The Korean sesame breeding programme uses the 'dt-45' mutant with determinate plant growth, which was developed by A. Ashri in Israel, for earlier maturity and synchronization of the flowering period. Many crosses were made with 'dt-45' and determinate advanced lines like Suwon 129, 131, 133, 134, and 135 were the result. All these lines possessed the determinate character, good resistance to several diseases, lodging resistance and early maturity. Nevertheless, due to a low number of capsule setting nodes the yield was not acceptable and did not exceed the existing varieties.

The search for the 'ideal plant type' continues. This means determinate growth, semi-dwarfness, lodging resistance, larger seed size, few and non-branching stems, early maturity and high yield potential. The mutant derived line 'SI 90033-2B' has most of these characteristics and is believed to be closest to the ideal sesame plant type for Korean agriculture.

(Contributed by KANG, C.W. and L. van ZANTEN*. National Crops Experiment Station, Rural Development Administration, Suwon 441-100, Republic of Korea; * Plant Breeding and Genetics Section, FAO/IAEA Joint Division, P.O. Box 100, Vienna, Austria)

TOPIC FOR DISCUSSION

70 YEARS INDUCED MUTATIONS - TO BE RECONSIDERED?

Almost 70 years ago researchers began to be successful in mutagenic treatments for altering genes. A mutation was detected phenotypically and verified genetically afterwards. In most cases monogenic recessive inheritance was established.

According to the prevailing concept at that time, "qualitative traits" were assumed to be controlled by one or very few genes, "quantitative traits" by many genes. One had already learned, that genes could freely recombine, unless they were tightly linked in a chromosomal section. Great attention was paid to "gene/environment interactions", separating traits with "high heritability" from those with "low heritability". Mutagenesis, however, was supposed to be capable of altering all genes irrespective of their chromosomal location, linkage group or level of heritability. Those with "high heritability" of course were easier to handle and identified as the more promising targets for mutation induction.

When plant breeders speak about gene/environment interactions, the environment is usually considered under the aspect of physical and chemical conditions outside the plant (e.g. location, year, stress), supporting or restricting performance. This neglects the fact that interaction among genes creates some kind of "genetic environment". Plant breeders tend to focus on particular genes assumed to be responsible for traits relevant for cultivar improvement. The other genes are downgraded by being lumped into the "genetic background". This thinking also prevailed so far in application of induced mutations in breeding programmes.

The possibility to alter individual genes, leaving the rest of the genome intact was always stated as one of the assets of mutagenesis. That the rest of the genome - which in fact is its major part - was also exposed to the mutagen and therefore also carries all kinds of mutations was too often forgotten or hushed up. At best, an attempt was made to eliminate unwanted "background mutations" by crossing, but besides linkages with unwanted mutations one experienced also correlations and pleiotropy.
Looking at "gene physiology" it is now common-place, that genes code for a particular protein possessing enzymatic function. Thereby, every gene is supposed to control a step in chemical processes which ultimately lead to physiological and morphological traits. Plant breeders usually relate the function of a gene directly to the phenotypic trait (like e.g. a gene for early flowering, a gene for high oil content, a gene for short culm), since for economically important genes there is hardly any knowledge about intermediate steps of gene expression. Such a simplistic view of genetic control certainly is not correct.

Geneticists now distinguish e.g. between structural genes, determining the structure and thereby the role of an enzymatic protein leading to a certain product (trait), and regulatory genes, which play the role of somehow controlling the function of other genes (such regulatory genes should not be confused with the regulatory DNA-sequences that are part of every gene and control its transcription and replication). In studying the role of a particular gene, usually the concept of "chemical pathways" is followed, in which consecutive steps are controlled by individual genes. It seems, however, that in most cases the concept of a "chemical and physiological network" would be more adequate, where genes depend on other genes for their functioning and the degree of expression. Here, only genes acting towards the end of a pathway can clearly be associated with a particular trait.

Induced mutations have been used effectively for identifying genes having a major phenotypic effect in certain pathways. Mutations in genes active within a network, however, are rather difficult to locate. If the network includes parallel pathways, it should be virtually impossible to detect mutated genes. Adopting the "network concept" therefore should have serious consequences for the strategy of using induced mutations for plant improvement. One possible approach was met with little success, namely using statistical methods for identifying positive changes in quantitative traits ("micro-mutation" experiments).

The recognition of a hierarchy among genes and as part of it the distinction between structural and regulatory genes, however, has still other consequences. For interpreting results of mutation induction experiments one would have several options, e.g.:

1) A mutated trait may be caused by a change in a directly related structural gene.
2) A mutated trait may be caused indirectly by a change in a regulatory gene which altered the expression of a structural gene. The difference to (1) could only be noticed in test crosses, not by selfing or by backcross to the original stock.
3) A mutated trait may be caused indirectly by a change in a regulatory gene which altered the expression of several structural genes. In this case pleiotropy of the mutation could be observed.
4) A gene perhaps could have both functions and therefore a "direct change" would be accompanied by influence upon the expression of other genes. In this case one would probably conclude, that several mutations occurred at the same time, which surprisingly would all be inherited together.

