

## Progress in research of seed germination inhibitors- A review article



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### Abstract:

Germination inhibitors substances that prevent the germination of seeds, spores, and other plant reproductive structures are widespread across the plant kingdom. These include compounds such as phenols, cyanides, alkaloids, essential oils, and amino acids. Based on their effects, these inhibitors can be categorized into **germination destructors** and **germination retarders**. Germination destructors damage the seed's morphology, structure, or physiological function, while germination retarders are more closely associated with seed dormancy. When applied at appropriate dosages, exogenous retarders can artificially extend the dormancy period of non-dormant or short-lived seeds, offering valuable potential for post-harvest preservation and long-term germplasm storage. In contrast, germination destructors highly effective but non-selective compounds have promising applications in the development of eco-friendly herbicides. Current research on germination inhibitors faces challenges, such as identifying specific endogenous inhibitory substances, determining the minimum concentration required to induce dormancy, and assessing whether exogenous applications cause physiological damage to seeds. Future research should focus on identifying and tracking germination inhibitors, elucidating their mechanisms of action at both the biochemical and molecular levels, and exploring innovative applications for both destructors and retarders.

**Keywords:** seed, germination inhibitor, germination retarder, germination destructor, dormancy

### 1. Introduction

Seeds are fundamental to agroforestry, serving as the starting point for plant growth and development and playing a critical role in the plant life cycle [1]. Seed germination is initiated when seeds absorb water, activating metabolic processes that eventually lead to the emergence of the radicle through the seed coat [2,3]. This process is regulated by various plant hormones—some promote germination, such as gibberellins [4,5], while others, like abscisic acid, inhibit germination by maintaining seed dormancy [6–8]. Additionally, certain chemical compounds found in different seed components—including the fleshy or dry pericarp, endosperm, seed coat, and embryo—can also suppress germination [9].

The presence of germination inhibitors is a major factor contributing to seed dormancy, particularly in seeds with physiological dormancy (PD) [10,11]. These inhibitors, often produced by the plant itself, are closely linked to the regulation of dormancy [12]. Therefore, a thorough understanding of germination inhibitors is essential for improving crop production techniques, conserving germplasm resources, and aiding in the protection and restoration of endangered plant species.

Although several germination inhibitors have been identified, comprehensive and systematic research on their nature and function remains in the early

stages. This paper introduces a classification framework for germination inhibitors based on current scientific advances and highlights key research challenges—focusing on qualitative identification, quantitative analysis, and experimental methodologies. These insights aim to provide a valuable reference for ongoing and future studies in seed science.

### 2. Definition of Germination Inhibitors

Germination inhibitors are substances present in plants that delay or suppress the germination of seeds. According to Evenari [13], it is often difficult to determine whether these substances merely delay growth or actively harm the plant, so the term “germination inhibitor” is broadly used to describe any compound that prevents or delays seed germination.

These inhibitors are not limited to angiosperms. For instance, butylated hydroxytoluene (BHT), found in the endosperm and embryo of *Pistacia chinensis* seeds, has been shown to inhibit both seed germination and cotyledon elongation [14]. Germination inhibitors are also present in gymnosperms, cryptogams, and other types of reproductive structures. For example, fenugreek spores are unable to germinate within their own spore pool [13,15].

Furthermore, Bingöl et al. [16] reported that ethanol extracts from *Xanthoparmelia somloensis* (gyelnik) significantly inhibited tomato seed germination. Recent studies have also identified non-plant-based compounds with germination-inhibiting properties. One such example is trimethylbutenolide (TMB), a smoke-derived lactone [17], which has been shown to strongly inhibit the germination of several weed species, including fleabane, hairy wild lettuce, bugweed, spilanthes, and fumeflower [18]. The successful large-scale synthesis of TMB [19] has broadened the traditional scope of germination inhibitors beyond plant-derived substance

### 3. Classification of Germination Inhibitors

#### Classification by Function

Kockemann [20] categorized germination inhibitors into two functional groups:

1. **Seed-based inhibitors** – Compounds located within seeds that are often sensitive to light and other environmental factors.
2. **Fruit-flesh-based inhibitors** – Substances found in the fleshy parts of fruit, whose physical and chemical characteristics remain poorly understood.

