tiring address, reviewing the work of the society and its relation to the history of science movement. Dr. S. E. Morison, of Harvard University, will speak on the background of Colonial culture. Dr. C. A. Browne, of the U. S. Bureau of Chemistry, will have as a topic the history of industrial chemistry of the period. Dr. Lao G. Simons, of Hunter College, will speak on the mathematical knowledge in the Colonies. Dr. Henry R. Viets, of Boston, will speak on medicine in the Colonies. A number of other prominent speakers will also give papers, making thus a general symposium on Colonial culture.

The preliminary announcement of the Section on Engineering (M) will be a subject of special note to be published later.

The Section on Medical Sciences (N) will meet on December 27, 28, 29 and 30. On the first two days symposia on "Pneumonia," "Sociology and Medicine," "Tuberculosis," "Appendicitis" and "Typhoid Carriers" will be held. On December 29 there will be a joint meeting with the American College of Dentists. On December 30 the meeting will be given over to a joint session with the American Society of Parasitologists.

The Section on Agriculture (O) will hold a joint session with the American Society of Agronomy on December 28 under the auspices of the Northeastern Section of the Agronomy Society. The program has been arranged by Dr. T. E. Odland, Dr. M. H. Cubbon and Dr. M. F. Morgan, consisting of a symposium on "Field and Microchemical Methods for Determining Soil Deficiencies." Invitation papers will be presented by a number of men active in developing soil-testing technique.

The section will cooperate with Sections M and K in a symposium on "The Engineer and the Farm Problem" to be held on Saturday. The section will also meet in joint session with the American Society for Horticultural Science and the Agronomy Society on Saturday afternoon.

The American Society for Horticultural Science plans joint sessions with other organizations as follows: (1) with the American Society of Plant Physiologists on Friday morning, the program being devoted to physiological problems with horticultural plants; (2) with Section O at which the retiring president, Dr. J. H. Gourley, will be the principal speaker.

The Section on Education (Q) plans four sectional programs and a joint dinner with Section I. One of the Friday programs consists of a series of papers concerning recent investigations of the components of mental ability. Among others, Dr. E. L. Thorndike and Dr. Truman Kelley will participate. A second session on Friday will consist of reports of research from members of the section. One of the sessions on Saturday will be organized around the central theme, "The Measurement of Individual Growth or Progress," under the direction of Dr. Walter F. Dearborn. Another session will consider reports of research from members of the section.

The Gamma Alpha Graduate Scientific Fraternity will hold its annual council meeting for the consideration of fraternity business on Friday afternoon. On Saturday morning all members of the fraternity will meet for breakfast and informal reunion at the Statler Hotel.

Pi Gamma Mu will meet for the annual luncheon at the Hotel Statler at 12:30 P. M. on Saturday. National officers and several distinguished guests will speak.

Sigma Delta Epsilon plans to hold a business session open to members alone, and a breakfast open to all women in science, at which a guest speaker to be announced later will preside.

The program is unusually condensed, so that one finds an unfortunate number of conflicts. For this reason readjustments are being sought and if secured may change somewhat the arrangements outlined above. Those in charge of special programs are earnestly requested to send notice of any changes to the office of the permanent secretary at the earliest possible moment in order that the final program may be free from errors.

DEATH AS THE RESULT OF CHANGE OF LIVING MATTER WITHIN THE PLANT CELL[1]

By Dr. JEAN DUFRENOY

STATION DE PATHOLOGIE VÉGÉTALE, BRIVE, FRANCE

LIVING is moving. Life implies perpetual change of form, perpetual interchange between the living matter and the inert surroundings. Movement, which we generally associate with life in animals, generally fails to be evident to us in the case of plants.

Still, much of the knowledge acquired during the recent years on the nature of living matter, and its

[1] Invitation address at A Century of Progress Meeting of the American Association for the Advancement of Science, Chicago, June 28, 1933.

changes, as conductive to death, has been acquired through the study of the plant cell.

The plant cells range in size from the enormous sap cells of the orange fruits, several millimeters long, to the ultramicroscopic viruses, for which the name Borellia was recently suggested, and which diameter probably is smaller than one fifth of one thousandth of one millimeter.

