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RESEARCH ARTICLE

DIVERSITY AND PATHOGENICITY OF FUSARIUM SPECIES CAUSING POSTHARVEST ROT IN CHERRIES AND THEIR CONTROL METHODS

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Abstract

The postharvest spoilage of sweet cherry fruit (Prunus avium) from three selected geographical locations, namely Henan, Shandong and Hebei, was investigated. The associated fungi was isolated from cherry fruits on Potatoe dextrose agar (PDA). A total of four (4) fungi were isolated,namely Fusarium fujikuroi,Fusarium lateritium,Fusariumgrami nearum, and Fusarium proliferatum. Fusarium proliferatum was the most prevalent of the four fungi isolated and appeared in all four locations. Cherry fruit spoilage was most severe in humid environments and was enhanced by wounds on fruit surfaces. The pathogenicity test revealed that all four isolates proved pathogenic when artificially inoculated into healthy cherry fruits. These organisms are, therefore, the causal agents of cherry fruit rot under the conditions of this study. C herry suffers from several diseases at all stages of its life. Cherry rot, caused by the fungus Fusarium species is the most important postharve st disease of cherry.An experiment was carried out to test the possibilit y of using some mefentrifluconazole and y19315 fungicide to reduce postharvest losses induced by Fusarium species in cherry fruits. In this study,the antifungal activity of fungicides under in vitro conditions was assayed by testing various concentrations(0,0.6, 0.3, 0.15, 0.075 µg/ml) for y19315 and (1, 0.5, 0.25, 0.125 µg/ml) for mefentrifluconazole and under in vivo condition by using different essential oil concentrations (0, 10,50 and 100 µg/ml) for both fungicides on inoculated cherry fruits.

Results of the in vitro showed that all fungicides at all tested concentrations were significantly reducing the fungal linear growth. In vitro, results showed significant antifungal activity of all tested fungicides on all fungal species. y19315 were the most effective with EC50 of 0.033 µl/ml, whereby inhibition percentages ranged from 60.2% to 87.7% against YTMZ 52-1 followed by mefentrifluconazole which had an EC50 of 0.354 µl/ml, whereby inhibition percentages ranged from 35% to 58.6% against H24-1 for the concentrations 0.6 to 0.075 µl/ml and 1 to 0.125 µl/ml respectively. The higher concentrations tested of fungicides significantly reduced lesion diameter. In vivo, studies showed that two fungicides, y19315 and Mefentrifluconazole, tested against different Fusarium isolates caused fruit rot in cherry fruits. In the case of y19315, at concentrations of 10, 50, and 100 µl/ml, it exhibited varying levels of efficacy against different isolates. For F. Graminearum (H19-1), the efficacy was 0.1%, 49.71%, and 100%, respectively, while for F. Proliferatum (HZZ 291), it was 95.82%, 100%, and 100%, respectively. F. Fujikuroi(B34-

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1) showed the efficacy of 50%, 89%, and 100%, and F. Lateritium (YTMZ 60-3) had efficacy of 91%, 100%, and 100%. Notably, at 50 µl/ml, all fungicides showed a significantly increased efficacy. At the highest 100 µl/ml concentration, both fungicides completely suppressed fruit rot for all isolates. In the case of Mefentrifluconazole, similar trends were observed, with varying efficacy levels against different isolates. At 100 µl/ml, it inhibited fruit rot, except for isolates H19-1 and B34-1. These findings suggest the potential of y19315 and Mefentrifluconazole as effective fungicides for controlling fruit rot caused by Fusarium isolates in cherry fruits, especially at higher concentrations.. The study suggests that using fungicides can positively affect disease control and the quality of cherry fruit, with higher concentrations being more effective. The findings can contribute to developing regulatory policies and disease control strategies for cherries and related fruits.

Introduction:-

This study focuses on cherries (Prunus spp.), edible fruits rich in vitamins, antioxidants, carbohydrates, and minerals, widely cultivated in countries like Turkey, China, the United States, and Iran. Cherries have significant health benefits, including weight loss, cancer prevention, and management of various ailments [1-3]. Despite their economic and ornamental value, cherry production faces challenges, notably due to postharvest diseases caused by fungi such as Botrytis cinerea and Monilinialaxa, which limit shelf life and marketability [4-7]. The chemical composition of sweet cherries (Prunus avium) greatly influences their sensory quality and consumer acceptance, with total soluble solids (TSS), titratable acidity (TA), and maturation index (TSS/TA ratio) being key indicators [8-31]. Sweet cherries contain high water content (~80-83%), moderate carbohydrates (12.2-17.0g/100g), proteins (0.8-1.4g/100g), low fats, and are good sources of potassium, calcium, vitamins C, E, and K [12-38]. Varieties such as Bing, Rainier, Lambert, and Royal Ann differ in flavor and appearance and are mainly harvested in late spring or early summer [26-30]. Postharvest diseases, predominantly caused by fungi, are major threats to fruit crops including cherries, apples, bananas, and papayas. These diseases are influenced by physical, physiological, mechanical, and environmental factors such as temperature and humidity [39-59].

Recent advances emphasize biological control and natural treatments, including edible coatings, as safer alternatives to chemical fungicides [40-62]. For example, coatings like sodium alginate help maintain cherry quality and antioxidant properties during storage [62,76]. Preharvest factors affecting cherry quality include cultivar, temperature, light intensity, ripening stage, and treatments with substances like salicylic acid, abscisic acid, and oxalic acid, which enhance phenolic content, anthocyanins, and antioxidant activity [63-73]. Postharvest handling involving rapid precooling, cold storage (optimal at 0°C, 90-95% humidity), and controlled atmosphere can prolong shelf life while maintaining quality [60,61,74-76].

Cherries also have medicinal and traditional uses, including anti-inflammatory effects and applications in folk medicine [78-82]. Globally, sweet cherry production is about 2.2 million tons annually, with Turkey as the leading producer followed by the United States, Iran, and others. Modern orchard systems and cultivars aim to optimize yield and fruit quality [83,84]. Fruit rotcaused by multiple fungal species including Fusarium spp., poses significant postharvest challenges, especially in China where such diseases are under-researched [85-87]. This study aims to isolate and identify Fusarium species causing cherry fruit rot using molecular techniques (tef and ITS sequencing), assess genetic diversity, analyze phylogenetic relationships, and evaluate pathogenicity. The findings are expected to inform biosecurity policies and disease control strategies to mitigate losses in cherry production.

ISOLATION AND IDENTIFICATION OF FUSARIUM SPECIES:

Cherries are essential and valuable fruits given by nature to us. So, it is necessary to protect it against bacterial and fungal pathogens during postharvest. Hence, the present work aims to understand the characteristics of the fungal pathogen, its infection and disease-causing ability, and the control of pathogens causing postharvest diseases using chemical and biological approaches.

Fusarium fungi infect cherry fruit through wounds caused by mechanical damage and environmental factors. Later in the infection process, white and cotton-like mycelia conidia are produced on infected cherry fruits, considered the typical postharvest disease symptom of cherry fruits. Cherries are susceptible to fungal attacks; recent research has focused on safer methods of preventing fungal spoilage. Fruit rot caused by Fusarium species is the most common postharvest disease in the cherry industry. Postharvest fruit rot, the primary cause of fruit decay, causes enormous economic losses worldwide every year and can account for up to a large proportion of total cherry postharvest losses. Cherries are susceptible to fungal attacks; recent research has focused on safer methods of preventing fungal spoilage.

Figure 2.1. Typical symptoms of postharvest decay of cherry fruits

Materials and Methods:-

Materials:-

Chemical Reagents

Agar powder, Potatoes and Glucose, ethanol, isopropanol, trichloromethane, MixTaq.

Methods:-

Sampling and fungal isolation

Fruit rot-affected cherry samples were collected from various sites across China (figure 2.1). The fruits were washed under tap water and surface sterilized in 70% ethanol for 10 seconds, then fixed in 5% sodium hypochlorite for 90 seconds, followed by three rinses with sterile distilled water and then blotted dry with sterilized filter paper [88]. The laboratory was disinfected, and the equipment was properly sterilized. The safety inoculation was correctly disinfected using a UV beam for 15 minutes and sprayed with Ethanol to avoid cross-contamination. The chamber was filled with filtered air, and the Bunsen burner flame was kept lit. With a sterile scalpel knife, tissue segments, including symptomatic and asymptomatic areas, were sliced and placed onto potato dextrose agar mixed with penicillium to prevent contamination during incubation. The plates were incubated at 26°C, and each isolate was purified by single spore isolation after being sub-cultured [89].

Morphological identification:

In this study, 12 isolates were used; these isolates were isolated from diseased cherries with postharvest rot symptoms and identified as Fusarium species using morphological and molecular biological methods. The isolates were identified morphologically using [90]. For further examination, the organisms were placed in Potato dextrose agar petri dishes. The fungus was grown on a potato dextrose agar (PDA) medium to investigate its morphological properties. The plates were kept at 25° C in an incubator. Fungi morphological features such as form, edge, surface, color, pigment, and diameter were studied beginning on day 7. After the fungi on the plates had matured, the fungal strains were examined under a microscope. Temporary slides of diseased tissues were made and observed under a light microscope. Fungi were identified after reference to [91], [92], [93], [94], [95], [96], [97], and [98].

DNA extraction by the CTAB method:

The genomic DNA of each strain was extracted according to the fungal DNA extraction using CTAB method [99].
Materials: water bath, grinder, small high-speed centrifuge (maximum centrifugal force ≥ 12000) × g), 1.5 mL centrifuge tube, 70% ethanol, isopropanol, trichloromethane, Liquid nitrogen, etc.
To ensure the extraction quality of DNA, the following protocol was followed. First, two grinding beads were added to a sterile 1.5 ml centrifuge tube. Then, 25-100 mg of purified pathogen hyphae were placed in the same tube. Next, 300 µl of liquid nitrogen was added, and the sample was ground for 1 minute using a grinder. The tube containing the pathogenic hyphae was ground with liquid nitrogen to aid in cell lysis. Afterwards, liquid nitrogen (CTAB)

solution was added to the centrifuge tube, and the mixture was shaken to facilitate DNA extraction. Subsequently, 600 µl of trichloromethane was added, and the tube was centrifuged at 12,000 rpm for 10 minutes at room temperature. Following centrifugation, 500 µl of the upper phase, which contained the DNA, was carefully transferred to a new tube. Then, 500 µl of isopropanol was added, and the tube was centrifuged again at 12,000 rpm for 10 minutes at 20 °C. The resulting DNA pellet was washed with 500 µl of 70% ethanol and centrifuged at 12,000 rpm for 5 minutes at 20 °C. The DNA pellet was subsequently dried for 50 minutes. Finally, the pellet was re-suspended in 50 µl of ddH2O to obtain the DNA sample. This extraction procedure ensured the isolation of high-quality DNA from the purified pathogen hyphae.

PCR amplification of DNA fragments:

Fusarium was identified as the fungus, sequencing the internal transcribed spacer (ITS) regions confirmed the primer sequence obtained after the laboratory test. After that, 50 L PCR reactions were prepared with primers ITS1 (5'-TCCGTAGGTGAACCTG CGG-3') and ITS4 (5'-TCCTCCGCTTATTGATATGC-3') [100]. Sangon Biotech Kit was used for the PCR experiment, which included an initial denaturation for 3 minutes at 95°C, 35 cycles of 1 minute at 95°C, annealing for 40 seconds at 54°C, extension for 40 seconds at 72°C, and final extension for 10 minutes at 72°C. PCR products were separated by electrophoresis through 1.2% agarose gels in TAE, and the extracted gel was detected using a gel electrophoresis imaging strip. The concentration of the DNA sample was measured at the same time.

Those meeting the specifications were sent to Hangzhou Shangya Biotechnology Co., Ltd. for sequencing. The universal primers for the Fusarium species complex, namely EF-1H and EF-2T, given by White et al. [101], were used to amplify the TEF region of the genomic DNA of fusarium isolates. Post-PCR protocol is the same as for ITS.

Table 2.1 Protocol of the Reaction system

ddH2O	20.0 µl
2 × Rapid Taq Master Mix	25.0 µl
Primer1 (10 µM)	2.0 µl
Primer2 (10 µM)	2.0 µl
Template DNA*	50 µl

Table 2.2 Primers used in this study.

Primer	Sequence (5'-3')	PCR product	Reference
ITS1	TCCGTAGGTGAACCTGCGG	570bp	[100]
ITS4	TCCTCCGCTTATTGATATGC		
TEF-F	ATGGGTAAGGARGACAAGAC	700bp	[101]
TEF-R	GGARGTACCAGSATCATGTT		

Phylogenetic relationship analysis:

Molecular Evolutionary Genetics Analysis software was used to perform phylogenetic analyses based on ITS and TEF sequences. Blastn software was used to align the sequences with other reference sequences from the genus Fusarium from the National Center for Biotechnology Information (NCBI). Fusarium species with high similarity sequences were downloaded from the GenBank database. Phylogenetic analyses were performed using Neighbor joining algorithm in Mega 11 software.

