Recovery of the resurrection plant *Craterostigma wilmsii* from desiccation: protection versus repair

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Abstract

*Craterostigma wilmsii* Engl. (homoiochlorophyllous) is a resurrection species that is thought to rely primarily on the protection of cellular components during drying to survive desiccation. The time taken for this protection to be instituted is thought to preclude recovery after rapid drying. Thus the response of *C. wilmsii* plants to rapid dehydration was investigated. The effect of rapid drying on sucrose accumulation was determined and the cellular ultrastructure was investigated during natural (slow) and rapid dehydration and on subsequent rehydration. The dependence of naturally and rapidly dried *C. wilmsii* on de novo transcription and translation during and after rehydration was determined by examining quantum efficiency, changes in photosynthetic pigments and subcellular organization of excised leaves with rehydration in water and using the metabolic inhibitors, distamycin A and cycloheximide. Slowly dried *C. wilmsii* required no new transcription or translation during rehydration in order to recover. With rapid dehydration, cells showed ultrastructural damage, which indicated that at least some protective mechanisms were affected (as evidenced by a reduced accumulation of sucrose). *C. wilmsii* was able to limit the damage and recover upon rehydration in water, but rapidly dried plants did not survive if mRNA or protein synthesis was inhibited by distamycin A or cycloheximide, respectively. This demonstrates an induction of repair mechanisms during rehydration, which enables recovery from rapid drying. Thus, although *C. wilmsii* does rely almost entirely on protection during natural drying, it apparently has the ability to repair if protection is inadequate and damage is incurred.

Key words: *Craterostigma wilmsii*, drying rate, homoiochlorophyllous, metabolic inhibitors, modified desiccation tolerance, transcription, translation.

Introduction

During periods of prolonged water deficit, it may not be possible for plants to avert the loss of turgor in the cells, resulting in mechanical stress, concomitant membrane damage, metabolic disruption and damage due to free radicals (reviews by Levitt, 1980; Smirnoff, 1993; McKersie and Leshem, 1994; Bohnert *et al*., 1995; Pammenter and Berjak, 1999). Resurrection or desiccation-tolerant plants, however, have the ability to tolerate severe water loss (>90% RWC) and resume normal physiological functioning on rehydration (Gaff, 1989). In general, tolerance requires either a protection against the stresses, repair of the resulting damage or a combination of the two (Oliver, 1996). It has been suggested that lower orders chiefly repair damage associated with water loss, while angiosperms, which have been termed modified desiccation-tolerant plants since their loss of water is slower, utilize a greater degree of protection, induced during dehydration (Oliver, 1996). It was therefore thought that the time taken for induction and establishment precludes survival of rapid drying (Gaff and Loveys, 1984; Bartels *et al*., 1990; Oliver *et al*., 1998; Tuba *et al*., 1998).

However, Farrant *et al*., (1999), in a study on angiosperm resurrection species, have shown that *Craterostigma wilmsii* Engl. (homoiochlorophyllous) recovers after...
rapid (6–8 h) dehydration, while Xerophyta humilis (Bak.) Dub. & Schinz (poikilochlorophyllous) does not. They proposed that the strategy of protection employed by X. humilis, which involves the dismantling of the photosynthetic apparatus, is impaired by rapid drying, resulting in excessive damage due to light stress. C. wilmsii, conversely, has a swiftly instituted protection strategy (involving leaf curling and accumulation of anthocyanins) to avoid light stress (Sherwin and Farrant, 1998; Farrant, 2000) which enables it to survive.

Dace et al. (1998) investigated the effect of inhibiting transcription and translation on the recovery of slowly dried X. humilis. The results indicated that mRNAs for chlorophyll synthesis and recovery of electron transport in chloroplasts are stored in a stable form in the dried leaves and are translated upon rehydration. However, additional evidence suggested that new genomic transcription was necessary after approximately 18 h for the complete recovery of photosystem (PS) II functioning (Dace et al., 1998).

The aim of this study was to establish whether dehydration-induced mechanisms of protection in Craterostigma wilmsii are affected by rapid drying and to establish, by the use of metabolic inhibitors, the extent to which this species is dependent on de novo transcription and translation on rehydration after slow and rapid drying.

