Return of the cloud chamber

Cosmic ray particles are repaying a debt they owe, says s ananthanarayanan

THE cloud chamber, a cavity containing a cooled, saturated vapour, records the path of a cosmic ray particle by a "vapour trail". The device helped track the movement of sub-atomic particles like protons, electrons and gamma rays in magnetic or electrical fields and even detected collision of particles or decay events. Cloud chambers had an important role in experimental particle physics from the 1920s till 1950s. The discovery of the *positron* in 1932, the first instance of *antimatter*, for which Carl D Anderson won the Nobel Prize, was made when cosmic rays passed through a lead sheet in a cloud chamber.

Particles get detected in the cloud chamber because high energy particles break the molecules of the saturated vapour into ions, or separate, charged portions which, in turn, act as nuclei for the vapour to condense. The condensed vapour along the path of the high-energy particle is the visible trail. A team of scientists at Cern, in Geneva, has now put the glove on the other hand, with streams of known particles illuminating the process of vapour condensation in cloud chambers with a different composition of vapours.

Condensation

The subject has grown in importance for understanding the weather. Winds and movements of air masses are generally understood and predictions of rainfall are generally feasible. But the weatherman remains the butt of jokes and ridicule because for all his work and equipment, like satellites and Doppler radar, there is great uncertainty in how rain clouds form and how cloudbursts happen. The nature and causes of nucleation, or the formation of centres for a vapour to condense and form droplets, has not been studied and weather-science is still based on general ideas of how droplets arise.

Water in the form of vapour, like in clouds, needs a surface on which it can carry out the change from vapour to liquid. This is the reason that pure, particle-free water vapour can be cooled even below freezing point before droplets form. At higher temperatures vapour can be 400 per cent saturated before it must condense if there are no *cloud condensation nuclei*, as such condensing centres are called. But if such centres become present, condensation can rapidly happen, leading to cloud droplets and then raindrops.

The typical raindrop is two millimetres in diameter, which is 100 times more than the cloud droplet, which forms on a cloud condensation nuclei, about 100 times smaller than a droplet. Cloud condensation nuclei, which are called aerosols (not the same as the base of perfume and other sprays), generally arise from dust, clay, soot from grass or forest fires, factory smokestacks or petrol engines, sea salt from ocean wave spray, sulphate from volcanoes, chemicals released by algae in the sea, organic matter or volatile compounds.

Apart from acting as centres of condensation, aerosols also reflect sunlight back into space and act as a heat shield for earth. With the onset of global warming, this is another reason, apart from their role in rain formation, to understand aerosols.

Cern research

Cern's Cosmics Leaving OUtdoor Droplets experiment is an international and interdisciplinary effort to study the effect of cosmic rays on the formation of atmospheric aerosols under controlled laboratory conditions. According to current estimates, about half of all aerosols are not injected into the atmosphere by sea spray or human emission, etc, but are formed by a clustering of trace atmospheric molecules, a process called *nucleation*. But the mechanics of nucleation are not understood and existing climate models are based on surmises that are adapted to match observation. Cloud is designed to understand nucleation and improve aerosol physics for better weather forecast.

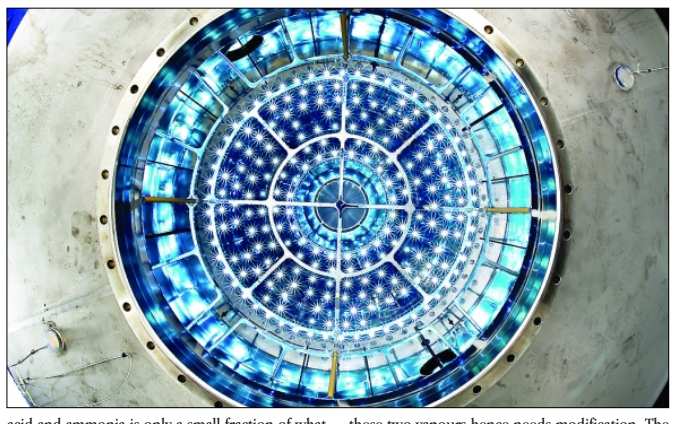
Cern's Cloud chamber permits a sensitive control of pressure, temperature, humidity, ionisation and concentration of different vapours, including the ones that promote nucleation. With low levels of contaminants, Cloud is able to assess nucleation due to selected trace gases, without distortions by other, undetected agents. Creation of cloud condensation nuclei by sulphuric acid and ammonia vapours, which are known as nucleating agents in the atmosphere, was studied, as also the creation of new particles from ions generated by ionising radiation from Cern resources. The facility has instruments to measure exceedingly trace presence of vapours and also the chemistry and growth of newly formed molecular clusters.

