EVALUATION OF OOMYCETE-TARGETED FUNGICIDES FOR CONTROLLING DOWNY MILDEW IN VEGETABLE CROPS

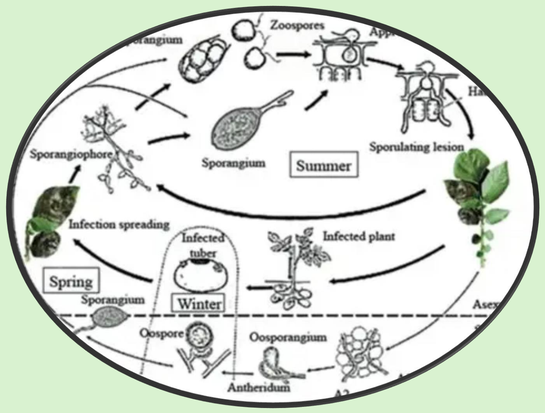
Biljana Kovacevik, Sasa Mitrev, Emilija Arsov, Dusan Spasov, Biljana Atanasova, Marija Taskova Kukutanov, Natalija Markova Ruzdik
Faculty of Agriculture, Goce Delcev University, Stip, Goce Delcev Str. No. 89, PO box 201, Stip 2000, Republic of North Macedonia
Contact author: biljana.kovacevik@ugd.edu.mk

Abstract

Currently, 56 distinct fungicide modes of action are recognized and classified by the Fungicide Resistance Action Committee (FRAC), including compounds with unknown mechanisms. This diversity supports effective resistance management. However, the availability of fungicides is often limited by regional registration and economic factors, restricting their use in certain crops. Consequently, key crop–pathogen systems may have few control options. Furthermore, many fungicides exhibit a narrow spectrum of activity, with a large proportion targeting specific pathogen groups such as oomycetes or even single species. Oomycetes are phylogenetically distinct from true fungi such as phytopathogenic Deuteromycetes, Ascomycetes, and Basidiomycetes, exhibiting significant biological and biochemical differences that influence their sensitivity to fungicidal compounds. These pathogens are responsible for severe diseases in economically important crops, causing substantial yield and quality losses. In this study, the efficacy of oomycete-targeted fungicides against downy mildew are evaluated, with emphasis on compounds from diverse modes of action (MoA) such as antitubulin agents affecting microtubule assembly, compounds interfering with cytoskeleton-associated proteins, carboxylic acid amides (CAAs) targeting cell wall biosynthesis, and novel inhibitors of oxysterol-binding proteins. Field and controlled environment assessments demonstrated that fungicides with specific activity against oomycetes provide effective disease suppression, especially when applied preventively or during early stages of infection. Variability in efficacy among MoA groups highlights the importance of strategic selection and rotation of fungicides to delay resistance development. The integration of these target fungicides within an integrated disease management framework is essential for sustainable control of downy mildew in crop production systems.

Introduction

Oomycetes are a distinct group of eukaryotic microorganisms commonly referred to as “water molds” because many species thrive in moist or aquatic environments. They are widely distributed in nature and play important ecological roles, particularly as decomposers that break down organic matter and recycle nutrients. At the same time, numerous oomycetes are notorious pathogens of plants and some animals, causing devastating diseases that affect agriculture, forestry, and natural ecosystems, often leading to serious economic losses. Despite their resemblance to fungi in growth form and lifestyle, oomycetes are not true fungi. One of the key differences lies in their cell wall composition: oomycetes possess cell walls made primarily of cellulose, whereas true fungi have cell walls composed of chitin. This fundamental distinction reflects their separate evolutionary origins. Their life cycle further highlights these differences. Oomycetes are predominantly diploid organisms, meaning their cells contain two sets of chromosomes throughout most of their life cycle. Oomycetes comprise major plant pathogens within the orders Saprolegniales, Pythiales, Peronosporales (“downy mildews” including species of Plasmopara, Phytophthora, Peronospora, Pseudoperonospora, Bremia), and Sclerosporales.



Resistance Development to Site-Specific Fungicides in Oomycete Pathogens

Management of diseases caused by oomycetes still depends largely on chemical control, with fungicides belonging to 16 different mode-of-action groups. Among these, phenylamides (PAs), quinone outside inhibitors (QoIs), carboxylic acid amides (CAAs), and multisite inhibitors are the most commonly applied. However, the extensive use of single-site fungicides has led to the widespread development of resistance in many oomycete pathogens. Resistance to phenylamides targeting RNA polymerase I has been reported in most oomycete species and is now linked to the Y382F mutation in the RNApolI gene. QoIs act by disrupting electron transport at complex III in the mitochondrial respiration chain; resistance is primarily associated with mutations in the cytochrome b (cyt b) gene, notably G143A in Plasmopara viticola and F129L in Pythium species. In contrast, resistance to QoIs has not been confirmed in Phytophthora species. Carboxylic acid amides inhibit cellulose biosynthesis, and resistance in field populations has been connected to mutations in the CesA3 gene, such as G1105S/V in P. viticola and G1105V/W in Pseudoperonospora cubensis. Overall, these examples highlight the strong selection pressure exerted by site-specific fungicides and the need for resistance management strategies.

Oomycete Targeted Fungicides

Fungicides used to control oomycete pathogens represent a diverse group of active substances specifically designed to interfere with key biological processes unique to these organisms. Oomycetes, although fungus-like, differ significantly from true fungi in their cell wall composition, metabolic pathways, and genetic makeup, which has enabled the development of selective fungicides with distinct modes of action. One of the earliest and most widely used groups are the phenylamides (PAs), Quinone outside inhibitors (QoIs), Carboxylic acid amides (CAAs), Cyanoacetamide oximes, Multisite fungicides, etc.

Phenylamides (PAs)

Phenylamides (PAs), such as metalaxyl and mefenoxam act systemically and inhibit RNA polymerase I, thereby blocking ribosomal RNA synthesis and protein production. Their high specificity and curative activity made them very effective; however, resistance developed rapidly in many oomycete populations due to single-point mutations, significantly reducing their long-term reliability.

Quinone Outside Inhibitors (QoIs)

QoIs, also known as strobilurins, target mitochondrial respiration by binding to the cytochrome bc1 complex (complex III), disrupting ATP production. Although highly effective against a broad range of pathogens, their activity against oomycetes is more limited. Resistance development, driven by mutations in the cytochrome b gene (e.g., G143A, F129L), is a well-documented issue, although some genera, such as Phytophthora, show lower frequencies of resistance.

Carboxylic Acid Amides (CAAs)

CAAs, such as mandipropamid and dimethomorph inhibit cellulose synthase, a critical enzyme in oomycete cell wall biosynthesis. Since cellulose is a major structural component of oomycete cell walls (unlike chitin in true fungi), CAAs exhibit high specificity. Resistance has been associated with mutations in the CesA3 gene, but it generally develops more slowly compared to older fungicide groups.

Cyanoacetamide Oximes

Cyanoacetamide oximes (e.g., cymoxanil) and oxathiapiprolin represent newer modes of action. Oxathiapiprolin, in particular, targets oxysterol-binding proteins (OSBP), disrupting lipid transport and membrane function. It is highly potent at low doses and effective against multiple oomycete species, but due to its single-site mode of action, resistance management is critical.

Multisite Fungicides

Multisite fungicides, such as copper compounds and mancozeb, remain essential in oomycete control programs. These compounds affect multiple metabolic pathways simultaneously, which greatly reduces the risk of resistance development. Although they are less specific and often less systemic, they are widely used in combination with single-site fungicides to delay resistance.