

DESTRUCTION PROCESSES IN THE CELLULAR MEDIUM OF A PLANT
- THEORETICAL APPROACH

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A b s t r a c t. A theoretical model of destruction of a material containing elastic cell wall surrounding a content consisting of liquid and gas is presented. The results show that a damage of the cellular plant material is the result of breaking a cell wall under the outer pressure and crack propagation as the consequence of fissure.

K e y w o r d s: damage of the cellular body, crack propagation in the plant tissue

INTRODUCTION

Physical properties, and even more so, mechanical features of agricultural plant materials have been in the centre of scientific interest for years. There are hundreds of papers dealing with those problems. But there is still a number of controversial theories on, and interpretations of the mechanical damage of plant tissue. It is the reason why we would like to continue the above discussion on the pages of our journal the *International Agrophysics*.

Studies on the mechanical properties of agricultural materials were started as empirical research. It is understandable since a growing level of mechanisation in farming and food processing required knowledge indispensable for designers and constructors of more and more aggressive machines that may generate substantial losses of up to several percent of raw material and yield due to mechanical damage. The

problem is so important that in the laboratories of multinational manufacturers of agricultural equipment and machinery there are experimentally worked out catalogues of the physical properties of biological materials or lists of requirements on that sort of studies that should proceed design work. Standards that are obligatory in such laboratories as for example John Deere, cover several types of research studies. It is equally obvious that empirical studies were often followed by attempts at theoretical interpretations and generation of mathematical models of the processes that take place in biological materials [1,2,4,6,8,10,11,14,15]. It followed not only from the researchers' interest in the phenomena that had not been fully recognised yet, but also from the fact that a good theoretical model would allow for avoiding numerous, often costly and time consuming, empirical studies since biological cycles are usually long. However, enormous difficulties were encountered while working on these theories.

The proposed theoretical models have to fulfil the condition of metatheoretical compatibility [16]. It means that the model must be probable, logically correct, possible to test empirically and fulfil the condition of intertheoretical compatibility, i.e., must agree with the

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previously checked and accepted theories. Attempts at adjusting numerous, already existing and well developed theories originating from the mechanics of the continuous media and rheological models for the description of, generally speaking, relations between stresses and strains in the materials of biological origin were undertaken.

However, it is not difficult to notice that the degree of structural complexity of these materials that is far higher than in the engineering constructions, excludes execution of the above mentioned compatibility conditions. Let us look at this problem more closely.

All, or almost all biological plant materials that are agriculturally produced have cellular structure. Two types of these materials can be distinguished.

First group includes those materials that are filled with water, gas or other liquids during harvest and any further processes. The group contains root plants, fruit, berries and many others. The same group includes also ripe seeds of oil plants such as: soya, sunflower and rape that have cells filled to a high percentage with liquid fat.

The other group are materials in which biological processes are more advanced and that are the media consisting of cells or cell contents (e.g., starch grains) totally devoid or to a large extent devoid of water in which life processes have almost totally been finished. These are, for example, cereal grains, ripe seeds of various plants and other materials like that.

Materials belonging to these two groups are especially susceptible to mechanical damage during harvest and processing which results, as has already been said, in enormous losses. These are not only direct losses that lower quantitative classification of the material, but also some damages that are initially invisible, often limited to some small inner fractures that are, however, the reasons for further infections and biochemical changes resulting in material damage during storage. These phenomena are widely known and often described in literature that presents the results of empirical data [8,12,13,15]

It is not difficult to notice that the attempts at the theoretical interpretation coming from the studies on the materials used in the design engineering practice, for which the mechanics of continuous media can be applied or any discussions on the damages resulting from the mechanisms of material fatigue on the microscopic level, cannot fulfil the conditions ensuring correct interpretation of phenomena taking place in the biological materials. In most of the cases researchers applying the above method limited their approach to treating biological materials as continuous, isotropic visco-elastic-plastic media and ascribed various rheological models to them, i.e., they assumed that they can be characterised by some definite relaxation and creeping.