Some plant cells, therefore, afford a most valuable material to work out the general problems of life and death.

Inert as any plant cell may look because it is generally encased in rigid cell wall, still the living matter within responds to stimuli readily, and that response may often be much easier to study under the microscope than the response of the animal cell.

Besides, some plant organs actually respond to stimuli by movements which are quite evident, even for the casual observer; such, for instance, are the movements of the leaf of some insectivorous plants after they have captured an insect, or those of sensitive leaves that fold to a sleeping position. Our knowledge of these remarkable manifestations of cell life dates back to 1875, when Darwin, with a precision and accuracy at which we must really marvel, considering the optical equipment he had to depend on at that time, described what he termed "aggregation phenomena," and which deVries years later interpreted as to the part of the cell involved, which he named "vacuoles."

Active cells of the glandular hairs covering the leaves of a carnivorous plant like Drosera, or active cells of pulvinus causing the movements of sensitive leaves, contain many small drops of a highly viscous red liquid. To these drops, containing a watery solution of anthocyan, deVries gave the name of vacuoles. Our own observations confirm that the more active the cell is, the more numerous and the smaller these drops are within the cell; in other words, the more intensive the life of the cell, the more finely divided is the watery vacuolar solution, and, as a result, the greater are the surfaces of contact between those tiny water drops (or so-called vacuoles) with the surrounding living matter, or the so-called cytoplasm.

Whenever the manifestations of activity become less intense in the cell, these numerous small vacuoles aggregate together into fewer lobe-shaped vacuoles, which eventually round up.

Few among the plant cells contain those colored vacuoles, but all cells contain vacuoles as made up of drops of water containing substances in the solution, within the cytoplasm. In most of the living cells, vacuoles are colorless within the colorless cytoplasm so that their boundary only can be made out under the microscope or the ultramicroscope as a light-breaking line.

Another way to demonstrate vacuoles within the living cell is to make use of some water-soluble dyes which, when made into dilute solutions, can enter the living cell and penetrate through the cytoplasm without staining, to accumulate within the vacuolar solution, and concentrate therein to the extent of developing an intense color. This vital staining of the vacuoles can be obtained in a moment by immersing a section of living tissue or a piece of epidermis stripped off a leaf into a solution of neutral red, or cresyl blue, or phenosafranin or alpha naphthylamine, made isotonic and isoionic to the vacuolar sap by adding minute quantities of salts.

We could also stain vacuoles in actively growing cells by germinating seeds in nutrient solutions to which vital dyes had been added. The presence of vacuoles is absolutely necessary to the life of any cell, from bacteria up to the higher plants and animals.

Any stimulus enhancing the activity of the life processes within the cell always causes the one large vacuole, or the few large vacuoles of the cell, to break down into a network of smaller vacuoles, and eventually into a multitude of tiny drops.

This general phenomenon becomes of great significance in interpreting morphologically the intensity of the life processes, of which the rate of respiration is a fairly good measure. Any moderate stimulus, not resulting soon in the death of the cell, results both in that exaggeration of respiration which is commonly spoken of as "fever" and in the division of the vacuoles of the affected cell. That both phenomena should be linked is to be expected in the light of Warburg's and DeVaux's recent theories to the effect that the life process as expressed by the equilibrium of concomitant chemical reactions is manifest mainly along the surface of the living substance "cytoplasm" and more especially at the contact surface between the cytoplasm and enclosed watery "vacuoles."

That interphase along which respiratory and other fundamental cell activities take place may be represented as a polarized monomolecular film.

To a certain limit a cell is the more active as the cytoplasm is made more spongy by a greater number of small vacuoles, since the interfacial contacts between cytoplasm and vacuoles are larger, and as the circulation of the cytoplasmic material brings about frequent and noticeable changes into the repartition of the vacuolar material.

Most adverse conditions inhibit the building up processes while they enhance the disintegrating, oxidative processes.

Moderate excitations seem to enhance first, at least locally, the respiratory activity, resulting in fever.

Living cytoplasm and its fundamentally active inclusions, "mitochondria" and "plastids," are built of

a homogeneous complex of proteins and lipoids (fats).

