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Senescence: Concepts and Synonyms

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Abstract: The study reviews on the concept of senescence and some important synonymous terms of it. The term ‘Senescence’ has deeper roots than Programmed Cell Death (PCD)’. Senescence can be regarded to occur at microscopic as well as microscopic levels. At microscopic levels, the process is continuous and can be viewed as Programmed cell death’. Thus both PCD and senescence can be viewed as synonyms at cellular level. Senescence is characterized by a number of catabolic processes as protein degradation, nucleic acid and nuclear degradation, lipid peroxidation, disruption of cell membranes etc., but at the same time, it prepares the plant to start the next generation by remobilizing essential nutrients to developing organs which makes it important for the success of subsequent generations. Although true apoptosis has not been found to occur in plants, but some features of it have been implicated to occur in plants. Recent studies have also revealed the differential expression of some genes in plant systems that were earlier found to express during animal cell death. It is therefore to be decided whether the term apoptosis’ will be still restricted to animal systems only. Ageing refers to degenerative changes that occur in all organisms without any reference to death, while senescence refers to the developmental stage at which close to death’ symptoms becomes apparent.

Key words: Ageing, apoptosis, DAD genes, degenerative, programmed cell death, senescence, synonyms, transdifferentiation

INTRODUCTION

The terms Senescence, Programmed Cell Death (PCD)’, Apoptosis’ and Ageing’ are often used synonymously in plant or animal systems. All these terms generally refer to death of cells, organs or organisms. The use of these terms has led to some confusion (Van Doorn and Woltering, 2004). Although the term PCD has existed for a long time (since 1960s), but the term senescence was used as early as 106-43 BC (Cicero, 106-43 BC). In this context the term senescence is much older than PCD. Apoptosis generally referred to animal cell death appears to be distinct from senescence as far as its features are concerned but the present studies have revealed some of its features in plant systems (Schweichel and Merkel, 1973; Nooden and Guiamet, 1996; Nooden *et al*., 1997; Baehrecke, 2003; Van Doorn and Woltering, 2005). Ageing has long been used as a synonym to senescence but its application has been restricted to those degenerative changes that occur without any reference to death (Nooden and Leopold, 1988). The present review intends to reflect on some important aspects of senescence, its associated synonymous terminology and their applicability in different living systems.

SENESCENCE

The term senescence’ is derived from a Latin word “Senescere” which means to grow old’. The term was apparently used in everyday Latin and is therefore much older. e.g. Cicero (106- 43 BC) wrote, in *De senecitute* (an old age): Temeritas est videlicet florentis aetatis, prudentia senescentis (of course rashness is the note of youth and prudence of old age) (reviewed in Van Doorn and Woltering, 2004). About a century ago the word senescence’ was conceived of, in scientific texts, as the end phase of differentiation, the phase that ends in death (Van Doorn and Woltering, 2004). Leopold (1961) defined senescence in plant cells, as the deteriorative processes that are natural causes of death. Senescence is a decline in age specific survival and reproduction with advancing age. The decline in reproduction and survival with advancing age reflects a decline in the performance of many different physiological functions and physiological studies of plant senescence have given valuable insights into these processes. Senescence in the modular sense is generally considered a beneficial process which increases the individual fitness by allowing a plant to get rid of the old redundant structures or as prelude to the onset of harsh environments (e.g., leaf fall) but in the evolutionary sense, senescence is a deleterious phenomenon which decreases the fitness of the individual (Roach, 1993).

Senescence comprises those processes that follow physiological maturity which lead to the event of death of a whole plant, organ or tissue at macroscopic level. At microscopic level the process, however is continuous, since there exists always a turnover of cell organelles at one or other places of the whole body (Voleti *et al*., 2000;

