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A Particle Based Model to Simulate Microscale Morphological Changes of Plant Tissues during Drying

H.C.P. Karunasena, W. Senadeera, R.J. Brown and Y.T. Gu*

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1. Introduction

Drying is one of the most popular and generally used food preservation techniques which is currently used on about 20% of the world’s perishable crops. Since plant food materials can originally contain water even up to 90% by weight, moisture removal is the key driving factor which causes food preservation during drying. As a result of such extensive moisture removal from the tissue structures of these food materials, they undergo excessive deformations which lead to significant changes of numerous food properties.
(FDM), which are primarily developed for basic cell micro-
mechanical behaviour studies rather than drying. Recently, a
FEM based microscale tissue drying model is reported which
couples water transport phenomenon and cellular deformations.

In this particular model, there are several key limitations such as
the limited moisture content range that the model can simulate
(only 30% of normalized moisture content reduction) and
inability to represent the wrinkling effects that the actual cell
walls undergo during drying. Further, it appears that their main
focus is on the turgor loss based cellular deformations rather than
the moisture content based deformations, where the latter one is
actually more critical to industrial drying applications. Also the
hybrid numerical simulation approach they have involved is fairly
complicated to implement. In addition to such models on drying,
there are several vertex cell models reported in literature which
are specifically developed to study division and reproduction of
cells during tissue development. Also, there are gel material
models used to simulate bulk level deformations of plant leaves
during dehydration.

However, considering the technical challenges and various
limitations, these models have limited applicability in the scope
of this research. Such fundamental shortcomings are: poor
representation of multiphase phenomena existing in plant
materials consisting of liquid, solid and gas phases, use of over-
simplified continuum approximations for the tissue materials by
neglecting the discrete nature of the cellular structure and its
microscale mechanisms, inability to model the excessive cellular
level deformations (cell wall wrinkling in particular) at excessive
moisture content reductions, and limited flexibility to model the
multiscale effects of plant material deformations during drying.
These shortcomings need to be well addressed in order to get a
clearer understanding on the underlying physics involved in real
industrial drying processes. When considering these challenges of
conventional grid-based modelling approaches, meshfree
methods seem to be more applicable since they fundamentally do
not use any interconnected grids as in case of FEM and FDM,
which are entirely based on grids.

When considering meshfree methods, Smoothed Particle
Hydrodynamics (SPH) is a popular particle-based technique,
which was originally developed for astrophysical applications. The
technique discretises a given problem domain as a set of non-
interconnected particles (without any mesh generation), where
the particles own system properties which can evolve with time. SPH
is also particularly adaptive such that the basic formulations can
easily be combined with new physics and mechanics in order to
solve different problems of interest. Using these capabilities,
SPH has been successfully used for plant cell and tissue models
recently by coupling with a Discrete Element Method (DEM) to
simulate compression, tension, shear of fresh cells and tissues
and even cell breakage. We have further improved the
modelling technique in a way it can be used to simulate dried
plant cells. In those models we mainly focused on two-
dimensional (2-D) single cells and in this work we present how a
2-D tissue structure can be developed with the use of such single
cell models along with several improvements, in order to simulate
morphological changes of tissues during drying. Also, we believe
that, these proposed methods and findings will contribute to the
development of meshfree methods itself and their applications
towards developing novel modelling techniques particularly
applicable to critical fields such as food drying.

This paper begins with the modelling concepts and
formulations used in a single cell model. Then, we discuss how the
tissue model can be developed using such individual cells.
Next, the approach used to simulate tissue dryness states is
presented. Then the experimental findings on dried tissues are
compared with simulation results for validation of the model and
several other simulations are provided along with a sensitivity
analysis of several model parameters. Finally, we present key
insights drawn from the study along with prospective future
improvements.

2. The particle based model

2.1 2-D representation of plant cells in a tissue

A plant tissue was approximated to an aggregate of cylindrical
shaped cells as shown in Fig. 1(a), where the top surfaces were
taken as a 2-D model representing each cell. To facilitate this
approximation, axial deformations of each cell in the Z direction
were assumed to be uniform and the Z directional velocity
components and plane stresses of the top and bottom surfaces
(in the XY plane) were neglected. Accordingly, as shown in Fig.
1(b), each of the individual cells were modelled assuming two
basic model components: cell fluid and cell wall such that the
fluid hydrodynamic pressure is counter balanced by the cell wall
tension as observed in real plant cells. Following a SPH-DEM based fresh cell modelling approach proposed by
some researchers, the cell fluid was treated as a low-Reynolds
number incompressible viscous fluid and modelled with SPH.
Further, the cell wall was modelled using a DEM Considering the
nature of the solid-dominated fibrous cell wall structure. A
particle scheme was used to represent both the cell fluid and the
cell wall and governing equations were applied to these particles.
In addition to those basic concepts, we further improved both the
cell wall model (see Fig. 2) and cell fluid model (see Fig. 3) in
our previous works, so that the single cell model could be
used to simulate dried conditions. The sections below summarise
these schemes and concepts along with the new improvements
done in this work.

2.2 DEM modelling of the cell wall

The cell wall was assumed to be a visco-elastic solid material and
a Neo-Hookean elastic model was used with a supplementary
viscous term. Several additional features were introduced by us
to improve the model performance and to account for drying
related physical changes. As presented in Fig. 2, the cell wall
model accounts for seven types of force interactions: cell wall
stiff forces ($F_e$), wall damping forces ($F_d$), wall-fluid repulsion
forces ($F_F$), non-bonded wall-wall repulsion forces ($F_{ww}$),
wall-fluid attraction forces ($F_a$), forces due to bending stiffness of
the cell wall ($F_b$) and forces to produce contractions of the cell wall
during drying ($F_f$). Thereby, the total force ($F_k$) on any wall
particle is derived using these force interactions as:
Fig. 1 2-D representation of a plant tissue: (a) A plant tissue is approximated to an aggregate of individual cylindrical cells. (b) Top surface of each cylindrical cell is taken as a 2-D representation of the whole cell which consists of two basic components: cell fluid and cell wall. (c) The problem domain is discretized and represented as two sets of particles: SPH particles for the cell fluid and DEM particles for the cell wall. (d) Individual elements of the discretized cell wall.