Factors which slowly interfere with the normal life of the cell cause that complex to be split into its constituents, so that we find lipoids separating out into fat globules, and insoluble proteins disintegrating into water soluble fractions which go into the vacuolar solution, to be partially used up for cell metabolism or translocated to meristematic cells in growing points.

But while the living cell can draw freely on its reserves of starch and sugars, it can use up only a small fraction of its proteins. Some of the proteins in the plastids seem to be mobile, being replaced if nutritional conditions become better after periods of stress, but the bulk of the plastids themselves and, to all appearance, the whole of mitochondria are still demonstrable in starved cells and in cells badly affected by virus diseases. While the cell as a "reservoir" can be depleted of most of the substances stored in metabolic products by starving under aseptic conditions, we can write as our conclusion the same lines as Professor H. S. Reed wrote a quarter of a century ago as the conclusion of his researches on nutritional deficiencies: "No case was found in which the absence of an essential element caused serious modifications in the form or structure of the strictly living part of the cell"; the living cell is an architectural complex where life is possible as long as the fundamental constituents each retain a certain physical structure, while all keep some definite interrelations.

The study of life and death should make use of the microscope, through which we can learn much concerning the arrangement of the constituents within the living cell, and of the wrecking down of these arrangements when the cell dies.

But we feel that the highest magnification available with the microscope fails to reveal what is the fundamental difference between the living and dead cell, though the proper killing methods of modern cytology preserve the finest details of the cell arrangements, changing only the staining properties of the various cell constituents. Therefore we must look further into the essential structure of the cell substance, and the ultramicroscope, following the microscope, helps us a step further on.

The Structure of the Living Cell

When a living cell is examined on the dark field of the ultramicroscope, neither the cytoplasmic material nor that of any of the cytoplasmic inclusions (mitochondria or plasts) nor even the vacuolar solution within the vacuoles show any illuminated particle; therefore the living parts of the cell, although chemically complex, are physically as homogenous as perfectly clean water, being also "optically empty." What is illuminated by the dark-field illumination is the boundary of the cytoplasm, where it is in contact with any of its inclusions. Therefore, mitochondria, plastids or vacuoles are brightly outlined by the dark-field illumination, although their inside, like that of the cytoplasm itself, containing no particle to deflect the light, is invisible under the ultramicroscope.

Death is concomitant with the breaking up of that perfectly homogeneous living complex into a less fine material; death therefore is the change of ultramicroscopically invisible into ultramicroscopically visible. Of course, since the ultramicroscope is unable to make out any structure out of the living matter, the microscope is more hopeless still, and even under the highest magnifications, the living cytoplasm looks like a perfectly transparent material. As we can not stain the living matter, we only know of it through its carrying along microscopically visible mitochondria, plastids and fat globules, as cytoplasmic currents are flowing around the "vacuoles" in the cell.

While the cell is living, the cytoplasm can not be stained, but a number of stains, the so-called "vital stains," can penetrate through the cytoplasm to accumulate into the vacuolar contents, where they develop a deep color. Even when their vacuoles are heavily stained the cells may keep all the activities of living cells: They grow, they multiply. But, as soon as the cell dies, the vacuolar solution breaks down into a number of vacuolar precipitates, while the cytoplasm breaks down into a granular stainable material. Death therefore is the change of the microscopically structureless living substance into a material built of microscopically visible parts.

The only parts which we can stain within the living cell are: (1) The watery solution in the vacuoles which absorb the vital dyes; (2) the mitochondria which Janus green stains green and dahlia stains violet within the unstained cytoplasm; (3) the oil droplets, within the cytoplasm or within the plastids, which can be stained blue by nascent indophenol blue. But indophenol blue is so toxic that it should be spoken of as a post-vital dye rather than a true vital dye. Indophenol blue stains the oil droplets in the dead cell as well as it stains them in the living; whereas vital dyes stain the vacuolar solution as such only as long as the cell is living. As soon as the cell dies, the vacuolar solution within undergoes such changes as it no longer absorbs vital dyes, while those same dyes which penetrated the living cytoplasm without staining it readily stain the dead cytoplasm and nucleus. These changes are perhaps the best criteria of the death of the cell. Death, therefore, is the change of homogeneous vitally stainable vacuolar

material into granular vacuolar precipitates as well as the change of homogeneous unstainable cytoplasm into readily stainable gel.

