# The Involvement of Marmesin in Celery Resistance to Pathogens During Storage and the Effect of Temperature on Its Concentration

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**ABSTRACT**

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We show evidences that (+)marmesin, rather than linear furanocoumarins (psoralens), may play the major role in celery resistance to pathogens during storage. (+)Marmesin, the precursor of psoralens in celery, has at least 100 times greater antifungal activity in vitro in the dark than psoralens. Increased susceptibility of celery to pathogens during 1 mo of storage was accompanied by a decrease in (+)marmesin concentration and a corresponding increase in psoralen concentration. An increase in celery decay was negatively correlated with (+)marmesin concentration and positively correlated with psoralen concentration. After 1 mo of storage at 0 or 2 C, the concentration of psoralens increased from 10 to 136 or 78 $\mu g\ g^{-1}$ fr. wt., respectively, while the concentration of (+)marmesin under the same storage conditions decreased from 33 to 4 or 11 $\mu g\ g^{-1}$ fr. wt., respectively. Incidence of decay after 1 mo of storage at 0 or 2 C was 62 or 27%, respectively.

*Additional keyword*: phytoalexins.

Psoralens, furanocoumarins produced by celery (*Apium graveolens* L.) have a number of biological effects, the most important of which are photosensitized reactions with nucleic acids. Photosensitized reactions of DNA with furanocoumarins cause lethal, mutagenic, and clastogenic effects in a wide variety of cellular systems (5,6,27,33). Photocarcinogenesis occurs in animals and, probably, also in humans exposed to the combined effects of psoralens and near-UV. Effective wavelengths for these phototoxic reactions are between 320 and 380 nm (16,27,30). PUVA (psoralen-UVA light) photochemotherapy for psoriasis is recognized by the World Health Organization to be causally related to human skin cancer (26).

Phytophotodermatitis, caused by psoralens, has been reported in field workers after contact with celery (7,14,20). Other studies (13,34) have found that phytophotodermatitis occurs in grocery workers. Pathak et al (32) reported that, in addition to celery, various plants, such as parsnips, fig, parsley, and citrus, produce psoralens.

Furanocoumarins are believed to be phytoalexins associated with celery resistance to pathogens (10,12,16). These chemicals were originally thought to be mycotoxins produced by *Sclerotinia sclerotiorum* (Lib). De Bary (4,7). Beier and Oertli (12) demonstrated that the production of furanocoumarins was also initiated by general elicitors including copper sulfate, UV, and low temperatures. Mechanical damage occurring during harvesting and storage has also been shown to increase furanocoumarin concentration from about 2 to 95 $\mu g\ g^{-1}$ fr. wt. (16).

The association of furanocoumarins and resistance causes celery breeders (31) to face a serious dilemma when attempting to develop disease-resistant varieties as an alternative to postharvest applications of pesticides, the use of which is restricted in many countries. Unfortunately, the development of such varieties, which would presumably contain high levels of psoralens, would pose hazards to humans.

(+)Marmesin has been reported to be the precursor of linear furanocoumarins in several species belonging to the families of the Apiaceae, Rutaceae, Moraceae, and Leguminoseae (15,21–24, 29,36). The main objective of this study was to determine whether (+)marmesin, rather than psoralens, plays the major role in celery resistance to pathogens during storage.

## MATERIALS AND METHODS

**Fungal and plant materials.** The fungi *Botrytis cinerea* Pers.:Fr., *Alternaria alternata* (Fr.:Fr.) Keissl. and *Sclerotinia sclerotiorum* (Lib). De Bary were used in the experiments. These fungi, which are major pathogens causing rot diseases to celery, were isolated from naturally infected celery (*Apium graveolens* L.), var. Tender Crisp, from Kibbutz Alumim, the Negev Desert, Israel, in January 1991. The fungi were cultured on potato-dextrose agar (PDA) at 24 C in darkness to serve as inoculum. Celery stalks from the external head side of the variety Early Bell were used for the experiments and for inoculations.