Fig. 2 Force interactions used in the DEM-based cell wall model: (a) Stiff forces. (b) Damping forces. (c) Wall-fluid repulsion forces. (d) Non-bonded wall-wall repulsion forces. (e) Wall-fluid attraction forces. (f) Wall bending stiff forces. (g) Wall contraction forces. (i: fluid particles; j, k & l: wall particles)

\[ F_k = F_{kj}^s + F_{kj}^d + F_{kj}^f + F_{kl}^w + F_{kl}^c + F_{kj}^a + F_{kj}^b, \]  

where: \( i \) is any neighbouring fluid particle, \( j \) is any bonded wall particle and \( l \) is any non-bonded wall particle. Here, the stiff force on any wall particle \( k \) due to any bonded wall particle \( j \) is defined using a spring model\(^{24}\) as:

\[ F_{kj}^s = G Z_0 T_0 \left( \frac{\lambda_0}{\alpha^2 \lambda_0^2} \right), \]  

where, \( G \) is the shear modulus (\( \approx E/3 \)) with \( E \) being the Young's modulus of the wall material, \( Z_0 \) is the initial cell height, \( T_0 \) is the initial cell wall thickness, \( \lambda_0 = L/L_0 \) is the extension ratio of any cell wall element at the current time step, \( L \) is the width of the wall element at the current time step (distance between particle \( k \) and \( j \) ) and \( L_0 \) is its initial un-deformed width. Further, the parameter \( \alpha \) is calculated with \( \beta = 0.5 \) for cylindrical cells as follows\(^{24}\):

\[ \alpha = \sqrt{\frac{\beta + \sqrt{\beta^2 - 4(\beta - 1)/\lambda_0^2}}{2}}. \]  

In Eq. 1, viscous forces acting on any wall particle \( k \) due to the neighbouring wall particles \( j \) are defined by using a linear dashpot model\(^{25}\):

\[ F_{kj}^d = -\gamma \nu_{kj}. \]  

where, \( \gamma \) is the cell wall damping constant and \( \nu_{kj} \) is the velocity of particle \( k \) relative to particle \( j \). In Eq. 1, the \( F_{kj}^f \), \( F_{kl}^w \) and \( F_{kj}^a \) forces are used to define the wall-fluid interactions and the boundary conditions. To avoid any fluid particle penetrations through the wall, the \( F_{ij}^f \) forces are used to create repulsive forces between the cell fluid and wall particles which act through the centre of any interacting wall-fluid particle pair of interest, in an equal and opposite manner. Therefore, the repulsion forces \( F_{kl}^w \) on any wall particle \( k \) from any other fluid particle \( i \) are defined as\(^{21, 24}\):

\[ F_{kl}^w = f_{kl}^w x_{kl}, \]  

where, \( f_{kl}^w \) is the magnitude of the repulsion force and \( x_{kl} \) is the position vector of particle \( k \) relative to particle \( i \). The \( f_{kl}^w \) is defined based on a Lenard-Jones (LJ) force type as\(^{24}\):

\[ f_{kl}^w = \begin{cases} f_0^w \left( \left( \frac{r_0}{r_{kl}} \right)^8 - \left( \frac{r_0}{r_{kl}} \right)^4 \right) \left( \frac{1}{r_{kl}} \right) & r_{kl} \geq 1 \vspace{1em} \cr 0 & \left( \frac{r_0}{r_{kl}} \right) < 1, \end{cases} \]  

where, \( r_0 \) is the initial gap between the two particles, \( r_{kl} \) is the current gap between them and \( f_0^w \) is the strength of the LJ contact. Further, in Eq. 1, self-penetration of the non-bonded wall-wall particles are prevented using a similar repulsion force: \( F_{kl}^c \) with an LJ contact strength of \( f_0^c \). In order to limit cell wall-fluid detachments during drying, wall-fluid attraction force \( F_{kl}^a \) was introduced by us\(^{28}\), which was also defined using the above LJ interactions with a contact strength of \( f_0^a \).
Next, to account for the resistance that a plant cell wall creates when it experiences local bending and wrinkling, bending stiff forces $F_{bk}$ were used and are defined on any wall particle $k$ within the $k$ and $j$ particle pair as:

$$F_{bk} = \frac{k_b}{L} \tan \left( \frac{\Delta \theta}{2} \right),$$

(7)

where, $k_b$ is the cell wall bending stiffness, $L$ is the width of any given wall element at any given time step, $\theta$ is the external angle between the particular wall element and the adjacent wall element as shown in Fig. 2 and $\Delta \theta$ is the change of the $\theta$ angle during time evolution.

Next, as given in Eq. 1, to incorporate experimentally observed cell perimeter reductions, cell wall contraction forces ($F^c$) were used in the model and are defined as:

$$F^c_{kj} = k^c_{wc} \left[ L - L'_0 \left( 1 - \frac{a}{b} \left( 1 - \frac{X}{X_0} \right) \right) \right],$$

(8)

where, $k_{wc}$ is the force coefficient of wall contractions, $L$ is the current width of any particular wall element (see Fig. 1(d)), $L'_0$ is the width of the wall element at fully turgid condition, $a$ and $b$ are empirical factors and $X/X_0$ is the normalized moisture content of the dried cell to be simulated. The $a$ and $b$ factors were set to 0.2 and 0.9 by considering the normalized cell perimeter trends of our experiments (Fig. 13(c) in Section 3), where the overall normalized perimeter reduction is about 0.9. Then the appropriate value for $k_{wc}$ was determined after a series of trial simulations in order to replicate the normalized perimeter observed from our experiments (see Table 1). Furthermore, we hypothesized that the cell wall itself will also undergo moisture reductions during drying and we simply considered such reductions to be proportional to the fluid mass reduction of the cell (see Section 2.5 for details). For fresh cells, the initial cell wall mass was taken as 10% of the fluid mass (see Table 1) which was calculated using the cell volume and fluid density.

### 2.3 SPH modelling of the cell fluid

Cell fluid was modelled using SPH by treating it as a high viscosity incompressible Newtonian fluid with low Reynolds number flow characteristics. Additionally the cell fluid-wall attraction forces were used to ensure the cell fluid was well attached to the cell wall when simulating dried cells. As shown in Fig. 3, the cell fluid model involves four types of force interactions: fluid pressure forces ($F_P$), fluid viscous forces ($F_V$), wall-fluid repulsion forces ($F_{rw}$) and wall-fluid attraction forces ($F_a$). The resultant of these forces $F_i$ on any fluid particle $i$ is defined as:

$$F_i = F^p_{ii} + F^v_{ii} + F^r_{rw_{ik}} + F^a_{ik}. $$

(9)

Here, the momentum equation of the standard Lagrangian type SPH formulations that is used to model weakly compressible low Reynolds number fluids was used to define the pressure forces ($F^p_{ii}$) and viscous forces ($F^v_{ii}$). Accordingly, for any given fluid particle $i$, $F^p_{ii}$ and $F^v_{ii}$ are defined using the properties of the neighbouring fluid particles $i'$ as:

$$F^p_{ii} = -m_i \sum_{i'} m_{i'} \left( \frac{P_i + P_{i'}}{\rho_i + \rho_{i'}} \right) \frac{1}{r_{ii'}} |\nabla W_{ii'}|. $$

(10)

$$F^v_{ii} = m_i \sum_{i'} m_{i'} \left( \frac{\mu_i + \mu_{i'}}{\rho_i + \rho_{i'}} \right) v_{ii'} \frac{1}{r_{ii'}} \left( \frac{\partial W_{ii'}}{\partial r_{ii'}} \right), $$