Materials and methods

Plant material

Whole plants of C. wilmsii (Scrophulariaceae) were collected and maintained in a greenhouse at the University of Cape Town, as previously described (Sherwin and Farrant, 1996; Dace et al., 1998) until the initiation of the experiments described below.

Dehydration and rehydration

Whole plants were slowly dried by withholding water, allowing the plants to dry naturally over approximately 8 d. Rapid drying in the order of 20 h was accomplished as described previously by Farrant et al. (1999), namely, whole plants were removed from the soil and suspended on nylon gauze over silica gel within a plastic container. Dry, compressed air was passed through a tube filled with silica gel (at a rate of approximately 0.13 dm$^3$ s$^{-1}$), over the plant and out through a perforated lid. During drying, the ranges of light intensity and temperature were 150–400 μmol m$^{-2}$ s$^{-1}$ and 18–33 °C, respectively.

Detached leaves were rehydrated for 48 h in Petri dishes containing either water, a 310 g m$^{-3}$ distamycin A solution (inhibitor of transcription) or a 220 g m$^{-3}$ cycloheximide solution (inhibitor of translation) in a plant growth room at 25 °C. The light intensity was 1200 μmol m$^{-2}$ s$^{-1}$ on a 16/8 h light/dark cycle. The concentrations of the inhibitor solutions were determined by testing the incorporation of tritiated uridine and radiolabelled amino acids ($^{35}$S-met,$^{35}$S-cys mixture).

At various intervals during drying and rehydration, leaves were sampled for the procedures described below. Relative water content (RWC) of at least three replicate sample leaves was determined by the standard formula

\[ \text{RWC} = \left( \frac{\text{water content}}{\text{water content} \times 100} \right) \]

Water content and dry weight were determined gravimetrically by oven-drying at 70 °C for 48 h. Mean water content at full turgor was determined from at least 40 replicate leaves taken from fully hydrated plants.

Sucrose content

The sucrose content of leaves from hydrated and dry plants, as well as at two stages during dehydration was determined for at least four replicate samples from different (at least three) plants. The RWC ranges for the drying stages were: drying stage (i) 70–65% RWC; drying stage (ii) 40–25% RWC.

Leaves were ground in liquid nitrogen and mixed with cold extraction solution consisting of 100 mM NaOH in 50% ethanol (v/v). The volume of extraction solution added was four (for hydrated tissue) to eight (dry tissue) times the weight of leaf material. Extracts were heated to 100 °C in a water bath for 10 min and then rapidly cooled on ice. The extract was neutralized by the addition of 100 mM HEPES in 100 mM acetic acid. This was centrifuged at 16 000 g for 20 min and the supernatant retained. The remaining pellet was re-extracted using the same procedure as described above and the supernatants of the two extractions combined.

The quantitation of the sucrose was carried out using a d-glucose/d-fructose sugar assay kit (Boehringer Mannheim, Germany) based on the methodology of Bergmeyer and Bernt (1974). The production of NADPH (determined by reading the absorbance at 340 nm with a spectrophotometer (Shimadzu UV-1601, Shimadzu Scientific Instruments Inc., USA)) was used to calculate the quantity of sucrose in samples.

Cellular ultrastructure

Cellular ultrastructure was investigated using transmission electron microscopy (TEM). Small pieces of leaf tissue (approximately 2 mm$^2$) were excised from at least four different leaves of a minimum of two different plants. Samples were taken from hydrated, dry and rehydrated tissue, as well as three stages during dehydration. These drying stages were (i) 85–70% RWC; (ii) 65–50% RWC; (iii) 40–25% RWC. The tissue was processed according to a method previously used for this tissue (Sherwin and Farrant, 1996). Fixation was in 2.5% glutaraldehyde in 0.1 M phosphate buffer (pH 7.4) containing 0.5% caffeine, post-fixed in 1% osmium in phosphate buffer. After dehydration in a graded ethanol series, the tissue was infiltrated with epoxy resin (Spurr, 1969) over 4 d. Samples were embedded in epoxy resin, hardened at 60 °C for 16 h and sectioned at a gold interference colour (95 nm) using a microtome (Ultracut-S, Reichert, Germany). Sections were stained with 2% uranyl acetate and 1% lead citrate (Reynolds, 1963) and viewed with a transmission electron microscope (EM-109, Zeiss, Germany). Observations were made of general cell structure in at least four different sections of all leaves prepared. Use of standard fixation techniques might have allowed slight rehydration of the desiccated leaves. Studies using cryofixation of dry leaves of C. wilmsii (Vicré et al., 1999) and freeze-substitution of dry tissue from both species used in this study (J Wesley-Smith, University of Natal Durban, personal communication) have given similar results to those observed in this study. However, those methods allowed the preservation of epidermis only and one or two cell layers below that. In order to view the subcellular changes in all leaf cells, standard fixation was used here. Furthermore, since all treatments in the current study were fixed in the same manner, comparisons among them are valid.

