Biotechnology and its Applications Flow Chart

Bt Toxin and Pest Resistance

Some strains of *Bacillus thuringiensis*

↓ **Produce**

Toxic insecticidal protein crystals during a particular phase of their growth

↓ **Kill insects such as**

Lepidopterans **(tobacco budworm, armyworm)**

Coleopterans **(beetles)**

Dipterans **(flies, mosquitoes).**

Bt toxin protein exists as **Inactive Protoxins**

↓ Converted into

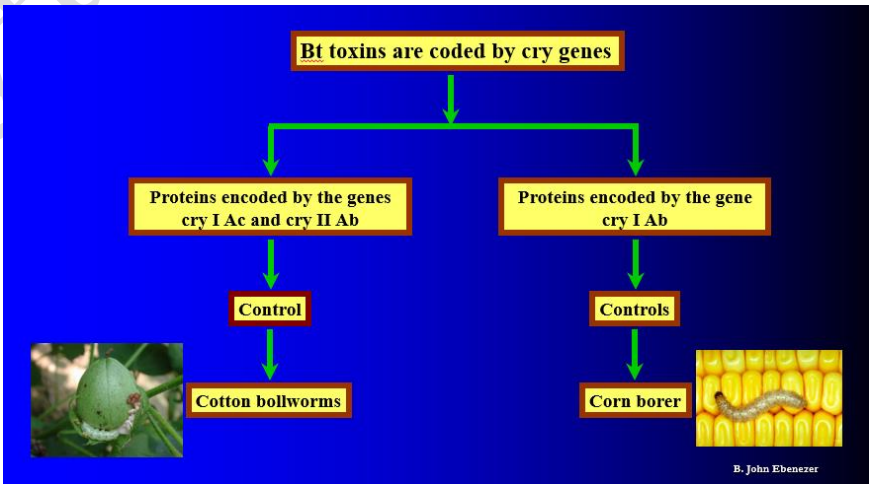
Active form of toxin due to the **alkaline pH** of the gut which solubilise the crystals.