(11)

where at any given time, $m, P, \rho, \mu, Z$ and $W$ are the particle mass, pressure, density, dynamic viscosity, cell height and the smoothing kernel. For the smoothing kernel $W$, the quartic smoothing kernel was used which is calculated for any given fluid particle $i$ as:

$$W_{ij} = \frac{15}{7 \pi h^2} \begin{cases} \frac{2}{3} \left( \frac{r_{ij}}{h} \right)^7 - \frac{9}{10} \left( \frac{r_{ij}}{h} \right)^6 + \frac{9}{24} \left( \frac{r_{ij}}{h} \right)^5 - \frac{5}{32} \left( \frac{r_{ij}}{h} \right)^4 \quad & 0 \leq S \leq 2, \\ 0 & S > 2, \end{cases} \quad (12)$$
where, $h$ is the smoothing length at the current time step, $S$ is the ratio of $r_{ii}/h$ and $r_{ii}'$ is the gap between particle $i$ and any surrounding fluid particle $i'$ within the influence domain of the particle $i$ ($0 \leq S \leq 2$). In order to improve computational accuracy, the $h$ should be such that approximately 20 particles are maintained within the influence domain of any fluid particle $i$ of this 2-D model. Considering the cellular shrinkage, to maintain the above condition on particle number, the smoothing length $h$ was dynamically adopted using a geometrical relationship as:

$$h = \left( \frac{D}{D_0} \right)^{1/4} h_0,$$

where, $D$ is the average cell feret diameter at the current time step, $D_0$ is the initial cell diameter and $h_0$ is the initial smoothing length (see Table 1). During the time evolution of the model, fluid particle pressure was evolved using a standard SPH state equation as:

$$P_t = P_T + K \left[ \left( \frac{\rho_t}{\rho_0} \right)^7 - 1 \right],$$

where, $P_T$ is the initial cell turgor pressure, $K$ is the fluid compression modulus that ensures the fluid behaves in a fairly incompressible manner, $\rho_t$ is the current density of each fluid particle and $\rho_0$ is its initial density assumed to be equal to the density of water. Next, the density of any fluid particle $i$ was evolved as:

$$\frac{d\rho_t}{dt} = \frac{1}{2} \frac{\partial \rho_t}{\partial Z} \frac{dZ}{dt} - \frac{m_t}{Z} \frac{d\rho_t}{dt} - \frac{m_t}{Z} \frac{\partial Z}{\partial t} + \frac{\partial}{\partial t} \left[ \frac{\partial}{\partial \rho_t} \right].$$

The first term in Eq. (15) accounts for slight density changes of the cell fluid as the cell deforms in XY plane and $\rho_t^i$ is the 2-D density of fluid particle $i$ defined as $\rho_t^i = Z \rho_t$. The $\rho_t^i$ fluctuations are defined using the standard SPH continuity equation as:

$$\frac{d\rho_t^i}{dt} = m_t \sum_v \nabla \cdot \mathbf{v}_w^i \cdot \nabla \mathbf{v}_w^i.$$

The second term in Eq. (15) ensures the density evolution compensates for any cell volume changes due to cell height variations and is defined as:

$$\frac{dZ}{dt} = \frac{Z_{1+\Delta t} - Z_t}{\Delta t}.$$

where, at any given time, $Z_{1+\Delta t}$ and $Z_t$ are the cell heights at the current and previous time steps and $\Delta t$ is the time step size. Cell height used in these equations were updated in each time step by considering the incompressibility property of the cell wall material as:

$$Z = (a \lambda_0) Z_0.$$

The third term in Eq. (15) accounts for the slight density changes within the SPH scheme as a result of the cell fluid mass transfer through the semi-permeable cell wall and is defined as:

$$\frac{dm_t}{dt} = - \frac{A_c L_P n_f}{n_f} (P_t + \Pi).$$

where $A_c$, $L_P$, $n_f$ and $\Pi$ represent the total surface area of the cylindrical cell at any given time, cell wall permeability, total number of fluid particles used and the osmotic potential of the cell fluid at a given dried cell state, respectively (see Section 2.5 for details). In Eq. (9), the remaining two terms are the repulsion forces $F_{iw}^{ik}$ and attraction forces $F_a^{ik}$ that act on a given fluid particle which are defined using the LJ force type as:

$$F_{iw}^{ik} = \sum_k f_{iw}^{ik} x_{ik},$$

$$F_a^{ik} = \sum_k f_a^{ik} x_{ik}.$$
adjacent cells. When the gap between any two interacting wall particles differ from the initially set pectin layer gap, the stiff force always act as a one-to-one contact-force only between the corresponding initially-adjacent wall particle pairs. Accordingly, the pectin layer stiff force on any wall particle \( k \) due to its initial neighbouring particle \( m \) on the adjacent cell wall is defined as:

\[
F_{km}^{pectin} = -k_{pectin}\Delta x_{km} ,
\]

where \( k_{pectin} \) is the pectin layer stiffness and \( \Delta x_{km} \) is the gap difference of the two particles compared to their initial gap. This force helps to maintain the gap between the wall particle pair equal to the initially set pectin layer thickness. Further, this is the only force acting in between cells if they try to separate each other beyond the initial pectin layer gap.

In case if the interacting cells become closer, pectin stiffness creates a repulsion force to separate the cells and tries to return them back to their initial relative positions. The intensity of this force is usually insufficient to fully prevent the cells become interpenetrated. Therefore, a LJ type force is used for this purpose \(^{23} \). However, compared to the approach of the previous researchers \(^{23} \), we propose a computationally far better LJ force field in this work. Here, the cells are repulsed only using wall particles rather than using the full array of fluid particles. Since the computational cost for SPH-DEM calculations increase nonlinearly with the particle number \(^{28} \), our proposed method should be computationally superior since the size of the involved wall particle array is far smaller compared to the cell fluid particle array. When implementing this LJ force field, the initial particle gap \( (r_0) \) used for LJ force calculations is set to half of the initial pectin layer gap (Fig. 4(d)). Accordingly, when the particle \( m \) approaches particle \( k \), until their gap is equal to half of the initial pectin layer gap, only the pectin layer stiff force acts on the two particles to separate them. However, in case if the two particles become further closer (beyond half of the pectin layer thickness), the LJ forces start to act, since the \( r_0 \) is set equal to half of the initial pectin layer gap. Thereby, since the LJ force is much stronger compared to the pectin layer repulsive stiff force, \(^{40} \) when the cells are about to penetrate, the LJ forces become stronger and tends to effectively separate the two approaching particles. Accordingly, the cell-cell repulsive LJ forces are defined as:

\[
F_{km}^{rc} = \sum f_{km}^{rc}x_{km} ,
\]

where, \( f_{km}^{rc} \) is the strength of the LJ force field and \( x_{km} \) is the position vector of particle \( k \) relative to particle \( m \). Here, the \( f_{km}^{rc} \) is defined similar to that of the cell wall LJ force field. It should be noted that, in order to effectively prevent cell-cell penetrations, \( f_{km}^{rc} \) is set comparatively higher than that of the other LJ force fields used in the cell wall model (see Table 1). Using these two force interactions, individual cells were aggregated and the tissue model was developed. The sections below describe how the model was computationally setup and time evolved.