Chlorophyll and carotenoid content

The photosynthetic pigment content of at least three replicates of leaf tissue from different (at least two) plants was determined as described by Sherwin and Farrant (1996). Pigment was extracted overnight in 100% acetone and the absorbance of the extract was measured at 661.6 nm, 644.8 nm and 470 nm using a spectro-
photometer (DU 650, Beckman, USA). Chlorophyll \((a + b)\) and carotenoid \((x + c)\) contents were calculated using extinction coefficients provided by Lichtenthaler (1987). The pigment content of hydrated material (which had not been previously dried) was used as a control.

**Quantum efficiency of photosystem II**

The quantum efficiency of PSII of plants that had been either rapidly or slowly dried was determined by chlorophyll fluorometry during rehydration. Five replicate leaves from different (at least two) plants were dark adapted for 10 min (as previously determined) using dark-adaptation cuvettes. \(F_v/F_M\) was measured using a portable, modulated fluorometer (OS-500, Opti-Sciences, USA) with a saturating light intensity of 7500 \(\text{mol} \text{m}^{-2} \text{s}^{-1}\) to obtain maximal fluorescence \(F_M\). This experiment was performed twice.

**Statistical analysis**

Where appropriate, statistical analyses were conducted with the Statgraphics Plus computer program (Statistical Graphic Corporation, 1997).

**Results**

**Dehydration and rehydration rates**

The two drying treatments resulted in markedly different rates of water loss (Fig. 1A). Slowly or naturally dried *C. wilmsii* took approximately 8 d to reach an air-dry state, whereas rapidly dried *C. wilmsii* achieved similar water contents in 12 h. In both cases the final water content achieved was similar for rapidly and slowly dried material.

Figure 1B shows the rehydration time-courses for leaves that were rehydrated in water and with the two metabolic inhibitors. Rehydration rates (for all treatments) of rapidly and slowly dried plants were not significantly different (Analysis of Covariance, \(P < 0.05\), \(n = 3–6\)) and, therefore, only data for slowly dried leaves are shown. Although there was considerable variability in RWC of leaves during the early stages of rehydration, all three rehydration treatments exhibited comparable rates of rehydration, reaching 100% RWC between approximately 15 h and 20 h.

**Sucrose contents during dehydration**

Figure 2 shows the sucrose contents of leaves of rapidly and slowly dried plants. This sugar showed a substantial increase in dry leaves from both drying treatments, with the induction of sucrose accumulation only occurring during the late stages of dehydration (below 25% RWC). Rapidly dried leaves, however, accumulated less sucrose than slowly dried ones, suggesting that the protection afforded by this molecule was incomplete in rapidly dried plants.

**Cellular ultrastructure during dehydration and on rehydration**

Figures 3 and 4 depict the subcellular organization of *C. wilmsii* mesophyll cells in hydrated, dry and rehydrated states and at several stages during dehydration. Control (hydrated) *C. wilmsii* cells (Fig. 3A) had a large central vacuole, with the cytoplasm and organelles situated at the periphery of the cell. There was little variation in cell appearance of tissues dried (regardless of rate) to at least 70% (data not shown). At water contents below this (65–50% RWC), the first signs of turgor loss and subcellular reorganization became evident. In general, there was little difference in ultrastructural changes of cells from rapidly and slowly dried tissues, although at these water contents, some cells from rapidly dried leaves had signs of subcellular damage (primarily rupturing of plasma and vacuolar membranes). The most marked changes in cellular ultrastructure were observed at water contents...
between 40% and 25% (Fig. 3B–D). In slowly dried tissues, considerable cell wall folding had occurred, there was substantial subdivision of the vacuoles and some plasma membrane withdrawal from the cell walls (Fig. 3B).

