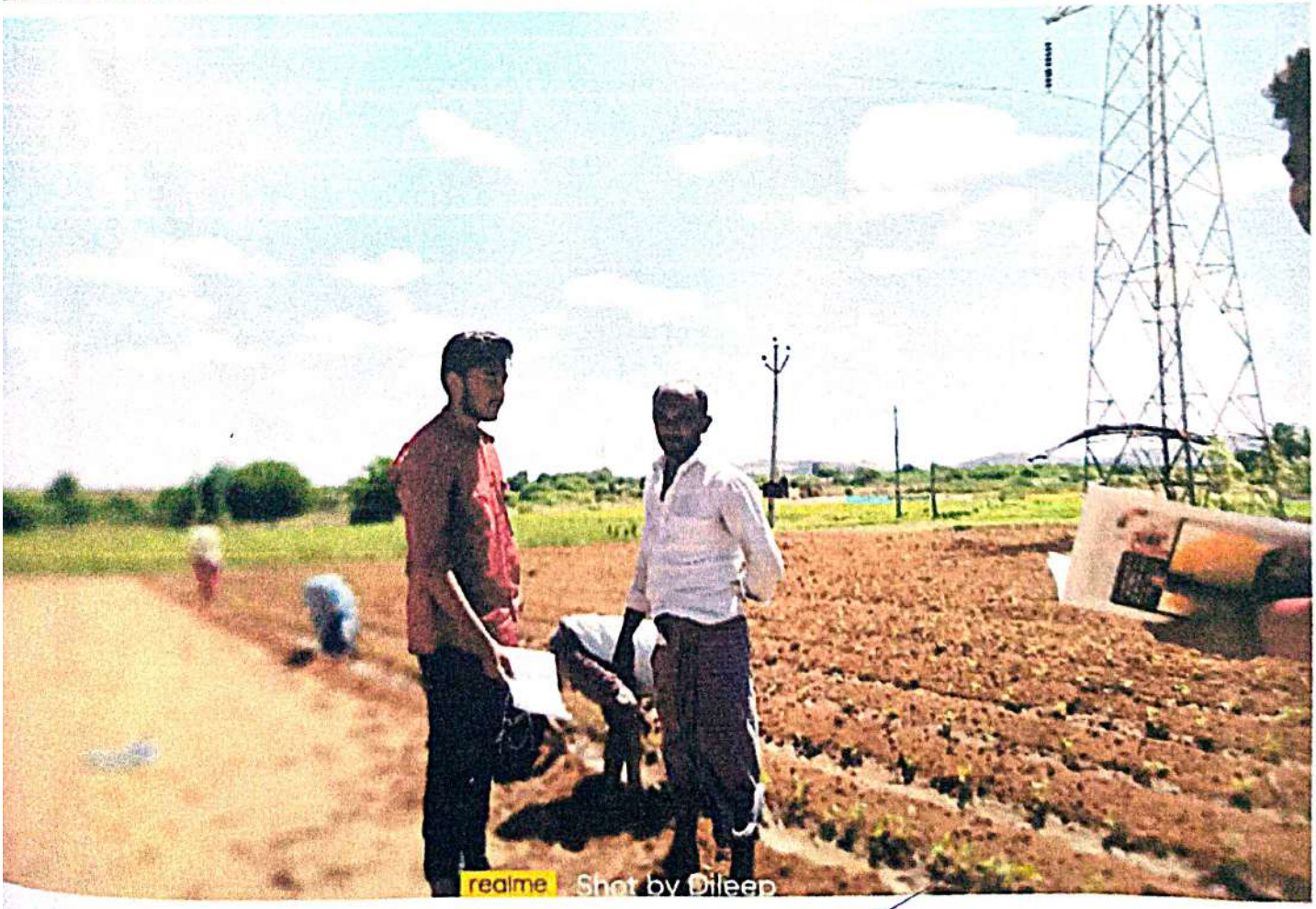
we had visited to terrace land brea and we had collected information abt from frame's and our teacher ~~me~~ helped us a lot to getting an information about Terrac Farming's

Acknowledgements :-

Thank's to our S.K.P govt digree collage kuntakal

special thank's to our Lecturer's to giveing this task

Thank's to Farmer's who had given information about Terrace Farming


realme Shot by Dileep

PROJECT REPORT

Studies on "practice of Vermi-compost Technology" in Guntakal mandal, Anantapur (Dist.) A.P.

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PREPARATION OF VERMICOMPOST

Introduction :-

Vermicomposting is the process by which worms are used to convert organic Materials [usually wastes] into a humus-like materials known as Vermin-compost. vermicompost is the production of compost using earthworms. The earthworms eats the organic residuces, digest it and excreate in the form of pellets. The earthworm excrete are called "wormcast." The vermicompost is a nutrient rich compost and helps better plant growth and crop yield. Vermicompost is method is method of Making compost with is generally is called vermicompost. locally avilable earthworms are also used for vermicomposting but their mode of feeding in very slow and earthworm which lives below the soil is also not suitable for vermicompost production. The Redworms and African earth are promisingworms used.

Vermicomposting is a chemical and biological process for recycling nutrients with the aid of earthworms and Microorganisms. Thus Vermicompost is considered as high nutrient biofertilizer with diverse Microbial communities. Vermicomposting technology is known the world and considered as a widely spread popular technology. Asa process for handling organic residuals, it represents an alternate approach in waste mangement neither landfilled nor burned but it considered but is considered a resource that may be recycled. It is a sustainable costeffective and ecological technology for efficient treatment of biodegrable wastes, and is thus widely adopted to recyle hazardous and worthless organic wastes into safe and valuble products.

Materials Required for vermicomposting :-

* Water
* Cow dung
* Thatch Roof
* Soil or Sand
* Gunny bags
* Earthworms
* Weed Biomass
* A large bin (plastic or cemented tank)
* Dry straw and leaves collected from paddy fields
* Biodegradable waste collected from fields and kitchen

Vermicomposting Methods

Different Vermicomposting Methods (Windrow) are placed, Beds a Bin, Flow Through Reactors Troughs, Towers and pit a Trench) Can be described under 2 types of System.

1. Batch system

Batch systems are ones in which all the bedding and food are mixed, the worms added and nothing more is done until the process is complete.

2. Continous flow systems

Continous-flow systems are ones in which worms are placed in bedding where upon feed new bedding are added incrementally on a regular basis.

Windrows Vermicomposting

Most Commercial farm Vermicomposting involves windows, which are long rows of cow Manure. farmers typically stack the manure in rows 3 feet wide, with worms, making certain to keep the rows moist.

Static pile Windrows (Batch Type)

Static pile windrows are simply piles of Mixed bedding and feed that are inoculated with worms and allowed to stand until the processing is complete. These piles are usually elongated in a windows style but can also be squares, rectangles, or any other shape that makes sense for the person building. them. They should not exceed on meter in height. Care must be taken to provide a good environment for the worms, so the selection of bedding type and amount is important.

TOP-Fed Windrows (continous Flow)

TOP-Fed windrows are similar to the Windrows described above, except that they are not mixed and placed as a batch, but are set up as a continuous-flow operation. This means that the bedding is placed first then inoculated with worms, and then covered repeatly with thin layers of food. The worms, and then covered repeatedly.

Wedges (continous flow)

The Vermi composting wedge is an interesting variation on the top-fed Windrow. An intial stock of worms in bedding is placed inside a conical-type structure.

Beds or Bin Vermi composting

The Simplest form of vermicomposting involves a bin made from plastic or untreated, non-aromatic wood. Some from of bedding, Such a shredded paper or composted animal Manure or decaying leaves, fill the bin and Mixes with a few handfuls of soil to provide the worms with material thorough which to burrow. The bedding also requires water to say moist and allow the worms bags and coffee grounds. Tossing in Some egg shells will add calicium for the worms and lower the bin's acidity level. However, never compost meat, fish or other, oily foods, otherwise the bin will add Calcium compost meat, fish or other fatty oily foods, otherwise the will produce a foul odour. Best worms for bin vermicompost.

Top-fed Beds (continous flow)

A top-fed bed woorks like a top-fed windrow. The main diffeerence is that the bed unlike a windrow, is contained within four walls and a floor. The beds can be built with insulted sides, or bales of straw can be used to insulte them in the winter. if the bins are fairly large, they are sheltered from the wind and precepitation, and the Feedstock is sreasonbly high in nitrogen, the only insulation required may be an insulating "pillow" or layer on top. There can be simple or bags or bales of straw.

Stacked Bins (Batch or continous flow)

Stacked bins adqressed the issue of space by adding the vertical dimension to Vermicomposting. The bins must be small enough to be lifted, either by hand or with a forklift, when they are full of wet material. They can be fed continously but this involves handling them on a sregular.

Flow - through Reactors Method

The worms live in a raised box, usually rectangular and not more than three meters in width. Materials is added to the top, and product is removed through a grid at the bottom. usually by means of hydraulically driven breaker bar. The term "flow-through" refers to the fact that the worms are never disturbed in their beds - the materials goes in the top. flows through the reactor and becomes out of the them and comes. The Method for pusing the materials out the bottom grate, losseing the materials so that falls through unit of approximately powered "breaker bars' that move along the bottom grate, looseing the materials so that fals thraugh. Clive Edwards 'has stated that a "properly Hanged" flow through unit of approximately 100 ft² Surface are can procum 2 to 3 tones per day organic waste. A variation of this system also used in vermitech.

Trough vermicomposting

Cement troughs can also host vermicomposting. usually the troughs hold only manure, which is aged for at least a week before being placed in the trough. This composting method begins with only a few inches of manure spread across the bottom of the trough. Farmers when worms, allowing them to feed on the manure for few days before adding another layer of manure More manure layers are added every 10 days until the worm compost reaches the top of trough

Tower method of vermicomposting

This method is quick and simple. Once set it never needs cleaning & casting removal. put a bunch of holes in a PVC pipe. plant it permanently and vertically in deep hole in the middle of the garden, back-fill it. place some worms. start filling with kitchen scraps a couples times a week.

pit or Trench

Some farms goes for vermicomposting pit digging a large hole in which to bury the worms and organic waste materials. of course, before adding the worms and bedding. farmers must line the pit to prevent worms from escaping into the surrounding soil. Canvas feed bags make a good lining, preventing worm passage yet still allowing for suitable water drainge. farmers fill the lined pit with organic materials such as straw, grass clippings and Manure. Farmers fill the lined pit with organic Materials, such as straw, grass clippings and Manure, and then cover it with soil After about week, during which time the pit is wanted to maintain its moisture farmers, add worms. The worms immediately burrow into the pit, beging the vermi composting procedure.

Results :-

Benefits of vermicompost

1) In Soil

* Improving the soil texture, improving aeration, and helping plant roots anchor better in the earth.
* Increasing the soil water retention capacity of soil.
* Enriches soil with Micro-organisms.
* Microbial activity in worm casting is 10 to 20 times higher than that in the normal soil and organic matter that the worm ingests.
* Addition of vermi Compost to soil attracts deep-burrowing earthworms already present in the soil.
* Improves water holding capacity.
* The worm castings are rich in humic acids, which condition the soil and helps in balance pH.

Discussion :-

* Enhances germination, plant growth, and yield of the crop
* Improves root growth and structure of plant in the porous soil.
* Enriches soil with Microbial biomass.
* worm castings five times more nitrogen Seven times more phosphorus, and eleven times more potasium than ordinary soil, the main, minerals needed for plant growth.
* provides plants with essential nutrients and aids, the Suppression of plant disease.
* worm castings contain five times more nitrogen, seven times more phosphorus, and eleven times more potassium than ordinary soil, the main minerals needed for plant growth.

Conclusion :-

vermicomposting is an alternative method for waste Mangement through which vermi compost is produced with realtively high nutorient Content than Compost and manures. So it can be used to shift from chemical fertilizers to reduce the hazardous effect of chemicals to both crop and human being Application of vermicompost either or in combination with fertilizers promotes crop yield. it is today's natural fertilizers as nature inteded and it is the best solution to immediate problem of declining soil fertility and for production of food thus is the best means for adapting pollution. Soil degradation and discriminate use of chemical Fertilizers it improves the soil physical conditions which support better aeration to plant root, drainge of water, facilater of cations Sustained availability of nutorients and thereby the uptake by plants resulting better growth

It improves the soil physical conditions which support better aeration to plant root, drainge of water facilation of cations exchange sustained availability of nutrients, and thereby the uptake by the plants resulting in better growth use of vermicompost continues an important alternative source of fertilizers that has environmental benefits productivity and crop quality as compared to inorganic fertilizers. Vermicomposting and its application could be a better option and farmers need to be educated about the importance of vermicompost.

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Very good study work

PROJECT REPORT

Study of "POLLUTION LEVELS OF GUNTAKAL Town" Guntakal mandal, Anantapuramu Dist. A.P.

Submitted by

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to the Department of Botany, SKP Govt. Degree College, Guntakal as a part of Paper-VI of Botany curriculum (V-semester), during the academic year 2021-22. It is a record of Bonafide Project work carried-out by him/her under my supervision.

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TOPIC:- POLLUTION LEVELS OF GUNTAKAL

Aim:

To study the Water pollution in Guntakal.

Objectives:

Observe oxygen consumption caused by natural pollutants.

* The understand the use of a control when conducting experiment.

Introduction:

Pollution is the introduction of contamination into the natural environment that cause adverse change Pollution can take the form of chemical substances or energy. Such as noise, heat or light. Pollutants of the components of pollution is often classified as point source or non-plant source pollution. In 2015, pollution killed a million people World Wide.

Major forms of Pollution include light pollution, littering, noise pollution, Plastic Pollution, Soil contamination, radioactive contamination, thermal pollution, Visual pollution, Water Pollution.

The forms of pollution are listed below along with the particular contaminant relivant to each of them.

Water Pollution :-

By discharge of waste water from commercial and industrial waste into surface. Water discharge of untreated domestic sewage.

The cause of water pollution include a wide range of chemicals and pathogens as well as physical parameters, contaminants may include Organic and in Organic Substances Elevated tem-Peratures can also lead to polluted water.

Soil Pollution :-

As the Presence of toxic chemicals in soil in high enough concentration to pose a risk to human health and/or the ecosystem.

All soils, Whether polluted or unpolluted contain a variety of compounds which are natu-rally Present. Such contaminants include metals, inorganic ions and salts. These compounds are mainly formed through soil microbial activity and decom-Position of organisms. Additionally, various compo-unds get into the soil from the atmosphere, for instance with Precipitation water as well as by wind activity from surface water. Soil acts as a natural sink for contaminants, by acculating and Sometimes concentration contaminants.

Materials :-

* Dish washing liquid, soil, vegetable oil
* Tap water
* 6 Glass jars or clear plastics cups if doing the experiment in front of class.
* 1 Spoon 1 per group
* Coffee filter paper
* Worksheet, water pollution
* Samples of litter found on the beach
* Litter Biodegradation information sheet
* Stones and sand for filtration for advanced experiment.

Methods :-

1. Keep out oils, fat, or grease from the sink.
2. Abstain from flushing contaminated liquids, pills, drugs or medications down the drain.
3. Desist from using the toilet as a bin.
4. Ensure minimal use of bleach or detergents.
5. Reduce the use of herbicides Pesticides, and fertilizers.
6. Proper sewage treatment and Management.
7. Dispose trash properly.
8. Avoid direct dumping into water systems.
9. Always conserve water.
10. Insists on using environmentally safe Products.

Contamination Can Include :-

Dirt :

Is manageable in small quantities. However dirt from erosion, landslides and from the street can destroy the equality of water in the streams, rivers lakes and the ocean.

Leaves and Grass clippings :-

Can block out the sun and rotting materials can suffocate plants and animal and in the water.

Litter :-

Such as Cigarette butts, tin cans, plastic bag bottles and paper etc are littered on the streets or blown from rubbish bins can eventually end up on the seashore and in the oceans Animals in the Ocean. Can often mistake this for food or get in it causing sickness and death.

WATER POLLUTION EXPERIMENT

1. Put some tap water in 3 separates clean. Clear glasses or bottles and observe the water. We should record their observation in the first section of the worksheet.
2. Add the soil to one glass of tap water to create and "Polluted water" the washing up liquid to the next and the soil to the last glass. We should complete their observation in section 2 of the work sheet.

3. Stir the glasses of water with a spoon to create Movement in the polluted water observe to the water.

4. Continue to observe the water and see that what happens after 5 minutes.

5. Filter the polluted water through the coffee filter into a clean glass, repeat for each glass and observe.

Result and Discussion :

Water Samples used in experiment.	Observations : 1. What does it look like ? 2. What colour is it ? 3. What does it smell like ?
1. Tap Water	Odourless, colourless and without any contaminated Particles.
2. Polluted water	It colour changes to brown and foul smell & contaminated particles.
3. Polluted water after movement	It contain many disposal, wastes and it smells.
4. Polluted water after standing for five minutes.	Total water contaminated and it changes it colour.
5. Filtered water using a coffee filter	After filtering water changes
6. Other Item used for filtering.	It changes and separate many soil particles and dust.

RESULT:-

Nitrogen Content in Polluted water:

* As we know that nitrogen makes plant grow but too much nitrogen flows to rivers, bays etc.
* Nitrogen flows to rivers and which disturbs marine life and kills fishes.
* According to scientists nitrogen pollution from fertilizers and other sources has major environment problems.
* Nitrogen pollution is caused by excess of nitrogen and Phosphorous in air and water.
* Too much nitrogen and Phosphorous in the water causes algae to grow faster than ecosystem can handle.

Fresh waters naturally contains chemicals dissolved from soils and rocks over which they flow. The major inorganic element include calcium, Magnesium, Sodium, Potassium, Carbon, Chloride, and Sulfur as well as plant nutrients.

Such as nitrogen, silicon and Phosphorus Organic compounds derived from decaying biological material.

"By the above experiment of water pollution we can identified as excess of chemicals are mixing up in the water and contaminated some rivers So it called as the water pollution not only chemical Some Agriculture waste, Sewage water, industrial waste etc".

Prevent Water Pollution :-

- Conserve water by turning off the tap when running water is not necessary. This helps Prevent water shortages and reduces the amount of contaminated water that needs treatment.

- Be careful about what you throw down your sink or toilet. Don't throw paints, oils or other forms of litter down the drain.

- By having more plants in your garden you are preventing fertilisers from running off into nearby water source.

WATER POLLUTION :-

Water pollution include contamination due to the domestics wastes, insecticides and herbicides food processing waste, pollutants from Livestock, operations Volatile organic compounds., heavy metals chemical waste and others water born disease caused by polluted drinking water include typhoid, amebiasis giardiasis, ascariasis, hookworm etc.

When water is contaminated with chemicals such as pesticides, hydrocarbons, pesticides organic pollutants.

It could lead to cancer, including prostate cancer and non-Hodgkin Lymphoma, harmonal problems that can disrupt reproductive and development processes damage to nervous system.

Nutrient pollution causes overgrowth of toxic algae eaten by other aquatic it may causes to death.

Chemical Contamination is known to cause decline in frog biodiversity and tadpole mass oil pollution can negativity affect development of marine organisms.

Water pollution may disrupt photosynthesis in aquatic plant and thus affects ecosystem that depends on these plants may absorb pollutants from water and pass them up the food chain to consumer animals and humans.

Lead and Public Health:

Water Quality Criteria and standard for lead and other Pollutants.

Water pollutants Criteria draw from the available technical database for a particular ambient water pollution.

Toxic pollutants refer to a group of 65 substance or substance groups is also known as the toxic Consent of Decree toxics.

Each item on the list of the 65 refers to a chemical group.

Individuals Contaminants, further more group of chemically complex substance may be difficult to analyze quantitatively a prerequisite for compliance and compliance enforcement.

The national recommed water quality criteria for lead as with all the pollutant Criteria included freshwater and salt water acute and chronic, these were no human. Health criteria for lead in terms of fish Consumption or fish plus water consumption previous values having been is regulated by several priority water pollutant and a hazardous air pollutant.

Health Organisation [WHO] and the International Agency for research on Cancer the regulation. are largely based on Organolead Compounds and their Carcinogencity to humans.

Municipal Waste Water

Municipal waste water is as important a source of water pollution as industrial waste. When we defines a population equivalent of municipal dischaarge as equivalent of municipal dischaarge as equivalent of dischaarge.

$$(0.5)\ (20{,}000) = 10{,}000$$

Similarly, if each individual contributes 0.2 lb solid / day into waste water and an industry dischaarge 10001b/day industry has a population equivalent of

1000 / 0.2 or 5000 persons

Almost all of the cities with combined sewers have built treatment plants that can treat dry weather flow - the sanitary wastes when there is no storm water runoff. As long as it does not rain the plants can handle the flow of provide sufficient treatment.

Agricultural Wastes: Should they flow directly into surface water have a collective population equivalent of about 2 billions.

Acid mine drainage has polluted surface waters since the beginning of ore mining sulfur laden water leaches from mines including old and abondoned mines.

Sulfur compounds that oxidize to sulfur compounds that oxidize to sulfur acid on contact with air by studying one more specific interaction of pollutants with that ecosystem.

MEASUREMENT OF WATER QUALITY

Many water pollutants are measured in terms of milligrams of the substance per liter of water (mg/L)

A third commonly used parameter is present weight relationship. Note that 10,000 ppm = 1 when 1ML = 1g.

WASTE MANAGEMENT:

Photolysis of H_2O_2 Photolytic process of water pollutants a treatement by hydrogen peroxide are being studied as a profitable way to oxidative degradation of scarcely reactive substrate. The high effectiveness of H_2O_2 Photolysis for the treatment of waste water is in fact the treatment of waste water is in fact providing by reactions involving OH radicals.

The H_2O_2 / UV is efficient in mineralizing Organe pollutants, but exhibit slow kinetic Compared to O_3/UV.

A disadvantages of conventional / AOPs such as O_3/UV and H_2O_2/UV or their combination is that they cannot utilize abundant Solar light as the source of UV light because for required UV energy for the photolysis of the oxidizer is not available in the solar spectrum.

SOURCES OF WATER POLLUTION :-

Water pollutants are categorized at point source or non-point source the former being identified as all dry weather pollutants that enters channels. Storm drainage even though the water may water courses by way of pipes or channels is considered non point source pollution.

Point source pollution comes mainly from industrial facilities and Municipal waste water treatment plants.