There exists substantial literature [3,5,7,9, 16] presenting results of empirical and theoretical studies on damaging centres with discreet structure and mechanisms of generating damage. Generally speaking, a term "continuous mechanics of damage" is used for them. It is worth mentioning, however, that the above papers describe, as has already been mentioned, mainly construction materials with discreet structure in the microscopic scale. They also deal, first of all, with damages resulting from material fatigue. However, the structure of biological materials is of varied character of discontinuity with relatively big sizes. We do not deal with the phenomenon of fatigue damage accumulation, either. The models that describe biological materials have to take into account factors such as: size and structure of cells, actual material constants and constitutional relations between stresses and relaxations in the materials that contain structural elements with the forms and states of matter decidedly different from construction materials.

For that reason the models proposed so far are based on incorrect assumptions as they do not take into consideration discreet, relatively macroscopic due to the cell size, stochastic and highly metamorphic structure of the material resulting from its anatomic variability.

However, even though the above remarks are related to the two groups of materials described above, the course of processes related to deformation and damage in both groups are considerably different. For that reason they should be treated separately. In the present work we concentrate only on the first group in which the cells contain liquid and gas.

DEFORMATION PROCESSES

The argument presented below is related, as has already been mentioned, to the materials from the first group that contains cells with elastic cell walls surrounding the content. Generally speaking and without entering into any details of the anatomic structure of their protoplast the content is made of liquid and gas. The cell wall is not tight but contains cytoplasmatic threads that join protoplasts. The threads are called plasmodesmas. They make the protoplasmatic cell system continuous as in the very centre of the plasmodesma there is a duct that is an extension of the intraplasmatic net of both neighbouring protoplasts. Similar connections appear between the cell interior, and intercellular spaces that are formed in the places where central plates joining the cells were dissolved and the cells moved away from one another. The intercellular spaces are joined to the outer space and form routs for gas and liquid penetration. The following general character of deformation can be assumed: under the influence of outer forces acting at the object filtration processes caused by the stress gradients appearing in its whole volume. They consist in the transfer of the cell content in relation to the pressure gradient between the neighbouring cells or from the cell into the intercellular spaces, and from there outwards of the material. Filtration velocity depends on the pressure gradient and permeability of the cell wall, and hence on the geometry of the cell, its plasmodesmas and liquid-gaseous viscosity of the cell content. The following conditions are fulfilled at the same time:

- Due to the fact that the cells are filled with liquid and gas, pressure in the cell is hydrostatic.

- Due to very small cross-sections and small velocity levels all the flow is of the laminar character.

- Due to its character cell membrane acts as a linear-elastic object till its damage (breakage).

The processes presented above allow for the generation of a qualitative picture of deformation that results from the process of laminar filtration and in principal depends on the increase of velocity stress. If the velocity is small enough, filtration processes lead to a gradual removal of the cell contents outside with relatively small elastic deformations. Then stresses in these walls do not reach threshold values that would cause their damage. The volume of all the cells decreases uniformly, hence a volumetric deformation of the whole centre takes place without damaging the cells. However, with the increase in the velocity of increase of the stress gradients, filtration processes are too slow and the pressure inside the cell increases up to the threshold value of the cell wall that gets broken and the whole content gets of the material subjected to loading through the intercellular spaces.

The maximum stress in the cell wall caused by the pressure inside the cell can change according to the cell shape (in the border cases it is a sphere or a cylinder) within the following limits:

$$\frac{p\rho}{g} \geq \sigma \geq \frac{p\rho}{2g} \quad (1)$$

where: σ - stress in the cell wall, ρ - radius of the cell wall curvature, g - thickness of the cell wall, p - pressure inside the cell.

Hence, assuming a constant level of the cell wall tensile strength, its damage depends on the time derivative of the pressure gradient, on the cell shape, thickness of the cell wall and conditions of filtration.

The cell that was broken and emptied of its contents is flattened as a result of outer pressure. A flat discontinuity in the form of a fissure is formed in this place with very small curve radii and that increases stresses accordingly.