Death is most conveniently diagnosed through the altered behavior of the vacuolar solution towards vital dyes. A living cell having been immersed in a weak solution of neutral red shows the vacuolar contents purple red within the unstained cytoplasm. A mechanical shock or the penetration of a poison killing the cell will result in the vacuoles immediately losing their red color, while the cytoplasm, nucleus and cell walls stain a dull, brick-red color.

The Effect of Hypotonic Solutions on Living Cells

Since Hugo deVries made known the osmotic properties of the living cell, we know that cell turgor results from the presence of the molecules of dissolved substances in the vacuolar sap. It is a well-known fact also that (except of course for water plants) when a living cell is immersed into tap water, the water from without tends to rush into the vacuole more quickly than the watery solution tends to flow out; not only is water taken in by the vacuolar sap, but also by some hygrophilous material (phosphatides) of the cytoplasmic inclusions, and especially so by mitochondria. As tap water penetrates into a plant cell, the mitochondria and eventually the plastids are seen to swell into spheres, the contour of which gradually fades.

That "vesiculization" of mitochondria and plastids is the commonest result of lethal effects which cause death of the cell within a few minutes.

As contrasted with the effect of plain water or of hypotonic solutions which are less concentrated than the solution within the cell vacuoles the effect of too concentrated (hypertonic) solutions is less likely to be rapidly fatal to the cell.

Poisoning Cells with "Vital Stains"

Vital stains are those which can be absorbed from dilute solutions by living cells to be stored into the vacuolar sap. Vital stains are more or less poisonous to the living cells so that any tissue may survive in a certain concentration of a certain vital stain, whereas a higher concentration will be more rapidly fatal.

Tobacco seedlings grow well under aseptic conditions in sterile nutrient media containing as much as 3 mg neutral red per liter. Higher concentrations result in the killing of meristematic cells at the root tip and in the inhibition of root growth, although the seedling, as a whole, survives.

It is therefore possible to poison certain cells within a tissue, without poisoning the bulk of the tissue.

It should even be possible to poison certain parts of a living cell without poisoning it whole, or at least it seems possible, through the effect of a proper concentration of vital stain, to obtain a local alteration of cytoplasm before the cell dies.

Sudden Death "Fixing the Cell" *vs.* Long Agony Altering Slowly the Cell to Death

Cells may die from a great many different causes, but the ultimate results in all cases are fundamentally alike: death is concomitant of the change from optically homogeneous living cytoplasm into a multitude of ultramicroscopic or even microscopic particles.

Sudden death only alters the ultramicroscopic structure of cytoplasm and of its inclusions, so that, as watched under the microscope, sudden death changes neither the general contour of cytoplasm (no shrinking) nor the shape of the nucleus, of the plastids, mitochondria or fat globules or of the vacuoles.

The staining reactions, however, are reverted so that the cytoplasm, which did not stain in the living, stains readily after death, while the vacuolar material, which absorbed readily vital dyes in the living, can no longer be stained as a whole after death (though, of course, the constituents, precipitated out of this vacuolar solution at death, can be microchemically detected by their specific reactions). Therefore, the picture of a cell stained after sudden death can be considered to be the negative of which the picture of vitally stained cell is the positive, and the photomicrograph of a cell stained by vital dyes could be superimposed to the photomicrograph of the same cell stained after sudden death (meaning a death such as is obtained by the killing mixtures which mitochondrial technique makes use of). On the contrary, great changes are noticeable in the picture of a cell which died of slow death, such as of a cell treated with fat solvents such as acetic acid or alcohol containing "killing fluids" or even more of a cell dying through the effect of imbibing water from dilute solutions or reversely of a cell dying from being deprived of water by bathing into concentrated salt solutions, and still more of a cell starved to death.

Local Death in the Cell

The living cytoplasm does not stain as such: it shows neither the staining reactions of lipoids nor those of proteins, although made up of a complex of both. Unmasking its lipoid and protein constituents is the result of the splitting of the cytoplasmic complex when the death process slowly advances. Such a disintegration generally is local and affects a certain area where the cytoplasm is honeycombed by a number of small vacuoles. As the lipoprotein complex breaks apart within the cytoplasm, water soluble

peptides go into the vacuolar solution, while lipoids are set free as fat droplets within the disintegrating cytoplasm, resulting in peculiar patterns which have been named "vacuolated bodies" when they were observed within cells affected by mosaic, but which resembled those in cells where proteolytic activity is locally enhanced as the result of parasitic action, or of starvation or malnutrition.

