Plant individuality: a solution to the demographer's dilemma

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Abstract   The problem of plant individuality is something which has vexed botanists throughout the ages, with fashion swinging back and forth from treating plants as communities of individuals (Darwin 1800; Braun and Stone 1853; Münch 1938) to treating them as organisms in their own right, and although the latter view has dominated mainstream thought most recently (Harper 1977; Cook 1985; Ariew and Lewontin 2004), a lively debate conducted mostly in Scandinavian journals proves that the issues are far from being resolved (Tuomi and Vuorisalo 1989b; Fagerström 1992; Pan and Price 2001). In this paper I settle the matter once and for all, by showing which elements of each side are correct.

Keywords   Biological individuality · Plants · Modular · Genet · Ramet · Selection

This paper presents a philosophical treatment of the nature of biological individuals, as assessed from the perspective of plants. I argue that plants violate the formal criteria given by the most popular ways of defining individuals, leaving us with the uncomfortable prospect of omitting this kingdom altogether from the domain of objects to which fitnesses can be assigned. I aim to show how universality can be restored to the concept of the individual by zeroing in on control of heritable variance in fitness as the basic criterion that the classical views are all pointing towards. The basic claim is that if plants have mechanisms which determine the hierarchical level at which selection is able to act, then they should qualify as

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individuals in virtue of those mechanisms, even if they look very different from the ones that do the same job in vertebrates. Accepting these individuation criteria forces us to accept that individuality comes in degrees, and can appear at numerous hierarchical levels simultaneously, meaning that sometimes plant biologists will have no choice but to adopt a multilevel selection approach, especially when assessing evidence for, and making predictions about, evolutionary change.

The first three sections of the paper set out some necessary groundwork for approaching the problem of plant individuality. In the first part I explain what it is about plants that causes individuation problems to arise. In part two I introduce the debate amongst plant demographers concerning the relative primacy of genets and ramets as plant individuals. Then in part three I discuss what I'll call 'classical criteria': those solutions that have gained widespread support in non-plant domains, especially with respect to higher vertebrate lineages. In the end we will see that, thanks to the properties outlined in part one, the classical criteria cannot help us to settle the genet/ramet debate. Furthermore, we appear to be stuck in a new dilemma, forced to choose between abandoning the classical criteria, or abandoning the idea that plants are organisms at all.

The second half of the paper presents a new definition of the biological individual and applies it to plants. I explain that if we focus, not on the classical criteria themselves, but instead on the *effect* of the properties on which they are based—on what consequences they have for the creatures that meet them—then we see that plants have their own idiosyncratic properties that nonetheless play the same role. In part four I argue that the classical individuation criteria succeed in vertebrates by picking out mechanisms which constrain a population's ability to exhibit heritable variance in fitness. In part five I identify some examples of mechanisms which play the same role in plants. In part six I give a quantitative argument to show what can go wrong when the action of plant individuation mechanisms is ignored, vindicating some of the insights that emerged in the ramet/genet debate, and showing why, when it comes to counting plants, a multilevel approach will sometimes be necessary.

First of all, a short note on semantics, before I start to explain why plants can be so very difficult to count. People who study plants and other modular organisms do not tend to bother using the word 'individual' all that much. Instead they invoke a plethora of more precise terms—zooid (Huxley 1852), ramet (Harper 1977), metamer (White 1979), individuoid (Van Valen 1978), module (Watkinson and White 1986), somatogen (Van Valen 1989), genet (Harper 1977), tiller, meristem and more (Pepper and Herron 2008). I am choosing to refer to the unit under study as 'biological individual' and 'individual organism', interchangeably, both of which have good precedent (Wilson 1999, 2007; Pepper and Herron 2008; Gardner and Grafen 2009; Queller and Strassmann 2009; Folse and Roughgarden 2010). The concept itself is relatively clear: a creature, one animal, a singular living thing which, if we were to take as a pet, we might name 'Fred' or 'Smoky'. In bygone times we might have said the concept picks out any single member of a species, one example of a type. There is much that can be said about the extent to which the concept under scrutiny is general; the extent to which it lines up with the philosophical notion of 'individual', or with the concepts brought to mind by

Table 1 Key terms

A *module* is a self-reproducing and semi-autonomous unit that is iterated to make up a larger unit or colony. In plants it will usually contain one or many meristems in a shoot or root.

A *meristem* is a plant tissue that remains undifferentiated and mitotically active. It is *totipotent* (can give rise to all parts of the embryo and adult (Campbell and Reece 2008)) and *immortal* (can mitotically divide an unlimited number of times (Michod and Nedelcu 2003)).

A *ramet* is a collection of modules that forms a physically coherent structural entity (a tree, or bush, for example) and is produced vegetatively, by another ramet (Harper 1977).

A *genet* is the collection of all those modules or ramets that have developed from a single zygote, i.e. all the mitotic products of a single sexual reproductive event (Harper 1977). Some readers may read the term 'genet' as implying genetic homogeneity, but I use a developmental definition because, as we will see later, the unit that develops from a zygote rarely stays genetically homogeneous for long.

We say an organism is *clonal* when whole ramets or structural individuals iterate themselves (Tuomi and Vuorisalo 1989a).

A *mosaic* individual is composed of two or more genotypes that originated from a single zygote but that diverged during mitotic growth (Gill et al. 1995).

'physiological individual' or 'unit of selection'. This discussion goes beyond the scope of the present paper, however. Although I choose not to use the term, you may think of the object as an 'evolutionary individual' only *if* you can be sufficiently careful not to confuse it with 'unit of evolution'—in the sense of species or other long term beneficiary of evolution (Mayr 1996; Lloyd 2005); or 'evolutionary module'—in the sense of sub-organismal unit whose evolution proceeds independently, to some extent, from the other parts of the organism (Brandon 1999). What is imperative for my argument is the following; I*f* we are happy to call animals 'individual organisms' in virtue of their possession of particular kinds of mechanisms, then we really ought to be willing to call plants individual organisms also, in so far as they possess mechanisms which do the same job. One major advantage of adopting this stance, in both domains, is that we pick out units from which we can generate accurate and predictive models of the evolutionary processes acting on populations.

The problem of individuality in plants

Scientists who study plant populations can have a variety of goals. Ecologists and comparative demographers measure plant fitness in order to compare strategies or phenotypes in different environments, and to predict optimal life history. Conservationists record the spread or success of different species. Evolutionary studies use fitness to assess selection pressures and evolutionary constraints. All of these types of study necessitate counting plant units—keeping a record of the number of births and deaths that take place within a give location over a given amount of time.

The trouble with counting plant units is that a decision has to be made, before the counting begins, about what to count. In many animal lineages this seems like no

problem at all because the relevant unit is just obvious. If we want to count pigs, for example, it is rather easy to tell which bits count as pig parts and which as new pigs. But plants, and other modular organisms, grow and develop in ways that cloud the issue, to say the least. In modular organisms, replication occurs at multiple hierarchical scales, and each scale constitutes a level at which the demographer might choose to count births and deaths.

My first task is to outline the features and habits of plants and other modular organisms that create a radically new context for the biological individuality problem. The essential points are these;

Plants are modular, so that their parts have some degree of ecological and reproductive autonomy. Some are capable of clonal iteration by vegetative growth from multicellular runners. Somatic mutations can be transmitted down cell lineages in the course of normal mitotic division. Finally, plants show somatic embryogenesis, meaning that many plant cells retain developmental potency throughout the lifetime of the plant. One significant consequence of this is that somatic mutations can be transmitted to future generations by sexual and apomictic[1] reproduction, as well as to mitotic offspring. This gives rise to the possibility of somatic evolution, in which selection acts on differences within plants as well as differences between plants. Finally, it is important to keep in mind that there is a huge amount of variability in the extent and manner in which different plant species exhibit these properties. In the rest of this section I explain these properties at greater length, and suggest that those readers who are already familiar with them may want to skip to part two.

Modularity

To call a plant 'modular' is to say that it grows by the accumulation of smaller constructional building blocks called modules (Table 1). Modules have been much discussed in many different contexts, but here I refer only to what can be termed 'structural modularity'. Unlike evolutionary or developmental modules, structural modules are self-reproducing and semi-autonomous[2] (Watkinson and White 1986; Tuomi and Vuorisalo 1989a). Structural modularity occurs in diverse lineages, including plants, fungi and bacteria (Andrews 1998) as well as many animal lineages, especially marine invertebrates (Jackson et al. 1985; Hughes 1989). So this isn't an obscure or minority group—modular organisms constitute the overwhelming majority of the planet's biomass (Townsend et al. 2003)—and many of the points made here apply to all of that group, as well as to plants.

Plant modules are the constructional building blocks of all vascular plants, including ferns, conifers and flowering plants (angiosperms) (Watkinson and White

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[1] Apomictic reproduction occurs when a new plant develops from a single celled zygote but without sexual fertilisation.

[2] In all cases 'modular' will be used here to refer to structural, rather than developmental or evolutionary modularity, and 'clonal' to refer to vegetative, rather than parthenogenetic growth. See Clarke (2011) for more on these distinctions.

1986). They are multicelled sub-units, whose iteration proceeds by mitotic division from a special totipotent and immortal group of cells known as apical meristems (Tuomi and Vuorisalo 1989b; Monro and Poore 2009). A typical plant module is found at the tips of roots and shoots. Each module has its own life cycle; its own program of growth and senescence. Unlike the cells that make up unitary multicellular organisms, plant modules are semi-autonomous, because provided they are supplied with the nutrients they need they can survive and sexually reproduce independently of the rest of the plant. The 'semi' is there because they do not usually live alone—they usually interact with and share resources with other modules.

Clonality

Many plants, including dandelions, grasses, aspen, bracken ferns and strawberry plants, are clonal *as well as* modular. This means that there is copying or iteration at two levels; at the level of modules, and also at the level of whole plants or trees. For example, quaking aspen (*Populus tremuloides)* have meristem modules in their roots from which they grow runners. These vegetative propagules grow away from the parent tree, and then up towards the surface of the soil, at which point module iteration proceeds until a whole new tree has been grown.

Clonal propagation allows a plant to start its developmental cycle anew without meiosis. This gives clonal plants a sort of potential immortality (Fagerström 1992) because they do not need to halve their genome and then recombine it with that of another organism in order to continue existing after the death of the parent plant. The genet only dies if all of its ramets die at once (Fig. 1).

Somatic mutations

The development of any multicellular organism involves a series of mitotic cell divisions. There are many mechanisms in place to ensure that the copying process is faithful, but they are not perfect, and errors sometimes occur, which are then
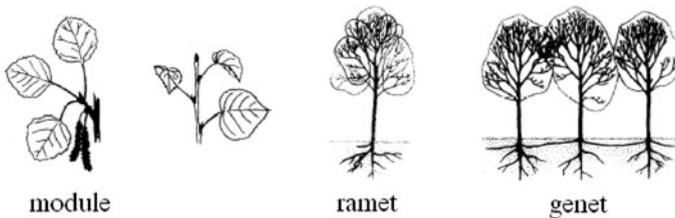

module   ramet   genet

Fig. 1 This schematic drawing from Tuomi and Vuorisalo (1989a) shows a typical plant shoot module composed of some leaves and flowering parts. These iterate to compose branches or stems, which iterate to form a ramet. In clonal species whole ramets are also iterated to form a genet

replicated down the cell lineage. The sheer numbers involved[3] mean that all but the tiniest organisms are genetic mosaics: their cells carry distinct genotypes (Otto and Hastings 1998). Different cell lineages or modules in each ramet, and/or different ramets in the clonal genet, can carry distinct alleles. Some kinds of mosaicism and chimerism are visible in the phenotype of a plant. For example, somatic mutation is responsible for leaf variegation, in which certain areas of the leaf carry a faulty gene involved in producing chloroplasts.