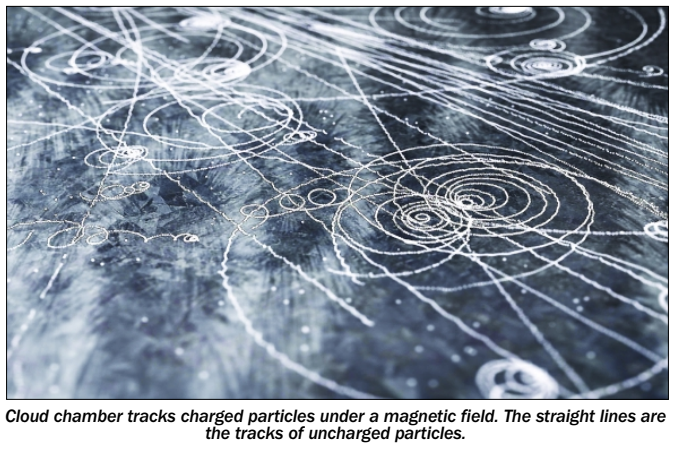
The results of the study have been significant. First, that the nucleation because of sulphuric acid and ammonia is only a small fraction of what is observed in the lower atmosphere. The current understanding that nucleation is almost all from these two vapours hence needs modification. The second result is that nucleation is substantially increased, by a factor of 10, by the action of cosmic rays. "These new results from Cloud are important because we've made a number of first observations of some very important atmospheric processes," said the experiment's spokesperson, Jasper Kirkby. "We've found that cosmic rays significantly enhance the formation of aerosol particles in the mid-troposphere and above. These aerosols can eventually grow into the seeds for clouds.

However, we've found that the vapours previously thought to account for all aerosol formation in the lower atmosphere can only account for a small fraction of the observations — even with the enhancement of cosmic rays."

This result suggests a major role of cosmic ray activity in climate change and control. "But the effect of cosmic rays on other nucleating vapours need to be studied before we can come to definite conclusions," says a release from the Cloud collaboration.

A team of scientists at Cern in Geneva has now put the glove on the other hand, with streams of known particles illuminating the process of vapour condensation in cloud chambers with a different composition of vapours.





Cloud chamber tracks charged particles under a magnetic field. The straight lines are the tracks of uncharged particles.

The writer can be contacted at simplescience@gmail.com

So much for theories on losing weight

jeremy laurance **reports on new finding by US researchers**

DIETING is harder than you think. If you cut out a chocolate bar each day you will lose only one-third of the weight that experts had thought. For decades, doctors have based their advice to those who want to lose weight on the assumption that cutting 500 calories a day will see the weight fall off at the rate of one pound a week.

"This is wrong," said Kevin Hill of the National Institutes of Health in the USA. "It does not happen." The error has arisen because the calculation did not take account of changes in metabolism as weight falls. The body adjusts to reductions in energy intake (calories eaten) by slowing its energy output (calories expended). The result is that forgoing that daily chocolate bar containing 250 calories will lead to a weight loss of about 25 pounds if it is sustained for three

years, much less than the 78 pounds predicted by the old dieting assumption.

A more sophisticated measure of weight loss, which takes account of metabolic changes and of differences between fat and thin people, has been developed by Dr Hill and colleagues of the National Institute of Diabetes and Digestive and Kidney Diseases. It shows that heavier people tend to lose weight faster than lighter people on the same diet, though they will take longer to reach the target weight than those who weigh less to begin with.

