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Review

Weeds: A Critical Challenge Confronting Direct-Seeded Rice

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Abstract

Direct-seeded rice (DSR) can reduce transplanting labor and, under suitable irrigation regimes, water use; however, these benefits vary among dry-, wet-, and water-seeded systems. Weed competition during crop establishment is a major constraint because rice and weeds emerge together and early flooding may be absent. This review critically examines herbicide modes of action, target-site resistance (TSR), non-target-site resistance (NTSR), and genetic strategies for herbicide-resistant rice, distinguishing field observations from functional validation. It further links resistance mechanisms to DSR-specific management constraints, including weed seedbanks, herbicide selection pressure, gene flow to weedy rice, and integration of chemical and nonchemical control. We identify research gaps in cross-resistance prediction, trait deployment, and field-scale durability, and propose an evidence-based framework for sustainable DSR weed management.

Keywords: direct-seeded rice; herbicide; target-site resistance; non-target-site resistance; gene editing; herbicide-resistance breeding

1. Introduction

Global population growth continues to increase pressure on rice productivity [1–3]. Rice (*Oryza sativa* L.) is established by transplanting or direct seeding [4]. Direct seeding encompasses dry seeding into nonpuddled soil, wet seeding into puddled soil, and water seeding into flooded fields; the associated irrigation regimes, weed flora, and greenhouse-gas outcomes differ [4,5]. In dry- and wet-seeded systems, rice and weeds may germinate simultaneously, and limited early flooding can favor competitive grasses, sedges, and broadleaf weeds. Therefore, water savings, labor savings, and methane reductions should be described relative to the establishment and irrigation system used, rather than as universal advantages of DSR. Weed competition is especially damaging during crop establishment, when small differences in emergence and early growth influence later canopy closure and yield. This review focuses on how herbicide resistance and rice breeding interact with these DSR-specific ecological constraints.

Weed management in DSR combines preventive measures (clean seed, machinery sanitation, and seedbank reduction), cultural practices (stale seedbeds, competitive cultivars, crop rotation where feasible, seeding density and timing), water management, mechanical removal, and herbicides. Herbicide-resistant rice traits and breeding strategies also broaden

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management options [6–8]. The feasibility of each practice depends on soil and irrigation conditions [9–11]. Straw burning is not a generally recommended weed-management option because of air-quality impacts and local restrictions. Rapid canopy closure can suppress weed emergence by reducing light availability [12–14], but claims about accelerated nitrogen cycling require direct evidence and should not be treated as an established weed-suppression mechanism.

Herbicides remain important for timely weed control in mechanized DSR, but repeated use of one effective mode of action selects for resistant weeds (Figure 1) [15,16]. We searched Web of Science, Scopus, PubMed, Google Scholar, the HRAC mode-of-action classification and the International Herbicide-Resistant Weed Database through 18 November 2026, using combinations of direct-seeded rice, dry/wet/water seeding, rice weeds, herbicide resistance, target-site resistance, non-target-site resistance, metabolism, gene editing and resistance breeding. We prioritized primary studies and authoritative databases, distinguishing DSR field populations from studies in other weeds, rice genotypes and model plants. Herbicide-resistant rice can expand management options [16–18], but crop tolerance alone does not prevent resistant weeds, resistant volunteer rice or gene flow into weedy rice. Unlike reviews focused primarily on resistance genes or editing tools, this article evaluates evidence strength and connects resistance mechanisms to DSR establishment conditions, breeding readiness and integrated weed management. Specifically, we (i) compare herbicide target pathways and resistance mechanisms, (ii) distinguish associative from causal evidence, (iii) evaluate herbicide-resistant rice breeding approaches and (iv) prioritize DSR-specific resistance stewardship.