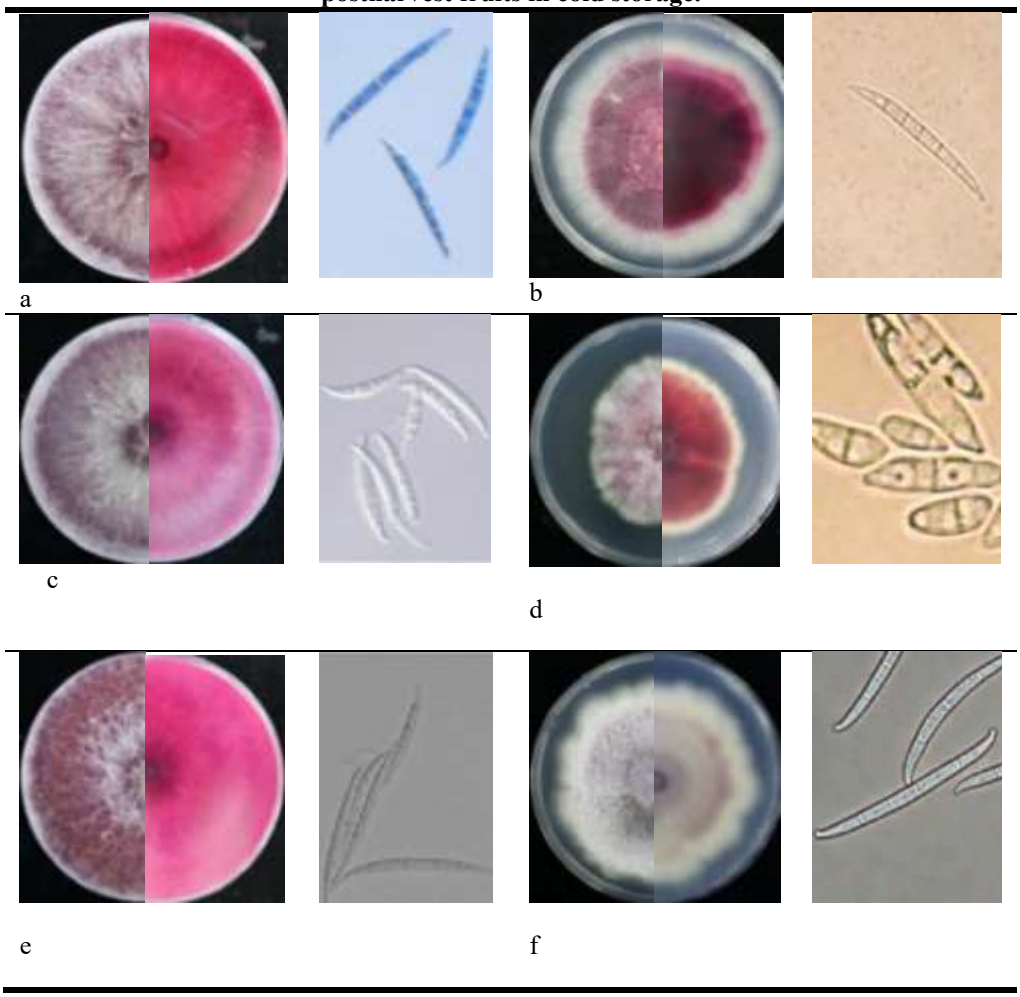
Data analysis and processing:
The obtained sequences were compared in NCBI (National Center for Biotechnology Information) nucleic acid database, the data were processed by Excel 2010, and the phylogenetic tree was constructed by neighbor joining (NJ) in mega 7.0 software.

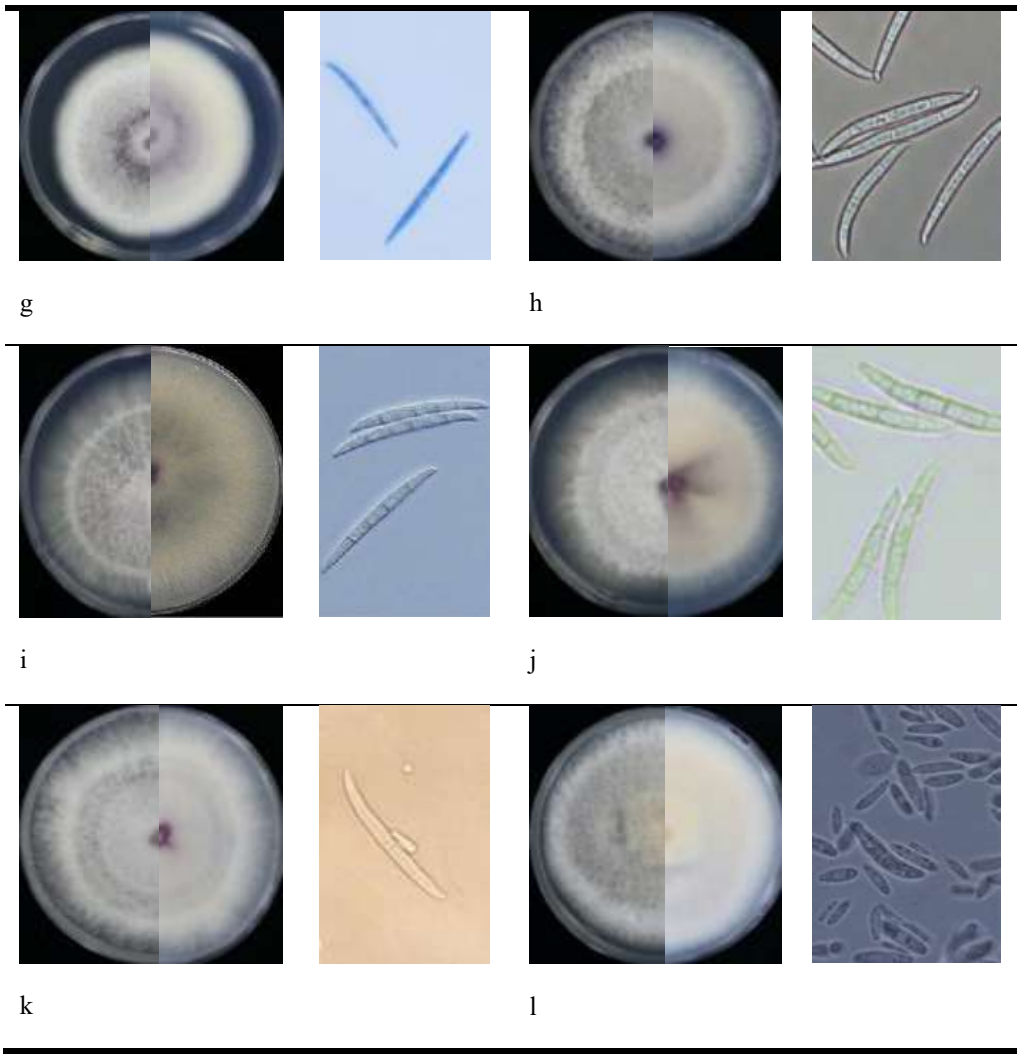
Results and Discussion:-

Identification of Fusarium spp.
12 Fusarium isolates were isolated from the diseased fruits with the postharvest rot symptoms on cherry fruits at 3 locations (table 2.4). The isolates originating from every culture were characterized based on their morphological characteristics, such as colonies, macroconidia and microconidia of Fusarium isolates, as shown in figure 2.2. According to the results, the sequences obtained in our laboratory was as determined to be Fusarium isolates namely Fusariumproliferatum, Fusarium lateritum, Fusarium fujkuroiand Fusarium graminearum belonging to four species [90].

Leyva-Madrigal et al (2015) reported that Fusarium spp is a widespread group of fungi that comprises many important pathogens that can cause severe yield losses [102]. Wang and Wang (2017) also reported that Fusarium equiseti caused postharvest rot in stored cherry fruits [103]. In this study, new symptoms were observed with cotton-like in diseased cherry fruits, different from the symptoms caused by Fusarium spp published before. Therefore, the new symptoms presented in this study could provide helpful information for the rapid diagnosis of this postharvest disease.

Figure. 2.2. Morphological characteristics of Fusarium species isolated from diseased Chinese cherry postharvest fruits in cold storage.





Isolated F. graminearum (a), F. graminearum (b), F. graminearum (c), F. fujikuroi (d), F. proliferatum (e), F. fujikuroi (f), F. fujikuroi (g), F. lateritium (h), F. lateritium (i), F. proliferatum (j), F. proliferatum (k), F. proliferatum (l) and F. proliferatum (m)

Table 2.3 Colonies and macroconidia features for Fusarium species isolated in this study.

Fusarium species	Colonies growth rate (mm/d)	Macroconidia features			
		Shape	Length (µm)	Width (µm)	Septum
F. proliferatum	8.4-8.9	slender	13–24	3.1–3.9	4-6
F. lateritium	6.9-7.4	falcate	37–45	2.9–4.9	2-4
F.fujikuroi	7.5–8.6	falcate	24.4–39	2.9–4.9	2-5
F. graminearum	8.8-9	oval	30-45	2.6-3.7	4-5

Sequences analysis:
ITS Sequences analysis of Fusarium isolates was sequenced and submitted in NCBI database. Based on the closest match of BLAST analysis, it showed 100% homology with F. graminearum, F.Fujikuroi, F. lateritiumand F. proliferatumwith the accession numbers of deposited in table 2.4.

TEF Sequences analysis of Fusarium isolates were sequenced and submitted in NCBI database. Based on the closest match of BLAST analysis, it showed 100% homology with F. graminearum, F. proliferatum, F. Fujikuroi and F. lateritium with accession numbers deposited in table 2.4. Hence, the pathogens were confirmed as Fusarium species.

Sequencing the ITS and TEF gene region is effective for identifying some species of Fusarium. The current fungal taxonomic systems have used macroconidia and microconidia in the asexual stages to identify fungal species. However, the plasticity and intergradation of the phenotypic traits has presented difficulties in identifying the filamentous fungi [104]. Similar studies by Wang et al. (2021) which involved isolation of 21 fungal species from infected cherries and performed PCR amplification of ITS and TEF gene region using universal ITS and TEF primers [105]. The fungal pathogens of cherries were identified as belonging to four species, Fusarium fujikuroi, Fusarium lateritium, Fusarium proliferatumand Fusarium graminearumbased on its cultural, morphological and molecular characteristics. Molecular profiling using ITS region sequencing is an indispensable method for identification studies as studied by [106].

Summerell et al (2010) and Shan et al (2019) reported that Fusarium genus contains about several species that are important plant pathogens worldwide [107], [108]. Accurate identification of pathogen plays a vital role in the development of disease management strategies [109]; [110]. [111] reported that Fusarium isolates are very difficult to differentiate based only on their morphological properties, but [89], [109], [112] and [113] suggested that both morphological characteristics and molecular approach are necessary to accurately identify Fusarium species.

Table 2.4 Information of the Fusarium species isolated from our lab

Isolate	Suggested identification	Geographical location	Year	GenBankaccession number (ITS)	GenBank accession number (TEF-1α)
H19-1	F. graminearum	Henan	2021	MK780229.1	MK896869.1
B30-1	F. graminearum	Henan	2020	MF800906.1	MG826888.1
SCHY 10-2	F. graminearum	Shandong	2021	MH299910.1	KY283902.1
H13-1	F. fujikuroi	Henan	2020	MW260108.1	MN223456.1
HZZ 29-1	F. proliferatum	Hebei	2020	OK325614.1	KF267266.1
B34-1	F. fujikuroi	Hebei	2020	MW314763.1	MK443268.1
YTMZ 60-3	F. lateritium	Hebei	2020	KC787693.1	OK484428.1
HZZ 14-2	F. lateritium	Shandong	2021	OK482905.1	MF521454.1
YTMZ 40-3	F. proliferatum	Shandong	2021	MW391506.1	MK952792.1
H24-1	F. proliferatum	Hebei	2021	MT372093.1	MN861758.1
YTMZ 29-1	F. proliferatum	Henan	2020	MN747996.1	KT239489.1
YTMZ 52-1	F. proliferatum	Shandong	2020	MT039382.1	MW091266.1

ITS, internal transcribed spacer; TEF1-α, translation elongation factor.

Phylogenetic Analysis:
Diseased cherry fruits were collected from three different regions in China for our study. All 12 Fusarium species isolates from 4 Fusarium species were successfully identified using morphological and molecular characteristics.

Morphological identification was carried out using multi-locus phylogenetic analyses. The ITS region is the most commonly used genomic region for fungal identification [114]. According to Nayyer et al (2018) and Guan et al (2020) ITS region can distinguish Fusarium from other fungi, and it is incapable of distinguishing between closely related Fusarium species [110]; [113]. TEF-1, a highly conserved protein-encoding region, was more effective in determining various closely related Fusarium species [115]; [116].

The F. graminearum, F. fujikuroi, F. proliferatum, and F. lateritium, sequences isolated from our lab was then compared with other fungal sequences to determine the relation of the pathogens.
In this study, a combination of ITS and TEF-1 gene regions were useful in successfully distinguishing the closely related Fusarium species in the phylogenetic tree.
Based on the phylogenetic findings, all Fusarium isolates were suitable for use in our research it was purified, cultured on PDA and stored at 4°C.

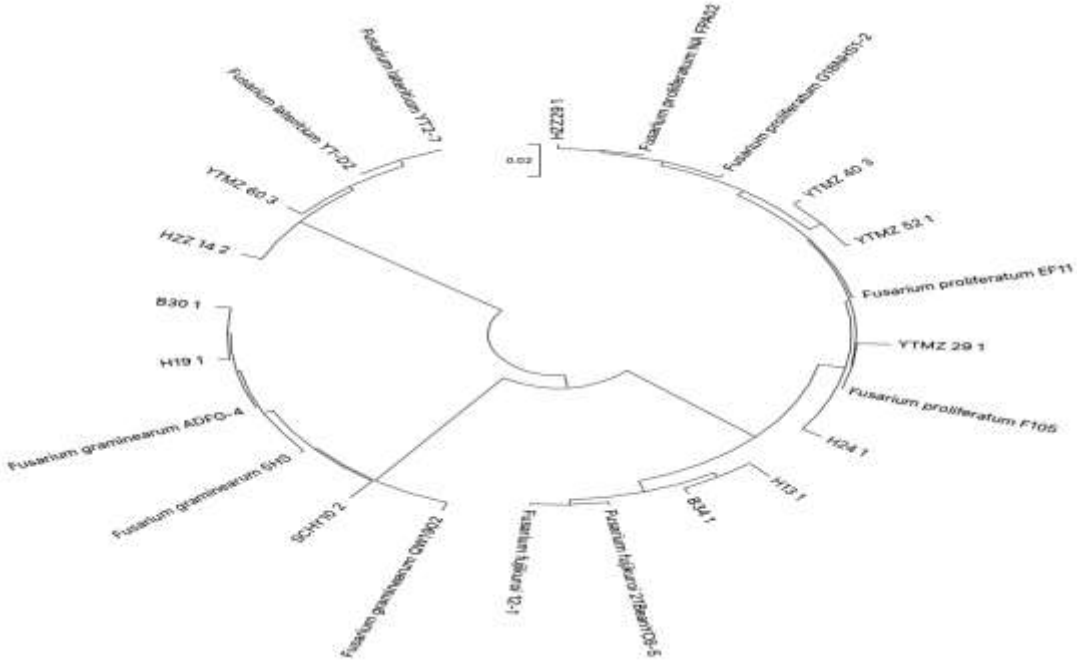


Figure 2.3. Phylogenetic tree using MEGA 7.2 showing fungal sequences

Following fungal identification and determining that the extracted fungi wasF. graminearum, F. fujikuroi, F. proliferatum, and F. lateritium, sequencing comparison using software Mega 7.2 with other sequences obtained from NCBI, all Fusariumisolates showed a phylogenetic relationship with other sequence.