Excessive local proteolysis within the cell is made evident cytologically through the shredding of cytoplasm into a number of strands (where mitochondria and fat globules are particularly numerous), surrounding so many small vacuoles. The local exaggeration of surface contents between cytoplasm and vacuole must enhance the processes of oxidation.

It is a well-known fact that starvation, among other factors, results in excessive oxidation of affected cells, making available energy for the remobilization of materials to be translocated away towards meristematic tissues.

Differentiated cells indeed may endure starvation or malnutrition for a while, using up part of the lipoproteids from their plastids, and even part of the lipoproteid from at least a certain cytoplasmic territory, not only to provide for their own metabolism, but even to supply material to the meristematic cells of buds near by; meristematic cells indeed must be provided for, or they die; they grow and divide into more meristematic cells, building a perfectly normal structure by using the latest trace of available material, after which they abruptly collapse. Whereas the change of living matter into non-living should be studied primarily where sudden death does not permit of any premortal rearrangement of the cell material, still much can be learned from the study of the gradual disturbance of the cell metabolism, such as results from systemic infection or deficiency nutritional diseases.

Most valuable information was obtained from the study of the affection of citrus known as mottle leaf, which has been shown by Professor Reed never to cause necrosis, as it does not directly kill affected cells; those cells, however, may show a definite lagging in their evolution from bud time to leaf time. Affected leaves are generally yellow between the veins, along which some green color generally develops. Transverse or tangential sections through those yellow interveinal areas show that the palisade tissue, instead of being built of rows of narrow cylindrical cells, each with numerous large green plastids, is made up of a crowd of isodiametric cells, twice as wide as the normal, but none normally elongated. This inhibition of histological differentiation is concomitant with an inhibition of physiological differentiation: affected cells never develop any marked photosynthetic efficiency; this, in turn, is linked with inhibition of cytological differentiation, mitochondria being generally unable to differentiate into plastids.

Death of the Cell as Caused by Deficiency of One Essential Nutrient

Dr. Haas, at the department of physiology of the Citrus Experiment Station, Riverside, California, could grow successfully Citrus in liquid media: roots develop normally in Hoagland's complete solution, apical growth resulting from the normal activity of the three dome-shaped layers of initial cells at root tip.

When potassium is omitted, however, roots soon cease to elongate, since groups of meristematic cells collapse at the root tip; the death of these meristematic cells may release enough potassium to keep meristematic cells (located deeper inside the root tissue) proliferating, although they are unable to differentiate, so that the root tip, instead of elongating, by pushing the root-cap as it grows, swells into a club-shaped mass of small embryonic cells.

These cells keep dividing for a while, forming new living cells with a normal nucleus and normal mitochondria. Then, some marginal cells may show a tendency for mitochondria to clump together in a part of the cytoplasm which becomes more finely vacuolated. These mitochondria become shorter and thicker; their substance, as well as that of the cytoplasmic strands along which they line up, take stains more and more strongly after fixation, so that staining eventually demonstrates within the cell a certain vacuolated part of the cytoplasm as a deeply stained "vacuolated body" (which is homologous to the "vacuolated body" of many virus-affected cells). One whole cell soon collapses and dies. Alternative collapsing of embryonic cells, with subsequent renewal of growth following remobilization of material from dead cells, is specially evident in roots from solution where potassium is lacking but magnesium is available; some cortical cells towards the root tip get loose through pectinization of the middle lamella of their cell wall and eventually die, making material available for deeper meristematic cells to outgrow that degenerated section by budding out from its set of initials. In that case, the inhibition of apical growth is periodic, and results in a piling up of successive periods of activity separated by periods of degeneration, instead of the usual situation of development of lateral rootlets following inhibition of terminal growth. Starvation, or lack of any essential element, generally results in a bushy type of twig growth and excessive branching of roots; meristematic cells in bud and root tips die as soon as they have used up all the available food supply; their death releases the

material which they had been using for the building of the living substance: when remobilized material becomes available, lateral buds and initials of lateral rootlets start developing, soon to die at their tips, as the available food is used up. And as the whole of the used material can not be remobilized, the supply available for each successive beginning of development is less and less, so that the successive lateral buds develop into shorter and shorter shoots, bearing smaller and smaller leaves, before they die.