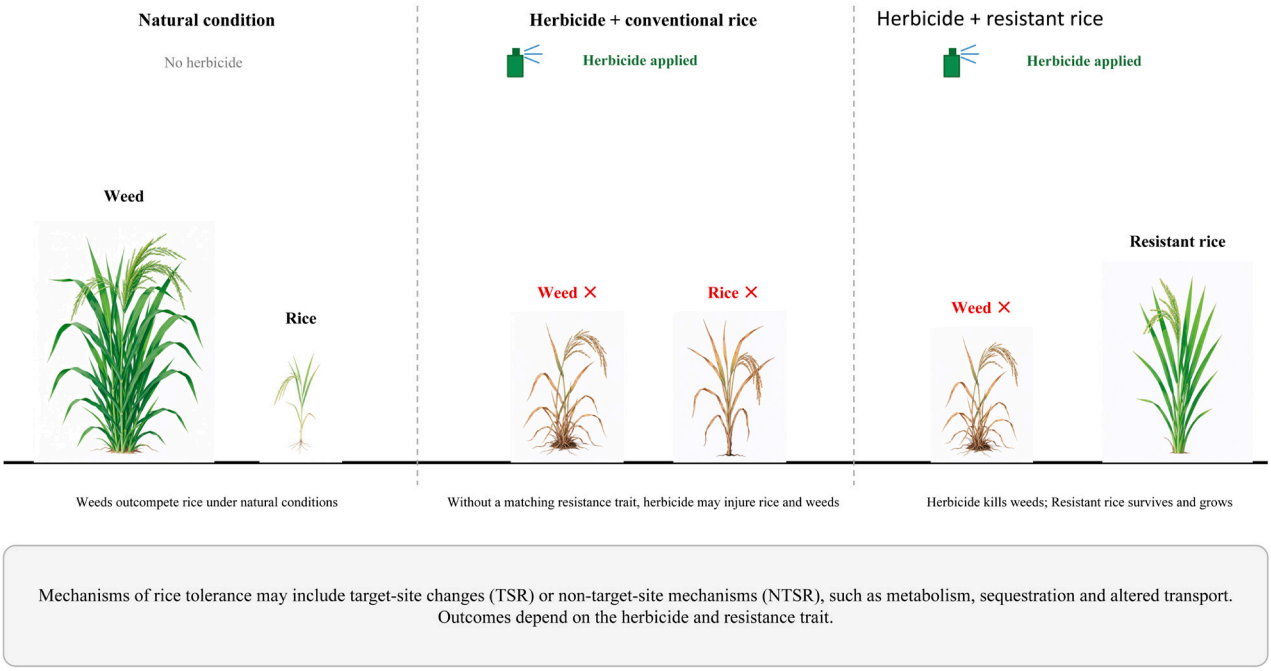

Figure 1. Conceptual comparison of weed competition and herbicide response in direct-seeded rice. Without herbicide, weeds may outcompete rice during establishment. A herbicide lacking crop selectivity can injure both rice and susceptible weeds, whereas a matching resistance trait may protect rice while susceptible weeds are controlled. Herbicide resistance can involve target-site resistance (TSR) or non-target-site resistance (NTSR), including metabolism, transport, and sequestration; the mechanism depends on the herbicide and rice genotype.

2. Classification and Application of Rice Herbicides

2.1. ACCase-Inhibitor Herbicides

Acetyl-CoA carboxylase (ACCase) catalyzes the carboxylation of acetyl-CoA to malonyl-CoA, a key step in fatty-acid biosynthesis [19–24]. Plants may possess plastidic heteromeric ACCase and homomeric ACCase isoforms; the distribution of these forms varies across taxa. The heteromeric enzyme contains biotin carboxylase, biotin carboxyl carrier protein, and carboxyltransferase components that associate as a functional complex rather than being universally linked by covalent bonds. ACCase-inhibiting herbicides primarily act on susceptible homomeric plastidic ACCase in grasses, whereas heteromeric plastidic ACCase is generally insensitive to these herbicides [22,23]. Binding at the carboxyltransferase region inhibits fatty-acid synthesis; membrane disruption and growth arrest are downstream consequences, not the primary binding mechanism.

ACCase-inhibiting herbicides include aryloxyphenoxypropionates (FOPs), cyclohexanediones (DIMs), and phenylpyrazolines (DENs). These families differ in chemical structure and binding interactions but share ACCase as the biochemical target [19,23]. Structure–activity claims concerning individual substitutions require verification against the original chemistry studies; unsupported generalizations about benzene-ring substitutions have been removed. The choice of active ingredient in rice must consider crop selectivity, application timing, registered uses, and resistance status of local grass-weed populations.

2.2. Hormone-Type Herbicides

Hormonal herbicides act through auxin perception and signaling. Representative rice-use examples include the quinolinecarboxylate quinclorac and the aryl picolinate florpyrauxifen-benzyl. Chemical family, active ingredient, crop selectivity, application timing and local resistance status should be specified separately because auxin-like plant growth regulators are not interchangeable with registered herbicides.

Quinclorac is used in rice to control *Echinochloa* spp. and other grass weeds, and resistant populations have been reported [25]. Florpyrauxifen-benzyl is an aryl picolinate auxin herbicide registered for rice. In a study of *Echinochloa crus-galli*, treatment induced ethylene biosynthesis, increased abscisic acid, antioxidant responses and malondialdehyde, with the magnitude and timing depending on the tested species and treatment conditions [26–28]. These observations describe a tested phytotoxicity response and should not be generalized to every weed or field situation.

2.3. Acetolactate Synthase (ALS)-Inhibiting Herbicides

Acetolactate synthase (ALS; also called acetohydroxyacid synthase) catalyzes early reactions in branched-chain amino acid biosynthesis [29]. ALS inhibitors block production of valine, leucine, and isoleucine, causing growth arrest, particularly in meristematic tissues. The absence of this pathway in animals contributes to biochemical selectivity, but does not by itself demonstrate human safety or environmental benignity. Toxicological profile, persistence, crop injury, and effects on nontarget organisms vary by active ingredient, dose, soil conditions, and use pattern. Sulfonylureas and imidazolinones are among the ALS-inhibiting chemical families relevant to rice systems [30].

