# Scent engineering: toward the goal of controlling how flowers smell

**Eran Pichersky[1] and Natalia Dudareva[2]**

[1] Department of Molecular, Cellular and Developmental Biology, University of Michigan, 830 North University Street, Ann Arbor, MI 48109, USA
[2] Department of Horticulture and Landscape Architecture, Purdue University, West Lafayette, IN 47907, USA

**Floral scent has an important role in the reproductive processes of many plants and a considerable economic value in guaranteeing yield and quality of many crops. It also enhances the aesthetic properties of ornamental plants and cut flowers. Many floral scent volatiles fall into the terpenoid or phenylpropanoid/benzenoid classes of compounds. Although the biochemistry of floral scent is still a relatively new field of investigation, in the past decade investigators have begun to identify 'scent genes'. Several of these genes, most of which, but not all, encode enzymes that directly catalyze the formation of volatile terpenoid or phenylpropanoid/benzenoid compounds, have now been used to manipulate, through genetic engineering techniques, the mix of volatiles emitted from the flowers of several plant species. The outcomes of these experiments, which are discussed here, have indicated that the genetic engineering approach to altering floral scents has potential; however, they have also revealed the limitations that result from our inadequate knowledge of the metabolic pathways responsible for scents and their regulation.**

## Why is it useful to introduce or change floral scents?

Floral scent is one of the adaptations that plants have evolved to attract pollinators. The volatiles emitted from the flowers – typically a mixture of several or even scores of compounds – provide potential insect and animal pollinators with information about the location and identity of the flowers. Efficient pollen dispersal, which results in maximum pollen transfer to conspecific flowers, increases fertilization rates and minimizes energy expenditure by the plant, thus increasing fitness. In crop plants where some edible parts of the plant (i.e. the fruit or seed) require fertilization to develop, and where complete fertilization of all the ovaries in a flower is sometimes required for fruits to develop the symmetrical shape favored by consumers, the success rate of fertilization influences both yield and quality.

Sub-optimal pollination rates are common in both cultivated and wild species [1]. The problem is often exacerbated in cultivated plant species where large numbers of conspecific individual plants are crowded together, which, in turn, require large numbers of their specific pollinators. In addition, some crop species are introduced from other parts of the world and do not have their co-evolved pollinators in their present locality [2]. Often, the morphology and biochemistry of such plants have been drastically changed during domestication, without concomitant adaptations in the pollinators.

In extreme cases, a lack of natural pollination can prevent plants from being commercially introduced as crop plants into a new territory [1]. Although breeding for self-pollination or for fruit development without pollination (apomixis) as well as achieving apomixis by chemical means could, in some cases, alleviate this problem [3], self-pollination leads to genetic uniformity and thus increases the probability of the spread of diseases.

In the USA and most other countries where large-scale monoculture agriculture is practiced, domesticated honeybees are used to pollinate many different crops. Beekeepers often derive a large portion of their income from their fees for placing beehives in orchards or crop fields. However, these bees (*Apis mellifera*) are, themselves, non-native to the USA and are not always well suited for the task [4]. Although it is known that the bees find flowers by cues from visual (color, shape) and olfactory signals, the relative importance of the various volatile, color and shape components, and their synergistic interactions, are far from understood [5]. It is clear, however, that bees do not visit all flower species with equal frequency or pollinate them with equal success. In some cases, a lack of scent has been implicated in the failure of flowers to be efficiently pollinated [6]. Recent outbreaks of diseases that have greatly reduced the numbers of domestic and wild honeybees in the USA have exacerbated the problem [7,8].

Genetic engineering of floral scent in crop plants could alleviate the problems listed above. It is envisioned that the scent of both local and introduced plant species could be enhanced to better appeal to local pollinators, thus increasing pollination efficiency and reproductive success.