Percentage of celery decay on naturally infected stalks during storage was determined as the number of infected stalks per total stalk number in each replicate. A replicate consisted of 100 stalks packed in polyethylene bags (30 $\mu$m thick) perforated with 20 holes (6 mm diameter) and stored in cold rooms with air circulation. Percentages of celery decay and concentrations of (+)marmesin, psoralen, bergapten (5-methoxypsoralen), xanthotoxin (8-methoxypsoralen), and isopimpinellin (5,8-methoxypsoralen) were measured weekly during 1 mo of storage at 0 or 2 C and 94% relative humidity in darkness.

Inoculation procedures were done as follows: four to five 3-mm-long incisions, 0.2–0.5 mm deep, were cut with a sterile scalpel into each celery stalk (5–30 cm long and 2–3 cm thick) 2–3 h after harvest. A 3-mm-diameter disk, cut from an actively growing PDA culture of *B. cinerea,* was placed over each incision, fungal side downward, and the inoculated stalks (100 in each replicate) were incubated in darkness in a chamber with air circulation held at 98% relative humidity and 24 C. Concentrations of (+)marmesin, psoralen, bergapten, xanthotoxin, and isopimpinellin were measured daily during 10 days after inoculation with *B. cinerea*.

**Extraction, purification, isolation, identification, and quantification of psoralens and (+)marmesin in celery.** Compounds were extraced 4 days after inoculation with *B. cinerea*. Slices of inoculated celery stalks with necrotic lesions cut from the margins of

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the wounds (220 g fr. wt.) were extracted with distilled water (10 ml/g fr. wt. tissue) for 2 h at 40 C. The extract was partitioned with ethyl acetate (EtOAc) and concentrated by evaporating the solvent at 40 C in a Rotovac evaporator. Preparative high-performance liquid chromatography (HPLC) separations were performed with an Applied Biosystems Inc. instrument, equipped with two Model 150 pumps and an 893 programmable detector. Nuclear magnetic resonance (NMR) spectra were recorded on a Bruker WM-360, operating at 360.132 MHz for $^1H$. Mass spectra were recorded on a Finnigan MAT ITD-800 GC-MS instrument equipped with a DB-1 capillary, minibore column (carrier gas: helium, 1 ml/min) and coupled with the PC version of the National Institute of Standards and Technology (NIST) mass-spectral database. This configuration allows comparison of the collected mass spectra with the spectra in the database.

The crude material (340 mg) was loaded onto a vacuum column (Merck Silica H, 5 g, packed into a 2-cm inside diameter, 30-cm tall, sintered ("porous silica") glass funnel; vacuum by a water aspirator) and eluted in petroleum ether with an increasing EtOAc step gradient, to yield 12 fractions (50 ml each). The fractions were assayed for antifungal activity against *B. cinerea*. Fraction 7, eluted from the column with 40% EtOac in petroleum ether, was the most active. Fraction 7 (27 mg) was purified on a preparative HPLC column (Altech Econsil $C_{18}$ reverse phase, 10 $\mu$m, 250 × 22 mm), using 70:30 MeOH/water as the eluant (5 ml/min) with UV detection at 254 nm. The chromatogram yielded 8 major components and the least polar was the active component and was identified as (+)marmesin. Psoralen, bergapten, xanthotoxin, and isopimpinellin were identified by comparison of their HPLC retention time, UV absorption, and $^1H$ NMR data with those of the authentic compounds (9,11,16,37).

The substances were also identified by thin-layer chromatography (TLC) (0.5 mm, Art. 7730, Kieselgel 60 GF254, E. Merck, Darmstadt, Germany). Ascending TLC was developed in a mixture of toluene/EtOAc (1:1, v/v), then dried and inspected under UV (365 nm). Quantifications of (+)marmesin and psoralens in celery during 1 mo of storage at 2 C were obtained by means of analytical HPLC, carried out with an L-6200 pump, an L-4200 UV-VIS detector, and a D-200 Chromat-Integrator (Merk-Hitachi, Tokyo, Japan). A Machery-Nagel (Duren, Germany) Nucleosil silica column (250 × 4 mm) containing $C_{18}$ reverse-phase packing of 5-$\mu$m particle size, with a 15 × 4 mm guard column filled with the same packing material, was used. All standards and samples were dissolved in methanol. For internal standards we used (+)marmesin from our own source, after purification and identification as described above, psoralen, bergapten, and xanthotoxin purchased from Sigma, and isopimpinellin kindly provided by R. C. Beier of the Veterinary Toxicology and Entomology Research Laboratory, USDA-ARS, College Station, TX. These standards were eluted isocritically with methanol/water (45:55, mixed by HPLC pump) at a flow rate of 0.4 ml/min. Peaks were monitored and quantified at 254 nm.

