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Fusaric Acid: A Potent Vivotoxin

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Abstract: Vivotoxin designates a secondary metabolite produced by the pathogen and/or its host during infection, produces disease symptoms, but is not oneself the initiating causal agent of the infection. Out of all, Fusaric-acid is the most studied pathogen produced wilt toxin classified as a non-specific vivotoxin. It does not produce all the symptoms of wilt. Many scientists all over the world including India were of the belief that, out of all other toxins involved in the infection process of wilting, fusaric acid was the most potent one. Infected tissue shows a marked increase in respiration process which is contrary to the host tissues doped with fusaric acid because it is a best-known respiratory depressant.

Key words: Fusaric-acid, Fusarium oxysporum, Potent, Vivotoxin, Wilting

1. INTRODUCTION

Dimond and Waggoner, 1953 gave the term "vivotoxin" to denominate "a substance produced in the infected host by the pathogen and/or its host, which functions in the production of disease, but is not itself the initial inciting agent of disease." They also notified that "a vivotoxin is a disease-producing entity and therefore a pathogenic agent." They enlisted 3 major criteria as the nominal need to set up vivotoxicity. These were -
(a) reproducible segregation from the infected host plant,
(b) purification, and
(c) re-production of at least a fraction of the disease symptoms by allocating the toxin in a same healthy plant.

Dimond, 1955 subsequently altered their criteria by defining that vivotoxin "not be present in the healthy host" because it produces during the host-pathogen interaction. Dimond as well explained that, instead of refinement, the toxin be defined chemically. Dimond & Waggoner seemed to be cognizant of this in consideration that vivotoxicity could be incontestable by observing only the 1st and 3rd of their criteria. Paradoxically, they said that "just as it is usually necessary to know the identity of a parasite to establish it as a cause of disease, so it is also necessary to purify and identify a vivotoxin to prove its complicity." We disagree with the postulate expressed in the first section or with the assumption attained in the last. What is required in both the cases is indicated in the conclusion that the parasite or the toxin perform a significant causative function during the occurrence of biotic infection. Here we want simply signalize that criteria which stipulate the requisite evidence are more adequate than those which prescript the processes by which the information is to be received. First step of Dimond & Waggoner's, which shows segregation of the toxin from the infected host plant, is based on the aforementioned criticism. Separation of most potent, coseismal toxin, existing in little bulk, seems impracticable, but tolerable illustration is that it utilizes in the creation of infection symptoms may be acquired by some different ways (Braun and Pringle, 1959). Despite the fact that these considerations betoken that the criteria of Dimond & Waggoner are little idealistic because they don’t explain straight on the query of their

cogency. Many scientists all over the world including India were observed that, even the presence of many other toxins during the infection process of wilting, fusaric acid was the highest powerful one and put forward the pursuing grounds to explicate why fusaric-acid didn't produce symptoms during the initiation of infection process:

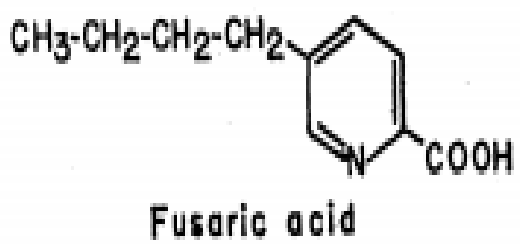
(a) fusaric acid alienates at above 6.0 pH and the pH of infected host plants sap during initial execute of infection process is 6.2;

(b) fusaric acid is produced in very little amounts during infection initiation (Subba-Rao, 1960).

Fusaric-acid

This secondary metabolite was first reported in 1934 from *Fusarium heterosporum*, but its toxic nature was recognized about two decades later by Gauman et al. in the year 1952, who also reported its occurrence from *Fusarium oxysporum* f.sp. *lycopersici*, *Fusarium oxysporum* f. sp. *vasinfectum* and *Gibberella fujikuroi*. Since then this phytotoxic metabolite has been detected in various *Fusarium* formae specialis of the elegance group which included *Fusarium oxysporum* f.sp. *lycopersici, batatis, conglutinans, cubense, lini, vasinfectum, udum* and *Fusarium moniliforme* (Gaumann, 1957; Kalyanasundaram, 1958; Heitefuss *et al.* 1960a, 1960b; Trione, 1960a, 1960b; Prasad and Chaudhary, 1974).

It is the most studied pathogen produced wilt toxin classified as a non-specific vivotoxin. It does not produce all the symptoms of wilt. Fusaric acid is a pyridine-carboxylic acid having empirical formula $C_{10}H_{13}O_2N$ and chemically this toxin is 5-n-butyl-picolinic acid.