2.4. Protoporphyrinogen Oxidase (PPO)-Inhibiting Herbicides

Protoporphyrinogen oxidase (PPO) catalyzes the oxidation of protoporphyrinogen IX to protoporphyrin IX (Proto IX), a shared intermediate in chlorophyll and heme biosynthesis (Table 1). HPPD inhibitors are a separate Group 27 class, represented by mesotrione and bicyclopyrone, and should not be conflated with PPO inhibitors [31]. Proto IX is a substrate for Mg-chelatase and Fe-chelatase, which direct tetrapyrrole metabolism toward chlorophyll

and heme, respectively. Plants contain PPO isoforms whose subcellular distribution can vary among species; generalizations concerning PPO1 and PPO2 localization should therefore be made cautiously [32,33]. PPO-inhibiting herbicides block this oxidation step, allowing protoporphyrinogen IX to accumulate and move to other cellular compartments, where it can undergo nonenzymatic oxidation to Proto IX. In illuminated tissues, excess Proto IX acts as a photosensitizer and generates reactive oxygen species, including singlet oxygen, leading to lipid peroxidation, membrane damage and tissue necrosis. Thus, phytotoxicity is associated with light-dependent oxidative damage, not with an inability of Proto IX to serve as a chelatase substrate. The activity, persistence and crop selectivity of individual PPO inhibitors vary with their chemical properties and application conditions [33,34].

2.5. Photosystem II Inhibitors

PSII mediates the transfer of electrons from water to plastoquinone, which further binds to the D1 protein (Table 1). The D1 protein is an essential subunit of the PSII reaction center. It provides binding sites for various co-factors and participates in primary charge separation and electron transport. Inhibition of electron transfer from PSII to PSI suppresses the production of nicotinamide adenine dinucleotide phosphate (NADPH) and ATP. Damage to the D1 protein blocks photosynthetic electron transport, impairs the PSII reaction center, and ultimately causes plant death due to disrupted photosynthesis. Photosystem II-inhibiting herbicides compete with plastoquinone for binding sites on the D1 protein. By binding to the D1 protein, they block electron transfer from the Q_a site to the Q_β site, suppress photosynthesis, and eventually kill weeds. Triazine herbicides represent the major group within this class and have been widely applied. As selective, systemic and broad-spectrum herbicides, triazines are absorbed by plant leaves and roots and translocated upward via the xylem. They possess advantages including a broad weed-control spectrum, chemical stability and low cost.

2.6. 5-Enolpyruvylshikimate-3-Phosphate (EPSPS)-Inhibiting Herbicides

5-Enolpyruvylshikimate-3-phosphate synthase (EPSPS) catalyzes the reaction between shikimate-3-phosphate (S3P) and phosphoenolpyruvate (PEP) to form 5-enolpyruvylshikimate-3-phosphate in the shikimate pathway. Glyphosate is competitive with PEP and binds preferentially to the EPSPS-S3P complex, forming a stable inhibitory EPSPS-S3P-glyphosate ternary complex [35]. This interaction blocks aromatic amino acid biosynthesis. Glyphosate is generally absorbed through foliage and translocated to growing tissues, although efficacy depends on species, developmental stage and environment.

2.7. Glutamine Synthetase (GS)-Inhibiting Herbicides

Glutamine synthetase (GS) catalyzes the ATP-dependent incorporation of ammonium into glutamate to form glutamine and is essential for nitrogen assimilation and reassimilation of photorespiratory ammonium. Glufosinate (phosphinothricin) inhibits GS, causing ammonium accumulation, altered amino acid homeostasis, oxidative stress and secondary impairment of photosynthesis [36]. During photorespiration, glyoxylate is converted to glycine by transamination. Glufosinate is predominantly contact-acting, but limited translocation can occur depending on species, application and physiological conditions. Assertions about neurotoxicity, livestock sensitivity, leaching or environmental safety require compound-specific toxicological and exposure evidence.

Table 1. Representative HRAC/WSSA herbicide groups, biochemical targets, selected active ingredients and DSR considerations.

