

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

BAYER CROPSCIENCE LP, MONSANTO COMPANY, AND
INARI AGRICULTURE, INC.
Petitioner,

v.

CORTEVA AGRISCIENCE LLC,
Patent Owner.

Case IPR2023-01038¹
Patent No. 11,299,745

PATENT OWNER'S NOTICE OF APPEAL

¹ The Board joined Inari Agriculture, Inc. as a party (Petitioner) to this proceeding via a Motion for Joinder in IPR2024-00181.

Pursuant to 35 U.S.C. §§ 141(c), 142, and 319, and 37 C.F.R. § 90.2(a), Patent Owner Corteva Agriscience LLC hereby provides notice of appeal to the United States Court of Appeals for the Federal Circuit from the Final Written Decision of the Patent Trial and Appeal Board (“Board”) entered on December 17, 2024, (Paper No. 43, Non-Public Version and Paper No. 45, Public Version) (“Final Written Decision”) and from all underlying orders, decisions, rulings, and opinions relating to the *inter partes* review of U.S. Patent No. 11,299,745 (“the ’745 patent”) in IPR2023-01038. This Notice is timely under 37 C.F.R. § 90.3(a)(1), having been filed no later than sixty-three (63) days after the issuance of the Final Written Decision.

In accordance with 37 C.F.R. § 90.2(a)(3)(ii), Patent Owner states that the issues on appeal include, but are not limited to:

- The Board’s determination that claims 1-29 of the ’745 patent are unpatentable;
- The Board’s application of an incorrect legal standard for written description;
- The Board’s incorrect and inconsistent reliance on post-filing date evidence;
- The Board’s error in assessing the knowledge of one skilled in the art;
- The Board’s error in assessing the scope of the challenged claims;

- Any Board finding decided adversely to Patent Owner unsupported by substantial evidence; and
- The Board's incorrect application of waiver, allocation of the burden of proof, and/or violation of the Administrative Procedure Act in rendering its Final Written Decision.

Patent Owner further reserves the right to challenge any finding or determination supporting or relating to the issues above, and to challenge all other issues decided adversely to Patent Owner in any orders, decisions, rulings, or opinions underlying or supporting the Final Written Decision. A copy of the Public Version of the Final Written Decision is attached to this Notice.

Pursuant to 35 U.S.C. § 142 and 37 C.F.R. § 90.2(a)(1), this Notice is being filed with the Director of the United States Patent and Trademark Office, and a copy of this Notice is being concurrently filed with the Board. In addition, a copy of this Notice and the required docketing fees are being filed with the Clerk's Office for the United States Court of Appeals for the Federal Circuit via CM/ECF in accordance with 37 C.F.R. § 90.2(a)(2), Federal Rule of Appellate Procedure 15, and Federal Circuit Rule 15.

Respectfully submitted,

Dated: February 18, 2025

/Pier D. DeRoo/
Pier D. DeRoo, Reg. No. 69,340
Lead Counsel for Patent Owner
Corteva Agriscience LLC

CERTIFICATE OF SERVICE

I hereby certify that on February 18, 2025, in addition to being filed and served electronically through the Patent Trial and Appeal Board's P-TACTS System, this **PATENT OWNER'S NOTICE OF APPEAL** was filed with and served on the Director of the United States Patent and Trademark Office via email at the following:

Office of the General Counsel
U.S. Patent and Trademark Office
efileSO@uspto.gov

I also hereby certify that on February 18, 2025, this **PATENT OWNER'S NOTICE OF APPEAL** and the requisite docketing fees were filed with the Clerk's Office of the United States Court of Appeals for the Federal Circuit via CM/ECF.

I further hereby certify that on February 18, 2025, this **PATENT OWNER'S NOTICE OF APPEAL** was served by electronic mail on the following counsel for Petitioner:

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Dated: February 18, 2025

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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

BAYER CROPSCIENCE LP, MONSANTO COMPANY, and
INARI AGRICULTURE, INC.,¹
Petitioners,

v.

CORTEVA AGRISCIENCE LLC,
Patent Owner.

IPR2023-01038
Patent 11,299,745 B2

Before MICHAEL J. FITZPATRICK, ROBERT A. POLLOCK, and
TIMOTHY G. MAJORS, *Administrative Patent Judges*.

MAJORS, *Administrative Patent Judge*.

JUDGMENT
Final Written Decision
Determining All Challenged Claims Unpatentable
35 U.S.C. § 318(a)
Granting Patent Owner's Motions to Seal (Papers 28, 33, 38)
37 C.F.R. § 42.14

¹ The Board joined Inari Agriculture, Inc. as a party (Petitioner) to this proceeding via a Motion for Joinder in IPR2024-00181. Paper 20.

I. INTRODUCTION

Bayer CropScience LP and Monsanto Company filed a Petition (Paper 2, “Pet.”) requesting *inter partes* review of claims 1–29 of U.S. Patent No. 11,299,745 B2 (Ex. 1003, “the ’745 patent”). Pet. 1, 16. Before institution, Corteva Agriscience LLC (“Patent Owner” or “Corteva”) filed a Preliminary Response (Paper 8). Petitioner also filed a Preliminary Reply (Paper 9) and Patent Owner filed a Preliminary Sur-reply (Paper 10).

We instituted trial on December 19, 2023. Paper 12 (“Inst. Dec.”). We later joined Inari Agriculture, Inc. (“Inari”) as a party. Paper 20. We refer to Bayer CropScience LP, Monsanto Company, and Inari collectively as “Petitioner” in this proceeding.

During trial, Patent Owner filed a Patent Owner Response. Paper 22 (“PO Resp.”). Petitioner filed a Reply (Paper 27 (“Pet. Reply”)) and Patent Owner filed a Sur-reply (Paper 34 (“PO Sur-reply”)). We held an oral hearing on September 24, 2024, and a transcript of that hearing is of record. Paper 42 (“Tr.”).

Patent Owner’s unopposed motions to seal also remain pending (Papers 28, 33, and 38). We resolve those motions in Section IV below.

We have jurisdiction under 35 U.S.C. § 6(b). After considering the full trial record, we determine that Petitioner has proved by a preponderance of the evidence that the challenged claims are unpatentable. *See* 35 U.S.C. § 316(e). Our reasoning is explained below, and we issue this Final Written Decision under 35 U.S.C. § 318(a).

II. BACKGROUND

A. *Real Parties-in-Interest*

Bayer CropScience LP and Monsanto Company identify themselves as real parties-in-interest; they also state that counsel for Bayer Intellectual

Property GmbH and Bayer U.S. LLC have been involved in this proceeding and that those Bayer entities are wholly-owned subsidiaries of Bayer AG.

Pet. 3. Petitioner Inari identifies itself as a real party-in-interest. IPR2024-00181, Paper 2, 3. Patent Owner identifies itself as the real party-in-interest. Paper 4, 1.

B. Related Matters

The parties identify the following lawsuit involving assertion of the '745 patent: *Corteva Agriscience LLC v. Monsanto Co.*, 22-cv-01046 (D. Del.); Pet. 4; Paper 4, 1.

The parties also identify related matters before the Board. Pet. 4; Paper 4, 1–2. Those matters include IPR2023-01036 and IPR2023-01037, in which Petitioner challenges, respectively, claims of U.S. Patent Nos. 10,947,555 (“the ’555 patent”) and 11,149,283 (“the ’283 patent”).² Pet. 4; Paper 4, 1–2. The ’283 and ’745 patents issued from continuation applications to the application that issued as the ’555 patent. Ex. 1003, code (63); Ex. 1002 (’283 patent), code (63); Ex. 1001 (’555 patent), code (21). Final Written Decisions in IPR2023-01036 and IPR2023-01037 are issued concurrent with this Decision.

Ex Parte Reexamination Control No. 90/019,130, filed by Inari, also involves the ’555 patent. Pet. 4; Paper 4, 2.

C. The ’745 Patent & Technology Background

The ’745 patent, titled “Herbicide Resistance Genes,” issued April 12, 2002. Ex. 1003, codes (45), (54). The application that matured into the ’745 patent was filed August 16, 2021, and claims the priority benefit of

² Inari was also joined as a petitioner in IPR2023-01036 and IPR2023-01038 via Motions for Joinder filed in IPR2024-00179 and IPR2024-00181.

several earlier applications. *Id.* at codes (22), (63). Those applications include U.S. Application No. 15/288,406 (filed October 7, 2016, which issued as the '555 patent), U.S. Application No. 11/587,893 (filed as PCT/US2005/014737 on May 2, 2005, and published as WO2005/107437 on November 17, 2005) and U.S. Provisional Application No. 60/567,052, filed April 30, 2004. *Id.*; Ex. 1021 (“Corteva ’437”), codes (22), (43).

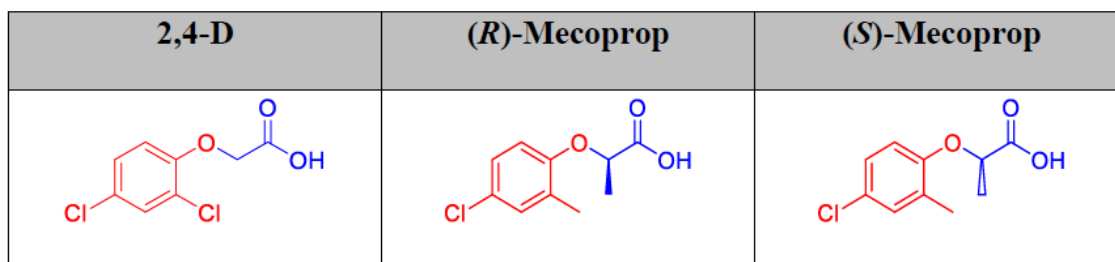
As the '745 patent explains, “[w]eeds can quickly deplete soil of valuable nutrients needed by crops” and “many different types of herbicides” are “used for the control of weeds.” Ex. 1003, 1:37–40. “One extremely popular herbicide is glyphosate.” *Id.* at 1:40. The patent explains that crops such as corn and soybean have been developed that are resistant to glyphosate, allowing glyphosate-resistant crops to be sprayed for weed control without significantly damaging the crop plants. *Id.* at 1:41–45. With glyphosate’s extensive use over time, however, “glyphosate resistant weeds” have emerged, including weeds of the broadleaf (dicot) and grass (monocot) variety. *Id.* at 1:46–2:35.

According to the '745 patent, in areas with glyphosate-resistant weeds, “growers can compensate for glyphosate’s weaknesses by tank mixing or alternating with other herbicides that will control the missed weeds.” *Id.* at 2:36–40. One popular herbicide is 2,4-dichlorophenoxyacetic acid (also known as “2,4-D”). *Id.* at 2:40–57 (noting 2,4-D’s use for “broadleaf weed control for more than 60 years,” but citing the sensitivity of broadleaf crops like soybean to 2,4-D as a “limitation” to its use); *see also id.* at 2:66–67 (“2,4-D has different levels of selectivity on certain plants (e.g., dicots are more sensitive than grasses).”), 2:59–63 (“2,4-D has been used in many monocot crops (such as corn, wheat, and rice) for the selective

control of broadleaf weeds without severely damaging the desired crop plants.”).

As disclosed in the ’745 patent, “2,4-D is in the phenoxy acid class of herbicides, as are MCPA, mecoprop, and dichlorprop.” *Id.* at 2:58–59. The patent describes these herbicides as part of a class called “phenoxy auxins.” *See, e.g., id.* at 3:65–67. According to the patent, “2,4-D is a synthetic auxin derivative that acts to deregulate normal cell-hormone homeostasis and impede balanced, controlled growth.” *Id.* at 2:62–65; Ex. 1054 ¶ 39 (testimony of Dr. Robert Hausinger) (“2,4-D is a widely used herbicide that acts by mimicking indolacetic acid, a naturally occurring plant growth hormone also known as an ‘auxin.’”).

As illustrative of the “phenoxy auxins,” the chemical structures of 2,4-D and mecoprop are depicted below.



Ex. 1054 ¶ 41; *see also* Ex. 1003, 8–11 (Table 1 (listing chemical name and structures for several phenoxy auxins)). The image above shows the structures of 2,4-D as well as two stereoisomeric or “enantiomeric” forms of mecoprop—(R)-Mecoprop and (S)-Mecoprop. Ex. 1054 ¶ 41. The image above, as highlighted by Dr. Hausinger, shows the sub-structures as including phenoxy (red) and carboxylic acid (blue) groups. *Id.* ¶¶ 41–42.

Some phenoxy auxins exist as enantiomers—“pairs of compounds that are mirror-images of one another, like a right hand and a left hand,” also known as “chiral” compounds. *Id.* ¶ 40. This can be seen in the image

above for mecoprop, with (*R*)-Mecoprop having a methyl (CH₃) group depicted as extending in front of the flat plane of the page (solid blue triangle); conversely, (*S*)-Mecoprop has a corresponding methyl group extending behind the plane of the page (hollow blue triangle). *Id.* The “(*R*)-enantiomers of these herbicides are herbicidally active, while the (*S*)-enantiomers are not.” *Id.* ¶ 41; *see also* Ex. 1003, 8:21–23 (describing the “*R*-enantiomers” as the “herbicidally active isomers” of “the chiral phenoxy auxins”). In contrast to mecoprop and other phenoxy auxins, 2,4-D is not chiral. Ex. 1054 ¶¶ 41–42.

The ’745 patent discloses that various bacteria have developed an efficient pathway for degradation of 2,4-D. Ex. 1003, 3:9–15 (noting degradation results in the “complete mineralization of 2,4-D”). “One organism that has been extensively researched for its ability to degrade 2,4-D is *Ralstonia eutropha*” and the “gene that codes for the first enzymatic step in the mineralization pathway is *tfdA*.” *Id.* at 3:19–23. According to the patent, the enzyme encoded by this gene, “TfdA[,] catalyzes the conversion of 2,4-D acid to dichlorophenol (DCP) via an α -ketoglutarate-dependent dioxygenase^[3] reaction.” *Id.* at 3:21–30 (noting “TfdA has been used in transgenic plants to impart 2,4-D resistance in dicot plants (e.g., cotton and tobacco) normally sensitive to 2,4-D”).

³ Papers and evidence herein describe various enzymes (such as TfdA, RdpA, SdpA, and TauD) as being part of a α -ketoglutarate-dependent dioxygenase family or subfamily. *See, e.g.*, PO Resp. 13–14; Ex. 2015 ¶¶ 60–63. To degrade 2,4-D, such enzymes require the presence of iron (Fe(II)), oxygen (O₂) and α -ketoglutarate as a co-substrate. PO Resp. 13–14 (citing Ex. 2001, 24316, Fig. 11 (model of TfdA active site indicating potential roles for histidine residues)). As short-hand, we may refer to these enzymes as “ α KGD enzymes” or “ α KGD dioxygenases” in this Decision.

Moreover, the '745 patent discloses, “[a] large number of tfdA-type genes that encode proteins capable of degrading 2,4-D have been identified from the environment and deposited into the Genbank database.” *Id.* at 3:31–33. Some of these homologues, the patent explains, are “similar to tfdA, (>85 amino acid identity),” with similar properties; other homologues “have a significantly lower identity to tfdA (25-50%), yet have the characteristic [amino acid] residues associated with α -ketoglutarate dioxygenase Fe^{+2} dioxygenases.” *Id.* at 3:36–39. According to the patent, “[i]t is therefore not obvious what the substrate specificities of these divergent dioxygenases are.” *Id.* at 3:39–41.