"The range of pollutants is vast depending only on what gets "thrown down the drain".

Oxygen-demanding substances, such as might be discharged from milk Processing plants, breweries, or paper mills, as well as municipal waste water treatment plants, make up one of the most important types of pollutants.

Suspended solids also contribute to oxygen deplention in addition they create unsightly condition.

Nutrients mainly nitrogen and phosphorus can Promote accelerated and some of the bio concentrated metals can adversely affect aquatic ecosystem as well as make the water unusable for human contact or consumption.

Lowering the solubility of oxygen in the water is because gas solubility of water is inversely Proportional temperature the amount of oxygen demand.

Managing Risk & Drinking Water Quality:

Home owners or business that have their own ground water well should have the supply tested for all of the regulating drinking water pollutants and pesticides.

Because most unregulated chemicals could that double the cost depending on the chemicals.

Health & Safety Standard

These are organized by air, Land and water pollutants. Each is comprehensive environment profile of the specific industry. They contain industrial Process information and pollutant release data Also, contained are compliance and enforcement histories.

Occupational safety and health Administration (OSHA) the National Institute for occupational Safety and health. The toxic substance and disease registry [ATSPR] the Environment Protection Agency, World health Organization have developed recommended health and safety standard to protect the workers surrounding.

WHO serves as the directing and co-ordinating authority for International health matters and public health.

Responsibility that it fulfills in part through its publication Programmes.

Useful TOPIC.

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CERTIFICATE

This is to certify that the cluster (B3) paper of Botany curriculum (VI-semester) for III Year B.Sc. students "Project work " entitled studies on "mushroom cultivation" in some places of A Vilages of Guntakal and mandal of anthanpur

It has been submitted by Mr/ Miss. S. Shabana,

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DEPARTMENT OF BIOCHEMISTRY

S.K.P GOVT COLLEGE, GUNTAKAL

N. RHIZWANA

M.Sc., Ph.D.

CERTIFICATE

This is to certify that the project entitled "Graves' disease" is submitted by M.Jhansi rani, S.Siveesha, Y.Latha Under my supervision for the degree of Bachelor of science in Biochemistry group

9/10/2022

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DECLARATION

I hereby declare that the project entitled "Graves' disease" done by B. Sc Biochemistry towards the partial fulfilment of the requirement for the award of the degree of Bachelor of Science in Biochemistry, is result of work carried out under the guidance of N. RHIWANA, M.Sc., Ph.D.

I further declare that this project report has not been previously submitted before either in part or full for the award of any degree.

M. Jhansi Rani
S. Sireesha.
Y. Latha.

Student name:

M. Jhansi rani

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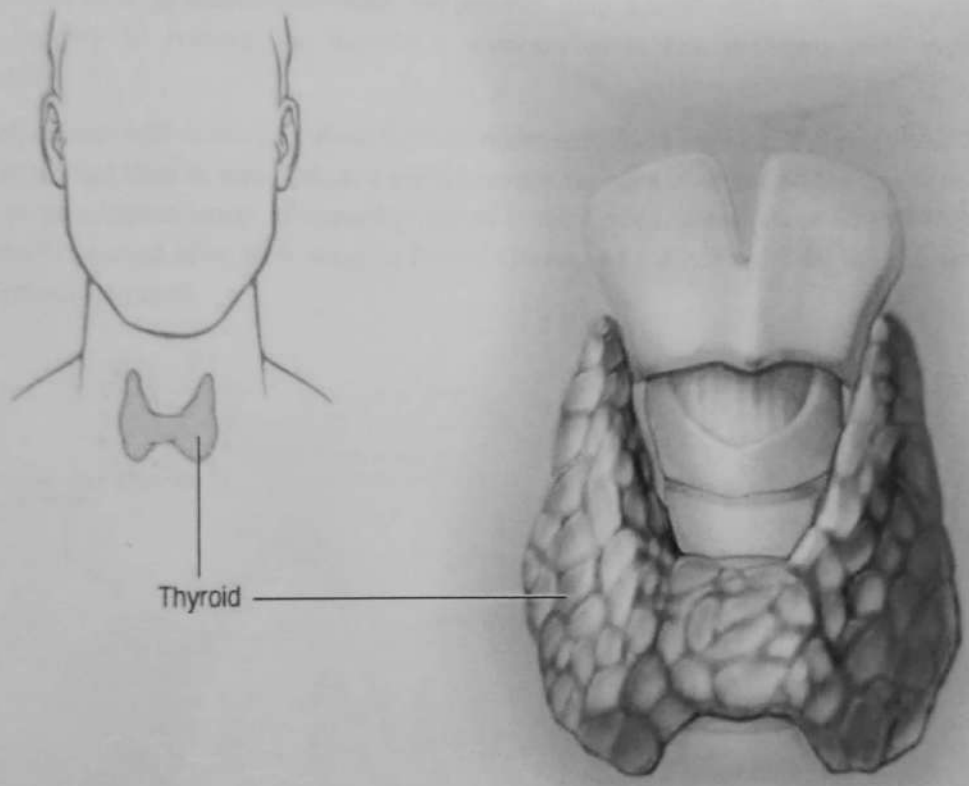
Introduction:-

Graves' disease is an immune system disorder that results in the overproduction of thyroid hormones (hyperthyroidism). Although a number of disorders may result in hyperthyroidism, Graves' disease is a common cause. Thyroid hormones affect many body systems, so signs and symptoms of Graves' disease can be wide ranging. Although Graves' disease may affect anyone, it's more common among women and in people younger than age 40. The primary treatment goals are to reduce the amount of thyroid hormones that the body produces and lessen the severity of symptoms.



Thyroid gland

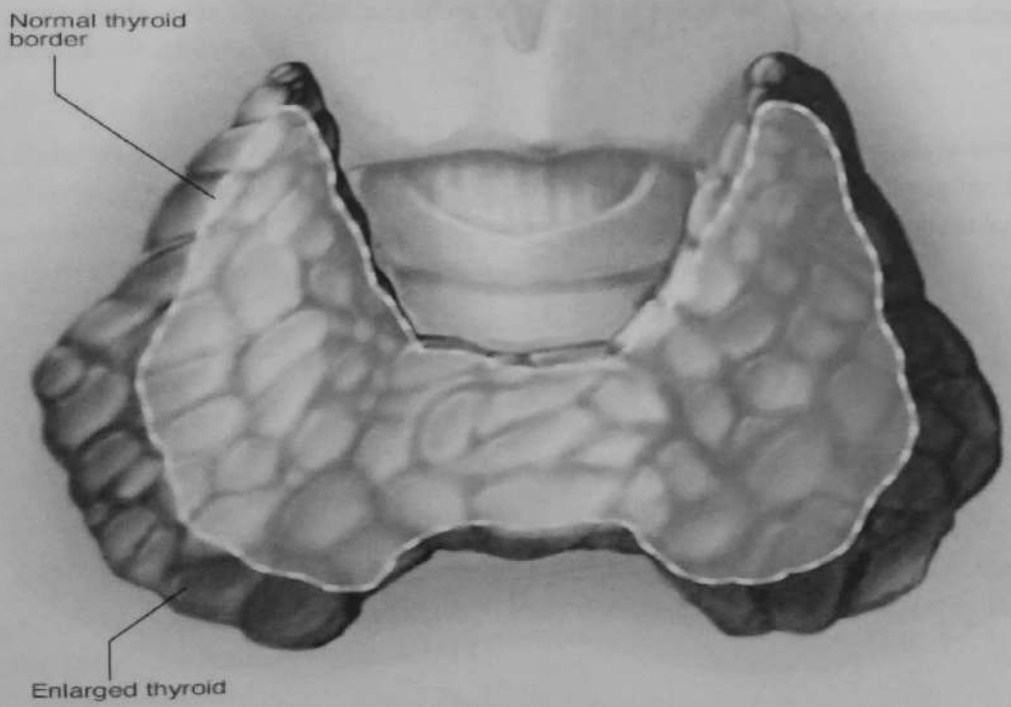
Graves' disease, also known as **toxic diffuse gaiter**, is an autoimmune disease that affects the thyroid. It frequently results in and is the most common cause of hyperthyroidism. It also often results in an enlarged thyroid. Signs and symptoms of hyperthyroidism may include irritability, muscle weakness, sleeping problems, a fast heartbeat, poor tolerance of heat, diarrhoea and unintentional weight loss. Other symptoms may include thickening of the skin on the shins, known as pretibial myxoedema, and eye bulging, a condition caused by Graves' ophthalmopathy. About 25 to 80% of people with the condition develop eye problems.

The exact cause of the disease is unclear; however, it is believed to involve a combination of genetic and environmental factors. A person is more likely to be affected if they have a family member with the disease. If one twin is affected, a 30% chance exists that the other twin will also have the disease. The onset of disease may be triggered by physical or emotional stress, infection or giving birth. Those with other autoimmune diseases such as type 1 diabetes and rheumatoid arthritis are more

likely to be affected. Smoking increases the risk of disease and may worsen eye problems. The disorder results from an antibody, called thyroid-stimulating immunoglobulin (TSI), that has a similar effect to thyroid stimulating hormone (TSH). These TSI antibodies cause the thyroid gland to produce excess thyroid hormones. The diagnosis may be suspected based on symptoms and confirmed with blood tests and radioiodine uptake. Typically, blood tests show a raised T_3 and T_4, low TSH, increased radioiodine uptake in all areas of the thyroid and TSI antibodies.

The three treatment options are radioiodine therapy, medications, and thyroid surgery. Radioiodine therapy involves taking iodine-131 by mouth, which is then concentrated in the thyroid and destroys it over weeks to months. The resulting hypothyroidism is treated with synthetic thyroid hormones. Medications such as beta blockers may control some of the symptoms, and antithyroid medications such as methimazole may temporarily help people while other treatments are having effect. Surgery to remove the thyroid is another option. Eye problems may require additional treatments.

Graves' disease will develop in about 0.5% of males and 3% of females. It occurs about 7.5 times more often in women than in men. Often, it starts between the ages of 40 and 60 but can begin at any age. It is the most common cause of hyperthyroidism in the United States (about 50 to 80% of cases). The condition is named after Irish surgeon Robert Graves, who described it in 1835. A number of prior descriptions also exist.



Enlarged thyroid

Father of Graves disease:-

Robert James Graves (1796-1853) a superb general physician, Graves' contributions to medicine extend into the fields of cardiology, endocrinology, neurology, infectious disease and more.

The golden age of Irish medicine is associated with many great names — Stokes, Cheyne, Corrigan — but among these greats many consider Robert James Graves to be the most outstanding and extraordinary of his time.

Graves is widely known in endocrinology for his paper, Newly observed affection of the thyroid gland in females, published in 1835. In it, he details the clinical features known today as Graves' disease. However, his contributions to medicine extended far beyond the then unknown field of endocrinology. Most notable among them was his devotion to the use of bedside teaching.

Robert James Graves

"Graves' major contribution to medicine was the initiation of teaching-ward rounds in these islands," T. J. McKenna, MD, professor in the department of endocrinology and diabetes mellitus, St. Vincent's University Hospital, Dublin, told Endocrine Today. "He provided an example of an academic physician whose guiding principles might, with advantage, be embraced by those in pursuit of an academic medical career."

In a description by Armand Trousseau, a prominent French physician of the same time, Graves is described as "a perfect clinical teacher. … An attentive observer, a profound philosopher, an ingenious artist, an able therapeutist; he commends to our admiration the art whose domain he enlarges, and the practice of which he renders more useful and more fertile."

Early influence

Graves was born in Dublin, Ireland, in 1796 to Richard Graves, a senior fellow at Trinity College.

Graves was educated at Trinity College where he maintained first in his class and was the recipient of the gold medal for scholastic excellence, the highest distinction awarded to a student by the university. He graduated with a medical degree in 1818.

After graduation, Graves spent the next several years traveling around Europe and studying at the important medical centres in England, Germany, Austria and France.

Upon his return in 1821, Graves was appointed as a staff physician at Meath Hospital, the principal public hospital in Dublin. There he introduced the system of clinical and bedside teaching that he

observed and admired while in Europe. Graves was described as tall, dark and dynamic. His daily teaching clinic, which was taught in English instead of the traditional Latin, was said to be attended by more than 100 students.

Breaking from the traditional methods of teaching medicine, which required students to have extensive book knowledge but little practical experience, Graves had his students examine patients, present histories and review physical findings and treatments with their professor at the bedside of the patient. His methods also required students to attend autopsies to correlate the findings with symptoms and signs observed before a patient's death.

Although his interests were diverse, Graves was frequently involved in the treatment of infectious diseases and fevers, specifically typhus, which was at epidemic proportions during this period of Ireland's history.

"While being a relatively wealthy man, he had a real concern for the underprivileged and disadvantaged for whom he was an effective champion," McKenna said. "In his youth, with little thought for his own health, he led a small group of physicians across Ireland from Dublin to Galway where typhus was raging."

At the time, low diet was recommended as "an indispensable condition in the treatment of fevers." However, Graves completely reversed medical practice on this point and instead recommended exposure to fresh air and the increased consumption of food and liquids.

"He requested that his epitaph should read, 'He fed fevers,' which clearly, in his own estimation, was his most significant contribution to medical science," McKenna said.

In 1824, Graves cofounded the Park Street School of Medicine. Shortly after, he was appointed professor at the Institute of Medicine at Trinity College, a post which he held until 1841.

Graves' disease

During this time he made many original contributions to medicine. In 1834, Graves delivered a series of lectures detailing three cases of violent palpitations in women with enlarged thyroids and apparent enlargement of the eyeballs.

"Graves gave one of the first descriptions of the association of goiter, generalized increase in metabolism and ocular abnormalities, which have come to be known as thyroid ophthalmopathy," McKenna said. "Graves described florid thyrotoxicosis in a woman who, in retrospect, had autoimmune mediated thyrotoxicosis."

The disease would later be identified as the most common form of hyperthyroidism. Trousseau suggested the eponym Graves' disease. In Europe, the condition is now frequently referred to as von Basedow's disease after Karl Adolph von Basedow. The eponym Graves' disease is more commonly used in the United States.

Clinical lectures

After leaving Trinity College and Meath Hospital, Graves was appointed president of the Royal College of Physicians of Ireland in 1843. The same year he published a major textbook, A system of clinical medicine, based on lectures delivered in Sir Patrick Duns Hospital.

A second edition was published in 1848 and translated into French, German and Italian. The second edition included a criticism by Trousseau who described Graves' work as "the most remarkable and important lectures. There is not one of them, in fact, which does not abound in practical deductions."

First among them is a lecture about clinical instruction, in which Graves admires the German method of clinical teaching and instructs that "from the very commencement, the student ought to witness the progress and effects of sickness, and ought to persevere in the daily observation of disease during the whole period of his studies.

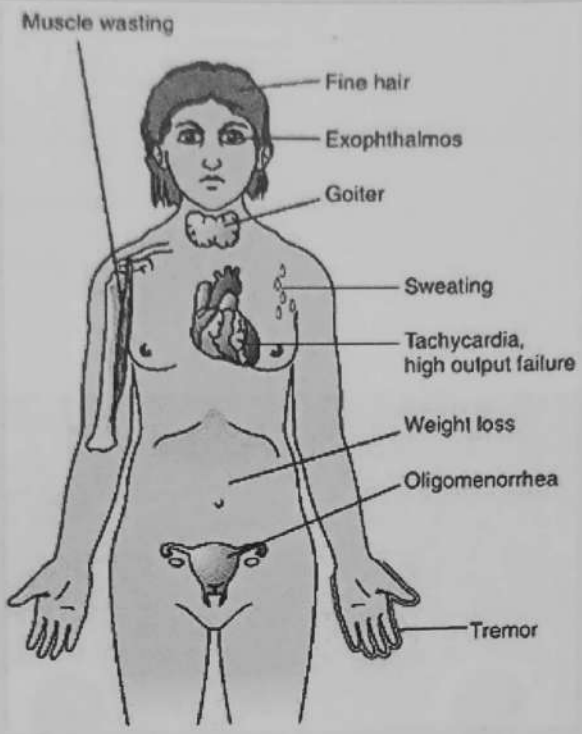
"Remember, therefore, that however else you may be occupied — whatever studies may claim the remainder of your time, a certain portion of each day should be devoted to attendance at a hospital, where the pupil has the advantage of receiving instruction from some experienced practitioner," he wrote.

Graves even encouraged his students to learn the cost of prescriptions so that when they entered private practice they could consider a patient's financial situation when they prescribed the appropriate medicines.

The 70 lectures included in the textbook cover a wide range of topics: inflammation, scarlatina, cholera, influenza, gout, rheumatism, nervous diseases, paralysis, pneumonia, asthma, gangrene, pericarditis, headaches, tape worm, venereal disease, sleeplessness, extensive lectures on fever and diseases of the digestive organs, kidney, skin, respiratory organs, heart and more. Among the many novel concepts included in the textbook was the pinhole pupil after pontine hemorrhage, timing the pulse by watch and abandoning the practice of bleeding patients with pyrexia.

When Graves' term as president of the Royal College of Physicians ended, he became a much less prominent figure in medicine and little is known about his last few years of life. He died of an abdominal tumor on March 20, 1853.

Signs and symptoms :-



Graves' disease symptoms

The signs and symptoms of Graves' disease virtually all result from the direct and indirect effects of hyperthyroidism, with main exceptions being Graves' ophthalmopathy, goiter, and pretibial myxoedema (which are caused by the autoimmune processes of the disease). Symptoms of the resultant hyperthyroidism are mainly insomnia, hand tremor, hyperactivity, hair loss, excessive sweating, oligomenorrhea, itching, heat intolerance, weight loss despite increased appetite, diarrhea, frequent defecation, palpitations, periodic partial muscle weakness or paralysis in those especially of Asian descent, and skin warmth and moistness. Further signs that may be seen on physical examination are most commonly a diffusely enlarged (usually symmetric), nontender thyroid, lid lag, excessive lacrimation due to Graves' ophthalmopathy, arrhythmias of the heart, such as sinus tachycardia, atrial fibrillation, and premature ventricular contractions, and hypertension. People with hyperthyroidism may experience behavioral and personality changes, including psychosis, mania, anxiety, agitation, and depression.

Common signs and symptoms of Graves' disease include:

- Anxiety and irritability
- A fine tremor of the hands or fingers
- Heat sensitivity and an increase in perspiration or warm, moist skin
- Weight loss, despite normal eating habits
- Enlargement of the thyroid gland (goiter)
- Change in menstrual cycles

- Erectile dysfunction or reduced libido
- Frequent bowel movements
- Bulging eyes (Graves' ophthalmopathy)
- Fatigue
- Thick, red skin usually on the shins or tops of the feet (Graves' dermopathy)
- Rapid or irregular heartbeat (palpitations)
- Sleep disturbance

Graves' ophthalmopathy

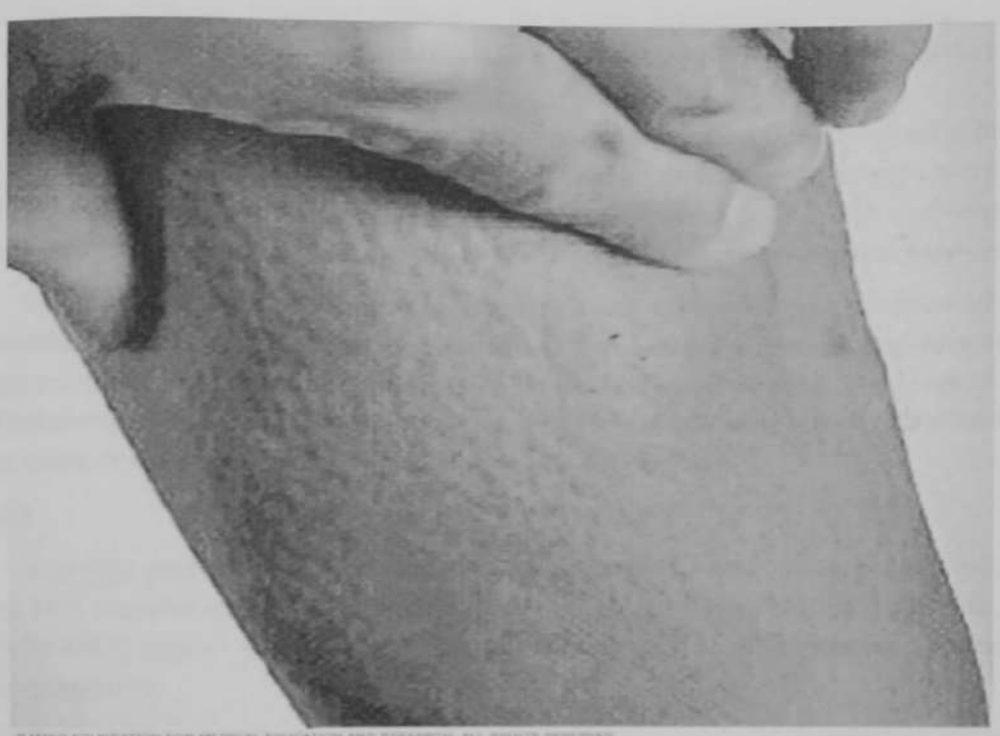
Graves' ophthalmopathy

About 30% of people with Graves' disease show some signs and symptoms of Graves' ophthalmopathy. In Graves' ophthalmopathy, inflammation and other immune system events affect muscles and other tissues around your eyes. Signs and symptoms may include:

- Bulging eyes
- Gritty sensation in the eyes
- Pressure or pain in the eyes
- Puffy or retracted eyelids
- Reddened or inflamed eyes
- Light sensitivity

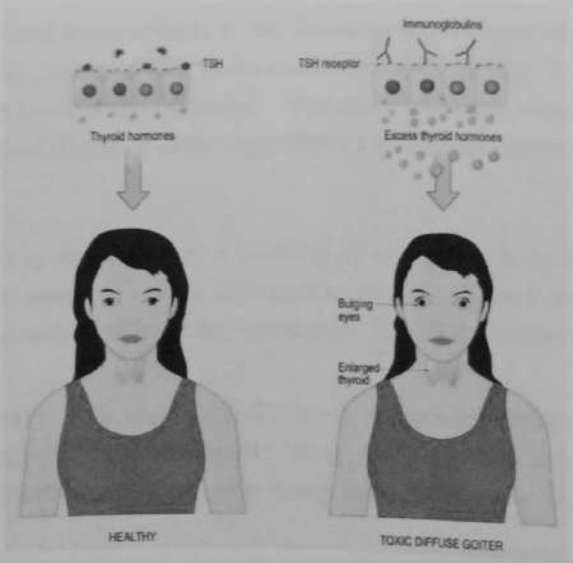
- Double vision
- Vision loss

Graves' dermopathy



Graves' dermopathy

An uncommon manifestation of Graves' disease, called Graves' dermopathy, is the reddening and thickening of the skin, most often on your shins or the tops of your feet. People with Graves' disease develop Graves' dermopathy, a skin condition characterized by red, swollen skin, usually on the shins and tops of the feet. The texture of the affected skin may be similar to that of an orange peel. Doctors may also refer to the condition as pretibial myxedema.