But while the shoots or leaves may be dwarfed, the constitution of each embryonic cell within is normal as long as, food being available, they survive, after which they abruptly die.

CYTOLOGY OF STARVED CELLS

Each physiological process in the cell is concomitant with an antagonist process: At the same time when a synthetic process builds up complex molecules, a reverse analytic process disintegrates complex molecules into simpler, translocable constituents.

Each process may be especially enhanced by exciting or slowed down by inhibitory agents, but, on the whole, analytic processes keep on going under conditions which prove adverse to synthetic processes.

While chloroplastids build starch out of the CO_2 from the atmosphere when receiving proper illumination, starch is being hydrolyzed into sugar. Under normal conditions the building of starch (amylogenese) is the faster process during the day. Amylolysis is the faster process during the night.

But starch is dependent on many factors besides light and CO_2. Lacking some of the necessary conditions, the plants build starch no more, while starch previously formed disappears as it is being hydrolyzed into soluble glucids to be used up by other cells.

Under normal conditions starch grains may dwindle during the night from the plastid from which they are being translocated.

In starved cells, starch grains seem to be slowly hydrolyzed from the periphery inwards, so that the central part of each grain may give the color reaction of starch when treated by the solution of iodine and potassium iodide, while the periphery of the grain gives a very faint or no color reaction, so that the stained central part of each starch grain is surrounded by a clear unstained halo. As this hydrolysis proceeds, the plastid seems to be hollowed out, and assumes a sieve-like appearance. At the same time, the lipoproteidic material (of which the plastid itself is normally built) may swell and disintegrate into its two main constituents; lipids and proteins. In fact, a starved cell draws on the plastids' material when proteins have to be remobilized within the cell. Proteins are primarily obtained through the disintegration of the lipoproteidic complexes of which the plastid is made up. As these lipoproteidic complexes disintegrate, water-soluble peptides are made available, and these go into solution in the vacuolar sap or move out of the cell, while the fatty part of the complex separates out in the form of so many fat droplets within the plastid. Moderate translocation of proteins leaves the plastid able to make up for that loss whenever conditions become favorable again. But if the disintegration proceeds too far, the plastid is irremediably impaired and "dead" to the cell for all purposes. Next to the lipoproteins in the plastid the cytoplasm depends on the lipoproteins which build up its own living substance: as starving is more severe, part of the cytoplasm itself disintegrates into proteins and fat globules. Not all of the proteins, however, seem to be available for new constructive processes. Part of it crystallizes out into cubic proteidic crystals in the very place where disintegration processes occur. Not only do certain cells in a tissue undergo a severe disintegrating process to make nutrients available to neighboring cells, but in one given cell, part of the cytoplasm may disintegrate, while the remaining parts of the cytoplasm keep living.

Disintegrating parts of starving cells are easily made out under the microscope because of the spongy appearance of involved cytoplasm, because of its higher refractory power as well as because it shows numerous fat globules and proteid crystals. After the cell has been properly fixed by cytological methods the disintegrated part of the cytoplasm can be made out because it stains more deeply with most stains, and because it shows the microchemical reaction for proteins.

STARVING A PLANT CELL TO DEATH

Cells of reserve tissues or cells from aged leaves, roots or twigs are normally deprived of some of their reserves for the benefit of meristematic tissues, as the plant grows. Senile cells, from which carbohydrates, proteins and even minerals have been translocated away, are easily infected by microorganisms and can hardly be expected to survive long under ordinary conditions. Therefore, we have been studying the impoverished cells of seedlings grown in sterile media, under aseptic conditions, when no microorganism could interfere with the long agony brought about through the only effect of starvation. Under these conditions the discriminating line between life and death may become more difficult than ever to draw, as some differentiated cells survive gradual depletion, for months or even years, while meristematic cells collapse in a very short time.