### 2.5 Computational setup and time evolution of the model

Firstly, in the case of a single cell, the fluid particles were placed inside a hexagonal cell wall. The hexagon size was set such that its surface area is equal to the surface area of a circle equal to the cell diameter given in Table 1. Then the model was time evolved using a Leapfrog integrator \(^{21} \) with a time step defined using the Courant-Friedrichs-Lewy (CFL) \(^{21} \) criteria. For the wall, a set of virtual particles were also used in order to avoid any undesirable fluid particle penetrations through the wall boundary and also to limit unrealistic fluid-wall separations \(^{24} \). During fresh cell simulations, according to Eq. (19), depending on the difference of the cell turgor pressure and the magnitude of the osmotic potential, the cell fluid mass varies in a similar way as observed in real cells that have semi-permeable cell walls. These fluid mass fluctuations result in slight fluid density variations as...
defined by Eq. (15), which eventually cause significant turgor pressure fluctuations as defined by Eq. (14). These turgor pressure fluctuations cause the cell wall to move inwards or outwards which produce cellular geometric changes. Now, as a result of such dimensional changes of the cell, the turgor pressure fluctuates again and it leads to cell fluid mass gains or losses according to Eq. (19). This cycle of model evolution repeats until the cell reaches steady state condition where the cell fluid turgor potential and thereafter any further moisture exchanges through the wall cease. By now, the cell shape is fairly circular which fairly replicates the top surface of a turgid cylindrical cell. This steady state particle arrangement is considered as the fresh cell state and is used to evaluate final cell fluid mass, turgor pressure and cell dimensions which are used to characterise the fresh cell state in the analysis described in Section 4.

For single cell dried state simulations, instead of a time-domain based approach, we used a moisture-content-domain based approach which was introduced in our previous works. Further, based on the experimental evidence of moisture content dependent positive turgor pressure existence in cells when they are subjected moisture deficiencies, we hypothesised that the turgor pressure would remain positive during drying and simply assumed that it would linearly reduce with the reduction of the cell fluid mass. So, for example, when 200 kPa is used as the fresh cell turgor pressure, the dried cells of: $X/X_0 = 0.6$, $X/X_0 = 0.4$ and $X/X_0 = 0.3$ were simulated by initially setting the turgor pressures to: 160 kPa, 120 kPa, 80 kPa and 60 kPa. At the same time, the magnitude of the osmotic potential was also set equal to the corresponding initial turgor pressure in each case and kept constant during the model evolution. As a result, when the model is time evolved as explained above, the final steady state turgor pressure of each dried cell state reaches the corresponding initial turgor pressure value. In addition to this, the cell wall drying effects were also incorporated in the model such that for each moisture content case mentioned above, the initial cell wall mass was set to 0.8, 0.6, 0.4 and 0.3 of the cell wall mass corresponding to the fresh condition. During the time evolution, the cell wall mass was kept constant at these initially set values. After time evolution, the final cell physical properties and other geometrical parameters were used to characterise each of the dried cell states and compared with the experimental results.

The above mentioned single cell model was used as a building block and the tissue model was setup by aggregating such cells according to the methodology presented in Section 2.4 with the model parameters given in Table 1. For each cell, the corresponding osmotic potential, initial turgor pressure and initial moisture content are set identically and allowed to undergo simultaneous inflation processes according to the time evolution cycle explained above. Since they are bonded by the pectin stiff forces, now the steady state 2-D cell shapes becomes quite closer.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial cell diameter ($D_0$)</td>
<td>150 μm</td>
<td>3</td>
</tr>
<tr>
<td>Initial cell height ($Z_0$)</td>
<td>100 μm</td>
<td>24</td>
</tr>
<tr>
<td>Wall thickness ($T_0$)</td>
<td>6 μm</td>
<td>15</td>
</tr>
<tr>
<td>Initial cell fluid mass</td>
<td>1.77 × 10^{-3} kg</td>
<td>calculated 27</td>
</tr>
<tr>
<td>Wall mass (10% of cell fluid mass)</td>
<td>1.77 × 10^{-3} kg</td>
<td>calculated 24</td>
</tr>
<tr>
<td>Fluid viscosity ($\mu$)</td>
<td>0.1 Pa s</td>
<td>21, 24</td>
</tr>
<tr>
<td>Initial fluid density ($\rho_0$)</td>
<td>1000 kgm^{-3}</td>
<td>set</td>
</tr>
<tr>
<td>Fresh cell turgor pressure ($P_t$)</td>
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<td>14, 24</td>
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<tr>
<td>Fresh cell osmotic potential ($\Pi$)</td>
<td>-200 kPa</td>
<td>$(\Pi = -P_t)$ 20, 24</td>
</tr>
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<td>Wall permeability ($L_p$)</td>
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</tr>
<tr>
<td>Wall shear modulus ($G_0$)</td>
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</tr>
<tr>
<td>Wall damping ratio ($\gamma$)</td>
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</tr>
<tr>
<td>Fluid compression modulus ($K$)</td>
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</tr>
<tr>
<td>Wall contraction force coefficient ($k_{con}$)</td>
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<td>set</td>
</tr>
<tr>
<td>LJ contact strength for wall-fluid repulsions ($f_{12}^{FF}$)</td>
<td>1 × 10^{-12} Nm⁻¹</td>
<td>set</td>
</tr>
<tr>
<td>LJ contact strength for wall-fluid repulsions ($f_{12}^{WW}$)</td>
<td>1 × 10^{-12} Nm⁻¹</td>
<td>set</td>
</tr>
<tr>
<td>LJ contact strength for wall-fluid attractions ($f_{12}^{WW}$)</td>
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<td>LJ contact strength for cell-cell repulsions ($f_{12}^{CC}$)</td>
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<tr>
<td>Pectin layer thickness ($T_{pect}$)</td>
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<tr>
<td>Pectin layer thickness ($T_{pect}$)</td>
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<tr>
<td>Initial smoothing length ($h_0$)</td>
<td>1.2 × initial fluid grid spacing</td>
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</tr>
<tr>
<td>Time step ($\Delta t$)</td>
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</tr>
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</table>
Fig. 5 SEM images of apple cells at different states of dryness: (a) \( X/X_0 = 1.0 \). (b) \( X/X_0 = 0.5 \). (c) \( X/X_0 = 0.2 \).

In each case, at the end of the time evolution, the dry basis moisture content \( X = \frac{\text{kg water}}{\text{kg dry material}} \) was computed and related with steady state cell deformations in the tissue which were quantified using a set of cellular geometrical parameters: cell area \( (A) \), ferret diameter \( (D) \), perimeter \( (P) \), roundness \( (R) \), elongation \( (EL) \) and compactness \( (C) \). Further, in order to facilitate analysis and comparison of the model performances, normalized parameters \( (X/X_0, A/A_0, D/D_0, P/P_0, R/R_0, EL/EL_0 \) and \( C/C_0) \) were finally used. For model validation, these results were compared with experimental data for apple cells obtained from our experiments \(^{37}\) (see Section 3) and literature \(^{6}\).