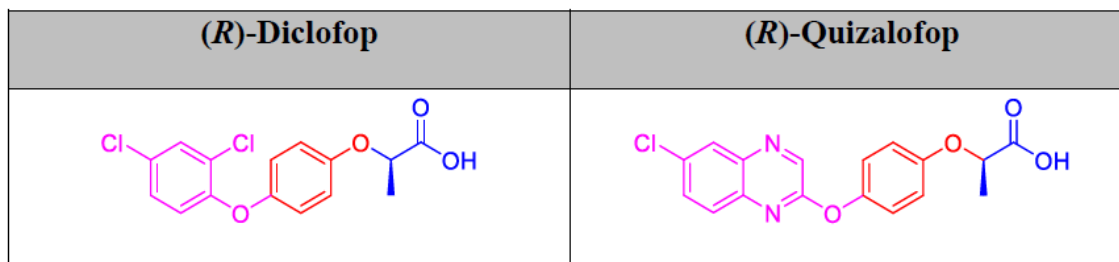
The '745 patent discloses that one known gene with “low homology to tfdA (28% amino acid identity) is rdpA” from the bacterium *Sphingobium herbicidovorans*. *Id.* at 3:42–46. The patent explains that “[t]his enzyme has been shown to catalyze the first step in (R)-dichlorprop (and other (R)-phenoxypropionic acids) as well as 2,4-D (a phenoxyacetic acid) mineralization.” *Id.* Yet, the patent discloses, “[h]eterologous expression of rdpA in other microbes, or transformation of this gene into plants, has not heretobefore been reported” and the “[l]iterature has focused primarily around close homologues of tfdA that primarily degrade achiral phenoxyacetic acids (e.g., 2,4-D).” *Id.* at 3:54–58.

The '745 patent discloses that “[a]ryloxyalkanoate chemical substructures are a common entity of many commercialized herbicides.” *Id.* at 3:65–4:5. Those herbicides include phenoxy auxins (e.g., 2,4-D), pyridyloxy auxins (e.g., fluroxypyr), and aryloxyphenoxypropionates (“AOPP”) acetyl-coenzyme A carboxylase inhibitors (e.g., quizalofop), among others. *Id.* “However,” the patent states, “these classes of herbicides are all quite distinct, and no evidence exists in the current literature for

common degradation pathways among these chemical classes.” *Id.* at 4:6–8. The patent states that “[d]iscovery of a multifunctional enzyme for the degradation of herbicides covering multiple modes would be both unique and valuable as an HTC [(herbicide-tolerant crop)] trait.” *Id.* at 4:8–11.

According to the ’745 patent, the invention relates to “novel plants that are not only resistant to 2,4-D, but also to AOPP herbicides.” *Id.* at 4:15–17. The patent states that, before the invention, “there was no expectation or suggestion that a plant with both of these advantageous properties could be produced by the introduction of a single gene.” *Id.* at 4:16–18. More specifically, the patent explains, “the subject invention relates to the use of an enzyme that is capable of degrading both 2,4-D and AOPP herbicides, in an enantiospecific manner. No α -ketoglutarate-dependent dioxygenase enzyme has previously been reported to have the ability to degrade herbicides of different chemical classes and modes of action.” *Id.* at 4:53–58. The patent indicates that “[t]he preferred enzyme and gene for use according to the subject invention are referred to . . . as AAD-1 (AryloxyAlkanoate Dioxygenase).” *Id.* at 4:58–60. And, the patent states, “[t]here was no prior motivation to produce plants comprising the AAD-1 gene . . . and there was no expectation that such plants could effectively produce an AAD-1 enzyme to render the plants resistant to not only phenoxy acid herbicides (such as 2,4-D) but also AOPP herbicides (such as quizalofop, haloxyfop, et al.).” *Id.* at 4:63–5:3.

The chemical structure for illustrative AOPP herbicides, also called “FOP” herbicides, is shown below.



Ex. 1054 ¶ 42; *see also* Ex. 1003, 11–16 (Table 2 (providing names and structures for many FOP herbicides)). The above image shows the structures for (R)-Diclofop and (R)-Quizalofop. Ex. 1054 ¶ 42. As highlighted by Dr. Hausinger, the two depicted FOPs include phenoxy (red) and carboxylic acid (blue) groups, similar to 2,4-D and mecoprop, discussed above. *Id.* The FOPs, however, include additional aryloxy structures shown in purple. *Id.* The two depicted FOPs are chiral compounds—the (R)-enantiomers are shown above. “Similar to the chiral phenoxy auxins mecoprop and dichlorprop, the (R)-enantiomers” of FOP herbicides “are herbicidally active, while the (S)-enantiomers are not.” *Id.*

As a preferred version of its AAD-1 enzyme, the ’745 patent identifies AAD-1 (v1), which is SEQ ID NO: 9—the native amino acid sequence expressed by the *rdpA* gene of *S. herbicidovorans*. Ex. 1003, 7:6–8, 139–141 (sequence information). The patent also identifies two AAD-1 variants as SEQ ID NO: 10 (AAD-1 (v2)) and SEQ ID NO: 11 (AAD-1 (v3)). *Id.* at 7:9–13 (describing AAD-1 (v3) as a plant-optimized sequence). The sequences of AAD-1 (v2) and AAD-1 (v3) differ from AAD-1 (v1) by two and one amino acid(s), respectively. *See id.* at 141–145 (sequence listings for SEQ ID NO: 10 and SEQ ID NO: 11, noting differences from v1).⁴ The

⁴ Compared to AAD-1 (v1), AAD-1 (v2) substitutes an alanine residue for histidine at amino acid position 2, and a cysteine substituted for arginine at

patent gives examples of *in vitro* and *in vivo* testing, including AAD-1 transformation in several plants and testing for expression of active protein and herbicidal tolerance. *See, e.g., id.* at 78:11–93:23 (Examples 7–9, related to transformations in maize/corn).

D. Illustrative Claims

Independent claim 1, reproduced below, is illustrative of the challenged claims. It reads:

1. A transgenic soybean plant cell comprising a recombinant polynucleotide that encodes an AAD-1 protein that exhibits aryloxyalkanoate dioxygenase activity wherein said activity enzymatically degrades a phenoxy auxin herbicide and an (R)-aryloxyphenoxypropionate herbicide, which are aryloxyalkanoate herbicides, further wherein said AAD-1 protein comprises:

- (i) an amino acid sequence having at least 85% sequence identity with SEQ ID NO: 9; and
- (ii) an AAD-1 motif having the general formula of:

HX₁₁₂D(X)₁₁₄₋₁₃₇T(X)₁₃₉₋₂₆₉H(X)₂₇₁₋₂₈₀R (SEQ ID NO: 34),
wherein

X₁₁₂ represents a single amino acid at position 112, relative to the sequence of SEQ ID NO: 9;

(X)₁₁₄₋₁₃₇ represents a sequence of 24 amino acids;

(X)₁₃₉₋₁₆₉ represents a sequence of 131 amino acids; and

(X)₂₇₁₋₂₈₀ represents a sequence of 10 amino acids,

wherein said AAD-1 protein when expressed in a soybean plant cell renders said soybean plant cell tolerant to a phenoxy auxin herbicide and an (R)-aryloxyphenoxypropionate herbicide as compared to an untransformed soybean plant cell.

position 212. Ex. 1054 ¶ 81. AAD-1 (v3) differs from AAD-1 (v1) based on an alanine insertion at position 2. *Id.*

Ex. 1003, 167:9–33 (reproduced with minor formatting changes).

Illustrating some of the dependent claims, claim 2 depends from claim 1 and adds “wherein said AAD-1 motif has the general formula of: $\text{HX}_{112}\text{D}(\text{X})_{114-137}\text{T}(\text{X})_{139-269}\text{H}(\text{X})_{271-280}\text{R},(\text{X})_{282-284}\text{R}$ (SEQ ID NO: 35), wherein $(\text{X})_{282-284}$ represents a sequence of 3 amino acids.” *Id.* at 167:26–30.

Independent claim 29 is similar to claim 1 and further recites that the AAD-1 motif “has 90% sequence identity with corresponding amino acids of position 111 to 281 of SEQ ID NO: 9.” *Id.* at 168:64–169:23.

Before moving on, a brief discussion about “motif” terminology (as claimed and as discussed in the art) may be helpful.⁵ With the exception of “X,” the alphabetical letters represent *specific* amino acids. Ex. 1054 ¶¶ 49–50. For example, “H” corresponds to histidine (His), “D” is aspartate (Asp), and “R” is arginine (Arg); a recitation of, for example, “D/E” would mean that the amino acid could be either aspartate or glutamate (Glu). *Id.* ¶ 49; *see also id.* ¶ 77 (noting the twenty naturally occurring amino acids). The letter “X” can be *any* amino acid. *Id.* ¶ 49.

The numerical subscripts in a motif may refer to the *position* of the amino acid(s) within a sequence (or relative to some reference sequence), and this is how the subscripts are used in claim 1.⁶ So, for example, X_{112}

⁵ Dr. Hausinger provides further explanation about motif terminology as used with the claimed “AAD-1 motif” and a “consensus motif” for α -KGD dioxygenases discussed in the parties’ papers. Ex. 1054 ¶¶ 49–50, 63–66. The “consensus motif” is discussed in more detail below. Patent Owner’s expert, Dr. Bollinger, states that skilled artisans “recognized amino acid sequence patterns—known as motifs—in enzymes to identify elements of their structures that are essential for their functions.” Ex. 2015 ¶ 39.

⁶ In related litigation, the parties appear to disagree whether the claimed subscripts require fixed positions for the motif amino acids of the AAD-1 protein or, instead, provide position reference points in SEQ ID NO: 9 for

corresponds to an unspecified amino acid “X” at position 112 within the protein sequence of SEQ ID NO: 9, which sequence is 295 amino acids long. *See* Ex. 1003, 139–141.

Alternatively, a motif may use subscripts to represent the *number* of amino acids within a segment of the motif. For example, for a motif reciting HX(D/E)X_{138–207}HX_{10–13}R, the terms X_{138–207} and X_{10–13} indicate a segment of the motif with between 138 to 207 unspecified amino acids and a subsequent segment with between 10 to 13 unspecified amino acids. In this format, the position of the motif’s amino acids within a broader consecutively-numbered amino acid sequence is not given. Ex. 1054 ¶¶ 49–50, 66 (testifying this terminology “specifies a pattern of amino acids, but does not provide information about their positions”). And, in this example, it is clear that the subscripts relate to the number of amino acids rather than their positions because the second subscript range includes lower numbers (i.e., 10–13) compared to the first subscript range (i.e., 138–207).

E. Prosecution History

We provide an overview of the prosecution history that gives context for arguments raised in this case, which overview focuses primarily on prosecution of the ’555 patent (i.e., the great-grandparent to the ’745 patent).

The claims of the ’555 and ’745 patents are similar, except that the ’555 patent’s broadest claims are directed to transgenic “plant cell[s]” or “plants” while corresponding claims of the ’745 patent concern transgenic

alignment with motif residues of the AAD-1 protein to determine the degree of sequence identity. Ex. 1056 (Jt. Claim Construction Chart). The parties do not, however, propose any claim construction here and it is not evident that the claims must be further construed to resolve the dispute before us. *See infra* Section III.C.

“soybean plant cell[s]” or “soybean plants.” *Compare* Ex. 1001, 165:2–19, 36–38, *with* Ex. 1003, 167:9–34, 38–41. Our overview of the relevant prosecution history below starts with the ’555 patent’s prosecution then moves to the more abbreviated prosecution of the ’745 patent.

I. Prosecution of the ’555 patent

On October 7, 2016, applicant filed U.S. Application No. 15/288,406 (“the ’406 Application”), which later issued as the ’555 patent. Ex. 1008 (file history for the ’555 patent). The ’406 Application claims priority through a series of earlier-filed applications—the earliest of which include a non-provisional application filed in May 2005, and a provisional application filed in April 2004. *See* Inst. Dec. 27 (depicting the claimed priority chain (as provided by Patent Owner)).

The ’406 Application was filed with a preliminary amendment that canceled claims 1–14, and added claims 15–48. Ex. 1008, 3, 5–10, 194–195 (original claims 1–14).⁷ Application claim 15 corresponds to issued claim 1 of the ’555 patent (Ex. 1001, 165:2–19), and its language is nearly identical. Ex. 1008, 5.⁸

In an Office Action dated November 13, 2018, the Examiner rejected the claims for, *inter alia*, lack of written description, anticipation based on Wright (the European counterpart of Corteva ’437), and obviousness over

⁷ Exhibit 1008 was submitted in nine parts. The pages of Exhibit 1008 include Bates numbering (e.g., COR-AAD1-00000005). We use a simplified citation format; for example, COR-AAD1-00000005 is cited as “Ex. 1008, 5,” COR-AAD1-00000655 would be “Ex. 1008, 655,” and so on.

⁸ Some of the subscripts in claim 15 were later amended to correct an apparent error. Ex. 1008, 481 (e.g., changing (X)₁₃₉₋₂₄₃ to read (X)₁₃₉₋₂₆₉, consistent with motif’s “general formula” recited earlier in the claim).

Jepson (Ex. 1030) and Kohler (Ex. 1016). Ex. 1008, 267–278. The written description rejection (and similar priority-date determination) related to the Examiner’s finding that the Specification of the pending application (as well as the priority applications) did not describe the newly-claimed “AAD-1 motif.” *Id.* at 270–271, 274–275. The Examiner found that the “instant specification refers to an α -ketoglutarate-dependent dioxygenase motif on page 17, paragraph 0085, but there is no description of a specific AAD-1 motif.” *Id.* (“Applicant does not describe the claimed genus of AAD-1 proteins or the AAD-1 motif (in the specification).”). The Examiner also questioned the sufficiency of the Specification’s description of the claimed AAD-1 protein “having at least 85% sequence identity” with SEQ ID NO: 9 because, the Examiner found, the applicant described AAD-1 (v1) (native RdpA/SEQ ID NO: 9) and only two variants “which are 98.8% and 99.7% identical” to SEQ ID NO: 9. *Id.* at 274–275.

An interview was held March 19, 2019. *Id.* at 452. The Interview Summary states that a correlation between the “general motif” and the “AAD-1 motif” was discussed, that applicant presented evidence in an “Attachment” about how applicant had “modified [the] required amino acids in the AAD-1 motif, demonstrating that these amino acids are critical for maintaining the claimed function,” and that “[n]o agreement was reached concerning the ‘at least 85% sequence identity’ limitation.” *Id.*; *see also id.* at 455 (“Attachment” to Interview Summary; slide titled “AAD-Variants Verifying Motif Validity”).

On April 12, 2019, applicant filed a response to the rejections. With its response, applicant amended paragraph [0086] of the Specification. *Id.* at 480. That amendment is reproduced below with underlining showing text the applicant added, and we also include paragraph [0085] for context:

[0085] The subject invention relates in part to the identification of an enzyme that is not only able to degrade 2,4-D, but also surprisingly possesses novel properties, which distinguish the enzyme of the subject invention from previously known *tfdA* proteins, for example. Even though this enzyme has very low homology to *tfdA*, the genes of the subject invention can still be generally classified in the same overall family of α -ketoglutarate-dependent dioxygenases. This family of proteins is characterized by three conserved histidine residues in a “HX(D/E)X₂₃₋₂₆(T/S)X₁₁₄₋₁₈₃HX₁₀₋₁₃R” motif which comprises the active site. The histidines coordinate Fe⁺² ion in the active site that is essential for catalytic activity (Hogan *et al.*, 2000). The preliminary *in vitro* expression experiments discussed herein were tailored to help select for novel attributes.

