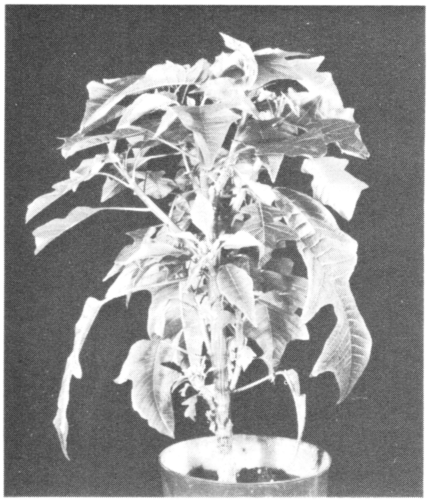

Fig. 4. Bud applications of BA at 25 mg/ml to every node induced axillary bud development. SD for flower induction was initiated after most of the axillary shoots elongated. The lower nodes on the woody stem developed more slowly and thus were abnormally effected by the SD treatment.

of either the main or lateral shoots. In addition, very little deformity was evident in growing shoots. Compared with growth in normal pinched plants, growth of lateral branches was slower in BA treated plants.

Carpenter et al. (2) showed that 74% of the axillary buds developed in 'Eckespoint C-1 Red' after a spraying with BA at a concentration of 500 ppm, and 80% developed at 1000 ppm. Approximately 60% of these buds developed into lateral branches. In contrast, direct bud application of BA at concentrations of 5, 25, and 50 mg/ml in lanolin induced 100% of the buds to develop into actively growing shoots (Fig. 3). Foliar sprays of BA promote growth of only the apical axillary buds, whereas BA in lanolin induced growth even in axillary buds near the base on woody stems (Fig. 4).

The treatment of dormant axillary buds with a BA lanolin suspension could have 2 possible commercial applications. First, this procedure would allow one to produce an increased number of cuttings from stock plants. Useful shoots for cuttings could even be obtained from woody stems. Second, this procedure produces a much fuller plant with a larger number of colored bracts than sprayed plants. These plants should demand a premium in the retail trade.

Since the poinsettia is a short day plant, timing of BA applications to obtain normal flowering shoots is very critical. Normal shoot growth will result if the BA treatment is given several months before the onset of short days required for flower bud initiation.

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Delay of Senescence in Carnations by Pyrazon, Phenidone Analogues, and Tiron

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Additional index words. aminooxyacetic acid, aminoethoxyvinylglycine, *Dianthus caryophyllus*, preservative solution, lipoxygenase inhibitors

Abstract. **Pyrazon [5-amino-4-chloro-2-phenyl-3(2*H*)-pyridazinone], phenidone (1-phenyl-3-pyrazolidone), BW 755 C(3-amino-1-[m(trifluoromethyl)-phenyl]-2-pyrazoline), and monophenylbutazone (4-butyl-1-phenyl-3, 5-pyrazolidinedione) at 0.1 mM increased the vase life of cut carnations (*Dianthus caryophyllus* L. 'White Sim') by 48% to 85% over that of control flowers. Tiron (1,2-dihydroxybenzene-3,5-disulfonate) also increased vase life about 38% at concentrations of 0.1 and 0.01 mM. These increases were additive to the beneficial effects of the control preservative solution which contained 2% sucrose, 0.02% 8-hydroxyquinoline citrate, and 0.02 M citric acid. A common feature of these compounds is that they are inhibitors of lipoxygenase.**