### 3. Experimental studies

In order to investigate the real cellular deformations experienced by plant tissues during drying, a series of experiments were conducted by us on the Galla Apple variety (\textit{Malus Domestica}) \(^{37}\). The samples were obtained from a local supermarket in Brisbane - Australia and the initial wet basis moisture content \( (\text{kg water}/\text{kg wet solid}) \) was measured to be 0.84 ± 0.01. Firstly, ring shaped slices were cut from the middle parenchyma region of the fruit such that the inner diameter was 25 mm and outer diameter was 60 mm. Prior to the drying phase conducted in a hot air oven dryer, a 3% citric acid solution was used to pre-treat the slices for about 1 minute. The dryer was set to maintain a temperature of 70 °C with a consistent air velocity of \( \frac{4A/\pi}{4\pi A/P^2} \) and 1 minute.

1. \[ \frac{4A/\pi}{4\pi A/P^2} \]
2. \[ \frac{4A/\pi}{(\text{major axis length})} \]
3. \[ \frac{4A/\pi}{(\text{minor axis length})} \]
of 1.5 ms\(^{-1}\). Then, dried tissue samples corresponding to 30, 60, 
90, 120, 150, 180 and 210 minutes of drying were obtained by 
drying independent samples individually. After each of the drying 
experiments, one half of obtained samples was used to quantify 
the dry basis moisture content and the other half was used for 
microstructural investigations. To determine the dry basis 
moisture content, weights of the oven-dried samples were 10 
recorded and those samples were further dried for about 18 hours 
at 100 °C to obtain the complete dry mass. These mass values 
were then used to calculate the dry basis moisture content of each 
 sample. All the experiments were repeated once to obtain average 
results.

Samples used for microstructural investigations were firstly cut 
in to cubic specimens of 10 mm \(\times\) 5 mm \(\times\) 5 mm. These were 
initially coated with a fixative solution containing 2.5% 
gluteraldehyde, 4% paraformaldehyde and 0.1 M cacodylate 
buffer (pH 7.2) and then kept stored for about 12 hours at 4°C. A 
0.1 M cacodylate buffer (pH 7.4) was used to rinse the samples 
before post fixing with cacodylate-buffered 1% osmium tetroxide 
at room temperature for 4 hours. Then, ethanol solutions of 
incremental concentrations of 50%, 70% and 90% were used 
twice for 10 minutes in each case to dehydrate the samples. These 25 
were finally dehydrated with a 100% ethanol solution for 10 
minutes. Next, a Critical Point Drying \(^{46, 47}\) apparatus was used to 
dry these dehydrated samples and it was conducted for 30 
twice, to obtain fresh-open cross sections for 
microscopic examinations, these samples were fractured by 
freezing in liquid Nitrogen. This method of fracturing helps to 
prevent mechanical damages to the tissue surface which can 
disrupt clear viewing of the microstructure. These samples were 
then mounted on metal stubs and sputter coated with gold up to 
10 \(\mu\)m using an automated sputter coater. These were then 
examined using a FEI Quanta 200 Environmental Scanning 
Electron Microscope (SEM) at 20 kV and imaged at 200\(\times\) 
magnification with an image size of 885 \(\times\) 1022 pixels. These 
images were processed by ImageJ software (version 1.46) and the 
cell-wise average area, ferret diameter, perimeter, roundness, 
elongation and compactness were quantified. These cellular 
geometrical variations were then analyzed against the variation of 
the dry basis moisture content of the samples. The model was 
validated by comparing its predictions against these findings.

As can be seen from Fig. 5, the tissue morphology strongly 
depends on the material moisture content. When the fresh tissue 
is considered as shown in Fig. 5(a), the cells are fairly circular 
compared to the dried states. This is mainly due to the presence 
of higher turgor pressure and moisture content in the cells, which 
apply higher forces on cell walls to make them stretched and 
thereby undergoing minimum wrinkling \(^{7, 14, 16, 32}\). When the 
material gets gradually dried, since the moisture content and the 
turgor pressure reduces considerably, cells become shrinked and 
cell walls experience a higher degree of wrinkling or warping 
(Fig. 5(b) and (c)). Further, it is observed that the deformations 
are irregular and the cell wall structure undergoes minimum 
damage or breakage \(^{4, 7, 8}\). In summary, these findings imply that 
the numerical modelling of plant tissues during drying should 
account for excessive moisture removal and cell wall wrinkling.

According to the best of our knowledge, even the most recent 
FEM based plant tissue drying model \(^{17}\) suffers from both of these 
shortcomings, which should be due to the fundamental limitations 
of the grid-based techniques involved. Section below describes 
how the meshfree model proposed in this work was used to 
simulate plant tissues by overcoming such limitations.

4. Results and discussion

4.1 Simulation of deformations during drying: single cell

Fig. 6 shows the fundamental model behaviour of a single cell in 
a tissue at fresh and dry conditions. Fig. 6(a) shows the initial 
hexagonal single cell model used and Fig. 6(b) corresponds to a 
fully turgid fresh cell. The dried cell conditions are presented in 
Fig. 6(c)-(f). When comparing Fig. 6(a) and Fig. (b), the inflation 
and size increment of the fresh single cell can be clearly 
observed. Further, the dried cell conditions indicate a clear 
reduction of the cell size which fundamentally agrees with the 
cellular shrinkage observed in plant cells during drying. This 
model behaviour is mainly influenced by three factors: lower 
turgor pressure, lower cell fluid and wall mass and higher cell

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Fig. 6 Single cell model: (a) Initial condition before simulations. (b) Turgid condition: \(X/X_0 = 1.0 & P_T = 200 \text{kPa}\). Dried conditions: (c) \(X/X_0 = 0.8 & P_T = 160 \text{kPa}\). (d) \(X/X_0 = 0.6 & P_T = 120 \text{kPa}\). (e) \(X/X_0 = 0.4 & P_T = 80 \text{kPa}\). (f) \(X/X_0 = 0.3 & P_T = 60 \text{kPa}\).
Fig. 7 7-cell tissue model: (a) Initial condition before simulations. (b) Turgid condition: \(X/X_0 = 1.0\) & \(P_T = 200\) kPa. Dried conditions: (c) \(X/X_0 = 0.8\) & \(P_T = 160\) kPa. (d) \(X/X_0 = 0.6\) & \(P_T = 120\) kPa. (e) \(X/X_0 = 0.4\) & \(P_T = 80\) kPa. (f) \(X/X_0 = 0.3\) & \(P_T = 60\) kPa.

Fig. 8 7-cell tissue model (enlarged view): (a) Initial condition before simulations. (b) Turgid condition: \(X/X_0 = 1.0\) & \(P_T = 200\) kPa. Dried conditions: (c) \(X/X_0 = 0.8\) & \(P_T = 160\) kPa. (d) \(X/X_0 = 0.6\) & \(P_T = 120\) kPa. (e) \(X/X_0 = 0.4\) & \(P_T = 80\) kPa. (f) \(X/X_0 = 0.3\) & \(P_T = 60\) kPa.

wall contraction forces. Since the turgor pressure remains positive during drying (see Section 2.5 for details), a fairly circular cell shape is maintained throughout the drying phases. Also since the cell is not externally restricted by any other cells or intercellular influences, this circular shape characteristic is further guaranteed (see Section 4.2 – 4.3 for intercellular influence in bonded cells in tissues).