For humans, scented flowers also constitute a commodity with strong aesthetic and emotional values. Unfortunately, floral scent has been a casualty of plant-breeding programs for the cut-flower market and ornamental plants in general. Despite the oft-expressed sentiment by consumers that they like scented flowers, the cut-flower industry operates under the assumption, based on actual market research, that 'the public in general will not pay an extra cent for scented flowers'. Consequently, breeders in this multi-billion dollar industry have concentrated on producing plants

*Corresponding author:* Pichersky, E. (lelx@umich.edu).
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with improved vase life, shipping characteristics and visual aesthetic values (i.e. color and shape). Owing to the lack of direct selection, or perhaps because of a negative correlation with any of these traits, many cultivated flowers have lost their scent [9]. Genetic engineering could, perhaps, restore scent to these varieties without sacrificing other important commercial traits and might, thus, produce extra value for the niche market of consumers who really do prefer scented flowers and are willing to pay extra to get them.

Here, we describe recent attempts to modify specifically floral scent by genetic engineering techniques rather than by traditional breeding approaches, with the emphasis on two important pathways that contribute a large number of floral volatiles – the terpenoid and phenylpropanoid/benzenoid pathways. Although the terpenoid pathway was the first and is still the most active area for such attempts to modify not only floral scent but also vegetative and fruit volatiles [10], recent progress in elucidating the phenylpropanoid/benzenoid pathways leading to plant volatiles and the isolation of genes involved in these pathways have opened up new opportunities for the modification of floral scent.

## Candidate pathways, general strategies and possible pitfalls

Most of the volatiles in plants belong to one of three major classes of compounds: terpenes, phenylpropanoids or fatty acid derivatives [11,12]. Other volatiles are derived from various amino acids. In general, plant volatiles are considered part of secondary, or specialized, metabolism because most of them are produced only in specific plant lineages and function in specific ecological roles unique to these lineages. They are not as widespread as primary metabolites, which are, by definition, found in almost all plants. However, primary and specialized metabolic pathways are not completely separate; rather, specialized metabolites are mostly produced in the terminal branches of the network of primary metabolism. Thus, in some cases a single reaction and a single enzyme will convert a primary metabolite into a volatile compound, whereas in other cases multiple steps are required [11,12].

For example, a single enzyme converts phenylalanine to phenylacetaldehyde – a volatile found in the floral scent of rose, petunia and many other species [13] – whereas eugenol, another volatile belonging to the phenylpropanoid class, is synthesized in two steps from coniferyl alcohol – an intermediate in the general lignin biosynthetic pathway of plants [14,15] (Figure 1). Likewise, many volatile monoterpenes and sesquiterpenes can be synthesized in a single reaction from geranyl diphosphate and farnesyl diphosphate, respectively; both of these are intermediates in the pathways leading to primary plant metabolites such as sterols, carotenes, chlorophylls, gibberellins and abscisic acid [12]. Numerous terpene synthase genes from various species have now been isolated and characterized. Several genes have also been identified that encode enzymes that convert non-volatile compounds into volatile ones by modification reactions such as methylation, acetylation, and decarboxylation [12]. Expressing these genes in the flower could potentially result in the production of new volatiles. In some cases, there are competing branches in a biosynthetic pathway that lead to volatile and non-volatile (or different volatile) products in the flower, whereby suppressing one branch could lead to enhanced production of the desired volatile.

The choice of specific genes to engineer floral scent thus depends on the availability of genes, the specific goal (i.e. the desired aroma effect for either human consumption or the targeted pollinator), and the ability to siphon compounds from primary metabolism while avoiding undesirable side effects. For example, expressing a gene encoding an enzyme that uses a common primary metabolite to make a volatile might not lead to a noticeable production of this volatile in the flower if the primary pathway is not highly active in this tissue. In addition, expressing such a gene under a constitutive promoter everywhere in the plant could lead to deleterious effects, either from the toxicity of the accumulated compound in non-flower tissue (or even in flowers if it is not emitted fast enough) or from the diversion of the flux of the primary metabolism pathway, which causes a deficiency in a needed compound.

## Successful and not-so-successful attempts to engineer scent

To date, the criteria for success in metabolic engineering of floral scent have been based on sensory evaluations by humans, whose odor threshold perception is much lower than that of most animals or insects [16,17]. Unfortunately, the impact of changes in the scent bouquet on insect and animal attraction has not yet been reported.