It is well-known that ethylene plays a central role in the senescence of many flowers and that inhibitors of ethylene synthesis, such as aminoethoxyvinylglycine (AVG) and aminooxyacetic acid (AOA), delay senescence in certain cut flowers (1, 2, 14). Other compounds, such as free radical scavengers, and EDU (N-[2-(2-oxo-1-imidazolidinyl)ethyl]-N′-phenylurea), an anti-ozonant, also delay senescence in cut carnations, and the effect of EDU is additive to that of AVG or AOA, suggesting that EDU acts on a pathway other than ethylene biosynthesis (14). EDU-enhanced ozone tolerance is correlated with increased levels of superoxide dismutase and catalase in bean leaf extracts (5). EDU also has been found to inhibit soybean lipoxygenase activity in vitro (D.E. Terlizzi, unpublished results). Lipoxygenase catalyzes the oxygenation of unsaturated fatty acids containing *cis, cis*-1,4-pentadiene systems, yielding fatty acid hydroperoxides that can undergo degradation via free radical chain reactions. It is known that this enzyme can catalyze the peroxidation of plant mitochondrial membrane phospholipids in situ, leading to loss of membrane integrity (7). It is possible that lipoxygenase is involved in the initiation or development of senescence in plant tissues. With this in mind, we investigated the effects of several known lipoxygenase inhibitors on senescence in carnations.

Fully opened carnation flowers from Colombia, S.A., were obtained from a local wholesale market. These flowers had not been treated with silver thiosulfate. Flowers were cut to 30 cm and placed in 1-liter jars with 500 ml of basal preservative solution containing 2% sucrose, 0.02% 8-hydroxyqui-

Table 1. Effect of pyrazon, phenidone analogues, and AOA on vase life of carnations at 20°C.

Treatment[z]	Vase life (days)[y]	%
Control (+ acetone)	9.0 a	100
Control (− acetone)	9.3 a	100.3
Pyrazon	16.6 c	184.4
Phenidone	17.0 cd	188.9
BW 755C	13.3 b	147.8
MPB	16.4 c	182.2
AOA	17.1 cd	190.0
AOA + pyrazon	18.1 de	201.1
AOA + phenidone	18.4 e	204.4
AOA + BW 755 C	18.0 de	200.0
AOA + MPB	16.5 c	183.3

[z]All treatments contained 2% sucrose, 200 ppm 8-hydroxyquinoline citrate, and 0.02 M citric acid buffer, pH 4.6. All except − acetone contained 0.5% acetone. AOA was present at 0.5 mM, and the other test chemicals at 0.1 mM.

[y]Mean separation by Duncan's multiple range test, 5% level.

Received 26 June 1984. The authors thank G.A. Higgs, Wellcome Research Laboratories, for providing BW 755 C. The cost of publishing this paper was defrayed in part by the payment of page charges. Under postal regulations, this paper therefore must be hereby marked *advertisement* solely to indicate this fact.

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Table 2. Effect of Tiron on vase life of carnations at 20°C.

Treatment[z]	Vase life (days)[y] Expt. 1	Expt. 2	Expt. 3	$\bar{x}$	%
Control	9.4	10.4	9.8	9.9 a	100
Tiron, 1.0 mM	11	15	12.7	12.9 b	130
Tiron, 0.1 mM	12.5	14.7	13.5	13.6 b	137
Tiron, 0.01 mM	12.5	13.9	14.8	13.7 b	138

[z]All treatments contained 2% sucrose, 200 ppm 8-hydroxyquinoline citrate, and 0.02 M citric acid buffer, pH 4.6.

[y]Mean separation by Duncan's mulitple range test, 5% level.

noline citrate, and 0.02 M citric acid buffer adjusted to pH 4.6 with KOH (control), or in basal solution at pH 4.6 containing one or a combination of the compounds to be tested (pyrazon, phenidone, BW 755 C, monophenylbutazone, AOA or Tiron). The pH of 4.6 was to provide an acidic medium, which is one of the criteria of a good floral preservative. Stock solutions of the 1st 4 compounds were made up in acetone, whereas AOA and Tiron were in basal solution.

Final concentration of acetone in solutions of treated flowers was 0.5%, which did not produce any significant deleterious effects on the flowers. Solutions were made up in deionized water. The control (basal) preservative solution typically increases the vase life of carnations about 80% over that obtained with deionized water alone (14). The flowers were placed randomly in a room maintained at 20°C, 50% to 60% RH, and with constant fluorescent light (300 μmol $s^{-1}m^{-2}$). Five flowers were placed in each jar, and 2 replicates were used for each treatment. Vase life of each flower was evaluated individually and was considered ended when the petals showed wilting or browning and had lost decorative value. At least 3 experiments were conducted with Tiron and the group of compounds comprised of pyrazon and phenidone analogues with similar results. The experiments with Tiron were conducted prior to evaluation of the other compounds, and hence the results are presented separately.