Most people on a diet achieve their maximum weight loss after six to eight months and it has been assumed this is a natural "plateauing" effect, resulting from slowed metabolism. But evidence shows that people find it hard to stick to a diet for longer than six months and that is why they stop losing weight. Body-weight plateauing occurs much later, after two to three years. There is nothing to choose between different diets that alter the fat, protein and carbohydrate balance, such as the Atkins diet that reduces carbohydrates, the researchers said. The body adapts rapidly to changes in these constituents with the result that all diets result in the same loss of body fat, at least in the short term. They said, "Little is known about the long-term effects."

It has been difficult to assess how closely people follow diets as research relies on self-reporting. The authors said, "Widespread past use of erroneous rules for estimation of human body-weight change have led to unrealistic expectations."

The Independent, London

Gene therapy

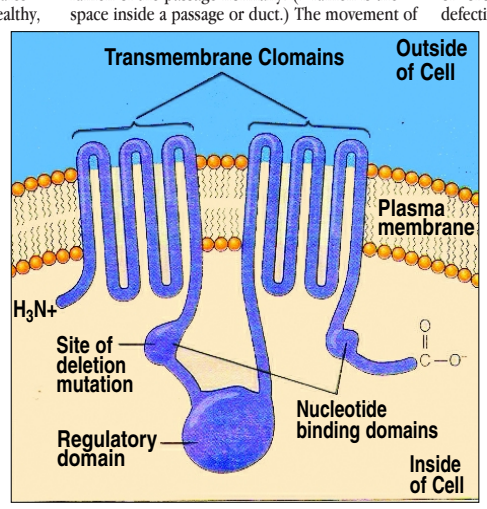
However slow the rate of progress, the results achieved are as secure as could be hoped for, says tapan kumar maitra

TRANSPORT proteins located in the plasma membrane play a critical role in speeding up and controlling the movement of molecules and ions into and out of cells. To remain healthy, our bodies depend on the proper functioning of many such membrane proteins. If any of these proteins is defective, the movement of a particular ion or molecule across cell membranes is likely to be impaired and disease may result.

An example that has attracted the attention of researchers and doctors alike is cystic fibrosis, a fatal disease caused by genetic defects in a transport protein in the plasma membrane. The parts of the body that are most noticeably affected are the lungs, pancreas and sweat glands. Complications in the lungs are the most severe medical problems because they are difficult to treat and can become life threatening. The airways of a patient are often obstructed with abnormally thick mucus and are vulnerable to chronic bacterial infections, especially by Pseudomonas aeruginosa.

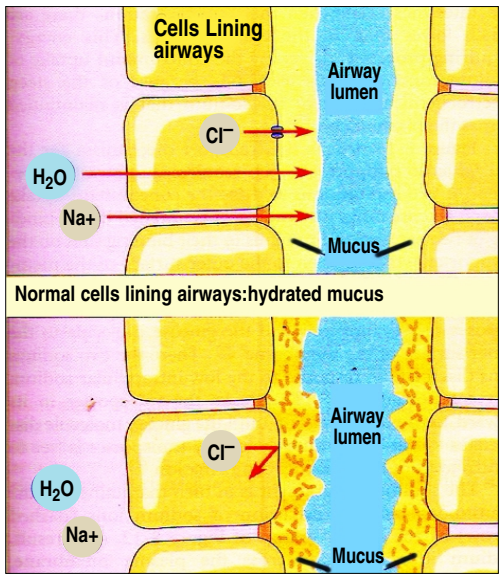
Using the tools of molecular and cellular biology, researchers have achieved a detailed understanding of this disease. During the 1980s, cells from patients were shown to be defective in the secretion of chloride ions (Cl^-). The cells that line unaffected lungs secrete chloride ions in response to a substance called cyclic AMI, whereas cells from patients do not. Experiments with tissue from patients suggested that this difference might be due to a defect in a membrane protein that normally served as a channel for the movement of chloride ions across the membrane.