↓

Binds to the surface of **midgut epithelial cells**

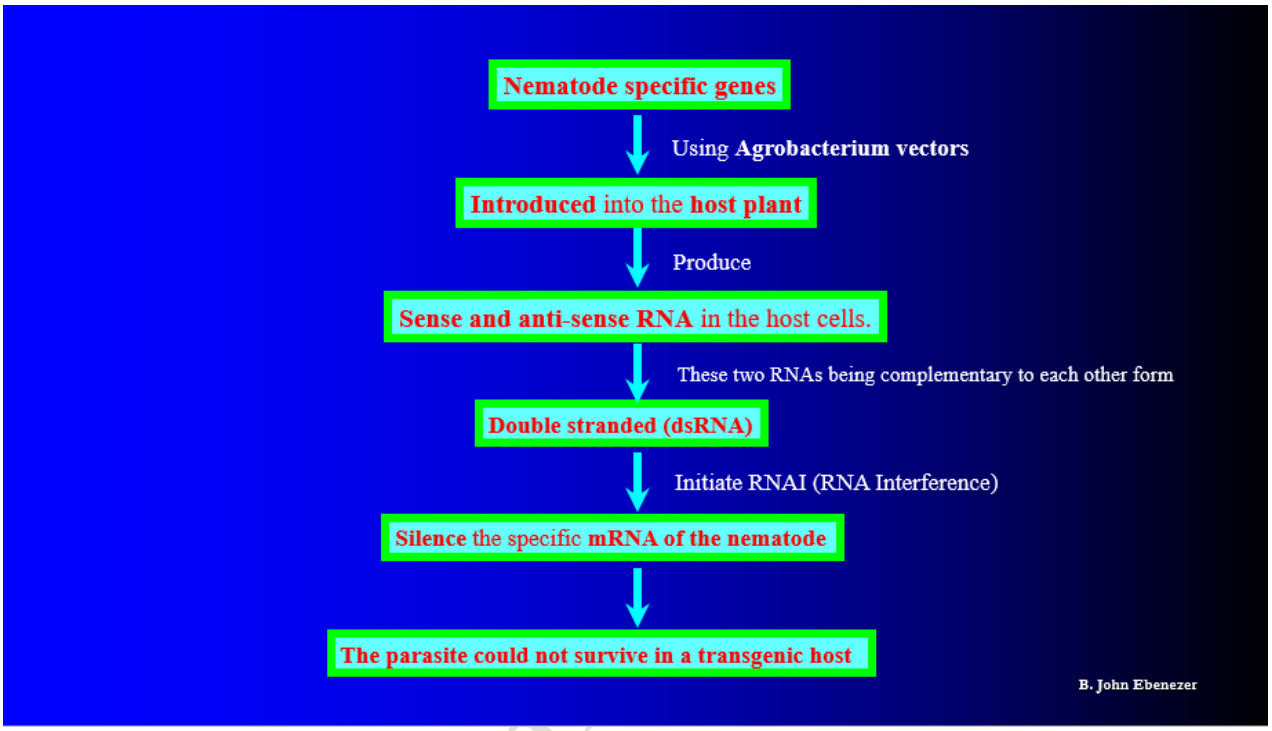
↓

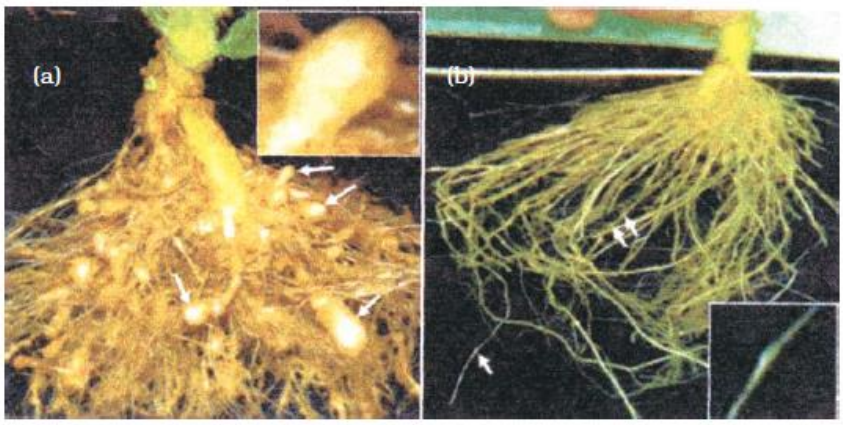
Cause **cell swelling and lysis** and leads to **death** of the insect.



Pest Resistant Plants

Strategy adopted to prevent infestation in Tobacco Plants caused by the Nematode Meloidegyne incognitia





Host plant-generated dsRNA triggers protection against nematode infestation.

(a) Roots of a typical control plant.