Pyrazon, a substituted pyridazinone which has been shown to inhibit soybean lipoxygenase 1 (9, 11), increased the vase life of carnations up to 84% (Table 1) at a concentration of 0.1 mM in the vase solution. Phenidone, a chemical used in the photographic industry, inhibits both the lipoxygenase and cyclooxygenase pathways of arachidonic acid metabolism in animals (3), and it also inhibits soybean lipoxygenase (9, 11). In our experiments, it consistently was one of the more effective chemicals in delaying senescence of carnations. As shown in Table 1, 0.1 mM phenidone increased vase life about 89% over that of control flowers. Reported Ki values for phenidone and pyrazon inhibition of soybean lipoxygenase 1 are 70 and 160 μM, respectively (11). BW 755 C also has been shown to inhibit both lipoxygenase and cyclooxygenase pathways of arachidonate metabolism in animal systems and associated inflammatory processes (4). It also inhibited soybean lipoxygenase 1 (D.E. Terlizzi, unpublished results). BW 755 C was less effective than phenidone and pyrazon in delaying senescence in carnations, increasing vase life about 48% in the experiment of Table 1. Monophenyl butazone, also an inhibitor of soybean lipoxygenase 1 (10), extended vase life of carnations about 68% (Table 1). Combination of AOA at 0.5 mM with phenidone resulted in a significantly higher value for vase life than either AOA or phenidone alone, but the increase was not strictly additive as in the case of AOA and EDU (14). Vase life values for AOA + pyrazon, BW 755 C, or MPB were not significantly different from those for AOA alone. Phenidone, pyrazon, and BW 755 C at 1.0 mM inhibited ethylene production in tomato slices only slightly during a 20-hr incubation (data not presented). It is therefore doubtful that these compounds delay senescence by inhibiting ethylene production of the flowers. Pistorius and Axelrod (8) showed that Tiron inhibited soybean lipoxygenase after long-term incubation, presumably by chelation and removal of iron from the enzyme. Tiron also inhibits ethylene production in a pea microsomal membrane system (6) but inhibits ethylene production in tomato fruit slices only slightly at 1.0 mM (J.E. Baker, unpublished results). Tiron at 0.01 and 0.1 mM increased vase life of carnations 37% to 38% in our experiments (Table 2). The maximal effect of Tiron was observed at the lowest concentration tested (0.01 mM). None of the chemicals produced any deleterious effects on stems or foliage of the flowers.

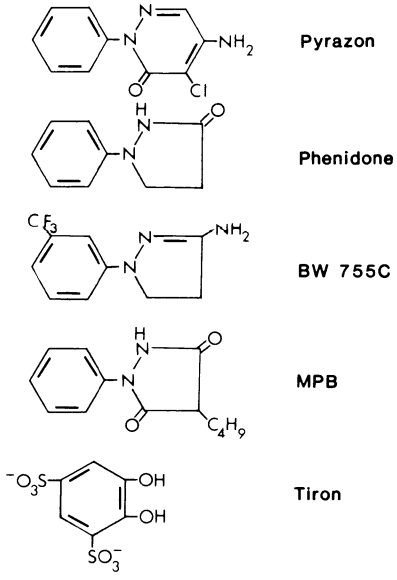

Fig. 1. Structure of chemicals used in this study.

The possibility that lipoxygenase plays a role in the regulation of plant senescence, either by a direct mediation of membrane lipid peroxidation, or via the production of jasmonic acid (13), a sensecence promoting compound, (12) led us to investigate the effects of some lipoxygenase inhibitors (Fig. 1) on senescence of carnations. Although all of the compounds tested delayed senescence of carnations, we have no evidence that they exert their effects by inhibiting lipoxygenase in vivo. Further work is needed to determine, among other things, whether or not membrane fatty acid composition is altered by the compounds.

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