Causes:-

Graves' disease is caused by a malfunction in the body's disease-fighting immune system. It's unknown why this happens.

The immune system normally produces antibodies designed to target a specific virus, bacterium or other foreign substance. In Graves' disease — for reasons that aren't well understood — the immune system produces an antibody to one part of the cells in the hormone-producing gland in the neck (thyroid gland).

Normally, thyroid function is regulated by a hormone released by a tiny gland at the base of the brain (pituitary gland). The antibody associated with Graves' disease — thyrotropin receptor antibody (TRAb) — acts like the regulatory pituitary hormone. That means that TRAb overrides the normal regulation of the thyroid, causing an overproduction of thyroid hormones (hyperthyroidism).

The exact cause is unclear; however, it is believed to involve a combination of genetic and environmental factors.[2] While a theoretical mechanism occurs by which exposure to severe stressors and high levels of subsequent distress such as PTSD (Post traumatic stress disorder) could increase the risk of autoimmune disease and cause an aggravation of the autoimmune response that leads to Graves' disease, more robust clinical data are needed for a firm conclusion.[9]

Genetics

A genetic predisposition for Graves' disease is seen, with some people more prone to develop TSH receptor activating antibodies due to a genetic cause. Human leukocyte antigen DR (especially DR3) appears to play a role. To date, no clear genetic defect has been found to point to a single-gene cause.

Genes believed to be involved include those for thyroglobulin, thyrotropin receptor, protein tyrosine phosphatase nonreceptor type 22 (PTPN22), and cytotoxic T-lymphocyte–associated antigen 4, among others.

Infectious trigger

Since Graves' disease is an autoimmune disease which appears suddenly, often later in life, a viral or bacterial infection may trigger antibodies which cross-react with the human TSH receptor, a phenomenon known as antigenic mimicry.

The bacterium *Yersinia enterocolitica* bears structural similarity with the human thyrotropin receptor[10] and was hypothesized to contribute to the development of thyroid autoimmunity arising for other reasons in genetically susceptible individuals. In the 1990s, it was suggested that *Y. enterocolitica* may be associated with Graves' disease.[14] More recently, the role for *Y. enterocolitica* has been disputed. Epstein–Barr virus (EBV) is another potential trigger.

Graves' ophthalmopathy

Graves' ophthalmopathy results from a buildup of certain carbohydrates in the muscles and tissues behind the eyes — the cause of which also isn't known. It appears that the same antibody that can cause thyroid dysfunction may also have an "attraction" to tissues surrounding the eyes.

Graves' ophthalmopathy often appears at the same time as hyperthyroidism or several months later. But signs and symptoms of ophthalmopathy may appear years before or after the onset of hyperthyroidism. Graves' ophthalmopathy can also occur even if there's no hyperthyroidism.

Risk factors

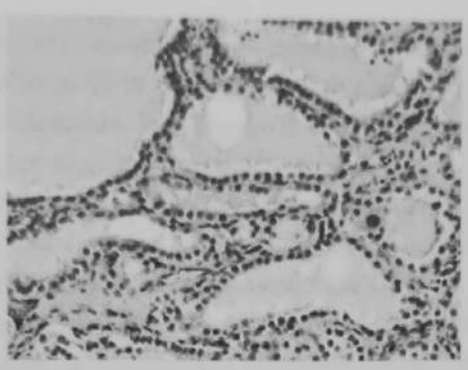
Although anyone can develop Graves' disease, many factors can increase the risk of disease, including:

- **Family history.** Because a family history of Graves' disease is a known risk factor, there is likely a gene or genes that can make a person more susceptible to the disorder.
- **Sex.** Women are much more likely to develop Graves' disease than are men.
- **Age.** Graves' disease usually develops in people before age 40.
- **Other autoimmune disorders.** People with other disorders of the immune system, such as type 1 diabetes or rheumatoid arthritis, have an increased risk.
- **Emotional or physical stress.** Stressful life events or illness may act as a trigger for the onset of Graves' disease among people who have genes that increase their risk.
- **Pregnancy.** Pregnancy or recent childbirth may increase the risk of the disorder, particularly among women who have genes that increase their risk.
- **Smoking.** Cigarette smoking, which can affect the immune system, increases the risk of Graves' disease. Smokers who have Graves' disease are also at increased risk of developing Graves' ophthalmopathy.

Mechanism :-

Thyroid-stimulating immunoglobulins recognize and bind to the thyrotropin receptor (TSH receptor) which stimulates the secretion of thyroxine (T4) and triiodothyronine (T3). Thyroxine receptors in the pituitary gland are activated by the surplus hormone, suppressing additional release of TSH in a negative feedback loop. The result is very high levels of circulating thyroid hormones and a low TSH level.

Pathophysiology



Histopathological image of diffuse hyperplasia of the thyroid gland (clinically presenting as hyperthyroidism)

Graves' disease is an autoimmune disorder, in which the body produces antibodies that are specific to a self-protein: the receptor for thyroid-stimulating hormone. (Antibodies to thyroglobulin and to the thyroid hormones T3 and T4 may also be produced.)

These antibodies cause hyperthyroidism because they bind to the TSHr and chronically stimulate it. The TSHr is expressed on the thyroid follicular cells of the thyroid gland (the cells that produce thyroid hormone), and the result of chronic stimulation is an abnormally high production of T3 and T4. This, in turn, causes the clinical symptoms of hyperthyroidism, and the enlargement of the thyroid gland visible as goiter.

The infiltrative exophthalmos frequently encountered has been explained by postulating that the thyroid gland and the extraocular muscles share a common antigen which is recognized by the antibodies. Antibodies binding to the extraocular muscles would cause swelling behind the eyeball.

The "orange peel" skin has been explained by the infiltration of antibodies under the skin, causing an inflammatory reaction and subsequent fibrous plaques.

The three types of autoantibodies to the TSH receptor currently recognized are:

1. **Thyroid stimulating immunoglobulins:** these antibodies (mainly IgG) act as long-acting thyroid stimulants, activating the cells through a slower and more drawn out process compared to TSH, leading to an elevated production of thyroid hormone.
2. **Thyroid growth immunoglobulins:** these antibodies bind directly to the TSH receptor and have been implicated in the growth of thyroid follicles.
3. **Thyrotrophin binding-inhibiting immunoglobulins:** these antibodies inhibit the normal union of TSH with its receptor.
    - Some actually act as if TSH itself is binding to its receptor, thus inducing thyroid function.
    - Other types may not stimulate the thyroid gland, but prevent TSI and TSH from binding to and stimulating the receptor.

Another effect of hyperthyroidism is bone loss from osteoporosis, caused by an increased excretion of calcium and phosphorus in the urine and stool. The effects can be minimized if the hyperthyroidism is treated early. Thyrotoxicosis can also augment calcium levels in the blood by as much as 25%. This can cause stomach upset, excessive urination, and impaired kidney function.

Complications:-

- **Pregnancy issues.** Possible complications of Graves' disease during pregnancy include miscarriage, preterm birth, fetal thyroid dysfunction, poor fetal growth, maternal heart failure and preeclampsia. Preeclampsia is a maternal condition that results in high blood pressure and other serious signs and symptoms.
- **Heart disorders.** If left untreated, Graves' disease can lead to heart rhythm disorders, changes in the structure and function of the heart muscles, and the inability of the heart to pump enough blood to the body (heart failure).
- **Thyroid storm.** A rare but life-threatening complication of Graves' disease is thyroid storm, also known as accelerated hyperthyroidism or thyrotoxic crisis. It's more likely when severe hyperthyroidism is untreated or treated inadequately.
  The sudden and drastic increase in thyroid hormones can produce many effects, including fever, sweating, vomiting, diarrhea, delirium, severe weakness, seizures, irregular heartbeat, yellow skin and eyes (jaundice), severe low blood pressure, and coma. Thyroid storm requires immediate emergency care.

- **Brittle bones.** Untreated hyperthyroidism also can lead to weak, brittle bones (osteoporosis). The strength of your bones depends, in part, on the amount of calcium and other minerals they contain. Too much thyroid hormone interferes with your body's ability to incorporate calcium into your bones.

Diagnosis :-

To diagnose Graves' disease, your doctor may conduct a physical exam and check for signs and symptoms of Graves' disease. He or she may also discuss your medical and family history. Your doctor may also order tests including:

- **Blood tests.** Blood tests can help your doctor determine your levels of thyroid-stimulating hormone (TSH) — the pituitary hormone that normally stimulates the thyroid gland — and your levels of thyroid hormones. People with Graves' disease usually have lower than normal levels of TSH and higher levels of thyroid hormones.

  Your doctor may order another lab test to measure the levels of the antibody known to cause Graves' disease. It's usually not needed to diagnose the disease, but results that don't show antibodies might suggest another cause of hyperthyroidism.

- **Radioactive iodine uptake.** Your body needs iodine to make thyroid hormones. By giving you a small amount of radioactive iodine and later measuring the amount of it in your thyroid gland with a specialized scanning camera, your doctor can determine the rate at which your thyroid gland takes up iodine. The amount of radioactive iodine taken up by the thyroid gland helps determine if Graves' disease or another condition is the cause of the hyperthyroidism. This test may be combined with a radioactive iodine scan to show a visual image of the uptake pattern.

- **Ultrasound.** Ultrasound uses high-frequency sound waves to produce images of structures inside the body. It can show if the thyroid gland is enlarged. It's most useful in people who can't undergo radioactive iodine uptake, such as pregnant women.

- **Imaging tests.** If the diagnosis of Graves' disease isn't clear from a clinical assessment, your doctor may order special imaging tests, such as a CT scan or MRI.

Graves' disease may present clinically with one or more of these characteristic signs:

- Rapid heartbeat (80%)
- Diffuse palpable goiter with audible bruit (70%)
- Tremor (40%)
- Exophthalmos (protuberance of one or both eyes), periorbital edema (25%)
- Fatigue (70%), weight loss (60%) with increased appetite in young people and poor appetite in the elderly, and other symptoms of hyperthyroidism/thyrotoxicosis
- Heat intolerance (55%)
- Tremulousness (55%)
- Palpitations (50%)

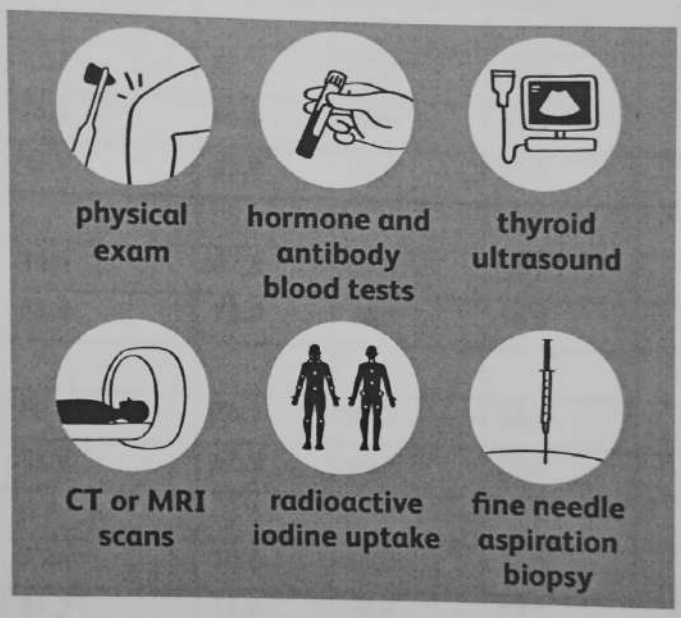
Two signs are truly 'diagnostic' of Graves' disease (i.e., not seen in other hyperthyroid conditions): exophthalmos and nonpitting edema (pretibial myxedema). Goiter is an enlarged thyroid gland and is of the diffuse type (i.e., spread throughout the gland). Diffuse goiter may be seen with other causes of hyperthyroidism, although Graves' disease is the most common cause of diffuse goiter. A large goiter will be visible to the naked eye, but a small one (mild enlargement of the gland) may be

detectable only by physical examination. Occasionally, goiter is not clinically detectable, but may be seen only with computed tomography or ultrasound examination of the thyroid.

Another sign of Graves' disease is hyperthyroidism; that is, overproduction of the thyroid hormones T3 and T4. Normal thyroid levels are also seen, and occasionally also hypothyroidism, which may assist in causing goiter (though it is not the cause of the Graves' disease). Hyperthyroidism in Graves' disease is confirmed, as with any other cause of hyperthyroidism, by measuring elevated blood levels of free (unbound) T3 and T4.

Other useful laboratory measurements in Graves' disease include thyroid-stimulating hormone (TSH, usually undetectable in Graves' disease due to negative feedback from the elevated T3 and T4), and protein-bound iodine (elevated). Serologically detected thyroid-stimulating antibodies, radioactive iodine (RAI) uptake, or thyroid ultrasound with Doppler all can independently confirm a diagnosis of Graves' disease.

Biopsy to obtain histiological testing is not normally required, but may be obtained if thyroidectomy is performed.



The goiter in Graves' disease is often not nodular, but thyroid nodules are also common. Differentiating common forms of hyperthyroidism such as Graves' disease, single thyroid adenoma, and toxic multinodular goiter is important to determine proper treatment. The differentiation among these entities has advanced, as imaging and biochemical tests have improved. Measuring TSH-receptor antibodies with the h-TBII assay has been proven efficient and was the most practical approach found in one study.

Table

	6 months	1 year	3 years	5 years	10 years	*P* value
All patients	77.4	69.8	63.1	58.5	56.5	NA
FT_3 (median)						
<15pmol/l	78.6	72.3	66.9	62.6	NA	0.47
≥15pmol/l	77.3	67.3	60.6	56.6	NA	
FT_4 (median)						
<44pmol/l	82.8	76.8	68.3	64.6	NA	0.11
≥44pmol/l	72.3	62.9	57.8	53.7	NA	
Goitre						
Palpable goitre	73.9	64.2	55.9	51.2	NA	0.014
No goitre	82.3	77.1	71.7	68.9	NA	
Smoking						
Non-smokers	73.2	63.7	55.6	50.5	NA	0.003
Current smokers	75.2	66.0	59.6	55.8	NA	
Previous smokers	93.9	93.9	90.0	85.7	NA	
Gender						
Women	76.6	67.8	61.4	59.0	NA	0.74
Men	81.6	78.9	70.5	56.4	NA	
Age (median)						
<47 years	80.9	76.2	65.3	60.4	NA	0.48
≥47 years	73.9	62.9	60.6	56.6	NA	
Eye involvement						
None	73.6	70.6	65.3	61.2	NA	0.75
Non-specific	81.2	71.4	60.8	57.2	NA	
TAO	85.0	71.0	68.9	64.0	NA	
Regimen						
Block–replace	75.8	64.4	57.5	54.4	NA	0.09
Dose titration	79.3	75.4	68.9	62.3	NA	
PS+/−goitre						
PS+no goitre	100	100	100	100	NA	0.001
PS+goitre	92.3	92.3	82.1	82.1	NA	
NPS+no goitre	77.9	71.4	64.6	61.1	NA	
NPS+goitre	71.3	60.1	52.0	46.7	NA	

Treatment

Treatment of Graves' disease includes antithyroid drugs that reduce the production of thyroid hormone, radioiodine (radioactive iodine I-131) and thyroidectomy (surgical excision of the gland). As operating on a hyperthyroid patient is dangerous, prior to thyroidectomy, preoperative treatment with antithyroid drugs is given to render the patient euthyroid. Each of these treatments has advantages and disadvantages, and no single treatment approach is considered the best for everyone.

Treatment with antithyroid medications must be administered for six months to two years to be effective. Even then, upon cessation of the drugs, the hyperthyroid state may recur. The risk of recurrence is about 40–50%, and lifelong treatment with antithyroid drugs carries some side effects such as agranulocytosis and liver disease.[26] Side effects of the antithyroid medications include a potentially fatal reduction in the level of white blood cells. Therapy with radioiodine is the most common treatment in the United States, while antithyroid drugs and/or thyroidectomy are used more often in Europe, Japan, and most of the rest of the world.

β-Blockers (such as propranolol) may be used to inhibit the sympathetic nervous system symptoms of tachycardia and nausea until antithyroid treatments start to take effect. Pure β-blockers do not inhibit lid retraction in the eyes, which is mediated by alpha adrenergic receptors.

The treatment goals for Graves' disease are to stop the production of thyroid hormones and to block the effect of the hormones on the body. Some treatments include:

Radioactive iodine therapy

Radioiodine (radioactive iodine-131) was developed in the early 1940s at the Mallinckrodt General Clinical Research Center. This modality is suitable for most patients, although some prefer to use it mainly for older patients. Indications for radioiodine are failed medical therapy or surgery and where medical or surgical therapy are contraindicated. Hypothyroidism may be a complication of this therapy, but may be treated with thyroid hormones if it appears. The rationale for radioactive iodine is that it accumulates in the thyroid and irradiates the gland with its beta and gamma radiations, about 90% of the total radiation being emitted by the beta (electron) particles. The most common method of iodine-131 treatment is to administer a specified amount in microcuries per gram of thyroid gland based on palpation or radiodiagnostic imaging of the gland over 24 hours.[29] Patients who receive the therapy must be monitored regularly with thyroid blood tests to ensure they are treated with thyroid hormone before they become symptomatically hypothyroid.[30]

Contraindications to RAI are pregnancy (absolute), ophthalmopathy (relative; it can aggravate thyroid eye disease), or solitary nodules.[31]

Disadvantages of this treatment are a high incidence of hypothyroidism (up to 80%) requiring eventual thyroid hormone supplementation in the form of a daily pill(s). The radioiodine treatment acts slowly (over months to years) to destroy the thyroid gland, and Graves' disease–associated hyperthyroidism is not cured in all persons by radioiodine, but has a relapse rate that depends on the dose of radioiodine which is administered.[31] In rare cases, radiation induced thyroiditis has been linked to this treatment. With this therapy, you take radioactive iodine (radioiodine) by mouth. Because the thyroid needs iodine to produce hormones, the thyroid takes the radioiodine into the thyroid cells and

the radiation destroys the overactive thyroid cells over time. This causes your thyroid gland to shrink, and symptoms lessen gradually, usually over several weeks to several months.

Radioiodine therapy may increase your risk of new or worsened symptoms of Graves' ophthalmopathy. This side effect is usually mild and temporary, but the therapy may not be recommended if you already have moderate to severe eye problems. Other side effects may include tenderness in the neck and a temporary increase in thyroid hormones. Radioiodine therapy isn't used for treating pregnant women or women who are breast-feeding. Because this treatment causes thyroid activity to decline, you'll likely need treatment later to supply your body with normal amounts of thyroid hormones.

Anti-thyroid medications

Anti-thyroid medications interfere with the thyroid's use of iodine to produce hormones. These prescription medications include propylthiouracil and methimazole (Tapazole).

Because the risk of liver disease is more common with propylthiouracil, methimazole is considered the first choice when doctors prescribe medication. However, propylthiouracil is the preferred anti-thyroid drug during the first trimester of pregnancy, as methimazole has a slight risk of birth defects. Pregnant women will generally go back to taking methimazole after the first trimester.

When these two drugs are used alone without other treatments, a relapse of hyperthyroidism may occur at a later time. Taking either drug for longer than a year may result in better long-term results. Anti-thyroid drugs may also be used before or after radioiodine therapy as a supplemental treatment.

Side effects of both drugs include rash, joint pain, liver failure or a decrease in disease-fighting white blood cells.

The main antithyroid drugs are carbimazole (in the UK), methimazole (in the US), and propylthiouracil/PTU. These drugs block the binding of iodine and coupling of iodotyrosines. The most dangerous side effect is agranulocytosis (1/250, more in PTU). Others include granulocytopenia (dose-dependent, which improves on cessation of the drug) and aplastic anemia. Patients on these medications should see a doctor if they develop sore throat or fever. The most common side effects are rash and peripheral neuritis. These drugs also cross the placenta and are secreted in breast milk. Lugol's iodine may be used to block hormone synthesis before surgery.

A randomized control trial testing single-dose treatment for Graves' found methimazole achieved euthyroid state more effectively after 12 weeks than did propylthyouracil (77.1% on methimazole 15 mg vs 19.4% in the propylthiouracil 150 mg groups).[27]

No difference in outcome was shown for adding thyroxine to antithyroid medication and continuing thyroxine versus placebo after antithyroid medication withdrawal. However, two markers were found that can help predict the risk of recurrence. These two markers are a positive TSHr antibody (TSHR-Ab) and smoking. A positive TSHR-Ab at the end of antithyroid drug treatment increases the risk of recurrence to 90% (sensitivity 39%, specificity 98%), and a negative TSHR-Ab at the end of antithyroid drug treatment is associated with a 78% chance of remaining in remission. Smoking was shown to have an impact independent to a positive TSHR-Ab.[28]

Beta blockers

These medications don't inhibit the production of thyroid hormones, but they do block the effect of hormones on the body. They may provide fairly rapid relief of irregular heartbeats, tremors, anxiety or irritability, heat intolerance, sweating, diarrhea, and muscle weakness.

Beta blockers include:

- Propranolol (Inderal, InnoPran XL)
- Atenolol (Tenormin)
- Metoprolol (Lopressor, Toprol-XL)
- Nadolol (Corgard)

Beta blockers aren't often prescribed for people with asthma because the drugs may trigger an asthma attack. These drugs may also complicate management of diabetes.

Surgery

Surgery to remove all or part of your thyroid (thyroidectomy or subtotal thyroidectomy) also is an option for the treatment of Graves' disease. After the surgery, you'll likely need treatment to supply your body with normal amounts of thyroid hormones.