[0086] More specifically, the subject invention relates in part to the use of an enzyme that is not only capable of degrading 2,4-D, but also AOPP herbicides. No α -ketoglutarate-dependent dioxygenase enzyme has previously been reported to have the ability to degrade herbicides of different chemical classes and modes of action. Preferred enzymes and genes for use according to the subject invention are referred to herein as AAD-1 (AryloxyAlkanoate Dioxygenase) genes and proteins. As disclosed herein a peptide of SEQ ID NO: 9 is provided that is capable of degrading 2,4-D, and AOPP herbicides. Mapping the α -ketoglutarate-dependent dioxygenase motif disclosed in the previous paragraph to the sequence of SEQ ID NO: 9 reveals the AAD-1 motif of HX₁₁₂D(X)₁₁₄₋₁₃₇T(X)₁₃₉₋₂₆₉H(X)₂₇₁₋₂₈₀R, wherein

X₁₁₂ represents a single amino acid at position 112, relative to the sequence of SEQ ID NO: 9;

(X)₁₁₄₋₁₃₇ represents a sequence of 24 amino acids;

(X)₁₃₉₋₂₆₉ represents a sequence of 131 amino acids; and

(X)₂₇₁₋₂₈₀ represents a sequence of 10 amino acids.

Id.

Applicant also responded with argument and evidence in an effort to traverse the written description and obviousness rejections. Ex. 1008, 486–

494.⁹ Applicant argued it had “described the complete amino acid sequence of AryloxyAlkanoate Dioxygenase (AAD-1) proteins (SEQ ID Nos: 9, 10, and 11) that have been found to exhibit activity in degrading 2,4-D as well as AOPP herbicides” and had “derived a specific ‘AAD-1 motif.’” *Id.* at 486–487 (arguing the motif was derived by “mapping” SEQ ID NO: 9 to the disclosed α KGD dioxygenase motif). Applicant argued “[o]ne of ordinary skill in the art would know that the motif provided on page 17 in paragraph [0085] provides a roadmap to identify the corresponding positions in the AAD-1 proteins of SEQ ID Nos: 9, 10, and 11 to allow one to generate a ‘AAD-1 motif’” as claimed and “that is specific to the claimed genus of AAD-1 proteins.” *Id.* at 488–489 (arguing the claims were “narrowly tailored . . . by requiring the claimed peptides to comprise the functional element of the AAD-1 motif” (i.e., dual enzymatic degradation of phenoxy auxins and (*R*)-FOPs)).

Applicant also filed a declaration from named inventor Dr. Terry Wright, which addressed the claimed AAD-1 motif and described an experiment showing the “importance and necessity” of the motif’s amino acid residues to the dual herbicide degradation activity. *Id.* at 463, 489; *id.* at 457–463 (“Wright I”), 472–474 (App’x III about testing of nine variants of AAD-1 v1 (similar to the aforementioned “Attachment” slide discussed at the March 19, 2019, interview)). For obviousness, applicant argued that a rationale for selecting the *rdpA* gene (of Kohler) and substituting it for the *tfdA* gene (of Jepson) was missing from the pending rejection. *Id.* at 490–

⁹ Applicant also argued that, upon traversing the § 112 rejection, the claims would be entitled to an earlier priority date and, thus, the asserted anticipatory references would not be prior art. Ex. 1008, 493–494.

493. Moreover, applicant argued, the invention produces surprising results insofar as the AAD-1 enzymes degraded herbicides in two different classes and modes of action—phenoxy auxins and (*R*)-FOPs. *Id.*

On July 12, 2019, the Examiner entered a Final Office Action, maintaining, in substantial part, the Examiner’s prior reasoning for rejecting the claims. Ex. 1008, 496–515. The Examiner further determined that the “generic” α KGD dioxygenase motif “does not itself define a specific function” and the Specification “does not describe what structure in the AAD-1 motif provides the specific activity that degrades a phenoxy auxin and an (*R*)-aryloxyphenoxypropionate herbicide.” *Id.* at 505. Moreover, the Examiner explained, applicant failed to describe adequately the variation within the broadly-claimed genus. *Id.* at 505–506.

Applicant then filed a Request for Continued Examination. Ex. 1008, 557–582. Applicant also submitted a declaration from Dr. Satish Nair. *Id.* at 590–598. Dr. Nair testified that a skilled person “would readily envision a disclosed AAD-1 motif” from a mapping of the generic α KGD dioxygenase motif against SEQ ID NO: 9, and that the AAD-1 motif “is readily apparent” from the Specification and background knowledge. *Id.* at 593–594. Dr. Nair cited an investigation of crystal structures for AAD-1 and AAD-2 proteins¹⁰ published in 2019 as validating the importance of the AAD-1

¹⁰ AAD-2 is another gene/protein described in the ’555 patent. Ex. 1001, 111:9–112:41 (“Another gene was identified . . . as a homologue with only 44% amino acid identity to *tfdA*.”); *id.* at 6:66–67 (“SEQ ID NO: 13 is the amino acid sequence for AAD-2 (v1) protein”). Dr. Nair testified AAD-2 has conserved amino acids within the generic α KGD dioxygenase motif, but is an (*S*)-specific enantiomer dioxygenase that does not degrade (*R*)-phenoxy auxins or (*R*)-FOPS. Ex. 1008, 594–595; Ex. 1001, 112:58–61 (citing data

motif residues and their spacing for AAD-1's dual-enzymatic activity. *Id.* at 594–595 (citing Ex. 1019, results of investigation by Drs. Nair and Wright).

On March 10, 2020, the Examiner again rejected the claims for lack of written description and for obviousness, raising similar points as discussed previously. Ex. 1008, 629–642 (withdrawing the § 102 rejections without substantive comment). *Id.* at 631. That Office Action prompted another interview and written response from the applicant. *Id.* at 650, 676–686.

In its September 9, 2020, response, applicant again argued that its claims were patentable. *Id.* at 676–686. Applicant argued it had disclosed common structural features for the genus claims, including the “AAD-1 motif” and SEQ ID NOs: 9, 10, and 11. *Id.* Applicant, citing a second declaration from Dr. Wright, also argued that applicant had conducted tests showing enzymes with the AAD-1 motif and “as low as 82% sequence identity” to SEQ ID NO: 9 exhibited 2,4-D and (*R*)-FOP degrading activity. *Id.* at 681–682 (discussing testing of eight AAD-1 variant enzymes with sequence identities between 82–90%) ; *see also id.* at 655–670 (“Wright II”). For obviousness, applicant argued there was no reason to combine the cited art, and that unexpected results supported patentability. *Id.* at 682–687.

On December 8, 2020, the Examiner withdrew the written description and obviousness rejections. *Id.* at 3844. For written description, the Examiner cited applicant's additional testing that “demonstrated variants of instant SEQ ID NO: 9 produced by truncations of both the N and C termini that retain the claimed function and that would describe the variation within

“that confirm that AAD-2 (v1) is specific for (*S*)-enantiomer substrates, in contrast to AAD-1 (v1) which is specific for (*R*)-enantiomers.”).

the genus.” *Id.*¹¹ In withdrawing the obviousness rejection, the Examiner cited “the ability of the AAD-1 gene to produce herbicide tolerance in a transgenic plant cell to two distinct types of herbicides.” *Id.*

2. *Prosecution of the '745 patent*

On August 16, 2021, applicant filed U.S. Application No. 17/403,476 (“the ’476 Application”), which later issued as the ’745 patent. Ex. 1011 (file history for the ’745 patent).

In an Office Action dated December 17, 2021, the Examiner rejected one of the pending claims for lack of written description. Ex. 1011, 5357–60 (rejecting pending claim 30). The Examiner found that such claim’s recitation of an amino acid sequence with “at least 85%, but less than 95% sequence identity with SEQ ID NO: 9” was not supported. *Id.* (noting, in a section titled “Priority,” the application’s relatedness to earlier applications but finding it “adds disclosure not presented in the prior applications” and suggesting the application be identified as a continuation-in-part); *see also id.* at 5325 (full text of application claim 30). The Examiner also entered rejections for obviousness (e.g., over Jepson and Kohler) like discussed above for the ’555 patent. *Id.* at 5360–65.

Applicant filed an amendment and response dated January 12, 2022. *Id.* at 5422–37. Applicant amended the Specification like it had done with the ’555 patent as explained above (i.e., adding the sentences about an “AAD-1 motif” allegedly revealed by “mapping” to another motif). *Id.* at 5423–24 (amending paragraph 87). Applicant also amended another

¹¹ Applicant later responded that the “referenced variants contained amino acid modifications throughout their sequences (but outside of the recited AAD-1 motif)—not truncations at the C- and N- termini.” Ex. 1008, 3897.

paragraph of the Specification, which paragraph had, in general, previously listed sequence identities from 49–99%. *Id.* at 5424 (amending paragraph 143). This further amendment added: “For example, in one embodiment an amino acid sequence is provided having at least 85%, but less than 95% sequence identity with SEQ ID NO: 9,” and also added the “general formula” for an “AAD-1 motif” like the other amended portion of the Specification discussed above. *Id.* Applicant then argued that the rejections should be withdrawn in view of the Specification’s disclosures and “surprising advantages” (i.e., degradation of 2,4-D and (R)-FOP herbicides) with the claimed invention. *Id.* at 5430–36.

In an interview, the Examiner proposed canceling claim 30 “to expedite allowance of the remaining claims.” *Id.* at 5466 (Interview Summary). “Applicant’s attorney approved [that] cancellation.” *Id.*

A Notice of Allowance was entered February 15, 2022. *Id.* at 5439–45. By an Examiner’s Amendment, claim 30 was canceled and the Examiner included no further substantive comment or specific reasons for allowing the claims. *Id.*

F. Prior Art and Asserted Grounds

Petitioner asserts that claims 1–29 are unpatentable based on the following grounds:

Claims Challenged	35 U.S.C. § ¹²	References/Basis
1–29	102	Corteva ’437 ¹³
1–29	103	Corteva ’437
1–6, 9–12, 29	103	Saari 1998, ¹⁴ Saari 1999, ¹⁵ Schleinitz, ¹⁶ Streber ¹⁷
7, 8, 13–28	103	Saari 1998, Saari 1999, Schleinitz, Streber, Jepson ¹⁸

Petitioner submits testimony from Robert P. Hausinger, Ph.D., in support of its challenge. Ex. 1054 (Hausinger Decl.); Ex. 1070 (Hausinger

¹² The Leahy-Smith America Invents Act (“AIA”), Pub. L. No. 112–29, 125 Stat. 284, 285–88 (2011), revised 35 U.S.C. §§ 102, 103 effective March 16, 2013. AIA § 3(b)–(c), (n). Petitioner’s grounds based on Corteva ’437 assume that the claims have a priority date no earlier than October 7, 2016, in which case post-AIA §§ 102, 103 would apply. Pet. 16–17. Petitioner’s other grounds assume a priority date of April 30, 2004, in which case pre-AIA §§ 102, 103 would apply. *Id.*

¹³ Wright et al., WO 2005/107437 A2, publ. Nov. 17, 2005 (Ex. 1021 (“Corteva ’437”)).

¹⁴ R.E. Saari, *Substrate Specificity and Spectroscopic Properties of 2,4-Dichlorophenoxyacetic Acid/α-Ketoglutarate Dioxygenase*, Doctoral Thesis, Mich. State Univ. (July 6, 1998) (Ex. 1040 (“Saari 1998”)).

¹⁵ R.E. Saari, et al., *Stereospecific Degradation of the Phenoxypropionate Herbicide Dichlorprop*, 6 J. Molecular Catalysis B: Enzymatic 421–428 (1999) (Ex. 1041 (“Saari 1999”)).

¹⁶ Accession No. AF516751, publ. Aug. 6, 2002 (Ex. 1013 (“Schleinitz”)).

¹⁷ Streber et al., US 6,153,401, issued Nov. 28, 2000 (Ex. 1043 (“Streber”)).

¹⁸ Jepson et al., US 2003/0041357 A1, publ. Feb. 27, 2003 (Ex. 1030 (“Jepson”)).

Reply Decl.). Patent Owner submits testimony from J. Martin Bollinger, Jr., Ph.D. Ex. 2015 (Bollinger Decl.). The testimony of Drs. Hausinger and Bollinger given during depositions is also of record. Ex. 1072 (Bollinger transcript); Exs. 2016 and 2066 (Hausinger transcripts).

III. ANALYSIS

A. *Legal Standards*

“In an [*inter partes* review], the petitioner has the burden from the onset to show with particularity why the patent it challenges is unpatentable.” *Harmonic Inc. v. Avid Tech., Inc.*, 815 F.3d 1356, 1363 (Fed. Cir. 2016) (citing 35 U.S.C. § 312(a)(3)).

Whether claims are entitled to the priority benefit of an earlier effective filing date is based on disclosure of the invention “in the manner provided by section 112(a)” in a related and earlier-filed application. *See* 35 U.S.C. §§ 119(e), 120. “[A] patent’s claims are not entitled to an earlier priority date merely because the patentee claims priority. . . . Rather, for a patent’s claims to be entitled to an earlier priority date, the patentee must demonstrate that the claims meet the requirements of 35 U.S.C. § 120,” which include satisfying the written description requirement of § 112. *In re NTP, Inc.*, 654 F.3d 1268, 1276–77 (Fed. Cir. 2011) (citations omitted).

“The essence of the written description requirement is that a patent applicant, as part of the bargain with the public, must describe his or her invention so that the public will know what it is and that he or she has truly made the claimed invention.” *AbbVie Deutschland GmbH & Co. v. Janssen Biotech, Inc.*, 759 F.3d 1285, 1298 (Fed. Cir. 2014). “[T]he test for sufficiency [of the written description requirement] is whether the disclosure of the application relied upon reasonably conveys to those skilled in the art that the inventor had possession of the claimed subject matter as of the filing

date sought.” *Ariad Pharm., Inc. v. Eli Lilly and Co.*, 598 F.3d 1336, 1351 (Fed. Cir. 2010) (en banc) (explaining that “the description must clearly allow persons of ordinary skill in the art to recognize that [the inventor] invented what is claimed”) (internal quotation marks omitted).

This “possession” test “requires an objective inquiry into the four corners of the specification from the perspective of a person of ordinary skill in the art.” *Ariad*, 598 F.3d at 1351 (noting that “[t]his inquiry, as we have long held, is a question of fact”). “[I]t is the specification itself that must demonstrate possession” and, although *in ipsius verbis* recitations are not required, “a description that merely renders the invention obvious does not satisfy the requirement.” *Id.* at 1352. Also, possession shown by evidence “outside of the specification is not enough.” *Id.* Indeed, “[b]ecause written description is determined as of the filing date . . . evidence of what one of ordinary skill in the art knew” after the filing date sought “cannot provide substantial evidence . . . that the [] claims were supported by adequate written description.” *Id.* at 1355; *Biogen Int’l GmbH v. Mylan Pharm., Inc.*, 18 F.4th 1333, 1342 (Fed. Cir. 2021) (“[E]vidence illuminating the state of the art subsequent to the priority date is not relevant to written description.”) (internal quotation marks omitted, alteration in original).