### *Engineering of terpenoid volatiles*

First attempts to engineer floral scent were focused on modifications of the terpenoid spectrum. The terpenoid pathway was an inviting target because isoprenoid precursors are ubiquitous molecules in plant tissues and, as described above, they also serve as precursors in the biosynthesis of several essential primary metabolites; therefore, they would be available for the synthesis of terpenoid volatiles using introduced terpene synthases. The most often used gene in these initial attempts was linalool synthase (LIS) from the flowers of *Clarkia breweri*, an annual native to California [18]. LIS converts geranyl diphosphate (GPP) to (3*S*)-linalool, a monoterpene alcohol with a sweet, pleasant fragrance that is found in the flowers of many species. Overexpression of LIS under the control of the constitutive 35S promoter in *Petunia hybrida* (petunia) [19] and *Dianthus caryophyllus* (carnation) [20], both of which do not emit this monoterpene from either their leaves or their flowers, indeed resulted in linalool production in both leaves and flowers. However, the synthesized linalool had no effect on the olfactory properties of the flowers or vegetative parts of the transformants. In petunia, most of the linalool was converted by an endogenous enzyme into non-volatile linalyl β-D-glucoside. In transgenic carnation, most of the synthesized linalool was further metabolized into the volatile *cis*- and *trans*-linalyl oxides. Although these extra terpenes constituted almost 10% of the total volatiles emitted from the transgenic flowers, this increase in scent emission was