THE ANTIFUNGAL EFFECTS OF FUNGICIDES ON FUSARIUM SPECIES:
Fusarium fungi are known to cause postharvest diseases in cherry fruits during storage, which can adversely affect their quality. Several Fusarium species are responsible for postharvest decay in cherry fruits. Therefore, managing postharvest diseases is a crucial aspect of cherry fruit production during storage.

There is no single effective method for controlling Fusarium fungi in fruits and other crops. Fungicides are commonly used to manage pathogenic Fusarium species. However, there are currently not enough varieties available that exhibit measurable resistance to Fusarium diseases. Some Fusarium species that infect cherry fruits can produce mycotoxins, resulting in severe postharvest decay.

Materials and Methods:-

Test reagents and methods:
The technical drugs for the tested drugs were dissolved in acetone and prepared into 10 mg/L drug mother liquor, respectively, which were stored in the refrigerator at 4℃ until ready for use. Please refer to the table below for the tested drugs and their respective concentration gradients.

For the fungicide sensitivity trials, twelve Fusarium isolates were selected. The recommended fungicides used in this study were y19315 and Mefentrifluconazole. Two hundred milliliters of each fungicide and 200 mL of penicillin were added to separate flasks containing 200 mL of Potato Dextrose Agar (PDA) medium. The fungicides were added at the concentrations specified in the table below, with each concentration tested in triplicate. The experiment was conducted using the laboratory test method recommended for fungicides [117]. Fungal colonies were grown in Petri dishes containing PDA mixed with fungicides and without fungicides (control experiment). The diameters of the colonies were measured after the colonies had grown to 50-70 mm in size.

Table 3.1 Fungicides and their concentration for tests

Fungicide	Concentration
y19315	0.6, 0.3, 0.15, 0.075
Mefentrifluconazole	1, 0.5, 0.25, 0.125

In vitro antifungal susceptibility to Fusarium isolates:
The sensitivity of each isolate to y19315 and Mefentrifluconazole was determined by comparing radial growth on media affected by fungicides to growth on unaffected media (control). y19315 was diluted in methanol to four concentrations, and Mefentrifluconazole was diluted in sterile distilled water to a concentration of 40 μg/mL. To flasks containing 200 mL Potato Dextrose Agar (PDA) medium, 200 μL of fungicides and 200 μL of penicillin were added. As a control, PDA plates containing only 200 μL of penicillin were used. Table 3.1 shows the concentrations of each fungicide used. Each fungicide concentration was mixed into autoclaved media.

The effect of the fungicides on mycelial growth in vitro was studied using PDA media supplemented with the fungicide concentrations listed in Table 3.1. A 5-mm diameter mycelial plug was transferred to the center of petri dishes (90 mm) with the fungicide-amended media and the non-amended media from the margin of a five-day-old actively growing culture of each isolate. The sensitivity of the isolates to the fungicides was determined by measuring the colony diameter when the control mycelial grew to 50-70 mm in diameter after incubation at room temperature in the dark. Each plate yielded two perpendicular colony diameter measurements, as well as the original plug diameter (5 mm). The experiment was repeated.

The diameter of each colony on fungicide-affected media was measured in comparison to the diameter of the colony on unaffected media. For each fungicide concentration, the relative growth reduction percentage was calculated as follows: 100-(diameter on fungicide-amended medium/diameter on non-affected medium) *100. The value for each isolate was calculated by determining the effective fungicide concentration that inhibited mycelial growth by 50% using SPSS. Isolates were considered sensitive if their EC_{50} values were equal to or less than 10 μg/mL, and resistant if their EC_{50} values were greater than 10 μg/mL [118]. The experiment was carried out using the method recommended for fungicide laboratory tests [117].

In vivo Experiment of fungicides:
Fully matured cherry fruits were obtained from a commercial orchard and transported immediately to the laboratory. Uniform-sized and unblemished cherry fruits with consistent color were selected for the test. To determine pathogenicity, ten healthy cherry fruits were randomly chosen, surface sterilized with 75% ethanol, rinsed three times with distilled water, and dried. After culturing strains in darkness at 25°C on PDA medium for 7 days, sterile water was added to the submerged colony surface to wash out the spores with an applicator. The spores were then filtered through three layers of filter paper to create a spore suspension, which was adjusted to a concentration of 10^6/mL. Using a sterilized fine needle, the dried cherry surface was punctured and inoculated with 10 μL of the spore suspension.

It was placed in a light incubator with alternating light and dark for 12 hours at 25°C for moisturizing culture. After 24 hours, each fruit was sprayed with 0, 10, 50, and 100 μg/mL of 200 μL of the test reagent, respectively, and samples that were not sprayed served as the control. Each treatment was repeated three times. The incidence was observed after 7 days, and the control effect was calculated using the following formula: E/% = [(Dc − Dt)/Dc] ×

100. Where E is the control effect as a percentage, DC is the diameter of the lesion in the control group in mm and DT is the diameter of the lesion in the treatment group in mm.

Data Analysis:-

The data processing software used for calculating the EC_{50} value of Fusarium species on mycelial growth was SPSS Statistics 23.0. The pathogenicity data were analyzed using one-way ANOVA based on a completely randomized design. Means were compared using Tukey's test ($P \leq 0.05$), also utilizing IBM SPSS Statistics 23.0

Results and Discussion:-

In vitro antifungal susceptibility to Fusarium isolates:

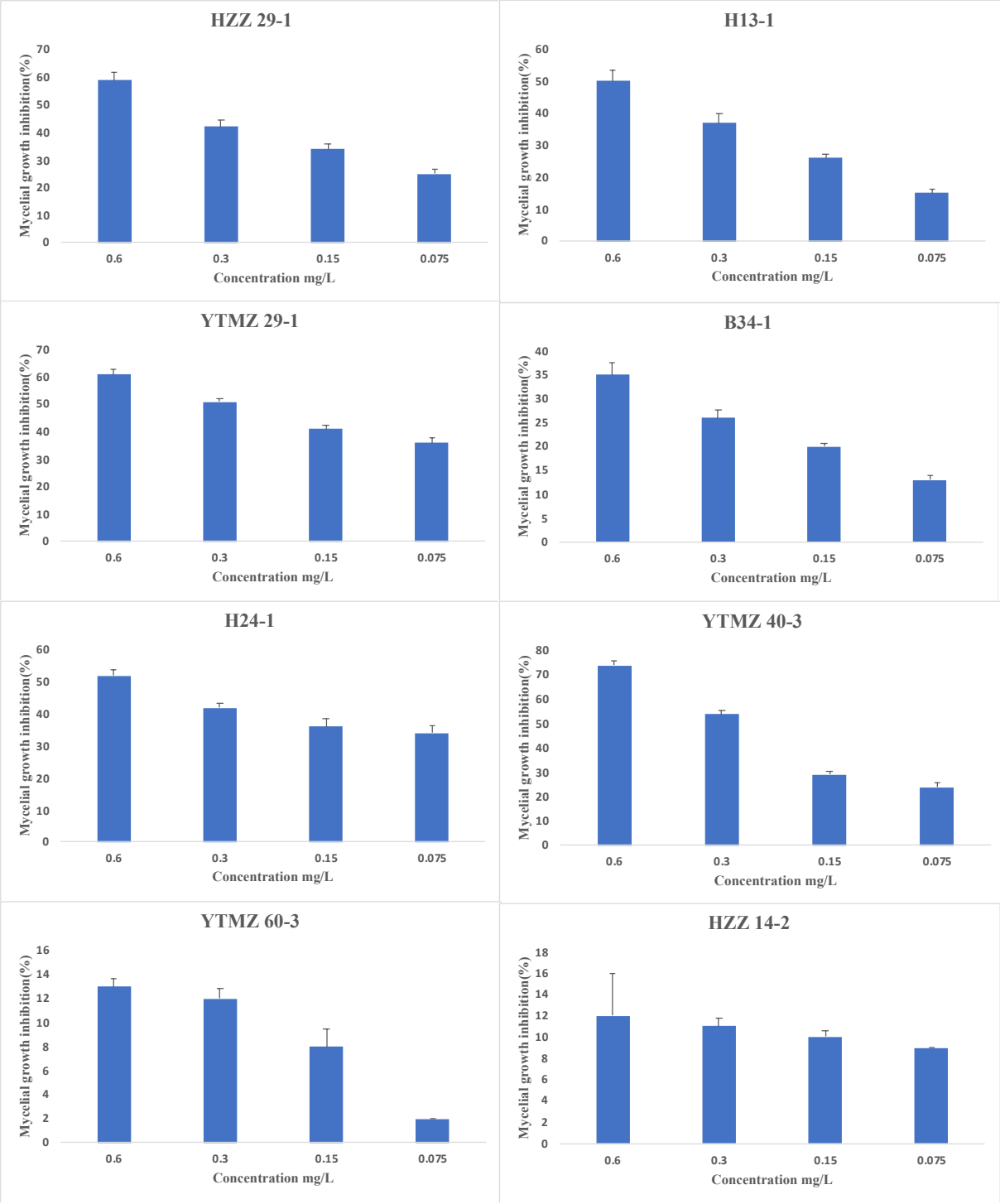
The effect of increasing fungicide concentration on mycelial growth of Fusarium species was evaluated. Table 3.2 presents the results obtained. The findings revealed a significant ($p<0.05$) reduction in mycelial growth for all tested fungicides across the concentration gradient.

Specifically, the mycelial growth of H19-1 and YTMZ 52-1 was notably suppressed by y19315, with reductions of 24.5 mm and 26.5 mm, respectively, observed at a concentration of 0.075 mg/L. These results corresponded to inhibitions ranging from 50% to 60%, as depicted in Figure 3.1. When the concentration was increased to 0.15 mg/L, y19315 exhibited the highest detrimental effect on mycelial growth, resulting in reductions of 13 mm for YTMZ 52-1, 19.17 mm for B30-1, and 19.17 mm for H19-1, with inhibitions ranging from 61% to 80%. Moreover, y19315 fungicide significantly ($p<0.05$) suppressed mycelial growth of YTMZ (8.88 mm) and H19-1 (15.83 mm) across the concentration gradient, resulting in inhibitions ranging from 67% to 87%, as outlined in Table 3.2. Notably, increasing the concentration to 0.6 mg/L led to even greater reductions in mycelial growth caused by y19315, as highlighted in Table 3.2.

Table 3.2. Effect of y19315 fungicide on Fusarium isolates mycelium growth along concentration gradient.

	Mycelial Growth diameter (mm)				
Isolates	Fungicide concentration (μL/mL)				
	CK	0.6	0.3	0.15	0.075
	y19315				
HZZ 29-1	58±3.69d	23.8±2.71e	33.5±2.51d	38.5±2.07dc	43.3±1.63d
H13-1	56.5±2.07e	28.3±3.5d	35.3±2.8c	42±1.1c	48±1.26b
YTMZ 29-1	59.3±3.01d	23±2.1e	29±1.26e	34.8±1.6d	37.8±1.83
B34-1	63±1.26c	41±2.45a	46.8±1.72a	50.2±0.75a	54.33±0.82a
H24-1	67.17±1.83b	32.43±1.9c	39±1.26b	43±2.37cb	44.5±2.35d
YTMZ 40-3	60.5±3.33d	15.83±1.94f	28±1.55e	42.83±1.47b	46±1.9c
YTMZ 60-3	46±1.26g	40±0.63a	40.6±0.82b	42.33±1.51b	45±0d
HZZ 14-2	44.5±2.07g	39.17±3.92b	39.67±0.82b	40.18±0.63c	41±0e
YTMZ 52-1	65.83±1.17c	8±1.55h	8.88±4.31h	13±1.55f	26.17±3.19g
B30-1	48.5±9.75f	12±0.89g	18.17±3.66f	19.17±2.56e	32.33±1.97f
H19-1	48.67±2.66f	12.5±1.05g	15.83±2.56g	19.17±2.64e	24.5±1.22g
SCHY 10-2	78.33±2.25a	20.83±0.98e	26.33±1.63e	39.83±1.17dc	46.17±0.75c

Mean values with the same lowercase letters in the same column did not differ significantly (p>0.05) according to Duncan's Multiple Range Test.



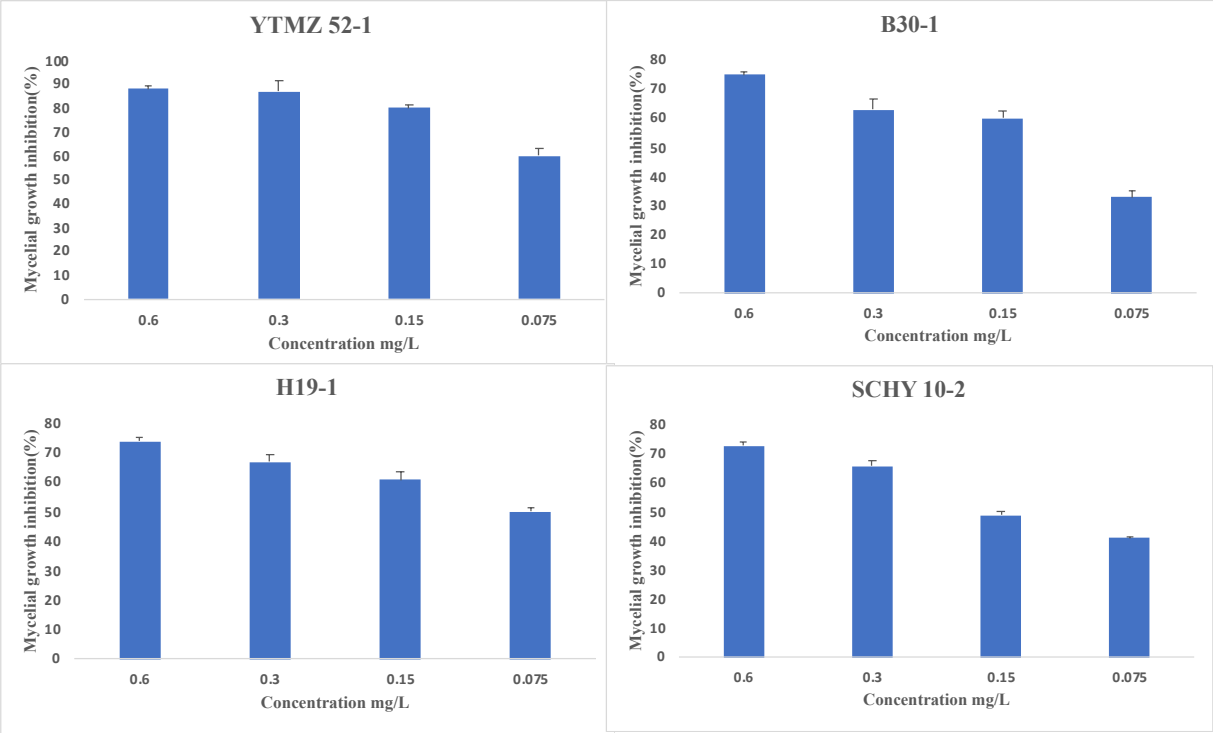


Figure 3.1. Inhibitory effect of y19315 fungicides on mycelial growth of Fusarium isolates.