To show anticipation under 35 U.S.C. § 102, each and every claim element, arranged as in the claim, must be found in a single prior art reference. *Net MoneyIN, Inc. v. VeriSign, Inc.*, 545 F.3d 1359 (Fed. Cir. 2008).

A claim is unpatentable under 35 U.S.C. § 103 if the differences between the claimed invention and the prior art are such that the claimed invention as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the relevant art. *KSR Int’l Co. v.*

Teleflex Inc., 550 U.S. 398, 406 (2007). The question of obviousness is resolved on the basis of underlying factual determinations including: (1) the scope and content of the prior art; (2) any differences between the claimed subject matter and the prior art; (3) the level of ordinary skill in the art; and (4) secondary considerations of nonobviousness when presented. *Graham v. John Deere Co.*, 383 U.S. 1, 17–18 (1966).

B. Level of Ordinary Skill in the Art

In determining the level of skill in the art, we consider the problems encountered in the art, the art’s solutions to those problems, the rapidity with which innovations are made, the sophistication of the technology, and the educational level of active workers in the field. *Custom Accessories, Inc. v. Jeffrey-Allan Indus., Inc.*, 807 F.2d 955, 962 (Fed. Cir. 1986).

Petitioner proposes that the person of ordinary skill in the art (“POSA”) would “have had knowledge and experience regarding protein biochemistry, including expression and characterization, amino acid sequences, three-dimensional structure, and enzyme activity and substrate selectivity, as well as methods of evaluating those properties.” Pet. 18 (citing Ex. 1054 ¶ 29). Petitioner contends, “[t]he POSA would be able to apply these concepts to expression of enzymes in transgenic plant cells and plants and degradation of herbicides” and “[t]he POSA generally would have an advanced degree in a relevant discipline and several years of advanced academic (e.g., post-doc or higher) and/or professional experience.” *Id.*

Patent Owner responds as follows:

For purposes of this proceeding, a skilled artisan would have typically possessed a Ph.D. in molecular biology, biotechnology, or biochemistry, and 3–5 years of experience in

the development of enzymes and enzyme variants as well as familiarity with α KG-dependent dioxygenases.

PO Resp. 26 (citing Ex. 2015 ¶ 75). Patent Owner contends that its proposed POSA level is “generally consistent” with Petitioner’s definition, and specifies the POSA’s “familiarity with α KG-dependent dioxygenases and structure-function relationships in enzymology.” *Id.* (citing, e.g., Exs. 1023 and 2025).

For purposes of this Decision, we apply Petitioner’s proposed definition of the POSA, with the further elaborations or clarifications suggested by Patent Owner (i.e., Ph.D. or higher educational level in, e.g., molecular biology, and 3–5 years of professional experience related to the development enzymes, including familiarity with α KG-dependent dioxygenases and structure-function relationships). We do not otherwise see a material difference in the parties’ proposals that would change the outcome of this Decision. Our applied POSA definition is also consistent with the level of skill shown in the prior art of record. *See Daiichi Sankyo Co. v. Apotex, Inc.*, 501 F.3d 1254, 1256 (Fed. Cir. 2007). And, with no argument to the contrary, we find that Drs. Hausinger and Bollinger have sufficient qualifications to testify from the perspective of the POSA relevant to the matters in dispute.¹⁹ Ex. 1054 ¶¶ 9–19; Ex. 1070 ¶ 7; Ex. 2015 ¶¶ 2–11.

¹⁹ Patent Owner states that Petitioner does not define any skill level for aspects of the ’745 patent about genetic transformation of plants, and suggests that Dr. Hausinger lacks such expertise. PO Resp. 26. Patent Owner does not, in any event, identify any such plant-based experience that Dr. Bollinger possesses yet Dr. Hausinger does not. Moreover, the alleged omission cited by Patent Owner is not decisive here because the disputed issues related to Grounds 1A and 1B concern enzymology and not, as Patent Owner acknowledges, transgenic-plant aspects of the claims. *Id.*

C. Claim Construction

We construe claims using the same claim construction standard used to construe claims in a civil action before the courts under 35 U.S.C. § 282(b), including construing claims in accordance with the ordinary and customary meaning as understood by the POSA and the patent’s prosecution history. 37 C.F.R. § 42.100(b). We need only construe terms that are in controversy and only as needed to resolve the matters in dispute. *Realtime Data, LLC v. Iancu*, 912 F.3d 1368, 1375 (Fed. Cir. 2019).

Neither party proposes any express claim construction in this proceeding. Pet. 17; *see generally* PO Resp. The plain language of the claims as written controls and it is not necessary to further expressly interpret those claims to resolve the present controversy.²⁰

D. The Corteva ’437 Grounds (Grounds IA & IB)

1. Priority Date of the Challenged Claims and the Prior-Art Status of Corteva ’437

Before turning to whether Corteva ’437 anticipates or renders obvious the challenged claims, we must address the threshold issue whether Corteva ’437 is prior art. According to Petitioner, for reasons discussed in more detail below, the priority date of the challenged claims is no earlier

²⁰ At the pre-institution stage, Patent Owner informed us that the district court had construed the claims in the parallel litigation. Paper 8, 20 n.4. We considered the district court’s order and supporting memorandum (Exs. 3003 and 3004), which does not change our analysis in this Decision. *See* PTAB Consolidated Trial Practice Guide at 49 (Nov. 2019) (“The Board also will consider . . . any prior claim construction determinations from the federal courts and ITC that have timely been made of record, before entering a final written decision that sets forth the final claim construction determination.”).

than October 7, 2016.²¹ Pet. 16, 23–41. October 7, 2016, is the filing date of the application (and accompanying preliminary claim amendment) that later matured into the ’555 patent. *See supra* Section II.E. If the priority date of the challenged claims is no earlier than October 7, 2016, Petitioner contends that Corteva ’437 is 35 U.S.C. § 102(a)(1) prior art as of Corteva ’437’s November 17, 2005, publication date. Pet. 19.²²

Patent Owner responds that Petitioner has not met its burden to show that Corteva ’437 is prior art. PO Resp. 28–60. Rather, Patent Owner contends, the challenged claims are entitled to priority to the May 2, 2005, PCT or April 30, 2004, provisional application filing dates. *Id.* at 28.

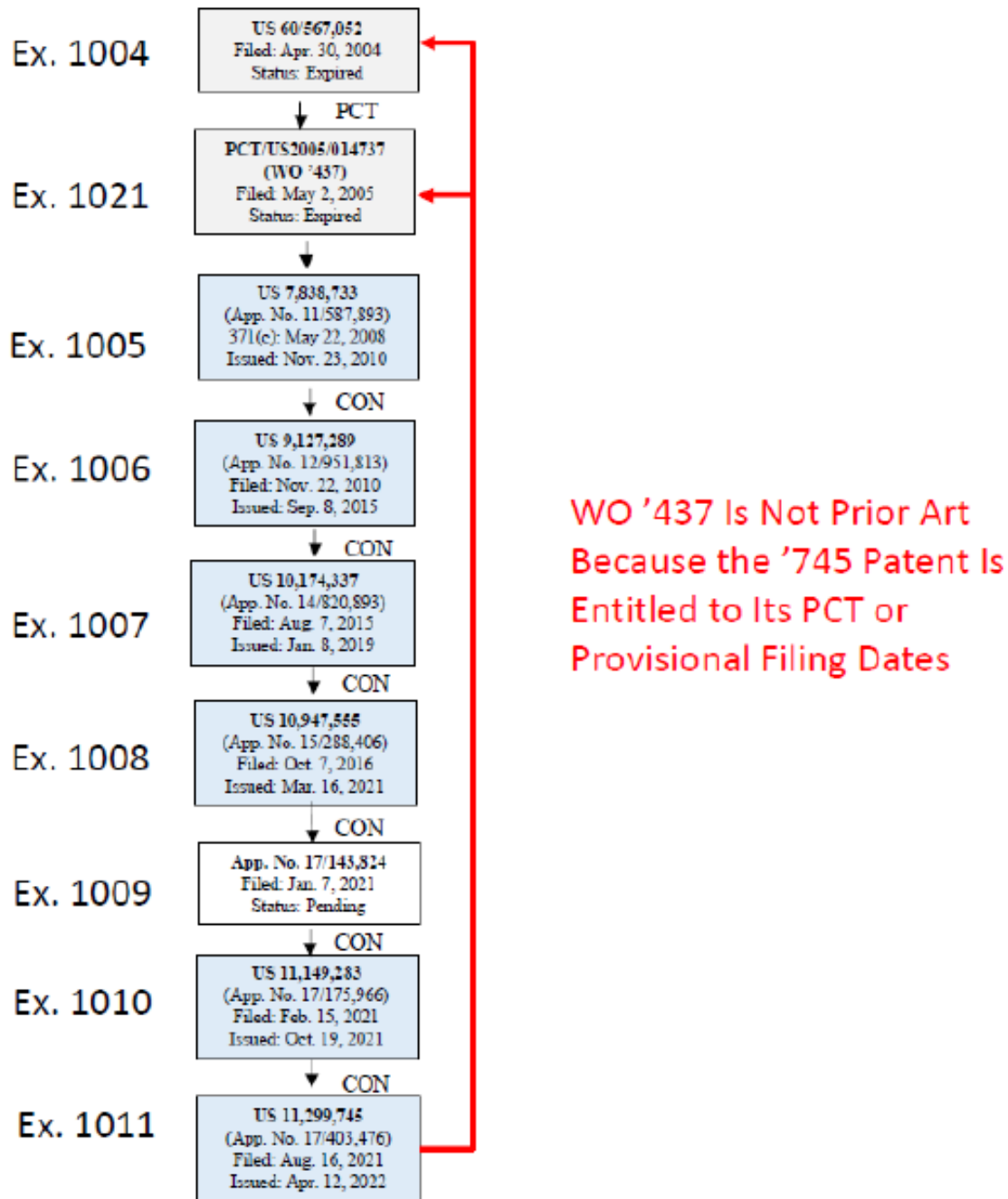
a) The ’745 Patent’s Asserted Priority Chain

“In order to gain the benefit of the filing date of the earlier application . . . each application in the chain leading back to the earlier application must comply with the written description requirement.” *Lockwood v. Am. Airlines, Inc.*, 107 F.3d 1565, 1571 (Fed. Cir. 1997).

The image below is a family tree and asserted priority chain for the ’745 patent.

²¹ For Grounds IA and IB, the Petition refers to both October 7, 2016, and October 17, 2016. *See, e.g.*, Pet. 17 (Oct. 7), 23 (Oct. 17); *see also* Ex. 1054 ¶¶ 60–61 (Oct. 7). The reference to October 17, 2016, appears to be an inadvertent error.

²² As noted in our Institution Decision, Petitioner’s priority arguments center on whether the claimed genus lacks adequate written description support in the priority applications. Inst. Dec. 26 n.20. Petitioner raises no argument based on lack of enablement in this proceeding and we express no opinion on whether the claimed subject matter is enabled.



PO Resp. 29 (Patent Owner's annotated family tree for the '745 patent). The image above shows the '745 patent at the bottom and the earlier-filed applications to which the '745 patent claims priority in a sequential chain above it. At the top is U.S. Provisional Application No. 60/567,052 (Ex. 1004 ("052 Provisional")), filed April 30, 2004, and below the

'052 Provisional is PCT application, PCT/US2005/014737, filed May 2, 2005, which claims priority to the '052 Provisional and which published as Corteva '437 (Ex. 1021). There are six intervening applications shown between the PCT application and the '745 patent—the national stage entry of the PCT application (Ex. 1005) and five continuation applications (Exs. 1006–1010).

If the '745 patent's challenged claims are entitled to priority to the PCT application (filed in 2005) or the '052 Provisional (filed in 2004), then Corteva '437 is not prior art as alleged in the Petition. Pet. 19–20, 23–24. The parties do not identify additional, distinct disclosures (or lack thereof) in the intervening applications (Exs. 1005–1010) that affect the priority analysis. Thus, the dispute here turns on whether the challenged claims have written description support in the PCT application (i.e., Corteva '437).²³

b) Overview of the Parties' Priority Arguments

(1) Petitioner's Arguments

Petitioner argues that the challenged claims lack sufficient written description in the priority applications. Pet. 23–41. According to Petitioner, the priority applications provide insufficient descriptive support for the broad genus of AAD-1 proteins that is claimed due to lack of adequate disclosure of structural features common to the members of the genus or

²³ Petitioner's priority analysis cites paragraphs from the '052 Provisional (Ex. 1004) and paragraphs in the intervening applications in group citation format (e.g., Exs. 1005–1010). *See, e.g.*, Pet. 23, 28–29; *see also* PO Resp. 30 (referring to "Priority Applications" as a group). Exhibit 1005 is the national stage entry of the PCT application and its disclosures are materially identical to the corresponding disclosures in Corteva '437 (Ex. 1021). We may refer to the applications collectively as "the priority applications."

sufficient representative species providing the recited dual-herbicide-degrading functionality. *Id.* at 30–40.

Petitioner argues, *inter alia*, that the priority applications do not describe a genus of proteins defined by an “AAD-1 motif” as recited in all the challenged claims.²⁴ Pet. 24–30. Petitioner contends that the priority applications instead refer to “a broader prior-art motif” characteristic of an “overall family” of α KGD dioxygenases, which motif is “HX(D/E) X_{23-26} (T/S) $X_{114-183}$ HX $_{10-13}$ R.” *Id.* at 24–26 (citing, e.g., Ex. 1005 [0085]); Ex. 1054 ¶¶ 64–70, 489 (testifying that the earlier applications disclosed “the same generic consensus motif” for α KGD enzymes “already known and published in the prior art”); Ex. 1021 ¶ 85.

According to Petitioner, this consensus motif is not claim 1’s “AAD-1 motif,” which applicant first added via claim amendment in 2016 during prosecution of the related ’555 patent. Pet. 24–28. Petitioner contends, for example, that the consensus motif permits the segments of unspecified (“X”) amino acids to vary in length yet the AAD-1 motif requires *specific* numbers of the unspecified amino acids in each segment. *Id.* (noting, for example, X_{23-26} in the consensus motif allows between 23 to 26 amino acids, versus $X_{114-137}$ (with subscripts reflecting *position* information) with exactly 24 amino acids); Ex. 1054 ¶¶ 65–67 (testifying the consensus motif “specifies a pattern of amino acids, but does not provide information about their positions” and noting other differences, including that the second and third conserved amino acids can be (D/E) and (T/S), not restricted to D and T like

²⁴ Except for dependent claim 2, which modifies the claimed AAD-1 motif by adding an extra (X) $_{282-284}$ R subsegment and for which the parties provide additional argument, the parties’ priority arguments treat the challenged claims as a group.

the claimed AAD-1 motif); *see supra* Section II.D (discussing differences in “motif” terminology). Petitioner also asserts that many proteins that have the consensus motif do not have the claimed functional activity against both phenoxy auxins and (*R*)-FOPs—a point applicant emphasized during prosecution. Pet. 26 (citing Ex. 1054 ¶¶ 64, 100–101, 489, 491); *see, e.g.*, Ex. 1008, 487, 491–492.