HRAC/WSSA Group and Mode of Action	Biochemical Target/ Primary Effect	Chemical Family	Representative Active Ingredient	DSR Use and Evidence Caveat	Ref.
ACCase inhibitors	ACCase; inhibit fatty-acid biosynthesis	FOPs/DIMs/DENs	Cyhalofop-butyl	Grass control; verify crop selectivity and local resistance	[19–24,37–39]
Synthetic auxins	Auxin perception and signaling	Quinolinecarboxylates/arylpicolinates	Quinclorac; florpyrauxifen-benzyl	Use depends on crop label, timing and local resistance	[25–28]
ALS inhibitors	ALS; inhibit branched-chain amino acid biosynthesis	Sulfonylureas/imidazolinones	Bensulfuron-methyl	Target-site and metabolic resistance reported	[29,30,40–42]
PPO inhibitors	PPO; photooxidative membrane injury	Diphenyl ethers and cyclic imides	Oxyfluorfen; flumioxazin	Crop safety and persistence vary with active ingredient	[32–34]
PSII inhibitors	D1 protein; inhibit electron transport	Triazines/triazinones	Atrazine (class example)	Rice use and registration must be verified	[43]
EPSPS inhibitors	EPSPS; inhibit aromatic amino acid synthesis	Glycines	Glyphosate	Nonselective unless a tolerant trait is present	[35,44–50]
GS inhibitors	GS; inhibit nitrogen assimilation	Phosphinic acids	Glufosinate	Predominantly contact-active; limited translocation can occur	[10,36,51,52]
HPPD inhibitors	HPPD; disrupt plastoquinone and carotenoid biosynthesis	Triketones/benzoylpyrazoles	Mesotrione; bicyclopyrone	HIS1-mediated detoxification and target-pathway tolerance are distinct mechanisms	[31,53,54]

3. Research Progress on Herbicide Resistance in Weeds

Herbicide-resistant weed populations have evolved through target-site resistance (TSR) and non-target-site resistance (NTSR), often in combination [37–39]. Counts of resistant species, active ingredients, and sites of action depend on the database, date accessed, and counting criteria; readers should consult the International Herbicide-Resistant Weed Database (weedscience.org) for dated records rather than treating undated counts as fixed. Evidence from DSR weed populations should be distinguished from the findings in other cropping systems and model plants, especially when drawing management conclusions.

3.1. Target-Site Resistance Mechanism

TSR results from alterations involving the herbicide target itself. Amino acid substitutions may reduce herbicide binding; target-gene amplification can increase gene dosage; and regulatory changes can elevate target expression without changing copy number. These are mechanistically distinct processes and should be reported separately. An observed expression increase does not establish causality without genetic, biochemical, or other functional validation.

3.1.1. Target Enzyme Gene Mutation

Target-site mutations alter the amino acid sequence of the herbicide target and can reduce binding. Examples include ALS, ACCase, PPO and EPSPS substitutions. In EPSPS, single substitutions such as Pro106 or Thr102 generally confer low-level glyphosate resistance, whereas combinations such as T102I + P106S can confer much higher resistance in particular weed populations [44–46]. The TAP-IVS combination in *Amaranthus palmeri* has also been reported [47,48]. The reported fold changes are resistance ratios or LD50-based comparisons from the cited studies, not universal values. In the PDS gene, Leu498Val and Leu498Val + Glu425Asp were associated with diflufenican resistance and cross-resistance to picolinafen in *Sisymbrium orientale* [55].

3.1.2. Increased Target Enzyme Gene Expression

Increased gene expression is generally caused by mutations in the gene promoter region or elevated gene copy number. Glyphosate resistance in *Conyza canadensis* collected from the Bohai-Rim Bay and Loess Plateau regions of China, as well as in *Eleusine indica* from the Dongting Lake and Poyang Lake surrounding areas, is associated with EPSPS gene overexpression [40,49]. Transgenic *Arabidopsis thaliana* was used as a model plant to investigate the effects of EPSPS gene overexpression on plant growth and reproduction. The results revealed that EPSPS overexpression in *Arabidopsis thaliana* incurred no fitness cost and might confer benefits under the tested growth conditions [50]. In recent years, a growing number of studies have focused on the expression of target genes at different growth stages or in distinct organs. Overexpression-mediated weed resistance of varying magnitudes has also been observed in target-enzyme genes other than EPSPS. ACCase gene overexpression was documented to confer resistance to cyhalofop-butyl in *Echinochloa crus-galli*. Resistant plants exhibited 2.0-fold and 2.8-fold overexpression of *ACCase1* and *ACCase3*, respectively [56]. Similarly, enhanced overexpression of the ALS gene was found to confer resistance to pyroxsulam in *Bromus sterilis* [41]. Subsequently, target-site ALS gene overexpression conferring resistance to the ALS-inhibiting herbicide nicosulfuron was also identified in *Digitaria sanguinalis* collected from Heilongjiang Province, China. The ALS gene expression level in resistant populations was 2.07-fold higher than that in susceptible populations [42].