The effect of increasing the concentration of Mefentrifluconazole fungicide on the mycelial growth of Fusarium species was investigated, and the results are presented in Table 3.3. The findings demonstrated a significant ($p<0.05$) decrease in mycelial growth for all tested fungicides across the concentration range.

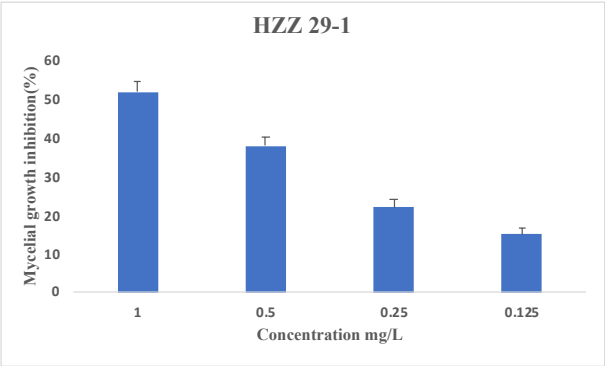
The mycelial growth of H24-1 was notably inhibited by Mefentrifluconazole, with reductions of 43.67 mm observed at a concentration of 0.125 mg/L. These results corresponded to a 35% inhibition, as shown in Figure 3.1. When the concentration was increased to 0.25 mg/L, Mefentrifluconazole had the most detrimental effect on mycelial growth, resulting in reductions of 41.33 mm for H24-1 and 41.83 mm for YTMZ 52-1, with inhibitions ranging from 36% to 38%. At a concentration of 0.5 mg/L, Mefentrifluconazole fungicide significantly ($p<0.05$) suppressed mycelial growth consistently across the concentration gradient, as outlined in Table 3.2. Importantly, increasing the concentration to 1 mg/L led to an even greater reductions in mycelial growth caused by Mefentrifluconazole, as highlighted in Table 3.2.

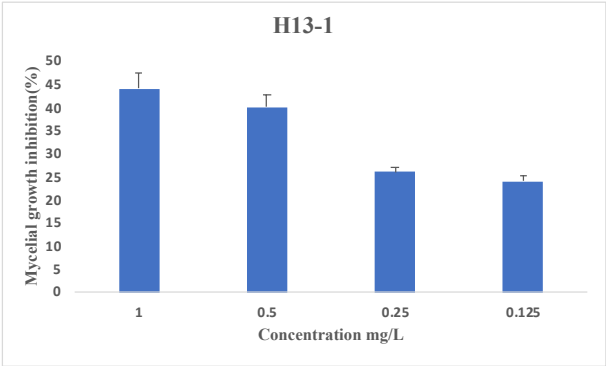
Table 3.3. Effect of Mefentrifluconazole fungicide on Fusarium isolates mycelium growth along concentration gradient.

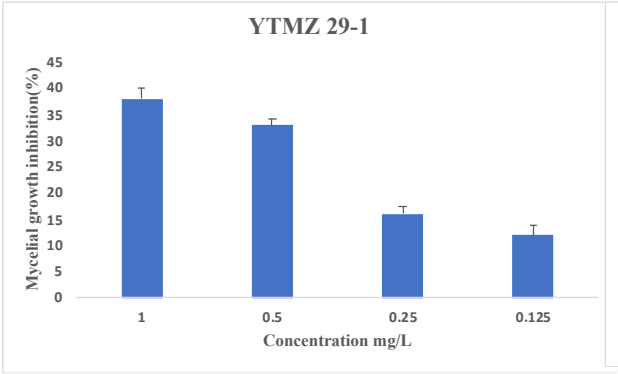
	Mycelial Growth diameter (mm)				
Isolates	Fungicide concentration				
	CK	1	0.5	0.25	0.125
	Mefentrifluconazole				
HZZ 29-1	58±4.1e	27.8±12.2f	36.2±2.04e	45.5±0.55d	49.3±1.63d
H13-1	56.5±2.07f	31.8±0.98e	33.8±0.75g	42±0.89e	43.17±0.41f
YTMZ 29-1	59.3±3.01e	37±0.63c	40±1.1d	49.8±0.41c	52±0c

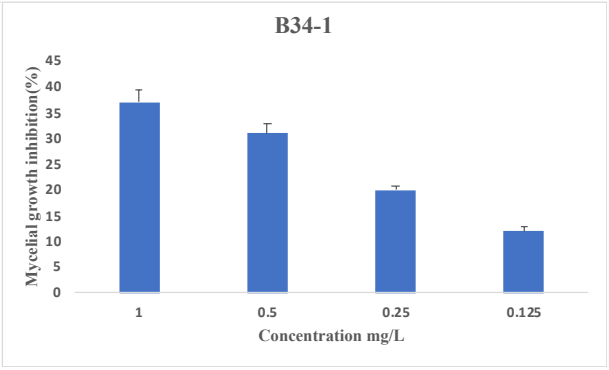
B34-1	63±1.26d	39.67±0.52b	43.33±1.03c	50.33±0.82c	55.33±4.18b
H24-1	67.17±1.83b	27.83±1.6	31.33±0.52g	41.33±3.33	43.67±1.97
YTMZ 40-3	60.5±3.33e	40.83±2.04b	49.83±3.13b	54.83±1.47b	55.5±1.64b
YTMZ 60-3	46±1.26h	40.17±1.94b	39.83±0.75d	42.33±2.07e	45±0e
HZZ 14-2	44.5±2.07h	40.5±1.22b	40.17±0.41d	43.5±2.07e	45±0e
YTMZ 52-1	65.83±1.17c	27.83±1.47f	33.67±1.03g	41.83±0.98f	49.17±0.41d
B30-1	48.5±9.75g	32.5±1.52d	35.33±0.82e	37.67±1.03g	39.67±0.82g
H19-1	48.67±2.66	31.83±3.19e	34.5±3.08f	37.17±2.32g	37.5±5.09g
SCHY 10-2	78.17±2.14a	52.67±1.21a	56±1.26a	61.5±1.64a	70.33±3.01a

Mean values with the same lowercase letters in the same column did not differ significantly ($p > 0.05$) according to Duncan's Multiple Range Test.









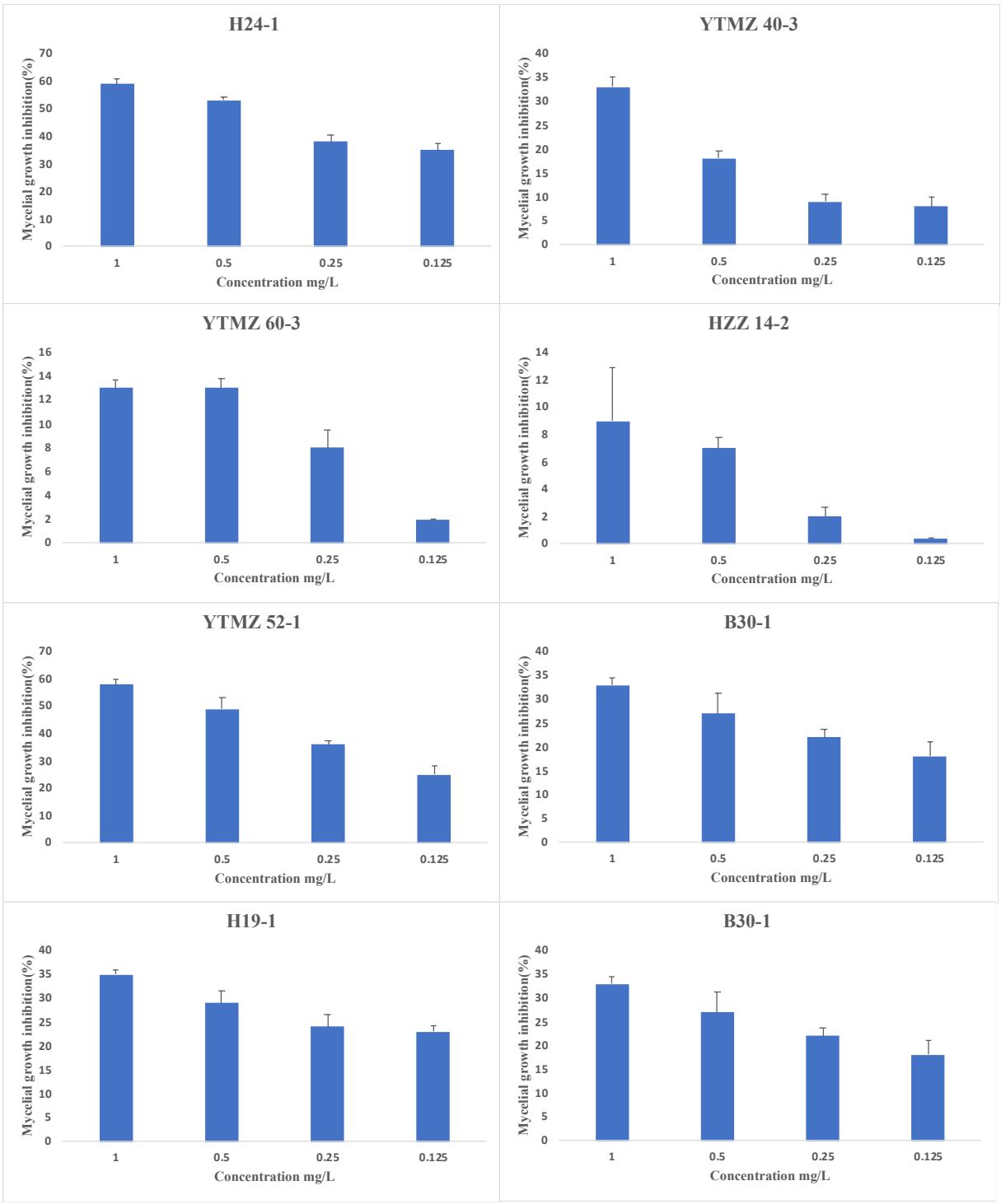


Figure 3.1. Inhibitory effect of fungicides on mycelial growth of Fusarium isolates.

Table 3.4 EC_{50} values corresponding to 50% inhibition of mycelial growth

Strain name	Fungicide name	EC_{50} values	Regression equation	r^2
HZZ 29-1	y19315	0.323	y=0.504+1.026x	0.993
	Mefentrifluconazole	1.452	y=-0.102+0.63x	0.887
H13-1	y19315	0.723	y=0.230+1.633x	0.968
	Mefentrifluconazole	1.151	y=-0.045+0.73x	0.914
YTMZ 29-1	y19315	0.198	y=0.543+0.772x	0.984
	Mefentrifluconazole	1.530	y=-0.194+1.05x	0.925
B34-1	y19315	1.608	y=-0.162+0.78x	0.980
	Mefentrifluconazole	1.727	y=-0.216+0.91x	0.929
H24-1	y19315	0.433	y=0.200+0.550x	1
	Mefentrifluconazole	0.354	y=0.341+0.756x	0.938
YTMZ 40-3	y19315	0.225	y=1.091+1.685x	0.980
	Mefentrifluconazole	2.523	y=-0.449+1.11x	0.996
YTMZ 60-3	y19315	1.862	y=-0.218+0.80x	0.919
	Mefentrifluconazole	2.627	y=-0.368+0.87x	0.881
HZZ 14-2	y19315	1.092	y=-0.051+1.34x	0.981
	Mefentrifluconazole	2.9	y=-0.489+1.05x	0.911
YTMZ 52-1	y19315	0.033	y=1.940+1.313x	0.771
	Mefentrifluconazole	0.484	y=0.319+1.012x	0.942
B30-1	y19315	0.152	y=1.179+1.439x	0.864
	Mefentrifluconazole	1.540	y=0.181+0.965x	0.951
SCHY 10-2	y19315	0.115	y=1.013+1.078x	0.864
	Mefentrifluconazole	2.389	y=-0.331+0.87x	0.951
H19-1	y19315	0.085	y=1.062+0.99x	0.901
	Mefentrifluconazole	0.947	y=-0.910+0.02x	0.935

EC_{50} values (µg m/L) of F. graminearum, F. fujikuroi, F. proliferatum, and F. lateritium isolates grown on potato-dextrose media amended with y19315 and mefentrifluconazole.

The inhibitory effects of two fungicides on mycelial growth of Fusarium graminearum were significantly different. Generally, higher concentrations of mefentrifluconazole were required to reduce colony growth by 50% compared to y19315. y19315 was more sensitive in its inhibitory effects compared to mefentrifluconazole. The EC_{50} values of y19315 for three strains of Fusarium graminearum ranged from 0.085 µg/mL to 0.152 µg/mL (see Table 3.2), with a mean of 0.117 µg/mL. The EC_{50} values of mefentrifluconazole for three strains of Fusarium graminearum ranged from 0.947 µg/mL to 2.389 µg/mL, with a mean of 1.625 µg/mL.

For five strains of Fusarium proliferium, the EC_{50} values of y19315 ranged from 0.033 µg/mL to 0.433 µg/mL, with a mean of 0.242µg/mL. The EC_{50} values of mefentrifluconazoleranged from 0.354 µg/mL to 2.523 µg/mL, with a mean of 1.2686 µg/mL. For two strains of Fusarium fujikuroi, the EC_{50} values of y19315 ranged from 0.723 µg/mL to 1.608 µg/mL, with a mean value of 1.1655 µg/mL. The EC_{50} values of mefentrifluconazole for two strains of Fusarium fujikuroi ranged from 1.151 µg/mL to 1.727 µg/mL, with a mean value of 1.439 µg/mL.

For two strains of Fusarium lateritium, the EC_{50} values of y19315 ranged from 1.092µg/mL to 1.862 µg/mL, with a mean value of 1.477 µg/mL. The EC_{50} values of mefentrifluconazole for two strains of Fusarium lateritium ranged from 2.627 µg/mL to 2.9 µg/mL, with a mean value of 2.7635 µg/mL.

In recent years, the planting area of cherry fruit has expanded rapidly, and the occurrence of postharvest disease has become more and more serious. However, effective fungicides for controlling Fusarium in cherry have not been reported. In this study, the indoor toxicity of two fungicides was determined using the hyphal growth rate method for 12 strains of four types of Fusarium.