Petitioner also contends that the alleged “mapping” that applicant argued during prosecution justified adding the recited “AAD-1 motif” and related 2016 amendment of the patent’s Specification is flawed. Pet. 28–30. According to Petitioner, the priority applications disclosed the sequences of three nearly-identical AAD-1 proteins (SEQ ID NOs: 9, 10, 11) but “not a genus of proteins characterized by an AAD-1 motif.” *Id.* at 27–29 (citing, *e.g.*, Ex. 1054 ¶¶ 94–95). That SEQ ID NO: 9 (*i.e.*, RdpA) may be a member of the α KGD enzyme family having amino acid residues within the scope of the α KGD consensus motif was already known in the art, Petitioner contends. *Id.* Thus, Petitioner argues, “mapping” of the consensus motif to SEQ ID NO: 9 provides no new information that describes a new and allegedly functionally-distinct genus of “AAD-1” proteins defined by an “AAD-1 motif,” much less a description that “reveal[s] what structural features within SEQ ID NO: 9 are necessary for the (*R*)-enantioselective activity against chiral phenoxy auxins and aryloxyphenoxypropionates covered by the claims.” *Id.*

Reinforcing the absence of descriptive support for the challenged claims including the “AAD-1 motif” in the priority applications, Petitioner contends that those applications describe sequence variation and amino acid substitution only in “general terms” without reference to any specific sequences, let alone identifying the amino acid positions or segments that

should or should not be varied or how they should be varied. Pet. 29–30 (citing, e.g., Ex. 1005 ¶¶ 138–145). At best, Petitioner contends, the priority applications merely suggest a “research plan” for exploring a broad genus of AAD-1 proteins without adequate description of variations that preserve the claimed functionality—requiring a POSA’s “extensive experimentation” to decipher the same. Pet. Reply 12–14 (asserting the priority applications (e.g., Ex. 1005 ¶¶ 144–145) suggest substitutions *of any type* can be made *so long as the substitutions do not adversely affect activity*); Ex. 1070 ¶¶ 28–32.

Continuing, Petitioner argues that the priority applications fail to support a genus of AAD-1 proteins with as little as 85% sequence identity compared to SEQ ID NO: 9 (i.e., AAD-1 (v1)) as claimed. Pet. 38–40. Petitioner contends that the only variants expressly described are AAD-1 (v2) and AAD-1 (v3), yet those variants differ from SEQ ID NO: 9 by only one or two amino acids (i.e., with $\geq 99\%$ similarity). According to Petitioner, those variants “lack the diversity needed to support the claims.” *Id.* at 38–39 (citing Ex. 1054 ¶¶ 76–86, 491). Further, Petitioner contends, the priority applications’ “laundry list” of possible sequence identity values ranging from 49% to 99% would not reasonably lead the POSA to any particular species and is, therefore, inadequate to describe the claims. *Id.* at 39 (citing, e.g., Exs. 1004–1009 ¶ 142; Ex. 1054 ¶¶ 71, 491).

Petitioner further argues that the priority applications do not disclose a representative number of species to support the genus of AAD-1 proteins having the claimed dual-herbicide-degrading activity. Pet. 30–31. According to Petitioner, the claims, even with the recited “AAD-1 motif” and percentage identity limitations, putatively cover at least 20⁴⁴ variant amino acid sequences—an “enormous number of proteins, which may or may not possess the claimed functional activities.” *Id.* (citing Ex. 1054

¶¶ 67, 74–91).²⁵ Again citing the three nearly-identical proteins that are described (i.e., AAD-1 (v1–v3)), Petitioner contends such limited species do not represent the breadth of the genus of AAD-1 proteins claimed, but instead evidence that the inventors possessed only “a corner” of that genus. *Id.* (citing *AbbVie*, 759 F.3d at 1300).

Petitioner also argues that applicant’s written description arguments made during prosecution were based on extrinsic and post-priority date evidence that cannot support an earlier priority date. Pet. 32–34. According to Petitioner, this evidence includes Dr. Wright’s and Dr. Nair’s testing and analysis of AAD-1 protein variants that took place well over a decade after the claimed 2004/2005 priority date. *Id.* at 32–38. What is most important, Petitioner contends, is the disclosure within the four corners of the priority applications, not later-discovered evidence or the teachings of uncited references. *Id.* at 31–32. Although a POSA’s “background knowledge” is available to inform what is actually disclosed in the priority applications, Petitioner contends that it is improper to use such knowledge to “supplement” or “teach limitations that are not in the specification, even if those limitations would be rendered obvious by the disclosure in the specification.” *Id.*; see also Pet. Reply 9 (quoting *Rivera v. ITC*, 857 F.3d 1315, 1322–23 (Fed. Cir. 2017)).

²⁵ Dr. Hausinger explains that 20⁴⁴ variant proteins is a lower-bound numerical estimate, reflecting a protein that is 295 amino acids long where an 85% sequence identity limitation would permit up to 44 substitutions with any of the twenty naturally-occurring amino acids at unspecified positions within the longer amino acid chain. Ex. 1054 ¶¶ 78–79.

(2) *Patent Owner's Arguments*

Patent Owner argues that the priority applications describe a genus of AAD-1 proteins with sufficient, function-correlated common structures. PO Resp. 28–55. Those common structures include, Patent Owner argues, AAD-1 proteins with at least 85% sequence identity to SEQ ID NO: 9, and an “AAD-1 motif” representing the active site of the genus of AAD-1 enzymes. *Id.* at 30–44.

Patent Owner contends that the priority applications describe a genus of AAD-1 enzymes with high sequence identity as a common, function-correlated structural feature. *Id.* at 30 (citing, e.g., Exs. 1005–1010, 1021 ¶ 95; Ex. 2015 ¶¶ 95–99). According to Patent Owner, the priority applications disclose that “[e]quivalent proteins will have amino acid similarity (and/or homology) with an exemplified protein,” and that the “amino acid identity will typically be at least 60% . . . , more preferably at least 80%, even more preferably at least 90%, and can be at least 95%.” Ex. 1021 ¶ 142 (listing sequence identity values ranging between 49% to 99%); PO Resp. 30. Patent Owner contends that the priority applications characterize other α KGD enzymes with percent identities between 85–95% as being “highly identical” and further state that “[t]raditionally only sequences with high homology would be expected to retain similar properties to the input sequence.” Ex. 1021 ¶ 171; PO Resp. 30–31.

Patent Owner argues the prior art recognized that enzymes with sequence identities of 85% or higher would be expected to exhibit the same function. PO Resp. 31–34. According to Patent Owner, by 2004/2005, research groups had studied various enzyme families and had described a “strong correlation” between high levels of sequence identity and conserved enzyme function. *Id.* at 31–33 (citing, e.g., Ex. 2015 ¶¶ 100–105; Ex. 2019,

864, 872)). Those studies include, for example, Tian,²⁶ which Patent Owner notes had disclosed that “60% sequence identity is needed to have at least 90% accuracy” in predicted function. *Id.* (citing Ex. 2019, 866, Fig. 1(B) (annotated by Patent Owner)); *see also id.* at 5–11 (background about an enzyme classification/coding system promoted by the Enzyme Commission or “EC”); Ex. 2019, 864 (“[S]equence identity is . . . widely used as an indication of functional similarity.”); Ex. 2028,²⁷ 604 (Fig. 5); Ex. 2015 ¶¶ 102–103 (testifying that Rost “found that at least 70% sequence identity was required to predict enzyme function”). According to Patent Owner, Petitioner’s expert fails to account for known structure-function correlations and the POSA’s capacity to predict enzyme function when sequence identity is high. PO Resp. 33–35.

Patent Owner argues that the claimed “AAD-1 motif” is described in the priority applications and was known in the prior art. PO Resp. 38–42. According to Patent Owner, the POSA would have known that each α KGD dioxygenase has a specific motif and the POSA “knew where the motif was located in RdpA/AAD-1—no information beyond its primary amino acid sequence was required.” *Id.* (citing Ex. 2015 ¶¶ 110–113). Patent Owner contends that the ’745 patent (and the priority applications) describes SEQ ID NO: 9 and, if more guidance was needed, the α KGD motif “orients a skilled artisan to the motif residues’ locations, and the role of the AAD-1 active site in degrading phenoxy auxin and (*R*)-FOP herbicides.” *Id.* at 38

²⁶ Weidong Tian and Jeffrey Skolnick, *How Well is Enzyme Function Conserved as a Function of Pairwise Sequence Identity?*, 333 J. Mol. Biol. 863–882 (2003) (Ex. 2019 (“Tian”)).

²⁷ Burkhard Rost, *Enzyme Function Less Conserved than Anticipated*, 318 J. Mol. Biol. 595–608 (2002) (Ex. 2028 (“Rost”)).

(citing Ex. 1003, 18:43–62, 49:20–50:45). Further, Patent Owner argues, RdpA’s motif residues and locations were already published in the prior art reference “Schleinitz 2004.”²⁸ *Id.* at 39–41 (citing Ex. 1042, 5360 (Fig. 2) (with Patent Owner’s annotations)); *see also id.* at 42–44 (arguing the “AAD-1 motif” is correlated to the claimed dual-degradation function).

Patent Owner also argues that Petitioner is demanding a structure-function “*causation*” standard that is too high, when only a structure-function “*correlation*” is needed to meet the written description requirement. PO Resp. 44–46 (citing, e.g., *Ajinomoto Co., Inc. v. ITC*, 932 F.3d 1342, 1359–60 (Fed. Cir. 2019); *AbbVie*, 759 F.3d at 1301). Patent Owner contends the written description requirement does not require ruling out all potentially inoperative embodiments. *Id.* at 48–50 (discussing the omission in claim 1’s AAD-1 motif of the R285 residue). And, Patent Owner argues that Petitioner erroneously relies on post-filing date evidence (e.g., publications from 2019) in support of the argument that the challenged claims lack written description in the 2004-2005 priority applications. *Id.* at 51–55 (arguing it was unnecessary for the priority applications to describe residues important to enantioselectivity of AAD-1 proteins or to provide three-dimensional structural information for such proteins). In any case, Patent Owner contends, post-filing evidence shows that Petitioner’s own dual herbicide-degrading RdpA variants retain the claimed AAD-1 motif and

²⁸ Kathleen M. Schleinitz et al., *Localization and Characterization of Two Novel Genes Encoding Stereospecific Dioxygenases Catalyzing 2(2,4-Dichlorophenoxy)propionate Cleave in Delftia acidovorans MCI*, 70:9 Applied and Environmental Microbiology, 5357–5365 (Sept. 2004) (Ex. 1042 (“Schleinitz 2004”)).

have high sequence identity (~89–90%), confirming those structures are correlated to function. *Id.* (citing, e.g., Ex. 2015 ¶ 136; Exs. 2012–2014).

Lastly, Patent Owner contends that the '745 patent's disclosed AAD-1 variants (or "species") are representative of the "narrow" genus claimed. PO Resp. 56–58; PO Sur-reply 20. Patent Owner contends that the claims cover a group of "*highly identical* enzymes" ($\geq 85\%$ sequence identity) with a "conserved active-site motif." PO Resp. 56 (citing Ex. 2015 ¶¶ 131–135). And again, Patent Owner cites Petitioner's 2019 publication of six "FT" (FOP tolerant) variants developed by Petitioner, which Patent Owner contends have the AAD-1 motif and greater than 85% sequence identity to SEQ ID NO: 9—"confirm[ing] that the claimed AAD-1 enzymes represent a genus of *highly identical* enzymes having the same or similar activity, just as the specification discloses." *Id.* at 57–58 (citing Ex. 1052,²⁹ 2087, 2090 (Fig. 3); Ex. 2015 ¶¶ 136–137 (determining that "each FT enzyme is 89-91% identical" to RdpA)).

We address the parties' arguments and related evidence as appropriate in further detail in the analysis below.

c) Analysis

For the reasons discussed below and based on the full record developed through trial, we find that the priority applications do not provide sufficient written description support for the challenged claims. Those claims are entitled to a priority date no earlier than October 7, 2016, and, thus, Corteva '437 is prior art as of its November 17, 2005, publication date.

²⁹ Clayton T. Larue et al., *Development of enzymes for robust aryloxyphenoxypropionate and synthetic auxin herbicide traits in maize and soybean crops*, 75 Pest Manag. Sci., 2086–2094 (2019) (Ex. 1052 ("Larue 2019")).

(1) “AAD-1 motif”

The phrase “AAD-1 motif” never appears in the priority applications. *See, e.g.*, Ex. 1005 ¶ 85; Ex. 1021 ¶ 85. Nor do the priority applications expressly describe a genus of AAD-1 proteins that is defined by “an AAD-1 motif having the general formula” of $HX_{112}D(X)_{114-137}T(X)_{139-269}H(X)_{271-280}R$ as recited in claim 1 of the ’745 patent. *See supra* Section II.D. We agree with Dr. Hausinger that “[t]he first mention of ‘an AAD-1 motif’ or a genus of proteins having it was in the claims that the applicant submitted to the USPTO on October 7, 2016.” Ex. 1054 ¶¶ 60–64. Nevertheless, because the written description requirement does not require claim language appear *in ipsius verbis* in the Specification (*Ariad*, 598 F.3d at 1352), we consider other disclosures in the priority applications and whether the POSA would have reasonably understood the inventors’ possession of the “AAD-1 motif” as claimed from such disclosures and background knowledge.

The priority applications identify a consensus motif related to an overarching family of α KGD enzymes, such as TfdA, TauD, and RdpA among others. Ex. 1021 ¶ 85 (disclosing that “genes of the subject invention can still be generally classified in the same overall family” despite very low homology to *tfdA*); Ex. 1054 ¶¶ 64–70 (describing as a “generic consensus motif already known and published in the prior art”). Those applications disclose: “This [α KGD] family of proteins is characterized by three^[30]

³⁰ The reference to “three” conserved histidine (H) residues here is unclear because the disclosed consensus motif specifies two histidines. Insofar as this disclosure in the priority applications cite Hogan (Ex. 1027), Hogan describes the α KGD family motif differently: $HX(D/E)X_{138-207}HX_{10-13}R$. Ex. 1027, 12401 (Table II, noting consensus motif for the Group II subgroup within the α KGD dioxygenase superfamily). A different reference (Elkins, 2002), which is not cited in the ’745 patent and does not discuss RdpA,

conserved histidine residues in a ‘HX(D/E)X₂₃₋₂₆(T/S)X₁₁₄₋₁₈₃HX₁₀₋₁₃R’ motif which comprises the active site. The histidines coordinate Fe⁺² ion in the active site that is essential for catalytic activity (Hogan *et al.*, 2000).”

Ex. 1021 ¶ 85.

The consensus αKGD motif is not, however, the same as the AAD-1 motif as later recited in the issued claims of the ’745 patent. For example, as Dr. Hausinger explains, the consensus motif indicates segments of variable lengths or numbers of unspecified amino acids (X₂₃₋₂₆, X₁₁₄₋₁₈₃, and X₁₀₋₁₃), but the claimed AAD-1 motif “selects one length from each of those generic ranges: 24, 131, and 10 unspecified amino acids, respectively.” Ex. 1054 ¶¶ 65–66 (testifying the AAD-1 motif recites positional information whereas the consensus motif does not). Moreover, “the AAD-1 motifs further restrict the second and third conserved amino acid residues, from (D/E) to D and from (T/S) to T, respectively.” *Id.* ¶ 67. And, the consensus αKGD motif identifies no comparable motif subsegment that specifies an “(X)₂₈₂₋₂₈₄R” portion, such as recited in the AAD-1 motif of challenged claim 2. *Id.* ¶ 68 (explaining that this AAD-1 motif subsegment was also not identified in the 2019 amendment of the Specification (*see supra* Section II.E.1); Ex. 1003, 167:34–37.

