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Desiccation damage, accelerated ageing and respiration in desiccation tolerant and sensitive seeds

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Abstract

Embryonic axes of tea (desiccation sensitive) and pea (desiccation tolerant) were dried at different rates or stored at different water contents to distinguish between damage associated with the immediate effects of water loss and the longer-term effects of a partially hydrated state. No loss of viability was observed if pea axes were dried sufficiently rapidly (from 1.8 to 0.1 g H_2O g^{-1} dry mass (g/g) within 5 d). However, viability was lost in tea axes dried below 0.5 g/g (approximately –15 MPa) even if axes were dried within 1 h. Death in tea axes dried to moisture contents less than 0.5 g/g probably resulted from the removal of water necessary for cellular structural integrity (i.e. desiccation damage *sensu stricto*). When axes of both species were dried at slower rates, viability losses were observed at water potentials between about –3 and –15 MPa. The timing for this type of damage was species dependent, occurring within 2 d for tea and after 5 d for pea, and may be explained by higher oxidative activity in tea compared to pea. Embryos of both species with water potentials below –3 MPa were lethally damaged if oxygen consumption exceeded 1000–5000 µmol O_2 g^{-1} dry mass. Recalcitrant seeds are different than orthodox seeds because the former do not survive drying below a critical water content, regardless of the drying rate. Rapid drying is required for accurate assessment of the critical water content. Slow drying leads to metabolic imbalance and artefactual assessment of the critical water content for desiccation damage. Both tea and pea seeds were susceptible to damage from metabolic imbalances, suggesting that the predominant stress from slow drying is ageing.

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Abbreviations: DSC = differential scanning calorimetry; dm = dry mass; g/g = g H_2O g^{-1} dm; mc = moisture content.

Keywords: accelerated ageing, desiccation damage, desiccation tolerance, germination, orthodox seeds, oxidative damage, recalcitrant seeds, respiration, seed, water content, water potential

Introduction

Desiccation damage occurs when water that is critical for survival is removed from cells. Desiccation tolerant organisms survive the removal of water because cellular constituents are either protected or can be repaired. The nature of the damage experienced by drying organisms and the mechanisms evolved to protect against such damage remain elusive.

Two major – and not mutually exclusive – hypotheses may explain cellular damage when water is removed (reviewed by Vertucci and Farrant, 1995; Pammenter and Berjak, 1999; Walters *et al.*, 2001). Damage *sensu stricto* results from mechanical stresses that perturb organelle structures at high (> –5 MPa) moisture levels (reviewed by Levitt, 1980) or macromolecule structures following more extreme drying (reviewed by Wolfe and Bryant, 1999). More recent research has also focused on damage incurred in metabolically active cells at intermediate moisture levels (Leprince and Hoekstra, 1998; Leprince *et al.*, 1999, 2000). These cells may continue to respire but may be incapable of scavenging toxic metabolic by-products that accumulate (Leprince *et al.*, 1990, 1992, 1993, 1994, 1999, 2000; Puntarulo *et al.*, 1991; Leprince and Hoekstra, 1998; reviewed by Hand and Hardewig, 1996) and cause free-radical-associated damage (McKersie *et al.*, 1988; Hendry *et al.*, 1992; Finch-Savage *et al.*, 1994). Dehydration causes several stresses to cells (reviewed by Walters *et al.*, 2001), but methods to distinguish among these stresses and to determine the factors limiting survival of desiccation sensitive tissues are not established.