Many symptoms of cystic fibrosis can be explained by faulty Cl^- secretion. In the lungs of an unaffected person, chloride ions are secreted from the cells that line the airways and enter the lumen of the passage normally. (A lumen is the space inside a passage or duct.) The movement of Cl^- out of the cell and into the lumen provides the driving force for the concurrent movement of sodium ions into the lumen. Osmotic pressure causes water to follow the sodium and chloride ions, resulting in the secretion of a dilute salt solution. The water that moves into the lumen in this way provides vital hydration to the mucus lining of the air passages. In the cells of a person with cystic fibrosis, Cl^- ions cannot exit into the lumen, so sodium ions and water do not move outward cither. As a result, the mucus is insufficiently hydrated, a condition that favours bacterial growth.



Cystic fibrosis and chloride ion secretion.

An exciting breakthrough in research came in 1989 when investigators in the laboratories of Francis Collins at the University of Michigan and of Lap-Chee Tsui and John Riordan at the University of Toronto isolated the gene that is defective in cystic fibrosis patients. The gene encodes a protein called the Cystic Fibrosis Trans-membrane conductance Regulator. The sequence of nucleotide bases in the gene was determined. Knowing the base sequence of the gene, scientists were able to predict the amino acid sequence and the structure of the protein. The protein is thought to have two sets of trans-membrane domains that anchor the protein in the plasma membrane and two nucleotide-binding folds that serve as binding sites for ATP, which provides the energy to drive transport of chloride ions across the membrane. In addition, the protein has a large cytoplasmic domain called the regulatory domain, which has several serine hydroxyl groups that can be reversibly phosphorylated. The protein has since been shown to function as a chloride channel in cells and channel function is known to be affected when the phosphorylation sites in the regulatory domain are changed as a result of a mutation in the gene.



Cells of a person with cystic fibrosis ~ dehydrated mucus infected with bacteria.

By sequencing the genes from patients, investigators have identified more than 600 mutations. The most common of these causes the deletion of a single amino acid in the first nudeotide-binding domain. The question of how this mutation causes cystic fibrosis remained unanswered until researchers examined the location of CFTR in cells with and without the mutation. Normal CFTR was found in the cell membrane, as predicted. In contrast, mutant CFTR was not detected in the plasma membrane.

The most likely explanation at present is that normal CFTR is synthesised on the rough Endoplasmic Reticulum, moves through the Golgi complex and is eventually inserted in the plasma membrane by a route. Mutant CFTR, on the other hand, is apparently trapped in the ER, perhaps because it is folded improperly. It is, therefore, recognised as a defective protein and degraded. Consequently, CFTR is not present in the plasma membrane of CF cells; chloride ion secretion cannot take place and disease results.

Armed with information about the gene and the protein, researchers are now trying to develop new treatments or perhaps even a cure for the disease. One such approach is gene therapy, in which a normal copy of a gene is introduced into affected cells of the body. Investigators would like to direct normal copies of the CFTR gene into the cells that line the airways of CF patients. These cells should then be able to synthesise a correct CFTR protein, which, unlike mutant CFTR, would be located in the plasma membrane, thereby allowing proper Cl^- secretion and correcting the disease.

Two kinds of practical problems must be overcome if gene therapy is to work: the CFTR gene must be delivered efficiently to the affected tissue and its expression must be regulated to achieve and maintain normal production of the protein. In most clinical studies to date, the normal CFTR gene has been introduced into patients by one of two means: either the CFTR is incorporated into the DNA of a virus called adenovirus T mixed with fat droplets called liposomes. The viral or liposomal preparation is sprayed as an aerosol into the nose or lungs of patients, who are then monitored for the correction of the chloride transport abnormality. Overall, however, progress has been slow in coming, though researchers and clinicians alike remain hopeful that gene therapy will eventually become a realistic therapeutic option for treatment of cystic fibrosis.

The writer is associate professor in botany, Anandamohan College, Kolkata