Even if the priority applications did not expressly describe a genus of proteins defined by an “AAD-1 motif,” Patent Owner contends that a POSA could derive the motif from other disclosed information. More specifically, Patent Owner argues that nothing more than RdpA’s primary amino acid sequence (SEQ ID NO: 9) was necessary because a POSA would have

discloses the αKGD consensus motif as it appears in the priority applications. *Compare* Ex. 1023, 5185, *with* Ex. 1021 ¶ 85.

known where to find the “AAD-1 motif” in that sequence. PO Resp. 38–39 (citing Ex. 2015 ¶¶ 110–113). Patent Owner contends that the disclosed α KGD family motif “orients” the POSA to the AAD-1 motif and its active-site residues. *Id.* at 38 (citing Ex. 1003, 18:43–62). Patent Owner’s contentions echo the “mapping” argument applicant raised during prosecution in support of the 2016 claim amendment and 2019 Specification amendment to recite an “AAD-1 motif.” Ex. 1008, 3–10 (preliminary amendment), 480, 486–487 (arguing that “mapping” SEQ ID NO: 9 to the consensus α KGD motif allows the POSA to derive the claimed AAD-1 motif), 489 (arguing a POSA “would recognize that the [α KGD] motif disclosed on page 17 could be used to identity an AAD-1 motif as a means of identifying important residues of the AAD-1 active site”); *see supra* Section II.E.1 (amending Specification in 2019 to add: “Mapping the α -ketoglutarate-dependent dioxygenase motif disclosed . . . to the sequence of SEQ ID NO: 9 reveals the AAD-1 motif” and then adding to the disclosure the “general formula” of the motif as recited in claim 1).

We disagree with Patent Owner’s contentions. As Dr. Hausinger persuasively explains, there is an “important distinction between observing that RdpA’s sequence includes an instance of the generic α KG dioxygenase consensus motif versus describing a new and unique . . . ‘AAD-1 motif’ that is characteristic of proteins like RdpA with (*R*)-enantioselective activity that degrades phenoxy auxin herbicides and (*R*)-FOP herbicides.” Ex. 1070 ¶ 13. Most α KGD enzymes do not have (*R*)-stereospecific activity and, as the ’745 patent and the priority applications acknowledge, none of this family of enzymes was previously known to degrade both phenoxy auxins and FOPs, much less the herbicidally-active (*R*)-FOPs required by the claims. *Id.*; Ex. 1054 ¶ 102; Ex. 1003, 4:52–58 (“[T]he subject invention relates to the

use of an enzyme that is capable of degrading both 2,4-D and [FOP] herbicides, in an enantiospecific manner. No α -ketoglutarate-dependent dioxygenase enzyme has previously been reported to have the ability to degrade herbicides of different classes and modes of action.”); Ex. 1021 ¶ 18 (same; stating this functionality distinguishes the invention from other α KGD enzymes like TfdA). Moreover, as Dr. Hausinger testifies, the “mapping” premise “incorrectly conflate[s] the ‘active site’ residues that are identified by the generic consensus of α KG motif with residues responsible for the [claimed] stereoselective activity,” i.e., “(R)-enantioselective activity against chiral phenoxy auxins and aryloxyphenoxypropionate herbicides.” Ex. 1054 ¶ 95. That RdpA may have shared canonical residues under an umbrella of the consensus α KGD motif—a motif and residues indicative of generic dioxygenase activity—does not sufficiently describe an allegedly new “AAD-1 motif” that characterizes a genus of proteins providing a never-before-seen functionality that is claimed.

Beyond the priority applications’ disclosure, Patent Owner also argues that, by 2004, other researchers had described the “AAD-1 motif” (including key residues and spacers in RdpA’s sequence). PO Resp. 16–17, 39–41 (citing Ex. 1042, 5360; Ex. 2016, 24:19–26:7). According to Patent Owner, the AAD-1 motif was published in Schleinitz 2004. *Id.* at 39–40 (annotating Schleinitz 2004’s Figure 2 (sequence alignments between TfdA, TauD, RdpA, and SdpA)). Thus, Patent Owner contends, “[a] skilled artisan therefore would have already known the RdpA (AAD-1) motif.” *Id.* (citing Ex. 2015 ¶¶ 114–115); *see also id.* at 43 (arguing that Schleinitz 2004 disclosed that the AAD-1 motif was “functionally important” and “functionally essential” (citing Ex. 1042, Abstr., 5362)); PO Sur-reply 8–9.

In its Reply, Petitioner argues that Patent Owner’s reliance on Schleinitz 2004 (published in September 2004) “abandons” any April 2004 priority date. Pet. Reply 2. Further, Petitioner contends, Patent Owner effectively concedes that the 2004 and 2005 priority applications, which Petitioner treats as having materially-identical disclosures, cannot support the AAD-1 motif as claimed. *Id.* at 2–3, 8–9; Tr. 11:13–12:6 (arguing Schleinitz 2004 published between the filing dates of the 2004 provisional and 2005 PCT applications, and “the only way Schleinitz [2004] can be prior art is if the AAD-1 motif is not described in the provisional . . . and [because] the PCT’s material[ly] identical it’s not in the PCT either”).

Patent Owner denies any concession about a 2004 priority date, arguing instead that it simply addressed whether the claims are entitled to a 2005 priority date that predates Corteva ’437’s publication. Tr. 32:22–33:4; PO Sur-reply 2 (arguing Corteva ’437 is not prior art if the claims are entitled to priority to “either” the 2004 or 2005 applications’ filing dates).

For purposes of our analysis, we treat Schleinitz 2004 as prior art, and need not additionally decide whether the claims are entitled to an April 2004 priority date. That being said, if the claims are ultimately not entitled to priority to the May 2, 2005, filing date of the 2005 PCT application (with Schleinitz 2004 facially qualifying as prior art), then it is not apparent the claims would be entitled to priority to the 2004 provisional application filing date (with Schleinitz 2004 unavailable as prior art to provide alleged written description support). Patent Owner cannot have it both ways—where the

claims are entitled to a priority date that is earlier than Schleinitz 2004 *and* Schleinitz 2004 is prior art to those claims.³¹

Even considering Schleinitz 2004 as prior art, Patent Owner’s argument about it supplying written description support for the claimed AAD-1 motif is unavailing. As an initial matter, Schleinitz 2004 is not cited in the Specification of the priority applications—despite the Specification identifying over a hundred references throughout (e.g., the aforementioned “Hogan”) and a listing of references that spans seven pages. Ex. 1021 ¶ 85, pp. 152–158. *Ariad*, 598 F.3d at 1351 (holding that the “hallmark of written description is disclosure” and “the test requires an objective inquiry into the four corners of the specification” from the POSA’s perspective). We also question whether Schleinitz 2004 qualifies as the sort of *well-known* background information that might adequately fill gaps in the disclosure of the priority applications relative to a newly-coined “AAD-1 motif.”³² Schleinitz 2004 published only eight months before the May 2, 2005, PCT application filing date. Ex. 1021, code (22); Ex. 1042, 5357. And, it

³¹ Petitioner argued in this case that Corteva ’437 is prior art with a § 102(a)(1) effective date that is November 17, 2005. Pet. 19. Petitioner never argued that Corteva ’437 is prior art with any earlier effective date (e.g., the April 30, 2004, filing date of the related 2004 provisional application). Our analysis of the effective prior art date of Corteva ’437 is limited accordingly. *Cf. SAS Inst. Inc. v. Iancu*, 138 S. Ct. 1348, 1355 (2018) (holding “petitioner is master of its complaint”).

³² Where gap-filling of a disclosure with the prior art’s teachings is involved, the Federal Circuit has been careful to limit such gap-filling to information that is *well-known* in the art. *See, e.g., Boston Scientific Corp. v. Johnson & Johnson*, 647 F.3d 1353, 1366 (Fed. Cir. 2011) (“[I]n some circumstances, a patentee may rely on information that is ‘well-known in the art’ for purposes of meeting the written description requirement.”) (quoting *Falko-Gunter Falkner v. Inglis*, 448 F.3d 1357, 1366–68 (Fed. Cir. 2006)).

describes RdpA and SdpA as “*novel* genes” that encode “enantiospecific dichlorprop and mecoprop.” Ex. 1042, 5362 (“The characterization of genes shown to be involved in the cleavage of these chiral herbicides *has not previously been reported.*”) (emphases added).

Accepting for argument’s sake that Schleinitz 2004 would have nevertheless informed the POSA’s perspective in the 2004/2005 timeframe, the reference compares sequences of several α KGD dioxygenases with varying substrates and stereospecificities and identifies five amino acids from the “functionally important” α KGD consensus motif in RdpA and other α KGD enzyme family members. Ex. 1042, 5357, 5360, 5362–63 (“The functionally-important [consensus α KGD] motif . . . is conserved throughout group II α KG-dependent dioxygenases . . . [and] is present in both RdpA and SdpA.”); Ex. 1070 ¶¶ 13–14 (testifying that Schleinitz 2004’s discussion about “functionally important” residues concerns α KGD dioxygenase activity “generally”), 19. As Dr. Hausinger testifies, “observing an instance of the generic consensus motif [in RdpA] is not the same thing as defining a new motif” that correlates to a new functionality across a genus of proteins—noting the absence of discussion about any new “motif” in Schleinitz 2004. Ex. 1070 ¶ 14 (testifying that, if Dr. Bollinger’s supposition about a newly-identified motif in RdpA were correct, “then Schleinitz 2004 surely would have discussed that motif”). We agree with Petitioner that “Schleinitz 2004 never discloses any motif that is ‘new and unique’ and generalizable from RdpA to other proteins.” Pet. Reply 9 (citing Ex. 1070 ¶¶ 8–10, 12–14 (testifying, *inter alia*, that a POSA would not have mistaken the generic α KGD residues or the spacing between them for the features that define the set of substrates that a particular α KGD enzyme such as RdpA will use)).

Petitioner's position and Dr. Hausinger's testimony about Schleinitz 2004 is also more consistent with Patent Owner's past treatment of that reference. Patent Owner's prosecution declarant, Dr. Nair, testified that Schleinitz 2004 described "[a] motif of the so-called Group II α -KG dioxygenases" (like the disclosure in paragraph 85 of the priority applications (e.g., Ex. 1021 ¶ 85)). *See* Ex. 1008, 590–598, ¶ 6. Dr. Nair never suggested Schleinitz 2004 itself described any new motif—much less the claimed AAD-1 motif. *Id.*; Pet. Reply 10.

Patent Owner suggests that Dr. Hausinger agreed that Schleinitz 2004 described the AAD-1 motif as claimed. PO Resp. 17 (citing Ex. 2016, 24:19–26:7). But, consistent with his testimony elsewhere that an instance of the consensus α KGD motif can be found in RdpA, Dr. Hausinger merely testified where the five conserved residues from the consensus motif appear in RdpA and how far apart they are, not that Schleinitz 2004 described a new AAD-1 motif that characterizes a new functionality across a genus of proteins broadly. Ex. 1070 ¶¶ 25–27.

Moreover, a structural basis for RdpA's herbicidal enantiospecificity is disclosed in neither Schleinitz 2004 nor the priority applications. We credit Dr. Hausinger's testimony that "it was not known which of the many differences" between RdpA and other α KGD enzymes "were responsible for RdpA's (*R*)-enantioselective activity."³³ Ex. 1070 ¶ 21. Schleinitz 2004

³³ Although some of the challenged claims (e.g., claim 1) encompass degradation of achiral phenoxy auxins, other claims (e.g., claim 10) require degrading an (*R*)-enantiomer of a phenoxy auxin. Ex. 1003, 167:66–67. And, all the claims require degradation of enantiomeric (*R*)-FOPs, which is not described in Schleinitz 2004. That the claimed genus of enzymes are able to degrade the (*R*)-enantiomers is not a trivial distinction because, as

itself discloses that the “basis of [RdpA’s] enantiospecificity . . . is not known.” Ex. 1042, 5362–63 (disclosing that it is likely that “substrate specificity is determined by only a few amino acids”). According to Schleinitz 2004, “[s]ite-specific mutagenesis studies are needed to reveal the molecular basis of the substrate specificity and stereospecificity” of these enzymes. *Id.*; Ex. 1070 ¶ 25 (testifying, *inter alia*, that “the [Schleinitz 2004] authors never suggest a connection between the spacing of the consensus motif depicted in Figure 2 and the activities of RdpA or SdpA on different enantiomers”). If anything, Schleinitz 2004 highlights a lack of understanding and unpredictability about the structural basis for (*R*)- and (*S*)- enantiomer substrate specificity among α KGD enzymes in the 2004/2005 timeframe. *Ariad*, 598 F.3d at 1351 (holding “predictability of the aspect at issue” is relevant to the written description inquiry).

Patent Owner also points to assay results disclosed in the ’745 patent as allegedly providing written description support for the “AAD-1 motif” as claimed. PO Resp. 38, 42 (citing Ex. 1003, 49:20–50:45); *see also* Ex. 1021 ¶¶ 192–198 (analogous disclosure in priority application); PO Sur-reply 10. Those results showed that AAD-1 (v1) degraded 2,4-D and two FOP herbicides, and corresponding phenol metabolites were detected. Ex. 1021 ¶¶ 192–198 (HPLC and bioassay results showing degradation of 2,4-D, haloxyfop, and diclofop, but no activity on pyridyloxy auxins (another class of herbicide with aryloxyalkanoate chemical substructures)); *id.* ¶ 15. According to Dr. Bollinger, the results “indicat[e] a similar mechanism of action on these substrates” and a “skilled artisan thus would have readily

discussed above, the (*R*)-enantiomers of the phenoxy auxins and FOPs are herbicidally active; the (*S*)-enantiomers are not. *See supra* Section II.C.

understood that the AAD-1 motif is essential to the claimed dual-herbicide degrading activity.” Ex. 2015 ¶¶ 88–90, 116–118.

We find the cited assay results suggest an α KG-dependent oxidation reaction is involved in the breakdown of 2,4-D and FOPs that were tested. *See* Ex. 2015 ¶¶ 89–90 (showing substrate cleavage and release of phenol products). The priority applications do not, however, tie those results to any “AAD-1 motif” generalizable across a new genus of proteins that, by virtue of including such motif, would be thus capable of degrading phenoxy auxins and (*R*)-FOPs as claimed. Ex. 1021 ¶¶ 192–198. The results show degradation of two classes of herbicides but the Specification offers little to explain what structural features of the AAD-1 (v1) enzyme are correlated to that dual-degradation function and should be maintained across a much broader genus of enzyme variants. Such efforts came years later—in testing and analysis missing in the disclosure of the priority applications, as we discuss below.