Fusaric acid

Biosynthesis of fusaric acid is accomplished even in synthetic media, which shows that no additional nutrition is required for its production. However, it has been noticed that its production is conditioned by the amount of zinc present in the culture medium. Kalyanasudaram and Saraswathi Devi (1955) noted that secretion of fusaric acid by *Fusarium oxysporum* f.sp. *vasinfectum* required 0.08 - 0.4 ppm of zinc (Zn), the optimum concentration being 0.24ppm. Prasad and Chaudhary (1974) also reported stimulatory influence of zince on fusaric acid production by *Fusarium oxysporum* f.sp. *udum*.

It is now well recognised that fusaric acid is produced during the rapid growth phase, and is not a product of autolysis (Sandhu, 1960). Its synthesis seems to be linked with the intermediates of Krebs cycle and is a primary metabolite. Although growing hyphae secrete fusaric acid, most of it is liberated after mycelial autolysis starts (Bohni, 2016). This metabolite has also been detected in the mycelial extracts of different strains of *Fusarium oxysporum* (Prasad and Chaudhary, 1974), which indicates that the entire quantity synthesized by the fungus is not secreted out, rather some of it is retained in the hyphae (Kumar, P. (2019); Kumar, D., Rameshwar, S. D., & Kumar, P. (2019); Dey, S. R., & Kumar, P. (2019); Kumar et al. (2019); Dey, S. R., & Kumar, P. (2019); Kumar, P., & Pathak, S. (2018); Kumar, P., & Dwivedi, P. (2018); Kumar, P., & Pathak, S. (2018); Kumar et al.,2018; Kumar, P., & Hemantaranjan, A. (2017); Dwivedi, P., & Prasann, K. (2016). Kumar, P. (2014); Kumar, P. (2013); Kumar et al. (2013); Prasann, K. (2012); Kumar et al. (2011); Kumar et al. (2014).

Synthesis of fusaric acid *in vivo* has also been studied and some isotopic data were obtained to demonstrate its production in the tissue of the host-plant (Kern and Sanwal, 1954; Kern and Kluepfel, 1956). Direct detection of this metabolite in the tissue-extract of diseased host plants has also been attempted and met with appreciable success (Lakshminarayanan and Subramanian, 1955; Kalyanasundaram and Venkata Ram, 1956).

Production of the toxin by some species in the rhizosphere soil of tomato plant is also reported (Kalyanasundaram, 1958). In contrast, there are certain reports (Heitefuss et al. 1960a), according to which no fusaric acid is produced in the host tissues, although the same pathogen produces fusaric acid in culture solution. They further concluded that this toxin had apparently no role in pathogenicity. Kuo and Scheffer (1964) have also doubted the role of fusaric acid in disease development, and full details are not clearly understood (ChitraMani & Kumar, P. (2020); Sharma, M., & Kumar, P. (2020); Chand, J., & Kumar, P. (2020); Naik, M., & Kumar, P. (2020); Kumar, P., & Naik, M. (2020); Kumar, P., & Dwivedi, P. (2020); Devi, P., & Kumar, P. (2020); Kumari, P., & Kumar, P. (2020); Kaur, S., & Kumar, P. (2020); Devi, P., & Kumar, P. (2020); Sharma, K., & Kumar, P. (2020); Kumar, S. B. P. (2020); Devi, P., & Kumar, P. (2020); Chand, J., & Kumar, P. (2020).

The toxin is active at 20-200mg/kg fresh weight. Sometimes, another toxin, dehydrofusaric acid is associated with fusaric acid which is easily converted into the latter. It mainly causes interveinal chlorosis. The role of fusaric acid in the plants is said to be of many types. It causes chelation of iron and copper in the host cells and alters the cell wall permeability. This disturbs the ionic balance of the cell. It also affects the enzymatic processes in the cell. By chelating the enzymes or by rendering respiratory enzymes ineffective, it alters respiratory pattern of the plant. However, whether fusaric acid is responsible for causing all the symptoms in the diseased host plant infected with wilt-fusaria is yet to be established because wilt syndrome in plants is produced by a combination of several toxins and metabolites.

This secondary metabolite is also toxic to bacteria, algae, fungi and angiospermic plants. Some of its notable effects are tabulated below:-

Table 1: Showing toxic effects of Fusaric-acid on various microbes and plants

Organisms	Effect	Concentration
Bacteria	Growth inhibited	10^{-4} to 10^{-3} M
Green Algae *Spirogyra nitida*	Permeability affected	5 x 10^{-3} M
Ustilago maydis	Germination of basidiospores effected	1.5 x 10^{-4} M
Rye, maize and pea plants	Injury caused	1.000 to 2000 mg/Kg fresh weight
Tomato plants	Injury caused	150 mg/Kg fresh weight
Cotton plants	Injury caused	10 to 20 mg/Kg fresh weight

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