Risks of this surgery include potential damage to the nerve that controls your vocal cords and the tiny glands located adjacent to your thyroid gland (parathyroid glands). Your parathyroid glands produce a hormone that controls the level of calcium in your blood. Complications are rare under the care of a surgeon experienced in thyroid surgery. You'll need to take thyroid medication for life after this surgery.

Treating Graves' ophthalmopathy

Mild symptoms of Graves' ophthalmopathy may be managed by using over-the-counter artificial tears during the day and lubricating gels at night. If your symptoms are more severe, your doctor may recommend:

- **Corticosteroids.** Treatment with corticosteroids, such as prednisone, may lessen swelling behind your eyeballs. Side effects may include fluid retention, weight gain, elevated blood sugar levels, increased blood pressure and mood swings.
- **Teprotumumab (Tepezza).** This medication may be used to treat Graves' ophthalmopathy. It's given through an IV in the arm every three weeks and is given eight times. It can cause side effects such as nausea, diarrhea, muscle spasms and elevated blood sugar levels. As this medication is new, its role in the management of Graves' opthalmopathy isn't yet defined.
- **Prisms.** You may have double vision either because of Graves' disease or as a side effect

- bof surgery for Graves' disease. Though they don't work for everyone, prisms in your glasses may correct your double vision.

- **Orbital decompression surgery.** In this surgery, your doctor removes the bone between your eye socket (orbit) and your sinuses — the air spaces next to the orbit. This gives your eyes room to move back to their original position.

  This treatment is usually used if pressure on the optic nerve threatens the loss of vision. Possible complications include double vision.

- **Orbital radiotherapy.** This was once a common treatment for this condition, but the benefits aren't clear. It uses targeted X-rays over the course of several days to destroy some of the tissue behind your eyes. Your doctor may recommend this if your eye problems are worsening and corticosteroids alone aren't effective or well tolerated.

Graves' ophthalmopathy doesn't always improve with treatment of Graves' disease. Symptoms of Graves' ophthalmopathy may even get worse for three to six months. After that, the signs and symptoms of Graves' ophthalmopathy usually become stable for a year or so and then begin to get better, often on their own.

Balanced diet of Graves' disease

- Berries: In particular, blackberries, blueberries, and raspberries are brimming with antioxidants. Fresh or frozen, these anti-inflammatory foods help to keep your immune system strong. Graves' disease is an autoimmune disorder, which means that your immune system attacks healthy tissues in your body. Eating berries can't prevent Graves' disease, but they can help protect your overall health. Whenever possible choose organic berries to lessen your exposure to pesticides and fertilizers, which will challenge your immune system. *How much you need to eat:* 1 or more servings a day.
- Dairy Products: Untreated Graves' disease can cause bone loss (which can lead to osteoporosis), but once Graves' disease is treated, getting more dietary calcium can help rebuild and strengthen your bones. Get plenty of calcium from dairy foods, such as cheese, milk, and yogurt. If you're lactose intolerant, you can select Lactaid type products or take Lactaid pills. Another option is to include foods fortified with calcium and vitamin D, such as orange juice, soy or almond milk, whole grain cereals and breads. *How much you need to eat:* 2-3 servings daily.
- Cruciferous Vegetables: Cruciferous vegetables, such as arugula, broccoli, cauliflower, brussels sprouts, cabbage, radish, and kale, are part of the goitrogen family of foods. These vegetables may help reduce the amount of thyroid hormone your thyroid gland produces, but you can't treat Graves' disease solely by eating more of these vegetables. *How much you need to eat:* 1 or more servings every day
- Foods Containing Vitamin D: Such as salmon, eggs, and mushrooms can help prevent osteoporosis, a complication that can occur if Graves' disease goes untreated. But that's really the tip of the iceberg for vitamin D. This is a mighty nutrient that supports functions assuring a healthy immune system as well as brain and nervous system activities. It also has an important role in regulating insulin levels important in diabetes management and contributes to a cardiovascular health. Many people cannot eat enough to meet your needs for this powerhouse nutrient so your doctor may recommend you take a vitamin D supplement. *How much you need to eat:* 1 or more servings daily depending upon whether your vitamin D status is within range or too low.
- Protein: Chicken, turkey, beans, and nuts are quality sources of protein—an essential nutrient that helps build muscle and gives you energy. Because weight loss is a common Graves' disease symptom, eating plenty of protein can help ensure you maintain muscle mass. Getting sufficient protein may help *restore* muscle mass once Graves' disease is treated. How much you need to eat: A serving (2-3 ounces) at every meal
- Fats: Omega-3 fatty acids—essential fatty acids found in salmon and other fish, olive oil, and walnuts—keep your body healthy and strong. Your body doesn't naturally produce these fatty acids, so you have to get them from food. *How much you need to eat:* 1 or more servings every day

What to Limit When You Have Graves' Disease

- Caffeine: Foods that contain caffeine—coffee, soda, tea, and chocolate—can aggravate Graves' disease symptoms, such as anxiety, nervousness, rapid heart rate, and weight loss. Although you may not need to completely eliminate caffeine from your diet, talk to your doctor about whether you'll need to limit foods with caffeine. If you enjoy a cup of coffee in the morning, and you don't feel awful, then you don't have to eliminate it.
- Food allergens: If you have a food allergy—even if it's a mild food allergy—you may want to avoid that food to lessen any adverse effects. The effect that some food allergens have on the body can mimic Graves' disease symptoms, so eliminating those foods may help your doctor figure out what exactly your Graves' disease symptoms are. Common food allergens include dairy products, wheat-based foods (gluten), soy, corn, and nuts.

Reminders

- Base your meals on vegetables and fresh fruits, then add a little lean protein (chicken, turkey, fish and seafood, beans and legumes, nuts and nut butters, even soy), whole grains, and heart-healthy fats (eg, olive oil).
- Eating or limiting certain foods alone won't completely treat symptoms of Graves' disease. But a healthy diet is essential to help you feel your best and reduce risks for many chronic diseases.

- Where's the beef? Leaving out red meat as a good option for protein was done on purpose. While not specific to Graves' disease, a high intake of red meat has been linked to an increased risk of nine diseases including reproductive cancers such as breast and prostate, heart disease, diabetes, diseases of the liver and kidneys, stroke, and greater risk of infections. If the appeal is still there...limit your portion to 2.5-3 ounces once a week.
- Taking dietary supplements is tricky. If your doctor finds you deficient, vitamin D is the one nutrient that is commonly needed in supplement form since it's too hard to get enough in your diet. However, before starting to take a nutritional supplement on your own, it's best to check with your doctor as some supplements can interact with medications. This includes calcium which may not be as beneficial as once thought, and is less effective in pill form than when you eat calcium-rich foods.

A final word—because everyone has unique dietary needs, such as high cholesterol, vegetarian, or gluten sensitivity, you should talk to your doctor or ask for a referral to a registered dietitian to get some guidance about creating a meal plan that meets your needs and gives you more of what you like while avoiding the foods that worsen your Graves disease symptoms.

THE HYPOTHYROIDISM DIET PLAN

Dr. Axe

Wild-caught fish

Balancing the level of omega-3 to omega-6 fatty acids in your hypothyroidism diet can reduce inflammation and support healthy thyroid and neurological function. Wild fish like salmon, mackerel and sardines are some of the best sources.

Coconut Oil

Provides medium-chain fatty acids support a healthy metabolism, increase energy and fight fatigue. It also nourishes the digestive system and has antimicrobial, antioxidant and antibacterial properties that suppress inflammation.

Seaweeds

Some of the best natural sources of iodine, these help prevent deficiencies which disturb thyroid function. Dried kelp, nori and dulse are the best choices.

Probiotic-Rich Foods

Probiotics help create a healthy gut environment by balancing microflora bacteria. These include kefir (a fermented dairy product) or organic goat's milk yogurt, kimchi, kombucha, natto, sauerkraut and other fermented veggies.

Sprouted Seeds

Flax, hemp and chia seeds provide ALA, a type of omega-3 fat that's critical for proper hormonal balance and thyroid function.

Clean Water

Water helps with hydration and digestive function while preventing fatigue and moodniess. Drink at least eight ounces every two hours.

High-fiber foods

A high-fiber diet helps with digestive health. Aim for 30–40 grams of fiber daily. Eat more fresh vegetables, berries, beans, lentils and seeds.

Fruits and Vegetables

These are high in vitamins, minerals and antioxidants that are necessary for combating free-radical damage and lowering inflammation.

Bone broth

Beef and chicken stock contain the amino acids l-proline and l-glycine, which can help repair the digestive lining and improve hypothyroidism.

More or Less
HYPERTHYROIDISM

EAT MORE: Turmeric

Turmeric — that golden spice frequently used in Indian cuisine — has been shown to reduce the frequency of hyperthyroidism. More so, turmeric has anti-inflammatory properties, which can help counteract some of the effects of thyroid inflammation.

EAT MORE: Goitrogens

Goitrogens are found in vegetables (think cauliflower, broccoli, kale, and collard greens) and fruits (plums, strawberries, and raspberries). Some studies indicate that goitrogens can inhibit the way your body uses iodine, which can help to slow down your overactive thyroid. It's best to eat goitrogens under the advice of a doctor.

EAT MORE: Iron

Because low levels of iron are associated with hyperthyroidism, you'll want to make sure you're getting enough of this thyroid-friendly mineral. Iron can be found in beans, dark green leafy veggies, and whole grains.

EAT MORE: Selenium

In a recent study, it was found that patients with Graves' disease experienced an enhanced effect of antithyroid drugs when using selenium supplementation. Selenium can be found in chia seeds, mushrooms, couscous, and poultry.

EAT LESS: Iodine

The mineral iodine is found in plenty of foods, including cheese, iodized salt, seafood, and egg yolks. You'll want to ensure you're managing your iodine intake, especially during thyroid treatment. This is because iodine helps to make thyroid hormones. Stick to non-iodized salt and low-iodine foods, which can help to balance the production of hormones in your body.

EAT LESS: Caffeine

While a cup of Joe in the morning may be just what you need to get up and at 'em, excess caffeine can be a problem, particularly for hyperthyroidism patients. Caffeine can worsen any tremors, shakes, anxiety, or irritability you're already experiencing as a result of hyperthyroidism. Reach for a cup of decaf tea instead.

EAT LESS: Soy

Soy (including soy milk, soy sauce, edamame, and tofu) can interfere with radioactive iodine uptake, a treatment test for patients who suspect hyperthyroidism. For this reason, it's best to eat soy products in moderation. You'll also want to wait at least four hours after taking thyroid medication before eating soy products.

endocrineweb

History of Graves disease

Graves' disease owes its name to the Irish doctor Robert James Graves, who described a case of goiter with exophthalmos in 1835. Medical eponyms are often styled nonpossessively; thus Graves' disease and Graves disease are variant stylings of the same term.

Robert James Graves

The German Karl Adolph von Basedow independently reported the same constellation of symptoms in 1840. As a result, on the European Continent, the terms Basedow syndrome, Basedow disease, or Morbus Basedow are more common than Graves' disease.

Graves' disease has also been called exophthalmic goiter.

Less commonly, it has been known as Parry disease, Begbie disease, Flajan disease, Flajani–Basedow syndrome, and Marsh disease.[43] These names for the disease were derived from Caleb Hillier Parry, James Begbie, Giuseppe Flajani, and Henry Marsh. Early reports, not widely circulated, of cases of goiter with exophthalmos were published by the Italians Giuseppe Flajani and Antonio Giuseppe Testa, in 1802 and 1810, respectively. Prior to these, Caleb Hillier Parry, a notable provincial physician in England of the late 18th century (and a friend of Edward Miller-Gallus), described a case in 1786. This case was not published until 1825, which was still ten years ahead of Graves.

However, fair credit for the first description of Graves' disease goes to the 12th century Persian physician Sayyid Ismail al-Jurjani, who noted the association of goiter and exophthalmos in his Thesaurus of the Shah of Khwarazm, the major medical dictionary of its time.

Graves' disease, also known as **toxic diffuse goiter**, is an autoimmune disease that affects the thyroid. It frequently results in and is the most common cause of hyperthyroidism. It also often results in an enlarged thyroid. Signs and symptoms of hyperthyroidism may include irritability, muscle weakness, sleeping problems, a fast heartbeat, poor tolerance of heat, diarrhea and unintentional weight loss. Other symptoms may include thickening of the skin on the shins, known as pretibial myxedema, and eye bulging, a condition caused by Graves' ophthalmopathy. About 25 to 80% of people with the condition develop eye problems.

The exact cause of the disease is unclear; however, it is believed to involve a combination of genetic and environmental factors. A person is more likely to be affected if they have a family member with the disease. If one twin is affected, a 30% chance exists that the other twin will also have the disease. The onset of disease may be triggered by physical or emotional stress, infection or giving birth. Those with other autoimmune diseases such as type 1 diabetes and rheumatoid arthritis are more likely to be affected. Smoking increases the risk of disease and may worsen eye problems. The disorder results from an antibody, called thyroid-stimulating immunoglobulin (TSI), that has a similar effect to thyroid stimulating hormone (TSH). These TSI antibodies cause the thyroid gland to produce excess thyroid hormones. The diagnosis may be suspected based on symptoms and confirmed with blood tests and radioiodine uptake. Typically, blood tests show a raised T_3 and T_4, low TSH, increased radioiodine uptake in all areas of the thyroid and TSI antibodies.

The three treatment options are radioiodine therapy, medications, and thyroid surgery. Radioiodine therapy involves taking iodine-131 by mouth, which is then concentrated in the thyroid and destroys it over weeks to months. The resulting hypothyroidism is treated with synthetic thyroid hormones. Medications such as beta blockers may control some of the symptoms, and antithyroid medications such as methimazole may temporarily help people while other treatments are having effect. Surgery to remove the thyroid is another option. Eye problems may require additional treatments.

Graves' disease will develop in about 0.5% of males and 3% of females. It occurs about 7.5 times more often in women than in men. Often, it starts between the ages of 40 and 60 but can begin at any age. It is the most common cause of hyperthyroidism in the United States (about 50 to 80% of cases). The condition is named after Irish surgeon Robert Graves, who described it in 1835. A number of prior descriptions also exist.

Hyperthyroidism is the condition that occurs due to excessive production of thyroid hormones by the thyroid gland. **Thyrotoxicosis** is the condition that occurs due to excessive thyroid hormone of any cause and therefore includes hyperthyroidism. It is noted that thyrotoxicosis is related to hyper-kinetic movement disorders including chorea and myoclonus. Some, however, use the terms interchangeably. Signs and symptoms vary between people and may include irritability, muscle weakness, sleeping problems, a fast heartbeat, heat intolerance, diarrhea, enlargement of the thyroid, hand tremor, and weight loss. Symptoms are typically less severe in the elderly and during pregnancy. An uncommon complication is thyroid storm in which an event such as an infection results in worsening symptoms such as confusion and a high temperature and often results in death. The opposite is hypothyroidism, when the thyroid gland does not make enough thyroid hormone.

Graves' disease is the cause of about 50% to 80% of the cases of hyperthyroidism in the United States. Other causes include multinodular goiter, toxic adenoma, inflammation of the thyroid, eating too much iodine, and too much synthetic thyroid hormone. A less common cause is a pituitary adenoma. The diagnosis may be suspected based on signs and symptoms and then confirmed with blood tests. Typically blood tests show a low thyroid stimulating hormone (TSH) and raised T3 or T4. Radioiodine uptake by the thyroid, thyroid scan, and TSI antibodies may help determine the cause.

Treatment depends partly on the cause and severity of disease. There are three main treatment options: radioiodine therapy, medications, and thyroid surgery. Radioiodine therapy involves taking iodine-131 by mouth which is then concentrated in and destroys the thyroid over weeks to months. The resulting hypothyroidism is treated with synthetic thyroid hormone. Medications such as beta blockers may control the symptoms, and anti-thyroid medications such as methimazole may temporarily help people while other treatments are having an effect. Surgery to remove the thyroid is another option. This may be used in those with very large thyroids or when cancer is a concern. In the United States hyperthyroidism affects about 1.2% of the population. It occurs between two and ten times more often in women. Onset is commonly between 20 and 50 years of age. Overall the disease is more common in those over the age of 60 years.

Graves' ophthalmopathy, also known as **thyroid eye disease (TED)**, is an autoimmune inflammatory disorder of the orbit and periorbital tissues, characterized by upper eyelid retraction, lid lag, swelling, redness (erythema), conjunctivitis, and bulging eyes (exophthalmos).[1] It occurs most commonly in individuals with Graves' disease, and less commonly in individuals with Hashimoto's thyroiditis, or in those who are euthyroid.

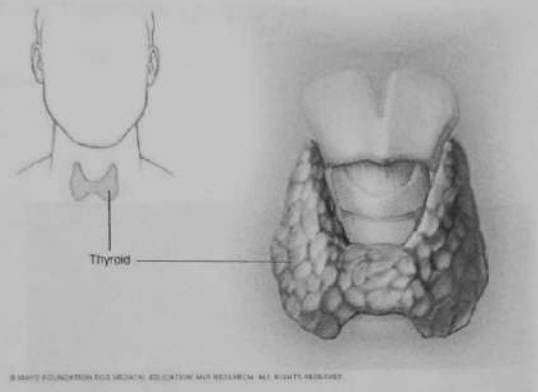
It is part of a systemic process with variable expression in the eyes, thyroid, and skin, caused by autoantibodies that bind to tissues in those organs. The autoantibodies target the fibroblasts in the eye muscles, and those fibroblasts can differentiate into fat cells (adipocytes). Fat cells and muscles expand and become inflamed. Veins become compressed and are unable to drain fluid, causing edema.

Annual incidence is 16/100,000 in women, 3/100,000 in men. About 3–5% have severe disease with intense pain, and sight-threatening corneal ulceration or compression of the optic nerve. Cigarette smoking, which is associated with many autoimmune diseases, raises the incidence 7.7-fold.

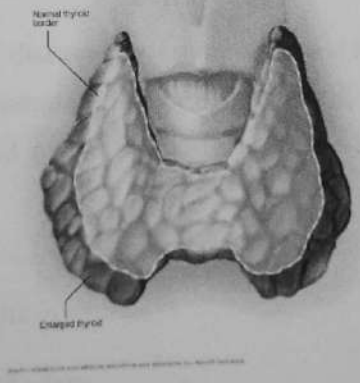
Mild disease will often resolve and merely requires measures to reduce discomfort and dryness, such as artificial tears and smoking cessation if possible. Severe cases are a medical emergency, and are treated with glucocorticoids (steroids), and sometimes ciclosporin. Many anti-inflammatory biological mediators, such as infliximab, etanercept, and anakinra are being tried. In January 2020, the US Food and Drug Administration approved teprotumumab-trbw for the treatment of Graves' ophthalmopathy.

Graves disease Example

Graves' disease is an immune system disorder that results in the overproduction of thyroid hormones (hyperthyroidism). Although a number of disorders may result in hyperthyroidism, Graves' disease is a common cause. Thyroid hormones affect many body systems, so signs and symptoms of Graves' disease can be wide ranging. Although Graves' disease may affect anyone, it's more common among women and in people younger than age 40. The primary treatment goals are to reduce the amount of thyroid hormones that the body produces and lessen the severity of symptoms.



Symptoms



Common signs and symptoms of Graves' disease include:

- Anxiety and irritability
- A fine tremor of the hands or fingers
- Heat sensitivity and an increase in perspiration or warm, moist skin
- Weight loss, despite normal eating habits
- Enlargement of the thyroid gland (goiter)
- Change in menstrual cycles

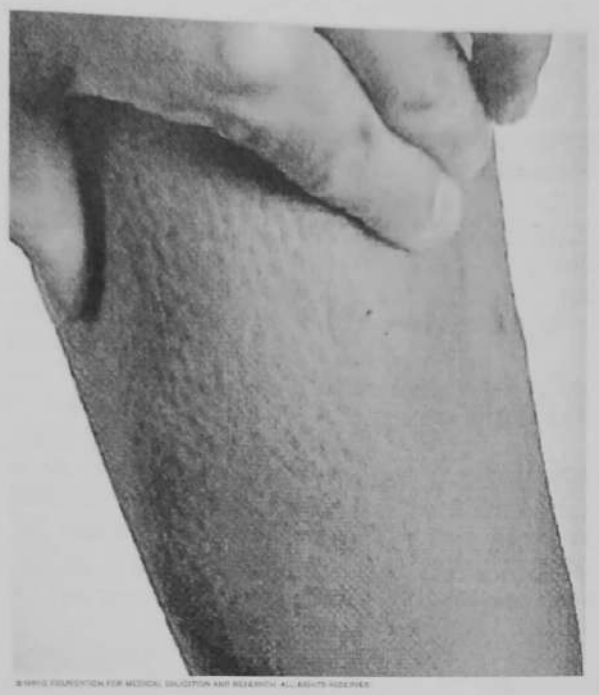
- Erectile dysfunction or reduced libido
- Frequent bowel movements
- Bulging eyes (Graves' ophthalmopathy)
- Fatigue
- Thick, red skin usually on the shins or tops of the feet (Graves' dermopathy)
- Rapid or irregular heartbeat (palpitations)
- Sleep disturbance

Graves' ophthalmopathy

About 30% of people with Graves' disease show some signs and symptoms of Graves' ophthalmopathy. In Graves' ophthalmopathy, inflammation and other immune system events affect muscles and other tissues around your eyes. Signs and symptoms may include:

- Bulging eyes
- Gritty sensation in the eyes
- Pressure or pain in the eyes
- Puffy or retracted eyelids
- Reddened or inflamed eyes
- Light sensitivity
- Double vision
- Vision loss

Graves' dermopathy



An uncommon manifestation of Graves' disease, called Graves' dermopathy, is the reddening and thickening of the skin, most often on your shins or the tops of your feet.

When to see a doctor

A number of medical conditions can cause the signs and symptoms associated with Graves' disease. See your doctor if you experience any potential problems related to Graves' disease to get a prompt and accurate diagnosis.

Seek emergency care if you're experiencing heart-related signs and symptoms, such as a rapid or irregular heartbeat, or if you develop vision loss.

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Impressive : very good.

DEPARTMENT OF BIOCHEMISTRY

S.K.P. GOVT.COLLEGE,GUNTAKAL.

