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Evidence from Polygene Mapping for a Causal Relationship between Potato Tuber Dormancy and Abscisic Acid Content[1]

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In previous studies polygene mapping of a backcross population derived from haploid potato (*Solanum tuberosum*) and a diploid wild species (*Solanum berthaultii*) showed at least eight quantitative trait loci (QTLs) associated with tuber dormancy. The same population was mapped for abscisic acid (ABA) content in tubers so that any QTLs identified could be compared with those detected previously. At least three distinct loci on three chromosomes (2, 4, and 7) were associated with variation in ABA content. One of the QTLs was detected only as a main (single locus) effect, and two QTLs were found through two-locus interaction analysis (epistasis). Interaction between QTLs at markers *TG234* (chromosome 2) and *TG155* (chromosome 4) explained 20% of total phenotypic variance for this trait. The interaction closely resembled one previously detected for dormancy, suggesting an association between high ABA content and long tuber dormancy. Although relationships between ABA level and dormancy could be demonstrated through polygene mapping, there was no indication of a relationship between these traits when they were subjected to a conventional correlation test. This illustrates the usefulness of polygene mapping as a tool to identify possible associations between hormone levels and plant development.

A well-tested approach for understanding growth and development is the analysis of differences between two phenotypes that contain different alleles of a single major gene. For example, analyses of dwarf and wild-type siblings have revealed hormonal differences associated with the mutant genotype (Reid and Howell, 1995). Although this approach has been productive, a limitation is that the control of many developmental processes is polygenic, i.e. inheritance is quantitative rather than qualitative. Thus, although it is interesting to know what physiological effect was produced by a dwarf mutation, it might be much more informative to learn about the physiological effects produced by the polygenes that control plant height. In the past the study of quantitative inheritance was so difficult that it yielded only very generalized information, and little could be learned that would elucidate the physiology of growth and development. The situation has changed dramatically with the availability of new methods for mapping polygenes (Tanksley et al., 1989; Tanksley, 1993; Young, 1993). The new mapping methods rely on DNA-based genetic markers, such as RFLP markers. Through such mapping, a number of QTLs may be found that are associated with the control of a trait such as plant height. A QTL denotes a region of chromosome linked to the marker gene that has a significant effect on the quantitative trait (Tanksley, 1993).

With the availability of this tool for mapping quantitative traits, polygenes can be used to study the physiology of growth and development in a manner analogous to, but more powerful than, the way single genes have been used in the past. Once the QTLs have been detected that are associated with the control of a given stage of plant development, it should be possible to learn about the physiology of the process by searching for QTLs that are associated with the levels of hormones or other biochemical components in the same population. Coincidence of a QTL for plant height with a QTL for a particular GA, for example, would suggest that the GA played a role in controlling plant height. The QTL analysis may also indicate how many loci controlled the GA content, how many of these were associated with the control of plant height, and how many other important factors in addition to the GA were operating for control of plant height.

We are using this approach to study the hormonal control of potato (*Solanum tuberosum* L.) tuber dormancy and the ability to tuberize under long photoperiods. We have examined two populations obtained by backcrossing to the respective parent species a hybrid of haploid potato by a wild species (*Solanum berthaultii*). RFLP mapping of these populations has been carried out (Bonierbale et al., 1994). We have used the mapping information to identify 11 QTLs associated with the ability to tuberize under long days (Van den Berg et al., 1996a) and 9 QTLs for tuber dormancy (Van den Berg et al., 1996b). Three of these QTLs coincide, raising the possibility that each of the 3 is the locus of a gene that controls a hormone or other substance affecting

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Abbreviations: cM, centiMorgan; QTL, quantitative trait locus; RFLP, restriction fragment-length polymorphism.

both tuberization and dormancy. Therefore, we have started to assay dormant tubers from each genotype in one population for hormones that may control dormancy. Assays for ABA have been completed, and the results are presented here to illustrate the usefulness of polygene mapping for understanding growth and development.