4.2 Simulation of deformations during drying: dense tissue (7 cells)

Fig. 7 shows the initial particle placement, fresh tissue condition and dried tissue conditions of the 7-cell tissue. Fig. 8 shows enlarged views of these 7-cell tissue configurations. From Fig. 8(a), the initial pectin layer gap is clearly observed. When the fresh tissue condition is considered as seen from Fig. 7(b) and 8(b), the cell shape has differed to a hexagonal shape from the previously mentioned full-circular shape observed in the single cell fresh condition. This is mainly due to the intercellular interactions. Further, it is observed that the pectin layer is stretched particularly at the cell wall positions which correspond to the initial hexagon edges. This is mainly due to the resistance created by the pectin layer on the fresh cell inflation which occurs at a higher turgor pressure. When considering the dried tissue states from both Fig. 7 and Fig. 8, a gradual cellular shrinkage pattern is clearly observed. Further, as the tissue gets further dried, the pectin layer tends to experience lower stretch conditions compared to the moist samples. This indicates that the tissue is more prone to deformations mainly due to the lower moisture content and turgor pressure.

4.3 Simulation of deformations during drying: dense tissue (19, 37 cells)

When considering the 19-cell and 37-cell tissues as seen from Fig. 9 - Fig. 12, similar shrinkage patterns can be observed during drying. But, it is clearly seen that the 37-cell tissue tend to experience some extensive shrinkage, which is due to the large number of intercellular interactions present in such models that causes cells to be more packed and attached to each other.

Further, when referring to Fig. 10(f) and Fig. 12(f), the cell wall has experienced some local wrinkling or warping. This clearly resembles the wrinkled cell walls of realistic dried cells in tissues (see Fig. 5(b) and(c)). Also, when referring to Fig. 9 and Fig. 11, the interior and exterior cells of the tissues have undergone different and irregular deformations, which should be due to the differences of intercellular contacts and force fields which they experience. To elaborate these trends quantitatively, the geometric parameters introduced in Section 2.5, were studied and are presented in Fig. 13. It should be noted here that only the
Fig. 9 19-cell tissue model: (a) Initial condition before simulations. (b) Turgid condition: $X/X_0 = 1.0$ & $P_T = 200$ kPa. Dried conditions: (c) $X/X_0 = 0.8$ & $P_T = 160$ kPa. (d) $X/X_0 = 0.6$ & $P_T = 120$ kPa. (e) $X/X_0 = 0.4$ & $P_T = 80$ kPa. (f) $X/X_0 = 0.3$ & $P_T = 60$ kPa.

Fig. 10 19-cell tissue model (enlarged view): (a) Initial condition before simulations. (b) Turgid condition: $X/X_0 = 1.0$ & $P_T = 200$ kPa. Dried conditions: (c) $X/X_0 = 0.8$ & $P_T = 160$ kPa. (d) $X/X_0 = 0.6$ & $P_T = 120$ kPa. (e) $X/X_0 = 0.4$ & $P_T = 80$ kPa. (f) $X/X_0 = 0.3$ & $P_T = 60$ kPa.

Fig. 11 37-cell tissue model: (a) Initial condition before simulations. (b) Turgid condition: $X/X_0 = 1.0$ & $P_T = 200$ kPa. Dried conditions: (c) $X/X_0 = 0.8$ & $P_T = 160$ kPa. (d) $X/X_0 = 0.6$ & $P_T = 120$ kPa. (e) $X/X_0 = 0.4$ & $P_T = 80$ kPa. (f) $X/X_0 = 0.3$ & $P_T = 60$ kPa.

Fig. 12 37-cell tissue model (enlarged view): (a) Initial condition before simulations. (b) Turgid condition: $X/X_0 = 1.0$ & $P_T = 200$ kPa. Dried conditions: (c) $X/X_0 = 0.8$ & $P_T = 160$ kPa. (d) $X/X_0 = 0.6$ & $P_T = 120$ kPa. (e) $X/X_0 = 0.4$ & $P_T = 80$ kPa. (f) $X/X_0 = 0.3$ & $P_T = 60$ kPa.
internal cells of the tissues were used for the analysis to calculate average cellular geometrical parameters and outer most cell layer was omitted in each case. It is because, the internal cells are the ones that fully experience the intercellular interactions which would fairly represent any realistic tissue. As seen from Fig. 13(a), (b) and (d), the cell area, ferret diameter and roundness trends further confirm that the bigger tissues experience higher level of shrinkage. Further, the model predictions are fairly in a good agreement with the experimental findings. Particularly the 19-cell tissue and 37-cell tissue shows a slightly rapid shrinkage during the latter part of drying ($X/X_0 < 0.6$) which replicates what is observed from the experimental curves. When the cell perimeter is considered, all models seem to experience almost identical cell perimeter reduction trends. This is because, the cell perimeter variation is less influenced by the tissue scale interactions and as defined in Eq. 8, the perimeter reduction was taken to be directly governed by the moisture content of the cell.

The selected parameters used in the cell wall contraction force formulations have ensured a good agreement between simulations and experimental findings. The cell elongation trends reconfirm that the 19-cell and 37-cell tissues can better mimic the real tissue drying behaviours than the single cell and 7-cell tissue models. Further, as observed from experimental curves, the elongation increases during drying, which implies that the cells are subjected to non uniform deformations along major and minor axes. This is clearly observed from the experiments (Fig. 5) and simulated tissue models (Fig. 10 and Fig. 12). Next, in the case of cell compactness, bigger tissues generally indicate a fairly good agreement with the gradually reducing compactness trend of experimental curves. All these observations highlight the capability of the proposed tissue model to replicate realistic tissue deformations during drying.

Further, we should mention here that in actual tissue drying, the cells that are located in the periphery of the tissue undergo...
intense moisture removal compared to the interior cells, leading to a case hardening phenomenon. This is particularly evident in tissues with finite size that involve a large number of cells, that undergo a rapid drying process such as in the case of forced convective drying. However, in our simulations, we simply assumed all the cells in the tissue undergo similar moisture removal, since our main objective was to demonstrate how the cells in a tissue would fundamentally deform when the moisture and turgor pressure varies. Although such a simplified approach was used in this work, the general tissue model introduced here is fully capable of simulating actual case hardening effects which can easily be achieved by separately time evolving the interior and exterior cells with customized moisture content and turgor pressure values.