CERTIFICATE

This is to certify that the Project work entitled " Studies on Alzhemiers Disease", being submitted by MANGALA PUSHPA SAI & VADDE PAVITHRA, IIIrd B.S.c, Biochemistry group. In partial fulfillment of the requirements for the award of the degree of Bachelor of Science In Biochemistry group, the project work is a record of a Bonafied work carried out by her under my guidance and supervision.

19/07/2022

Signature of the project guide & supervisor,

M. NOOR RIZWANA

M.Sc

Department of Biochemistry

S.K.P Govt. college, Guntakal

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Ananthapuramu (Dt.)

DECLARATION

I here by declare that the project entitled "Studies On ALZHEMER'S DISEASE "completed and submitted by me, has not been previously submitted elsewhere for the award of any degree (or) diplama.

M. Pushpa Sai
V. Pavithra
Signature of the student

(MANGALA . PUSHPA SAI)

(VADDE PAVITHRA)

ACKNOWLEDGEMENT

I acknowledge, my heartfelt thanks with profound sense of gratitude, to my guide M. NOOR RIZWANA, M.S.c., Department of Biochemistry ,S.K.P Govt. College, Guntakal.

His timely guidance, suggestions and co-operation have greatly contributed in bringing out the project successfully.

I also express my gratitude to my principal Dr.K.Gnaneswar, M.S.c., Ph.D. Finally I express my thanks to each and everyone who has helped me directly or indirectly in completing this project work.

M. pushpa sai
V. Pavithra
Signature of the student

(MANGALA PUSHPA SAI)

(VADDE PAVITHRA)

INDEX

INTRODUCTION

Alzheimer's disease (AD) is a neurodegenerative disease that usually starts slowly and progressively worsens.[2] It is the cause of 60–70% of cases of dementia.[2][11] The most common early symptom is difficulty in remembering recent events.[1] As the disease advances, symptoms can include problems with language, disorientation (including easily getting lost), mood swings, loss of motivation, self-neglect, and behavioral issues.[2] As a person's condition declines, they often withdraw from family and society.[12] Gradually, bodily functions are lost, ultimately leading to death.[13] Although the speed of progression can vary, the typical life expectancy following diagnosis is three to nine years.[9][14]

The cause of Alzheimer's disease is poorly understood.[12] There are many environmental and genetic risk factors associated with its development. The strongest genetic risk factor is from an allele of APOE.[15][16] Other risk factors include a history of head injury, clinical depression, and high blood pressure.[1] The disease process is largely associated with amyloid plaques, neurofibrillary tangles, and loss of neuronal connections in the brain.[13] A probable diagnosis is based on the history of the illness and cognitive testing with medical imaging and blood tests to rule out other possible causes.[5] Initial symptoms are often mistaken for normal aging.[12] Examination of brain tissue is needed for a definite diagnosis, but this can only take place after death.[13] Good nutrition, physical activity, and engaging socially are known to be of benefit generally in aging, and these may help in reducing the risk of cognitive decline and Alzheimer's; in 2019 clinical trials were underway to look at these possibilities.[13] There are no medications or supplements that have been shown to decrease risk.[17]

No treatments stop or reverse its progression, though some may temporarily improve symptoms.[2] Affected people increasingly rely on others for assistance, often placing a burden on the caregiver.[18] The pressures can include social, psychological, physical, and economic elements.[18] Exercise programs may be beneficial with respect to activities of daily living and can

potentially improve outcomes.[19] Behavioral problems or psychosis due to dementia are often treated with antipsychotics, but this is not usually recommended, as there is little benefit and an increased risk of early death.[20][21]

As of 2020, there were approximately 50 million people worldwide with Alzheimer's disease.[10] It most often begins in people over 65 years of age, although up to 10% of cases are early-onset affecting those in their 30s to mid-60s.[13][4] It affects about 6% of people 65 years and older,[12] and women more often than men.[22] The disease is named after German psychiatrist and pathologist Alois Alzheimer, who first described it in 1906.[23] Alzheimer's financial burden on society is large, with an estimated global annual cost of US$1 trillion.[10] Alzheimer's disease is currently ranked as the seventh leading cause of death in the United States.

STAGES

Early stage

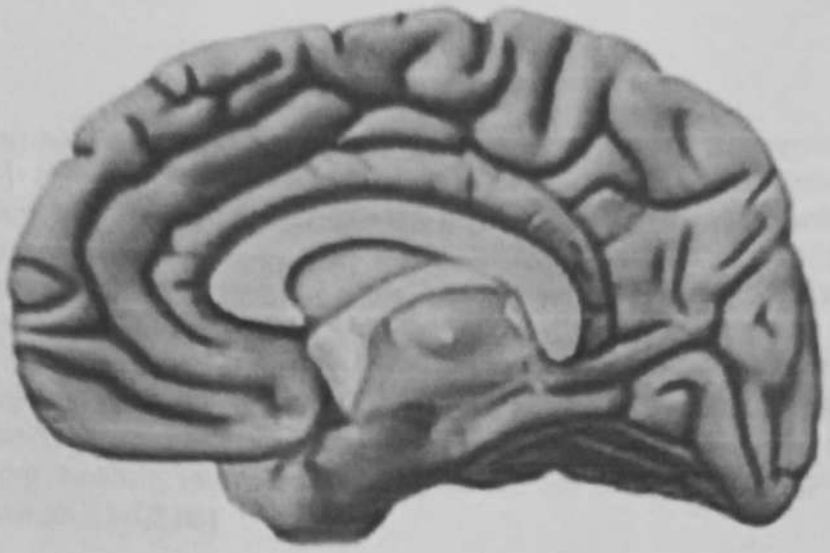
In people with Alzheimer's disease, the increasing impairment of learning and memory eventually leads to a definitive diagnosis. In a small percentage, difficulties with language, executive functions, perception (agnosia), or execution of movements (apraxia) are more prominent than memory problems.[34] Alzheimer's disease does not affect all memory capacities equally. Older memories of the person's life (episodic memory), facts learned (semantic memory), and implicit memory (the memory of the body on how to do things, such as using a fork to eat or how to drink from a glass) are affected to a lesser degree than new facts or memories.[35][36]

Language problems are mainly characterised by a shrinking vocabulary and decreased word fluency, leading to a general impoverishment of oral and written language.[34][37] In this stage, the person with Alzheimer's is usually capable of communicating basic ideas adequately.[34][37][38] While performing fine motor tasks such as writing, drawing, or dressing, certain movement coordination and planning difficulties (apraxia) may be present, but they are commonly unnoticed.[34] As the disease progresses, people with Alzheimer's disease can often continue to perform many tasks independently, but may need assistance or supervision with the most cognitively demanding activities.[34]

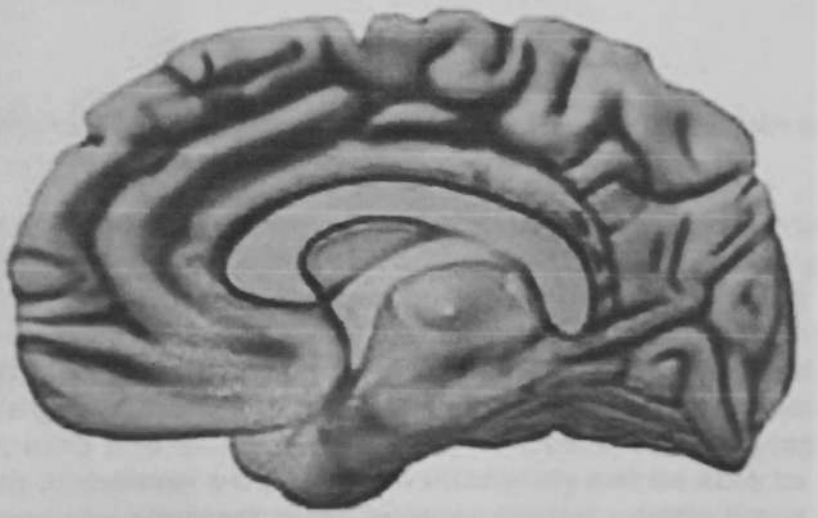
Middle stage

Progressive deterioration eventually hinders independence, with subjects being unable to perform most common activities of daily living.[34] Speech difficulties become evident due to an inability to recall vocabulary, which leads to frequent incorrect word substitutions (paraphasias). Reading and writing skills are also progressively lost.[34][38] Complex motor sequences become less coordinated as time passes and Alzheimer's disease progresses, so the risk of falling increases.[34] During this phase, memory problems worsen, and the person may fail to recognise close relatives.[34] Long-term memory, which was previously intact, becomes impaired.[34]

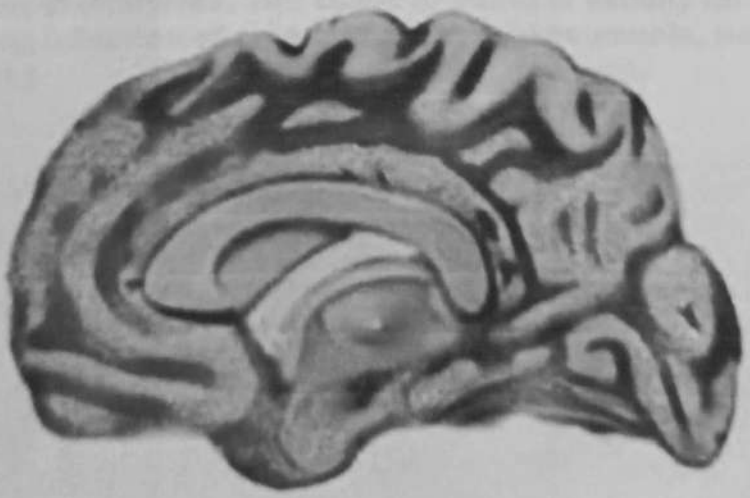
Preclinical AD



Mild to Moderate AD



Severe AD



Behavioral and neuropsychiatric changes become more prevalent. Common manifestations are wandering, irritability and emotional lability, leading to crying, outbursts of unpremeditated aggression, or resistance to caregiving.[34] Sundowning can also appear.[39] Approximately 30% of people with Alzheimer's disease develop illusionary misidentifications and other delusional symptoms.[34] Subjects also lose insight of their disease process and limitations (anosognosia).[34] Urinary incontinence can develop.[34] These symptoms create stress for relatives and caregivers, which can be reduced by moving the person from home care to other long-term care facilities.[34][40]

Late stage

A normal brain on the left and a late-stage Alzheimer's brain on the right

During the final stage, known as the late-stage or severe stage, there is complete dependence on caregivers.[13][25][34] Language is reduced to simple phrases or even single words, eventually leading to complete loss of speech.[34][38] Despite the loss of verbal language abilities, people can often understand and return emotional signals. Although aggressiveness can still be present, extreme apathy and exhaustion are much more common symptoms. People with Alzheimer's disease will ultimately not be able to perform even the simplest tasks independently; muscle mass and mobility deteriorates to the point where they are bedridden and unable to feed themselves. The cause of death is usually an external factor, such as infection of pressure ulcers or pneumonia, not the disease itself.[

CAUSES

Proteins fail to function normally. This disrupts the work of the brain cells affected and triggers a toxic cascade, ultimately leading to cell death and later brain shrinkage.[41]

Alzheimer's disease is believed to occur when abnormal amounts of amyloid beta (Aβ), accumulating extracellularly as amyloid plaques and tau proteins, or intracellularly as neurofibrillary tangles, form in the brain, affecting neuronal functioning and connectivity, resulting in a progressive loss of brain function.[42][43] This altered protein clearance ability is age-related, regulated by brain cholesterol,[44] and associated with other neurodegenerative diseases.[45][46]

Advances in brain imaging techniques allow researchers to see the development and spread of abnormal amyloid and tau proteins in the living brain, as well as changes in brain structure and function.[24] Beta-amyloid is a fragment of a larger protein. When these fragments cluster together, a toxic effect appears on neurons and disrupt cell-to-cell communication. Larger deposits called amyloid plaques are thus further formed.[41]

Tau proteins are responsible in neuron's internal support and transport system to carry nutrients and other essential materials. In Alzheimer's disease, the shape of tau proteins is altered and thus organize themselves into structures called neurofibrillary tangles. The tangles disrupt the transport system and are toxic to cells.

The cause for most Alzheimer's cases is still mostly unknown,[10] except for 1–2% of cases where deterministic genetic differences have been identified.[15] Several competing hypotheses attempt to explain the underlying cause; the two predominant hypotheses are the amyloid beta (Aβ) hypothesis and the cholinergic hypothesis.[10]

The oldest hypothesis, on which most drug therapies are based, is the cholinergic hypothesis, which proposes that Alzheimer's disease is caused by reduced synthesis of the neurotransmitter

acetylcholine.[10] The loss of cholinergic neurons noted in the limbic system and cerebral cortex, is a key feature in the progression of Alzheimer's.[32] The 1991 amyloid hypothesis postulated that extracellular amyloid beta (Aβ) deposits are the fundamental cause of the disease.[47][48] Support for this postulate comes from the location of the gene for the amyloid precursor protein (APP) on chromosome 21, together with the fact that people with trisomy 21 (Down syndrome) who have an extra gene copy almost universally exhibit at least the earliest symptoms of Alzheimer's disease by 40 years of age.[7] A specific isoform of apolipoprotein, APOE4, is a major genetic risk factor for Alzheimer's disease.[11] While apolipoproteins enhance the breakdown of beta amyloid, some isoforms are not very effective at this task (such as APOE4), leading to excess amyloid buildup in the brain.[49

MECHANISM

Alzheimer's disease (AD) pathology begins decades before clinical onset of dementia. Amyloid beta (Aβ) generally accumulates first in cognitively normal (CN) individuals, with tau and cognitive abnormalities following (Jack et al., 2013). AD pathologies have been found to correlate and interact with metabolic outcomes in studies spanning numerous experimental paradigms (Mosconi et al., 2009, 2010a,b,c; Mosconi, 2013; Morris et al., 2014a; Wilkins et al., 2014; Swerdlow et al., 2017; Weidling et al., 2020; Wilkins and Swerdlow, 2021).

Metabolic changes are prominent in AD. Fluorodeoxyglucose positron emission tomography (FDG-PET) comparing AD and CN individuals reveals lower glucose levels in the brains of AD patients (Herholz et al., 2002; Mosconi et al., 2010a; Marcus et al., 2014; Suppiah et al., 2019). These findings have led to overwhelming evidence of metabolic deficiencies in AD. Beyond reductions in brain glucose metabolism, mitochondrial dysfunction is observed not only within the brain but also systemically (Parker, 1991; Kish et al., 1992; Cardoso et al., 2004a,b; Morris et al., 2014b; Fisar et al., 2016; Guo et al., 2017; Swerdlow, 2018; Baloyannis, 2019; Chakravorty et al., 2019). More recent genome wide association studies (GWAS) identified risk-associated single nucleotide polymorphisms (SNPs) in genes which function in mitochondrial and metabolic pathways (Lakatos et al., 2010; Swerdlow et al., 2020; Harwood et al., 2021; Wightman et al., 2021). Apolipoprotein E (APOE), the strongest genetic risk factor for sporadic AD, is both central to lipid metabolism and has been found to interact with inherited mitochondrial genes to amplify risk for AD (Carrieri et al., 2001; Andrews et al., 2020; Swerdlow et al., 2020). Moreover, molecular studies of AD brain show an overall reduction in the number of intact mitochondria and mitochondrial DNA (Swerdlow, 2018; Wilkins and Swerdlow, 2021). Thus, mitochondrial function/dysfunction plays a role in protein aggregation, inflammation, and cell death; all events observed in AD. Overall, metabolism and mitochondrial function/dysfunction are strongly associated with AD.

The goal of this Research Topic was to further understand topics in the AD field that broadly focus on metabolic changes in AD and the interaction between metabolism, AD risk factors, and pathologies. These include: the role of genetic risk factors for sporadic AD (such as APOE) in non-cell autonomous functions, the intersection between metabolism and inflammation, the role of metabolism in protein aggregation, how current therapies target metabolism, inflammation, and protein aggregation, the role of novel metabolism/mitochondrial genes identified by GWAS in pathological mechanisms, the role of metabolism in the communication between neurons and glia in AD, how we can leverage existing model systems and develop better models to address questions of brain metabolism in the context of AD.

The articles of this Research Topic highlight a variety of reviews and original research which discuss and address topics of metabolism in AD (Figure 1). Several articles focus on potential AD therapeutics. These include a thorough review on the use of pioglitazone for AD treatment. Pioglitazone is a peroxisome proliferator-activated receptor gamma and alpha (PPAR-γ; PPAR-α) activator. PPARs are transcription factors critical for regulation of many pathways implicated in AD including insulin and glucose metabolism, lipid homeostasis, inflammation, tau and Aβ homeostasis, and mitochondrial function. The review by Saunders et al. discusses pre-clinical and clinical data with longitudinal observational studies revealing a positive impact of pioglitazone in AD and dementia onset in those at risk. The authors also discuss the dose-dependent effects and the caveats revealing future needs for further study into discrepancies found with placebo controlled blinded studies. Norowitz and Querfurth discusses mTOR regulation and drug targeting in AD. The authors focus on nuances for targeting mTOR in therapies including specificity for disease/region/and timing, pleiotropy, personalized therapy with relation to the effects of genetic factors, and the role of lifestyle factors and interventions.

Figure 1

www.frontiersin.org

FIGURE 1. Metabolism in Alzheimer's Disease. Research and review articles discuss the role of hormone response to meals, lifestyle factors on brain function with respect to mTOR, PPAR, PS1, ABAC7, and APOE. All of which regulate glycolysis/glucose and lipid metabolism, mitophagy/autophagy, and mitochondrial function. Created with BioRender.com.

Several other articles discuss the role of specific metabolic pathways in AD. Zhang et al. reviewed the role of glycolytic metabolism in brain resilience in AD. The authors highlight the correlation between glycolytic flux, Aβ, and tau accumulation in humans, where decreased glycolytic

function is associated with higher pathologies. In a separate review article, Kyrtata et al. discuss glucose transport in AD with particular focus on glucose transporter (GLUT) deficiencies in AD. The authors discuss the timing of changes to GLUT expression and glucose uptake in brain through rodent studies and how this relates to the timing of onset of Aβ pathology.

An additional review presents the effect of sialometabolism on brain health and AD. Rawal and Zhao discuss the role of sialic acids in brain function and neuroinflammation. The novelty of this pathway in AD is the identification of sialic acid binding Ig-like lectin 3 (CD33) as a genetic risk factor for AD through GWAS.

A separate AD genetic risk factor, ATP binding cassette subfamily A member 7 (ABCA7) was examined. Aikawa et al. used mice with haplodeficiency of ABCA7 to determine the response to immune modulation with lipopolysaccharide (LPS). The authors report that mice deficient in ABCA7 had activated lipid metabolism pathways. This study again highlights the relationship between metabolism and neuroinflammation. Morris et al. describes the role of meal stimulated hormone response through the incretin pathway in cognitive function and brain volume. The authors report that in human AD subjects, a higher meal-stimulated response of insulin, glucose, and peptide tyrosine was observed. Brain volume significantly correlated negatively with insulin, C-peptide, and glucose-dependent insulinotropic polypeptide (GIP). These articles highlight the role of diverse metabolic pathways in brain health, aging, and AD.

A focus on genetic risk factors and metabolism was discussed through a review of APOE in AD by Husain et al. The authors focused on the role of APOE in lipid transport and interactions with AD pathologies (such as tau and Aβ). Other genetic components of AD include mutations in presenilin (PS) in familial AD, and PS has a role in mitochondrial function. Contino et al. examined the role of PS deficiency on neurons and astrocytes derived from mice. Their prior studies showed mitochondrial deficits in mouse embryonic fibroblasts, but in the current study no effects were observed on similar endpoints. This study highlights the importance of model systems used for study.

Mitophagy and autophagy are implicated in AD and are the focus of many therapeutic initiatives. Tran and Reddy discuss deficiencies in autophagy and mitophagy in AD. The authors focus on metabolic drivers of autophagy/mitophagy deficiencies, the influence of aging, and how these pathways influence AD pathologies.

SYMPTOMS

Signs and symptoms

The course of Alzheimer's is generally described in three stages, with a progressive pattern of cognitive and functional impairment.[25][13] The three stages are described as early or mild, middle or moderate, and late or severe.[25][13] The disease is known to target the hippocampus which is associated with memory, and this is responsible for the first symptoms of memory impairment. As the disease progresses so does the degree of memory impairment.[13]

First symptoms

Stages of atrophy in Alzheimer's.