**Toxicity assays.** Toxicity assays were carried out for psoralen, bergapten, xanthotoxin, isopimpinellin, and (+)marmesin. $EC_{50}$ values for *B. cinerea, A. alternata,* and *S. sclerotiorum* were determined by adding increasing concentrations of marmesin and psoralens to cooled molten PDA immediately before pouring into petri plates. Three-millimeter-diameter disks, cut from actively growing PDA cultures of each fungus, were placed on the center of plates, fungal side downward. Plates were then incubated for 5 days at 24 C in darkness. The $EC_{50}$ values for colony area growth in these plates were calculated from the regression lines obtained by plotting the percent inhibition of growth against the log concentration of the compounds.

**Statistical analysis.** All experiments were conducted in a completely randomized design with 5 replicates for each treatment. Data were analyzed by analysis of variance procedures and regression analysis, using the Statistical Analysis System (SAS) package (Cary, NC). Experiments were conducted three times and similar results were obtained each time.

## RESULTS

Fraction 7, the most active against *B. cinerea,* was purified by preparative HPLC and the chromatogram yielded eight components, with relative retention times ($t_R$) of 7.5, 8.5, 9.3, 11.5, 13, 14, 14.8, and 18.1 min. The last component ($t_R$ 18.1 min, 3.1 mg) was found to be the active principle of the mixture and was identified by spectral data as (+)marmesin: GC-MS data ($t_R$ 21.13 min, 70eV) $m/z$ 246 ($M^+$, 100%), 213, 187, 160, 131, 102, 77, and 59 (identified by comparison with NIST database); $^1H$ NMR data ($CDCl_3$:$CD_3OD$ 1:20) and (multiplicity, J in Hz, assignment): 6.22 (d, J = 9.3 $H_z$, H-3), 7.60 (d, J = 9.3 $H_z$, H-4), 7.22 (s, H-5), 6.75 (s, H-8), 3.20 (dd, J = 8.6 and 15.7 $H_z$, H-9), 3.22 (dd, J = 9.1 and 15.7 $H_z$, H-9), 4.74 (dd, J = 8.6 and 9.1 $H_z$, H-10), 1.24 (s, $H_3$-12 and 1.37 (s, $H_3$-12). These values are identical with published data for marmesin (35). (+)Marmesin and psoralens were also identified by TLC. Chromatograms developed with a mixture of toluene/EtOAc (1:1, v/v), were examined under UV illumination, and gave fluorescent spots at $R_f$ = 3 for (+)marmesin and $R_f$ = 7 for psoralens. Quantification of (+)marmesin and psoralens was done by analytical HPLC; $t_R$ were as follows: (+)marmesin, 6.15; psoralen, 6.40; bergapten, 6.85; xanthotoxin, 6.66; isopimpinellin, 7.09.

(+)Marmesin, the precursor of psoralens, has at least 100 times greater antifungal activity in vitro in the dark than psoralens (Table 1). Increasing decay of celery in storage was accompanied by a decrease in (+)marmesin concentration and a corresponding increase in psoralen concentration (Fig. 1). An increase in celery decay was found to be negatively correlated with (+)marmesin concentration ($r^2 = 0.927$; $y = 33.2 - 0.28x$) and positively correlated with psoralen concentration ($r^2 = 0.956$; $y = 8.3 + 0.47x$). After 1 mo of storage at 0 C (this temperature causes chilling injury in celery) or 2 C (a normal temperature for celery storage) the concentration of psoralens increased from 10 to 136 or 78 $\mu g^{-1}$ fr. wt., respectively. However, the concentration of (+)marmesin decreased, under these storage conditions, from 33 to 4 or 11 $\mu g\ g^{-1}$ fr. wt., respectively. Incidence of decay after 1 mo of storage at 0 or 2 C was 62 or 27%, respectively (Fig. 1).