In any cell that dies slowly, the plastids are the

first part to be evidently affected. They are the reservoir which the cell draws upon for carbohydrates and proteids. Depleted plastids have a tendency to agglutinate as the neighboring cytoplasm assumes the structure of a network around a number of small vacuoles, which contents become richer in water soluble peptides, as first the plastids, and then the cytoplasm itself, undergo proteolysis.

Death may abruptly affect the cell as a whole, "fixing" the various cell constituents in the place they occupied at the moment death occurred; or death may affect the cell slowly during which agony the constituents have time to undergo changes. Premortal changes are fundamentally alike, whatever the cause of death: their results mainly depend on how long the cell endured.

Lethal factors which act very slowly (malnutrition of adult cells, infection by endophytes or viruses), will first produce increased respiration in the affected cell when part at least of the cytoplasm will become spongy. Proteolytic processes, being enhanced, result in the formation of more water soluble peptides which may accumulate in the vacuolar sap. Finally, large vacuoles are partitioned off into a number of smaller ones by the extension and branching of cytoplasmic strands.

CONCLUSION

Death is the change from the clearly visible harmonious arrangement of homogeneous living parts of the cell into crowding of microscopically heterogeneous material.

The living cell is a harmonious building, coordinating a number of homogeneous materials, the contour of which can be made out under the microscope or the ultramicroscope, making the architectural design of the living cell observable. Killing the cell suddenly by proper cytological technique preserves the architectural disposition of the cell materials, making those materials themselves visible through ultramicroscopical changes of structure admitting of staining. Slow death of the cell preserves neither the architectural disposition of the cell material nor even its microscopical structure, as premortal changes are mainly concerned in the splitting of the homogeneous unstainable living complex into a coarse, granular collection of its constituents.

SCIENTIFIC EVENTS

REFORESTATION BY THE TENNESSEE VALLEY AUTHORITY

TEN thousand bushels of pine cones and other seeds are being harvested by the members of the Civilian Conservation Corps for use in reforestation work in the Tennessee Valley, according to an announcement made by Robert Fechner, director of emergency conservation work. The seeds are being gathered largely in Virginia, West Virginia, North Carolina and Arkansas. The program calls for planting the seeds in nurseries this winter. Later the seedlings will be transplanted in the areas to be reforested by the Tennessee Valley Authority.

Five thousand members of the Civilian Conservation Corps have been assigned to Tennessee Valley work by Director Fechner. These men will be distributed among twenty camps in Tennessee and five in Alabama. One of the major tasks assigned will be that of combatting soil erosion through tree planting, this being part of a general erosion control program to be carried on in the central, southern and western states.

To obtain the amount of seed necessary for the reforestation of the valley, the collection has been apportioned among the four national forests in the states specified on a quota basis. The total harvest will include 600 bushels of yellow poplar seed pods, 4,000 bushels of short-leaf pine, 2,600 bushels of Virginia pine, 2,900 bushels of black locust pods, all capable, under proper care, of producing 2,000,000 yellow poplar, 10,000,000 short-leaf, 8,000,000 Virginia pine and 6,000,000 black locust tree seedlings. Pitch pine cones, of which there is a limited supply in the forests of this region, will be collected in lots of 25 bushels or more.

As soon as the ripe cones are picked, they are shipped to the three southern nurseries of the U. S. Forest Service for drying and seed extraction. These nurseries are at Parsons, West Virginia; Russellville, Arkansas, and Catahoula, Louisiana, which is a new federal nursery. The prepared seeds are forwarded to the Tennessee Valley Authority and probably will be planted in two new nurseries which that organization is planning to establish in the Tennessee Valley.

Although it is often easier to pick cones from trees felled in lumbering operations, most of this year's collections will have to be picked from standing timber. Where it is necessary to climb high trees or ladders the Forest Service has ordered that safety belts be supplied. State and private land camps in the vicinity of the national forests are to be enlisted with those on the national forests during the harvest.

AN AMERICAN DECIMAL-METRIC CODE

PROPOSING that the United States shall make general use of metric weights and measures, an American Decimal-Metric Code has been drafted and urged for adoption, to assist the administration in effecting eco-