The extent of this may have been an artefact of the aqueous processing of samples, but there was no breakage of the membranes. In this water content range, cells of rapidly dried leaves also had conspicuous wall folding (Fig. 3C, D).
D), but certain cells showed membrane breakage (Fig. 3C), while the membranes of other cells appeared intact (Fig. 3D). In the dry state, the majority of rapidly dried cells again showed much the same appearance as slowly...
dried cells, as reported in Farrant et al. (1999), with folded cell walls, U-shaped chloroplasts and numerous vesicles (Fig. 3E). There were, however, small pockets of cells in rapidly dried tissue that did show damage (Fig. 3F). Upon rehydration in water, the tissue of both slow and rapid drying treatments reverted to the appearance of the control leaves, with no visible damage (as exemplified in Fig. 3G).

The cellular organization of mesophyll cells of slowly and rapidly leaves rehydrated in solutions of distamycin A or cycloheximide are shown in Fig. 4. The subcellular organization of slowly dried leaves rehydrated in distamycin A (Fig. 4A, B) or cycloheximide (Fig. 4C, D) showed almost identical results to those rehydrated in water (not shown). Cells were vacuolated and the plasma membrane had become re-appressed to the cell wall, having incurred no visible damage (Fig. 4A, C). Thylakoids were intact within the chloroplasts and starch grains were visible (Fig. 4B, D). Possibly the only visible difference was in the cell walls of leaves rehydrated in the metabolic inhibitors, which exhibited slight folding as exemplified in Fig. 4A, something not evident in leaves rehydrated in water. In addition, chloroplasts lacked boundary membranes (Fig. 4E, G). Indeed, in some cases, there were few intact organelles present at all (Fig. 4F, H).

Chlorophyll and carotenoid content of rehydrated tissue

There was a loss of approximately 60% of chlorophyll and 20% of carotenoids during slow drying of C. wilmsii (Fig. 5A, B) as has been previously reported (Farrant et al., 1999). During rehydration of these plants, there was very little variation among the three treatments in chlorophyll and carotenoid content, with both pigments being resynthesized within 48 h of rehydration. It therefore seems that the proteins required for synthesis of photosynthetic pigments are protected during drying and activated during rehydration.

Following rapid drying, only plants rehydrated in water were able to resynthesize photosynthetic pigments (Fig. 5A, B). When leaves were rehydrated in either inhibitor, there was further breakdown and total loss of chlorophyll and carotenoids. This could be attributed to photo-bleaching caused by free radical action. The inability to recover pigments suggests that the components required for pigment synthesis had not survived rapid drying.
Quantum efficiency of photosystem II of rehydrated tissue

There was no statistical difference in the rate of recovery of PSII functioning among the three rehydration treatments of slowly dried material (Fig. 6A) (Analysis of Covariance, P <0.05, n=5). The similarity of the curves indicates that the inhibitor solutions did not prevent full recovery of the leaves and F\textsubscript{V}/F\textsubscript{M} increased to control levels (>0.7) within 15 h. The variability seen in the initial stages of rehydration corresponds with the variability of RWC before 20 h of rehydration (Fig. 1B). Furthermore, there was no drop in quantum efficiency for up to 72 h of rehydration in either inhibitor. This suggests that the proteins present were adequate to maintain normal photosynthetic metabolism for a considerable period subsequent to rehydration.

Figure 6B shows the pattern of chlorophyll fluorescence during rehydration of rapidly dried material. Only those leaves rehydrated in water survived. Although both inhibitors prevented eventual recovery of F\textsubscript{V}/F\textsubscript{M}, leaves rehydrated in transcriptional inhibitor (distamycin A) were able to initiate recovery at the same rate as that of the water controls during the first 2 h of rehydration (Analysis of Covariance, P <0.05, n=5). This might suggest that some transcripts coding for proteins (and/or pigments) involved in PSII activity were present and translated to allow the initial recovery. However, for full recovery new transcription must have been necessary. Leaf explants rehydrated in cycloheximide exhibited no recovery of PSII activity upon rehydration (Fig. 6B), suggesting that when dried rapidly, protein translation on rehydration is vital for recovery of C. wilmsii.