A broken cell remains surrounded by undamaged cells that form the front part of the crack. Pressure level in the cells makes their energy quite intensive (which is understood as the product of the energy contained in the element of volume and the distance between this element and the peak of the crack). This situation favours propagation of the crack, i.e., it creates conditions for further material breaking. In this case Mott's solution describing velocity of the crack can be applied with a certain degree of approximation. It seems, though, that in the first approximation, the problem can be simplified as much as possible. The more so that we are not interested in the velocity of crack propagation as it is almost two orders of magnitude bigger than the changes in the pressure gradient. We are interested in answering the question whether propagation of the crack will take place at all, how far the crack will be propagated, and whether energetic conditions will not result in bifurcation.

symmetrical in relation to the long axis and deformation in the direction of the z axis is not limited. (*Limitation of the deformation in the direction of the z axis would limit the problem to the plane state of strain, which is worth noting since most of the laboratory studies have been carried out with the sample placed in a container with stable side walls when the plane state of strain exist and breaking requires less energy, i.e., takes place with smaller inner stresses*). In the case discussed here the crack can be presented as an ellipse with 2a and 2b axes, 2a axis being longer. The crack is located in the space subjected to outer stresses σ_1 and σ_2 , respectively. We assume that these stresses are exclusively compression ones as these type of stresses appear almost exclusively with real loading. The crack is located inside the material and its longer axis is inclined in relation to the bigger main stress σ_1 by the angle β . Figure 1 presents distribution of normal stresses σ_x and σ_y and tangential stresses $\tau_{xy} = \tau_{yx}$.

SIMPLIFIED EQUATION OF THE STRESS CONDITION

Let us consider the case of a crack resulting from the process described above for simplification treating it as a flat stress state which is justified by the assumption that the cell shape is

Assuming that $\sigma_1 > \sigma_3$ we arrive at:

$$2\sigma_y = (\sigma_1 + \sigma_3) - (\sigma_1 - \sigma_3) \cos 2\beta \quad (2)$$

and

$$2\tau_{xy} = (\sigma_1 - \sigma_3) \sin 2\beta. \quad (3)$$

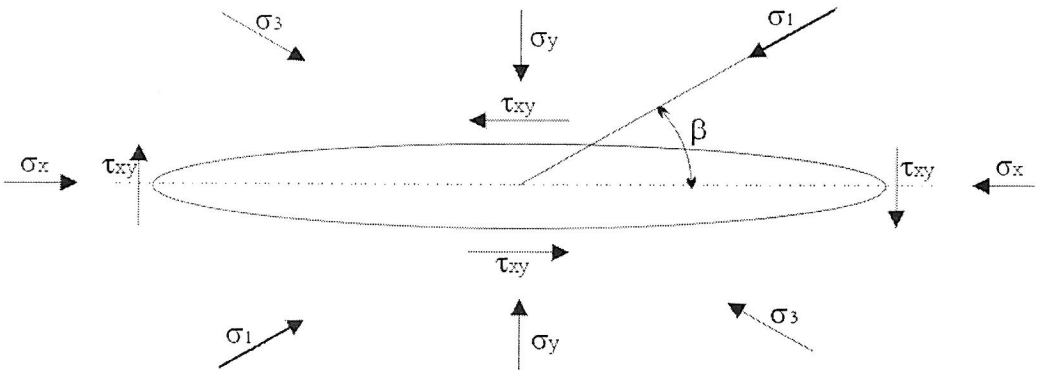


Fig. 1. A crack located inside the space subjected to outer compression stresses σ_1 and σ_3 , where: β - angle of inclination of the longer axis to bigger main stress, σ_x, σ_y - normal stresses, $\tau_{xy} = \tau_{yx}$ - tangential stresses.

Stress σ_x has little influence on the crack propagation and can be neglected. Crack parameters have been shown in Fig. 2, where σ_a is a tensile stress tangential to the crack surface at point A. If we denote: $m = \frac{b}{a}$, then it follows from the simple geometrical relations that $\text{tg } \theta = m \text{ tg } \alpha$. If we neglect the value σ_x that is of no importance for the crack propagation, the tangential stress σ_a at the point A on the ellipse is equal to:

$$\sigma_a = \frac{\sigma_y \left[m(m+2) \cos^2 \alpha - \sin^2 \alpha \right] - \frac{\tau_{xy} 2(1+m^2) \sin \alpha}{m^2 \cos^2 \alpha + \sin^2 \alpha}}{m^2 \cos^2 \alpha + \sin^2 \alpha} \quad (4)$$

The crack becomes flat under the pressure of the neighbouring undamaged cells. In this case m is most often very small. The biggest stress σ_a will appear at the small values of θ . When $\alpha \rightarrow 0$; $\sin \alpha \approx \alpha$; $\cos \alpha \approx 1$.