As research progresses, an increasing variety of chemical compounds have been identified as germination inhibitors. These include hydrogen cyanide, ammonia, ethylene, mustard oil, organic acids, unsaturated lactones, aldehydes, aromatic oils, and various alkaloids.

In terms of origin, germination inhibitors can be classified as **endogenous** or **exogenous**:

- **Endogenous inhibitors** are produced internally by the plant itself.
- **Exogenous inhibitors** originate from external sources, such as other plants, organisms, or synthetic processes. Evenari [13] initially defined exogenous inhibitors as those produced by neighboring plants or seeds. However, with

advancements in synthetic chemistry, the definition has expanded to include all inhibitors not synthesized by the plant in question.

Another classification is based on the effect on seed viability:

1. **Germination retarders** – These delay germination temporarily. Seeds exposed to these inhibitors can resume normal germination when the inhibitory condition is removed or altered.
2. **Germination destructors** – These cause irreversible physiological damage to seeds, ultimately preventing them from germinating.
3. Most germination retarders are naturally occurring compounds produced by dormant seeds under natural conditions. Examples include abscisic acid (ABA) and 1,2,3-benzenetriol, which are found in *Cercis chinensis* seeds and play a role in regulating seed dormancy [21]. Treatments such as warm water soaking, gibberellin application, or cold stratification can reduce or neutralize the effects of these inhibitors, thereby restoring the seeds' ability to germinate normally [22,23].
4. For instance, coumarin inhibits the germination of *Brassica parachinensis* and *Oryza sativa* by promoting ABA synthesis and reducing the accumulation of reactive oxygen species (ROS). It achieves this by enhancing the activities of antioxidant enzymes such as superoxide dismutase (SOD) and peroxidase (POD). However, this inhibition can be reversed by applying exogenous gibberellins, such as GA<sub>4</sub> or a combination of GA<sub>4</sub>+7 [24,25].
5. Since germination retarders are closely linked to the regulation of seed dormancy, they can also be applied externally to extend the dormancy period of non-dormant or short-lived seeds. This makes them highly valuable for post-harvest seed preservation and for the long-term storage of germplasm resources.

Classification of Germination Inhibitors by Chemical Structure

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| Chemical Class | Name of Germination Inhibitor | Locations of Existence             | Form of Existence                                       | Particular Plants Affected                          | Inhibitory Effects                             |
|----------------|-------------------------------|------------------------------------|---------------------------------------------------------|-----------------------------------------------------|------------------------------------------------|
| Phenols        | Caffeic acid                  | Seeds, fruits, other plant tissues | Free or conjugated with sugars as glucosides and esters | <i>Cucumis melo</i>                                 | Retarder – delayed germination                 |
|                |                               |                                    |                                                         | <i>Asparagus</i>                                    | Destructor – identified as inhibitory compound |
|                | Chlorogenic acid              | -                                  | -                                                       | <i>Sida spinosa</i> , <i>Sorghum bicolor</i>        | Destructor – slightly reduced germination      |
|                |                               |                                    |                                                         | <i>Amaranthus retroflexus</i> , <i>Sida spinosa</i> | Retarder – delayed germination                 |
|                |                               |                                    |                                                         | <i>Arabidopsis thaliana</i>                         | Destructor – reduced                           |