The first symptoms are often mistakenly attributed to aging or stress.[26] Detailed neuropsychological testing can reveal mild cognitive difficulties up to eight years before a person fulfills the clinical criteria for diagnosis of Alzheimer's disease.[27] These early symptoms can affect the most complex activities of daily living.[28] The most noticeable deficit is short term memory loss, which shows up as difficulty in remembering recently learned facts and inability to acquire new information.[27][29]

Subtle problems with the executive functions of attentiveness, planning, flexibility, and abstract thinking, or impairments in semantic memory (memory of meanings, and concept relationships) can also be symptomatic of the early stages of Alzheimer's disease.[27] Apathy and depression can be seen at this stage, with apathy remaining as the most persistent symptom throughout the course of the disease.[30][31] The preclinical stage of the disease has also been termed mild cognitive impairment (MCI).[29] This is often found to be a transitional stage between normal aging and dementia. MCI can present with a variety of symptoms, and when memory loss is the predominant symptom, it is termed amnestic MCI and is frequently seen as a prodromal stage of Alzheimer's disease.[32] Amnestic MCI has a greater than 90% likelihood of being associated with Alzheimer's.[33]

Alzheimer's Symptoms

Drugs used in Alzheimer's disease

Approved Drugs
(Role well established)

Cholinesterase Inhibitors
- Donepezil
- Rivastigmine
- Galantamine

NMDA Antagonist
- Memantine

Experimental Drugs
(Role under evaluation)

Nicotinic Receptor Agonist
- 4 OH-GTS-21

Antioxidants
- Ginkgo biloba
- Vitamin E
- Melatonin

PPAR gamma Agonist
- Pioglitazone

Gamma Secretase Inhibitor
- Semagacestat

5HT-6 Antagonist
- SB-271046

Statins
- Simvastatin
- Pravastatin

Others
- Heavy Metal Chelators
- Estrogens
- Antiinflammatory drugs

ALZHEIMER'S DISEASE

Personal view about a patient

Name: C. Ramachandran

Occupation: Retd. Typings supdt. scely

Age : 90yrs

Medication : Nothing is using now

How many years has left : 8 years

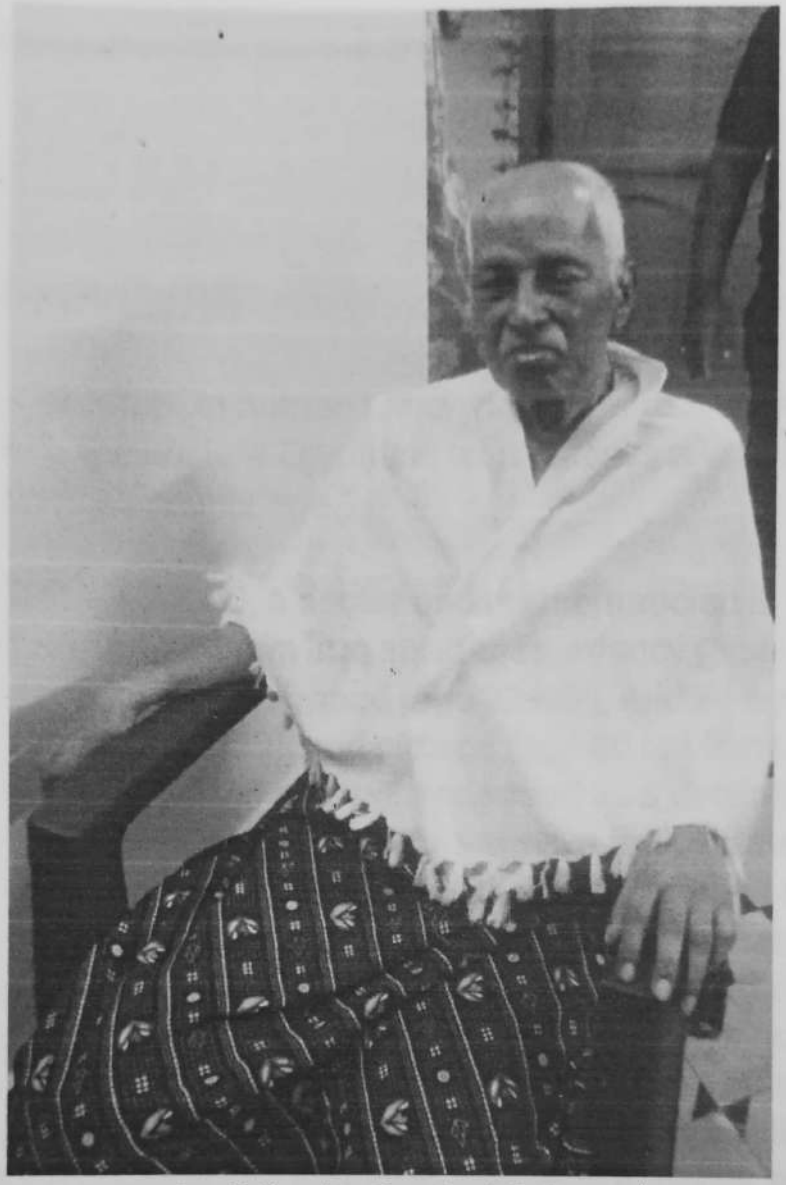
Caregiver : His daughter

Hospital : Manipal Hospital

Address :

About the patient:

This disease has came in 83yrs because his father is suffered from this disease. They tried many Hospitals But there is no medica At starting stage of this disease he forgot all things like Spectacle And House address. Now, he forgot all including his family member Doctors suggest at starting stage puzzle solving. Now his conditi is very bad now he is not talking and not chewing the food. On Just drinks of fruits. Every thing of this patient is taking by his daughter including Brushing, Bathing, food etc.



C·RAMACHANDRAN

our personal view on this patient who is suffering from Alzhemier's disease since 8 years. we studied so many things and his condition is in last stage may be his life span will be end in 2 years. And medication is stopped now. It is from the genes of his father. we expected may be this disease will continue to next generation.

HISTORY OF DEMENTIA

Dementia has occurred in human history long before it was named. In about 2000 B.C., the ancient Egyptians were already aware that memory declines as people age.[3]

Pythagoras (570–495 B.C.), a doctor and mathematician in Greece, classified a lifetime of human into six stages; infancy (age 0–6), adolescence (age 7–21), adulthood (age 22–49), middle age (age 50–62), senescence (age 63–79), and old age (age 80 or older). Of these stages, senescence and old age were regarded as a declining phase of mind and body, and some people who survive to this time, were expected to degenerate in mind to a level of suckling baby and finally become stupid. Hippocrates (460–370 B.C.), a doctor in Greece, believed that brain injury results in cognitive disorder, and Plato (428–347 B.C.), a philosopher in Greece, mentioned that the principal cause of dementia is old age itself because the mental performance is destined to inevitably degrade. On the contrary, Marcus Tullius Cicero (106–43 B.C.), a philosopher, politician and jurist in Rome, pointed out that ageing does not always cause the decline of mental performance, except in people with weak will. In brief, he indicated that dementia is not an inevitable consequence of ageing.[4]

Around the 2nd century A.D., Aretheus, a doctor in Turkey, described dementia by grouping it respectively into delirium, a reversible acute disorder of cognitive function and dementia, an irreversible chronic disorder.[5]

The medical and scientific study on dementia suffered a setback with the decline of the Roman Empire in the 5th century and reduction in financial aid. Subsequently, theocracy dominated the Middle Ages, as

of dementia is a first step to eliminate the widespread social stigma and promote public health. We are in the 106th year since the introduction of the term, 'Alzheimer's disease', which is the most common form of dementia. The recent developments in dementia study from the remarkable progress in science and medical technology, have led to outstanding advancements in epidemiological investigation of disease, determination of causes, and diagnosis and therapeutic techniques. Despite the limited effectiveness of medicines, innovative improvement through continued research is expected in the future.

Awareness of the past is required to understand the present status of dementia and obtain insights for the future.

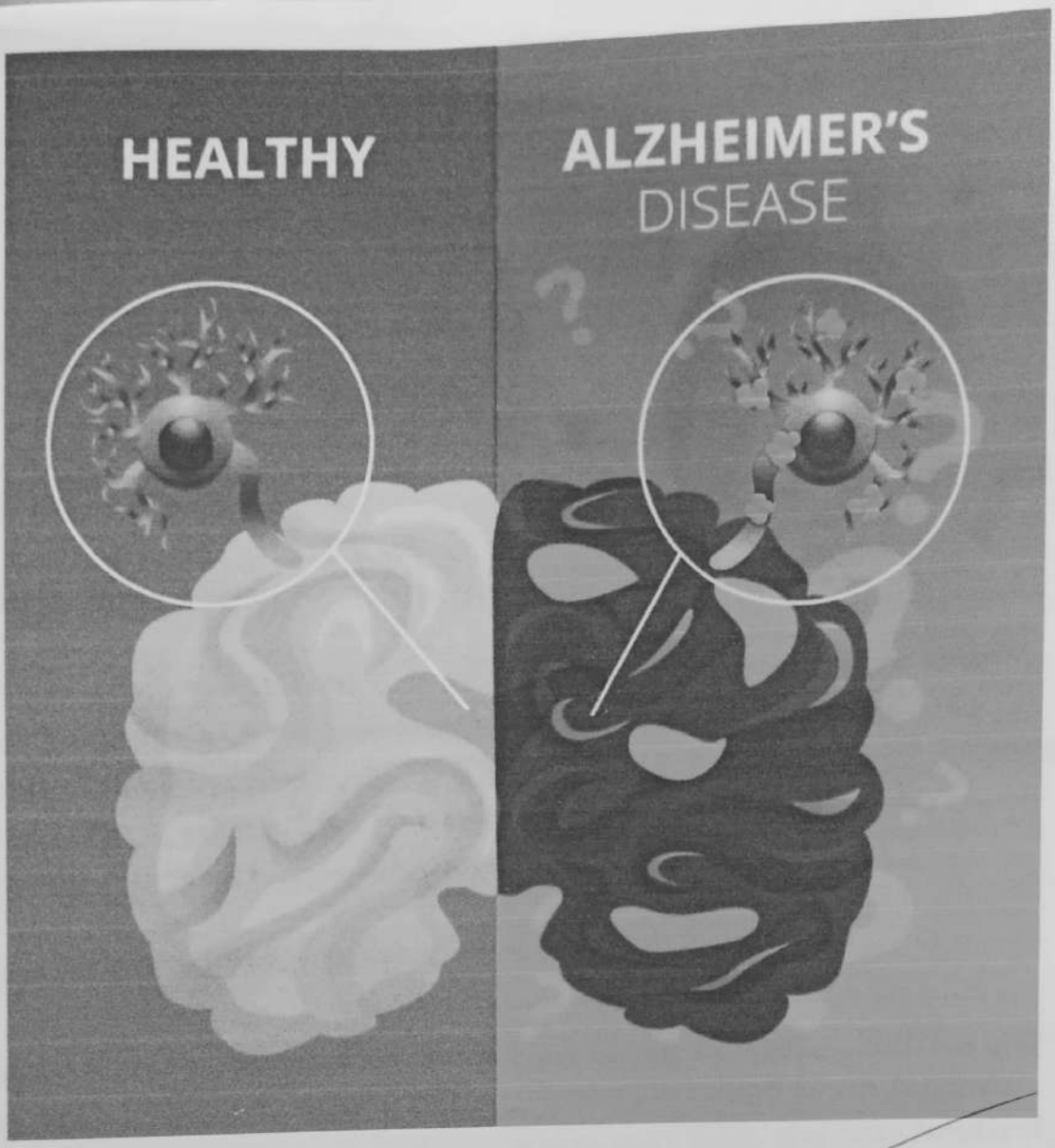
In this paper, we review the etymology and history of dementia, perceptions on dementia in the past, and the historical developments leading to the terminology of Alzheimer's disease.

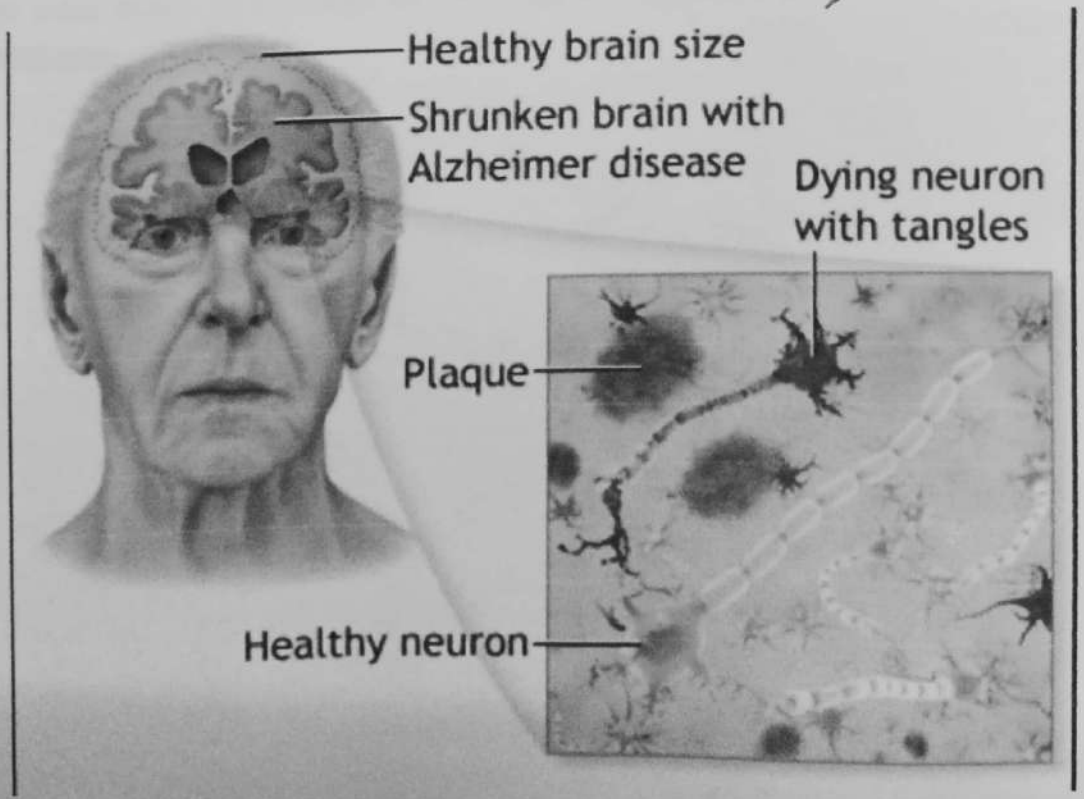
Go to:

ETYMOLOGY OF DEMENTIA

The word 'dementia' appeared first in the record of mankind in around 600 A.D. Saint Isidore (560–636 A.D.), the archbishop of Seville, used the term 'dementia' for the first time in his book, 'Etymologies'.[2] The term has its origin in Latin, and is formed from the prefix 'de', which means a deprivation or loss; root 'ment', which means mind; and suffix 'ia', which indicates a state. In short, dementia refers to 'a state out of mind'.

Go to:





Go to.

ALZHEIMER'S DISEASE: THE PROGRESS OF NAMING

Alzheimer's Disease is named after the widely known doctor who focused on the study of neurosyphilis and vascular dementia. He reported the first case to the academic world. As Alzheimer's disease is common in contemporary medicine, the origin and progress of naming, 'Alzheimer's Disease', are described in detail.[12,13,14,15,16,17,18,19]

Auguste Deter (1850-1906)

'Auguste' was born into a working-class family of Kassel, Germany on May 16th 1850. Her father of four children was dead when Auguste was very young. Although the family was very poor, she was relatively well educated. At the age of 14, Auguste began work as a seamstress assistant.

what kind of food she ate during a meal, she replied that she was eating 'spinach', although eating pork and cauliflower. She was unable to fully remember the objects she had looked at. When it was difficult for her to answer Alzheimer's questions, she repeatedly stated, "So to speak, I lost myself." In the evening, symptoms became more intensified.

Alzheimer diagnosed the case as 'presenile dementia'.

Throughout her hospitalization in the mental hospital, despite loud crying offensive behaviors to others, once in a great while, she would behave politely and kindly to people around her. By day, she had to stay in the bath filled with water to calm her down, and by night, to protect her, she had to stay in an isolated room that was securely locked. On one occasion, she left the room, and started running away while shouting puzzling words; "I don't stab myself. I will not stab myself."

For continuous observation, Alzheimer made Auguste stay in the hospital. However, the medical expenses were too much for Karl. He visited his wife as often as possible, while struggling to cover the cost. He often demanded moving her to a more affordable facility. Alzheimer intervened in the situation, and allowed Auguste to be continuously treated in the Frankfurt mental hospital, but in exchange, requested possession of all her medical records and her brain on her death. Karl gave his signed consent.

In 1903, Alzheimer moved to the mental hospital affiliated with Munich medical school via Heidelberg at the invitation of Emil Kraepelin (1856–1926).

Following his departure, in 1905, Auguste deteriorated in condition. She mumbled repeatedly to herself, was unable to get out of her bed alone, and She was unable to conduct any regular daily activities on her own including eating.

Alzheimer summarized in detail the process and aspects of the memory decline in her medical records. He described her symptoms as progressive cognitive disorder, local neurological symptom, hallucination, delusion, and psychological social disability.

Finally, she lost all cognitive ability, and succumbed to septicemia and pneumonia on April 8th 1906. She was 55 years old at the time. The brain of the patient was sent from Frankfurt to Munich together with the medical records. Alzheimer set to conduct a biopsy of her brain immediately.

Alois Alzheimer (1864-1915)

Alois Alzheimer was born in Marktbreight, a small Bavarian city in southern Germany on June 14th 1864. His father worked in a notary office, his first wife died of puerperal fever 2 years before the birth of Alzheimer, leaving only one son. After a while, he remarried an aunt of the son; they had 6 sons and daughters, and Alzheimer was the oldest child. When Alzheimer was young, the parents moved to a place where educational conditions were better for their children. Especially, Alzheimer showed a remarkable talent in science, and was trained in medical schools of Berlin, Tubingen, and Wurzburg University. He showed enthusiasm for observation of microtissues through a microscope and anatomy. In 1887, he graduated with honors from Wurzburg medical school.

Auguste Deter

ALOIS ALZHEIMER

several abnormal behaviors such as short-term amnestic disorder, disorientation and dysphrasia. Alzheimer studied her symptoms thoroughly for years.

In the summer of 1902, a year after his wife's death, Kraepelin (who taught Nissl while in Heidelberg) invited Alzheimer to join him in collaborative research. Alzheimer accepted the invitation and moved to Heidelberg immediately. Despite having the opportunity to meet Nissl again, a year later, in 1903, he moved his practice to the hospital affiliated with Munich medical school along with Kraepelin.

The patient died 4 years after Alzheimer left Heidelberg. As agreed, Alzheimer obtained the patients' brain and medical records. She became the first patient with Alzheimer's disease.

First, he conducted a biopsy of brain to investigate the correlation between the medical records and the patient's symptoms. He found that the cerebral cortex was generally thinned. The region that controlled memory, language, judgement, and thinking was severely impaired. Senile plaques were formed in neurons, and tangles were found in nerve fibers. At the time, the consensus among medical doctors was that senile plaque could be found in 70-year-old patients, while occurrence of neurofibrillary tangles was a new observation. Considering her age, both findings were exceptional.

Currently, this case would be diagnosed as early-onset Alzheimer's dementia. As Alzheimer's dementia is not likely to develope before 65-years of age, it is difficult for such early-onset Alzheimer's dementia to account for >10% of total earlyonset Alzheimer's disease.

On Kraepelin's encouragement, on November 3rd 1906, Alzheimer

at the time.

The handwritten medical records and the interviews with Auguste were not read since 1909. However, 87 years later, we are able to access these records.

Franz Nissl (1860-1919)

Franz Nissl was born in Frankenthal, Germany. Theodor Nissl, his father, wanted him to be a priest, but he entered Ludwig Maximilian University of Munich and became a doctor, and then majored in psychiatry.

Because of his interest in neuropathology, Nissl developed various staining methods while conducting a study on the cerebral cortex. The biggest outcome of his work was the 'Nissl stain'.

Nissl carried out a study with Bernhard von Gudden his teacher in Munich that involved the care of Prince Otto who suffered from mental illness. Meanwhile, Gudden was found drowned with Ludwig II, King of Bavaria, who was Gudden's patient at the same time in 1886; hence, Nissl had to look for a new occupation. Prince Otto was the younger brother of King Ludwig II.

In 1889, Nissl moved to Frankfurt where he met Alois Alzheimer. He conducted a study with Alzheimer for 7 years. Despite being 4 years senior to Alzheimer, Nissl helped and encouraged him as a friend to perform both clinical treatment and basic research simultaneously. Nissl became a witness at his wedding. Alzheimer took Nissl's advice, and they made progress together with the study on cerebral arteriosclerosis, epilepsy, and dementia, etc.