Triazoles are sterol-inhibiting fungicides that have no immediate effect on the respiratory mechanism. Therefore, they do not inhibit spore germination [119], but they are effective in preventing mycelial growth. These fungicides inhibit DNA synthesis [120].

The radial growth assay was utilized in this study [121] to test the sensitivity of all fungicides. Variations in sensitivity to fungicides in vitro were observed among isolates. While all isolates were sensitive to the tested fungicides, sensitivity varied among the isolates and species. A new fungicide, y19315, which is still under development, reduced radial growth by 50% for all Fusarium species at lower concentrations than mefentrifluconazole. This indicates that y19315 would likely be more effective at controlling Fusarium species at lower concentrations than mefentrifluconazole. Ma et al. (2020) reported that mefentrifluconazole did not have a significant influence on the sensitivity change of C. cassiicola[122]. Although y19315 and mefentrifluconazole have similar modes of action, a greater concentration of mefentrifluconazole was needed to reduce growth by 50% for F. graminearum, F. lateritium, F. proliferatum, and F. fujikuroi species.

A large number of studies have shown that there are significant differences in the sensitivity of different Fusarium strains to fungicides. Masiello et al. (2019) reported that the fungicide isopyrazam required a higher concentration to inhibit mycelial growth of Fusarium species [123]. Similar results were observed in our study when Fusarium species were treated with mefentrifluconazole. Tini et al. (2020) observed that Fusarium species did not inhibit mycelial growth at lower concentrations [124]. This proves that Fusarium species are challenging to control. As a result, a new fungicide, y19315, was used in this study and showed positive results as it was able to inhibit Fusarium growth at lower concentrations compared to the already-developed fungicide mefentrifluconazole.

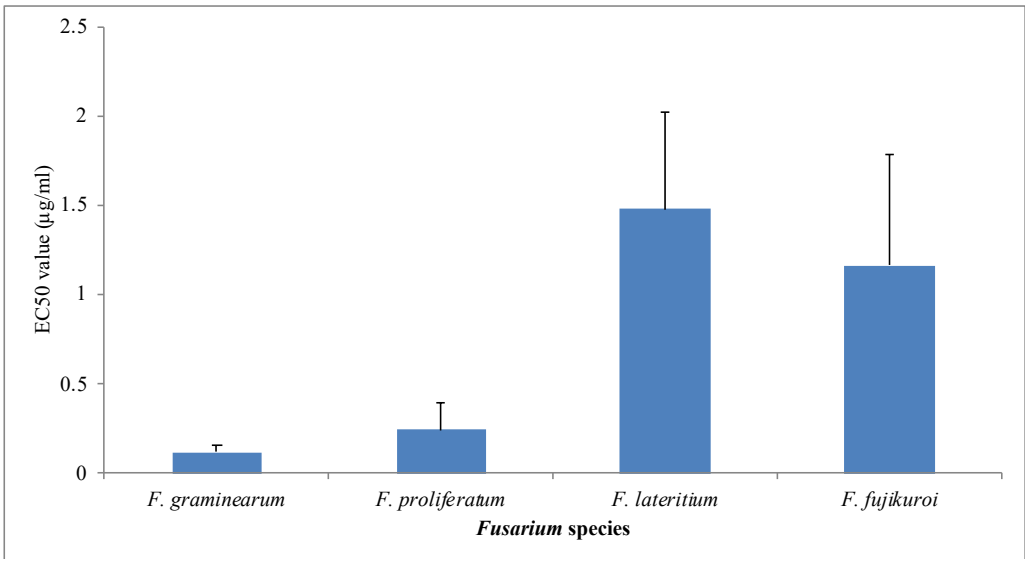


Figure. 3.1. Mean in vitro EC_{50} values of y19315 for isolates of F. graminearum, F. proliferatum, F. lateritiumand F. fujikuroi.

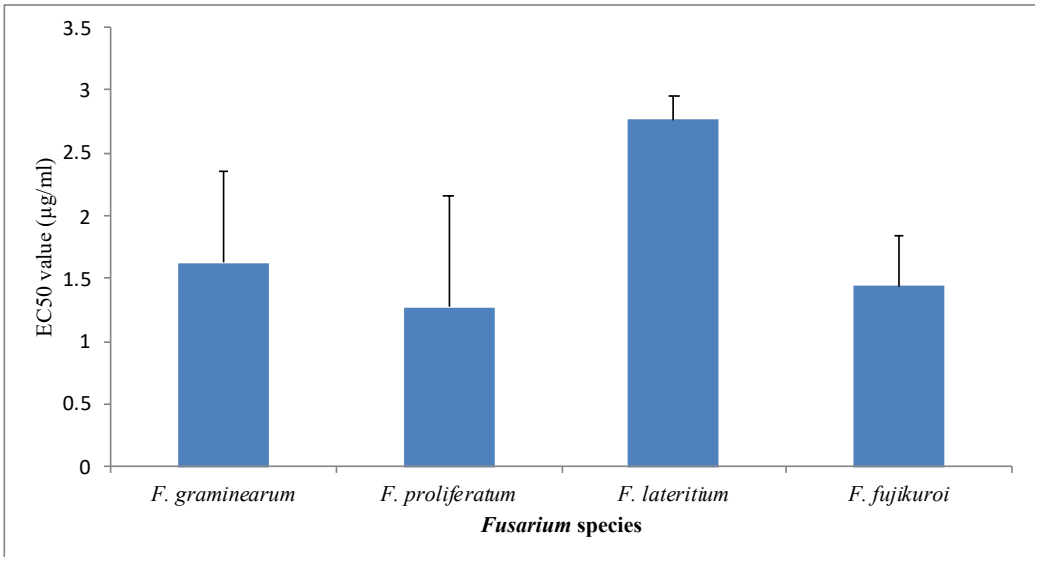


Figure. 3.2. Mean in vitro EC_{50} values of Mefentrifluconazole for isolates of F. graminearum, F. proliferatum, F. lateritiumand F. fujikuroi.

In vivo experiment of fungicide results:

The results of the experiment indicate that cherry fruits inoculated with Fusarium isolates and incubated at 25°C showed a significant increase in fungal rot. In contrast, cherry fruits coated with y19315 had a low lesion diameter compared to those treated with Mefentrifluconazole. Upon close observation of the surface morphology of the inoculated fruits with Fusarium isolates, fruit tissue began to rot around the inoculation point after 24 hours. The control samples showed decay incidence rapidly increasing from the point of inoculation, with the lesion diameter engulfing the fruit on day 7, while treated samples inhibited spore germination on the tissue.

According to figure 3.3, the fungicide y19315 exhibited fruit rot efficacy of 0.1%, 49.71%, and 100% for F. graminearum isolate (H19-1) at concentrations of 10, 50, and 100 µL/mL, respectively. Similarly, for F. proliferatum isolate (HZZ 29-1), the fungicide had fruit rot efficacy of 95.82%, 100%, and 100% at concentrations of 10, 50, and 100 µL/mL, respectively. F. fujikuroi isolate (B34-1) had fruit rot efficacy of 50%, 89%, and 100%, while F. lateritium isolate (YTMZ 60-3) had fruit rot efficacy of 91%, 100%, and 100% at concentrations of 10, 50, and 100 µL/mL, respectively. However, at 50 µL/mL, all fungicides showed a highly significant increase in efficacy. At 100 µL/mL, all fungicides exhibited a 100% inhibition of disease efficacy in artificially wounded and inoculated cherry fruits, resulting in 0% fruit rot.

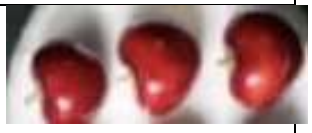
	CK	10	50	100
H19-1				
HZZ 29-1				

Figure 3.3. Effect of cherry fruits coating with various concentrations of y19315 on Fusarium rot disease incidence after 7 days of storage

Overall, the results suggest that y19315 fungicide can effectively inhibit the growth and spread of Fusarium isolates in cherry fruits, with a concentration of 50 μL/mL showing the highest efficacy. These findings have important implications for the management and prevention of fungal diseases in cherry fruits.

Table 3.5. In vivo control efficacy of y19315 on cherry fruits after the inoculation of F. graminearum, F. proliferatum,F. lateritiumand F. fujikuroifungi.

Isolate	Mass concentration of y19315	Control efficacy/%
H19-1	10	0.1±1.33d
	50	49.71±7.83c
	100	100.00 ± 0.00a
HZZ 29-1	10	95.82 ± 11.61ab
	50	100 ± 0.00a
	100	100 ± 0.00a
B34-1	10	50.00 ± 5.09c
	50	89.00 ± 10.59ab
	100	100.00 ± 0.00a
YTMZ 60-3	10	91.1±9.67ab
	50	100.00 ± 0.00a
	100	100.00 ± 0.00a

Mean values followed by different letters within the column are significantly different according to Tukey's test ($p < 0.05$).

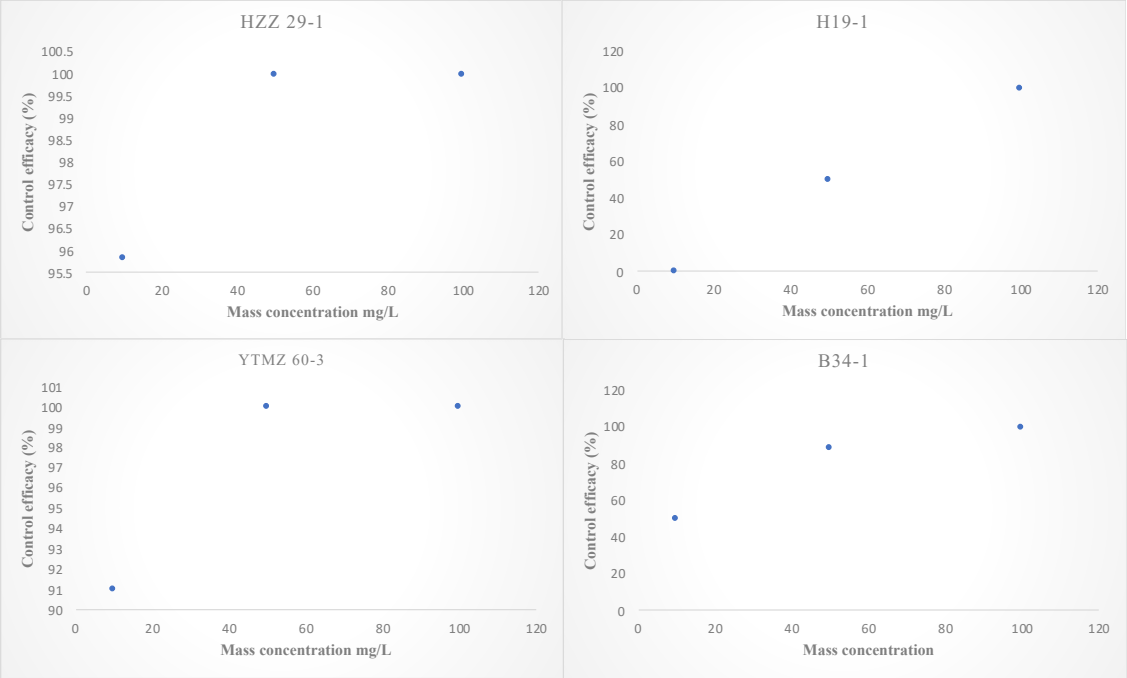


Fig 3.4. In vivo control efficacy of y19315 on cherry fruits after the inoculation of F. graminearum, F. proliferatum, F. lateritiumand F. fujikuroifungi.

According to the results presented in Table 3.4, Mefentrifluconazole fungicide exhibited fruit rot efficacy of 0.01%, 20.77%, and 81% for F. graminearum isolate (H19-1) at concentrations of 10, 50, and 100 µL/mL, respectively. For F. proliferatum isolate (HZZ 29-1), the fungicide y19315 demonstrated fruit rot efficacy of 0.84%, 95%, and 100% at concentrations of 10, 50, and 100 µL/mL, respectively. F. fujikuroi isolate (B34-1) had fruit rot efficacy of 19.01%, 90.45%, and 99.5%, while F. lateritium isolate (YTMZ 60-3) demonstrated fruit rot efficacy of 88.87%, 100%, and 100% at the respective concentrations of 10, 50, and 100 µL/mL. Notably, at the concentration of 50 µL/mL, the fungicides exhibited a significant increase in efficacy. Moreover, at the concentration of 100 µL/mL, Mefentrifluconazole fungicide completely inhibited disease efficacy in artificially wounded and inoculated cherry fruits, except for H19-1 and B34-1 isolates.

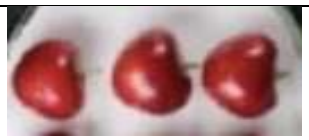
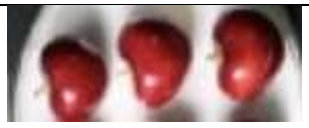
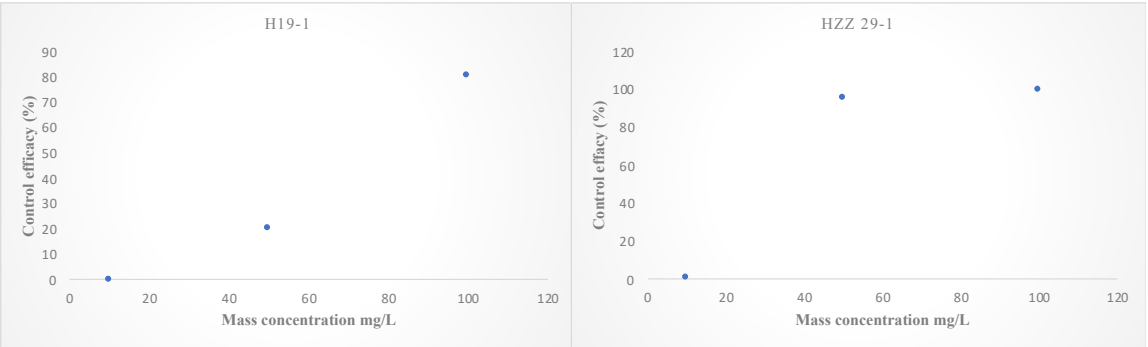
	CK	10	50	100
H19-1				
HZZ 29-1				
B34-1				
YTMZ 60-3				

Fig 3.5. Effect of cherry fruits coating with various concentrations of Mefentrifluconazole on Fusarium rot disease incidence after 7 days of storage

Table 3.6. Invivo control efficacy of mefentrifluconazole on cherry fruits after the inoculation of F. graminearum, F. proliferatum,F.lateritiumand F. fujikuroifungi.