Clonal plants are predicted to show a greater degree of mosaicism than other plants, just because their cells can continue mitotically dividing indefinitely (Hadany 2001) with each and every mitotic event providing another opportunity for errors to occur. Another reason why clonal plants can be expected to be less genetically uniform than unitary organisms is that the runners or propagules by which vegetative cloning occurs are multicellular, and so are efficient carriers of mosaic genotypes. This is in contrast to apomictic or sexual reproduction, in which the single celled stage ensures that only one cell with one genotype serves as the template for all the subsequent cells in the organism.

Somatic embryogenesis

In unitary organisms only very few cells are capable of developing into a whole new organism. The rest are differentiated; restricted to expressing one or a few cell phenotypes. This 'germ-soma separation' limits the significance of mosaicism in unitary organisms. Most mutations will occur in the differentiated, somatic tissues, just because there are many more of them than there are germ cells. These mutations will be passed on to daughter cells, but can't cross over into the germ cells. This means that they cannot be passed on to offspring organisms.[4] Unless the mutant has some way of being transmitted to other organisms, as is the case with viral cancers such as the infectious Tasmanian Devil facial cancer DFTD (Murchison 2008), it will disappear when the organism dies. There is a thus a firm limit on the long term heritability of somatic mutations.

Plants, along with many other living things,[5] show 'somatic embryogenesis' (Tuomi and Vuorisalo 1989a; Buss 1983; Lyndon 1990), which is just a way to say that they lack germ soma separation (Jerling 1985; Sutherland and Watkinson 1986). Instead of sequestering their germ cells, plants retain a stock of undifferentiated tissue, mixed in with all their other tissues, which are capable of producing all the phenotypes necessary to build a whole ramet (Stewart et al. 1958; Tuomi and Vuorisalo 1989a). Somatic embryogenesis makes vegetative or clonal propagation possible, as well as giving plants great powers of regeneration. Most cells taken

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[3] Estimates of the mutation rate per gene per individual generation fall between 10^{-7} and 10^{-4} (Otto and Hastings 1998, p. 510).

[4] At least this is the view of the modern synthesis. See Jablonka and Lamb (2005) for ways in which somatic variation may affect the germ line after all.

[5] Buss describes twenty seven out of fifty living taxa as showing some somatic embryogenesis (Buss 1987, p. 21) but he also shows that germ line sequestering and embryogenesis are not discrete alternatives. Rather, all living things fall somewhere on a spectrum where at one extreme the unitary organisms sequester the germ line early and preformistically and at the other no sequestering occurs at all.

from a tree can, with careful enough treatment, be prompted to de-differentiate and grow into a whole new tree (Verdeil et al. 2007).

Somatic embryogenesis also alters the status of somatic mutations. Rather than being fated to disappear once the ramet's life cycle is complete, plant mutations can be transmitted to new plants, by both vegetative and sexual means. If they show up in a clonal propagule, or during adventitious growth,[6] then they will be transmitted to a new ramet. But they can also occur in, or be transmitted to, a shoot meristem, from where they can spread into the inflorescence and ultimately a zygote.

Note that unitary organisms are also composed out of many smaller building blocks—cells, and these cells also iterate themselves and their life cycle by dividing mitotically to produce copies of themselves. But because almost all of a unitary organism's cells are differentiated, this iteration is something less than full reproduction—the cell is not capable of producing cells with all the phenotypes necessary to produce the entire organism.

Somatic selection

Many authors have tried to draw attention to the possibility, and potential evolutionary significance, of what they call 'somatic selection' or 'intraorganismal selection', which occurs when heritable differences between cells or other plant parts cause their differential survival or proliferation within the plant (White 1979; Klekowski et al. 1985; Antolin and Strobeck 1985; Hardwick 1986; Sutherland and Watkinson 1986; Hughes 1989; Hastings 1991; Acosta et al. 1993; Otto and Orive 1995; Fagerström et al. 1998; Hadany 2001; Orive 2001; Poore and Fagerström 2001; Klekowski 2003; Monro and Poore 2004). These authors are thinking about plants, but somatic selection has also been discussed in the context of other clonal lineages such as corals (van Oppen et al. 2011) and aphids (Loxdale 2008). Somatic plant selection is selection that acts between cell lineages or other plant parts, instead of (or as well as) between plants. Somatic selection can result in sub-organismal evolution, in which gene frequencies change within the lifetime of the plant, rather than across successive generations of plants, as occurs in organismal evolution.

This in itself is nothing surprising—any organism suffering from cancer can show a shift in the frequency of a particular allele over time, after all. However, the significant difference is that in plants the sub-generational changes are *heritable* because the victors of somatic -selective battles can be transmitted to subsequent generations. The transmission can be either sexual, if the mutation is expressed in the flowering parts, or vegetative, if it is expressed in the vegetative propagule. Somatic selection in plants can have long term evolutionary consequences.

Several authors argue that intraorganismal selection is evolutionarily significant and even adaptive at the organism level (Buss 1983; Gill et al. 1995; Otto and Hastings 1998; Fagerström et al. 1998; Pineda-Krch and Fagerström 1999;

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[6] This is growth that occurs outside of the 'normal' developmental program, if there is one, and which originates from non-meristematic tissue. For example in the practice of coppicing, a tree is cut down above the ground, and adventitious shoots grow from around the trunk.

Marcotrigiano 2000; Lushai et al. 2003; Pineda-Krch and Lehtilä 2004; Clarke 2011; Folse and Roughgarden 2010). For example, Otto and Hastings construct a model which shows that as long as selection is concordant between hierarchical levels, intraorganismal selection can act as a sieve which reduces the genetic load by removing deleterious mutations and increases the evolutionary rate by promoting beneficial mutations.

Empirical confirmation of somatic selection in plants is thin on the ground[7] however (exceptions are Breese et al. 1965; Whitham and Slobodchikoff 1981; Monro and Poore 2009) and most of the work done so far has been theoretical (see Folse III and Roughgarden 2011 for a review), leading some to be sceptical that somatic selection is really a significant evolutionary phenomenon (Hutchings and Booth 2004).

Variation

There is huge variety in the pattern and degree of modular iteration among different species of plant. In the most extreme cases, modular plants iterate many units at once in a branching pattern, with each module having a high degree of autonomy and perhaps even its own independent root connections or stem. They are clonal as well as modular so that iteration of functional units occurs at multiple hierarchical scales. And they are iteroparous, so that development is uncoordinated across modules.[8] The accumulation of modules is open-ended and can go on indefinitely, without progressing towards any fixed adult form (Begon et al. 2006).

At the other extreme are plants that are more unitary: they have a determinate growth form, so that form is linked to age or life stage (Jerling 1985; Hughes 1989). They are short lived and a clonal, reproducing only sexually, with only one growing axis or a single shoot module producing seeds or spores, and a single shared stem.

Different plant species occupy different positions between these two extremes. Some organisms such as grasses actually switch between modular and clonal modes within the lifetime of the genet, according to changing environmental conditions. The different modes are described as phalanx and guerrilla strategies respectively (Lovett Doust and Lovett Doust 1982).

It is important to keep all this variation in mind, because it implies that all plants are far from equal when it comes to individuation problems. While some plants possess all the features listed above, this does not deny that there are many plants which are much more tractable. Thale cress (*Arabidopsis thaliana*) will tend to be much less badly behaved than quaking aspen (*Populus tremuloides*), for example. Some plants just aren't particularly ambiguous when it comes to individuality, especially those that tend to be focus of evolutionary studies: short lived herbaceous sexual reproducers (although see Weinig et al. 2007). But neither ought the

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[7] Although there is substantial evidence for evolution in clonal lines in aphids, mites, and bacteria, which are relevantly similar (Weeks and Hoffman 1998; Wilson et al. 2003).

[8] As opposed to 'semelparous' plants, in which module development is coordinated so that all the parts flower at once, after which the whole plant or group of modules dies.

tractability of chosen examples be thought to undermine the seriousness of the problems for the general concept of individuality.

Now it should start to become clear why plants constitute a peculiar case for the problem of individuality. Many plants are constructed by iteration at both modular and clonal scales, which produces a hierarchical organization. Each level of the hierarchy is a level at which replication or copying occurs, at which births and deaths can be counted. The units at each level can carry mutations and transmit them to future generations. Which units should the botanist count? Which are individuals?

In part two I introduce the two rival responses to these questions, showing that there is a genuine choice to be made when counting plants.

The demographer's dilemma

This section introduces the competing genet and ramet approaches to plant individuality and gives just enough of the substance of the debate to convince the reader that the issue is still live. Anyone familiar with these arguments may wish to skip to part three.

In the 18 and 19th centuries a metapopulation view of plant individuality was dominant, on which the macroscopic objects which we call plants or trees were treated as communities of smaller scale individuals. In 1721, Richard Bradley wrote that "the twigs and branches of trees are really so many plants growing one upon the other." (Quoted in Solbrig 1980, p. 22).In 1853 Alexander Braun insisted that "in so far as we are justified in speaking of vegetable individuality at all, we must hold fast to the individuality of the shoot: the shoot is the morphological vegetable individual." (ibid). In 1938 it was argued that only competition amongst branch-individuals could explain the shape of trees (Münch 1938, quoted in White 1979).

Subsequent advances in evolutionary theory made people realise that understanding the action of natural selection on plant populations requires a unification of theoretical knowledge about population genetics—changes in gene frequency—with ecological observations about changes in the numbers of individuals in a population. Measuring natural selection requires determining the phenotypic and genotypic composition of a population just before and just after exactly one round of selection has occurred (i.e. after a single reproductive cycle). We then make inferences about the extent to which gene frequencies have changed as a consequence of selection for phenotypic traits. In unitary organisms the zygote cycle is generally taken to delimit a single generation or reproduction cycle, allowing for one round of selection. In plants, seeds or spores[9] are generally taken to be the key actors in the life cycle which must be counted. The theoretic ideal is that the genotypic and phenotypic characters of all the seeds in a population are measured (Solbrig 1980). Then after a period of time determined by the average time it takes members of this population to complete one life cycle from seed to seed, all the seeds *produced by* the seeds in the

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[9] I'll talk about seeds for simplicity, but this should be taken as standing in for any sexually produced plant propagule.

first count will be counted and have *their* genotypic and phenotypic properties measured.

There are many reasons why actual empirical practice never meets this theoretic ideal. Analysing the genotype of seeds is an expensive and laborious process and one that is impossible to perform in a non-destructive manner (i.e. without becoming a kind of selective agent). Sampling and statistical techniques are therefore used to make inferences from a small sample. These inferences are upset by the fact that just calculating the size of the population to be sampled is very difficult. Seed counts are inaccurate because of the ways in which seeds get carried in and out of spatial areas by water, wind and various motile organisms, and because vast numbers are buried below ground where they are invisible. Furthermore, performing a second count after a single zygote cycle has elapsed is in practice impossible for many plant species, because many have life times that far exceed that of a normal research project.