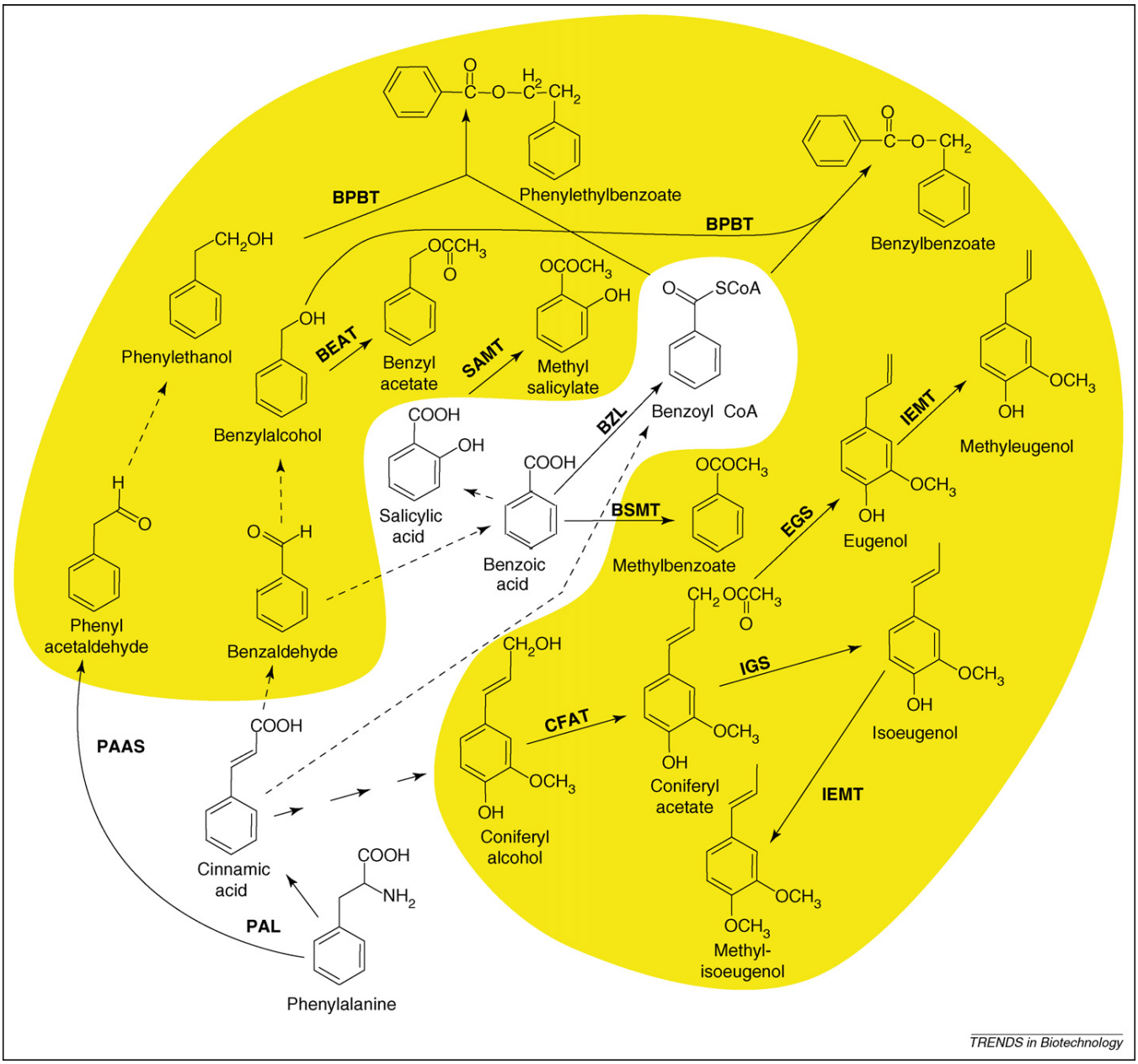


**Figure 1**. An overview of the biochemical reactions leading to the synthesis of volatile benzenoids/phenylpropanoids found in floral scents of various plants. This chart is a compilation of reactions and enzymes discovered in several plant species that have served as model organisms in the study of floral scent, but all the reactions and volatiles shown here occur in either *Clarkia breweri* or petunia, or both. Solid lines indicate established biochemical reactions, and broken lines indicate possible steps for which enzymes have not yet been characterized. Salicylic acid is shown here as possibly derived from benzoic acid, although it might also be derived from isochorismate [34]. BEAT, acetyl-coenzyme A:benzyl alcohol acetyltransferase; BPBT, benzoyl-CoA:benzyl alcohol/2-phenylethanol benzoyltransferase; BSMT, benzoic acid/salicylic acid carboxyl methyltransferase; IGS, isoeugenol synthase; BZL, benzoate:CoA ligase; CFAT, coniferyl alcohol acyltransferase; EGS, eugenol synthase; IEMT–*S*-adenosyl-L-methionine:(iso)eugenol O-methyltransferase; IGS, (iso)eugenol synthase; PAAS, phenylacetaldehyde synthase, PAL, phenylalanine ammonia-lyase. SAMT, salicylic acid carboxyl methyltransferase. Volatile compounds are shown with a yellow background.

still not enough for most humans to detect a change in floral aroma in smell tests [20]. These pioneering experiments revealed additional problems that have to be considered in the genetic engineering of flower fragrance: the modification of the scent compound into a non-volatile form by endogenous, non-specific enzymes; insufficient levels of emitted volatiles for olfactory detection by humans; or masking of introduced compound(s) by other volatiles [20].

In more recent experiments, successful changes in the terpenoid volatile profile were achieved in *Nicotiana tobacum* (tobacco) plants through the introduction of three lemon monoterpene synthases under the control of the constitutive 35S promoter [21]. These monoterpene synthases use GPP as a substrate but produce multiple products in varying ratios. In addition to the usual terpenoids emitted by the parental line, the transgenic tobacco plants also produced and emitted β-pinene, limonene, γ-terpinene and several other products from their leaves and flowers. Importantly, the emission levels of the new products were sufficient for detection by the human nose [22]. In flowers, the comparably high levels of the introduced monoterpenes did not reduce the emission of linalool,

which was already emitted by the non-transgenic plants. The introduced monoterpene synthases competed for the same GPP substrate, suggesting that the substrate pool is not limiting for monoterpene production [21]. Crosses between transgenic lines harboring different introduced monoterpene synthases resulted in progeny with an even more complex monoterpene emission spectrum [21].

In contrast to LIS-transformed petunia and carnation plants [19,20], no further modification products of the primary monoterpenes were detected in these transgenic tobacco plants. In subsequent experiments, the monoterpenoid profile in these transgenic tobacco plants was further modified by introducing the mint limonene-3-hydroxylase, which catalyzes the hydroxylation of (+)-limonene to form (+)-*trans*-isopiperitenol [23].