MATERIALS AND METHODS

Plant Population

The study was performed on a population obtained by backcrossing a haploid *Solanum tuberosum* (HH1–9) to a hybrid of haploid *S. tuberosum* (USW2230) × diploid *Solanum berthaultii* (PI 473331), where the hybrid clone was the female. The population had the mean genome ratio of 75% *S. tuberosum*, with a range from 50 to 87%. RFLP mapping was carried out on this population by Bonierbale et al. (1994), and it is the same population of 155 progenies that was used to find QTLs for tuberization and dormancy (Van den Berg et al., 1996a, 1996b).

Because of the heterozygous nature of the parental clones used in these experiments, segregating alleles from both parents contributed to the genetic variation of progenies. Thus, two maps were constructed by Bonierbale et al. (1994): one based on segregation from the hybrid parent (*S. berthaultii* alleles, B) and the other based on segregation from the recurrent parent (*S. tuberosum* alleles, T^R). The framework map, based on segregation from the hybrid parent, consists of 81 loci at average intervals of 10 cM. The map based on recombination from the recurrent parent, consists of 35 markers (Bonierbale et al. (1994). These 35 markers are not as uniformly distributed as are the 81 markers, and chromosomes 1, 7, and 12 have only 1 marker each.

Plants were transplanted into soil from in vitro plantlets on May 27, 1994, and grown in the greenhouse until harvest 15 weeks later. Photoperiod was shortened to 8 h by covering plants with black cloth from July 29 to August 12 to induce tuberization in all clones, and tuber formation occurred from August 14 to 22. Tubers were stored in the dark at 3 ± 1°C. Approximately 120 d after tuberization of a given genotype, tubers were frozen in liquid nitrogen and stored at −25°C prior to analysis. The storage period was timed to end shortly before the end of the dormant period for the clones previously identified as having the shortest dormancy (Van den Berg et al., 1996b).

ABA Analysis

Tubers were available from 144 genotypes for ABA analysis. One tuber, ranging from 4 to 12 g fresh weight, was chosen from each clone. Tissue was homogenized in ice-cold methanol:water (90:10 [v/v] using 5 mL g^{-1}), and the homogenate was left overnight at 4°C prior to vacuum filtration through a Büchner funnel that contained a celite bed. The filtrate was dried in vacuo at 40°C, resuspended in 50 μL of methanol, and 5 mL of water:acetic acid (99.8:0.2 [v/v]) was added. The sample was loaded onto an ODS-5 C_{18} cartridge column (Whatman), washed with 3 mL of water:acetic acid (99.8:0.2 [v/v]), and then eluted with 3 mL of methanol. The content of ABA in the sample was analyzed by ELISA (Ober et al., 1991). Triplicate ELISAs were performed on each sample, with very close agreement between repetitions.

Before the ELISA was performed on tuber samples from the backcross population, a preliminary test was carried out on 8 tubers from one genotype to check for the effect of tuber size on ABA content. There was no correlation ($P = 0.50$) between tuber size in the range between 4 and 12 g and ABA content (nanomoles per gram fresh weight) of tubers. After the ELISA was performed we checked for regression between tuber size and ABA content for each of the 144 tubers. Again, there was no evidence that size differences of tubers affected the content of ABA ($R^2 = 0.013$; $P = 0.17$).

As a further precaution we checked for false-positive QTLs that might be related to the tuber size taken for ABA analysis. It might be argued that spurious QTLs for ABA could be claimed when the QTLs were actually associated with the size of tuber taken for assay. If smaller tubers differed from larger tubers in ABA content, we then might falsely conclude that a QTL was associated with ABA content when it was actually associated with the size of tuber chosen for assay. To guard against this, we performed a QTL analysis for tuber size used in the assay. Because no QTL was detected even at $P = 0.10$, we concluded that the individual sizes of tubers used for ELISA had a random distribution in the population and were unrelated to QTLs detected for ABA content.