EPSPS copy-number increases should be reported as copy-number or relative quantitative differences, not as expression fold changes unless the study measured expression [57–62]. In *Amaranthus palmeri*, copy-number amplification may involve extrachromosomal circular DNA, whereas other weeds show tandem duplication or chromosomal rearrangement [63–66]. ACCase copy-number changes and increased expression are distinct from metabolic detoxification and require direct evidence before being assigned a metabolic mechanism [67]. In a glufosinate-resistant *Amaranthus palmeri* accession, GS2 copy-number and expression increases were reported, but their contribution should be interpreted with the study's resistance phenotype and other mechanisms [51].

3.2. Non-Target-Site Resistance Mechanisms

NTSR encompasses mechanisms that reduce the biologically effective herbicide dose at the target without altering the target enzyme itself, including metabolic conversion, reduced uptake or movement, and sequestration. These mechanisms can confer cross-resistance across chemically distinct herbicides, although the spectrum depends on the substrate specificity and regulation of the underlying genes. Transcript accumulation or association with a resistant population is hypothesis-generating; causal assignment requires functional tests and, where feasible, direct measurements of herbicide disposition.

3.2.1. Detoxification Metabolism of Herbicides

Herbicide metabolism may involve oxidation, reduction, hydrolysis, conjugation, and compartmentation, but these reactions do not always follow a universal three-phase sequence. Some initial transformations may generate metabolites that retain or increase biological activity. Cytochrome P450 monooxygenases, glutathione S-transferases, and aldo-keto reductases are candidate mediators of metabolic resistance, yet their roles must be evaluated for the specific herbicide and weed population using metabolism assays, genetic manipulation, or complementary functional evidence.

A set of specific P450 enzymes belonging to the CYP81 family have been proven to mediate resistance to multiple herbicides in grass weeds. For instance, *CYP81A10v7* confers broad-spectrum resistance to herbicides of diverse modes of action, including ALS inhibitors (chlorsulfuron), ACCase inhibitors (diclofop-methyl), HPPD inhibitors (mesotrione), PSII inhibitors (atrazine), and tubulin inhibitors (trifluralin) in *Lolium rigidum* [43]. *CYP81A68* in *Echinochloa crus-galli* is associated with resistance to ALS- and ACCase-inhibiting herbicides (Table 2) [68]. Transgenic rice overexpressing *CYP81Q32* exhibits enhanced tolerance to mesosulfuron-methyl, pyriminobac-methyl and bispyribac-sodium [69]. Six CYP81A family genes (*CYP81A12*, *CYP81A14*, *CYP81A15*, *CYP81A18*, *CYP81A21* and *CYP81A24*) have been identified in *Echinochloa phyllopogon* (water barnyardgrass) to possess metabolic functions toward herbicides with diverse modes of action. Among them, *CYP81A12* and *CYP81A21* are associated with the metabolism and resistance to multiple ALS- and ACCase-inhibiting herbicides, whereas *CYP81A15* and *CYP81A24* reduce the sensitivity of transgenic *Arabidopsis thaliana* to clomazone [70–73]. Apart from P450 enzymes of the CYP81 family, studies have reported that *CYP709C69* mediates resistance to diclofop-methyl in *Echinochloa phyllopogon*. In terms of broadleaf weeds, a set of P450 genes potentially associated with herbicide resistance (*CYP77B34*, *CYP709B8*, *CYP81F5*, *CYP96A146* and *CYP96A1-47*) have been identified in *Descurainia sophia* populations [74,75]. Among them, both *CYP96A146* and *CYP77B34* mediate resistance to tribenuron-methyl. Transgenic *Arabidopsis thaliana* overexpressing *CYP96A146* exhibits strong metabolic activity toward carfentrazone-ethyl and oxyfluorfen. Transgenic *Arabidopsis thaliana* overexpressing *CYP77B34* also displays certain metabolic capacity against herbicides belonging to PPO inhibitors (carfentrazone-ethyl and oxyfluorfen), very-long-chain fatty acids (VLCFAs) inhibitors (pretilachlor), and PSII inhibitors (bromoxynil) [76].

Table 2. Representative weed problems and management options in direct-seeded rice.

Nonchemical Complements	Resistance Evidence to Monitor	Common Herbicide Options/HRAC-WSSA Group	Representative DSR Weed	Ref.
Stale seedbed, water management, early scouting, remove escapes	ACCase, ALS, quinclorac and metabolic resistance reported	Cyhalofop-butyl; penoxsulam; quinclorac	*Echinochloa crus-galli* and *E. colona*	[22,25,38,68]
Competitive stand, rotary weeding, prevent seed return	ACCase, ALS and HPPD metabolic resistance reported	Cyhalofop-butyl; penoxsulam; tripyrasulfone	*Leptochloa chinensis*	[53,77]
Water depth and timing, hand removal of survivors	Cross-resistance and ALS target-site or metabolic mechanisms	Bensulfuron-methyl, penoxsulam and bispyribac-sodium	*Cyperus difformis*	[47,50,51]