Isolate	Mass concentration of mefentrifluconazole	Control efficacy/%
H19-1	10	0.01±4.31d
	50	20.77±4.36c
	100	81.00 ± 6.21a
HZZ 29-1	10	0.84 ± 17.1ab
	50	95.78 ± 3.55a
	100	100 ± 0.00a
B34-1	10	19.01± 4.07c
	50	90.45 ± 9.89ab
	100	99.50 ± 1.11a
YTMZ 60-3	10	88.87±4.77ab
	50	100.00 ± 0.00a
	100	100.00 ± 0.00a

Mean values followed by different letters within the column are significantly different according to Tukey's test ($p < 0.05$).



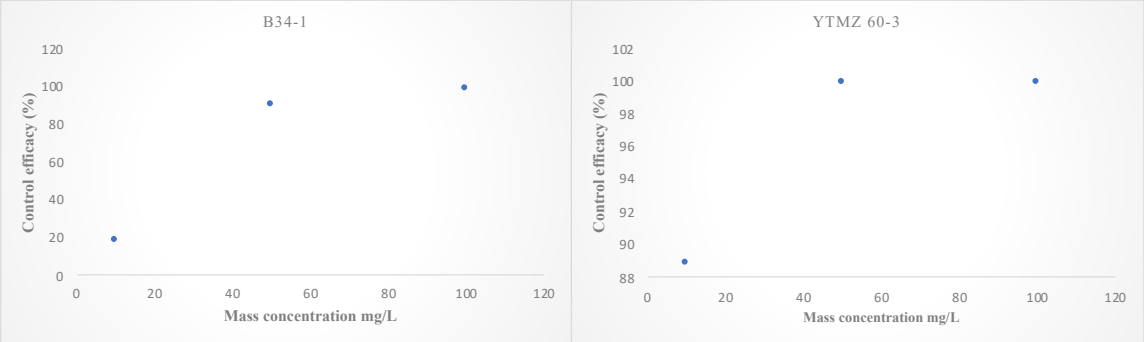


Fig 3.6In vivo control efficacy of mefentrifluconazole on cherry fruits after the inoculation of F. graminearum, F. proliferatum, F. lateritiumand F. fujikuroifungi.

In the in vivo study, the two fungicides chosen from the in vitro trials inhibited both Fusarium species in artificially damaged and inoculated cherry fruit. The fungicides had no phytotoxic effect on the tissues of the fruit at any of the levels examined. Furthermore, these fungicides proved their efficacy against Fusarium isolates and considerably reduced the incidence of fruit rot. There is currently limited research on the in vivo effect of fungicides in cherry fruits.

Romero et al. evaluated the in vivo antifungal efficacy of volatile organic compounds on cherry tomatoes infected with the phytopathogen F.oxysporum[125]. The concentration of the volatile organic compounds ranged from 12.5 to 1000 µg/mL; all treatments resulted in significant inhibition of F. oxysporum growth in a concentration-dependent manner. For concentrations greater than 125 µg/mL, the inhibitory effect was over 40% for all the volatile organic compounds except ocimene. However, this monoterpene was the only terpene with 100% growth inhibition at the largest evaluated concentration (1000 µg/mL). The mixture of six volatile organic compounds, the mixture of alcohols, and the three individual alcohols showed the greatest antifungal effect, with significant percent inhibition of 30.5, 17.1, 40.7, 44.5, and 47.5% for the minimal concentration of 12.5 µg/mL. Our findings revealed that the two fungicides used in our study also had 100% efficacy at higher concentrations compared to lower concentrations. This shows that mefentrifluconazole and y19315 had a significant impact on cherry rot at higher concentrations, which is similar to the findings of other researchers who examined the in vivo effects of volatile organic compounds on the phytopathogen F. oxysporum in several cherry tomato studies.

REFERENCES:-

[1]T. Chen, X.-R. Wang, H. Luo, C.-T. Wang, J.-Z. Zhang, and M.-M. Luo, "[Chloroplast DNA trnQ-rps16 variation and genetic structure of nine wild Chinese cherry (Cerasus pseudocerasus Lindl.) populations]," Yi Chuan Hered., vol. 34, no. 11, pp. 1475–1483, Nov. 2012, doi: 10.3724/sp.j.1005.2012.01475.

[2]Q. Zhang, G. Yan, H. Dai, X. Zhang, C. Li, and Z. Zhang, "Characterization of Tomentosa cherry (Prunus tomentosa Thunb.) genotypes using SSR markers and morphological traits," Sci. Hortic., vol. 118, no. 1, pp. 39–47, Sep. 2008, doi: 10.1016/j.scienta.2008.05.022.

[3]D.-O. Kim, H. J. Heo, Y. J. Kim, H. S. Yang, and C. Y. Lee, "Sweet and Sour Cherry Phenolics and Their Protective Effects on Neuronal Cells," J. Agric. Food Chem., vol. 53, no. 26, pp. 9921–9927, Dec. 2005, doi: 10.1021/jf0518599.

[4]D. Spadaro and S. Droby, "Development of biocontrol products for postharvest diseases of fruit: The importance of elucidating the mechanisms of action of yeast antagonists," Trends Food Sci. Technol., vol. 47, pp. 39–49, Jan. 2016, doi: 10.1016/j.tifs.2015.11.003.

[5]J. Børve, L. Sekse, and A. Stensvand, "Cuticular Fractures Promote Postharvest Fruit Rot in Sweet Cherries," Plant Dis., vol. 84, no. 11, pp. 1180–1184, Nov. 2000, doi: 10.1094/PDIS.2000.84.11.1180.

[6]M. P. Tarbath, P. F. Measham, M. Glen, and K. M. Barry, "Host factors related to fruit rot of sweet cherry (Prunus avium L.) caused by Botrytis cinerea," Australas. Plant Pathol., vol. 43, no. 5, pp. 513–522, Sep. 2014, doi: 10.1007/s13313-014-0286-7.

[7]A. Spadoni, A. Ippolito, and S. M. Sanzani, "First report of Stemphylium eturmiunum causing postharvest rot of sweet cherry in Italy," Crop Prot., vol. 132, p. 105112, Jun. 2020, doi: 10.1016/j.cropro.2020.105112.

[8]C. H. Crisosto, G. M. Crisosto, and P. Metheney, "Consumer acceptance of 'Brooks' and 'Bing' cherries is mainly dependent on fruit SSC and visual skin color," Postharvest Biol. Technol., vol. 28, no. 1, pp. 159–167, Apr. 2003, doi: 10.1016/S0925-5214(02)00173-4.

[9]A. A. Hayaloglu and N. Demir, "Physicochemical Characteristics, Antioxidant Activity, Organic Acid and Sugar Contents of 12 Sweet Cherry (Prunus Avium L.) Cultivars Grown in Turkey: Chemical constituents in sweet cherry …," J. Food Sci., vol. 80, no. 3, pp. C564–C570, Mar. 2015, doi: 10.1111/1750-3841.12781.
[10]D. Faniadis, P. D. Drogoudi, and M. Vasilakakis, "Effects of cultivar, orchard elevation, and storage on fruit quality characters of sweet cherry (Prunus avium L.)," Sci. Hortic., vol. 125, no. 3, pp. 301–304, Jun. 2010, doi: 10.1016/j.scienta.2010.04.013.
[11]F. A. Tomás-Barberán et al., "Health Benefits from Pomegranates and Stone Fruit, Including Plums, Peaches, Apricots and Cherries," in Bioactives in Fruit, M. Skinner and D. Hunter, Eds., Oxford, UK: John Wiley and Sons, Ltd, 2013, pp. 125–167. doi: 10.1002/9781118635551.ch7.
[12]K. Damerau, K. Waha, and M. Herrero, "The impact of nutrient-rich food choices on agricultural water-use efficiency," Nat. Sustain., vol. 2, no. 3, pp. 233–241, Mar. 2019, doi: 10.1038/s41893-019-0242-1.
[13]L. M. McCune, C. Kubota, N. R. Stendell-Hollis, and C. A. Thomson, "Cherries and Health: A Review," Crit. Rev. Food Sci. Nutr., vol. 51, no. 1, pp. 1–12, Dec. 2010, doi: 10.1080/10408390903001719.
[14]G. Ferretti, T. Bacchetti, A. Belleggia, and D. Neri, "Cherry Antioxidants: From Farm to Table," Molecules, vol. 15, no. 10, pp. 6993–7005, Oct. 2010, doi: 10.3390/molecules15106993.
[15]D. Valero and M. Serrano, Postharvest biology and technology for preserving fruit quality. Boca Raton: CRC Press, 2010.
[16]D. González-Gómez, M. Lozano, M. F. Fernández-León, M. J. Bernalte, M. C. Ayuso, and A. B. Rodríguez, "Sweet cherry phytochemicals: Identification and characterization by HPLC-DAD/ESI-MS in six sweet-cherry cultivars grown in Valle del Jerte (Spain)," J. Food Compos. Anal., vol. 23, no. 6, pp. 533–539, Sep. 2010, doi: 10.1016/j.jfca.2009.02.008.
[17]B. Girard and T. G. Kopp, "Physicochemical Characteristics of Selected Sweet Cherry Cultivars," J. Agric. Food Chem., vol. 46, no. 2, pp. 471–476, Feb. 1998, doi: 10.1021/jf970646j.
[18]V. Goulas et al., "1H NMR Metabolic Fingerprinting to Probe Temporal Postharvest Changes on Qualitative Attributes and Phytochemical Profile of Sweet Cherry Fruit," Front. Plant Sci., vol. 6, Nov. 2015, doi: 10.3389/fpls.2015.00959.
[19]C. Grafe and M. Schuster, "Physicochemical characterization of fruit quality traits in a German sour cherry collection," Sci. Hortic., vol. 180, pp. 24–31, Dec. 2014, doi: 10.1016/j.scienta.2014.09.047.
[20]L. C. Rodrigues, M. R. Morales, A. J. B. Fernandes, and J. M. Ortiz, "Morphological characterization of sweet and sour cherry cultivars in a germplasm bank at Portugal," Genet. Resour. Crop Evol., vol. 55, no. 4, pp. 593–601, Jun. 2008, doi: 10.1007/s10722-007-9263-0.
[21]N. Papp et al., "Main quality attributes and antioxidants in Hungarian sour cherries: identification of genotypes with enhanced functional properties," Int. J. Food Sci. Technol., vol. 45, no. 2, pp. 395–402, Feb. 2010, doi: 10.1111/j.1365-2621.2009.02168.x.
[22]M. J. Serradilla, A. Hernández, M. López-Corrales, S. Ruiz-Moyano, M. De Guía Córdoba, and A. Martín, "Composition of the Cherry (Prunus avium L. and Prunus cerasus L.; Rosaceae)," in Nutritional Composition of Fruit Cultivars, Elsevier, 2016, pp. 127–147. doi: 10.1016/B978-0-12-408117-8.00006-4.
[23]İ. Damar and A. Ekşi, "Antioxidant capacity and anthocyanin profile of sour cherry (Prunus cerasus L.) juice," Food Chem., vol. 135, no. 4, pp. 2910–2914, Dec. 2012, doi: 10.1016/j.foodchem.2012.07.032.
[24]V. Rakonjac, M. F. Akšić, D. Nikolić, D. Milatović, and S. Čolić, "Morphological characterization of 'Oblačinska' sour cherry by multivariate analysis," Sci. Hortic., vol. 125, no. 4, pp. 679–684, Jul. 2010, doi: 10.1016/j.scienta.2010.05.029.
[25]D. E. Guyer, N. K. Sinha, T.-S. Chang, and J. N. Cash, "PHYSICOCHEMICAL AND SENSORY CHARACTERISTICS OF SELECTED MICHIGAN SWEET CHERRY (Prunus avium L.) CULTIVARS," J. Food Qual., vol. 16, no. 5, pp. 355–370, Oct. 1993, doi: 10.1111/j.1745-4557.1993.tb00121.x.
[26]A. A. Hayaloglu and N. Demir, "Physicochemical Characteristics, Antioxidant Activity, Organic Acid and Sugar Contents of 12 Sweet Cherry (Prunus Avium L.) Cultivars Grown in Turkey: Chemical constituents in sweet cherry …," J. Food Sci., vol. 80, no. 3, pp. C564–C570, Mar. 2015, doi: 10.1111/1750-3841.12781.
[27]V. Usenik, N. Fajt, M. Mikulic-Petkovsek, A. Slatnar, F. Stampar, and R. Veberic, "Sweet Cherry Pomological and Biochemical Characteristics Influenced by Rootstock," J. Agric. Food Chem., vol. 58, no. 8, pp. 4928–4933, Apr. 2010, doi: 10.1021/jf903755b.
[28]A. Wojdyło, P. Nowicka, P. Laskowski, and J. Oszmiański, "Evaluation of Sour Cherry (Prunus cerasus L.) Fruits for Their Polyphenol Content, Antioxidant Properties, and Nutritional Components," J. Agric. Food Chem., vol. 62, no. 51, pp. 12332–12345, Dec. 2014, doi: 10.1021/jf504023z.
[29]M. Schuster, C. Grafe, B. Wolfram, and H. Schmidt, "Cultivars Resulting From Cherry Breeding in Germany," Erwerbs-Obstbau, vol. 56, no. 2, pp. 67–72, Jun. 2014, doi: 10.1007/s10341-014-0204-8.