Van Doorn and Woltering, 2008; Yamada *et al*., 2009). Senescence is an integral part of the normal developmental cycle of plants and can be viewed on a cell, tissue, organ or organization level. It is the final event in the life of many plant tissues and is highly regulated process that involves structural, biochemical and molecular changes that in many cases bear the hallmarks of programmed cell death, PCD (Makrides and Goldthwaite, 1981; Noh and Amasino, 1999; Buchanan-Wollaston and Morris, 2000; Rubinstein, 2000; Xu and Hanson, 2000; Mahagamasekera and David, 2001; Leverentz *et al*., 2002; Wagstaff *et al*., 2003; Jones *et al*., 2005; Rogers, 2006; Xu *et al*., 2006; Hoeberichts *et al*., 2007; Yamada *et al*., 2009; Shahri and Tahir, 2010a). Senescence though a terminal developmental stage, can be accelerated by an array of both biotic and abiotic factors such as light, temperature, nutrients, ethylene, pathogens and pollination etc. (Taverner *et al*., 1999; Van Doorn and Woltering, 2005; Wagstaff *et al*., 2005; Jones, 2008; Zhou *et al*., 2008; Shahri *et al*., 2009; Shahri and Tahir, 2010b). It is a dynamic and closely regulated developmental process which involves highly coordinated changes in gene expression and requires active gene transcription and protein translation (Shahri and Tahir, 2010c, d). A genetically controlled senescence programme allows for the ordered degradation of organelles and macromolecules with the remobilization of essential nutrients (Hensel *et al*., 1993; Yamada *et al*., 2003; Hoeberichts *et al*., 2005; Jones, 2008; Chapin and Jones, 2007; 2009). It is largely an oxidative process involving a general degradation of cellular structures and the mobilization of the products of degradation to other parts of the plants or organs (Nichols and Ho, 1975; Feller and Keist, 1986; Bieleski, 1995; Fischer *et al*., 1998; Van Doorn and Woltering, 2008).

Senescence is mainly characterized by cessation of photosynthesis, disintegration of organelle structure, intensive loss of chlorophyll and proteins, upregulation of tonoplast localized cytochromes, a dramatic increase in lipid peroxidation, proteolytic activity, protease gene expression, poly-galactouronidase activity, nuclease activity, nuclear degradation, vacuolar autophagy, membrane leakage, disruption of cell membranes leading to cellular decompartmentalization and loss of tissue structure (Buchanan-Wollaston and Ainsworth, 1997; Mahagamasekera and David, 2001; Yamada *et al*., 2003; Hoeberichts *et al*., 2005; Rogers, 2006; Xu *et al*., 2006; Van Doorn and Woltering, 2008; Shibuya *et al*., 2009; Shahri and Tahir, 2010c, d). The physiological consequences of physical and chemical changes in the membrane lipids include modifications in the membrane permeability and loss of membrane bound enzymatic activity (Mazliak, 1981). The identification of DAD (defender against apoptotic cell death) gene in plants suggests that N-linked glycosylation of proteins could be an important control point of programmed cell death (Van der Kop *et al*., 2003). Tunicamycin (inhibitor of N-linked glycosylation of proteins) and Brefeldin A (inhibitor of protein trafficking from the Golgi apparatus) strongly induce a form of PCD showing apoptotic features (Cristo *et al*., 2001). The central senescence process seems to begin in the nucleus with the senescence of RNA's which inturn, are used to make certain proteins in the cytoplasm, ultimately resulting in the alteration of plasma membrane and loss of homeostatic ability (Nooden and Leopold, 1988; Hoeberichts *et al*., 2005).

SENESCENCE AND PROGRAMMED CELL DEATH (PCD)