Statistical Analyses

Because the frequency distribution of ABA level was skewed toward low-ABA content, data were subjected to logarithmic and to arcsine transformations to obtain normal distributions. The same QTLs were found with either type of transformed data as with the original data; therefore, the original data are presented here. Statistical analyses of the linkage between RFLPs and ABA level segregating from the hybrid parent were performed by qGene software (Nelson, 1997) and by the MAPMAKER/QTL program (Lincoln and Lander, 1989). The two programs gave the same results. A threshold of $P = 0.01$ was set for declaring an interval significant in a QTL model.

Because the number and distribution of markers segregating from the recurrent *S. tuberosum* parent did not cover the genome homogeneously, an analysis of linkage between ABA level and RFLP segregation was performed in qGene by single-point analysis. A P value of 0.01 was chosen for the threshold significance level. Epistatic effects among the marker loci linked to QTLs that had P values ≤ 0.10 from interval or single-point analysis were also tested by qGene. This led to testing all combinations of 19 markers for epistasis, or 171 tests. However, 6 of the 19 markers were less than 10 cM from the others and may have represented the same QTLs. On this basis 13 QTLs were included in the tests for epistasis, which means that only 78 of the 171 combinations involved markers that were not closely linked. A threshold value of $P \leq 0.01$ was consid-

ered significant for interaction. The population analyzed was not large enough to test for interactions involving more than two loci (Tanksley, 1993).

The QTLs detected for ABA content were compared with QTLs previously detected for tuber dormancy (Van den Berg et al., 1996b) and for the ability to tuberize under long photoperiods (Van den Berg et al., 1996a). We determined the mean lengths (in days) of tuber dormancy for 2 successive years. Tuberization data were from the same population (Van den Berg et al., 1996a) and consisted of ratings for tuberization on one-node cuttings the 1st and 2nd years and percentage of plants that tuberized early.

RESULTS

Population Relationships

The ABA content in the population analyzed ranged from 0.18 to 4.35 nmol g^{-1} fresh weight, with a mean value of 1.17 nmol g^{-1} fresh weight. The frequency distribution of ABA content among individuals was skewed toward low ABA content (Fig. 1). No significant correlation (R^2 = 0.013) was detected between ABA content and the length of dormancy in year 1 (Fig. 2) or in year 2 (R^2 = 0.008; not shown). Similarly, there was no correlation between ABA content and any of the tuberization traits (R^2 < 0.01 for each of three traits; not shown).

QTLs for ABA

Four QTLs on three chromosomes were significantly associated with ABA content in tubers (Fig. 3). One of the four, located on chromosome 4, was detected while following T^R alleles segregating from the *S. tuberosum* parent. The other three QTLs for ABA were found by following alleles segregating from the hybrid parent. These were on chromosomes 2, 4, and 7. The original parents were no longer available for ABA analysis, but both *S. tuberosum* and *S. berthaultii* were sources of alleles for high ABA content.

Of the QTLs detected by following segregation from the hybrid parent, two were found as main (direct) effects (Table I). The presence of a B allele at marker *TG123* was associated with an increased ABA content of 0.31 nmol g^{-1} fresh weight. The presence of a B allele at *TG499* was

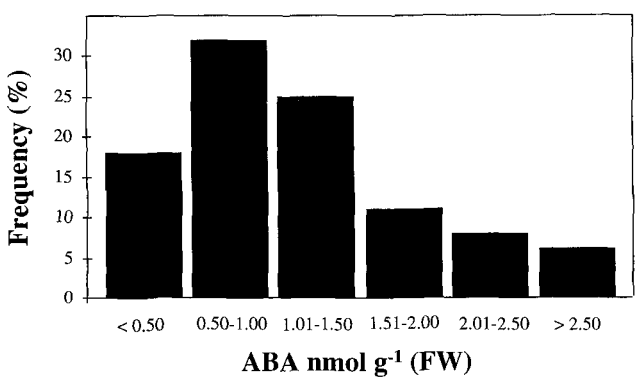

Figure 1. Frequency distribution of ABA content among individuals in the backcross population. FW, Fresh weight.