Notwithstanding the assay results now cited by Patent Owner, the priority applications lack sufficient description of the functional significance of the motif residues and their exact spacing as later recited in the “AAD-1 motif” of the 2016 amended claims and 2019 amended Specification. Patent Owner sought to address those issues during prosecution—not based on the 2004/2005 priority applications’ disclosures, but with *extrinsic evidence* created over a decade later. Ex. 1008, 455. In 2019, Patent Owner submitted experimental results from Dr. Wright alleged to show the “importance and necessity” of the AAD-1 motif residues. *See, e.g., id.* at 452, 455, 462 ¶ 14, 474 (showing that 2,4-D and *R*-quizalofop degrading activity was maintained in certain sequences when the AAD-1 motif was intact but, in variants where single residues of the defined motif were

mutated, most variants exhibited 90–100% reduced activity).³⁴ No such data or analysis is described in the 2004/2005 priority applications. Yet even that 2019 data shows that a single substitution of a residue that is *not part of* claim 1’s “AAD-1 motif” renders the variant enzyme *inactive* against both phenoxy auxins and FOPs. Ex. 1008, 455, 462–63, 474 (substituting an alanine residue for arginine at position 285 (R285A)); Ex. 1054 ¶¶ 96–97 (testifying “the claim 1 motif does not require this essential arginine, and the original application does not otherwise indicate that it is necessary”).³⁵

³⁴ *Lifescan*, cited by Patent Owner, is distinguishable. See, e.g., PO Resp. 49–50, 52 (citing *Lifescan Glob. Corp. v. Ikeda Food Rsch., Ltd.*, PGR2019-00032, Paper 11, 29 (PTAB Aug. 15, 2019)). In *Lifescan*, unlike here, the specification itself described a specific six-amino-acid subsequence (AGVPWV) that “was necessary” for enzyme activity and “confirmed that the activity was substantially lost when at least one amino acid residue in the amino acid sequence was deleted.” See, e.g., *Lifescan*, Paper 11 at 5, 15–16 (discussing, e.g., the specification’s express description of nine enzymes—six that included the subsequence and were active, and three without it that lacked activity—and further description of specific acceptable variants of the AFVPWV sequence). In *Lifescan*, the Board expressed skepticism about the written description challenge but was persuaded that petitioner was likely to succeed on a related enablement challenge, and instituted trial on all grounds. *Id.* at 11, 24–25, 41–42. The sole claim at issue was later canceled in favor of a proposed substitute/amended claim, which claim the Board determined through trial would have been obvious. *Lifescan*, Paper 38, 7–8, 31–33. *Yeda*, cited by Patent Owner (PO Resp. 41–42), is distinguishable too. *Yeda Rsch. & Dev. Co. v. Abbott GmbH & Co.*, 837 F.3d 1341 (Fed. Cir. 2016). As noted by Petitioner, it was “undisputed” in *Yeda* that the claims encompassed only one protein, not a broad genus like involved here. *Yeda*, 837 F.3d at 1345; Pet. Reply 11.

³⁵ It was only in 2016, with the addition of the “AAD-1 motif” of claim 2 and in Dr. Wright’s 2019 experiments that this residue is identified as “critical” to the claimed function. Ex. 1008, 452. We address claim 2 in further detail below.

Altogether, we find that the priority applications do not describe a new and functionally-distinct genus of proteins defined by an “AAD-1 motif” as later recited in challenged claim 1. The cited disclosures fail to adequately describe a new active site “motif” for a newly-coined genus of Aryloxy Alkanoate Dioxygenase “AAD-1” enzymes with a newly-claimed ability to degrade herbicides in different chemical classes and modes of action, including (*R*)-FOPs. *See, e.g.*, Ex. 1021 ¶ 85 (describing α KGD family motif only). The priority applications disclose that RdpA has dioxygenase activity and degrades phenoxy auxins and (*R*)-FOPs, but missing is any meaningful description of the specific structural features of the enzyme that correlate to this new and allegedly “surprising” activity and that might characterize a whole genus of proteins that exhibit it. Ex. 1021 ¶¶ 18 (disclosing the invention involves an enzyme that “surprisingly possesses novel properties” that distinguish it from other α KGD enzymes—degradation of 2,4-D and FOP herbicides “in an enantiospecific manner”), 192–198 (assays showing 2,4-D and FOP degradation); Ex. 1054 ¶¶ 73–75, 92–95 (“Nothing in the specification identifies the residues that correspond to the claimed activity.”).

Schleinitz 2004 does not alone, or in combination with the priority applications, fill this gap for the reasons noted above. Patent Owner’s efforts to stitch the internal and external disclosures together to support a broad genus of proteins characterized by the “AAD-1 motif” looks like obviousness at best. *Lockwood*, 107 F.3d at 1571–72 (“Entitlement to a filing date [for priority] does not extend to subject matter which is not disclosed, but would be obvious over what is expressly disclosed.”); *see also Novozymes A/S v. DuPont Nutrition Biosciences APS*, 723 F.3d 1336, 1349 (Fed. Cir. 2013) (explaining that written description support cannot be

provided merely by pointing to “an amalgam of disclosures plucked selectively” from the priority document). *Flash-Control, LLC v. Intel Corp.*, 2021 WL 2944592, *4 (Fed. Cir. 2021) (noting “the specification must present each claim as an ‘integrated whole’” to meet the description requirement) (quoting *Novozymes*, 723 F.3d at 1349).

(2) “at least 85% sequence identity”

As we explained at institution, there is some literal descriptive support in the priority applications for AAD-1 proteins with at least 85% sequence identity. Inst. Dec. 38–39. We find that AAD-1 v1 (SEQ ID NO: 9) and its variants (AAD-1 v2 and v3) would be understood as “exemplary” proteins. *See, e.g.*, Ex. 1021 ¶¶ 50–52, 207–217 (Example 4, AAD-1 v1); Ex. 2015 ¶¶ 106–109. The priority applications also disclose that “the subject invention comprises variant or equivalent proteins . . . having the same or similar activity of the exemplified proteins” and that “[e]quivalent proteins will have amino acid similarity (and/or homology) with exemplified proteins.” Ex. 1021 ¶ 142. The disclosure continues: “The amino acid identity [with exemplified proteins] will typically be at least 60%, . . . more preferably at least 80%, even more preferably at least 90%, and can be at least 95%.” *Id.* Continuing, the applications disclose, “the identity and/or similarity can be 49, 50, 51, . . . **85**, 86, 87, . . . 97, 98, or 99% as compared to a sequence exemplified or suggested herein.” *Id.* (emphasis added).

Although this disclosure is broad, a literal description of a protein with at least 85% sequence identity to SEQ ID NO: 9 can be discerned without further blaze marks. PO Resp. 36–37 (citing, e.g., Ex. 2015 ¶¶ 108–109; Ex. 1021 ¶ 142). That does not end the inquiry, however, because, just as literal language is not necessary to satisfy the written description requirement, literal language is not necessarily sufficient to meet the

requirement. *Enzo Biochem, Inc. v. Gen-Probe Inc.*, 323 F.3d 956, 968 (Fed. Cir. 2002) (rejecting “argument that the written description requirement . . . is necessarily met as a matter of law because the claim language appears *in ipsius verbis* in the specification”). As Petitioner and Dr. Hausinger explain, at least 20⁴⁴ different sequence permutations (including artificial variants) are encompassed within the scope of the claims even with the “at least 85% sequence identity” limitation. Yet the only enzymes in the priority applications disclosed as actually degrading phenoxy auxins and (*R*)-FOPs involved a mere one or two amino acid changes and are $\geq 99\%$ identical.³⁶ Pet. 30–31; Ex. 1054 ¶¶ 77–83; Pet. Reply 15–16.

On whether there is a sufficient description of a structure-function correlation between the recited “at least 85% sequence identity” and the claimed dual-degradation of phenoxy auxins and (*R*)-FOPs, Patent Owner cites further disclosure in the priority applications and points to preexisting third-party research as supporting a prediction about enzyme function. PO Resp. 30–34 (arguing, e.g., that the Specification describes α KGD enzymes with $\geq 85\%$ as “highly identical,” and Patent Owner cites an external study by Tian (Ex. 2019) as indicating that common enzyme function could be predicted in enzymes with 60% sequence identity); Ex. 1021 ¶ 171. We address that argument and evidence below.

³⁶ Dr. Hausinger testifies that the limitations in claim 29 of the ’745 patent do not meaningfully narrow the scope of the claims compared to claim 1, and a “rough lower estimate of the amino acid sequences satisfying claim 34’s sequence requirements would also be 20⁴⁴ proteins.” Ex. 1054 ¶¶ 80 (addressing in relation to claim 34 of the ’555 patent), 561–563 (noting the correspondence between relevant language of claim 34 of the ’555 patent and claim 29 of the ’745 patent). Patent Owner provides no evidence that rebuts Dr. Hausinger’s estimate.

Patent Owner cites a disclosure in the priority applications about enzymes with α KGD activity and describing sequence identities of 85–98% as “highly identical.” PO Resp. 30–33 (citing, e.g., Ex. 1021 ¶ 171); Ex. 2015 ¶¶ 82–83, 108. Some context is, however, appropriate. The cited disclosure relates to a method used to identify genes that impart 2,4-D resistance, and patentee’s search for, and selection from public databases of, genes with low homology to another known gene/enzyme having α KGD activity—specifically, TfdA. Ex. 1021 ¶¶ 171–173. In its more complete context, the disclosure about “highly identical” sequences reads:

Using default settings, this search returns upwards of 100 homologous proteins at varying levels. These range from highly identical (85-98%) to very low identity (23-32%) at the amino acid level. Traditionally only sequences with high homology would be expected to retain similar properties to the input sequence. In this case we chose only those sequences with ≤ 50 homology. We go on to exemplify that cloning and recombinantly expressing homologues with as little as 27% amino acid conservation can be used to impart commercial levels of resistance not only to the intended herbicide [(i.e., 2,4-D)], but also to substrates never previously tested with these enzymes.

Id. ¶ 171; *see also id.* ¶¶ 172–173 (describing identification of a “single gene (*rdpA*)” from the database as a homologue with only 28% amino acid identity to *tfdA*, and then obtaining a strain of *S. herbicidovorans* containing the *rdpA* gene for subsequent analysis), 12 (disclosing that a “large number of *tfdA*-type genes that encode proteins capable of degrading 2,4-D have been identified from the environment and deposited into the Genbank database” and “[m]any homologues are similar to *tfdA* (>85% amino acid identity) and have similar enzymatic properties to *tfdA*.”).

Importantly, this is not a disclosure that sequences (including artificial variants) with at least 85% sequence identity to RdpA/SEQ ID NO: 9 will

retain the newly-described dual-herbicide degradation function of claim 1. Moreover, the disclosure relates to a search for proteins with general α KGD activity to impart resistance to the achiral compound 2,4-D, not using sequence identity to predict enantioselectivity for (*R*)-FOPs like claimed. Ex. 1070 ¶ 36 (explaining how this interpretation is reinforced by a reading of the title of the example (“Method for Identifying Genes that Impart Resistance to 2,4-D in Planta”) and other context (citing Ex. 1001, 44:10–13, 44:17–20)); *see also* Ex. 1003, 44:5–30 (same).

Other disclosure in the priority applications suggests that sequence identity would not alone be sufficient to predict function because individual residue changes may or may not negatively impact activity of a particular RdpA variant. That disclosure states that *conservative* or *non-conservative* amino acid substitutions can be made if the changes do not adversely affect the protein’s biological activity. Pet. Reply 13–14; Ex. 1021 ¶¶ 144–145; Ex. 1070 ¶¶ 29–30 (testifying the problem with using sequence identity threshold in the manner suggested by Dr. Bollinger “is that it treats each amino acid change as equivalent to, and interchangeable with, any other change,” yet “the POSA would have known, the nature of the changes to a given variant matters”).

We credit Dr. Hausinger’s testimony that the priority applications do not identify sufficient structural information or instances of specific acceptable changes that do (or do not) maintain the claimed function—“leaving it up to the POSA to test each artificial variant to evaluate whether there has been an adverse change to the biological activity.” Ex. 1070 ¶ 30; Ex. 1054 ¶ 90 (testifying the POSA would have known that different types of changes at different locations in the sequence can have varied and unpredictable effects); *PureCircle USA Inc. v. SweeGen, Inc.*, 2024 WL

20567 at *4 n.10 (Fed. Cir. Jan. 2, 2024) (explaining that “*Ajinomoto* stands for the idea that where there are structural features common to a genus, the structure-function correlation does not need to be perfect and some testing—appropriate to the knowledge of a POSA—is allowed, not that an unknown structure-function correlation along with extensive testing can satisfy written description”). We agree with Petitioner that, because the claims facially permit both conservative and non-conservative substitutions of any type and at nearly any location, sequence identity values would not be sufficient to predict the dual activity claimed. Pet. Reply 14 (citing Ex. 1070 ¶¶ 28–31).

With respect to the third-party papers cited by Patent Owner, there is no question that, between the late 1990s and early 2000s, other researchers had published results related to predicting function of various enzymes from comparison of protein sequences and sequence identity. PO Resp. 8–11, 31–32 (citing, e.g., Ex. 2023³⁷ (Chung), 1123; Ex. 2026³⁸ (Devos), 99; Ex. 2019 (Tian), 866; Ex. 2028 (Rost), 604). As Patent Owner points out, researchers like Tian suggested that enzyme function was conserved with about 90% accuracy when sequence identity was above 60%. See PO Resp. 9–10 (citing Ex. 2019, 866 (Fig. 1B)). According to Patent Owner, other researchers including Rost showed that enzyme function diverged more than originally thought when sequence identity was below 70%, but that there

³⁷ Su Yun Chung and S Subbiah, *A Structural explanation for the twilight zone of protein sequence homology*, 10:4 Structure 1123–27 (1996).

³⁸ Damien Devos and Alfonso Valencia, *Practical Limits of Function Prediction*, 41 Proteins: Structure, Function, and Genetics 98–107 (2000).

was generally strong conservation of function above that level. *Id.* at 9 (citing Ex. 2028, 604 (Fig. 5)); Ex. 2015 ¶¶ 36–40.³⁹

We agree with Petitioner and Dr. Hausinger, however, that Tian and these other third-party studies are insufficient to show written description support in the priority applications. Pet. Reply 4, 15–17. Tian and the other studies are not cited within the four corners of those applications. That aside, the studies have an important limitation relative to the issue here because they describe the relationship between *naturally occurring proteins* not the artificial variants encompassed by the claims. *See, e.g.*, Ex. 2019, 863–65, 876–77; Ex. 1070 ¶¶ 33–34, 37–41, 45 (testifying, for example, that Swiss-Prot is a curated database of naturally occurring protein sequences that excludes synthetic or artificial sequences (citing Ex. 1074, 53; Ex. 1075)). As Dr. Hausinger explains, naturally-occurring enzymes are the product of natural selection and would be expected to preserve activity or functionality and are *unlike* the challenged claims, which are not limited to

³⁹ Although not decisive in any way here because it involved a different patent and overall record, and was resolved on a different statutory basis, we note that the Board recently rejected a similar argument made by Patent Owner (and relying on Tian (Ex. 2019), Rost (Ex. 2028), etc.) about predicting a claimed dual-herbicide degrading function for a genus of AAD enzymes based on sequence identity $\geq 85\%$, citing, e.g., the experimentation required to test for such functionality. *Inari Agriculture, Inc. v. Pioneer Hi-Bred Int’l, Inc.*, PGR2023-00022, Paper 50, 27–34 (PTAB Oct. 10, 2024) (finding claims unpatentable for lack of enablement; not reaching written description challenge); *see also id.* at 4–5 (claims to transgenic plant cell and recombinant polynucleotide encoding an “AAD-12 protein” that degrades phenoxy auxins and pyridyloxy auxin herbicides, wherein protein has an “AAD-12 motif” and “at least 85% sequence identity” to a reference sequence); PGR2023-00022, Paper 48, 1 (noting Corteva’s assignment of the patent to its affiliate Pioneer Hi-Bred Int’l, Inc., which occurred shortly before the final written decision issued).

naturally occurring RdpA proteins; instead the claims encompass a huge set of artificial sequences that can be generated by making changes throughout SEQ ID NO: 9. Ex. 1070 ¶¶ 38–40 (testifying it would not make sense for the POSA to extend Tian’s conclusions from a biased set of amino acid sequences to a larger dataset of proteins that have not been the product of natural selection).