These sorts of problems plague studies of many animals too, and population biologists have developed various ways to try to overcome them. The most established for plant populations is the demographic method, founded by John Harper in 1977. In some ways Harper's method can be seen as an extension of the metapopulation view, because it treats plants as populations, taking their parts as individuals to be counted. For example, we might be looking at a small group of trees, and we would count the number of new leaves produced each spring by each tree. But more commonly, demographers take ramets or trees to be the countable units.[10] With respect to a species like quaking aspen (*Populus tremuloides*), the demographer would mark off an area of forest. He would then return to the area at regular intervals and record the numbers of births and deaths of trees within the area.

Unlike the metapopulation view however, Harper's demographic method essentially takes genets to be the individuals in plant populations. It is important that in recording new births, we note whether the new tree has grown from seed or was vegetatively propagated. They *could* in principle use genetic analysis to actually record the genotype of a genet, but in species where the parts of a genet remain visibly connected to one another, in practice they will often work on the assumption that plants grown as independent seedlings have a novel genotype, whereas new vegetatively produced plants share that of the plant to which they are connected. The purpose of the survey is to record the growth rate of each genet, where its growth rate is just the number of new clonal parts produced, minus the number that have died. The central tenet of demographic analysis is summed up in this simple equation which gives the size of a genet at a future time as;

$$\eta t + 1 = \eta t + B - D$$

where η = number of modules, B = number of module births and D = number of module deaths (Harper 1977).

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[10] In fact genet growth can be ascertained by counting any genet part—ramets, branches, buds, leaves, tillers, flowers—any countable unit will do. The main criteria used when choosing a focal unit are practical—is it easily accessible? Are the numbers tractable? (Wikberg 1995).

But why does the demographer assume that measuring the size of a genet has anything to do with reproduction? How can something so far from a count of seeds tell us anything about the fitness of genets? It is entirely usual for biologists in all fields to use some easily measurable trait as a proxy for fitness, rather than trying to measure actual lifetime reproductive success (Niklas 1997). It is justifiable so long as the trait measured can be assumed to correlate well with actual fitness. What demographers essentially do is take genet growth (rate of increase by vegetative expansion) to be a proxy for fitness. This is justified on the grounds that in clonal plants genet size is closely corollated with the lifetime sexual reproductive output of the genet, because a genet's seed production is equivalent to its number of ramets, multiplied by the average seed production of each ramet. So long as the new parts grown are such that they will produce seeds, then growing larger is a way of becoming fitter.

So the demographic method is essentially a kind of *genet view* (Janzen 1977; Harper 1977, 1985; Cook 1979a; Harper et al. 1979; Cook 1985; Jackson and Coates 1986; Eriksson and Jerling 1990; Ariew and Lewontin 2004) which views the individual as the whole mitotic product of a single sexually produced zygote. "Through the eyes of a higher vertebrate unaccustomed to asexual reproduction, the plant of significance is the single stem that lives and dies, the discrete physiologically integrated organism that we harvest for food and fibre. From an evolutionary perspective, however, the entire clone is a single individual that, like you or me, had a unique time of conception and will have a final day of death when its last remaining stem succumbs to age or accident." (Cook 1980, p. 91) Vegetative iteration of the parts of a genet is viewed as a form of growth, rather than genuine reproduction. This growth is used as a proxy for fitness, because it is taken to correlate with reproductive output, but it is not taken as constituting reproductive output. In the same way that healthy heart cells might correlate with reproductive fitness in humans, a high rate of vegetative growth is seen as contributing to the viability and fecundity of a plant individual.

Clonal growth is posited to be adaptive at the level of the genet in several hypotheses. It is suggested that an individual genet can use clonal growth to increase its life span, or reduce the risk of mortality (Cook 1979a). So long as a single module remains viable, the genet can survive, so having parts spread about spatially reduces the probability of a single event destroying them all. Cook also claims that clonal plants might actually be spreading themselves out in order to exploit a wider range of environmental resources (Cook 1985) especially where those resources have a patchy distribution in space or time. This has been called a kind of foraging behaviour in clonal plants (Silvertown and Charlesworth 2001). There is evidence that some plants might actively bias their clonal proliferation in an environmental gradient in order to maximise resource efficiency in a kind of active habitat selection (Salzman 1985; Williams 1986).

The genet view has failed to secure a lasting victory over metapopulation views however. Recent authors have argued that there is no basis to the claim that vegetative propagation is an inferior or pseudo-form of reproduction. Supporters of a *ramet* or *module view* (Hamilton et al. 1987; Fagerström 1992; Pan and Price 2002; Pedersen and Tuomi 1995; Poore and Fagerstrom 2001; Tuomi and Vuorisalo

1989b; Winkler and Fischer 1999) would claim that when a new aspen ramet grows from the root stock of its clonemates a new individual has truly been born. They claim that it is wrong to think that the parent clone has simply grown one more part. "Growth alone is an important component of fitness in modular organisms" (Tuomi and Vuorisalo 1989a, p. 230). If this is right, then clonal iteration doesn't just correlate with fitness, it actually partially constitutes it. Fagerström recommends defining generations according to the meristem cycle, rather than the zygote cycle. Ramets are then considered to have two types of offspring—seeds and clonal ramets (Fagerström 1992). On this view both seeds and daughter ramets need to be counted, and the fitness of a ramet is the sum of these numbers[11] (whereas on the genet view, ramets are counted and multiplied by average number of seeds to get a genet-level fitness attribution.)

Some pragmatic or intuitive reasons are invoked in support of a ramet or module perspective on plant individuality. The lower level units are more obviously analogous to unitary organisms in various ways. Ramets and modules have proper life cycles—they reach maturity and senesce, while for genets the notion of a life history stage is largely meaningless (Watkinson and White 1986). Ramets have specialized, differentiated parts and reproductive autonomy. Tuomi and Vuorisalo argue that modules are the only units which can truly be said to reproduce, and that larger scale units are significant only in so far as they constitute domains of interaction, which don't necessarily overlap with genets (Tuomi and Vuorisalo 1989b).Fagerström argues that the significant fact in deciding whether to call some part a new individual or not is not its origin, but its evolutionary potential. He says it doesn't matter what size a propagule is, or how it was produced: only whether it is totipotent. On these grounds, vegetatively produced ramets qualify as genuine individuals (Fagerström 1992).

Is there any substance to this conceptual dispute between the ramet[12] and genet views? Just as some people treat disputes over 'the' individual as largely superficial or language-based, and respond with some version of pluralism (Wilson 1999) or promiscuous realism (Dupré 1995), we might be tempted to think that there ought to be some compromise available where the ramet and genet views are seen as equally valid conceptual alternatives.[13] However, Pedersen and Tuomi demonstrate the mathematical nonequivalence of these views, whenever vaguely realistic assumptions are included about for example, density dependence (Pedersen and Tuomi 1995). As I will demonstrate in part six, the choice of one view over the other can have real empirical consequences.

One case over which the supporters of these views are going to have a concrete disagreement is one in which a species is known to be obligately asexual, or in which the actual rate of seedling establishment is observed to be so low that the

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[11] Usually the contribution of each type of offspring is weighted by relatedness (see Fagerström 1992) to account for the difference in heritability between sexual and clonal reproduction.

[12] For simplicity I'll use 'ramet view' from now on to include all lower level views, in opposition to 'genet view'.

[13] Wikberg argues for what she calls pluralism with respect to unit choice in plants (Wikberg 1995). However, on closer inspection her account is firmly in the genet camp—she advocates a pragmatic sort of pluralism with respect to which unit is chosen as the proxy for fitness.

species is de facto asexual. In such an instance, a demographer will have good reason to deny that the growth of the genet will correlate with its sexual reproductive output, which precludes using genet growth as a proxy for fitness. In fact any supporter of a genet view has to treat an obligately asexual organism as having zero fitness, no matter how vigorous or long-lived it proves to be. If the genet does not produce any sexually produced daughter genets, then it is an evolutionary dead end.

A supporter of a ramet based measure of fitness will, on the other hand, make a fitness attribution according to the rate at which plants give 'birth' to new, vegetatively propagated plants, and so can generate high fitness scores even when ramets rarely or never produce seeds that become established. They claim that this is appropriate for species which seem to have a high level of vigor or longevity, such as the many alien species which become very invasive and fast spreading in new environments, despite totally lacking one of the genders necessary for sexual reproduction. For example, all the *Hydrilla verticillata* in Florida are female and so cannot produce seeds, yet thanks to a combination of tuber propagation and production of 'turions', buds which drop off the plant, *Hydrilla* has become an incredibly successful invasive plant, choking water ways throughout the state (Silvertown 2005).

Critics counter that such plants are only doing well in the short term. Whilst vegetative reproduction might be advantageous in the short term, because plants can clone genotypes that are adaptively superior, in the long term these plants should suffer because they lack the recombinatorial access to new genotypes that sex provides. If the environment suddenly changes, asexual plants should be unable to adapt quickly enough to the changing conditions and will be eradicated in competition with sexual plants. Ramet theorists might deny that empirical research has borne this out. Some point out that somatic selection might be able to compensate clonal plants for the loss of sex, by providing an alternative route to evolvability (Lushai et al. 2003; Neiman and Linksvayer 2006; Clarke 2011).

These disputes remain fairly empirically intractable for a number of reasons. Both sides can find comparative studies, that compare extinction or diversification rates in sexual and asexual clades, in their favour (for example Beck et al. 2011; Johnson et al. 2011). Similarly, genet supporters predict that asexual lineages should suffer decreased fitness as a consequence of a gradual accumulation of deleterious mutations (Klekowski 2003), but researchers have failed to find particularly high levels of mutation in clonal plants (Cloutier et al. 2003; Ally et al. 2008), although this result is difficult to interpret. It could be that the *rates* of mutation are low: perhaps clonal plants have particularly effective DNA repair mechanisms, for example. Alternatively, it could be that rates are as high as elsewhere, but that the resulting genetic heterogeneity is removed by somatic selection (Pineda-Krch and Lehtilä 2004). Ally et al. do find that *sexual* fitness, i.e. the capacity to reproduce sexually, becomes degraded in long-lived, but this only proves that clonality is deleterious *on the assumption that* vegetative growth is not itself genuinely constitutive of fitness.

Plant ecologists therefore face a dilemma. Should they count genets or ramets? They know that the predictions they make about the evolutionary dynamics of the

species they are examining might well vary according to their choice. Of course, they can always just do both, wait many years, and then see which prediction was closest to the mark. Not only is this hugely time consuming and impractical, but it completely robs the concept of the individual organism of its predictive usefulness. Perhaps the ecologist could console herself that at least she will know which unit to count next time. But can she be sure that the same count will be relevant in other species? Even in the same species? Even in the same population at a later date? In part five below we will see why such assumptions are probably not generally supportable.

In this section we have seen that there are two competing views about individuality in plants. Those who adopt a genet view identify a whole clone as a plant individual, and take clonal growth to be a more or less reliable correlate of plant fitness, while those who argue for a lower level view say that modules or ramets are plant individuals, and that vegetative growth actually constitutes plant fitness. In order to properly evaluate these views, it is necessary to think about how we solve the analogous problem in unitary organisms.

In part three I introduce several formal criteria that have been used with much success to define individuality in unitary organisms, and explain why they fail to give any resolution to the plant individuality debate.