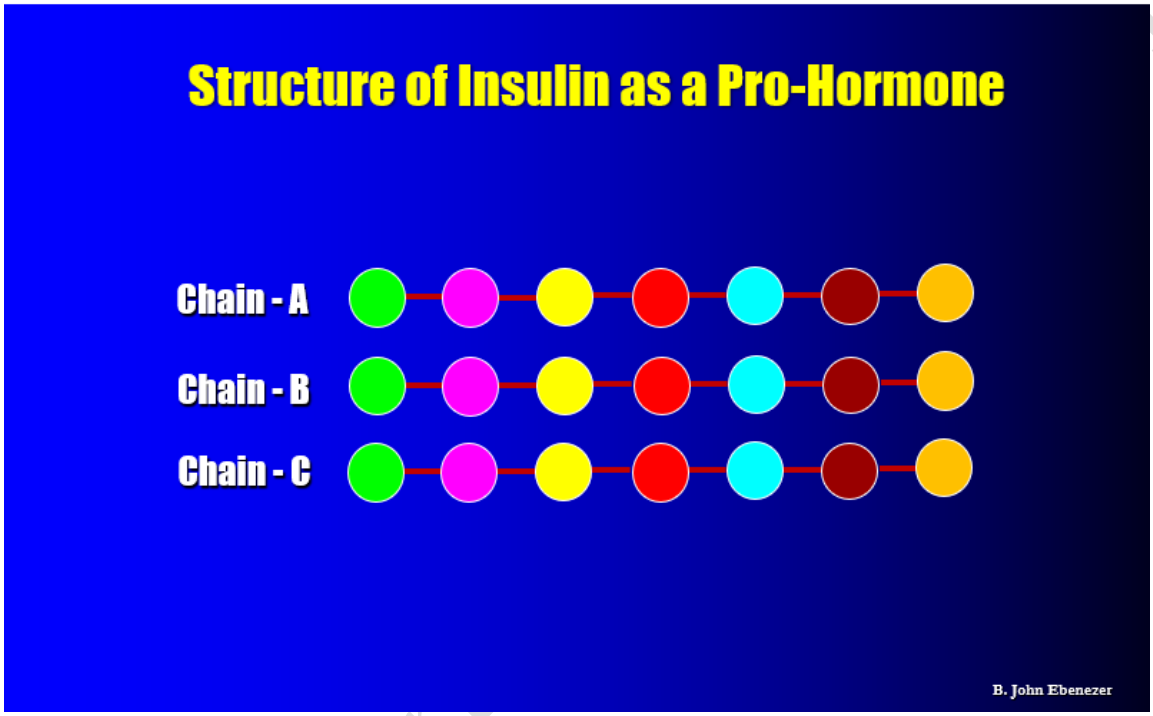
(b) Transgenic plant roots 5 days after deliberate infection of nematode but protected through novel mechanism.

Insulin as a Pro-Hormone

In mammals, including human insulin is synthesised as a prohormone.

The pro-hormone consists of two short polypeptide chains: chain A and chain B.

The pro-hormone also consists of an extra stretch called the C peptide.



Genetically Engineered Insulin

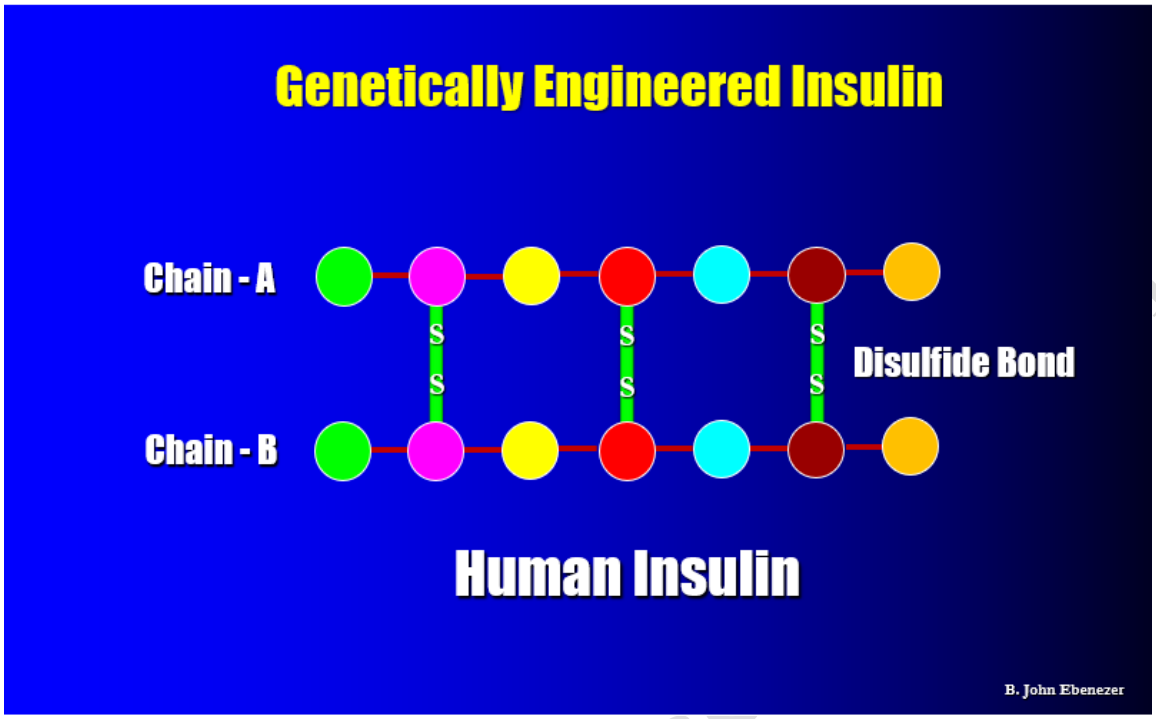
In 1983, Eli Lilly an American company prepared

