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Senescence Inhibition and Respiration

Abstract. Freshly harvested asparagus spears treated with N^6-benzyladenine and then held in the dark at 21°C for 136 hours showed a lower respiration rate, as measured by CO_2 evolution, than nontreated spears. Associated with the lower respiration rate were proportional decreases in postharvest spear elongation and weight loss through desiccation.

Since the discovery of the biological activity of kinetin (6-furfurylaminopurine) in cell division (*1*), many studies have been made in which this and similar compounds have been evaluated in cell division of tobacco tissue, germination of lettuce seeds, and cell enlargement in leaf disks of radish (*2*). In addition, some rather striking effects on protein metabolism, chlorophyll breakdown, and the capacity of excised leaves to withstand stress have been reported (*3*). N^6-benzyladenine, a compound in which the furan ring in kinetin has been replaced by a benzene ring, has shown some effect in preventing postharvest breakdown of head lettuce when applied shortly before harvest or as a postharvest dip (*4*). This compound reportedly has a similar effect on other green vegetables, including asparagus (*5*).

Freshly harvested asparagus spears were obtained from a commercial processor and held for 24 hours at 5°C; the apical ends were trimmed to 5-inch length and then dipped in a $5 \times 10^{-5} M$ solution of N^6-benzyladenine or water. Eight 500-g samples of the treated spears and like samples of nontreated spears were placed in respirometers (*6*) and held in the dark at 21°C. The CO_2 evolution was measured every 8 hours for 136 hours, whereupon the spears were removed and their weight loss and length were determined.

The observed mean values and the fitted regression lines describing CO_2 evolution in treated and nontreated asparagus spears (Fig. 1) indicate that treatment with N^6-benzyladenine materially decreased respiration. Theoretical values of total CO_2 evolution during the 136-hour interval, obtained by integration of the regression equations, were 303 mg/kg for the nontreated and 257 mg/kg for the treated spears. These calculated values correspond very closely to the observed values of 295 and 253 mg/kg. The area between the curves constitutes the total respiration inhibition. This calculation yielded a value of 48 mg of CO_2 per kilogram, indicating about a 16-percent reduction in respiration through the first 120 hours in the N^6-benzyladenine-treated samples. Observed values for these differences were 47 mg of CO_2 per kilogram and 16 percent, respectively. After 120 hours, the nontreated samples evolved CO_2 at a lesser rate than did the treated, as a probable consequence of an initially more rapid depletion of the metabolites in the nontreated samples.

Weight losses in 136 hours were 7.25 and 5.95 percent in the nontreated and treated samples, respectively. Though the differences in weight loss were largely a function of the degree of desiccation, it is noteworthy that the decrease in water loss of approximately 18 percent in the treated samples was comparable to the decrease in respiration. Furthermore, nontreated spears elongated an average of 8.2 percent, and treated spears, of 7.0 percent. This difference (15 percent less elongation in the N^6-benzyladenine-treated spears) was again directly proportional to the amount of respiration inhibition.

These data suggest that respiration as measured by CO_2 evolution, desiccation as measured by weight loss, and growth as measured by elongation are all proportionally inhibited in harvested asparagus spears following postharvest application of N^6-benzyladenine.

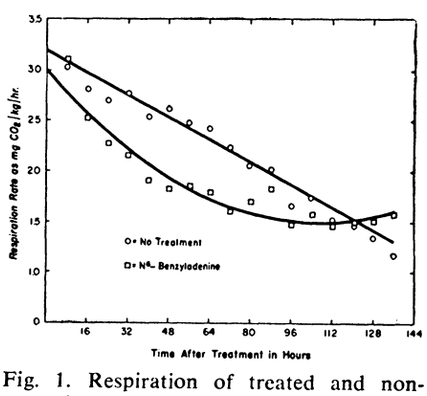

Fig. 1. Respiration of treated and nontreated asparagus held in the dark at 21°C.

It is further suggested that many of the phenomena observed by others, such as chlorophyll retention, changes in nitrogen metabolism, and general inhibition of senescence in green plant tissues subsequent to treatment with kinetin and related compounds, may be a consequence of respiration inhibition of the type described in this report (*7*).

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3 July 1961

Explanation of Cocaine Desensitization of Blood Pressure Responses to Ephedrine

Abstract. It is suggested that cocaine desensitizes ephedrine blood pressure responses by depletion of norepinephrine depots. This was shown by reversing cocaine desensitization through the administration of agents such as bretylium, which has a sparing effect on norepinephrine, and by the infusion of norepinephrine itself.

A number of workers (*1*), since the original discovery by Tainter (*2*), have shown that cocaine in large doses diminishes or abolishes the pressor responses to ephedrine. The exact mechanism of this antagonism has not been explained. We therefore attempted in the present study (*3*) to elucidate this phenomenon according to the theory of one of us (T.K.). This theory postulates that large doses of cocaine exhaust the sympathetic neurohumor depots and, owing to this mobilization, may change physicochemical parameters of cellular membranes (*4*). Therefore, three alternatives were considered: ephedrine should be protected from cocaine desensitization by the administration of (i) substances which prevent such neurohumoral release, or (ii) substances which protect the neurohumor from metabolic destruction, or (iii) the neurohumor itself.

Male mongrel dogs, weighing from 6 to 10 kg, were anesthetized intraven-