Classical individuation criteria and the failure of plants as individuals

Anyone who wants to count organisms needs to make decisions about which things to consider as mere parts of organisms, as well as about which things to treat as collections of organisms, rather than as organisms in their own right. There is no general consensus as to the correct way to make these judgments, or even as to whether a single unitary conception is possible, but there are several popular competitors (see Clarke 2010 for an extensive review). Each of the views outlined here acts as a criterion of individuality, by identifying an essential property that all organisms must possess. One of the reasons it is illuminating to ask about individuality in plants is that the usual favoured definitions of individuality are inapplicable. We then have a choice to make—fail the plants or fail the definitions. I'll choose to reject the definitions rather than to say that the notion of an individual doesn't apply to plants, but it is worth taking the time to explain exactly what those definitions are and why they fail when it comes to individuating plants.

Germ soma separation

According to this view the essential property of a biological individual is that there is a reproductive division of labour so that some parts are sterile and carry out only somatic functions (behaviours necessary for survival and growth) but not reproduction (Weismann 1885; Buss 1983, 1987; Michod 1999; Michod and Nedelcu 2003; Michod and Herron 2006; Godfrey-Smith 2009; Fagerström 1992; Martens 2010). This definition picks out all unitary organisms as individuals, as well as many social insect societies, and other higher-level groups which show a

reproductive division of labour, such as naked mole rat groups. Those entities that lack reproductive independence, so they can only increase their (inclusive) fitness by contributing to the success of some larger whole, are considered a mere part of a higher level organism, on this view.

The definition fails with respect to plants because, as we saw earlier, all plants have somatic embryogenesis, rather than terminal somatic differentiation. Mutations that occur in just about any plant part can be passed on to subsequent plant generations, both mitotically—by stoloniferous or adventitious growth—and meiotically—if they end up appearing in the specialized ova or pollen. No plants parts are complete evolutionary dead ends, in the way that nerve cells or some worker ants are.

Germ soma separation is probably the most popular criterion for defining the individual organism but it can offer no reason for preferring either of the demographer's positions on plant individuality.

Developmental bottleneck

This is another very popular definition. The bottleneck view identifies the entire mitotic product of a bottleneck stage in the life cycle as the individual (Dawkins 1982; Maynard Smith and Szathmáry 1995; Godfrey-Smith 2009). The organisms of any species which is obligately sexual count as individuals by this criterion, because a fertilised zygote is always unicellular. But development from an apomictically produced seed, as in self-compatible plants, or parthenogenetically as in aphids, also qualifies under this definition. The bottleneck view is able to accommodate insect societies and also separate identical twins as individuals so long as the embryo splits into sufficiently small pieces (Huxley 1912).

The definition isn't very helpful when it comes to plants, however. Apomictic and sexual reproduction include a single celled stage, whereas vegetative reproduction always involves a multicelled propagule such as a stolon or bulb. So we might interpret the bottleneck view as saying that new plant individuals are born from seed, and all other forms of expansion in between are just growth. This is close to the genet view, but not identical, since the genet view considers only development from sexually produced, not apomictic, seeds to produce new individuals, at least in theory.

However, Dawkins, a notable defender of the bottleneck view, understands matters differently. With respect to *clonal* plants in particular, he denies that the whole mitotic product of a seed is an individual organism, on the grounds that multicellular runners are too efficient at transmitting mutations. He claims that the appropriate unit, whenever levels of mosaicism are high, is the cell (Dawkins 1982, p. 260). So the bottleneck view does not give an unequivocal verdict with respect to plants—it depends on what the motivation behind the view is.

An additional problem with the bottleneck definition is that plants have not one but two single celled stages in their life cycle. They alternate between two multicellular generations—a gametophyte and a sporophyte—with two single celled stages in between (a spore and a gamete or zygote). Different forms are dominant in different types of plant. The bottleneck definition of the individual would seem to

have to say that the gametophytes and sporophytes are distinct individuals, with unpalatable implications for the notion of parent-offspring similarity (Godfrey-Smith 2009, p. 78).

Appealing to bottlenecks won't give us a straightforward resolution to the demographer's dilemma either.

Sexual reproduction

Sex is the process from which genets are born, so this conception identifies the individual with the genet: the entire mitotic product of a sexually fertilised zygote (Huxley 1852; Janzen 1977; Cook 1979b). Many people have objected that this criterion is useless in groups such as bacteria, where sex, as normally understood, does not occur (Godfrey-Smith 2009). Furthermore, the definition must confront the fact that sexual and asexual reproduction are really two ends of a continuum, rather than discrete alternatives (Sterelny and Griffiths 1999). It is a further question *which* precise aspect of sex—syngamy, for example, or meiosis—is significant. More generally its not clear whether a sexual view can offer any additional justification for the genet view, or whether it simply restates it. What arguments are there for taking a sexual view?

Janzen's version of the sexual view, on the other hand, was motivated by the idea that individuals should be genetically unique (Janzen 1977).[14] In so far as this is true, they ought to recognise that other mechanisms enhance an organism's genetic uniqueness too. Somatic mutation and selection can cause ramets to become genetically distinct from each other, even if they are mitotic descendants from a common zygote. Polyploidy is another source of genetic novelty that doesn't depend upon sex, although when it is passed on sexually it can create new organisms that are much *more* genetically unique, than can regular recombination alone (Niklas 1997). Polyploidy can create organisms that are so genetically different from their parents that they are unable to breed with organisms from their parent species.

The sexual view is probably more popular with botanists than it is with other contributors to the individuality debate, but in so far as it reiterates the genet standpoint, rather than situating that view on some firmer theoretical foundations, it doesn't really contribute anything to the genet/ramet debate. Digging into the motivations behind a sexual view is more profitable however, and makes us see that other genetic considerations are likely to count for as much as sex. If somatic selection can make ramets genetically unique, just as sex makes genets unique, then both units are still on the table as potential individuals.

Physical boundaries

Many authors have argued that individual organisms are always physiologically discrete, spatially bounded and/or localized (Hull 1978, 1980; Brasier 1992; Huxley 1912; Gould 1991; Sterelny and Griffiths 1999; Leigh 2010; Buss 1987). Relatively

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14 Though see, Gorelick and Heng (2011) who are motivated instead by the significance of epigenetic reset.

little theoretic underpinning has been offered for this view; it is supposed to be close to an everyday or intuitive notion, according to which organisms have edges and are not gappy. Social insect colonies and swarms of aphids are *not* individuals, on this view.

Aclonal plants are often well individuated, physically. Trees, plants and bushes have fairly clear edges, often wrapped in bark, ending in leaves at one end and roots at the other. Clonal plants, however, may or may not maintain physical connections amongst their ramets. The boundary view would say that genets are individuals only when their ramets remain attached. This is *not* an obvious matter, to be settled by simple observation. Aspen forests look like they are composed of physically separated trees, but if we look underground, we see a network of propagating runners between them. Furthermore these connections are rather chancy. The runners are routinely interrupted by land subsidence, or the activities of burrowing animals. A bounded individual today may be a mere collection of individuals tomorrow.

Groups of phylogenetically distinct parts, such as the plant chimeras created by grafting, can be individual organisms on this view. Many grape varieties, for example, are in fact chimeras: in which one vine has been grafted or fused with the stem of another. Grafting occurs naturally too. Aspen frequently graft roots with unrelated genets. In the site studied by Jelinkova et al. inter-clonal grafts were found to be just as common as intra-clonal grafts (Jelinkova et al. 2009).

The boundaries view isn't an easy option for plants, and it makes plant individuals somewhat contingent and arbitrary, but it can deliver clear verdicts: sometimes genets will be individuals and sometimes ramets. Sometimes they will even be multi-genet groups.

Immune response

According to this view, parents are distinguished from offspring and from other organisms in terms of immune response or allorecognition (Loeb 1921, 1937; Medawar 1957; Pradeu 2010; Tauber 2009; Burnet 1969; Metchnikoff 1905).

Plants differ from vertebrates in that they lack an adaptive immune system, but they do still have the capacity to mount an immune response against threats to their integrity. Each cell has an innate immunity based on proteins which can recognize 'modified self' (Jones and Dangl 2006). This enables the cell to respond defensively to parasites and herbivores. The proteins are probably used to prevent self-fertilisation as well (Nasrallah 2005).

Plants also exhibit various forms of 'induced resistance', in which an immune response is elicited in undamaged parts of a plant. Signals are carried between cells, via transmembrane receptors, but also between different genets, by air transport of volatile compounds (Vallad and Goodman 2004; Eyles et al. 2010).

If we interpret the immune response view as individuating organisms according to shared immunity, then the indiscriminate communication of induced resistance means that the plant individuals can be very large: as far as the wind can blow the volatile hormone molecules.

Pradeu's version of the immune response view spells out the criterion in terms of immune *rejection,* however. He specifies that anything that is not rejected by the organism's immune system, despite being in physical contact with it, should be

considered a proper part of that organism (Pradeu and Carosella 2006; Pradeu 2010). On this formulation systemic acquired resistance fails to confer individuality on whole fields of plants at a time. Instead, the immune response view overlaps with the boundaries view.

Reject the category?

The conclusion of this brief survey is that the classical criteria for individuality do not help us to choose between the ramet and the genet view of plants. The different criteria pull in different directions, and in some cases we were even pulled in different directions by a single definition.

A plausible verdict might be that for at least some plants (the more modular ones) no single notion of the biological individual applies. Maybe there is no such thing as *the* individual organism in the plant Kingdom? This conclusion does not help anyone who needs to count individuals in order to generate accurate and predictive models of selective dynamics. There might be numerous correct ways to *represent* those dynamics—where higher and level models are mathematical isomorphisms of one another, for example (Kerr and Godfrey-Smith 2002). But where two models of plant evolution are *not* mathematically isomorphic, because taking genets to be individuals has different empirical consequences from taking ramets to be individuals, time will prove at most one of these models to be correct. We want to be able to say something general about which one it will be.

So given a choice between abandoning the criteria or abandoning the relevance of the organism concept for plants, I am going to think seriously about failing the classical criteria. In other words, I will argue that those definitions to do not identify properties that are essential to biological individuals after all. On the other hand, I don't want to throw away those criteria altogether: that they are popular suggests they are getting something right. My strategy will be to conduct a closer examination of the classical criteria to try to find out the reason why they are successful in non-plant domains.

Here is a sketch of the argument to come;

First I will argue that the classical criteria achieve their success by homing in on mechanisms which constrain the hierarchical level at which selection is able to act. I say that it is this *effect* of the mechanisms picked out by the criteria that is really doing the work, and that accordingly if we find mechanisms in plants that have the same effect, then we should call them individuating mechanisms too, however different they look. Then in part five I will describe the sorts of mechanisms I have in mind, which I think are at the root of the demographer's dilemma.

Part Two

Towards a new criterion of individuality

In this section I first argue that we can usefully reinterpret the classical criteria of individuality as identifying mechanisms which constrain the extent to which

populations of biological units exhibit heritable variance in fitness. Then I develop a new criterion of individuality which centres on the identification of mechanisms with this effect.

I am going to look again at the classical criteria for defining the organism, this time in the context of a straightforward vertebrate with which we are all familiar: a pig. The aim is to set out what the properties identified by the classical criteria actually do—what effect they have on the biological objects that instantiate them.