Two DNA sequences corresponding to the chains A and B of human insulin.

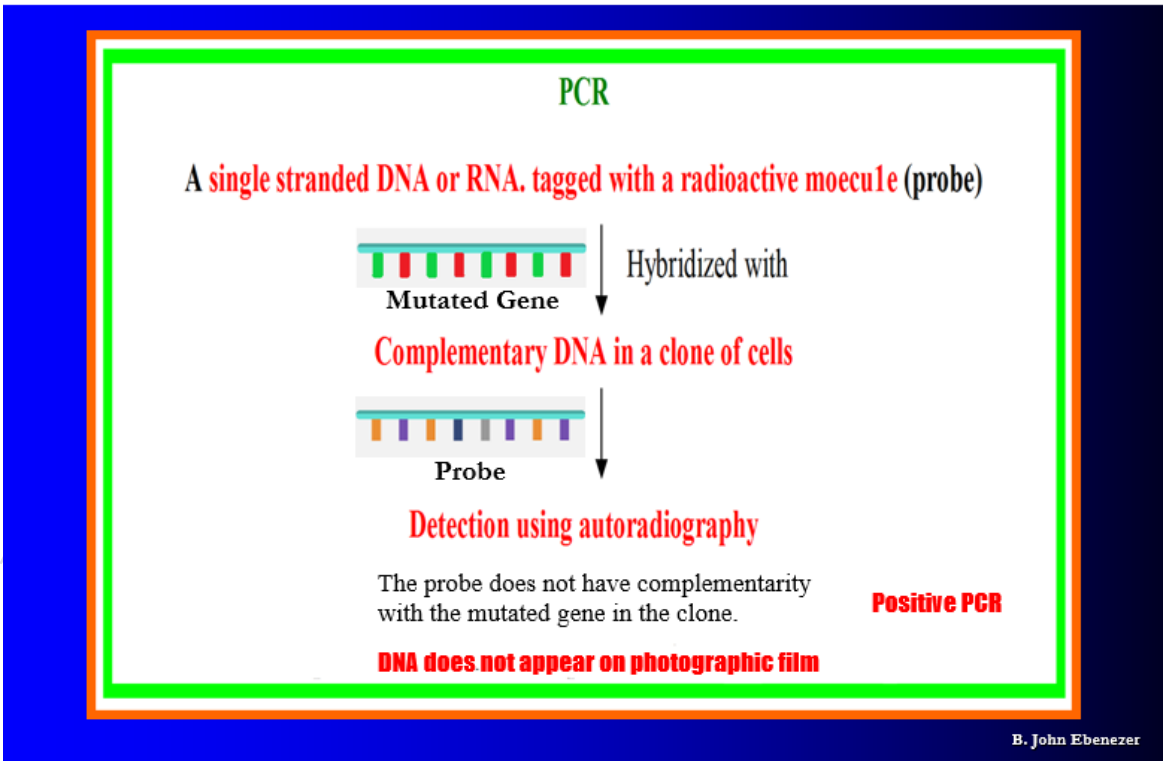
Introduced the DNA sequences in plasmids of *E. coli* to produce insulin chains.

Chain A and Chain B were produced separately.