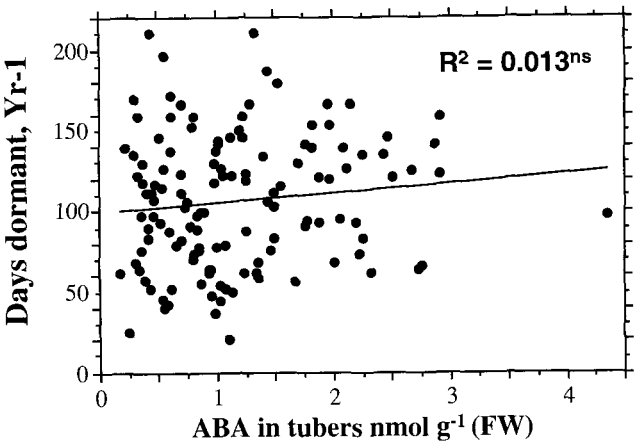

Figure 2. Test for correlation between ABA content and the length of the dormant period in year 1. Data show no significant correlation (P = 0.21). FW, Fresh weight.

associated with the opposite effect, a decrease in the ABA content of 0.33 nmol g^{-1} fresh weight. Each of these QTLs explained only about 5% of the total variance in ABA level. The QTL detected by following segregation from the recurrent parent was linked to *TG155* and *TG65* (Fig. 3) and was found as significant both through main effects (Table II) and through epistasis with a QTL linked to *TG234* and *TG276* (Fig. 4). The main-effect model, using the QTLs linked to *TG123*, *TG499*, and *TG155*, explained 18% of the total phenotypic variance in ABA content.

The epistasis between the QTL linked to *TG155* and the QTL linked to *TG234* accounted for 20% of the phenotypic variance. The interaction was most significant (P = 0.001) at *TG234* × *TG155* but could also be detected at *TG234* × *TG65* (P = 0.034), *TG276* × *TG155* (P = 0.009), and *TG276* × *TG65* (P = 0.054). This is to be expected, since on the map for segregation from the recurrent parent *TG65* is 4.7 cM from *TG155*, and on the map for segregation from the hybrid parent *TG234* is 8.0 cM from *TG276* (Fig. 3). The multiple-analysis model, which includes the main-effect QTLs together with epistasis, explained 25.4% of the total phenotypic variance (P < 0.0001).

Coincidence with QTLs for Other Traits

Neither the QTL near *TG123* nor the one near *TG499* coincided with QTLs previously found for dormancy or tuberization in this population (Van den Berg et al., 1996a, 1996b). However, the QTL linked to *TG155* and *TG65* coincided with one detected for tuberization on two-node cuttings (Van den Berg et al., 1996a), and through epistasis we detected one at that location for tuberization on one-node cuttings (Van den Berg et al., 1996a) and for dormancy (see below). The epistasis found for ABA level matched epistasis found earlier for length of tuber dormancy in the same backcross population (Van den Berg et al., 1996b). Longer dormancy was associated with a B allele at *TG276* solely in the concurrent presence at *TG65* of a T^R allele segregating from the recurrent *S. tuberosum* parent. The epistasis was significant only at P = 0.081, but the