Pigs and germ soma separation

Germ soma separation is supposed to give us a reason to call pigs organisms, but not to call pig cells organisms. But what is it about germ line separation that excuses us from the, at best, laborious, and, at worst, impossible, task of counting and genetically profiling pig cells in order to work out what is going on in pig evolution?

The Weismannian orthodoxy is that somatically differentiated cells are irrelevant to evolution: they act as a mere transient vehicle for the germ line (Weismann 1885). Whilst there is general agreement that the evolution of germ line separation is significant in major transitions because it eliminates conflict amongst the cells of multicellular organisms (Buss 1987; Michod 1999; Godfrey-Smith 2009), there is less consensus on the details of how it does it.[15] It is sometimes said that soma cells are evolutionary dead ends, because the only way they can modulate the representation of their genes in future generations is by influencing the fitness of the aggregate individual: the pig itself.

I argue that the key thing here is that somatic cells cannot pass their traits on to future pigs. While somatic mutants (cancers) can transmit their traits to mitotic offspring, this is heritability with a limited shelf life. When the pig dies, which at some point it surely will, the mutant will go extinct. Germ cells, on the other hand, lack this death sentence—their lineages can go on and on indefinitely. This gives their traits a sort of long-term or open-ended heritability that somatic cells lack. And because heritability is one of the necessary ingredients of evolution by natural selection (Lewontin 1970), a population of cells will not evolve—it will not exhibit any response to selection—without it.

I suggest we understand germ soma separation as relevant to individuality in so far as it constrains the action of natural selection in the populations of cells which instantiate it, by limiting the heritability of somatic traits. Neglecting to track evolution at the cell level will only result in a skewed picture of evolution by natural selection if selection operates within populations of cells. Counting germ soma separated entities then, will give an undistorted representation of pig evolution *in so far as* germ soma separation successfully prevents selection from acting at the between-cell level in pig populations.

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[15] For example Michod says germ separation prevents somatic cells from having fitness at all, whereas Godfrey-Smith says it merely decouples their fitness from their intrinsic character. See Clarke (2010) for more details.

Pigs and developmental bottlenecks

The primary argument given for the role of bottlenecks as individuating mechanisms points to their role as mutation sieves. Dawkins (1982) defends the bottleneck view because of the role bottlenecks play in removing genetic variation. When a lineage passes through a single celled bottleneck, then the genome of only one cell is transmitted to the next generation, regardless of how many different genomes were present in the parent. All the cells in the new individual will be derived mitotically from this one cell, and this increases the degree to which that individual will be genetically homogeneous in adulthood.

Selection can only act on populations in which there is heritable variance in fitness and, by removing genetic variance, bottlenecks remove a major source of such variance in biological populations. So once again, we can see that in so far as bottlenecks incapacitate natural selection from acting on those populations of pig cells that we call pigs, they legitimise us in electing not to bother counting pig cells.

Pigs and sexual reproduction

Why is sexual reproduction relevant to pig individuality? For Janzen sex is relevant because of its effect on the genetic uniqueness of pigs. He argued that aphids born from parthenogenesis are mere parts of a single large, spatiotemporally scattered evolutionary individual (Janzen 1977). The rationale of his view is that sexual reproduction produces novel (unique) genotypes by putting genes in new partnerships with other genes. Where this is absent, a clonally produced organism is just more of the same as its parent.

We can understand this in terms of heritable variance in fitness again. Sexual reproduction increases the capacity for populations of pigs to undergo evolution by natural selection, by increasing the extent to which those populations exhibit genetic variance. If we count only at the level of pig groups, without bothering to track change at the level of pigs themselves, we will overlook this selection. So we had better not neglect to count the products of sexual reproduction.

Pigs and physical boundaries

Pigs have parts that are physically connected to or contiguous with each other, and they are separated from everything else by skin. But what effect does this have on the evolutionary dynamics of pig populations?

One effect is to fix the boundaries of different populations of pig cells—skin prevents the cells in one pig from migrating over to a new pig. In group selection theory, we know that one factor that is highly significant in determining the power of group selection, relative to that of lower level selection, is the amount of mixing or migration across groups. If the rate of migration is too high then group-level heritability is too low for there to be a group-level response to selection. Physical boundaries or barriers around a collection of parts can help to keep within-boundary variance lower than across boundary variance simply by preventing mixing or migration between the groups (Leigh 2010).

Contiguity can also fix the possibilities for selection on populations of objects simply by eliminating variance between them. If two entities are physically stuck together, and cannot separate, so that even when they replicate, they produce a daughter entity each and these daughters are also physically stuck together, then there is no room for variance *in fitness* between the two entities. The rate of reproduction for one is precisely tied to the rate of reproduction of the other. Not just any physical attachment will do though—it must be permanent, and of a kind that reproduction becomes perfectly synchronised—then the genes of the two entities are 'co-dispersing' (Frank 1997).

So physical boundaries can be important in determining the scope for natural selection to operate on biological populations in so far as they influence the amount of variance within and between groups. Pig cells cannot migrate in and out of distinct pigs, while pigs usually can migrate between different pig groups. So pig skin gives us a justification for counting pigs, but not pig cells or pig groups, in so far as it makes it easier for selection to act between pigs, than between pig cells or pig groups.

Pigs and immune response

Vertebrates such as pigs have an adaptive immune system which allows them to accept skin grafts and other organ transplants from themselves, and from close relatives, but not from other conspecifics (Medawar 1957).[16] Although the main function of the vertebrate immune system is widely accepted as being defence against pathogens, it also plays a role in policing against cancers by identifying and destroying somatic mutants which fail particular identity checks. In this latter role we can recognise the immune system as complementing the developmental bottleneck in reducing the amount of genetic heterogeneity present in vertebrates. Immunity also plays a similar role to boundaries: by policing the borders of an organism, controlling what comes in, the immune system influences the level of migration into an organism. Many foreign entities are admitted—most organisms healthily carry around a vast number of exogeneous entities within their bodies, especially their digestive tracts (Pradeu 2010)—but the aim is to exclude those who would compete for resources with the host. The role that immunity plays in restricting migration across an organism's borders can be seen very clearly in the case of marine invertebrates such as *Stylophora pistillata,* in which unrelated individuals are able to fuse with one another to form new, larger units with shared vascular systems (Amar et al. 2008). The control of variation is also fairly central for plant immunity, because one of its central functions is to discriminate between self and non-self in order to support self-incompatibility (Nasrallah 2005).

It may not be reasonable to say that control of heritable variance in fitness is the primary function of the immune system in pigs. But it is in virtue of this effect that the immune individual overlaps with the evolutionary individual: the unit which we should count in order to understand evolutionary dynamics.

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[16] Unless immune-suppressant drugs are administered to prevent the graft from being rejected.

Unity in pigs

A great deal of unification between these classical views on biological individuality can be achieved, if we interpret them in evolutionary terms. It is easy to see why the property picked out by each view is salient, and generates an effective criterion, if your primary goal is to identify the unit that needs to be counted in order to generate accurate and predictive models of selective dynamics. Each definition succeeds in picking out the optimal unit for evolution tracking purposes in the same way: by identifying a mechanism which successfully manipulates heritable variance in fitness amongst pig parts so that evolution by natural selection can only occur at one level.

On the other hand, this analysis should also make it obvious that the actual properties picked out by the classical views are red herrings, in so far as we are interested in finding *general* criteria for counting organisms. Pig cells are redundant as demographic units, not because bottlenecks prevent selection from acting within pigs, but just because there is no selection acting within pigs. In other words, it doesn't really matter what property or mechanism succeeded in preventing intra-pig evolution, only that *something* did. So if we want to decide whether or not we need to count plant ramets, we ought to be asking, not whether or genets have bottlenecks, but whether or not they have *some* property or mechanism which succeeds in preventing intra-genet evolution.

Next I develop a methodology for identifying the counterparts of the classical criteria in plants, based on the central insight that individuation mechanisms function to constrain heritable variance in fitness.

Individuation in the abstract

To decide what units to count we need to locate the levels at which selection is able to act. I suggest we do this by looking for mechanisms which eliminate heritable variance in fitness. The existence of such mechanisms enables us to rule out certain units as worth counting, just as the bottleneck gives us reason to rule out cells as worth counting in pigs. We can break this down, because there are different components to heritable variance in fitness, and therefore different properties on which the relevant mechanisms might act.

As is so often the case in philosophy, half of the battle in solving the problem of biological individuality is won if we can just succeed in asking the right questions. The questions that need to be asked in order to establish whether a given population should be considered to be composed of separate biological individuals, which need to be counted separately, are;

Question 1: Are there non genetic but heritable variations in fitness within the population?
Question 2: Is there genetic variance within the population?
Question 3: Does the genetic variance give rise to fitness differences in the population?

Question 4: Is the genetic variance within the population heritable, in the long term?

If a collection of living things exhibits a mechanism which causes the answer to one of these questions to be negative, then we will call it an individuating mechanism. Furthermore, we will say that the collection in fact constitutes an individual organism, in virtue of the mechanism, and we will count it, but not its parts, when we conduct demographies of that species of living thing.[17]

In the next section I turn the attention back specifically to *plant* individuality, and to the demographer's dilemma. Firstly, I identify two insights which originated in the ramet/genet debate which I interpret as valuable precisely because they focus on the existence of properties which, through their consequences for the heritable variance in fitness of different plant units, invalidate some of the assumptions of the genet view. Then I apply my methodology more systematically, identifying and classifying some concrete examples of plant-specific individuation mechanisms.

Individuation criteria for plants

The ramet/genet debate yields two key insights that bear on these questions.

Two insights

The insight that I attribute to (Fagerström 1992) but see also (Hadany 2001) gives us reason to worry about calling genets individuals. The point is that because of the combined phenomena of mosaicism and somatic embryogenesis, the answers to questions two (is there genetic variance?) and four (is the variance heritable?) are positive, to a greater or lesser extent, for *all* multicellular plants. Fagerström's scepticism about genet-based demographies stems from his awareness that mosaicism and somatic embryogenesis together make somatic selection possible, and this, in turn, makes counting genets an unreliable method of tracking evolution by natural selection in plants.

As I indicated in part one, the jury is still out on somatic selection. It might be that intraorganismal selection acts as a sort of evolutionary engine, accelerating the rate of genetic change and adaptive evolution in plants. This would be hard to show empirically however. One way to test the evolutionary power of somatic selection is to compare the rate of molecular evolution in different groups, but it is difficult to measure this rate in plants (Whittle 2006). And while it would be nice to have more comparative data—comparing rates of evolution in clonal and non clonal plants, and in angiosperms compared to gymnosperms, for example –it is also not obvious how to interpret such data. It might be that there is an increase in speed thanks to somatic

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[17] This is framed negatively, for simplicity. We might also find mechanisms whose effect is to *increase* the extent to which these questions gain positive answers. Those qualify as individuating mechanism also, but for a different hierarchical level. Those mechanisms give us reason to call the collection of parts which possess them a collection of organisms, rather than an individual in its own right, and to count at the lower level. Both kinds of mechanism might interact to determine the level at which evolution by natural selection occurs.

selection which exactly counterbalances the decrease in speed due to deleterious/neutral mutations causing drift. This is why quantitative models of somatic selection are valuable (Folse III and Roughgarden 2011). Yet even if somatic evolution is primarily a matter of drift this could still undermine genet-level heritability in plants. To see this, consider a self-incompatible, aclonal plant, with many branches, which produces seeds on each branch. Now suppose there is mosaicism present: some branches will carry an allele of interest, lets call it allele a, and some will not. There may or may not be a visible difference between the types, but assume that branches of both type produce seeds. The genet thinker comes along and counts the number of offspring, i.e seeds produced by the genet—the whole plant. He assumes that around half the seeds will carry allele a because meiosis is fair. But this isn't right: less than half the seeds will carry allele a because some of the seeds were produced by branches that don't carry allele a. So in this very minimal sense, within-plant heritable variation reduces plant-level heritability.