The term senescence and PCD have led to some confusion. Senescence as visibly observed in, for example, leaf yellowing, petal wilting, has often been taken to be synonymous with the programmed death of the constituent cells. The term PCD refers to the underlying programme in cells leading to their death. PCD actually refers to the processes that lead to the moment of death and the degradation; such as in the nucleus and cell walls (Van Doorn and Woltering, 2004). In animal science the term senescence' was initially used for the processes leading to the death of individuals (Child, 1915) but later on it also became used for the processes at the level of organs and cells (Witten, 1983). The term PCD is now applied to death of cells, both in culture and in the intact organism and is used as a synonym for senescence at the cellular level. Plant scientists, applied the term PCD mainly to the death of cells and tissues and the term senescence' to the death of individuals and organs, as also in conjuction with death of tissues (e.g., endosperm senescence) or sometimes individual cells (Pollen tapetal cells). Senescence is considered as a special case of plant cell differentiation designated as Transdifferentiation' which may be defined as change of a cell or tissue from one differentiated state to another' (Delorme *et al*., 2000; Thomas *et al*., 2003). In this context, the conversion of chloroplasts into geronoplasts in senescing cells is comparable to the conversion of chloroplasts into chromoplasts in ripening fruit and developing flower petals and can be seen as a differentiation process. Nooden (2004), however restricted the term senescence to the developmental processes in which the close to death symptoms are visible to the naked eye (as in leaf yellowing, petal wilting and withering of whole plants at the end of their life span). In this view, the term PCD applies to all other forms of cell death including apoptosis, as in root aerenchyma formation after flooding, or the death of the embryo suspensor cells after the development of embryo or even the degeneration of tapetal cells after pollen maturation. There is agreement that both PCD and senescence are terms that denote the processes that lead to the programmed death of individual cells during the early stages of development and to the death of cells in organs and whole plants at the end of their life span. However, it is asserted that senescence in cells is the same as PCD and the two are fully synchronous at cellular level (Van Doorn and Woltering, 2004).

SENESCENCE AND APOPTOSIS

Senescence resembles animal cell death called apoptosis in many features but the characteristic signs of apoptosis have not been convincingly demonstrated in plants (Nooden and Guiamet, 1996; Nooden *et al.*, 1997; Yamada *et al.*, 2003; Hoeberichts *et al.*, 2005; Reape and McCabe, 2008; Yamada *et al.*, 2009). Three morphological features apparently define apoptosis: (1) nuclear fragmentation, (2) formation of apoptotic bodies and (3) engulfment and final degradation of the apoptotic bodies in the lysosome of another cell (Schweichel and Merkel, 1973; Baehrecke, 2003). So far engulfment of apoptotic bodies and degradation in another cell has not been convincingly found in the PCD of any plant cell, so true apoptosis apparently does not occur in plants. However, several other features that have often been taken to be characteristic for apoptosis include (1) the role of caspases, (2) chromatin condensation, (3) nuclear blebbing, (4) DNA degradation and (5) DNA laddering (Van Doorn and Woltering, 2005). In the tapetum and pollen-tubes, there is compelling evidence to support an important role for the mitochondrion and involvement of caspases suggesting a mechanism similar to animal apoptosis, although engulfment of cellular remains by other cells have not been found to occur in plants (reviewed in Van Doorn and Woltering, 2005). Following its nutritive role during pollen development, the tapetum degenerates. This has been reported to be due to chromatin condensation in *Lobivia rauschii* and *Tillandsia albida* (Papini *et al.*, 1999) and by DNA fragmentation in barley anthers (Wang *et al.*, 1999). Another example of apoptotic like PCD in floral organs is in the death of the pollen tube during self-incompatible pollination interactions. In *Papaver*, Thomas and Franklin-Tong (2004) showed that Self Incompatibility (SI) stimulated increase in the cytosolic Ca^{2+} activating the release of cytochrome C into the cytosol and inducing caspases-3- like activity. Recently homologs of genes associated with programmed cell death in animal cells have been found to be differentially expressed during senescence of *Ipomoea* nil petals (Yamada *et al.*, 2009).

SENESCENCE AND AGEING

The term ageing is often used as a synonym to senescence. A distinct difference, however, exists between the two. The former comprises all those degenerative changes and cellular wear and tear that occur in time without any reference to death, the latter is considered as a final developmental phase which culminates in death (Nooden and leopold, 1988). Senescence in higher plants involve a series of physiological processes integrated with developmental programme which normally lead to the loss of the maternal plant or most of its organs and the plant is prepared to start the next generation. This makes senescence important for the success of subsequent generations (Feller and Keist, 1986). One important difference between senescence and other types of PCD is that senescing cell has a very specific role to play before death. e.g. In senescing leaves, chlorophyll is degraded only to remove this potential toxic compound from the cell to prevent premature cell death. The products of chlorophyll degradation are then stored in the vacuoles of senescing cell (Matile, 1997). Besides, increased transcription of a number of genes encoding enzymes involved in removal of reactive oxygen species have been demonstrated during senescence (Buchanan-Wollaston and Ainsworth, 1997).

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