Discussion

C. wilmsii, being an angiosperm, is classified as a modified desiccation-tolerant plant (Oliver, 1996). As such, its ‘strategy’ in surviving desiccation is primarily to protect the subcellular milieu during drying, so as to limit the damage incurred (Oliver, 1996). Previous research has indeed found that such protective mechanisms play a significant role in the survival of this resurrection species (Farrant and Sherwin, 1998; Sherwin and Farrant, 1998; Vicré\textit{et al.}, 1999; Farrant, 2000). Despite the hypothesis that this reliance on protection necessitates slow drying (in order to allow sufficient time to institute the required mechanisms), Farrant \textit{et al.} (1999) have shown that \textit{C. wilmsii} does survive rapid drying in the order of 6–8 h. In that study, slowly and rapidly dried plants showed virtually no difference in recovery of photosynthetic functioning, pigment contents and electrolyte leakage during dehydration. However, from the data presented here, it is evident that rapid drying does have some effect on \textit{C. wilmsii}.

Folding of the cell walls in this species has been proposed to be a mechanism to minimize mechanical stress (Vicré\textit{et al.}, 1999; Farrant, 2000). In the current study wall folding occurred only below a RWC content of 50% and was relatively rapidly induced, with almost complete wall folding visible in the 40–25% RWC range. While wall folding occurred in both rapidly and slowly dried tissues, some cells within the rapidly dried leaves did incur some mechanical damage, indicated by membrane tearing and some disarrangement of cell contents. This suggests that not all cells were able to institute suitable protective mechanisms to maintain membrane integrity. However, damaged cells were sufficiently few so as not to increase electrolyte leakage significantly (Farrant \textit{et al.}, 1999).

Another indication that rapid drying possibly prevented full accrual of protection mechanisms is the reduced sucrose accumulation in leaves of rapidly dried as compared with those of slowly dried plants. This sugar is thought to play a number of protective functions in dry tissues, such as by replacing water on membranes and macromolecules (Crowe \textit{et al.}, 1988), vitrification of the cytoplasm (Leopold and Vertucci, 1986; Vertucci and Farrant, 1995) and filling and stabilization of vacuoles (Farrant, 2000). Thus the shortfall of sucrose in rapidly dried leaves could have allowed lesions to develop in a number of places. In general, the quantity of sucrose was greater than previously reported for other resurrection plants (Ghasempour \textit{et al.}, 1998; Norwood \textit{et al.}, 1999). This difference is probably attributable to the low light intensities in which the experimental plants used in those studies were maintained prior to and during desiccation. Possibly, this resulted in lower accumulation of photosynthetic products, with fewer reserves available for sucrose production in the leaves.

Impairment of complete protection by rapid drying has indirectly been shown to occur in another \textit{Craterostigma} species. Alamillo and Bartels (1996) have shown that young \textit{C. plantagineum} Hochst plants have reduced accumulation of desiccation stress proteins (dps). They proposed that young plants dry more rapidly than the adult plants and therefore cannot accumulate the typical quantity of dps.

However, insufficient protection mechanisms, whatever the nature, did not compromise the survival of rapidly dried \textit{C. wilmsii}, when rehydrated in water, these plants survived. Since there was little ultrastructural evidence of the subcellular damage noted during drying in rehydrated tissues, it is likely that this damage was repaired on rehydration.

 Intriguingly these data show that slowly dried plants rehydrated in distamycin A (inhibitor of transcription) or cycloheximide (inhibitor of translation) recovered completely. Photosynthetic pigments lost during desiccation were recovered, as was full functioning of PSII. \textit{C. wilmsii}, being homoiochlorophyllous, does not lose all of its photosynthetic pigments during drying and can probably continue photosynthesis during early recovery without the
need for immediate resynthesis of these pigments. However, these data show full recovery (within 48 h) of both carotenoid and chlorophyll contents in both inhibitors. In a similar study on Xerophyta humilis, the mRNAs required for chlorophyll and carotenoid synthesis accumulated during slow drying and were stored in the dry state, avoiding the necessity for de novo transcription on rehydration (Dace et al., 1998). However, translation was required for full recovery of the plants. It is possible that in C. wilmsii, the mRNA and enzymes required for pigment biosynthesis are accumulated prior to or during desiccation, and are sufficiently protected in the dry state not to require new transcription or translation during early rehydration. Alternatively, the photosynthetic pigments may have been partially disassembled into colourless intermediates (Matile et al., 1999; Takamiya et al., 2000) needing only reconstitution into an active state on rehydration. Again, however, this would necessitate the presence during drying and activity upon rehydration of enzymes involved in the reconstitution process. Added to this, photosynthesis (as measured by PSII activity), starch accumulation in plastids (ultrastructural observation), and probably all other metabolism (although not measured in this study) continue upon rehydration independently of the de novo transcription and translation.