For these reasons, higher-level assays of genetic or trait-type in plants are potentially misleading. Sampling methods which generalise across large units only work if there is some reason to believe that the sample is representative: that there is no variation, so that the unobserved parts are the same as the observed parts, or that an unbiased portion of the variation has been sampled. Mosaicism alone undermines these assumptions in the plant case. In germ separated organisms we have good reason to ignore most of that variance, because the gametes are the only cells whose traits are heritable, and their type is most likely to be identical to the majority of the soma. In plants we have no such excuses.

This insight has been obscured by proponents of the genet view, who treat the genet as an adequate approximation of the unit that bears a unique and homogeneous genotype. A key criticism they make of lower level approaches is that they tend to ignore genetics.[18] Given the aim of understanding how natural selection acts on plant populations, a neglect of genetic data might seem inexcusable. But the genet view, on the other hand, makes improper inferences about the genetic structure of a population, in assuming that all the mitotic products of a single zygote share a single genotype. *If* this were true, then it would be right to treat all a genet's parts as mere parts. After all, no selection can occur in a population if that population lacks heritable variance in fitness.[19] However, as we have seen, this is not the reality. Modules, ramets and genets may all form populations whose parts possess heritable variance in fitness, which means that we need to take account of the possibility that each of these populations itself undergoes evolution by drift or by natural selection.

Some might reply that nonetheless a *genetic* view—which equates the individual with whichever unit is genetically unique and homogeneous, for whatever reason—is a correct view on individuality. A biomass view, on which expansion alone can constitute fitness (Van Valen 1989), is similar. There are two reasons why a genetic

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[18] Although this is not true of all advocates of the lower level view, many of whom insist on an integrated fitness measure that incorporates genetic information with the count of clonal offspring (Pan and Price 2002).

[19] Setting non-genetic sources aside for a moment.

view is not worth adopting. One problem is that genetic variation is not the only possible source of heritable variance in fitness in a population. The second is that a genetic view is not operational. If there was a way to accurately and constantly monitor the genotype of every cell in a given sample of biological matter, then a genetic view would be useful. Defining individuals directly in terms of heritable variance in fitness fares no better in these, pragmatic, terms. Homing in on *mechanisms,* however, changes things. By following a simple checklist, we can rule various units out simply and straightforwardly, according to the presence of common and well understood mechanisms.

Scientists deal with idealized concepts all the time, and might feel that mere operational difficulties aren't relevant. However, the seriousness of the operational obstacles, combined with the widespread tendency to forget that the genetic individual is an idealized, not an actual, genet, starts to render the concept not just useless but actively misleading. In the real world, acting on the assumption that the genetic individual is approximated by a genet—the matter that is derived mitotically from a single zygote—will not do.

The second key insight, which I attribute to Tuomi and Vuorisalo, bears on question 3, concerning whether or not genetic variance within a unit gives rise to fitness differences (Tuomi and Vuorisalo 1989a, b; Pedersen and Tuomi 1995). The insight is that certain types of interaction and integration between units can constrain the extent to which genetic variance gives rise to fitness differences. They say that "interactive units (modules, organisms and groups) may provide potential levels of phenotypic selection if they have a distinguishable causal impact on reproduction at the level of reproductive units." (Tuomi and Vuorisalo 1989b) Genetic variance is only going to be decisive in determining individuality if it gives rise to heritable variance in fitness. If competition between the units is suppressed so that fitness differences cannot arise, then genetic variance is not relevant to individuality, because it is not sufficient for the operation of natural selection. In some plants interactions between different parts or ramets might act to suppress fitness differences amongst them. When the modules of a plant are entirely dependent on external resources translocated to them from other parts of the structural individual, Tuomi and Vuorisalo claim that it is the entire structural individual, rather than the modules, which acts as the ecological interactive unit. The insight I derive from this is that certain sorts of interactions between ramets, which may not be salient from the perspective of someone who is fixed on counting ramets, may influence plant evolution. Note that it is not appropriate to automatically think of the unit whose parts are engaging in fitness suppressing interactions as the *genet*, because the unit whose parts are interacting may be larger or smaller than the mitotic product of a zygote.

This insight effectively introduces an additional unit into the genet/ramet dispute. Tuomi and Vuorisalo refer to the unit whose parts are engaging in interactions that suppress fitness differences as the *structural unit* (Tuomi and Vuorisalo 1989a). The suggestion is that sometimes a structural unit, rather than a ramet *or* a genet, may be the appropriate counting unit in plants.

Anyone who endorses genets as exclusive levels of selection overlooks Fagerström's insights, yet Tuomi and Vuorisalo's insight gives us caution against

rushing to a simple ramet view. Although these constitute very general and important insights, the question of individuality with respect to concrete cases can only be settled via detailed examination of the case in hand.

Filling in the empirical details

I now survey some empirical detail to pull out examples of the sorts of things which plant scientists might usefully seek out when they are trying to ascertain which units to count. However, it should be clear that the main conclusions of my argument do not depend on the empirical claims made here. A conditional claim is all that is needed:

> *If* it is the case that the mechanisms described here have a robust effect upon the extent to which plant units are able to vary in ways that are heritable and which affect their fitness, *then* these mechanisms are hallmarks of individuality.

They are the plant equivalents of bottlenecks and germ sequestration. They can be used as flags or markers, instructing the biologist where to count. My purpose in this section is to flesh out this claim a little, and make my own suggestions about what sorts of things are prime candidates for playing the individuator role in plants. But my central argument does not stand or fall with the accuracy or veridity of these particular suggestions. Only if it is shown ultimately that no such mechanisms exist at all, then I will have to revise my account, and say that plants, after all, do *not* express biological individuality.

In order to generate plant-specific criteria for individuality, to complement those that we have for unitary animals, we need to identify mechanisms or properties which determine the answers to the four questions about heritable variance in fitness. In other words, we need to look for evolved plant features that determine the level at which selectable populations of units occur. The questions give us four ways in which that determination can work: by controlling extra-genetic variance, or by controlling genetic variance, and so on. For example, we might ask: Do aspen ramets have mechanisms/properties which eliminate genetic variance amongst their parts?

Note that the questions are unlikely to have answers that generalise across all plants. There is likely to be a lot of variation across different plant species about the hierarchical level at which most variance occurs. On the other hand, we may be optimistic that there will be patterns to this variation, as different species respond to features of their environment in predictable ways.

Here I will not say much about the first question, concerning extra-genetic sources of heritable variance in fitness. Transgenerational epigenetic inheritance is known to be an important source of heritable phenotypic variation in clonally propagated crops and trees where it can create management problems. For example, in Norfolk Island Pine (*Araucaria heterophylla*), cuttings from orthotropic (vertical) or plagiotropic (horizontal) axes inherit the growth trait of the axis from which they were prepared (Robbins 1964). For now I direct the reader to the growing literature on ecosystem engineering or niche construction (Odling-Smee et al. 2003), on

epigenetics in plants (Rival et al. 2010) and on the extent to which epigenetic effects are significant factors in evolution by natural selection (Jablonka and Lamb 2005; Jablonka and Raz 2009).

I also assume that question four—concerning whether genetic variance is heritable in the long term—is practically always positive in plants, because of somatic embryogenesis. There are just two questions left to examine and I will treat them in turn.

Question 2: Are there mechanisms which constrain the amount of genetic variance that occurs at different hierarchical levels in plants?

If my reasoning is correct so far, then plant genets, ramets and modules have some individuality if they exhibit mechanisms whose effect is to decrease the amount of genetic variance within the unit, or, equivalently, to *increase* variance *across* units. Sex is one such mechanism. Sexual outcrossing increases the extent to which the product of the zygote (the genet) is different from other genets, creating a novel genotype by recombination. Clonal plant genets, whose development includes a very large number of cell divisions because they can have very long lifetimes and grow to very large sizes, are normally likely to exhibit much more within-genet genetic variance than genets of non-clonal, determinate and short lived plants. This means that they will often have a lower degree of individuality than do non-clonal or short lived plants. The efficacy of sex in individuating plants depends on the amount of genetic variation available in the genet population. In very tiny populations, genetic variation can be too low for sexual recombination to have much influence. For example, the population of Wollemi Pines is so small that even though trees reproduce sexually, the trees in the population are about as genetically similar to each other as they would be if they were clones (Peakall et al. 2003).

Apomictic reproduction will decrease genetic variation within genets by acting as a bottleneck, but it will not produce additional variation between genets, compared with sexual reproduction. Whether the apomictically produced plant varies from its 'parent' depends on the degree of somatic mutation in the parent.

Another mechanism that will affect the amount of genetic variation present in a plant population is polyploidy. This is a means by which sterile species hybrids can become fertile when the chromosome number in their germ cells spontaneously doubles (Niklas 1997, p. 88). This offers a route by which genet-level variance can be dramatically increased, even for plants whose populations have become severely inbred.

Other mechanisms that will affect the amount of genetic variance exhibited by populations of plant units are DNA repair mechanisms, somatic selection and bottlenecks. In vegetatively reproducing plants, larger propagules will have the effect of increasing within –ramet genetic variation, because larger runners are more efficient at carrying mosaicism.

An additional candidate for an evolved mechanism whose effect is to constrain the extent to which different plant units contain genetic variance is the meristem itself. This is the multicelled plant module from which most plant growth originates. Meristem structure varies across different plant groups, and can influence levels of genetic variance by controlling the extent to which meristem cells are able to

compete with one another. Monopodial meristems, as found in Pteridophytes (ferns, horsetails and lycopods), do not support cell-level selection at all because they have a single apical meristem cell which acts effectively as a single celled bottleneck (Hughes 1989; Lyndon 1990).

Stochastic meristems, which are found in gymnosperms (Cycads, conifers and Gingkos) do support a limited amount of between-cell selection because there is a pool of meristem cells which divide several times before the ones destined to differentiate are picked stochastically. Mutants which divide faster can easily spread through the pool of meristems and gain themselves better than average chances of being picked to remain in the germ line (Klekowski and Kazarinova-Fukshansky 1984; Pineda-Krch and Fagerström 1999).

Angiosperms, or 'flowering plants,' share a more complex meristem structure. They are stratified, so that the cells are compartmentalised into isolated subpopulations (Fig. 2).

Cells can compete within their layers, so that mutants can spread through the very local population, but no further. This means that the meristems themselves can become mosaics, with different layers being dominated by different mutants. These effects are occasionally visible in flowers or fruits that have stripes or patches of different colouration. Meristem stratification therefore increases a plant's capacity for carrying genetic variance.