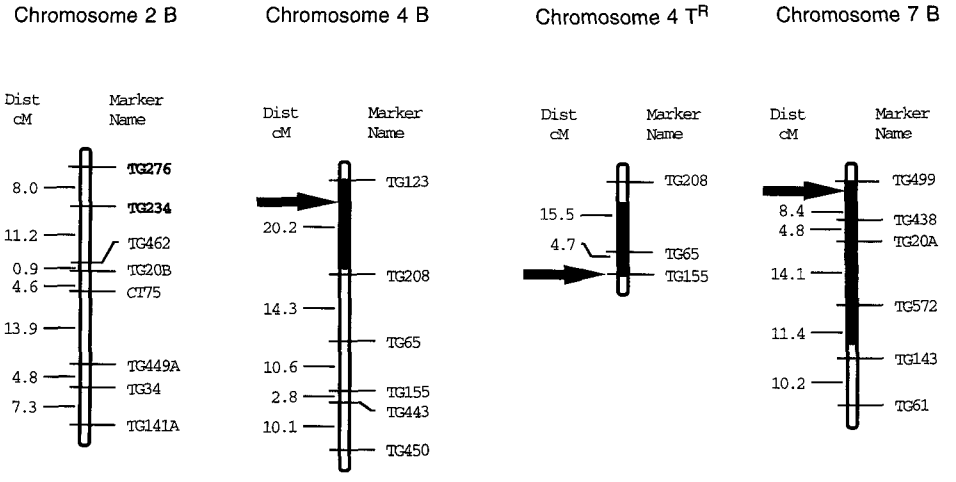

Figure 3. Location of QTLs associated with ABA content in potato tubers. Maps are based on the segregation of B alleles from the hybrid parent for all three chromosomes shown, but for chromosome 4 a QTL was detected through segregation of T^R alleles from the recurrent *S. tuberosum* parent. Arrows denote the estimated positions of QTLs detected by main-effect analysis. For main-effect QTLs, the black area on the chromosome shows the width of the peak before the likelihood decreases 10-fold. On chromosome 2, a QTL in the vicinity of *TG234* and *TG276* was detected through epistasis with a QTL in the vicinity of *TG155* and *TG65*. The letter after the chromosome number indicates whether the map is based on segregation from the hybrid parent (*S. berthaultii* alleles, B) or from the recurrent parent (*S. tuberosum* alleles, T^R).

same interaction was present when data from a 2nd year of dormancy testing were analyzed (P = 0.075). Even though the P values were high, the interaction was obtained both years and the shape of the response was the same, i.e. long dormancy was observed only with the concurrent presence of a B allele at *TG276*, together with an allele from the recurrent parent at *TG65*. Thus, the interaction did not appear to be a chance effect. Results were similar for the interaction between the linked QTLs, *TG234* × *TG155* (P = 0.069 for the 1st year of dormancy testing and P = 0.120 for the 2nd year). Because *TG65* and *TG155* are linked in repulsion, it is the absence of a T^R allele at *TG155*, in combination with the presence of a B allele at *TG234*, that is associated with long dormancy (Fig. 4).

Presence of More than One QTL per Chromosome

Two of the apparent QTLs we found for ABA were located on chromosome 4. One QTL, linked to *TG123*, was found through segregation from the hybrid parent, and the other, linked to *TG65*, was found through segregation from the recurrent parent. Because the 35 markers used for the map based on recurrent parent segregation do not allow precise localization of QTLs, we cannot be certain that these two QTLs are distinct. By this more conservative reckoning, our studies reveal at least three distinct QTLs for ABA content in potato tubers.

DISCUSSION

Evidence for a Causal Relationship

The coincidence of epistatic relationships between dormancy and ABA levels in tubers raises the question of causality. Evidence for a causal relationship between traits such as ABA content and potato dormancy has usually been based on correlative evidence from observations or treatments within a single genotype (Ji and Wang, 1988; Cvikrová et al., 1994; Suttle and Hultstrand, 1994). As noted by Lebreton et al. (1995), identifying the location of the genes that regulate the expression of two or more traits may provide a more precise test of whether the traits are causally related than is possible by studies of whether the traits merely vary in association. If trait X has a causal relationship to trait Y, then the two traits should have at least one QTL in common. These authors point out, how-

Table I. *Phenotypic effects of the QTLs detected (P ≤ 0.01) for tuber ABA content through tests for direct (main) effects, segregation of B alleles from the hybrid parent (BT)*

Chromosome	Locus	P Value	Variance Explained	Presence[a] of B	ABA Content (mean ± SE)
			%		*nmol g^{-1} fresh wt*
4	*TG123*	0.0081	4.8	+	1.35 ± 0.09
				0	1.04 ± 0.07
7	*TG499*	0.0072	5.1	+	1.02 ± 0.07
				0	1.35 ± 0.11

[a] Presence or absence of the B allele is indicated by + or 0, respectively.