[30]D. Milatović, J. Milivojević, D. Nikolić, and International Society for Horticultural Science, Eds., Proceedings of the III Balkan Symposium on Fruit Growing, Belgrade, Serbia September 16 - 18, 2015. volume 2. in Acta horticulturae, no. number 1139,2. Leuven: International Society for Horticultural Science (ISHS), 2016.
[31]M. J. Serradilla, A. Martín, S. Ruiz-Moyano, A. Hernández, M. López-Corrales, and M. D. G. Córdoba, "Physicochemical and sensorial characterisation of four sweet cherry cultivars grown in Jerte Valley (Spain)," Food Chem., vol. 133, no. 4, pp. 1551–1559, Aug. 2012, doi: 10.1016/j.foodchem.2012.02.048.
[32]S. Pacifico et al., "Chemical composition, nutritional value and antioxidant properties of autochthonous Prunus avium cultivars from Campania Region," Food Res. Int., vol. 64, pp. 188–199, Oct. 2014, doi: 10.1016/j.foodres.2014.06.020.
[33]C. Bastos et al., "Chemical characterisation and bioactive properties of Prunus avium L.: The widely studied fruits and the unexplored stems," Food Chem., vol. 173, pp. 1045–1053, Apr. 2015, doi: 10.1016/j.foodchem.2014.10.145.
[34]V. Usenik, J. Fabčič, and F. Štampar, "Sugars, organic acids, phenolic composition and antioxidant activity of sweet cherry (Prunus avium L.)," Food Chem., vol. 107, no. 1, pp. 185–192, Mar. 2008, doi: 10.1016/j.foodchem.2007.08.004.
[35]M. J. Serradilla, M. Lozano, M. J. Bernalte, M. C. Ayuso, M. López-Corrales, and D. González-Gómez, "Physicochemical and bioactive properties evolution during ripening of 'Ambrunés' sweet cherry cultivar," LWT - Food Sci. Technol., vol. 44, no. 1, pp. 199–205, Jan. 2011, doi: 10.1016/j.lwt.2010.05.036.
[36]G. Ballistreri, A. Continella, A. Gentile, M. Amenta, S. Fabroni, and P. Rapisarda, "Fruit quality and bioactive compounds relevant to human health of sweet cherry (Prunus avium L.) cultivars grown in Italy," Food Chem., vol. 140, no. 4, pp. 630–638, Oct. 2013, doi: 10.1016/j.foodchem.2012.11.024.
[37]M. Esti, L. Cinquanta, F. Sinesio, E. Moneta, and M. Di Matteo, "Physicochemical and sensory fruit characteristics of two sweet cherry cultivars after cool storage," Food Chem., vol. 76, no. 4, pp. 399–405, Apr. 2002, doi: 10.1016/S0308-8146(01)00231-X.
[38]M. Mitic, M. Obradovic, D. Kostic, R. Micic, and E. Pecev, "Polyphenol content and antioxidant activity of sour cherries from Serbia," Chem. Ind. Chem. Eng. Q., vol. 18, no. 1, pp. 53–62, 2012, doi: 10.2298/CICEQ110701046M.
[39]D. Singh and R. R. Sharma, "Postharvest Diseases of Fruits and Vegetables and Their Management," in Postharvest Disinfection of Fruits and Vegetables, Elsevier, 2018, pp. 1–52. doi: 10.1016/B978-0-12-812698-1.00001-7.
[40]J. P. Rodrigues, C. C. De Souza Coelho, A. G. Soares, and O. Freitas-Silva, "Current technologies to control fungal diseases in postharvest papaya (Carica papaya L.)," Biocatal. Agric. Biotechnol., vol. 36, p. 102128, Sep. 2021, doi: 10.1016/j.bcab.2021.102128.
[41]C. L. Xiao and R. J. Boal, "Preharvest Application of a Boscalid and Pyraclostrobin Mixture to Control Postharvest Gray Mold and Blue Mold in Apples," Plant Dis., vol. 93, no. 2, pp. 185–189, Feb. 2009, doi: 10.1094/PDIS-93-2-0185.
[42]Y. Chen, B. Li, Z. Zhang, and S. Tian, "Pathogenicity Assay of Penicillium expansum on Apple Fruits," BIO-Protoc., vol. 7, no. 9, 2017, doi: 10.21769/BioProtoc.2264.
[43]B. C. Deka, S. Choudhury, A. Bhattacharyya, K. H. Begum, and M. Neog, "POSTHARVEST TREATMENTS FOR SHELF LIFE EXTENSION OF BANANA UNDER DIFFERENT STORAGE ENVIRONMENTS," Acta Hortic., no. 712, pp. 841–850, Jun. 2006, doi: 10.17660/ActaHortic.2006.712.110.
[44]R. Ozkan, J. L. Smilanick, and O. A. Karabulut, "Toxicity of ozone gas to conidia of Penicillium digitatum, Penicillium italicum, and Botrytis cinerea and control of gray mold on table grapes," Postharvest Biol. Technol., vol. 60, no. 1, pp. 47–51, Apr. 2011, doi: 10.1016/j.postharvbio.2010.12.004.
[45]L. Lassois, M. H. Jijakli, M. Chillet, and L. De Lapeyre De Bellaire, "Crown Rot of Bananas: Preharvest Factors Involved in Postharvest Disease Development and Integrated Control Methods," Plant Dis., vol. 94, no. 6, pp. 648–658, Jun. 2010, doi: 10.1094/PDIS-94-6-0648.
[46]A. El Ghaouth, C. L. Wilson, and M. Wisniewski, "Control of Postharvest Decay of Apple Fruit with Candida saitoana and Induction of Defense Responses," Phytopathology®, vol. 93, no. 3, pp. 344–348, Mar. 2003, doi: 10.1094/PHYTO.2003.93.3.344.
[47]Z. Yang, S. Cao, Y. Cai, and Y. Zheng, "Combination of salicylic acid and ultrasound to control postharvest blue mold caused by Penicillium expansum in peach fruit," Innov. Food Sci. Emerg. Technol., vol. 12, no. 3, pp. 310–314, Jul. 2011, doi: 10.1016/j.ifset.2011.04.010.
[48]K. Abeywickrama, C. Wijerathna, N. Rajapaksha, K. Sarananda, and S. Kannangara, "Disease control strategies for extending storage life of papaya (Carica papaya), cultivars 'Red Lady' and 'Rathna,'" Ceylon J. Sci.

Biol. Sci., vol. 41, no. 1, pp. 27–34, Aug. 2012, doi: 10.4038/cjsbs.v41i1.4535.
[49]D. Shtienberg, "Effects of Host Physiology on the Development of Core Rot, Caused by Alternaria alternata , in Red Delicious Apples," Phytopathology®, vol. 102, no. 8, pp. 769–778, Aug. 2012, doi: 10.1094/PHYTO-09-11-0260.
[50]J. M. Gardner, Y. Kono, and J. L. Chandler, "Bioassay and host-selectivity of Alternaria citri toxins affecting rough lemon and mandarins," Physiol. Mol. Plant Pathol., vol. 29, no. 3, pp. 293–304, Nov. 1986, doi: 10.1016/S0048-4059(86)80046-7.
[51]K. K. Jacobi and J. E. Giles, "Quality of `Kensington' mango (Mangifera indica Linn.) fruit following combined vapour heat disinfestation and hot water disease control treatments," Postharvest Biol. Technol., vol. 12, no. 3, pp. 285–292, Dec. 1997, doi: 10.1016/S0925-5214(97)00053-7.
[52]F. Cladera-Olivera, G. R. Caron, A. S. Motta, A. A. Souto, and A. Brandelli, "Bacteriocin-like substance inhibits potato soft rot caused by Erwinia carotovora," Can. J. Microbiol., vol. 52, no. 6, pp. 533–539, Jun. 2006, doi: 10.1139/w05-159.
[53]Z. Volcani, "Bacterial Soft Rot of Avocado Fruit," Nature, vol. 174, no. 4430, pp. 604–605, Sep. 1954, doi: 10.1038/174604b0.
[54]Y. Sm, "Review of Post-Harvest Losses of Fruits and Vegetables," Biomed. J. Sci. Tech. Res., vol. 13, no. 4, Jan. 2019, doi: 10.26717/BJSTR.2019.13.002448.
[55]S. M. Yahaya et al., "The effect of washing with water and detergent on increasing the shelf life of pepper (Capsicum annum) on sale at Rimi and Tarauni markets of Kano State, Nigeria," Dutse J. Pure Appl. Sci., vol. 7, no. 4a, pp. 201–208, Feb. 2022, doi: 10.4314/dujopas.v7i4a.21.
[56]"Review the Extent and Cause of Post Harvest Loss of Fruits and Vegetables in Ethiopia," J. Biol. Agric. Healthc., Jul. 2021, doi: 10.7176/JBAH/11-13-01.
[57]FAO, Ed., Women in agriculture: closing the gender gap for development. in The state of food and agriculture, no. 2010/11. Rome: FAO, 2011.
[58]C. N. Williams, J. O. Uzo, and W. T. H. Peregrine, Vegetable production in the tropics. in Intermediate tropical agriculture series. Essex, England: Longman Scientific and Technical, 1991.
[59]S. M. Yahaya et al., "A Study of Pathogenic Fungi Causing Post Harvest Losses of Pineapple Sold at Wudil and Yan Lemo Markets of Kano State," J. Exp. Agric. Int., pp. 1–6, Jun. 2019, doi: 10.9734/jeai/2019/v38i230297.
[60]G. A. Manganaris, I. F. Ilias, M. Vasilakakis, and I. Mignani, "The effect of hydrocooling on ripening related quality attributes and cell wall physicochemical properties of sweet cherry fruit (Prunus avium L.)," Int. J. Refrig., vol. 30, no. 8, pp. 1386–1392, Dec. 2007, doi: 10.1016/j.ijrefrig.2007.04.001.
[61]"Sweet cherry quality in the horticultural production chain," Stewart Postharvest Rev., vol. 2, no. 6, pp. 1–9, 2006, doi: 10.2212/spr.2006.6.2.
[62]H. M. Díaz-Mula et al., "THE FUNCTIONAL PROPERTIES OF SWEET CHERRY AS A NEW CRITERION IN A BREEDING PROGRAM," Acta Hortic., no. 839, pp. 275–280, Jul. 2009, doi: 10.17660/ActaHortic.2009.839.34.
[63]M. Schreiner and S. Huyskens-Keil, "Phytochemicals in Fruit and Vegetables: Health Promotion and Postharvest Elicitors," Crit. Rev. Plant Sci., vol. 25, no. 3, pp. 267–278, Jul. 2006, doi: 10.1080/07352680600671661.
[64]S. Y. Wang, "EFFECT OF PRE-HARVEST CONDITIONS ON ANTIOXIDANT CAPACITY IN FRUITS," Acta Hortic., no. 712, pp. 299–306, Jun. 2006, doi: 10.17660/ActaHortic.2006.712.33.
[65]M. Serrano, F. Guillén, D. Martínez-Romero, S. Castillo, and D. Valero, "Chemical Constituents and Antioxidant Activity of Sweet Cherry at Different Ripening Stages," J. Agric. Food Chem., vol. 53, no. 7, pp. 2741–2745, Apr. 2005, doi: 10.1021/jf0479160.
[66]B. Mozetič, M. Simčič, and P. Trebše, "Anthocyanins and hydroxycinnamic acids of Lambert Compact cherries (Prunus avium L.) after cold storage and 1-methylcyclopropene treatment," Food Chem., vol. 97, no. 2, pp. 302–309, Jul. 2006, doi: 10.1016/j.foodchem.2005.04.018.
[67]B. Gonçalves et al., "Effect of Ripeness and Postharvest Storage on the Phenolic Profiles of Cherries (Prunus avium L.)," J. Agric. Food Chem., vol. 52, no. 3, pp. 523–530, Feb. 2004, doi: 10.1021/jf030595s.
[68]T. Mahmood, F. Anwar, M. Abbas, and N. Saari, "Effect of Maturity on Phenolics (Phenolic Acids and Flavonoids) Profile of Strawberry Cultivars and Mulberry Species from Pakistan," Int. J. Mol. Sci., vol. 13, no. 4, pp. 4591–4607, Apr. 2012, doi: 10.3390/ijms13044591.
[69]M. J. Giménez et al., "Quality and antioxidant properties on sweet cherries as affected by preharvest salicylic and acetylsalicylic acids treatments," Food Chem., vol. 160, pp. 226–232, Oct. 2014, doi: 10.1016/j.foodchem.2014.03.107.