In models the overall effect of meristem stratification is to facilitate the selective loss of deleterious mutations, as well as the fixation of beneficial mutations (Gill et al. 1995; Pineda-Krch and Fagerström 1999; Pineda-Krch and Lehtilä 2002; Folse III and Roughgarden 2011), which makes meristem stratification a good candidate for an adaptation whose function is to increase a plant's capacity for intra-organismal selection. This is still controversial, however, as is the general evolutionary significance of somatic evolution (Clarke 2011). What seems clear is that plant meristems have an effect upon the extent to which plants carry heritable variance in fitness. If this is so, then I argue that consistency obliges us to call
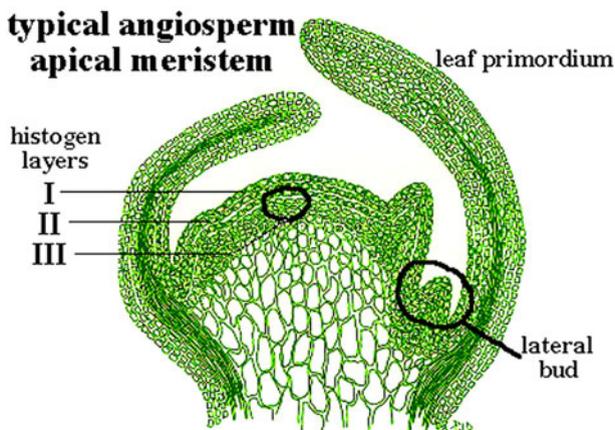

Fig. 2 Stratified plant meristem. Photo credit to Dan Lineberger (http://aggie-horticulture.tamu.edu/tisscult/chimeras/chimeralec/chimeras.html)

stratified meristems individuating mechanisms, and to consider plant cells as having a degree of individuality in virtue of them.

Question 3: Are there mechanisms which constrain the extent to which genetic variance gives rise to fitness differences in plant units?

Tuomi and Vuorisalo point out that "a continuum of modular organisations can be envisaged, from those in which the modules are completely autonomous units to those in which they are entirely dependent on external resources translocated from other parts of the structural individual" (Tuomi and Vuorisalo 1989a, p. 228). If the parts of a genet are unattached to each other and have their own stems, so that they are self sufficient in terms of both root and shoot products, then they might very easily start to transmit mutations to subsequent generations at a different rate from their vegetatively produced 'clones'. When plant parts share a stem, on the other hand, so that they are entirely dependant on one another, then its hard for any part to do much better than the others. Similarly, when ramets remain connected to each other via the stolons or runners from which they were originally grown, they can exchange resources and nutrients, and then disaster in one part of the structural unit will be felt in all the other parts. "Trees, even seed-regenerated ones like jack pine, are not independent individuals, and may directly affect the growth of their neighbors by forming root grafts." (Tarroux and DeRochers 2011). These connections are just the sort of phenomena which can individuate a higher-level structural plant unit, by decreasing the extent to which genetic variance between the parts gives rise to fitness differences among them.

Some clonal plant species invest in maintaining the connections between ramets, while others do not. It is always costly to prevent the connections between ramets from decaying, so why do it? Pitelka and Ashmun predict that different environments will favour different integration strategies. Single stemmed units are more likely to be found in dense and competitive forest canopies, where the advantage of increased height is key. In environments that are patchy in both space and time, such as sand dunes, we expect integration in which young ramets in favourable sites are supported, but old ramets in unfavourable sites are not. The 'phalanx strategy' is probably best in stable environments where competition for space is intense, and resources are diverted to prop up weak ramets in order to prevent competitor species from gaining a foothold. Environments that are patchy in space but not time, on the other hand, should select for a 'guerilla strategy', in which integration persists in smaller groups (Pitelka and Ashmun 1985; Oborny et al. 2000).

However, investment in connections does not automatically confer individuality at the level of the structural unit. This is because the connections can be utilized for different types of interactions. Sometimes resources are exchanged in a manner we might term 'egalitarian' because all ramets are guaranteed to have their resource needs met, regardless of their actual contribution to the common pool. This sort of interaction transfers nutrients from source to sink, which often results in extra assistance going to ramets or parts that are struggling. In these plants, fitness differences between ramets will be suppressed to some extent, regardless of genetic variance between them. Other plants exchange resources in a different way, which we might view as capitalistic. Here ramets that are 'successful' in that they have a fast

growth rate, caused either by environmental advantage or by an idiosyncratic genotype, can exert apical dominance over the other ramets in the network, which guarantees them a disproportionate share of the resources. Here resources are actually drawn away from weaker or less successful ramets, so that fitness differences are exaggerated. In goldenrods (*Solidago altissima*), for example, sister ramets compete for nutrient donations from their parent, and large ramets do better than small ones (Abrahamson et al. 1991). Hadany describes how such systems can give rise to conflict between different parts of a tree, resulting in overutilization of resources by branches, or overgrowth causing the trunk to collapse (Hadany 2001).[20]

The style of resource sharing, like the amount of investment in maintaining connections, seems to be a species and even environment-specific adaptation to particular circumstances (Pitelka and Ashmun 1985). Abrahamson et al. argue that capitalistic sharing is a gene level adaptation to poor environments, which makes the genet more efficient in utilizing resources (Abrahamson et al. 1991). Others claim that resource patchiness is what selects for capitalistic sharing, by allowing the genet to avoid wasting resources on ramets who occupy localized poor patches (Hellström et al. 2006).

The type of interaction amongst modules within a ramet also varies, with implications for the individuality of ramets. Allocation of resources amongst modules is often capitalistic, with more resources going to modules that produce a larger amount of some hormone (often auxin). In some plants such as conifers a single growing point will suppress the growth of all the others in a phenomenon called apical dominance (Jerling 1985). In these plants there can be large fitness differences between parts of a single tree. "The effect of (a) somatic mutation will be determined in a "double or nothing'' manner." (Hadany 2001) If it gives its branch a significant enough advantage it can gain apical dominance and become the main growing point. On the other hand, if the advantage is not large enough then the mutation stands an almost zero chance of being inherited.

It is worth noting that the boundaries, and immune response, views on individuality gain significant vindication here, in so far as physical contiguity and immunity are important in mediating fitness-affecting interactions.

Plant individuation

There are almost certainly other factors that have been neglected here, but the factors determining the individuality of plants fall into two, interrelated, categories: sources of variance; and interactivity. Interactivity is irrelevant if there are absolutely no heritable differences between two objects—they cannot be independently selected. But likewise, genetic variance can be irrelevant if ecological factors prevent it from giving rise to fitness differences, so two genetically diverse objects can nonetheless form part of a single individual. Fagerström and Tuomi and

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[20] He also describes how conflicts can be expected to have certain paradoxical consequences. For example, "harmful somatic mutations concerning the roots are expected to accumulate in the branches (and therefore the seeds) as the tree grows older". The roots themselves would eliminate these mutants by somatic selection, so that "the roots of vegetative offspring would be more resistant to root parasites, for example, than the roots of a young tree germinating from seed." (Hadany 2000, 519).

Vuorisalo's insights can be seen as necessary correctives to views that make the mistake of focusing only on genetic factors. It is wrong to assume that clones are true genetic copies of one another, and it is also wrong to forget that genetic variance will not be selected if it does not cause variance in fitness.

This table offers a very rough summary of the sorts of mechanisms which are significant to plant individuality;

Mechanisms which constrain sources of heritable variance	Mechanisms which constrain fitness differences via interactivity
Non-genetic: epigenetics? niche construction?	Investment in root connections, shared stems, egalitarian vs capitalistic resource interactions, auxin dominance, synchronization of flowering, inter-genet root grafting, immune response
Genetic: sex, self-incompatibility vs. apomixis, bottlenecks, polyploidy, meristem structure, somatic selection, DNA repair systems, meiotic driver systems, cell walls, bark and other boundaries.	

In the end then, after careful consideration of these factors, we will decide that some plants have the sort of properties that give us reason to say that modules are individuals, while other plants will have different properties and would be best viewed as having individuality at a higher level. Many more are going to have a significant degree of individuality at multiple levels.

What does this survey of plant individuation mechanisms have to teach us about individuality in general? Perhaps the most important lesson we can learn about individuality in general is not to assume that criteria of individuality are transferable across different examples. It might be that none of the features I have so far singled out are going to be relevant in determining individuality in bacteria, for example, or social insect societies. On the other hand, many of the conclusions found here are consequences of the modularity of plant growth, and will therefore generalise to modular organisms in other kingdoms.

Another general lesson we should extract is about what forms individuality can take. Looking at plants makes it obvious that individuality is a matter of degree. In fact this is true for unitary organisms also but plants make it even clearer that there won't always be a single unique level of selection. Similarly, the structure of modular organisms makes it very apparent that biological individuals can be hierarchical.

We can identify biological individuals, in plants as elsewhere, on the basis of mechanisms they have evolved for the purpose of constraining relative levels of selection. These mechanisms are species-specific, and they fix the degree to which units at any particular hierarchical level are individuals by fixing their capacity to vary from one another in ways that heritably affect their fitness.

Solving the demographer's dilemma

Earlier I said that anyone who endorses genets as exclusive levels of selection overlooks Fagerström's insights, but anyone who endorses ramets or modules as

exclusive levels, on the other hand, ignores Tuomi and Vuorisalo's insights. Now I want to show that if we ignore either Fagerström's insights (that lower level units might be undergoing considerable selection) or Tuomi and Vuorisalo's insights (that higher level units might show the kind of integration which allows them to act as significant levels of selection despite lower level genetic variance) then we can end up making serious mistakes. The lesson I will ultimately draw is that in so far as either higher or lower level views are monistic—countenance only one exclusive individual or level of selection—they are mistaken. Plant demographers will sometimes be forced to recognise multiple focal units as potential individuals, and set to work on establishing the degree to which different levels are significant.

Two distinct kinds of mistake can occur as a result of assuming a single level of selection when there are actually two. One the one hand, if there is a level *below* the focal level that you are failing to acknowledge, then predictive errors can occur. On the other hand, if you are failing to take account of a level *above* the focal level of selection, then an explanatory problem can occur.

The genet horn

Suppose we want to work out how natural selection is operating on a particular species of plant in a particular environment. Suppose further that we are assuming a monistic genet view, so we assume that selection occurs exclusively at the level of the genet. We carry out an experiment in which we use the demographic method to track the covariance between traits and fitness at the genet level. Bear in mind that we should read 'genet' to mean 'unit that has developed from a single zygote here'. So as stated in section two, we are likely to use the presence of connecting vegetative runners to decide which ramets are parts of which genets, and assume that new seedlings have a novel genotype. Then we would record the growth rate of each genet, or measure its total seed output. But we wouldn't take any measurements that would enable us to pick up on differential growth rates or seed outputs within the genets. This is because a genet-only view implicitly assumes that there is no heritable variation within a genet, because it assumes erroneously that vegetatively propagated parts are genetically identical to their 'parent'.

Price's equation allows us to see very clearly the mistake that we would be making (Price 1970). The equation is based on the idea that selection is driven by variation in organismal traits causing variation in organismal fitness. Price saw that this causal connection will show up as covariance between organisms' traits and their fitness. Over several generations it can result in a change in the relative frequencies of different traits. Price proved that the amount of evolution—change in the population average value of a character (such as stamen length)—from one generation to the next that is due to selection is equal to the statistical covariance between individuals' character values and their relative reproductive success (their fitness compared to the population average). Taking z to be an entity's character value, ω its relative fitness, and $E_W(\Delta z)$ to be the fitness-weighted average of the quantity Δz, we express this like so;

$$\Delta \bar{z} = Cov(\omega, z) + E_w(\Delta z) \tag{1}$$

From this we multiply both sides by $\bar{w}$ to derive an equation which is concerned with absolute fitness, w;

$$\bar{w}\Delta \bar{z} = Cov(w, z) + E(w\Delta z) \tag{2}$$

Equation 2 describes how the average properties of a population of entities change over time to the extent that the traits of those entities covary heritably with their fitness.[21] This covariance between traits and fitness is determined by the properties of the collection of objects—specifically, their heritable variance in fitness—as demonstrated by the fact that with some minimal assumptions[22] we can derive Lewontin's conditions for evolution by natural selection from the Price equation (Okasha 2006).