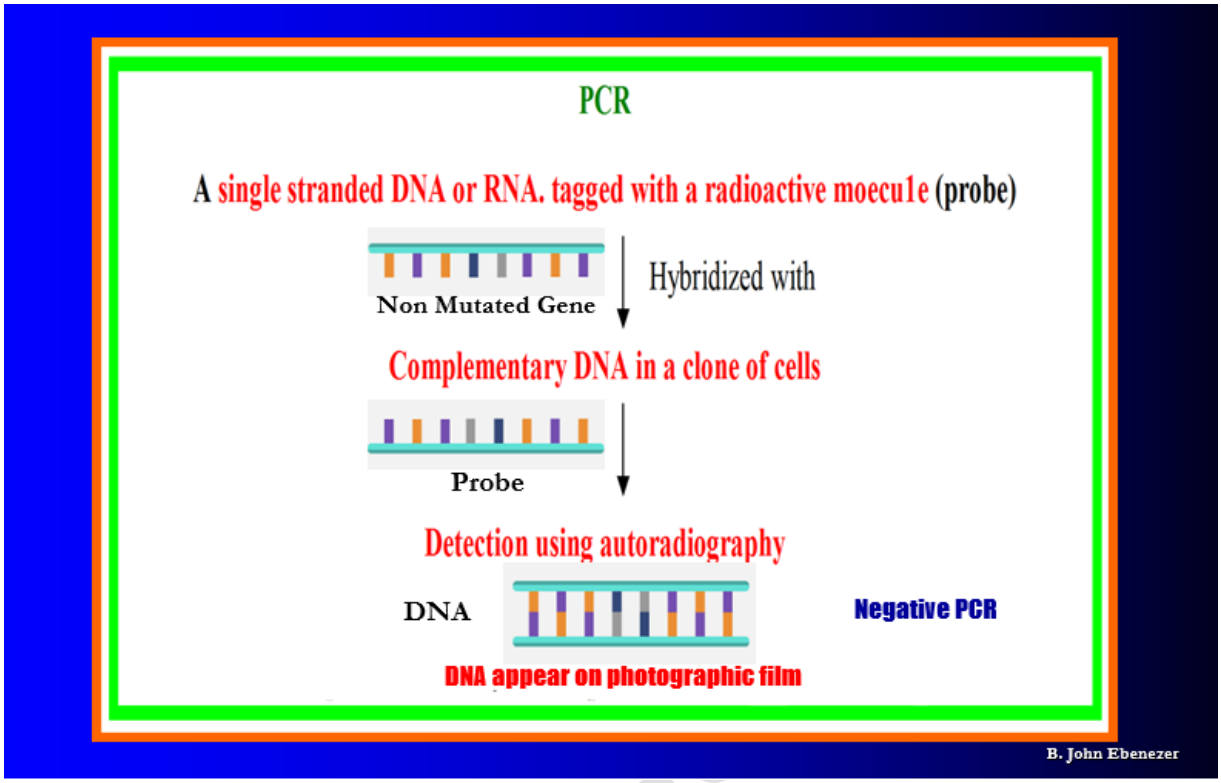
They were isolated and combined by disulfide bonds to form human insulin.



Polymerase Chain Reaction



The clone having the mutated gene does not appear on the photographic film, because the probe does not not have complementarity with the mutated gene.



Treatment for ADA Deficiency in children

The first clinical gene therapy was given in 1990 to a 4-year old girl with adenosine deaminase (ADA) deficiency.

The enzyme adenosine deaminase (ADA) is crucial for the immune system to function.

The disorder is caused due to the deletion of the gene for adenosine deaminase enzyme.

Treatment for ADA Deficiency in children

1. Bone marrow transplantation:

In some children ADA deficiency can be cured by bone marrow transplantation.

2. Enzyme replacement therapy:

In others it can be treated by enzyme replacement therapy, in which functional ADA enzyme is given to the patient by injection.

The problem with both of these approaches are, they are not completely curative.

3. Gene therapy

Gene Therapy Strategies