Table 2. *Cont.*

Nonchemical Complements	Resistance Evidence to Monitor	Common Herbicide Options/HRAC-WSSA Group	Representative DSR Weed	Ref.
Certified seed, rogue volunteers, rotate crops and modes of action	Gene flow, volunteers and seedbank persistence	Imidazolinones in tolerant-rice systems	Weedy rice (*Oryza sativa* complex)	[5,14,78]
Flooding where feasible, stale seedbeds, mechanical removal	Population- and compound-specific resistance testing required	ALS inhibitors and registered auxin options	*Monochoria vaginalis* and other broadleaf weeds	[11,26–28,30]

GSTs represent a supergene-family-encoded group of metabolic enzymes. They catalyze the conjugation reaction between glutathione (GSH) and herbicide molecules containing alkyl sulfoxide, phenolic or halogen functional groups, degrading primarily metabolized herbicides into more polar and less toxic metabolites, and thereby play a vital role in herbicide metabolism and resistance [79]. GSTs exert direct herbicide-detoxifying effects by catalyzing the conjugation reaction between herbicide molecules and glutathione. Alternatively, they mitigate or protect plants from injury by scavenging various reactive oxygen species generated during herbicide action. In addition, GSTs can respond to abiotic stress by modulating redox-signaling pathways that transcriptionally activate defense-related genes [80]. Recent studies have revealed that overexpression of *GSTF1* and *GSTU6* is associated with resistance to cyhalofop-butyl in *Leptochloa chinensis* [77]. Resistance to quizalofop-p-ethyl in *Polypogon fugax* is linked to the overexpression of *GST2c* and *GSTL3*, and overexpression of *GSTU3* is related to glyphosate resistance in *Eleusine indica* [81,82]. Some studies have found that overexpression of *GSTF1* is associated with resistance to the 4-hydroxyphenylpyruvate dioxygenase (HPPD)-inhibiting herbicide tripyrasulfone in *Leptochloa chinensis* [53]. At present, most evidence for GST-mediated herbicide metabolism or resistance is derived from RNA-seq or RT-qPCR assays. Both RNA-seq and RT-qPCR detect gene expression differences in one or multiple genes between susceptible and resistant plants at a given time point. Such differences may result from variations in plant genetic background, growth status, culture conditions, resistance traits, and herbicide treatment effects. Expression differences in GST genes between susceptible and resistant plants can partly indicate the involvement of GSTs in weed herbicide resistance. Nevertheless, further, more direct evidence from genetic transformation and herbicide metabolic assays is required to ultimately confirm the participation of specific GST isoenzymes in herbicide metabolism or resistance.

AKR evidence includes both heterologous functional assays and weed-population studies. Vemanna et al. reported enhanced glyphosate tolerance in plants expressing bacterial or rice AKR genes [83]. Pan et al. identified EcAKR4-1 and EcAKR4-2 in glyphosate-resistant *Echinochloa colona* and showed higher expression, AKR activity and glyphosate metabolism than in susceptible plants [84]. These results support a functional contribution in that population, while the species, allele and assay context should not be generalized to all Echinochloa or all glyphosate-resistant weeds [85].

3.2.2. Herbicide Sequestration

Herbicide sequestration can reduce the biologically active dose by restricting movement or exporting herbicide and metabolites to the apoplast or vacuole. Vacuolar or cellular sequestration is documented for particular glyphosate-resistant populations, but its prevalence varies among species and mechanisms. In *Echinochloa colona*, EcABCC8 overexpression

in rice, maize and soybean increased glyphosate tolerance, whereas knockout of the endogenous rice ortholog OsABCC8 increased rice sensitivity, supporting a transporter-mediated efflux mechanism [86].

3.2.3. Reduced Herbicide Absorption and Translocation

Reduced uptake or translocation can contribute to resistance for particular herbicide-population combinations. In *Amaranthus hybridus*, lower uptake and translocation of glyphosate and imazethapyr were measured in the resistant population [87]. Reduced 2,4-D translocation was reported in *Sisymbrium orientale* [88], and rapid leaf necrosis or restricted movement was reported for 2,4-D in *Conyza sumatrensis* [89]. These studies do not establish impaired translocation as a mechanism for all auxin analogs.

4. Application of Herbicide-Resistant Genes in Rice Breeding

With the continuous advancement of molecular biology technologies, herbicide-tolerant crop breeding has gradually evolved from natural screening and random mutagenesis to precision design. Three major technical systems have been established: conventional mutation breeding, transgenic breeding and gene-editing breeding. The integrated model of mutation breeding combined with molecular marker-assisted selection and other technologies has become mainstream, driving the upgrading of herbicide-tolerant crop germplasms from single resistance toward multiple-resistance and stacked traits.