One should accept the fact that most mutagen induced DNA changes (particularly those by ionizing radiations) are genetic lesions. The interference of mutagens with the complicated structure of a gene with its thousands of base pairs and the meticulously conserved nucleotide sequence may only seldom lead to an altered enzyme, that is still able to function (missense mutation). In most cases one has to assume that the damaged gene can no longer properly code for the original or a related enzyme and has therefore lost its function (non-sense mutation). A mutation leading to a non-functioning gene will be classified as recessive, although in most cases the term "deleted" would be more appropriate. Of course at the molecular level one can detect great differences between such genetic lesions (mutant alleles), depending to some extent on the mutagen used and probably also on repair systems.
At the physiological level, one should ask which kind of genetic lesion a cell/plant may tolerate, and -of course- also, which kind of mutation could bring benefit in adaptability, competitiveness etc. One could certainly distinguish between "essential" genes and what might be called "peripheral" genes. A dominant or homozygous-recessive mutational lesion in an essential gene could not easily be tolerated by the plant and would probably have a lethal effect, unless the plant's genome contains intact copies of the affected gene elsewhere. Such multiple genes have been considered by breeders in the past mostly in relation to polyploids, but in the meantime it is well known, that duplicated sequences and even chromosome segments are a common phenomenon in the genomes of higher plants. Moreover, phylogenetic studies using molecular techniques unveiled often unknown evolutionary polyploidy as well as homoeologies among genomes of unrelated species (syntheny). Thus it seems that mutagenesis has only little chances to create useable genetic variation in essential genes.

It follows as a consequence, that mutagenesis studies during the past 70 years have probably mostly dealt with those genes that are not "essential", not protected by copies, and for which a plant could tolerate drastic changes, inactivation or even the loss. It needs to be investigated, however, how many of the "essential" genes are structural genes and how many of the "peripheral" genes are actually genes with a regulatory function.

At this point another clarification may be useful: although plant breeders are used to describe the genotype of a plant by its traits, the traits assumed to be the result of specific genes, one should recall, that genes do not function throughout the whole life of a plant, do not function in all tissues, do not even function all day. Many genes only function in response to a particular trigger, maybe from outside the plant, maybe from inside the cell. The ultimate phenotype of a plant then is not simply the sum of individual gene effects, but the product of numerous short and long, single and recurrent processes, of differential gene activities in specialized tissues, of gene responses to environmental stimuli. Mutations do not have to alter structural genes to obtain a mutant plant. It would be sufficient and probably more efficient, to alter the timing of gene activities, the location of gene activities, the intensity and speed of response to stimuli from outside (this might well be an interesting aspect for explaining natural evolution).

In the event of a mutation in a gene with regulatory functions it is likely, that - as usual - the "wild type" will be dominant and the mutant recessive. However, among regulatory genes there will be some that promote and some that inhibit the expression of another gene. If for example, by mutagenesis an inhibitor gene is inactivated or removed, this will uncover another gene's function, a gene one was perhaps completely unaware of. The mutation, although being recessive, results in promoting a particular gene product. Whether the particular inhibition requires two gene doses and therefore is already lifted in the heterozygous mutant would depend on the individual case. In selfing the heterozygous mutant, also after a "backcross" with the original stock, one will see as expected a monogenic recessive segregation, with 25% of individuals showing the "new mutant character". If, however, the mutant plant is crossed with other genetic stocks (which may not carry the inhibitor gene), e.g. to test for allelic identity, the whole F₁ may show what seemed to be a recessive mutated character and the F₂ might segregate for 75% of the mutant phenotype, simulating a dominant mutation. Reported changes in dominance in literature may be explainable by the dual control of a particular phenotype through a structural and a regulatory gene.

If the "wild type" regulatory gene has a promoting function and its deletion leads to the inhibition of another gene, the reverse situation would be seen: depending upon the individual case, the heterozygous mutant may still possess the wild type phenotype (one gene dose sufficient for the promoting effect, inhibition completely recessive). But it could also
be, that the heterozygous mutant would show a reduced function (semidominance) or a complete inhibition (dominance) of the controlled gene, in which case one would wrongly conclude to have induced a dominant or semi-dominant mutation.

In induced mutations for disease resistance, these considerations could be of far reaching relevance. Quite often, reports of mutants with improved resistance were questioned, if the mutant gene was described as dominant or when it was claimed that the mutation improved simultaneously the resistance against more than one "race" of a particular pathogen or pest. When afterwards in test crosses it could be shown, that the mutant gene was identical with one already known, the mutation work was often discredited and the researcher accused of not preventing outcrossing.

Thus, in the light of the possibility that induced mutations may happen in regulating genes and not in the genes normally associated with a trait, it may be justified to reconsider many mutation experiments of the past 70 years and to re-examine results that looked like false claims of success of application of induced mutations in breeding.

*(Contributed by A. MICKE, Salmannsdorfer Str.94, A-1190 Vienna, Austria)*