Although slowly dried C. wilmsii seems almost unaffected by inhibitor treatments, an exception was the mechanism of cell wall unfolding. The folding of cell walls is a controlled process, which is thought to involve the accumulation during drying of components that increase tensile strength (Vicrê et al., 1999). Upon rehydration, the quantity of these molecules (e.g. xylotolucan and pectins) decline and the walls ‘unfold’ (Vicrê et al., 1999). It is conceivable that although many of the proteins needed for the reversal of this process are synthesized before or during desiccation, newly synthesized components (requiring transcription and translation, since wall unfolding does not occur in either inhibitor) are required upon rehydration for completion. Similarly, osmophilic material remains accumulated in vacuoles of plants rehydrated in both inhibitors, suggesting that the components necessary to eliminate these from vacuoles are probably synthesized de novo during rehydration. Alternatively, these may be by-products of metabolism, which was disrupted due to the inhibition in metabolic inhibitors.

In general, it would appear that C. wilmsii lays down virtually complete protection, including the components needed for recovery on rehydration, during desiccation. Furthermore, C. wilmsii is able to maintain photosynthetic functioning for at least 72 h of rehydration in either distamycin A or cycloheximide. This indicates the robust nature of the protection of the photosynthetic metabolism and slow turnover of the enzymes involved in photosynthesis and/or the operation of effective repair processes. Indeed, Schneider et al. (1993) found that translation products accumulated during drying remained for many hours after rehydration in the tissues of C. plantagineum. It has been proposed that the nature of protection of the genome against desiccation- and rehydration-induced damage is such that its reorganization into an active state on rehydration might require more time than most other metabolism (Dace et al., 1998). Having all the components necessary for recovery in place, without the need for new genome-derived information, allows for rapid recovery.

Rapidly dried plants did not survive rehydration in the presence of the metabolic inhibitors, distamycin A or cycloheximide, with little recovery of photosynthesis being recorded. It was proposed above that rapidly dried plants of C. wilmsii do not have sufficient time to institute the protection needed completely, resulting in injury during desiccation. These damaged components are not repaired nor replaced when rehydrated in inhibitors, since this would require de novo transcription and translation, and thus viability is lost. It is not possible at this stage to differentiate whether rapid drying also prevents the establishment of repair systems, or damages repair enzymes, or whether repair is only ever initiated de novo on rehydration. However, if protection is complete enough, then repair probably does not need to be put into place during drying and would only be upregulated on rehydration if needed.

In conclusion, it is proposed that the survival strategy of C. wilmsii relies on the production, during natural (slow) drying, of full cellular protection and of the components required for recovery. This ensures that little repair is required on rehydration, although repair is possible if injury is sustained. The study’s data show that while some repair processes could be constitutively present, any replacement of damaged components requires de novo transcription and translation. Such a strategy would enable an extremely rapid recovery, especially of carbon metabolism, when water is once again available. Furthermore, C. wilmsii can institute protection relatively rapidly, a strategy probably necessitated due to its lack of xerophytic morphological features which serve to retard water loss. These plants occur in an environment in which they undergo frequent cycles of drying and rehydration (Gaff, 1977) and the ability to induce protection rapidly, much of which might even be constitutive, would be essential to its survival. In this respect, C. wilmsii does not conform to the character of a typical modified desiccation-tolerant plant.

This is a departure from the conventional assumptions regarding classifications of desiccation-tolerance and may extend to other desiccation-tolerant species. Certainly, the findings of Proctor and Smirnoff (2000) indicate that the photosystems of ‘fully’ desiccation-tolerant bryophytes are kept intact during desiccation and that repair systems are not required for initial recovery of PSII quantum efficiency. Only with long-term rehydration in light conditions does the need for repair arise, most likely due to photo-oxidative
stress. Although increased complexity of angiosperm resurrection plants does indeed engender a greater degree of protection, rapid drying does not necessarily constrain these mechanisms to such a degree as to prevent survival. In addition, the role of repair in these species may have been underestimated. In the light of these findings, it seems appropriate to re-evaluate the general classifications of modified versus full desiccation-tolerance.

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