4.1. Developing Herbicide-Resistant Rice by Transgenic Technology

Transgenic herbicide-tolerant crops include traits based on CP4-EPSPS, glyphosate acetyltransferases or oxidoreductases, PAT or Bar, and other trait systems. The commercial status and regulatory treatment of a trait depend on crop, country and year, so a global claim that only three commercial traits exist is not supportable. Nontransgenic imidazolinone-tolerant crops produced by mutagenesis should be distinguished from transgenic traits. Transgenic methods can provide strong tolerance but require transformation, event selection, regulatory review and stewardship. Comparisons of time, cost or success rate should be made only with defined evidence.

4.2. Development of Herbicide-Resistant Rice Using Gene Editing Technology

The gene-editing system precisely modifies genes to express specific herbicide-resistance genes or regulate the expression of relevant genes, thereby enhancing plant tolerance to herbicides. To date, remarkable progress has been achieved in developing herbicide-resistant crops using gene-editing technology.

4.2.1. Non-Homologous End Joining and Homology-Directed Repair

Genome editing can create herbicide-tolerance alleles through different DNA-repair or nucleotide-conversion routes. Classical NHEJ commonly generates insertions or deletions, but the intron-targeting CRISPR-Cas9 method reported by Li et al. used paired sgRNAs and a donor template to replace an OsEPSPS exon through NHEJ-mediated targeted replacement. The study obtained the T102I + P106S (TIPS) substitutions at about 2.0% frequency, confirmed the intended sequence and glyphosate-resistant phenotype, and observed transmission to the next generation [90]. This example should be distinguished from HDR, base editing and prime editing. Editing of OsCNGC12 and the OsHPPD 3′ untranslated region concerns different loci and herbicide-response phenotypes [54,91].

In maize (*Zea mays*), CRISPR/Cas9 and homology-directed repair replaced a ZmEPSPS fragment with a GATIPS sequence containing three amino acid substitutions, rather than disrupting the conserved domain. The edited plants showed enhanced glyphosate toler-

ance [92]. In rice, targeted OsALS substitutions including W548L and S627I were obtained by gene targeting, and Cas9-VirD2 was used to facilitate HDR in another study [93,94]. These examples illustrate distinct replacement and donor-directed strategies, while HDR efficiency remains context-dependent.

4.2.2. Development of Herbicide-Resistant Rice by Base Editing

Using the rice adenine base editor rBE14, the endogenous rice tubulin gene (*OsTubA2*, tubulin alpha 2) was targeted. Among the 63 obtained 'Kitaake' rice lines, eight lines harbored an A-to-G base substitution in *OsTubA2*, resulting in an amino acid substitution from Met to Thr at residue 268 (M268T). Further resistance assays demonstrated that rice plants carrying the M268T mutation in *OsTubA2* acquired resistance to the herbicides trifluralin and pendimethalin, and this trait was stably inherited in subsequent generations [95]. Multiple crop varieties tolerant to ALS-inhibiting herbicides have been successfully developed using the CBE technology [96–101], including wheat (*Triticum aestivum*), rice, maize, watermelon (*Citrullus lanatus*), rapeseed (*Brassica napus*), tomato (*Solanum lycopersicum*), and potato (*Solanum tuberosum*) [102–105]. Targeting *GmALS1* and *GmALS3* genes in soybean (*Glycine max*), the cytosine base editor (BE4max-dCas12-SF01) was used to induce C-to-T base substitutions at the P178 site of GmALS1 and the P172 site of GmALS3. The base editor was delivered into the elite soybean cultivar 'Xudou18' via *Agrobacterium*-mediated transformation, generating single mutants ALS1(P178S), ALS3(P172S), and the double mutant ALS1(P178S)/ALS3(P172S), designated as *als1*, *als3*, and *als1/als3*, respectively. Wild-type and *als1/als3* plants were treated with different concentrations of the herbicide flucarbazone-sodium. Wild-type plants suffered severe damage under the treatment of twice the recommended field dose. In contrast, the *als1/als3* plants showed no significant differences in agronomic traits compared with untreated wild-type plants [106]. Studies have shown that targeted mutation of the DNA sequences encoding Pro-171 (P171) and Gly-628 (G628) of rice ALS using CBE generated a set of mutant lines resistant to ALS-inhibiting herbicides. Among them, four distinct P171 mutants, namely P171S, P171A, P171Y and P171F, exhibited varying levels of resistance after spraying with five ALS-inhibiting herbicides: nicosulfuron, imazapyr, pyrazosulfuron-ethyl, fenpyrazone and bispyribac-sodium (Table 2). When the application rate of bispyribac-sodium increased from 1-fold to 32-fold of the recommended field dose, growth arrest was aggravated in P171S and P171A mutants, whereas P171Y and P171F mutants remained unaffected. In addition, the triple-amino-acid mutant P171F/G628E/G629S displayed high-level resistance to all five tested ALS-inhibiting herbicides. Field trials demonstrated that mutants P171F and P171F/G628E/G629S possessed favorable herbicide resistance under field-rate herbicide application, without any yield penalty (Table 2) [107]. Recent studies have uncovered the potential of TadDE, a high-performance cytosine and dual-base editor derived from TadA-8e. TadDE was employed for base editing of *OsALS*, and two herbicide-resistance sites, *OsALS-R190H* and *OsALS-G628E/G629S*, were validated via herbicide screening [108].