[70]Z. Aryanpooya, G. H. Davarynejad, and L. Lakatos, "POSTHARVEST QUALITY OF SOUR CHERRY FRUITS SPRAYED BY ETHEPHON," Acta Hortic., no. 1020, pp. 121–126, Feb. 2014, doi: 10.17660/ActaHortic.2014.1020.15.
[71]S. Kondo and K. Inoue, "Abscisic acid (ABA) and 1-aminocyclopropane-l-carboxylic acid (ACC) content during growth of 'Satohnishiki' cherry fruit, and the effect of ABA and ethephon application on fruit quality," J. Hortic. Sci., vol. 72, no. 2, pp. 221–227, Jan. 1997, doi: 10.1080/14620316.1997.11515509.
[72]H. Luo et al., "The Role of ABA in the Maturation and Postharvest Life of a Nonclimacteric Sweet Cherry Fruit," J. Plant Growth Regul., vol. 33, no. 2, pp. 373–383, Jun. 2014, doi: 10.1007/s00344-013-9388-7.
[73]A. Martínez-Esplá, P. J. Zapata, D. Valero, C. García-Viguera, S. Castillo, and M. Serrano, "Preharvest Application of Oxalic Acid Increased Fruit Size, Bioactive Compounds, and Antioxidant Capacity in Sweet Cherry Cultivars (Prunus avium L.)," J. Agric. Food Chem., vol. 62, no. 15, pp. 3432–3437, Apr. 2014, doi: 10.1021/jf500224g.
[74] "Antioxidant compounds in fruits and vegetables and changes during postharvest storage and processing," Stewart Postharvest Rev., vol. 7, no. 1, pp. 1–10, 2011, doi: 10.2212/spr.2011.1.1.
[75]M. Serrano et al., "Maturity Stage at Harvest Determines the Fruit Quality and Antioxidant Potential after Storage of Sweet Cherry Cultivars," J. Agric. Food Chem., vol. 57, no. 8, pp. 3240–3246, Apr. 2009, doi: 10.1021/jf803949k.
[76]D. Valero et al., "Postharvest Treatments with Salicylic Acid, Acetylsalicylic Acid or Oxalic Acid Delayed Ripening and Enhanced Bioactive Compounds and Antioxidant Capacity in Sweet Cherry," J. Agric. Food Chem., vol. 59, no. 10, pp. 5483–5489, May 2011, doi: 10.1021/jf200873j.
[77]M. S. Fernandez-Panchon, D. Villano, A. M. Troncoso, and M. C. Garcia-Parrilla, "Antioxidant Activity of Phenolic Compounds: From In Vitro Results to In Vivo Evidence," Crit. Rev. Food Sci. Nutr., vol. 48, no. 7, pp. 649–671, Jul. 2008, doi: 10.1080/10408390701761845.
[78]F. Güneş, "Medicinal plants used in the Uzunköprü district of Edirne, Turkey," Acta Soc. Bot. Pol., vol. 86, no. 4, Dec. 2017, doi: 10.5586/asbp.3565.
[79]H. Wang, M. G. Nair, G. M. Strasburg, A. M. Booren, and J. I. Gray, "Antioxidant Polyphenols from Tart Cherries (Prunuscerasus)," J. Agric. Food Chem., vol. 47, no. 3, pp. 840–844, Mar. 1999, doi: 10.1021/jf980936f.
[80]D. A. J. Connolly, "Efficacy of a tart cherry juice blend in preventing the symptoms of muscle damage * Commentary 1 * Commentary 2," Br. J. Sports Med., vol. 40, no. 8, pp. 679–683, Apr. 2006, doi: 10.1136/bjsm.2005.025429.
[81]W. R. Pigeon, M. Carr, C. Gorman, and M. L. Perlis, "Effects of a Tart Cherry Juice Beverage on the Sleep of Older Adults with Insomnia: A Pilot Study," J. Med. Food, vol. 13, no. 3, pp. 579–583, Jun. 2010, doi: 10.1089/jmf.2009.0096.
[82]S.-Y. Kang, N. P. Seeram, M. G. Nair, and L. D. Bourquin, "Tart cherry anthocyanins inhibit tumor development in ApcMin mice and reduce proliferation of human colon cancer cells," Cancer Lett., vol. 194, no. 1, pp. 13–19, May 2003, doi: 10.1016/S0304-3940(02)00583-9.
[83]S. Sansavini and S. Lugli, "SWEET CHERRY BREEDING PROGRAMS IN EUROPE AND ASIA," Acta Hortic., no. 795, pp. 41–58, Aug. 2008, doi: 10.17660/ActaHortic.2008.795.1.
[84]G. Bujdosó and K. Hrotkó, "Cherry production.," in Cherries: botany, production and uses, J. Quero-García, A. Lezzoni, J. Puławska, and G. Lang, Eds., Wallingford: CABI, 2017, pp. 1–13. doi: 10.1079/9781780648378.0001.
[85]R. O. Olatinwo, A. M. C. Schilder, and A. N. Kravchenko, "Incidence and Causes of Postharvest Fruit Rot in Stored Michigan Cranberries," Plant Dis., vol. 88, no. 11, pp. 1277–1282, Nov. 2004, doi: 10.1094/PDIS.2004.88.11.1277.
[86]D. G. Alvindia, T. Kobayashi, K. T. Natsuaki, and S. Tanda, "Inhibitory influence of inorganic salts on banana postharvest pathogens and preliminary application to control crown rot," J. Gen. Plant Pathol., vol. 70, no. 1, pp. 61–65, Feb. 2004, doi: 10.1007/s10327-003-0084-5.
[87]R. Kristensen, M. Torp, B. Kosiak, and A. Holst-Jensen, "Phylogeny and toxigenic potential is correlated in Fusarium species as revealed by partial translation elongation factor 1 alpha gene sequences," Mycol. Res., vol. 109, no. 2, pp. 173–186, Feb. 2005, doi: 10.1017/S0953756204002114.
[88]K. Kouame, K. Abo, E. Dick, E. Bomisso, D. Kone, and S. Ake, "Artificial wounds implication for the development of mango (Mangifera Indica L. Anacardiaceae) fruit disease caused by Colletotrichum gloeosporioïdes (Penz.) Sacc. (Glomerellaceae)," Int. J. Biol. Chem. Sci., vol. 4, no. 5, Apr. 2011, doi: 10.4314/ijbcs.v4i5.65579.
[89]Z. Zhu, L. Zheng, L. Pan, T. Hsiang, and J. Huang, "Identification and Characterization of Fusarium Species

Associated with Wilt of Eleocharis dulcis (Chinese water chestnut) in China," Plant Dis., vol. 98, no. 7, pp. 977–987, Jul. 2014, doi: 10.1094/PDIS-08-13-0805-RE.
[90]J. F. Leslie and B. A. Summerell, Eds., The Fusarium Laboratory Manual, 1st ed. Wiley, 2006. doi: 10.1002/9780470278376.
[91]H. L. Barnett and B. B. Hunter, Illustrated genera of imperfect fungi, 4th ed. St. Paul, Minn: APS Press, 1998.
[92]C. Booth, The genus Fusarium. Farnham Royal: Commonwealth Agricultural Bureaux [for the] Commonwealth Mycological Institute, 1971.
[93]G. P. Chamuris and D. Counterman, "Dung-Inhabiting Fungi in the High School Biology Laboratory," Am. Biol. Teach., vol. 61, no. 8, pp. 605–609, Oct. 1999, doi: 10.2307/4450776.
[94]L. Bettucci, I. Malvarez, J. Dupont, E. Bury, and M.-F. Roquebert, "Paraná river delta wetlands soil microfungi," Pedobiologia, vol. 46, no. 6, pp. 606–623, Jan. 2002, doi: 10.1078/0031-4056-00163.
[95]F. M. Dugan, The identification of fungi: an illustrated introduction with keys, glossary, and guide to literature. St. Paul, Minn: American Phytopathological Society, 2006.
[96]Wm. B. Cooke, "Terminology of the fungi imperfecti," Mycopathol. Mycol. Appl., vol. 53, no. 1–4, pp. 45–67, Aug. 1974, doi: 10.1007/BF02127197.
[97]D. C. Erwin and O. K. Ribeiro, Phytophthora diseases worldwide. St. Paul, Minn: APS Press, 1996.
[98]P. E. Nelson, T. A. Toussoun, and W. F. O. Marasas, Fusarium species: an illustrated manual for identification. University Park: Pennsylvania State University Press, 1983.
[99]Y. J. Zhang, S. Zhang, X. Z. Liu, H. A. Wen, and M. Wang, "A simple method of genomic DNA extraction suitable for analysis of bulk fungal strains: Fungal DNA isolation by thermolysis," Lett. Appl. Microbiol., p. no-no, May 2010, doi: 10.1111/j.1472-765X.2010.02867.x.
[100]T. J. White, T. Bruns, S. Lee, and J. Taylor, "AMPLIFICATION AND DIRECT SEQUENCING OF FUNGAL RIBOSOMAL RNA GENES FOR PHYLOGENETICS," in PCR Protocols, Elsevier, 1990, pp. 315–322. doi: 10.1016/B978-0-12-372180-8.50042-1.
[101]K. O'Donnell, H. C. Kistler, E. Cigelnik, and R. C. Ploetz, "Multiple evolutionary origins of the fungus causing Panama disease of banana: Concordant evidence from nuclear and mitochondrial gene genealogies," Proc. Natl. Acad. Sci., vol. 95, no. 5, pp. 2044–2049, Mar. 1998, doi: 10.1073/pnas.95.5.2044.
[102]K. Y. Leyva-Madrigal et al., "Fusarium Species from the Fusarium fujikuroi Species Complex Involved in Mixed Infections of Maize in Northern Sinaloa, Mexico," J. Phytopathol., vol. 163, no. 6, pp. 486–497, Jun. 2015, doi: 10.1111/jph.12346.
[103]C. W. Wang and Y. Wang, "First Report of Postharvest Fruit Rot Caused by Fusarium equiseti on Stored Cerasus pseudocerasus in China," Plant Dis., vol. 101, no. 6, pp. 1041–1041, Jun. 2017, doi: 10.1094/PDIS-10-16-1451-PDN.
[104]A. Duggal, M. T. Dumas, R. S. Jeng, and M. Hubbes, "[No title found]," Mycopathologia, vol. 140, no. 1, pp. 35–49, 1997, doi: 10.1023/A:1006816025113.
[105]C. Wang, Y. Wang, L. Wang, X. Li, M. Wang, and J. Wang, "Fusarium species causing postharvest rot on Chinese cherry in China," Crop Prot., vol. 141, p. 105496, Mar. 2021, doi: 10.1016/j.cropro.2020.105496.
[106]I. M. Singha, Y. Kakoty, B. G. Unni, J. Das, and M. C. Kalita, "Identification and characterization of Fusarium sp. using ITS and RAPD causing fusarium wilt of tomato isolated from Assam, North East India," J. Genet. Eng. Biotechnol., vol. 14, no. 1, pp. 99–105, Jun. 2016, doi: 10.1016/j.jgeb.2016.07.001.
[107] B. A. Summerell, M. H. Laurence, E. C. Y. Liew, and J. F. Leslie, "Biogeography and phylogeography of Fusarium: a review," Fungal Divers., vol. 44, no. 1, pp. 3–13, Oct. 2010, doi: 10.1007/s13225-010-0060-2.
[108]L. Shan et al., "A loop-mediated isothermal amplification (LAMP) assay for the rapid detection of toxigenic Fusarium temperatum in maize stalks and kernels," Int. J. Food Microbiol., vol. 291, pp. 72–78, Feb. 2019, doi: 10.1016/j.ijfoodmicro.2018.11.021.
[109]X. Chang et al., "Identification of Fusarium species associated with soybean root rot in Sichuan Province, China," Eur. J. Plant Pathol., vol. 151, no. 3, pp. 563–577, Jul. 2018, doi: 10.1007/s10658-017-1410-7.
[110]B. G. Nayyar et al., "Identification and pathogenicity of Fusarium species associated with sesame (Sesamum indicum L.) seeds from the Punjab, Pakistan," Physiol. Mol. Plant Pathol., vol. 102, pp. 128–135, Apr. 2018, doi: 10.1016/j.pmpp.2018.02.001.
[111] H. Suga et al., "A single nucleotide polymorphism in the translation elongation factor 1α gene correlates with the ability to produce fumonisin in Japanese Fusarium fujikuroi," Fungal Biol., vol. 118, no. 4, pp. 402–412, Apr. 2014, doi: 10.1016/j.funbio.2014.02.005.
[112]K. R. Kintega, P. E. Zida, V. W. Tarpaga, P. Sankara, and P. Sereme, "Identification of Fusarium Species Associated with Onion (and lt;iand gt;Allium cepaand lt;/iand gt; L.) Plants in Field in Burkina Faso," Adv.

Biosci. Biotechnol., vol. 11, no. 03, pp. 94–110, 2020, doi: 10.4236/abb.2020.113008.
[113]Y. M. Guan, J. C. Deng, Y. Y. Ma, Y. Li, and Y. Y. Zhang, "Seed-Associated Fungal Diversity and the Molecular Identification of Fusarium with Potential Threat to Ginseng (Panax ginseng) in China," Plant Dis., vol. 104, no. 2, pp. 330–339, Feb. 2020, doi: 10.1094/PDIS-09-19-1817-RE.
[114]M. Moumni, M. B. Allagui, V. Mancini, S. Murolo, N. Tarchoun, and G. Romanazzi, "Morphological and Molecular Identification of Seedborne Fungi in Squash (Cucurbita maxima , Cucurbita moschata)," Plant Dis., vol. 104, no. 5, pp. 1335–1350, May 2020, doi: 10.1094/PDIS-04-19-0741-RE.
[115]F. R. Flores-de La Rosa, E. De Luna, J. Adame-García, L. G. Iglesias-Andreu, and M. Luna-Rodríguez, "Phylogenetic position and nucleotide diversity of Fusarium oxysporum f. sp. vanillae worldwide based on translation elongation factor 1α sequences," Plant Pathol., vol. 67, no. 6, pp. 1278–1285, Aug. 2018, doi: 10.1111/ppa.12847.
[116]Y. Wang et al., "Development of loop-mediated isothermal amplification (LAMP) assay for rapid detection of Fusarium proliferatum causing ear and kernel rot on maize," Crop Prot., vol. 132, p. 105142, Jun. 2020, doi: 10.1016/j.cropro.2020.105142.
[117]K. L. Chadha and R. K. Pal, Eds., Managing postharvest quality and losses in horticultural crops. New Delhi: Daya Publishing House, 2015.
[118]L. M. Kawchuk, J. D. Holley, D. R. Lynch, and R. M. Clear, "Resistance to thiabendazole and thiophanate-methyl in canadian isolates ofFusarium sambucinum andHelminthosporium solani," Am. Potato J., vol. 71, no. 3, pp. 185–192, Mar. 1994, doi: 10.1007/BF02849053.
[119]M. R. Siegel, "Sterol-Inhibiting Fungicides: Effects on Sterol Biosynthesis and Sites of Action," Plant Dis., vol. 65, no. 12, p. 986, 1981, doi: 10.1094/PD-65-986.
[120]L. C. Davidse, "Benzimidazole Fungicides: Mechanism of Action and Biological Impact," Annu. Rev. Phytopathol., vol. 24, no. 1, pp. 43–65, Sep. 1986, doi: 10.1146/annurev.py.24.090186.000355.
[121]R. Estrada Jr, N. C. Gudmestad, V. V. Rivera, and G. A. Secor, "Fusarium graminearum as a dry rot pathogen of potato in the USA: prevalence, comparison of host isolate aggressiveness and factors affecting aetiology: Dry rot of potato caused by Fusarium graminearum," Plant Pathol., vol. 59, no. 6, pp. 1114–1120, Dec. 2010, doi: 10.1111/j.1365-3059.2010.02343.x.
[122]D. Ma et al., "Evaluation of Sensitivity and Resistance Risk of Corynespora cassiicola to Isopyrazam and Mefentrifluconazole," Plant Dis., vol. 104, no. 11, pp. 2779–2785, Nov. 2020, doi: 10.1094/PDIS-02-20-0384-RE.
[123]M. Masiello, S. Somma, V. Ghionna, A. F. Logrieco, and A. Moretti, "In Vitro and in Field Response of Different Fungicides against Aspergillus flavus and Fusarium Species Causing Ear Rot Disease of Maize," Toxins, vol. 11, no. 1, p. 11, Jan. 2019, doi: 10.3390/toxins11010011.
[124]F. Tini, G. Beccari, A. Onofri, E. Ciavatta, D. M. Gardiner, and L. Covarelli, "Fungicides may have differential efficacies towards the main causal agents of Fusarium head blight of wheat," Pest Manag. Sci., vol. 76, no. 11, pp. 3738–3748, Nov. 2020, doi: 10.1002/ps.5923.
[125]Y. M. Medina-Romero, G. Roque-Flores, and M. L. Macías-Rubalcava, "Volatile organic compounds from endophytic fungi as innovative postharvest control of Fusarium oxysporum in cherry tomato fruits," Appl. Microbiol. Biotechnol., vol. 101, no. 22, pp. 8209–8222, Nov. 2017, doi: 10.1007/s00253-017-8542-8.
[126]L. Gao and G. Mazza, "Characterization, Quantitation, and Distribution of Anthocyanins and Colorless Phenolics in Sweet Cherries," J. Agric. Food Chem., vol. 43, no. 2, pp. 343–346, Feb. 1995, doi: 10.1021/jf00050a015.