|                   |                       |       |                                    |                                                                                                                                                                      |                                                                                |
|-------------------|-----------------------|-------|------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------|
|                   |                       |       |                                    |                                                                                                                                                                      | germination and radicle elongation                                             |
|                   |                       |       |                                    | <i>Oryza sativa</i>                                                                                                                                                  | Retarder – mediated by ABA catabolism suppression and reduced ROS              |
|                   |                       |       |                                    | <i>Brassica parachinensis</i>                                                                                                                                        | Retarder – decreased GA <sub>4</sub> and ROS production                        |
|                   | p-Coumaric acid       | -     | -                                  | <i>Zea mays</i>                                                                                                                                                      | Destructor – significantly decreased germination                               |
|                   | Ferulic acid          | -     | -                                  | <i>Gossypium hirsutum</i>                                                                                                                                            | Destructor – significantly decreased germination                               |
|                   | Fumaric acid          | -     | -                                  | <i>Sida spinosa</i>                                                                                                                                                  | Destructor – slightly reduced germination                                      |
|                   | Gallic acid           | -     | -                                  | <i>Cucumis sativus</i>                                                                                                                                               | Destructor – reduced germination rate, radicle/hypocotyl growth, seedling mass |
|                   | Hydrocinnamic acid    | -     | -                                  | <i>Amaranthus retroflexus</i>                                                                                                                                        | Retarder – delayed germination                                                 |
|                   | Pyrocatechol          | -     | -                                  | <i>Amaranthus retroflexus</i>                                                                                                                                        | Retarder – delayed germination                                                 |
|                   | p-Hydroxybenzoic acid | -     | -                                  | <i>Chenopodium album</i> ,<br><i>Plantago lanceolata</i> ,<br><i>Amaranthus retroflexus</i> ,<br><i>Solanum nigrum</i> ,<br><i>Cirsium</i> ,<br><i>Rumex crispus</i> | Retarder – significantly inhibited germination                                 |
|                   | Juglone               | -     | -                                  | <i>Zea mays</i>                                                                                                                                                      | Retarder – delayed germination                                                 |
|                   |                       |       |                                    | <i>Abutilon theophrasti</i> ,<br><i>Sida spinosa</i> ,<br><i>Amaranthus retroflexus</i>                                                                              | Destructor – seed growth destroyed                                             |
| <b>Flavonoids</b> | Flavonols             | Seeds | Low molecular weight polyphenolics | <i>Zea mays</i>                                                                                                                                                      | Retarder – involved in seed maturation and dormancy                            |
|                   | Proanthocyanidins     | -     | -                                  | <i>Arabidopsis thaliana</i>                                                                                                                                          | Retarder – promotes ABA, maintains dormancy                                    |
|                   | Dihydroflavonoids     | -     | -                                  | <i>Brassica campestris</i> ,<br><i>Echinochloa</i>                                                                                                                   | Destructor – inhibits embryo growth                                            |

### Problem of Germination Inhibitor Research Identifying Germination Inhibitors in Dormant Seeds

Germination inhibitors are distributed throughout various parts of the plant, including the pulp, peel, endosperm, seed coat, embryo, leaves, bulbs, and roots [13]. While these compounds can inhibit seed germination in one or more species, accurately identifying the specific inhibitors present in dormant seeds remains a significant challenge. Most current research has only confirmed the presence of germination inhibitors in dormant seeds without determining their exact chemical identities.

For example, Bian et al. [26] demonstrated that extracts from *Taxus yunnanensis* seeds exhibited strong inhibitory effects on *Brassica campestris* germination, yet the specific endogenous compounds responsible for this inhibition could not

be clearly identified. Furthermore, changes in endogenous substance levels during dormancy release do not necessarily pinpoint the germination-inhibiting agents. Some inhibitors exert rapid effects in very small quantities, while others may fluctuate dramatically during dormancy yet play no regulatory role in germination.

The release of seed dormancy is a highly complex physiological and biochemical process. Even when the key triggers for dormancy release are identified, successful germination still requires favorable external conditions—such as appropriate temperature, moisture, and oxygen levels [2]. Once these conditions are met, seeds undergo changes in water absorption, reactivation of metabolic pathways, mobilization of storage reserves, and the initiation of seedling development—all involving intricate metabolic transformations [27].

Additionally, indirect metabolic changes can also hinder germination. For instance, paclobutrazol inhibits gibberellin biosynthesis and, in turn, prevents seed germination [28]. Seeds are highly responsive to environmental changes and can adjust their dormancy status accordingly [29], further complicating efforts to determine which substances are directly or indirectly disrupting germination pathways.

In many studies, once the presence of germination inhibitors is suspected, researchers employ solvent extraction methods on whole seeds or specific tissues (e.g., seed coat or endosperm). The extracts are then partitioned into various solvent phases, such as petroleum ether, diethyl ether, methanol, and water (Figure 2) [30]. The chemical constituents of each phase are subsequently analyzed using techniques like gas chromatography–mass spectrometry (GC-MS) and high-performance liquid chromatography (HPLC). Inhibitors are inferred based on changes in the concentration of individual compounds. However, this approach has significant limitations, as it only allows for indirect inference rather than definitive identification.