4.4 Simulation of deformations during drying: tissues with cellular voids (19, 37 cells)

In addition to the dense tissues simulations, another set of simulations were conducted in order to predict how the tissues with voids would deform during drying. For this purpose, only the 19-cell and 37-cell tissues were used and the voids were created by removing one cell from each tissue. The qualitative results are presented in Fig. 14 - Fig. 17. From Fig. 14 and Fig. 16, it is evident that the void geometry also experiences shrinkage in a fairly similar manner, to that of the cells, which fundamentally agrees with experimental findings\textsuperscript{12, 21, 23}. Also it is observed that the cells that are located in the periphery of the voids, tend to deform in a different manner compared to the normal cells within the dense regions of the tissues. This is due to the difference of the intercellular interactions, where cells in the periphery of the void have no contacts towards the void. Further, the enlarged views shown in Fig. 15 and Fig. 17 indicate that the cells in the 36-cell tissue tend to undergo limited irregular deformations compared to the 18-cell tissue. It should be due to the higher number of intercellular interactions present in the larger tissue which resist irregular local deformations. In addition to these qualitative analysis, the geometric parameter trends were also studies as presented in Fig. 18. The cell area and roundness trends indicate this deformation difference up to some extent. However, due to the averaging of the parameters, the localized changes observed in tissue images are not clearly reflected from cell elongation and compactness trends. These findings imply that the overall deformation characteristics of tissues are influenced by local voids, but in bigger tissues the effect is not so significant both qualitatively and quantitatively, provided that the void percentage is not very high (in this case it is 2\% - 5\%). Also, as described in Section 4.3, the different boundary conditions can be used in case of the voids also, by having customized moisture content and turgor pressures for the cells in the periphery of the tissue and around the void. However, considering the scope of this work, these potentially interesting developments were not further studied, but will be addressed in our future works.
Fig. 16 36-cell tissue model with voids: (a) Initial condition before simulations. (b) Turgid condition: $X/X_0 = 1.0$ & $P_T = 200$ kPa. Dried conditions: (c) $X/X_0 = 0.8$ & $P_T = 160$ kPa. (d) $X/X_0 = 0.6$ & $P_T = 120$ kPa. (e) $X/X_0 = 0.4$ & $P_T = 80$ kPa. (f) $X/X_0 = 0.3$ & $P_T = 60$ kPa.

Fig. 17 36-cell tissue model with voids (enlarged view): (a) Initial condition before simulations. (b) Turgid condition: $X/X_0 = 1.0$ & $P_T = 200$ kPa. Dried conditions: (c) $X/X_0 = 0.8$ & $P_T = 160$ kPa. (d) $X/X_0 = 0.6$ & $P_T = 120$ kPa. (e) $X/X_0 = 0.4$ & $P_T = 80$ kPa. (f) $X/X_0 = 0.3$ & $P_T = 60$ kPa.

Fig. 18 Influence of moisture content reduction for cellular geometric property variations of tissues with voids: (a) $A/A_0$. (b) $D/D_0$. (c) $P/P_0$. (d) $R/R_0$. (e) $E/L/LE_0$. (f) $C/C_0$. (Error bars indicate one standard deviation)
4.5 Sensitivity analysis

The tissue model involves a number of physical and non-physical parameters. A sensitivity analysis was conducted for three parameters that mainly influence the model behaviour. For easy interpretation of results, only a 19-cell tissue model was used for this analysis. Also, to better illustrate the model sensitivity to each parameters of interest, both the fresh cell condition (i.e. $X/X_0 = 1.0$) and the critically dried condition (i.e. $X/X_0 = 0.3$) are presented as tissue images. Additionally, quantitative data on cell area, perimeter and roundness are presented for all states.

4.5.1 Model sensitivity to cell wall Young’s modulus ($E$)

Depending on the biological variability and maturity level of materials, the cell wall Young’s modulus ($E$) can vary. Since the $E$ mainly governs how the cell wall responses to different force interactions, it can highly influence the model behaviour. To study the model sensitivity for the cell wall $E$, a series of simulations was conducted by varying $E$ from 1 $E$ to 2 $E$ in steps of 0.25 $E$. From Fig. 19 - Fig. 22, it is observed that the influence of $E$ is dominant in dried tissues than fresh tissues. Further, when comparing 1 $E$ and 2 $E$ cases from Fig. 21 and Fig. 22, it is clearly observed that the dried tissue deformations are resisted by stiffer cell walls and the resulting dried tissue are comparatively larger. Additionally, the 1 $E$ and 1.25 $E$ tissues have undergone considerable local wrinkling compared to 2 $E$ tissue, which also supports the above mentioned behaviour of stiffer cell walls. This is further evident from Fig. 23 where cell area and roundness of 1.75 $E$ and 2 $E$ models in particular are subjected to limited shrinkage compared to other less stiff tissue models.

4.5.2 Model sensitivity to pectin layer stiffness ($k_{pectin}$)

Pectin layer stiffness mainly governs the extent to which the cells are bonded to each other. It is clearly evident from Fig. 24 and Fig. 25, that the fresh cells are tightly bonded to each other in case of higher $k_{pectin}$. When comparing Fig. 25(a) and Fig. 25(e), it is observed that the cell shape also tend to be fairly hexagonal when higher $k_{pectin}$ values are used. Further, when referring to Fig. 26 and Fig. 27, it is observed that higher $k_{pectin}$ values tend to have the cells in dried tissues attached to each other with fairly consistent gaps between them. This is clearly observed from Fig. 26(e) and Fig. 27(e), where the spacing around the centre-most cell is almost equal to the initially set pectin layer gap. Fig. 26 and Fig. 27 further indicate that $k_{pectin}$ particularly influences localized changes of cell shapes in dried tissue rather than global...
Fig. 21 Sensitivity of 19-cell tissue at $X/X_0 = 0.3$ for cell wall Young's modulus ($E$): (a) $1 \times E$ ($\approx 54$ MPa), (b) $1.25 \times E$, (c) $1.5 \times E$, (d) $1.75 \times E$, (e) $2 \times E$.

Fig. 22 Sensitivity of 19-cell tissue at $X/X_0 = 0.3$ for cell wall Young's modulus ($E$) (enlarged view): (a) $1 \times E$ ($\approx 54$ MPa), (b) $1.25 \times E$, (c) $1.5 \times E$, (d) $1.75 \times E$, (e) $2 \times E$.

Fig. 23 Effects of cell wall Young's modulus ($E$) on normalized cellular parameters of a 19-cell tissue: (a) $A/A_0$, (b) $P/P_0$, (c) $R/R_0$. (Error bars indicate one standard deviation.)
Fig. 24 Sensitivity of 19-cell tissue at $X/X_0 = 1.0$ for pectin layer stiffness ($k_{pectin}$): (a) $0.5 \ k_{pectin}$, (b) $1 \ k_{pectin} (= 20 \ \text{Nm}^{-1})$, (c) $1.5 \ k_{pectin}$, (d) $2.0 \ k_{pectin}$, (e) $2.5 \ k_{pectin}$.

Fig. 25 Sensitivity of 19-cell tissue at $X/X_0 = 1.0$ for pectin layer stiffness ($k_{pectin}$) (enlarged view): (a) $0.5 \ k_{pectin}$, (b) $1 \ k_{pectin} (= 20 \ \text{Nm}^{-1})$, (c) $1.5 \ k_{pectin}$, (d) $2.0 \ k_{pectin}$, (e) $2.5 \ k_{pectin}$.

Fig. 26 Sensitivity of 19-cell tissue at $X/X_0 = 0.3$ for pectin layer stiffness ($k_{pectin}$): (a) $0.5 \ k_{pectin}$, (b) $1 \ k_{pectin} (= 20 \ \text{Nm}^{-1})$, (c) $1.5 \ k_{pectin}$, (d) $2.0 \ k_{pectin}$, (e) $2.5 \ k_{pectin}$.