When celery was artificially inoculated with *B. cinerea* the concentration of (+)marmesin increased during the first 4 days and then started to decline. The concentration of psoralens did not start to increase until 5 days after the inoculation (Fig. 2).

## DISCUSSION

We hypothesize that (+)marmesin, rather than psoralens, may play a major role in celery resistance to pathogens during storage. Our finding, which contrasts with other studies that have reported psoralens to be the principal compounds (phytoalexins) involved in the defense mechanisms of celery against pathogens (10,12,16), suggests a new mechanism. (+)Marmesin, the precursor of psoralens,

TABLE 1. Effective concentrations of psoralens and their precursor, (+)marmesin in darkness, for obtaining 50% growth inhibition ($EC_{50}$) of three pathogenic fungi of celery

| | Psoralens | | | | |
|---|---|---|---|---|---|
| | | $EC_{50}$ values ($\mu g\ ml^{-1}$) | | | |
| Pathogens | Psoralen | Bergapten | Xanthotoxin | Isopimpinellin | Marmesin |
| *Botrytis cinerea* | 4,150 | 4,740 | 4,550 | 5,270 | 28 |
| *Alternaria alternata* | 5,040 | 5,890 | 5,540 | 6,330 | 43 |
| *Sclerotinia sclerotiorum* | 3,620 | 3,970 | 3,310 | 4,050 | 19 |

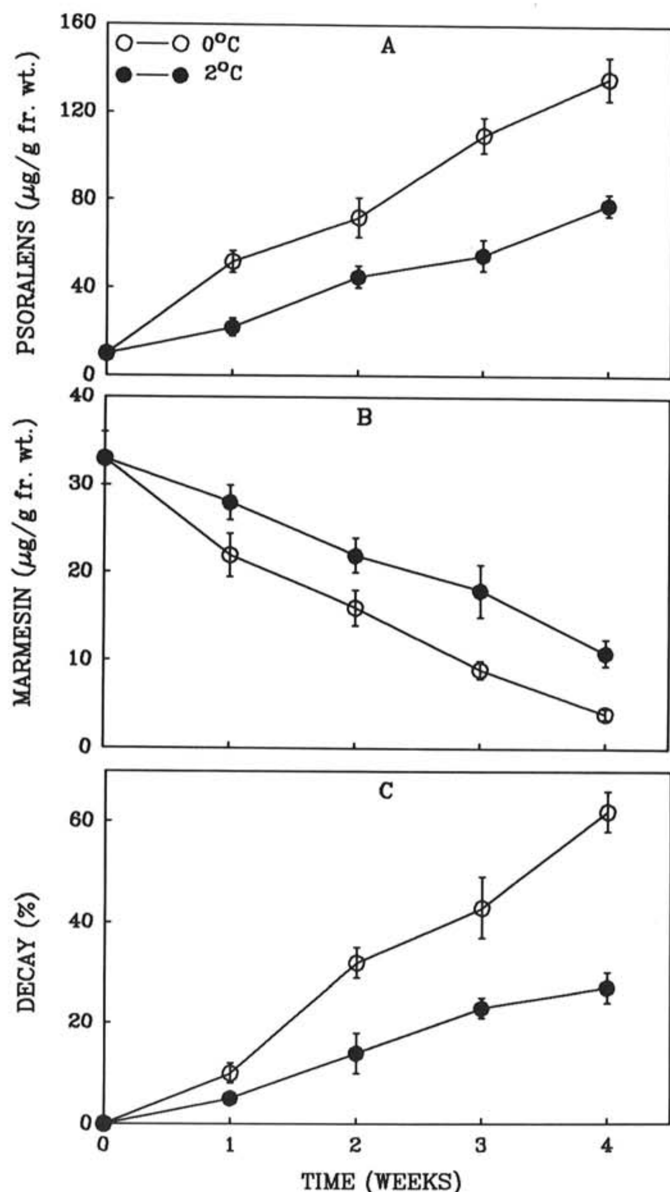


**Fig. 1.** Concentration of **A,** total psoralens and **B,** (+)marmesin, and **C,** incidence of decay in celery during 4 wk of storage at 0 and 2 C. Vertical bars indicate standard error.