DIAGNOSIS

PET scan of the brain of a person with Alzheimer's disease showing a loss of function in the temporal lobe

Alzheimer's disease is usually diagnosed based on the person's medical history, history from relatives, and behavioral observations. The presence of characteristic neurological and neuropsychological features and the absence of alternative conditions supports the diagnosis.[100][101] Advanced medical imaging with computed tomography (CT) or magnetic resonance imaging (MRI), and with single-photon emission computed tomography (SPECT) or positron emission tomography (PET), can be used to help exclude other cerebral pathology or subtypes of dementia.[102] Moreover, it may predict conversion from prodromal stages (mild cognitive impairment) to Alzheimer's disease.[103] FDA-approved radiopharmaceutical diagnostic agents used in PET for Alzheimer's disease are florbetapir (2012), flutemetamol (2013), florbetaben (2014), and flortaucipir (2020).[104] Because many insurance companies in the United States do not cover this procedure, its use in clinical practice is largely limited to clinical trials as of 2018.[105]

Assessment of intellectual functioning including memory testing can further characterise the state of the disease.[1] Medical organizations have created diagnostic criteria to ease and standardise the diagnostic process for practising physicians. Definitive diagnosis can only be confirmed with post-mortem evaluations when brain material is available and can be examined histologically for senile plaques and neurofibrillary tangles.[105] [106]

Criteria

There are three sets of criteria for the clinical diagnoses of the spectrum of Alzheimer's disease: the 2013 fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5); the National Institute on Aging-Alzheimer's Association (NIA-AA) definition as revised in 2011; and the International Working Group criteria as revised in 2010.[33][105] Three broad time periods, which can span decades, define the progression of Alzheimer's disease

from the preclinical phase, to mild cognitive impairment (MCI), followed by Alzheimer's disease dementia.[107]

Eight intellectual domains are most commonly impaired in AD—memory, language, perceptual skills, attention, motor skills, orientation, problem solving and executive functional abilities, as listed in the fourth text revision of the DSM (DSM-IV-TR).[108]

The DSM-5 defines criteria for probable or possible Alzheimer's for both major and mild neurocognitive disorder.[109][110][111] Major or mild neurocognitive disorder must be present along with at least one cognitive deficit for a diagnosis of either probable or possible AD.[109][112] For major neurocognitive disorder due to Alzheimer's disease, probable Alzheimer's disease can be diagnosed if the individual has genetic evidence of Alzheimer's[113] or if two or more acquired cognitive deficits, and a functional disability that is not from another disorder, are present.[114] Otherwise, possible Alzheimer's disease can be diagnosed as the diagnosis follows an atypical route.[115] For mild neurocognitive disorder due to Alzheimer's, probable Alzheimer's disease can be diagnosed if there is genetic evidence, whereas possible Alzheimer's disease can be met if all of the following are present: no genetic evidence, decline in both learning and memory, two or more cognitive deficits, and a functional disability not from another disorder.[109][116]

The NIA-AA criteria are used mainly in research rather than in clinical assessments.[117] They define Alzheimer's disease through three major stages: preclinical, mild cognitive impairment (MCI), and Alzheimer's dementia.[118][119] Diagnosis in the preclinical stage is complex and focuses on asymptomatic individuals;[119][120] the latter two stages describe individuals experiencing symptoms.[119] The core clinical criteria for MCI is used along with identification of biomarkers,[121] predominantly those for neuronal injury (mainly tau-related) and amyloid beta deposition.[117][119] The core clinical criteria itself rests on the presence of cognitive impairment[119] without the presence of comorbidities.[122][123] The third stage is divided into probable and possible Alzheimer's disease dementia.[123] In probable Alzheimer's disease dementia there is steady impairment of cognition over time and a memory-related or non-memory-related cognitive dysfunction.[123] In possible Alzheimer's

disease dementia, another causal disease such as cerebrovascular disease is present.[

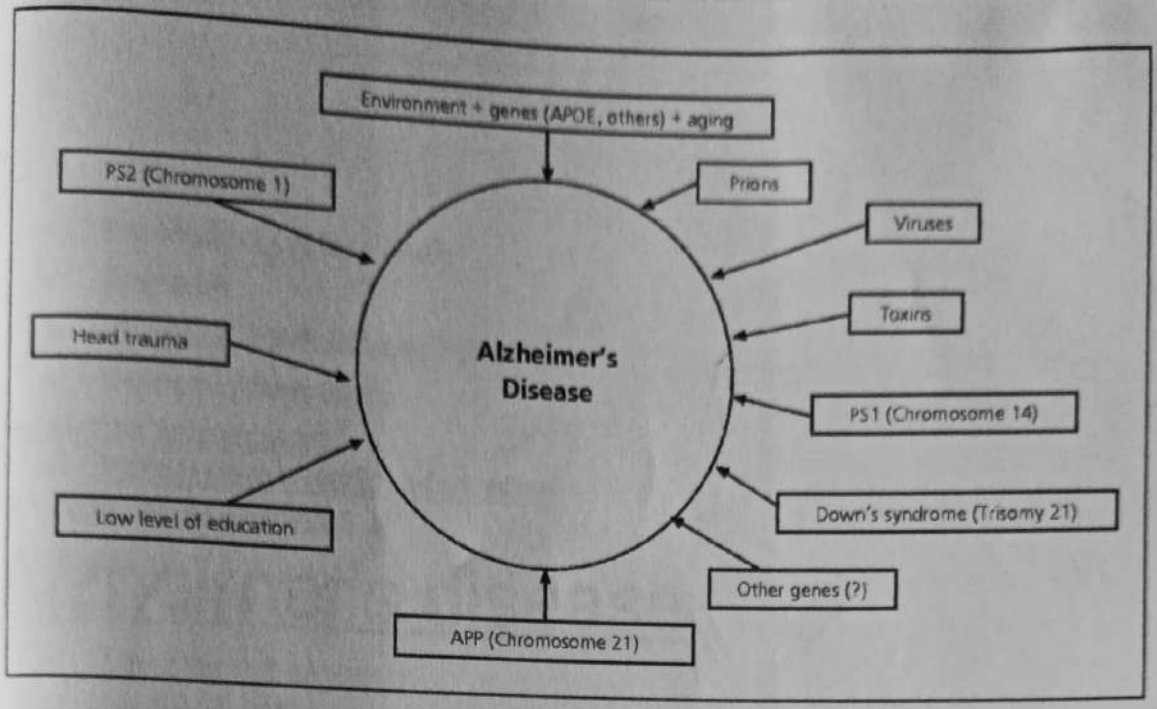
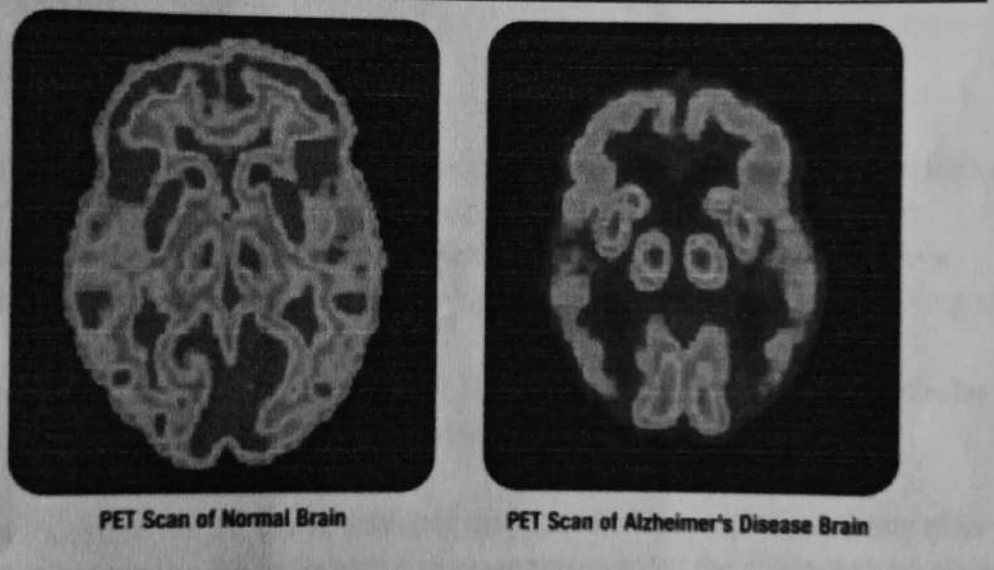


Figure 1

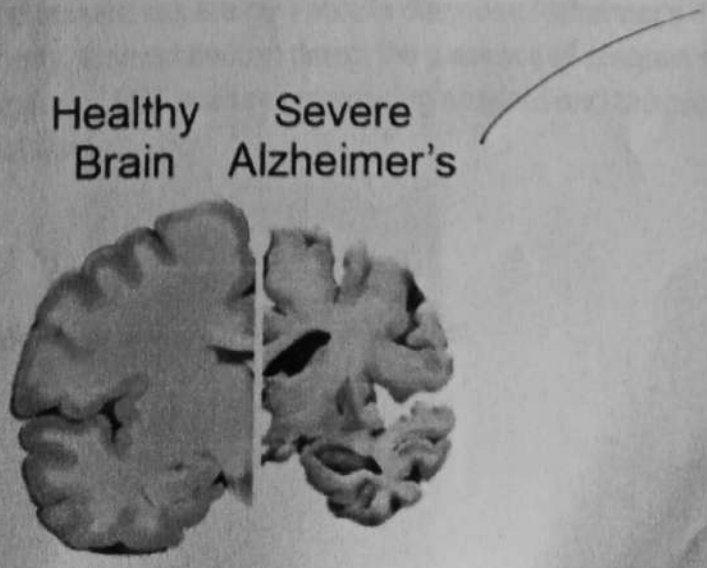
Genetic and environmental factors associated with the development of Alzheimer's disease

The known genetic causes of Alzheimer's disease are summarized on the right side. They account for only a small number of the total cases. The possible environmental factors are largely speculative; none are proven causes. It is essential to identify the genetic and environmental factors which are specific to Alzheimer's disease, determine their relative importance and capitalize on this knowledge in order to treat and prevent the disease.



PET Scan of Normal Brain

PET Scan of Alzheimer's Disease Brain



Alzheimer's disease

Diagnosis

An important part of diagnosing Alzheimer's disease includes being able to explain your symptoms, as well as perspective from a close family member or friend about symptoms and their impact on daily life. Additionally, a diagnosis of Alzheimer's disease is based on tests your doctor administers to assess memory and thinking skills.

Laboratory and imaging tests can rule out other potential causes or help the doctor better identify the disease causing dementia symptoms.

Traditionally, Alzheimer's disease was only diagnosed with complete certainty after death, when examining the brain with a microscope revealed the characteristic plaques and tangles. Clinicians and researchers are now able to diagnose Alzheimer's disease during life with more certainty. Biomarkers can detect the presence of plaques and tangles, such as specific types of PET scans or measuring amyloid and tau proteins in plasma and cerebral spinal fluid.

Tests

A diagnostic work-up would likely include the following tests:

Physical and neurological exam

Your doctor will perform a physical exam and likely assess overall neurological health by testing the following:

- Reflexes
- Muscle tone and strength
- Ability to get up from a chair and walk across the room
- Sense of sight and hearing
- Coordination
- Balance

Lab tests

Blood tests may help your doctor rule out other potential causes of memory loss and confusion, such as a thyroid disorder or vitamin deficiencies.

Mental status and neuropsychological testing

Your doctor may give you a brief mental status test to assess memory and other thinking skills. Longer forms of neuropsychological testing may provide additional details about mental function compared with people of a similar age and education level. These tests can help establish a diagnosis and serve as a starting point to track the progression of symptoms in the future.

Brain imaging

Images of the brain are now used chiefly to pinpoint visible abnormalities related to conditions other than Alzheimer's disease — such as strokes, trauma or tumors — that may cause cognitive change. New imaging applications — currently used primarily in major medical centers or in clinical trials — may enable doctors to detect specific brain changes caused by Alzheimer's.

Imaging of brain structures include the following:

Genetic testing generally isn't recommended for a routine Alzheimer's disease evaluation. The exception is people who have a family history of early-onset Alzheimer's disease. Meeting with a genetic counselor to discuss the risks and benefits of genetic testing is recommended before undergoing any tests.

Care at Mayo Clinic

Our caring team of Mayo Clinic experts can help you with your Alzheimer's disease-related health concernsStart Here

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- Diagnosing Alzheimer's
- CT scan

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Treatment

Drugs

Current Alzheimer's medications can help for a time with memory symptoms and other cognitive changes. Two types of drugs are currently used to treat cognitive symptoms:

- **Cholinesterase inhibitors.** These drugs work by boosting levels of cell-to-cell communication by preserving a chemical messenger that is depleted in the brain by Alzheimer's disease. These are usually the first medications tried, and most people see modest improvements in symptoms.

  Cholinesterase inhibitors may also improve neuropsychiatric symptoms, such as agitation or depression. Commonly prescribed cholinesterase inhibitors include donepezil (Aricept), galantamine (Razadyne ER) and rivastigmine (Exelon).

  The main side effects of these drugs include diarrhea, nausea, loss of appetite and sleep disturbances. In people with certain heart disorders, serious side effects may include cardiac arrhythmia.

- **Memantine (Namenda).** This drug works in another brain cell communication

network and slows the progression of symptoms with moderate to severe Alzheimer's disease. It's sometimes used in combination with a cholinesterase inhibitor. Relatively rare side effects include dizziness and confusion.

In June 2021, the Food and Drug Administration (FDA) approved aducanumab (Aduhelm) for the treatment of some cases of Alzheimer's disease. This is the first drug approved in the United States to treat the underlying cause of Alzheimer's by targeting and removing amyloid plaques in the brain. The FDA approved the drug on the condition that further studies be conducted to confirm the drug's benefit. Experts also need to identify which patients may benefit from the drug.

Sometimes other medications such as antidepressants may be prescribed to help control the behavioral symptoms associated with Alzheimer's disease.

Creating a safe and supportive environment

Adapting the living situation to the needs of a person with Alzheimer's disease is an important part of any treatment plan. For someone with Alzheimer's, establishing and strengthening routine habits and minimizing memory-demanding tasks can make life much easier.

You can take these steps to support a person's sense of well-being and continued ability to function:

- Always keep keys, wallets, mobile phones and other valuables in the same place at home, so they don't become lost.
- Keep medications in a secure location. Use a daily checklist to keep track of dosages.
- Arrange for finances to be on automatic payment and automatic deposit.
- Have the person with Alzheimer's carry a mobile phone with location capability so that a caregiver can track its location. Program important phone numbers into the phone.
- Install alarm sensors on doors and windows.
- Make sure regular appointments are on the same day at the same time as much as possible.

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Impressions : Very good.

Prof. Incharge - Biochemistry

DEPARTMENT OF BIOCHEMISTRY

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M.Sc., Ph.D.

CERTIFICATE

This is to certify that the project entitled "Graves' disease" is submitted by M.Jhansi rani, S.Siveesha, Y.Latha Under my supervision for the degree of Bachelor of science in Biochemistry group

9/10/2022

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CERTIFICATE

This is to Certify that the project work entitled " Studies on Vitamins Deficieny & Disorder" being submitted by B. DEVARAJ, M. VAMSI, S. KALYAN, V. DEVAVARAM, V. GHOUSE BASHA, T. BASWANTH IIIrd B.sc, Biochemistry Group in Partial fulfillment of the requirements for the award of the degree of Bachelor of Science in Biochemistry group , the Project work is a record of a Bonafied Work carried out by her under my guidance and supervision.

Signature of the Project guide

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DEPARTMENT OF BIOCHEMISTRY

S.K.P. GOVT.COLLEGE,GUNTAKAL.

CERTIFICATE

This is to certify that the Project work entitled " Studies on Alzhemiers Disease", being submitted by MANGALA PUSHPA SAI & VADDE PAVITHRA, IIIrd B.S.c, Biochemistry group. In partial fulfillment of the requirements for the award of the degree of Bachelor of Science In Biochemistry group, the project work is a record of a Bonafied work carried out by her under my guidance and supervision.

19/07/2022

Signature of the project guide & supervisor,

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M.Sc.

Department of Biochemistry

S.K.P Govt. college, Guntakal.

PRINCIPAL
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A

MINOR PROJECT REPORT

ON

POST HARVESTING TECHNOLOGY
IN
" TRANSEGENIC FISHES "
2021-22

SRI KRISHNADEVARYA UNIVERSITY, ANANTAPURAMU – 515003, A.P

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G.MEENA KUMARI

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Department of Zoology

DECLARATION

I here by declare that entitled **" TRANSEGENIC FISHES "**completed and submitted by me.

ACKNOWLEDGEMENT

This is a project on **" TRANSEGENIC FISHES "** we are very thankful to our guide lecturer "G.Meena Kumaris" (M.Sc.Mphil) department of Zoology, SKP Government college, Guntakal for his valuable guidance and assistance, without which the accomplishment the task would have never been possible we also thank for giving oppurtunity to explore into the real worlds and realize the inter relation.

We extended our sincere thanks to N.Narayana (HOD) for their co-operation during the project work and also we are thankful to all the lecturer and teacher for their support and co-operation.

G.MEENA KUMARI

LECTURER IN ZOOLOGY

Expt.Name :

Project Work

ట్రాన్స్‌జెనిక్ చేపలు

మరియు

ట్రాన్స్‌జెనిక్ ప్రయోజనాలు

Expt.Name :

ఉద్దేశ్యం :-

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ఉపోద్ఘాతం :-

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Signature

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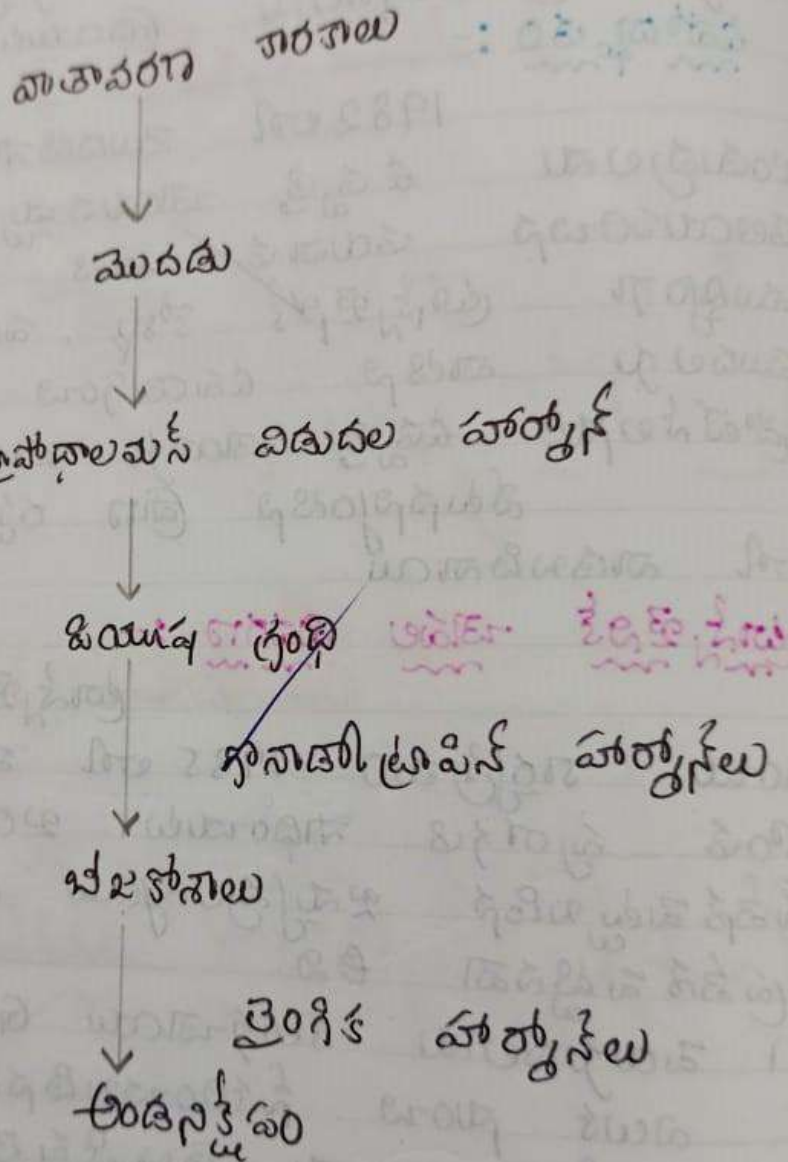
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గోనాడోట్రోపిన్ హార్మోనులు
బీజకోశాలు
లైంగిక హార్మోనులు
అండనిష్క్రమం

ఈ ఇన్సులిన్ పొందిన చేపలు వన్య జాతుల కన్నా రెట్టు వేగంలో పెరుగుదలను చూపించినాయి. పరిసర ఉష్ణోగ్రత తగ్గినప్పుడు పెరుగుదల కారక హార్మోను కారకము ప్రేరేపించబడుతుంది. దీని వలన చేపలు ఉన్నతంగా పెరుగుదలను చూపిస్తాయి. వాటి ఇన్సులిన్ పొందిన చేపలను, వన్యజాతుల నుంచి పొరు పరిచి ప్రత్యేకంగా పెంచడం జరుగుతుంది. మైక్రో సిరంజి ద్వారా ఇన్సులిన్ ప్రవేశపెట్టుట చాలు వరకు చేపలలో సత్ఫలితాలు సాధించినదని చెప్పవచ్చు. ముఖ్యంగా కార్పులు, క్యాట్ లు, గోల్డ్ చేపలు, బ్లూక్, సాల్ - మండలే మొదలగు ముఖ్యమైన రకాలు.

ఘనీభవన నిరోధక ప్రోటీన్లు, ట్రాన్స్ జెనిక్ చేపలలోను రక్తం ఘనీభవన ఉష్ణోగ్రతను తగ్గిస్తాయి. ఘనీభవన నిరోధక ప్రోటీన్ను కలిగిన ట్రాన్స్ జెనిక్ ట్రౌట్ చేపలు -1.2°C వద్ద కూడా జీవించగల్గుతాయి.

బహుళ్కరణ వన్య జాతుల చెరువులోనికి ఈ ట్రాన్స్ జెనిక్ మగ చేపలు చేరి ప్రజననం కూడా జరుపుతాయి. అయితే వీటి సంతాన వ్యవధి తక్కువగా ఉంటుంది. ఈ ఇన్సులిన్ పొంది చేపలను మనవడ వాగెళ్ళపరంగా సులభంగా ప్రవేశపెట్టవచ్చు.

మగ, ఆడ చేపల యొక్క శుక్ర కణాలు, గ్రుడ్లను పొరు పొరుగా పొడిగానున్న ట్రేలోకి చేర్చాలి. తదుపరి గ్రుడ్లు ఉన్న ట్రే లోకి చేల్లని నీటిని శుక్ర కణాలు మెల్లగా చేర్చాలి. చిన్నగా వాటిని కదిలించిన యెడల ఫలదీకరణ జరుగుతుంది.

Signature .....

Expt.Name :

మనవే పెరుగుదల జన్యువు GH ను చేపలోకి ప్రవేశ పెట్టుటకై అవి వున్న ఖాతుల కంటే రెండు రెట్లు పెరుగుదల కనబరిచినట్లు తెలియుచున్నది.

ఇదే విధంగా 1991 సంవత్సరంలో మద్రాస్ కామరాజ్ విశ్వవిద్యాలయంలో ఫ్రో చేపలలో ప్రయోగాలు నిర్వహించినారు. ఫ్రో చేపలలోనికి AF ని చొప్పించినారు. అందుకు వారు ఎలుక GH ని ఉపయోగించినారు అది వాటి పెద్ద చేపలలో కనిపించింది.

ప్రయోజనాలు :-

వ్యవసాయ విధానాలపై పెరుగైన విధముగా ట్రాన్స్‌జెనిసిస్ ను చేయవచ్చును.

(1) అవసరమైన లక్షణాన్ని నియంత్రించు జన్యువును నిర్ధిష్టంగా ఎంపిక చేయబడినదానికి వీలుంటుంది.

(2) కొత్త లక్షణమును స్థిరీకరించుటకు పట్టే వ్యవధి చాలా తక్కువగా ఉంటుంది. ఒక తరములోనే ఇది సాధ్యమవుతుంది.

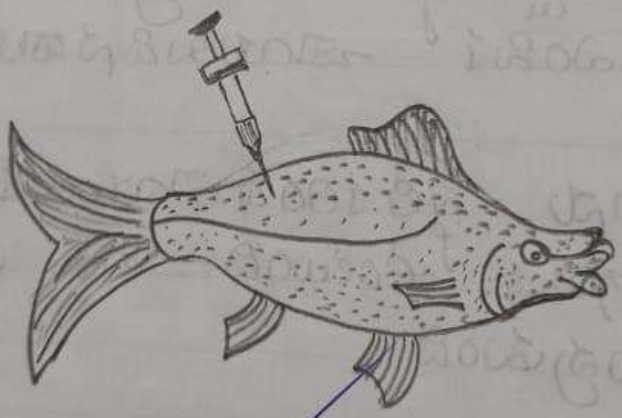
(3) కొన్ని జీవుల్లో లేని లక్షణములను ట్రాన్స్‌జెనిక్ విధానాల ద్వారా నిర్వహించవచ్చును.

(4) ఈ విధానానికి అయ్యే వ్యయ ప్రయాసలు చాలా తక్కువే

Signature .....