Fig. 27 Sensitivity of 19-cell tissue at $X/X_0 = 0.3$ for pectin layer stiffness ($k_{pectin}$) (enlarged view): (a) $0.5 \ k_{pectin}$, (b) $1 \ k_{pectin} (= 20 \ \text{Nm}^{-1})$, (c) $1.5 \ k_{pectin}$, (d) $2.0 \ k_{pectin}$, (e) $2.5 \ k_{pectin}$. 
tissue shape changes (Fig. 26(a) and Fig. 26(e) has almost same tissue geometry even the used $k_{pectin}$ values are significantly different). The qualitative results are presented in Fig. 28 and only cell area and roundness indicates some level of a difference between different $k_{pectin}$ models. This should be mainly due to the cancellation of parameter effects during the averaging and normalisation process.

4.5.3 Model sensitivity to cell wall contraction force coefficient ($k_{wc}$)

As described in Section 5.1, the cell wall contractions are directly influenced by the $k_{wc}$ and four different $k_{wc}$ values were tested in this study against the original model. By referring to the Eq. (10), it is clear the model is not influenced by $k_{wc}$ when $X/X_0 = 1.0$. This is reconfirmed by Fig. 29 and Fig. 30, where fresh tissues are of identical configurations even at different $k_{wc}$ values. In case of dried tissues, as seen from Fig. 31 and Fig. 32, a clear difference is observed. Fig. 31 implies that higher $k_{wc}$
Fig. 31 Sensitivity of 19-cell tissue at $X/X_0 = 0.3$ for cell wall contraction force coefficient ($k_{wc}$): (a) $0 \ k_{wc}$, (b) $0.5 \ k_{wc}$, (c) $1 \ k_{wc}$ ($= 4 \times 10^{-4} \text{Nm}^{-1}$), (d) $1.5 \ k_{wc}$, (e) $2 \ k_{wc}$.

Fig. 32 Sensitivity of 19-cell tissue at $X/X_0 = 0.3$ for cell wall contraction force coefficient ($k_{wc}$) (enlarged view): (a) $0 \ k_{wc}$, (b) $0.5 \ k_{wc}$, (c) $1 \ k_{wc}$ ($= 4 \times 10^{-4} \text{Nm}^{-1}$), (d) $1.5 \ k_{wc}$, (e) $2 \ k_{wc}$.

Fig. 33 Effects of the cell wall contraction force coefficient ($k_{wc}$) on normalized cellular parameters of a 19-cell tissue: (a) $A/A_0$, (b) $P/P_0$, (c) $R/R_0$.

10 Values tend to increase tissue shrinkage which can even lead to bulk level tissue shape changes. This is particularly evident when comparing Fig. 31(a) and Fig. 31(e), where the former tissue configuration is fairly hexagonal and the latter one is quite circular. Further, when referring to Fig. 32, we can argue that the cell-wise shrinkage differences should be the main cause for these bulk level shrinkage differences of the tissue. Also in Fig. 32, in case of higher $k_{wc}$, the cells have undergone local
wrinkling and warping, which resemble actual cell behaviours during drying. These differences are further evident from the geometric parameters as shown in Fig. 33, where the cell area, perimeter and roundness trends indicate that higher $k_{wc}$ models tend to experience lower shrinkage. Also, particularly in cell perimeter trends as shown in Fig. 33(b), the $k_{wc} = 0$ model only subjects to very limited cell perimeter reductions and deviates significantly from experimental curves which indicate a general decreasing trend. This highlights the importance of $k_{wc}$ in the model, which should be carefully tuned to ensure better model performance.

5. Conclusions and outlook

A 2-D meshfree particle based model has been developed by coupling SPH with DEM to simulate microscale morphological changes of plant tissues during drying. We specifically used a hexagonal cell shape and a positive pectin layer gap in the tissue model in order to represent the frequently observed honeycomb cellular structure of plant tissues which also owns middle lamella of a finite thickness. The main findings of this work can be summarized as:

• Using a meshfree approach, this work have clearly overcome the key shortcomings of grid-based plant tissue drying models that has limited capability of accounting for excessive moisture reduction during drying and cell wall wrinkling behaviour. Also, the simulation approach used here is comparatively straight forward.

• Compared to the state of the art SPH-DEM based plant tissue models, the hexagonal initial cell arrangement and the positive gap used for the middle lamella in this work resulted in a more realistic tissue structure with improved cell-cell contacts.

• To ensure stability of this novel implementation, a careful selection of cell wall pectin layer and LJ repulsion force parameters are recommended is as the smooth time evolution by initially omitting cell wall bending stiffness effects and cell wall contraction force effects.

• In order to avoid cell-cell penetrations in the tissue model, only the cell wall particles can be used rather than the large array of fluid particles which was used by the previous SPH-DEM tissue models. By this means, the computational cost can be significantly reduced as a result of lower number of particle interaction calculations.

• We introduced a means of simulating dried tissues using a SPH-DEM particle scheme by varying the cell moisture content and turgor pressure. A series of simulations were conducted on different size tissues (7-cell, 19-cell and 37-cell) using a generic dense arrangement and it was observed that the tissue model undergoes cellular and bulk level shrinkage. Particularly in critically dried states, cell wall wrinkling and warping was clearly observed. Both effects were found to be in close agreement with experimental findings of dried plant tissues.

• Qualitative studies on several key cellular geometric parameters indicate that the bigger tissue models are fairly capable of replicating most of the realistic cellular geometrical changes during drying.

• Tissue models with voids resemble a basic porous cellular structure which can be observed in real tissues and it was observed that the voids undergo similar deformation as of actual cells, which is in good agreement with the experimental findings in the literature.

• The model is highly sensitive to the cell wall Young’s modulus particularly in dried conditions than fresh conditions. Stiffer walled cells tend to resist shrinkage and resulted in larger dried tissue dimensions.

• Higher pectin layer stiffness tends to maintain fairly uniform gaps between cells particularly in dried tissue.

• The model is also highly sensitive to the force coefficient of cell wall contractions and it mainly influences the dried tissue deformations by enhancing cellular shrinkage and cell wall warping.

Considering all these, we conclude that the proposed SPH-DEM tissue model provides a reliable means of numerically studying the cellular drying phenomena which involves complex physics. The model can be further improved by using advanced cell wall models, fluid-wall interaction models and incorporating multiscale techniques to model bulk tissues in a computationally efficient manner. Tissue porosity effects, bulk material deformations and shape changes can be further studied and related with product and process parameters in the drying industry. Also, 3-D models can be developed using the fundamental conceptual framework proposed in this work. This model can be regarded as a generic model valid for most of the plant materials and could be used to predict deformation characteristics during drying upon the availability of relevant model parameters. Also, temperature variations can be fundamentally incorporated into the SPH-DEM formulations to account for temperature influence for dried plant tissue deformations.

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SPH-DEM based microscale drying model can predict shrinkage and cell wall wrinkling of plant cells in tissues at different moisture contents and turgor pressures during drying (top row: full tissue view, bottom row: enlarged view).