Although the terpene synthase genes used in the experiments were introduced under the control of the 35S promoter, the analyzed plants, in general, did not appear to be negatively affected by the expression of the heterologous genes. However, the amounts of the new terpenes synthesized by the transgenic plants was relatively low; and it is probable that plants expressing the introduced genes at a higher level did suffer adverse effects [24] and were, therefore, not selected for further analysis.

### *Engineering of phenylprpanoid/benzenoid volatiles*

Because many of the enzymes for volatile biosynthesis can use multiple substrates, the particular volatiles produced in the flowers of transgenic plants will depend on the substrates available in the floral cells in which the transgene is expressed [11,25]. This is also true for endogenous genes. For example, in petunia flowers the endogenous benzoic acid/salicylic acid carboxyl methyltransferase (PhBSMT) apparently has higher catalytic efficiency with salicylic acid than benzoic acid, but the flowers do not emit methylsalicylate as a result of the small internal pool of free salicylic acid. Thus, the enzyme is responsible for the formation of methylbenzoate using the substantial amount of benzoic acid present within the cells [26,27] (Figure 1). Petunia flowers also emit low levels of benzyl acetate and phenylethyl acetate. When *Rosa hybrida* (rose) alcohol acetyltransferase (RhAAT), which catalyzes the formation of geranyl acetate from geraniol and acetyl-CoA in rose flowers [28], was expressed under the control of the 35S promoter in petunia, it used the endogenous phenylethyl alcohol and benzyl alcohol instead of the unavailable geraniol, and significantly increased the emitted levels of benzyl acetate and phenylethyl acetate in transgenic flowers [29]. Feeding of transgenic flowers with geraniol, the preferred substrate in the *in vitro* assays, or with 1-octanol, an additional potential RhAAT substrate [28], led to the production of their respective acetates, confirming the previous conclusion that the function of the introduced gene *in planta* depends on substrate availability.

To date, the metabolic engineering of floral scent has mainly concentrated on the introduction of the genes responsible for the final steps of the formation of volatile compounds. The redirection of metabolites by restricting the specific fluxes is another approach that has occasionally been tried. For example, antisense suppression of flavanone 3-hydroxylase, an enzyme in the biosynthetic pathway leading to the formation of anthocyanin pigments, resulted in an unpredicted rise in the levels of emitted methylbenzoate in transgenic carnations; the difference could be detected by the human nose [30]. Because both benzoic acid and flavones are ultimately derived from the shikimate pathway, the blockage in the anthocyanin pathway led to an increase in flux in the pathway leading to benzoic acid and, ultimately, methylbenzoate.

Phenylpropanoid/benzenoid floral scent profiles have also been modified by the elimination of some volatile compounds from the scent bouquet. This work has, so far, only been done in petunia. RNAi-mediated silencing of the PhBSMT gene resulted in transgenic petunia plants that lack the major scent component methylbenzoate, with minimal changes in the emission of other volatiles [27]. The change was easily detected by a human sensory panel, which reacted negatively to the decrease in floral scent [27]. More recently, RNAi silencing of the petunia phenylacetaldehyde synthase gene (PhPAAS) not only led to the complete elimination of the emission of phenylacetaldehyde but also of 2-phenylethanol, for which it is a precursor [13] (Figure 1). Silencing the petunia benzylalcohol/phenylethanol benzoyltransferase (PhBPBT) (Figure 1) by RNAi resulted in plants whose flowers did not emit benzylbenzoate or phenylethylbenzoate, although emission of all other volatiles remained unchanged [31]. Interestingly, plants with fully suppressed PhBPBT expression also had clear morphological differences, such as bigger flowers and larger leaves. These differences, which are probably due to an, as yet, unexplained interaction between the benzenoid pathway and auxin [31], demonstrate the unpredictable nature of metabolic engineering in general, but scent engineering in particular. Finally, silencing of coniferyl alcohol acyltransferase (CFAT), the enzyme that catalyzes the formation of coniferyl acetate (the precursor of isoeugenol and eugenol; Figure 1) [14], led to almost complete elimination of isoeugenol emission in petunia flowers, with little effect on the emission of other phenylpropanoid/benzenoid volatiles [15].