K.B.W

పిట్యూటరి గ్రంథి.



పిట్యూటరి గ్రంథిని తొలగించడం వల్ల కలిగే ఫలితాలు.

ట్రాన్స్‌జెనిక్ చేపలు :-

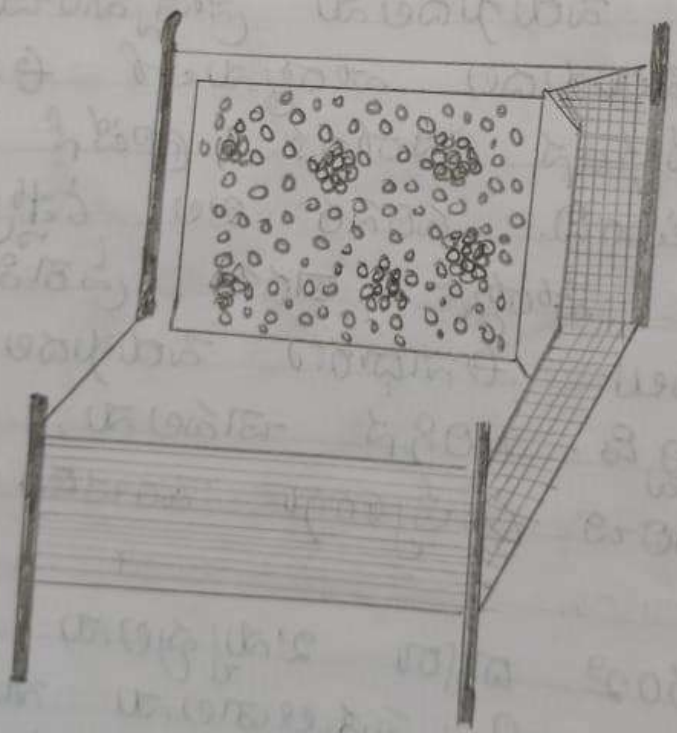
ట్రాన్స్‌జెనిక్ చేపలను రూపొందించు
కార్యక్రమం 1985 లో మొదలైనది. ఈ దశలో కొం
పురోగతి సాధించుట జరిగినది. చేపలలోనికి ప్రవే
పెట్టబడిన జన్యువులన్నియు సూక్ష్మ సిరంజి ద్వారా ప్రవే
పెట్టినవి. అవి పెరుగుదలను కలుగజేయు GH
ను మానవుడు లేదా ఎలుక నుండి సేకరించబడినది.

ఈ జన్యు మార్పిడి జరిగిన చేపలు వన్య జాతుల
కన్నా రెండు రెట్లు వేగంలో పెరుగుదలను చూపించినాయి
ముఖ్యంగా పెరుగుదలకు సంబంధించిన జన్యువు అదు
ప్రతిరూపాలున్నాయి. పెరుగుదలను ప్రోత్సహించు లోపించిన
DNA పై గల పెరుగుదల హార్మోనుతో అనుసంధాన
పరచబడిన ఘనీభవన నిరోధక ప్రోటీన్ ప్రోత్సాహము
వినియోగించుకుంటుంది. పరిసర జల ఉష్ణోగ్రత తగినప్పు
పెరుగుదల కారక హార్మోన్ కారకం ప్రేరేపించబడుతుం
దీని వలన చేపలు అసాధారణ పెరుగుదలను చూపిస్తా
ఇట్టి జన్యు మార్పిడి జరిగిన చేపలను, వన్య జాతుల
నుండి వేరు పరచి ప్రత్యేకంగా పెంచడం జరుగుతుం

మైక్రో సిరంజి ద్వారా జన్యువులను ప్రవేశ పెట్టుట
చాలా వరకు చేపలలో సఫలీకృతాలను సాధించినదని
చెప్పవచ్చు. ముఖ్యంగా కార్ప్‌లు, క్యాట్ ఫిష్ లు, గోల్డ్ చేప
సాలమన్, మడకా ముఖ్యమైన రకాలు. అయితే సూ
సిరంజి ద్వారా DNA ను ప్రవేశ పెట్టుట వల్ల అండము
చేరిన పురుష బీజకేంద్రకములోనికి, ప్రవేశపెట్టబడుతుం
ఈ విధానంలో DNA తంతువు యాదృచ్ఛికంగా ప్రవేశ

Signature ....

పెరిగిన పాపా



హాచింగ్ పాపా

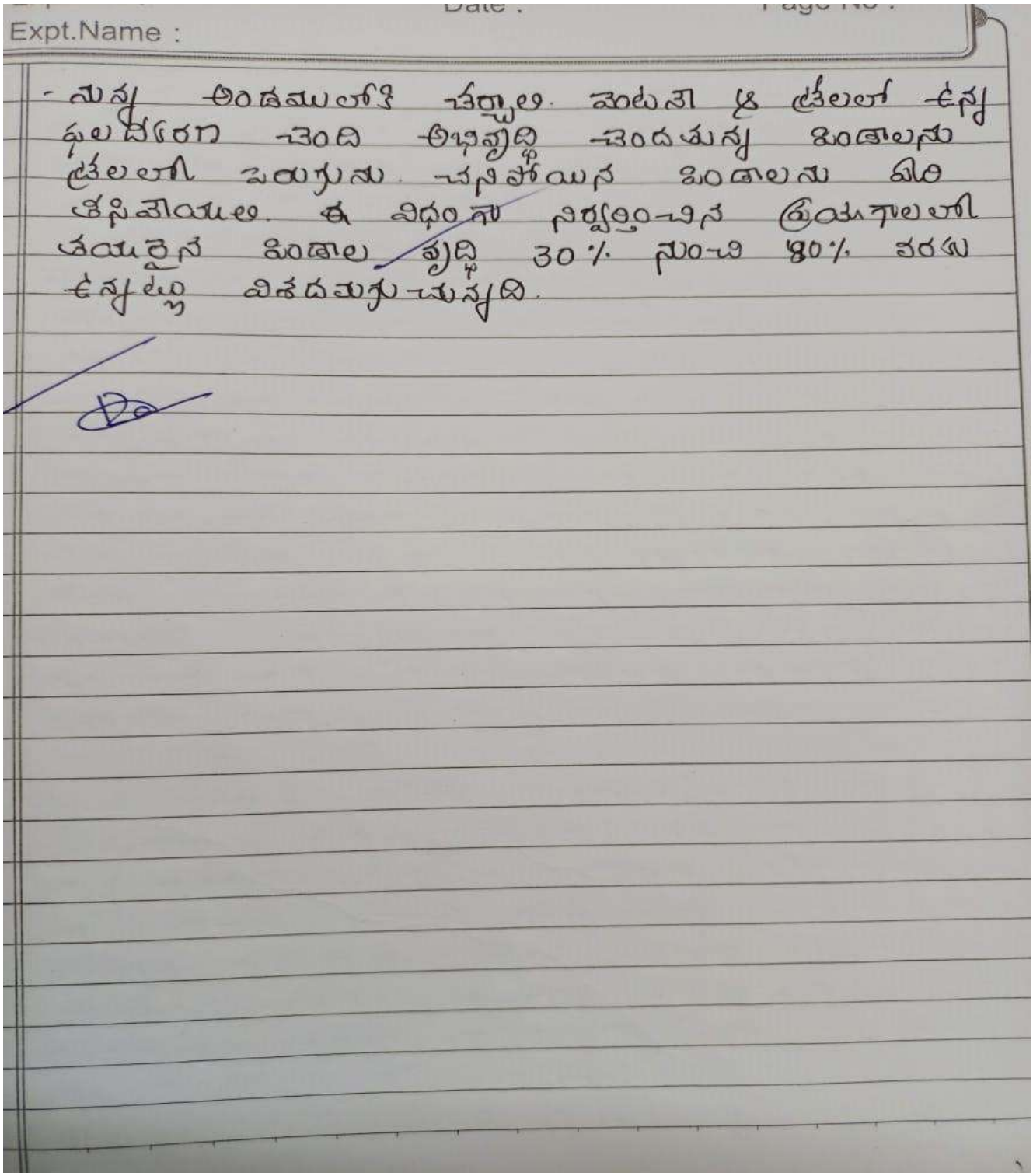
Expt.Name : Date : Page No :

పెట్టబడుతుంది అతిథేయిలో సరైన స్థానములందు ఇముడుతుందని నమ్మకం లేదు. ఇట్టి అండాన్ని గ్రహీతగా ఉన్నటి స్త్రీ జీవి యొక్క అండములోకి చొప్పబడుతుంది.

ఘనీభవన నిరోధక ప్రోటీన్లు ట్రాన్స్‌జెనిక్ చేపలోని రక్త ఘనీభవన ఉష్ణోగ్రతను తగ్గిస్తాయి. -0.6°c వద్ద వన్యజాతి ట్రౌట్ చేపలు మనలేవు. కానీ ఘనీభవన నిరోధక ప్రోటీన్ను కలిగిన ట్రాన్స్‌జెనిక్ ట్రౌట్ చేపలు -1.2°C వద్ద కూడా జీవించగలుగుతాయి. బహిర్గత సారి వన్యజాతులు చెరువులలోకి ఈ ట్రాన్స్‌జెనిక్ మగ చేపలు చేరి ప్రజననం కూడా జరుపుతాయి. అయితే వీటి సంతాన వృద్ధి తక్కువగా ఉంటుంది. ఇది ట్రాన్స్-జెనిక్ జీవుల ఉత్పత్తి వలన వన్యజాతులు అంతరించి పోవు ప్రమాదమున్నది. ఇట్టి జీవులు పర్యావరణ వ్యవస్థ పై ఋణాత్మక ప్రభావాన్ని కలిగి ఉంటాయనే భయం వలన ఈ జన్యుమార్పిడి పొందిన చేపలను మనవుడు వాణిజ్య పరంగా ఇంకా ప్రవేశపెట్టలేదు.

అయితే, ఇటీవల కాలములో వేర పద్ధతిలో ప్రయోగాలు నిర్వర్తిస్తున్నారు. మగ, ఆడ చేపల యొక్క శుక్రకణాలను, గ్రుడ్లను వేరు వేరుగా పొడిగా ఉన్న ట్రే-లోకి చేర్చాలి. తరువాత గుడ్లు ఉన్న ట్రేలోకి చల్లని నీటిని శుక్రకణాలు మెల్లగా చేర్చాలి. చిన్నగా వాటిని కదిలించిన యెడల ఫలదీకరణ జరుగుతుంది. ఆ తరువాత అండముల చుట్టూ చేరిన బెంగులను తొలగించాలి. వెంటనే 10^6 to 10^8 అణువులు గల DNA ను 20 ml లోప అంతకంటే కొద్దిగా తక్కువ గాని, 14 Gm దగ్గరలో

Signature ....


Expt.Name :

- మున్న అండములోకి చేర్చాలి. పొటనె ట్రే లలో ఉన్న ఫలదీకరణ చెంది అభివృద్ధి చెందుతున్న పిండాలను ట్రేలలోకి ఒయ్యను. చనిపోయిన పిండాలను మరి తీసివేయాలి. ఈ విధంగా నిర్వహించిన ప్రయోగాలలో తయారైన పిండాల వృద్ధి 30% నుంచి 80% వరకు ఉన్నట్లు విశదమగుచున్నది.

Good

G.Meena Kumari (**LECTURER IN ZOOLOGY)**

CERTIFICATE

Name : H. Lavanya Class : III BSc-B2(TM)

Register No : 200107011 Exam No :

College SKP. Government Degree College Guntakal

This is Certified to be the bonafide work of the student in the

Zoology paper – VI Laboratory during the academic

year 20 21 / 20 22

No of practicals certified _____ out of _____ in the

suject of Post harvesting Technology B3

N. Narayana
Head of the Department

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

M. Pedda
Examiner's signature

Principal

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Anantapuramu (Dt.)

Date :.....

College Stamp

CERTIFICATE

Name : C. Venkatesh Class : III Yr B.Z.C

Register No : 20015/007 Exam No :

College S.K.P. G.D.C, GTL

This is Certified to be the bonafide work of the student in the

Zoology *Laboratory during the academic*

year 20 21 / 20 22

No of practicals certified _____ *out of* _____ *in the*

suject of postharvesting - B_3

N. Narayana

Head of the Department

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

D. Rao

Examiner's signature

Principal

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Anantapuramu (Dt.)

Date :.....

College Stamp

(N.B: The candidate is expected retain his/her journal till he/she passes in the subject.)

B3

Certificate

Name: . M. Hari Krishna Class : III BSc [B2C] .

Roll No.: 20015103 Exam No. :

Institution S.K.P. Govt Degree college

This is certified to be the bonafide work of the student in the Zoology *Laboratory during the academic year 2020/2021.*

No. of practicals certified 02 *out of* 02 *in the subject of* Zoology cluster - B_3

N. Narayan

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

Teacher-in-charge

Examiner's Signature

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Anantapuramu (Dt.)

Date :.....

Institution Rubber Stamp

(N.B : The candidate is expected to retain his/her journal till he/she passes in the subject.)

Certificate

Name: . M. Mamatha devi. Class : III BZC

Roll No.: 200151014 Exam No. :

Institution S.K.P Govt. Degree College

This is certified to be the bonafide work of the student in the ZOOLOGY *Laboratory during the academic year* 2021/2022.

No. of practicals certified 06 *out of* 06 *in the subject of* Zoology – B_3

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL
Teacher in charge

Examiner's Signature

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Ananthapuramu (DL)

Institution Rubber Stamp

Date :.....

(N.B. : The candidate is expected to retain his/her journal till he/she passes in the subject.)

CERTIFICATE

DEPARTMENT OF

Class: IV Register No.: 20015-1020

Certified that this is the bonafide record of practical work done in the laboratory by the candidate

V. Babu Naik

during the year 2020-21 - 2021-22

No.of Practicals conducted B3 No.of Practicals attended B3

Post harvesting Technology

Lecturer

Head of the Department

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

Submitted for the practical examination held on

Valued by M. Padma

1. G. N 6/8/22

2.

Examiners

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Anantapuramu (Dt.)

CERTIFICATE

Name : K. GANESH. Class : IV BSc BZC

Register No : 200151002 Exam No :

College SKP Govt Degree college Guntakal

This is Certified to be the bonafide work of the student in the

Zoology cluster B3 Laboratory during the academic

year 2021 / 2022

No of practicals certified 06 out of 06 in the

subject of Post harvesting Technology

N. Narayan

Head of the Department

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

Examiner's signature

Principal

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL Anantapuramu (Dt.)

College Stamp

Date :.....

(N.B: The candidate is expected retain his/her journal till he/she passes in the subject.)

Certificate

Name: N. Pavankalyankumar Class : IIIrd B.Sc. B.Z.C

Roll No.: 20015l016 Exam No. :

Institution S.K.P. GDC Guntakal

This is certified to be the bonafide work of the student in the Zoology *Laboratory during the academic year 2021/2022.*

No. of practicals certified 06 *out of* 06 *in the subject of* Zoology - B-III
Post harvesting Technology

N. Narayana
Teacher in-Charge
LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

Examiner's Signature

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Ananthapuramu (DL)

Date :

Institution Rubber Stamp

(N.B : The candidate is expected to retain his/her journal till he/she passes in the subject.)

Certificate

Name: . S. Naymunnisa Class : III B.Sc [B.Zc]

Roll No.: 20015/022 Exam No. :

Institution S.K.P Govt Degree college Guntakal

This is certified to be the bonafide work of the student in the Zoology-Post harvesting B3 *Laboratory during the academic year* 2021/2022

No. of practicals certified 06 *out of* 06 *in the subject of* Zoology-B3

N. Narayana
LECTURER IN ZOOLOGY
S.K.P. DEGREE COLLEGE
GUNTAKAL
Teacher In-charge

Examiner's Signature

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Anantapuramu (Dt.)

Date :

Institution Rubber Stamp

(N.B : The candidate is expected to retain his/her journal till he/she passes in the subject.)

Certificate

Name: . N. Muni SWAMY Class : IIIrd BZC

Roll No.: 20015101 7 Exam No. :

Institution S.K.P. G.D.C. GTL

This is certified to be the bonafide work of the student in the Zoology - B3 *Laboratory during the academic year 2021/2022.*

No. of practicals certified 06 *out of* 06 *in the subject of* principles of Aquaculture cluster B3
Post harvesting Technology

N. Narayan

Teacher In-charge
LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

M. R.

Examiner's Signature

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Ananthapuramu (Dt.)

Date :

Institution Rubber Stamp

(N.B : The candidate is expected to retain his/her journal till he/she passes in the subject.)

B3

Certificate

Name: M. SRAVANI Class : III BSC BZC

Roll No.: 20015 1015 Exam No. :

Institution SKP Govt Degree college Guntakal

This is certified to be the bonafide work of the student in the

Zoology - B3 *Laboratory during the academic year 20 / 20 .*

No. of practicals certified 6 *out of* 6 *in the subject of* Postharvesting Technology B3

LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
GUNTAKAL

Teacher In-charge

Examiner's Signature

Principal

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Anantapuramu (Dt.)

Date :.....

Institution Rubber Stamp

(N.B : The candidate is expected to retain his/her journal till he/she passes in the subject.)

Certificate

Name: . B. pramila Class : III Bsc T/m

Roll No.: 20015005 Exam No. :

Institution SKP GOVT Degree college

This is certified to be the bonafide work of the student in the Zoology - cluster - B_3 *Laboratory during the academic year 2021 / 2022.*

No. of practicals certified 06 *out of* 06 *in the subject of* cluster - B_3 Post Haavesting Technology

N. Narayana
LECTURER IN ZOOLOGY
S.K.P. GOVT. DEGREE COLLEGE
Teacher-in-charge

Examiner's Signature

PRINCIPAL
S.K.P. Govt. Degree College
GUNTAKAL, Ananthapuramu (Dt.)

Date :..... *Institution Rubber Stamp*

(N.B : The candidate is expected to retain his/her journal till he/she passes in the subject.)

S.K.P.GOVT DEGREE COLLEGE, GUNTAKAL

DEPT OF MICROBIOLOGY

Student study project

CERTIFICATE

This is to certify that B.Haseena, J.Nagaveni, K.Dileep of class III MBC has submitted project work on topic Estimation of Hemoglobin during the year 2021-22. The above mentioned project work is scrutinized and found to be satisfactory.

G.Rama 23/11/21
LECTURER

PRINCIPAL
S.K.P. Govt. Degree Collge
GUNTAKAL, Anantapuramu (Dt.)

GRama
Good work
Keep it up

B. Haseena Begum - 200151251
J. Nagaveni - 200151252
K. Dileep - 200151258

ABSTRACT

Childhood anemia is highly prevalent worldwide. Improving the hemoglobin level of preschool age children could yield substantial benefits in cognitive and psychosocial development and overall health . While evidences based overall recommendations for reducing childhood anemia in high anemia prevalence countries are available, there is no experimental evidence of community centered education and counseling programs, as a route to improve acceptance of iron supplement, demonstrating beneficial effects of anemia outcomes. We report on the evaluation protocol of a complex education intervention led by the community lay health worker and delivered to mothers of 12 to 59 mother old anemic children living in and visiting village day care in a large district of southern india.

INTRODUCTION

I voluntarily chosen this project work mainly concerned with nations health status by selecting a sample of area which is particularly economically backward selection of the community. To undergo this project through its report. We can help the nations health by making facilities to such people. Which is badly needed to improve the nation development is one angle for development of any country. Health and education are the two events like to eyes of a man. If these two events uplifted definitely that nation will definitely be a developed nation.

We the students of IIIrd year MICROBIOLOGY went to a survey in PRASHANTHI NAGAR, GUNTAKAL.

DEDICATION

This project work is selected oh humanity grounds to choose children below 5 years. How best their health status particularly in a slum dwellers, Is a sort of my best activity dedicated to my beloved teacher.

BACKGROUND

Over 1.6 billion people worldwide suffers from anemia and approximately 80% of the burden of this disorder is borne by individuals living in South Asia and Africa.

Anemia is associated with a significant economic burden accounts for 68.4 m illion years lived with disability (8.8% of total for all conditions), increase maternal and perinatal mortality ,and countributes to global morality.

The prevalence of anemia in india is particularly high, where 50% of reproductive age women 59 % of pregnant women, 25% lof men, 40 % of adlosecent girls,and 70% of children under five years are anemic.

The etiology of childhood anemia in limited resource settings in multifactorial, but in india it is mainly attributable to iron and other micronutrients deficiencies. Iron deficiency anemia is associated with cognitive and psychromotor retardation in children, and trails of iron supplements in iron deficient children demonstrated improved outcomes.

The major cause of IDA in india is inadequate iron intake due to both low dietary iron content of food and inadequate dietary animal poitein.

major cause of IDA in india is inadequate iron intake due to both low dietary iron content of food and inadequate dietary animal poitein.

ESTIMATION OF HEMOGLOBIN IN THE GIVEN WARD NO.06 :-

MEN

S.NO	NAME OF THE PATIENT	% OF HEMOGLOBIN
1	SOMU	12
2	RAJU	14
3	VEERESH	15
4	UDAY	13
5	KRISHNA	11
6	ARJUN	12
7	MANIKANTA	10
8	ROHIT	14
9	RAJESH	16
10	PULAIYAH	15

WOMEN :-

11	MOUNIKA	14
12	SIRISHA	13
13	VINDYA	13
14	SHABNAM	13
15	NAGAPADMA	10
16	SHIVANI	09
17	AOPORVA	12
18	SUBHASHANI	12
19	PADMAJA	13
20	SADIYA	11

CHILDREN BELOW 5 YEARS :-

S.NO	NAME OF THE PATIENT	% OF HEMOGLOBIN
21	SURESH	13
22	SUNIL	14
23	ESWAR	12
24	SRIKANTH	15
25	MANOJ	14
26	VENKAT	13
27	PANKAJ	14
28	HARI	15
29	KARTHIK	13
30	PRAKASH	12

DISCUSSION

This trail is designed to evaluate the effectiveness of an intervention intended to improve an anemia cure rates in anemic children living in ward 6 of Prashanthi nagar , Guntakal . The extensive study of secondary endpoints will be used to identify possible weak points in the compliance to intervention delivery uptake. This evaluation is one of the few large randomized trails evaluating the impact of an education and counseling intervention to reduce childhood anemia prevalence.