

## INTERACTION OF COMPOUNDS WITH THE ETHYLENE RECEPTOR: HOW AND WHY THEY ACT?

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### ABSTRACT

In 1901 Neljubov reported that ethylene causes responses in plants. Since that time it has been shown that many other compounds including propylene, carbon monoxide, isocyanides, acetylene and a number of other olefins also induce these responses. Burg and Burg (1967) showed that compounds giving ethylene like responses (agonists) in plants are in the same order as they bind to silver ion (Muhs and Weiss, 1962). Burg and Burg (1967) tested the compounds ethylene, propylene, and 1-butene for biological activity and the required concentrations for a ½ maximum biological response were 0.1 for ethylene, 130 for propylene and 27, 000 for 1-butene. Based on this it was thought that the site was restricted and only small molecules could bind to the site.

In 1973, Sisler and Pian reported that cyclic olefins interacted with plants and counteracted ethylene responses (antagonists). Sisler and Yang (1984) showed that the inhibitory compounds were active in the same order as they bind to silver ion. Chemists have attributed binding of cyclic compounds to silver to ring strain (Traynham and Sehnert, 1956). The more strained they are, the stronger they bind to silver ion. The fact that both ethylene agonists and ethylene antagonists both increase in activity in the same order that they bind to silver is a paradox that is not completely understood, but the use of strain energies has proven very useful in selecting ethylene antagonists in plants.

The strain values of many compounds are known (Greenberg and Liebman, 1978; Wiberg, 1987; however, a strain value alone is not very informative about the biological activity of compounds. Cyclopropene has a reported strain value of 55.2 kcal mol<sup>-1</sup> and is an excellent ethylene receptor blocker and blocks the receptor for several days by a single exposure. Cyclopropane has a strain value of 27.5 kcal mol<sup>-1</sup> but does not induce an ethylene response, nor does it block ethylene responses because it does not have a double bond. Methylenecyclopropane has a high strain value of 40.9 kcal mol<sup>-1</sup> and it has a double bond, but it is an active ethylene agonist probably because the double bond is outside the ring. *trans*-Cyclooctene has a strain value of 16.7 kcal mol<sup>-1</sup>, and 2,5-norbornadiene has reported values of 34.7 and 31.6 kcal mol<sup>-1</sup>, but it is distributed over 2 double bonds. *trans*-Cyclooctene has a far higher number for binding to silver, and far greater biological activity than 2,5-norbornadiene. Cyclobutene has a strain value of 28.4 kcal mol<sup>-1</sup> and blocks ethylene responses, but like *trans*-Cyclooctene and 2,5-norbornadiene requires continuous exposure to inhibit ethylene responses. The strain energy is apparently too small to remain bound for long periods of time. The amount of strain can also be modified by substituents on ring compounds. Electron donating groups will weaken the strain and electron-withdrawing groups should increase the strain thus altering binding and the time of protection (Sisler *et al.*, 2001).

Since molecular size was thought to be an important factor in limiting the activity of ethylene agonists, it seemed logical that the same would be true for ethylene antagonists. To test this idea a series of cyclopropenes substituted in the 1 position were prepared and tested for activity. This series consisted of cyclopropenes with an alkane in the 1-position from 0-10 carbons long. Rather than seeing the large decline in activity seen with ethylene agonists, in

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general the activity increased and the time of inactivation of banana insensitivity to ethylene increased from about 12 days for cyclopropene to 40 days for 1-decylcyclopropene. The site appeared not to be restricted. This created another paradox. How could the site be restricted for ethylene agonists but not for ethylene antagonists?

To solve this paradox, it was decided to repeat and expand the work of Burg and Burg (1967). A series of alkenes starting with ethylene and extending to 1-decene were tested. Two tests were used. One of these was with etiolated pea plants and one with banana fruit ripening. For ethylene, propylene and 1-butene the results with pea plants were the same as obtained by Burg and Burg (1967). 1-pentene was inactive as an ethylene agonist as were 1-hexene, 1-octene, 1-decene and 1-dodecene. These compounds were also tested as antagonists. 1-butene was an ethylene antagonist as were the other longer chain alkenes. The longer the chain, the more inhibitory they were. These results show that 1-butene is a pivotal compound being both an agonist and an antagonist of ethylene responses. The results with bananas were essentially the same, as with pea plants except 1-butene did not appear to be an active agonist at the concentrations tested.

This series shows that rather than the ethylene-binding site being a restricted site the alkenes are getting to the ethylene binding site but change from being agonists to being antagonists. All of these alkenes have the same functional group and differ only by the length of the hydrophobic chain. They would be more hydrophobic as the chain length increases. Alkanes have no effect.

This reveals that there are at least two forces involved in the binding of antagonists. There is an effect of the double bond and there is an effect of the hydrophobic tail. The compound is more active when the hydrophobic part is present. Binding to the putative metal in the receptor need not be different for an agonist and an antagonist. Exactly what is different is not known. It could be the length of time the compound remains bound, or it could possibly restrict movement. As other data suggests (Sisler *et al.* 2003; Grichko *et al.*, 2003) the ethylene binding site is not very restricted.

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