Table II. *Phenotypic effects of the QTL detected ($P \leq 0.01$) for tuber ABA content through tests for direct (main) effects, segregation of T^R alleles from the recurrent S. tuberosum parent (TT^R)*

Chromosome	Locus	P Value	Variance Explained	Presence[a] of T^R	ABA Content (mean ± SE)
			%		*nmol g^{-1} fresh wt*
4	*TG65*	0.0001	10.9	+	1.46 ± 0.11
				0	0.97 ± 0.06

[a] Presence or absence of the T^R allele is indicated by + or 0, respectively.

ever, two other scenarios apart from a causal relationship that could account for common QTLs between traits: (a) the traits could be controlled by separate genes, linked too closely to be separated by the test procedures used, or (b) the traits could be controlled by the same gene but independent of one another.

These alternative scenarios must certainly be considered for QTLs detected through main effects for two traits in common. However, both scenarios are improbable in the case of matching epistatic effects, which we report here for dormancy and ABA levels (Fig. 4). If the commonality of QTLs is to be explained by linked genes, then not only would such linkage have been required at both loci involved in the epistasis but the interactions of the two pairs of linked genes would have had to be identical, with long dormancy accompanying high ABA. The other scenario is equally unlikely, namely that ABA and dormancy are independent of one another, even though (a) both traits are controlled by the same two genes on two different chromosomes, (b) the interactions of these genes are the same for dormancy as for ABA, and (c) high ABA level is associated with long dormancy, as might be expected based on physiology.

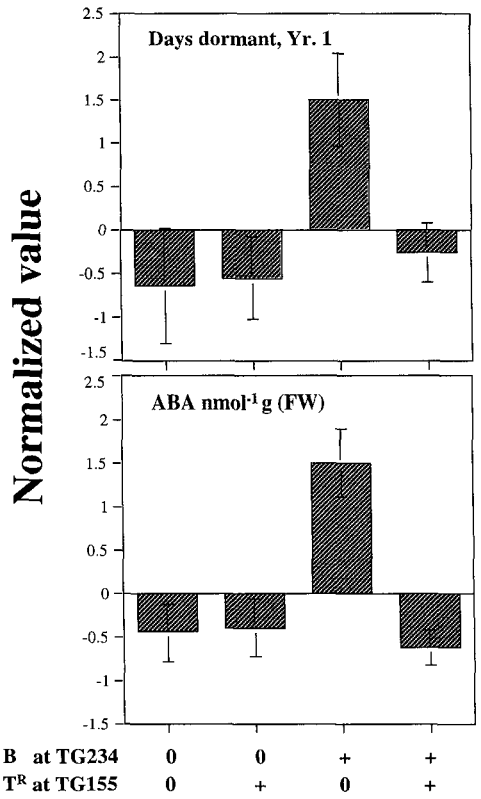

Figure 4. Epistasis of QTLs linked to *TG234* and *TG155* for both ABA and dormancy. Presence or absence of indicated alleles is denoted by + or 0, respectively. Data have been normalized by the formula: $Z = (X - \mu)/\sigma$, where μ is the mean of the four values plotted and σ is their SD. For ABA, μ = 1.17 nmol g^{-1} fresh weight and σ = 0.31 nmol g^{-1} fresh weight. For dormancy in year 1, μ = 106 d and σ = 15 d. Bars denote SE. FW, Fresh weight.

The probability is remote that this could happen by chance alone. With 81 markers for segregation from the hybrid parent and 35 markers for segregation from the recurrent parent, there were 2835 possible cases of epistasis between QTLs from these two sources. Because of linkage some of the possible cases were not independent. If we arbitrarily count markers that are <10 cM apart as a single linkage group, then there were 45 groups of such markers for segregation from the hybrid parent and 29 groups for segregation from the recurrent parent. This gives 45 × 29 = 1305 combinations of groups of markers spaced at least 10 cM apart that could be involved in the epistasis. Only 40 of the possible combinations of QTLs for ABA level were tested for epistasis, but the decisions as to which ones were tested and which ones were declared significant were made by objective criteria. Tests were made only among QTLs, the main effects of which had $P \leq 0.1$. Similar criteria were applied to epistasis tests for dormancy (Van den Berg, 1996b). Since the declarations of significance were objectively and independently made for the two traits, the odds that the epistasis found for ABA level would coincide by chance alone with one of the two cases of epistasis found for dormancy is only 2/1305, and the P value is 0.0015.