which has at least 100 times greater antifungal activity in vitro in the dark than psoralens, was isolated and identified from celery. $EC_{50}$ values for psoralen toxicity to *B. cinerea, A. alternata,* and *S. sclerotiorum* growth in vitro were found to be more than 3,000 μg/ml (Table 1). The concentration of psoralens before storage in vivo is 10 μg/g fr. wt. (Fig 1A). This is only 0.3% of the concentration required for growth inhibition of celery pathogens, indicating that psoralens cannot play any role in the defense mechanism of celery against these pathogens during storage. However, the $EC_{50}$ value of (+)marmesin for toxicity to these fungi in vitro was found to be less than 43 μg/ml (Table 1). This concentration is close to the normal amount of (+)marmesin in vivo (Fig. 1B), and could play a major role in the defense mechanism of stored celery against pathogens. In support of our findings, two other research groups reported that psoralens may not play the major role in disease and insect resistance of celery (18,19,25). (+)Marmesin may be considered as a phytoalexin, since its concentration increased during the first 4 days after inoculation of celery with *B. cinerea,* and then declined (Fig. 2). Such a pattern of accumulation and degradation is typical of phytoalexins in plants (8).

Chaudhary et al (17) reported on increased psoralen content of celery during storage. Similarly, results of the present study

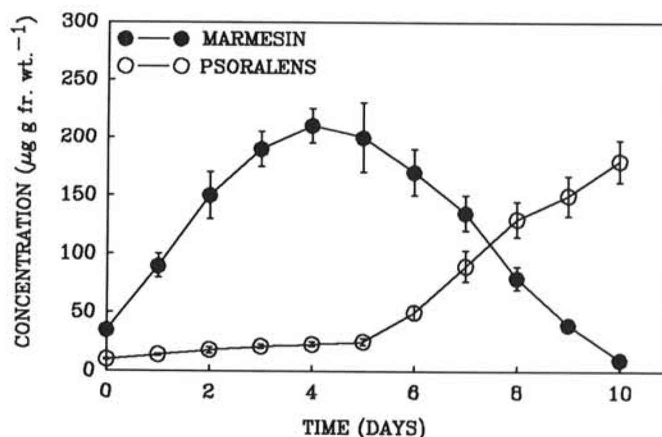


**Fig. 2.** Accumulation of (+)marmesin and total psoralens in celery stalks after inoculation with *Botrytis cinerea* and incubation at 24 C. Vertical bars indicate standard error.

show that the concentration of psoralens in celery stalks increased during storage. In addition, increasing decay of stored celery was accompanied by a decrease in (+)marmesin concentration and a corresponding increase in total psoralen concentration (Fig. 1). An increase in celery decay was found to be negatively correlated with (+)marmesin concentration and positively correlated with psoralen concentration. Based on these results, we suggest that (+)marmesin, and not psoralens, is the major compound involved in celery resistance to pathogens during storage.

(+)Marmesin has been recognized as the precursor of linear furanocoumarins in several species belonging to the families of the Apiaceae, Rutaceae, Moraceae, and Leguminoseae (15,21–23, 36). The conversion of (+)marmesin to the respective linear furanocoumarins involves the oxidative cleavage of the isopropyl alcohol side chain and the introduction of a double bond to the five-member ring moiety (15,24,29). We consider, therefore, that following this reaction, the resulting products (psoralens), derived from (+)marmesin, lose about 99% of their antifungal activity. Results of the present study indicate that celery stored at 2 C is more resistant to pathogens than that stored at 0 C. Similarly, several studies have reported on the effect of temperature on plant resistance (1–3,28). We hypothesize that the higher temperature retarded celery decay during storage by maintenance of a high level of (+)marmesin and low levels of psoralens, presumably thereby increasing resistance to pathogens. This may happen through the slowing down of the conversion of (+)marmesin to psoralens in the celery biosynthesis pathway. Storage at 0 C, however, which causes chilling injury in celery, may increase the conversion of (+)marmesin to psoralens.

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**NOTE FROM THE PUBLISHER:** An article by U. Afek, N. Aharoni, and S. Carmeli that appeared in the June issue (85:711-714) "The Involvement of Marmesin in Celery Resistance to Pathogens During Storage and the Effect of Temperature on Its Concentration" contains text that is out of order. The corrected article is reprinted in its entirety.