Seed dormancy release involves a highly integrated and dynamic series of biochemical processes, including the breakdown and synthesis of numerous substances [31]. As a result, identifying germination inhibitors based solely on changes in compound levels is inherently problematic. Moreover, in some cases, dormant seeds can germinate after the removal of the peel or seed coat, suggesting that inhibitors in those tissues may not be the direct cause of dormancy [32]. It is also important to note that the mere presence of endogenous inhibitors in seeds or fruits does not necessarily indicate dormancy, as some non-dormant seeds also contain these compounds [9].

### Examination of Germination Inhibitors

The presence of germination inhibitors is commonly assessed by diluting extracts from dormant seeds and applying them to non-dormant seeds—such as cabbage, wheat, or mung bean—to observe any inhibitory effects. For example, Cutillo et al. demonstrated that an aqueous methanol extract (10 mg/L) from *Brassica fruticulosa* inhibited the germination of *Lactuca sativa* seeds by 50%, illustrating a widely accepted method for verifying inhibitor presence in seed extracts.

However, this method has notable limitations. First, the correlation between inhibitor concentration and the germination response of non-dormant seeds is not always reliable. Dormant seeds act as complex environmental sensors, adjusting their dormancy status based on multiple environmental cues [33]. Consequently, even non-toxic substances

at high concentrations may inhibit germination, making it difficult to determine the minimum effective concentration necessary to induce dormancy.

Another overlooked issue is whether seed extracts applied to non-dormant seeds cause irreversible physiological damage. To evaluate this, ungerminated seeds should undergo a re-germination test after the removal of the inhibitor. This step helps assess whether the inhibitory effect was temporary or caused lasting damage. Unfortunately, many studies fail to consider this, potentially leading to inaccurate conclusions. For example, studies using cabbage seeds as biological models to assess the dormancy of *Daphne giraldii* and *Quercus* species did not evaluate the minimum inhibitory concentration nor confirm the non-toxic nature of the inhibitors. Therefore, ensuring the harmlessness of endogenous inhibitors should be a critical criterion in such tests.

### 5. Research Prospects of Germination Inhibitors Clarifying the Link Between Inhibitors and Germination Suppression

Currently, much of the research on germination inhibitors is based on earlier studies from the previous century, and a comprehensive theoretical framework or standardized methodology has yet to be fully established. Even after confirming the presence and effect of germination inhibitors, accurately identifying the specific inhibitory compounds remains a challenge. Moreover, some germination inhibitors exhibit species-specific effects, showing inhibition in certain seeds but not others. Therefore, it is essential to verify the general inhibitory action of known compounds across different types of dormant seeds.

#### Improving Testing and Evaluation Methods

Once specific germination inhibitors are identified, further research is needed to characterize their chemical properties, biological targets, and precise mechanisms of action. It is also critical to determine the minimum inhibitory concentration and assess whether the use of exogenous inhibitors results in physiological damage to the seeds. These efforts are fundamental to advancing the controlled regulation of seed dormancy and germination, with potential applications in seed preservation, agriculture, and ecological restoration.

#### Study on the Mechanism of Action of Germination Inhibitors

Currently, various omics approaches, such as proteomics, have been applied to investigate the mechanisms of seed germination inhibitors. For example, jasmonic acid (JA), methyl jasmonate (MeJA), and 12-oxophytodienoic acid (OPDA) have

been shown to promote the interaction between COI1 and JAZ1 proteins. This interaction triggers the ubiquitin-dependent degradation of JAZ1, which suppresses the transcription of JA-responsive genes [34], ultimately inhibiting seed germination in species like *Solanum lycopersicum*, *Brassica napus*, and *Linum usitatissimum* [35,36].

Additionally, the inhibitory mechanism of abscisic acid (ABA) on seed germination has been further elucidated through multiple experimental approaches [37]. However, the modes of action for most germination inhibitors remain poorly understood. Generally, their inhibitory effects involve mechanisms such as suppression of the electron transport chain, inhibition of seed swelling, and interference with transcription and translation processes [38].

Since different germination inhibitors operate via diverse mechanisms, further application of omics technologies is essential to uncover the specific molecular pathways involved and to deepen our understanding of their regulatory networks at the molecular level.

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