Furthermore, if the coincidence of epistasis for dormancy and ABA level had been a chance effect, there would be no reason to expect similarity in the patterns of epistasis. All four possible combinations at *TG234* and *TG155* showed a good correlation between ABA levels and length of dormancy: three of the four possible allelic combinations at *TG234* and *TG155* were associated with low levels of ABA and short dormancy, and the remaining allelic combination was associated with a high ABA content and long dormancy (Fig. 4). By permutation analysis (a conservative approach), the probability of a finding by chance alone an equally good or better fit between the patterns for dormancy and for ABA level is represented by P = 0.208. Multiplying the two P values gives an overall P value of 0.0003, which estimates the probability that the same case of epistasis would be found for ABA level as for dormancy and that the pattern fit would be as good. Thus, to explain

the similarity between the dormancy and ABA results by chance alone would require a very remote combination of coincidences.

If two of the QTLs detected for ABA coincided with those detected for dormancy, it is reasonable to ask why the other QTL did not. There could be various explanations. One is that differences in ABA found for *TG234* and *TG155* might reflect the status of ABA in those parts of the tubers (or cells) most responsible for controlling dormancy, whereas ABA differences responsible for the QTL linked to *TG499* might be located in parts that would have little effect on dormancy. Suttle (1995) excised the apical bud clusters from tubers, taking a thin slice of periderm and buds for ABA analyses. It would be interesting to repeat the polygene mapping using ABA levels from tissue around the apical buds to see whether the same QTLs would be found. Sorce et al. (1996) analyzed tubers for ABA content in pith and in what they termed "eyes" and "subeyes." No measurements were made during the important interval between harvest and the end of dormancy, but ABA levels at harvest and at the end of dormancy were much lower in eyes than in subeyes. Values for pith were similar to or slightly lower than eyes (Sorce et al., 1996).

Another possible reason why the QTL linked to *TG499* did not coincide with a QTL for dormancy is that there may be QTL(s) for other hormone(s) linked to the QTL for ABA in such a manner that the effects on dormancy are masked. The analyses under way for GAs and other hormones may be informative in this regard.

Nature of Causality

Evidence for a causal relationship does not itself distinguish which trait is the cause and which is the effect. Could it be that long dormancy promotes high ABA rather than the reverse? The possibility cannot be ruled out, although the known properties of ABA as an inhibitor would make the reverse case seem more likely. It should also be noted that the QTLs for ABA and dormancy linked to markers *TG65* and *TG155* coincided with a QTL found for tuberization on cuttings (Van den Berg, 1996a). A reasonable hypothesis would be that in this backcross a gene linked to *TG155* controls the level of ABA in both shoots and in tubers, that higher ABA controlled by this gene in tubers prolongs dormancy, and that higher ABA level in shoots promotes better tuberization on cuttings. To test this hypothesis we plan to search for QTLs for ABA content in leaves. If it turns out that ABA level in both leaves and tubers is associated with the QTL linked to *TG155*, then the reasonable conclusion would be that ABA has a causal relationship to dormancy. (Since tuberization precedes dormancy, it would be illogical that longer dormancy could cause better tuberization or that longer dormancy in tubers would cause higher ABA, which would lead to better tuberization.)