The right hand side of the equation contains two terms—the first gives the covariance between the traits and fitness of genets, while the second term gives the transmission bias—the phenotypic change that comes about during reproduction of genets (Frank 1997). We would have to use an estimated figure for the second term, based on artificial experiments done on similar plants to measure heritability. We could then generate a prediction about how the traits will change in the future.

The problem is that if there are lower levels of selection in effect, such as somatic selection or between-ramet selection, then this will create a further amount of transmission bias. As Price noted, selection at a level below the focal level always shows up in the Price analysis as transmission bias at the focal level (Okasha 2006) because lower level selection acts to undermine heritability at the higher level. But in the imaginary experiment above we haven't taken this into account. We are likely to underestimate, therefore, the value of the transmission bias, which will cause us to overestimate the total amount of evolutionary change that is going to take place. This assumes that the two levels of selection are antagonistic, i.e. the two left hand terms in the multilevel Price equation are opposite in sign. If they have the same sign then neglecting the lower level selection most likely leads us to *underestimate* the overall change.

In cases where there is a lower level of selection in effect, such as cases where there is a considerable amount of either somatic selection or between-ramet selection, then calculations based only on the covariance of traits and fitness at the genet level are likely to generate false predictions about how gene frequencies will change over time. Ignoring lower levels of selection can lead one to overestimate the effect of selection at higher levels. This situation is essentially analogous to the case of genic selection caused by selection between the genes within a single organism, as in cases of segregation distortion, or intra-genomic conflict more generally (Okasha 2006). What is important is that neglecting a lower level of selection actually falsifies the ecologist's empirical hypothesis.

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[21] I am using Okasha's derivations of these equations (Okasha 2006), p. 22.

[22] The assumption is that the covariance relation is locally transitive, so that covariance between an organism's traits and its fitness, as well as covariance between an organism's traits and its offspring's traits, guarantees covariance between an organisms' fitness and its offspring's traits.

The ramet horn

Now suppose instead that we take the contrary view and assume that selection takes place exclusively at the ramet level. We would now measure the covariance between fitness and traits of ramets, where ramet fitness is equivalent to their combined sexual and vegetative reproduction. Here we would also use the single level Price equation to make predictions about how selection will affect our chosen species. We will assess the covariance over the whole (global) population of ramets, ignoring their assortment into genets and/or connected groups. In order to keep things simple, let us assume that we don't make any mistakes with respect to even lower levels of selection. We get the right figure for transmission bias, somehow. Now we are in a better situation than before, because we are going to get our sums right. Assuming we apply Eq. 2 correctly, we can make correct predictions about how the frequencies of different traits are going to vary over time. However, if there is a significant degree of higher level selection, then we may not be perfectly content with this accurate prediction.

The problem is that if population structure is having a big effect on individual fitness, then we will come up with a correlation between traits and fitness that seems counterintuitive, because a phenotypic trait, which *seems like* it should impair an organism's ability to survive and reproduce, has high fitness. For example, suppose we are looking at a population of ramets of which some show a mutation C which increases metabolic rate, resulting in a faster conversion of resources into new growth. Based on knowledge of the effects of mutation C alone, we might guess that ramets carrying C will increase in frequency relative to the wildtype A. In fact, we observe that C-type ramets are often bigger and more fecund than their neighbours. However, after measuring the covariance of fitness with traits C and A, we would be surprised to calculate that trait A will increase in frequency in the population, because ramets carrying A actually have a higher average fitness than do ramets carrying C. Now we might be perplexed—how can having a higher metabolic rate end up decreasing a ramet's fitness on average?

This case is supposed to be exactly analogous to a model for the evolution of altruism, and also to Simpson's paradox (Simpson 1951).[23] The 'paradox' can be resolved by taking population structure into account in order to acknowledge the effects of higher level selection. If the ramets that we observe are compartmented into mixed groups, then it is possible for a mutant to actually decrease in frequency over time, even though it always outcompetes the wildtype members of its group, so long as groups with a high frequency of wildtypes do better on average than groups with a high frequency of mutants. These are precisely the sorts of conditions under which altruistic traits can evolve.

We fail to see the result as so surprising if we take into account the fact that the ramets are assorted into groups which maintain physiological connections between their roots to exchange resources. The C-type ramets are cheaters, because their fast metabolic rate is achieved by cutting out the work of producing resources, and

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[23] Or the 'Berkeley Admissions Paradox'. The philosophical mistake given rise to has been termed 'The Averaging Fallacy' (Sober and Wilson 1999).

instead taking them from the common stock shared with the other ramets to which they are connected below ground. A-types are altruists, who donate resources to the cheaters. C-types take advantage of A-types by taking resources without generating any of their own, which allows them to grow and reproduce much faster than any of the A-type ramets to which they are connected. *However,* because C-types don't produce vital resources, in groups which have a high frequency of cheaters, the fitness of the whole group is depressed, because neither A nor C types have sufficient resources to carry out their essential functions. Now it is not surprising that C types might decrease in frequency over time, exactly because their perceived 'success' is frequency dependent. As soon as the group –level effects are taken into consideration it seems perfectly plausible that altruists will do better than cheaters, because altruistic structural groups have a higher fitness than cheater groups.

Once again, the Price equation makes this mistake perfectly clear. Our mistake was to use the single level Price equation;

$$\bar{w}\Delta\bar{z} = Cov(w, z) + E(w\Delta z) \tag{2}$$

In the thought experiment we observed a positive covariance between ramet fitness and character values, where the variance was measured over ramets in the global population, but it was hard to interpret this correlation as causal, because the C trait doesn't seem like the sort of trait that should decrease a ramet's fitness. We worried, therefore, that the observed covariance is some sort of statistical artefact (Okasha 2006). And in fact given the backstory about C-types stealing all the resources without putting any back, we think that being of a C type doesn't on its own *cause* a ramet to have low fitness. The real cause is located at a higher level—it is being in a group that has a high proportion of C-types that causes a ramet to have low fitness. What we needed to do to make this explicit is partition the covariance in the equation into it's between-and within-group components;

$$Cov(w, z) = E(Cov(w, z)) + Cov(W, Z) \tag{3}$$

This is the multilevel form of Price's Equation, where the first term on the right hand side expresses the evolution that is due to selection within groups, and the second term expresses the evolution that is due to selection between groups (Okasha 2006). This allows us to give a more causally accurate interpretation of the evolution of C and A types. The multilevel Price equation would tell us that selection within groups favours C-types, but antagonistic selection between groups favours A-types. So long as the second component outweighs the first, C-types will decrease.

Even where the inequality between the two terms is not so great, so that selection at the higher level is just partially counteracted by lower level selection, or where the two components act concordantly, rather than antagonistically, it is still more philosophically accurate to say that the value generated by summing the two components is *caused* by the combined effects of two selective levels, rather than to simply interpret the correlation revealed at the higher level as causal.

Notice that this second mistake has a totally different flavour from the first, even though both are a consequence of neglecting a selective level. In the first instance, we made false predictions about how the frequency of traits in the population of

ramets would change. The second error, on the other hand, was philosophical rather than empirical—we got the predictions right, but couldn't come up with an appropriate causal story to explain why traits would change as we predicted.

Another complication is that the two errors are not symmetrical. The genet-only view is misled when selection acts at the ramet-level. However the ramet-only view is misled when selection acts at the *group* level, where the interaction group may or may not overlap with the genet (either developmentally or genetically defined). *Interaction effects* cause problems for the ramet-level view, but *genetic effects* cause problems for the genet-level view.

In this section I have shown how neglecting levels of selection can lead to different sorts of errors in our understanding of selective processes. If we neglect lower levels of selection we make book keeping errors, whilst if we neglect higher levels of selection, we omit significant causes of and are thus unable to explain gene frequency change. We see that extreme versions of all the genet, ramet and module views are wrong, in so far as they treat their focal unit as an exclusive level of selection, and fail to countenance the possibility and significance of selection at other levels. In both cases, the mistake was to assume that only a single level of selection was in action. The lesson is that plant ecologists must be careful not to assume that *any* level, whether the genet, ramet, module or structural unit, always acts as an *exclusive* level of selection.

Conclusions

I suggested that it is reasonable to explain the success of popular individuation criteria within vertebrate lineages as lying in the fact that they identify mechanisms—bottlenecks, germ soma separation and the rest—whose effect is to constrain the extent to which populations at different compositional scales exhibit heritable variance in fitness. Given this, we can identify plant-specific criteria of individuality by searching for mechanisms which have the same effect.

I argued that there are two classes of factors that together fix individuality in plants. The availability of heritable variance is determined by mechanisms including niche construction and other epigenetic factors, bottlenecks, DNA repair mechanisms, meristem structure, somatic selection, sex, polyploidy, boundaries and the dominant mode of reproduction (i.e the incidence of sexual versus asexual propagation). The extent to which variation causes differences in fitness is determined by mechanisms which mediate interactions amongst parts, including physical contiguity, investment in root connections, modes of resource allocation, immune responses, auxin dominance, synchronization of flowering, and determinacy of growth habit.

We see in the end that both horns of the demographer's dilemma are to be avoided. Neglecting levels of individuality can have negative consequences for our attempts to understand the way selection acts. Where there is reason to suspect that somatic selection might be causing a substantial amount of within-genet change in gene frequencies, a genet-only view (such as the demographic method) is liable to misrepresent the evolutionary process. On the other hand, where there is reason to

suspect that ramets might take part in fitness-affecting interactions within population-structured groups, then a ramet-only view is liable to misrepresent the causes of evolutionary change. The solution to the dilemma lies in flexibility: wherever ecologists see the hallmarks of individuality they should include an analysis of the extent to which traits covary with fitness at *that* level within their overall model of selection. As Pedersen and Tuomi say, "there is no simple or general solution to the problem of identifying units to which fitness should be attributed" (Pedersen and Tuomi 1995). No single level can provide any exhaustive description of selective processes in modular organisms. On a case by case basis, one must separately find the most appropriate way of measuring fitness as well as the level of the hierarchy of demographic units at which one should analyze specific aspects of adaptation in these organisms. The optimal method of modelling evolutionary dynamics in modular and clonal plants is going to involve some kind of multilevel selection analysis (Pedersen and Tuomi 1995; Stevens et al. 1995; Hadany 2001; Aspi et al. 2003; Weinig et al. 2007; Monro and Poore 2009; Folse III and Roughgarden 2011).

The degree to which different hierarchical levels are individuated in plants will be variable across and within species (perhaps even within the lifetime of a single clone), and different factors will be significant in different cases. In some species there will be a single unique level of individuation, but in many more individuality will be hierarchical, so that an intermediate level of individuality will coexist at multiple levels simultaneously. Yet despite failing the criteria which unitary organisms meet, plants are biological individuals too.

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