If the small values are neglected in the numerator of the second and higher magnitude, we arrive at:

$$\sigma_a = \frac{2(m\sigma_y - \tau_{xy}\alpha)}{m^2 + \alpha^2} \quad (5)$$

Pressure gradients are not big as a rule due to the fact that the cells are filled with liquid and gas. Hence, τ_{xy} takes on small values. However, due to very small values of the denominator, the σ_a value can exceed the value of outer stresses many times. In this situation the walls of the neighbouring cells will always be subjected not only to the stresses resulting from the pressure gradients, but also to the stresses caused by the appearance of the fissure. That in turn may result in breaking further cells and fissure propagation up to the outer wall of the studied object.

The sequence of cell breaking next to the fissure depends first of all on their size. Bigger cells, in which tensile stresses are higher according to the Eq. (1), will be more susceptible to damage in the first place. Assuming that the gradient of pressure is approximately constant in the vicinity of the crack, and assuming that the resistance of the cell walls in the anatomically uniform zone is constant (it follows from

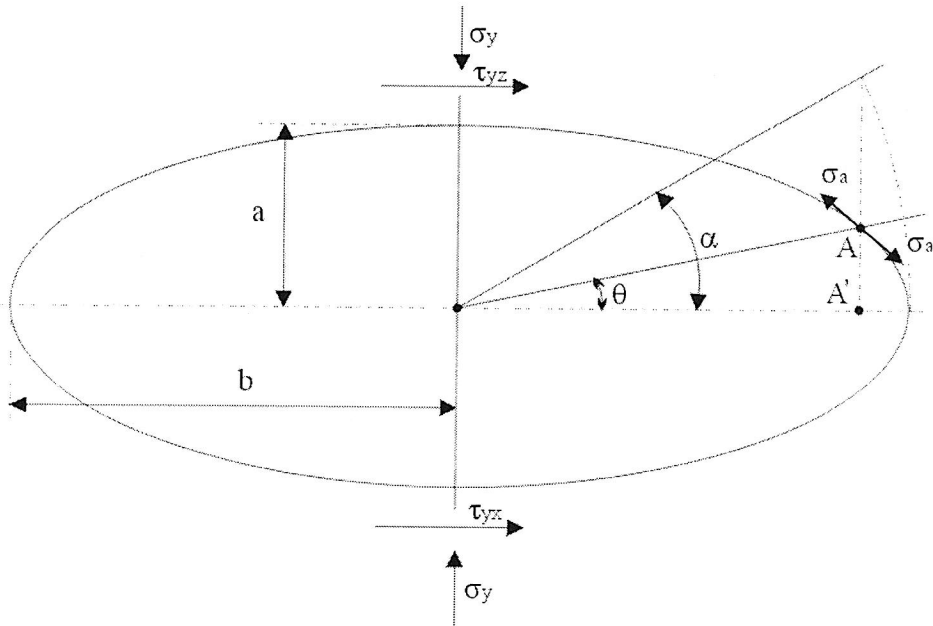


Fig. 2. Parameters of the crack, where: σ_a – tensile stress tangential to the crack surface at point A with respective angles θ , and α ; a, b – axis of the crack, longer and shorter, respectively; another symbols according to Fig. 1.

the microscopic studies that the thickness of cell walls is not correlated with the cell size), it can be stated that the process of cell breaking depends to a large extent on the distribution of the probability of their sizes. It can also happen, that the crack will reach a group of cells strong enough to stop its further propagation.

It should be stressed that no real biological object is anatomically homogenous in the macroscopic scale. Hence, different ways of crack propagation are possible. If the cell structure of the outer layer is weaker than the inner layer, the fissure can be formed on the surface of the object (e.g., potato bulb, root or fruit) and reach inside. If the structure of the outer layer is more resistant (which generally is the case), the fissures appear inside the object and do not appear outside at all. In the border cases crack propagation causes partition of the object into two parts.

CONCLUSIONS

So far no data allowing for the determination of probability distribution of cell sizes, geometry of cell walls that determine conditions of filtration, distribution of the material constant values that characterise cell walls and contents are available, and they are the condition for the verification of the theoretical model of material damage. However, even though data is not available, it seems obvious that in the material with cellular structure, i.e., discrete structure, a typical damage is crack formation that leads to breaking, and not elastic or visco-elastic deformations of the medium as has been suggested in the papers released on the above subject so far.

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