Because the markers for segregation from the recurrent parent were not as numerous or well distributed as those for segregation from the hybrid, there is a good chance that still other QTLs for ABA segregating from the recurrent parent were not detected. This could explain in part why the combined model accounted for only 25% of phenotypic variation. A large number of minor QTL effects might also be responsible for variability in the assay. No correlation was found between ABA and dormancy (Fig. 2). We could expect the correlation to be low if ABA is only one of many causal factors, which is consistent with current speculation that both tuberization (Ewing, 1995) and dormancy (Ludford, 1995; Suttle, 1995) are controlled by a balance of hormones; i.e. if two QTLs are associated with dormancy because of effects on ABA levels, and seven other dormancy QTLs are associated with the levels of other factors such as GAs, cytokinins, tuberonic acid, or polyamines, then it would not be surprising if variation in dormancy caused by the other factors makes it impossible to detect correlation between ABA level and dormancy. The fact that a relationship between ABA and dormancy could be demonstrated through polygene mapping, even though none was detected by a conventional correlation test, illustrates the superiority of the mapping approach, a conclusion also reached by Lebreton et al. (1995).

Other Evidence on ABA and Tuber Dormancy

Control of tuber dormancy likely involves an interplay of various hormones and other substances (Ludford, 1995). GAs, cytokinins, and even polyamines may well be involved, but ABA has received special attention as a part of the β-complex of inhibitors (Holst, 1971; Hemberg, 1985). Coleman and King (1984) found a decline in tuber ABA levels in two of three cultivars at the end of dormancy but no evidence of a threshold level below which sprouting could not occur. Suttle (1995) monitored ABA in tubers of one cultivar under three storage regimes. Even though in other studies the sustained presence of ABA was considered essential for the induction and maintenance of tuber dormancy (Suttle and Hultstrand, 1994), breaking of dormancy could not be attributed to the decline in ABA below a threshold level (Suttle, 1995). He concluded that other internal processes or factors were responsible for the loss of dormancy under natural conditions (Suttle, 1995). This is compatible with our observation that only two of the eight QTLs for dormancy in this backcross coincided with QTLs for ABA, especially if we assume that changes in GAs, cytokinins, or other factors may have eclipsed the effects of ABA in Suttle's experiments. In this connection we have found a QTL for content of spermine in potato leaves (Mueller et al., 1996), different from the QTLs detected for ABA content in tubers, that is linked to QTLs for both tuberization (Van den Berg et al., 1996a) and tuber dormancy (Van den Berg et al., 1996b). If a balance of growth promoters and inhibitors controls dormancy, then the changes in any one hormone viewed in isolation from the others is not likely to yield the answer.

Other Polygene Mapping

Freyre et al. (1994) detected QTLs for tuber dormancy in a hybrid population between a haploid of *S. tuberosum* × *Solanum chacoense* crossed with *Solanum phureja*. *S. phureja* has much shorter dormancy than *S. tuberosum* and contrib-

uted dominant genes for tuber dormancy (Freyre et al., 1994), whereas *S. berthaultii* in our study had a much longer dormancy than *S. tuberosum* and contributed recessive genes for long dormancy (Van den Berg et al., 1996b). Although there were too few common markers to permit a precise comparison of the populations analyzed in these two studies, Van den Berg et al. (1996b) noted that five of the chromosomes with significant QTLs were the same in both investigations. Chromosome 7, which contained the most important QTLs for segregation from the *S. tuberosum* × *S. chacoense* hybrid, was not detected as having significant QTLs in our population (Van den Berg et al., 1996b). Nevertheless, in the present ABA analysis we have found a QTL (*TG499*, Fig. 3) on chromosome 7 that appears to be at least close to the QTL for dormancy located by Freyre et al. (1994) at marker *Got-2*. The other QTL with a direct effect on ABA content, located on chromosome 4, could correspond to *Pgm-2*, a dormancy QTL detected by Freyre et al. (1994). Our data indicate that the QTL for ABA is between *TG123* and *TG208*, 8 cM from *TG123* (Fig. 3), and that *Pgm-2* is located between the same two markers, 11 cM from TG123 (Tanksley et al., 1992).

Lebreton et al. (1995) found commonality in maize between QTLs for ABA and QTLs for root-pulling force, stomatal conductance, and turgor pressure, all of which imply modifications in morphology. The present study establishes through the polygene mapping of a single segregating population an association between a plant hormone and a specific developmental change.

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