## Future prospect

The examples described above (and summarized in Table 1) show that metabolic engineering of floral scent is now feasible. However, whether newly introduced 'scent enzymes' will find appropriate substrates, and whether the intended products will be produced and emitted at levels that can be detected by humans and other animals, including insects, will depend on the specific plant–animal pair interactions. These factors cannot be predicted presently because of insufficient understanding of plant metabolic pathways as well as animal olfactory systems. The generation of metabolic flux models of the relevant pathways will provide information for rational metabolic engineering. Useful data for these models are now being obtained from many types of experiments, including those using transgenic technology to increase or change scent production [31], and from the identification and characterization of earlier steps in the scent biosynthetic pathways.

The observation that flowers coordinately synthesize many different scent volatiles that are often derived

**Table 1. Approaches used for metabolic engineering of floral scent**

| Approach | Engineered species | Gene used | Result achieved | Olfactory effect | Refs |
|---|---|---|---|---|---|
| Introduction of a single gene | Petunia | CbLIS | Linalyl glucoside | No | [19] |
| | Carnation | CbLIS | Linalyl oxides | No | [20] |
| | Petunia | RhAAT | Benzyl acetate and phenylethyl acetate | ND | [29] |
| Introduction of multiple genes | Tobacco | CITER, CILIM, CIPIN | γ-terpinene, limonene, and β-pinene and side products | Yes | [21,22] |
| Introduction of multiple steps | Tobacco TERLIMPIN | MsLIM3H | Isopiperitenol and derivatives | ND | [23] |
| Elimination of some compounds | Petunia | PhBSMT RNAi | Lacks methylbenzoate | Yes | [27] |
| | | PhBPBT RNAi | Lacks benzylbenzoate and phenylethylbenzoate | ND | [31] |
| | | PhPAAS RNAi | Lacks phenylacetaldehyde and phenylethanol | ND | [13] |
| | | PhCFAT RNAi | Lack of isoeugenol | ND | [15] |
| Blocking of competitive pathways | Carnation | Anti-DcF3′H | Increased methylbenzoate emission | Yes | [30] |
| Down-regulation of transcription factor | Petunia | PhODO1 | Reduced levels of volatile benzenoids | ND | [32] |

Abbreviations: CILIM, limonene synthase; CIPIN, β-pinene synthase; CITER, *Citrus limon* γ-terpinene synthase; CbLIS, *Clarkia breweri* linalool synthase; DcF3′H, *Dianthus caryophyllus* flavanoid 3′-hydroxylase; MsLIM3H, *Mentha spicata* limonene-3-hydroxylase; ND, not determined; PhBPBT, benzylalcohol/phenylethanol benzoyltransferase; PhBSMT, petunia benzoic acid/salicylic acid carboxyl methyltransferase; PhCFAT, coniferyl alcohol acyltransferase; PhODO1, ODORANT1 transcription factor; PhPAAS, phenylacetaldehyde synthase; RhAAT, *Rosa hybrida* alcohol acetyltransferase. Tobacco TERLIMPIN is a tobacco transgenic line expressing CITERM, CILIM and CIPIN.

from multiple pathways indicates that the main 'switch' in this process occurs upstream of individual metabolic pathways. Transcription factors that are able to control the multiple pathways leading to the formation of fragrance have not yet been identified. However, the first transcription regulator of the formation of volatile benzenoids in petunia, ODORANT1, was recently discovered [32]. Additional progress in discovering transcription factors will hopefully result in additional tools that can offer an efficient strategy to manipulate the flux through the metabolic pathways to improve scent emission.

Present technology for the genetic engineering of plants in general requires the ability to regenerate plants from callus tissue. To date, successful transformations have been developed for several cut flowers, including commercially important roses, chrysanthemums, carnations and gerbera, although for most varieties it is still an 'art form' [33].

Overall, it is clear that genetic manipulation of floral scent is possible but will require a more rational design based on the correct choice of species, previous knowledge of the pathways involved, including their cellular and subcellular localization, judicious use of promoters, and empirical testing.


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