4.2.3. Development of Herbicide-Resistant Rice Using Prime-Editing Technology

Prime editing uses a Cas nickase fused to reverse transcriptase and a pegRNA to encode intended substitutions or short insertions/deletions. For the HIS1 example, the reported system was GRAND editing, which uses paired nickase complexes and complementary reverse-transcription templates to restore the 28 bp sequence deleted in some Indica rice. In the S1035 transformation, nine of 78 RTT-10 T0 plants were herbicide-resistant (11.5%); in MingHui86, 13 of 84 RTT-10 T0 plants were resistant (15.5%). Target-site sequencing identified precise insertions and byproducts, mesotrione treatment provided phenotypic selection, and T-DNA-free homozygous progeny were identified in the T1 generation [109,110]. These

percentages refer to resistant T0 plants divided by transformed T0 plants and do not by themselves establish field-level durability (Table 3).

4.2.4. Generation of Herbicide-Resistant Crops by Gene-Editing-Mediated Upregulation

A donor-DNA-free CRISPR/Cas9 study generated a 911 kb inversion that swapped the CP12 promoter onto OsPPO1 and a 338 kb duplication that placed the Ubiquitin2 promoter before OsHPPD. Homozygous structural-variant plants showed tens-fold increases in expression and resistance to flumioxazin and bicyclopyrone in field trials [111]. PrimeRoot is a separate system for precise insertion of large DNA sequences, including an Act1 promoter upstream of OsHPPD [112]. The inversion and duplication should therefore be reported as distinct structural outcomes.

Table 3. Reported rice gene-editing examples associated with herbicide response. Direct target alterations, metabolic protection and indirect sensitivity changes are separated.

Mechanistic Category and Locus	Edit or Phenotype Outcome	Evidence Stage and Reference	Ref.
ACCase target: OsACC1	Base/prime-edited substitutions; resistance to selected Group 1 ACCase inhibitors	Rice screens or greenhouse assays	[113–117]
Auxin perception: OsTIR1/OsAFB	CRISPR loss-of-function; altered auxinic-herbicide sensitivity	Functional rice assays	[118]
PPO target: OsPPO1	911 kb inversion places CP12 promoter before OsPPO1; flumioxazin tolerance	Field-tested structural variants	[112]
HPPD target: OsHPPD	3′ UTR editing or 338 kb duplication; mesotrione/bicyclopyrone tolerance	Controlled assays; field trial for duplication	[54,112]
EPSPS target: OsEPSPS	T102I + P106S targeted replacement and other edited alleles; glyphosate resistance	Sequence, phenotype and inheritance for TIPS	[90,91,119]
ALS target: OsALS1	HDR/base-edited W548L, S627I, P171 and G628 alleles; ALS-inhibitor resistance	Herbicide assays; some field trials	[94,108]
Indirect sensitivity: OsCNGC12; OsGLR2; OsARF18; OsSPL10	KO or base editing; altered sensitivity to tested Group 2 or 10 herbicides	Controlled rice assays	[92,120–122]
Metabolism: HIS1; OsACE2; CYP genes	Sequence restoration or expression changes; active-ingredient-specific tolerance	Phenotype plus biochemical or expression evidence	[54,111,123–125]

5. Discussion

The evidence reviewed here supports a DSR-specific conclusion. Target-site substitutions are often supported by sequence and functional tests, whereas many proposed non-target-site mechanisms remain based on expression differences. For DSR, a candidate gene matters only when its effect is demonstrated in the relevant weed or rice background and the resulting trait improves control under field conditions.

TSR and NTSR also differ in management implications. A different mode of action may reduce selection for one target-site mutation, but cross-resistance and multiple resistance can limit alternatives. Resistance to 2,4-D and dicamba has been documented in weeds [28,85]. In DSR, mixtures or rotations should be used only when each component is independently

effective against the local population, with cultural practices that reduce weed recruitment and seed return.

Breeding value depends on more than editing precision. Functionally validated alleles still require stable inheritance, dose–response data, agronomic evaluation, and assessment of gene flow and regulatory requirements. Plant origin alone does not establish environmental safety or public acceptance.

Herbicide-resistant rice should therefore be deployed within integrated weed management. Clean seed and equipment, competitive establishment, suitable water management, early scouting, removal of escapes, seedbank reduction, and monitoring of weedy rice and volunteers should accompany any resistant cultivar. The priority is to connect molecular evidence with local weed phenotypes and field durability.

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