Patent Owner also cites Minshull⁴⁰ as disclosing the use of homologous sequences as a potentially valuable source of information for protein engineering. PO Resp. 10–11, 31 (citing Ex. 2025, 202–204); PO Sur-reply 18–19 (citing a “successful philosophy of using substitutions found in homologs”). We agree with Petitioner, however, that Minshull, if anything, “invites trial and error experimentation” and itself cautions that “[p]rediction of protein function from sequence and structure is a difficult problem.” Ex. 2025, 203; Pet. Reply 4, 17–18; Ex. 1070 ¶¶ 46–48 (testifying that even Minshull recognizes that artificially-engineered variants require screening for activity (citing Ex. 2025, 202–203)).

Petitioner also argues, and we agree, that even if Tian and these other third-party studies supported making a “prediction” about activity from sequence identity values, written description support for the claims would still be lacking. Pet. Reply 12–13. On this record, a bare prediction about dual-degradation activity and theory about techniques that a skilled artisan might have employed to identify suitable artificial RdpA variants is not a

⁴⁰ Jeremy Minshull et al., *Predicting enzyme function from protein sequence*, 9 Current Opinion in Chemical Biology 202–209 (2005). Petitioner also points out that Minshull published April 28, 2005, and is nowhere cited in the ’745 patent, and that Patent Owner does not explain why Minshull was “well known” by the May 2, 2005, PCT application date. Pet. Reply 17–18.

written description of what is claimed—it is a research plan with a potentially obvious result. *Id.* at 13 (asserting that Dr. Bollinger invokes obviousness-based reasoning when testifying that, if a POSA “follow[ed] established principles” for “designing enzymes variants” in an effort to “conserve function,” one would “have reasonably expected” to obtain proteins with the claimed dual activity (quoting Ex. 2015 ¶ 134)); *see also id.* ¶¶ 52–59). But “[a] patent is not a hunting license. It is not a reward for the search, but compensation for its successful conclusion.” *Ariad*, 598 F.3d at 1353 (quoting *Brenner v. Manson*, 383 U.S. 519, 536 (1966)). Indeed, the written description requirement “limits patent protection to those who actually perform the difficult work of ‘invention’—that is, conceive of the complete and final invention and all its claimed limitations—and disclose the fruits of that effort to the public.” *Id.* at 1354–1355.

Patent Owner contends that Dr. Hausinger fails to account for the POSA’s perspective and disregards their ability to foresee enzyme function. PO Resp. 34 (citing Ex. 2016, 29:25–30:23). We disagree with Patent Owner’s characterization of Dr. Hausinger’s analysis. Rather, he consistently testified that even small residue changes to the enzymes in question can have a significant impact on enzyme function so, in his opinion, the POSA would want “data” not mere hypotheses or predictions. *See* Ex. 2016, 29:14–31:9; Ex. 1054 ¶¶ 89–91; Ex. 1070 ¶¶ 32, 56.

Petitioner also submits evidence from the prior art disclosing that herbicide-degrading α KGD enzymes can have divergent activity even when those enzymes share sequence identities above 85%. Ex. 1041, 421, 427; Pet. Reply 18–19. Saari 1999 describes TfdA homologs sharing even greater levels of sequence identity but exhibiting different properties. Ex. 1041, 421, 427 (describing a TfdA-like protein from *A. denitrificans*

with 86% and 95% amino acid identity to two other TfdA proteins but exhibiting opposite stereoselective degradation activities); Ex. 1054 ¶¶ 54, 90–91, 109. We credit Dr. Hausinger’s testimony that a POSA “would have considered studies of enantioselective herbicide-degrading α KG dioxygenases” such as Saari 1999 “to be more relevant and informative to the claimed RdpA variants” compared to a generic prediction derived from Tian and the other third-party studies that “do[] not address the structure or function of α KG dioxygenases.” Ex. 1070 ¶¶ 34, 51 (testifying that Saari 1998 (Ex. 1040, Abstr., 71–74, 86–94) and Schleinitz 2004 are consistent); Ex. 1042, 5362–63 (reporting it “likely” that “substrate specificity is determined by only a few amino acids”).

Patent Owner questions Saari 1999’s data because it was allegedly based on testing of cell extracts not purified protein. PO Resp. 50 (citing, e.g., Ex. 2015 ¶¶ 125–127); Ex. 2016, 42:7–16 (testifying that, using cell extracts, some assay results “can be compromised to some extent”). Patent Owner’s attempt to undermine Saari 1999 is unavailing. Dr. Hausinger, who co-authored Saari 1999, testifies that cell extracts were commonly used, and in any event, Saari 1999 employed several approaches to ensure that the reported activity was due to an (*R*)-enantioselective-herbicide degrading α KGD dioxygenase. Ex. 1070 ¶¶ 53–54 (citing, e.g., Nickel (Ex. 1036), Kohler (Ex. 1031), and the patent itself (Ex. 1001, 22:11–22, 46:8–28) as using extracts; discussing additional testing beyond colorimetric assays that were used in Saari 1999 (Ex. 1041, 425–427, Fig. 4)). We also agree with Dr. Hausinger that the other references cited by Patent Owner do not present data or findings inconsistent with those in Saari 1999. *Id.* ¶¶ 55–56; *see also id.* ¶¶ 58–59 (explaining how the teachings in Saari 1998 and Saari 1999 about how reaction stereochemistry can depend on only a handful of amino

acids has precedent in the art (Saari 1999 (Ex. 1041, 427–428 (“The ability for closely related enzymes to recognize opposite stereoisomers has precedent”) (citing Nakajima 1998 (Ex. 2053))).

Patent Owner also suggests Saari 1999’s teachings are “irrelevant” because RdpA and SdpA only have about 30% sequence identity. PO Resp. 50 (describing SdpA as RdpA’s “(*S*)-enantiospecific counterpart enzyme”) (citing Ex. 2053); *see* Ex. 1042, 5357 (describing two novel genes *rdpA* and *sdpA* encoding enantiospecific α KGD enzymes in *D. acidovorans*). That RdpA and SdpA may have relatively low homology is not a disclosure that artificial variants of RdpA with ~ 85% sequence identity will yield active protein product—much less protein with the same activity as the native sequence and degrade both phenoxy auxins and (*R*)-FOPs as claimed. Ex. 1070 ¶ 57 (testifying the “POSA had no way to know without trial-and-error experimentation whether any given variant with at least 85% identity to RdpA would degrade (*R*)-herbicides”). Moreover, as Dr. Hausinger testifies, “[i]f anything, the large number of differences between the RdpA and SdpA amino acid sequences would have made it harder, not easier to identify the amino acids that are important to RdpA’s (*R*)-enantioselective activity.” *Id.* (citing Ex. 1042, 5362–63).

As explained herein, we find that the priority applications lack a sufficient written description of a structure-function correlation supporting the broad genus recited in the challenged claims, even when we consider the alleged disclosures about the “AAD-1 motif” and “at least 85% sequence identity” limitations, and the POSA’s asserted background knowledge about the same.

(3) *Representative Species*

Petitioner persuades us that the genus of AAD-1 proteins encompassed by claim 1, even with the “AAD-1 motif” and the “at least 85% sequence identity” limitations, is vast. It is uncontested that it includes at least 20⁴⁴ permutations comprising myriad protein/sequence variants. Ex. 1054 ¶ 79. Beyond the five specific amino residues that appear in the “AAD-1 motif” formula of claim 1, up to 44 amino acid substitutions could be made among the other 290 amino acids making up SEQ ID NO: 9 and “[e]ach of those substitutions could be any amino acid, at almost any position in the protein.” *Id.* ¶ 78. Neither the challenged claims nor the disclosure of the priority applications make any restriction on the type of substitutions that could be made to any amino acid residue corresponding to an “X” within the boundaries of the “AAD-1 motif” (even assuming the AAD-1 motif were adequately described) or to other amino acids outside the motif. Any or all such substitutions could be “conservative” (i.e., changing to an amino acid of the same type or class), all could be “non-conservative,” or some combination of conservative and non-conservative substitutions might be made—the only caveat is that such changes should not be “adverse to” or “significantly detract from” the variant’s desired biological activity. Ex. 1021 ¶¶ 144–145; Ex. 1070 ¶¶ 29–31 (Dr. Hausinger testifying that “extensive experimentation would be required to evaluate which artificial variants” would have the claimed activity).

The claim scope is, therefore, quite broad yet the disclosure of species alleged to demonstrate possession of that full scope is comparatively narrow. The priority applications expressly describe only three virtually-identical AAD-1 proteins (with $\geq 99\%$ sequence similarity) within the claims’

otherwise broad scope that degrade both phenoxy auxins and (*R*)-FOPs.⁴¹ As Petitioner contends, SEQ ID NO: 9 and the two AAD-1 proteins that differ from SEQ ID NO: 9 by just one or two amino acids do not fairly represent the diversity of proteins within a genus of at least 20⁴⁴ variants. Pet. 30–31; Ex. 1054 ¶¶ 81–82 (showing the minimal sequence differences in AAD-1 v2 (alanine substituted for histidine at position 2, and cysteine for arginine at position 212), and v3 (alanine insertion at position 2)); Ex. 1003, 6:55–60 (describing v2’s substitution of cysteine at position 212 as an “inadvertent change”); *id.* at 141–145 (amino acid sequences for SEQ ID NO: 10 (AAD-1 v2) and SEQ ID NO: 11 (AAD-1 v3)).

Dr. Hausinger testifies persuasively that the $\geq 99\%$ identity shared by the three AAD-1 variants that are described is “simply not representative of proteins having as little as 85% sequence identity to AAD-1 v1 (SEQ ID NO: 9).” Ex. 1054 ¶¶ 83–86 (testifying, e.g., that the Specification “does not provide any structural information that might have allowed the POSA to make inferences about the effect of a given change” and nothing in the disclosure “would have suggested to the POSA that changes at [the positions in which v2 and v3 differ] would be particularly informative.”). On this record, we agree with Dr. Hausinger that “[t]he POSA would recognize that changing two amino acid residues, as in v2, cannot begin to represent the degree of amino acid sequence variation allowed in the claimed genera of AAD-1 proteins.” *Id.* ¶¶ 76, 81–86. Although the priority applications’ disclosure need not actually show that every other variant protein works, the

⁴¹ Patent Owner also references “twenty-six examples” described in the patent (PO Resp. 22–23) but, as Petitioner points out, those examples (e.g., expressing protein in different plant types) still involve only the same three enzyme species. Pet. Reply 21 (citing Inst. Dec. 47 n.27).

disclosure must adequately support the breadth of the claimed genus. *Capon v. Eshhar*, 418 F.3d 1349, 1359 (Fed. Cir. 2005) (“It is not necessary that every permutation within a generally operable invention be effective in order for an inventor to obtain a generic claim, provided that the effect is sufficiently demonstrated to characterize a generic invention.”).

Patent Owner argues that Petitioner fails to establish that any significant structure-function diversity exists within 85% identity to SEQ ID NO: 9. PO Resp. 56–58 (citing, e.g., Ex. 2015 ¶¶ 131–135); PO Sur-reply 20. We disagree. Petitioner highlights—and Patent Owner’s own extrinsic testing data shows—that enzymes with ~ 94–95% sequence identity to SEQ ID NO: 9 but without the “critical” (X)₂₈₂₋₂₈₄R subsequence of amino acids are *inactive* against both phenoxy auxins and (R)-FOPs. Ex. 1008, 452, 455 (describing R285 among other amino acids as “critical” to activity), 462–463 ¶ 15 (Dr. Wright testifying to the “importance and necessity” of the motif residues as well as the X₃R segment (i.e., R285 residue) for the claimed dual-herbicide degradation); Pet. 32–33; Pet. Reply 1–2, 10.

Patent Owner’s internal testing documents also show at least three sequences with the AAD-1 motif and 85% identity to SEQ ID NO: 9 that “[REDACTED].” Pet. Reply 23 (citing Ex. 2002, 433–444; Ex. 2004; Ex. 1070 ¶¶ 65–68 (testifying that Patent Owner [REDACTED]
[REDACTED]
[REDACTED]
[REDACTED]); *id.* at 16–17 (arguing that Dr. Bollinger’s opinion neglects to consider sequence changes yielding no product, i.e., inactive variants (citing Ex. 1072, 7:14–9:5)). Given the dearth of disclosure in the priority applications about any specific variants with less than 99% sequence identity

that exhibit the claimed activity, we are unpersuaded that [REDACTED] [REDACTED] represent irrelevant, anecdotal exceptions as suggested by Patent Owner. PO Resp. 51; PO Sur-reply 19–20.

Patent Owner criticizes Dr. Hausinger for allegedly not “evaluat[ing] whether potentially deleterious substitutions had been made to highly conserved residues in those [REDACTED].” PO Sur-reply 20 (citing Ex. 2066, 72:10–78:14). This criticism does not weaken Petitioner’s challenge because, as already discussed, the claims and priority disclosures put no restriction on the type of substitutions that can be made to any of the 290 non-specified (i.e., “X”) amino acids. Also, those variants were all apparently prepared by inventor Dr. Wright in an effort to support his first prosecution declaration and at a time when Patent Owner and Dr. Wright had an interest in showing that variants with ~ 85% sequence identity exhibit the claimed activity. Ex. 1008, 274–275 (Nov. 13, 2018, written description rejection citing lack of support for the “at least 85% sequence identity” and “AAD-1 motif” limitations), 433–438 (Patent Owner slides dated Jan. 30, 2019, about internal testing of variants); Ex. 1070 ¶ 66; Ex. 1008, 452, 455, 457, 462, 474 (disclosing to Patent Office, in March/April 2019, results with Seq 1–10 ($\geq 95\%$ identity variants) *but not* [REDACTED] [REDACTED])). That Dr. Wright would, under those circumstances, have made deleterious substitutions to non-motif but otherwise highly-conserved residues (even if that was an accurate characterization of the changes made) undercuts the notion that a POSA would have known to avoid and, thus, would not have made such substitutions. PO Sur-reply 18. We are also unpersuaded by Patent Owner’s suggestion that a POSA would have modified the subject ~ 85% variants through “routine techniques” including fusing them to a maltose-binding protein to potentially improve solubility

and enable further testing. *Id.* at 19–20 (citing Ex. 2066, 34:25–42:12).

There is no evidence in the record showing that Patent Owner and/or Dr. Wright made the alleged “routine” changes to its variants to reveal any herbicide-degrading activity as claimed.

Patent Owner also cites Petitioner’s own later development of RdpA-variant “FT” (FOP tolerant) enzymes as described in Larue 2019. PO Resp. 57–58 (citing Ex. 1052, 2087, 2090). According to Patent Owner, all “six dual herbicide-degrading FT enzymes have the AAD-1 motif” and “are within 85% sequence identity of RdpA.” *Id.* (citing Ex. 2015 ¶¶ 136–137 (noting that the six cited variants are 89–91% identical to RdpA)). These examples, Patent Owner argues, confirm that its claimed genus comprises highly identical rather than diverse proteins “just as the specification discloses.” *Id.* (citing, e.g., Ex. 1003, 44:12–24; Ex. 1021 ¶¶ 142, 171).

This argument does not support that the priority applications disclose sufficient species representative of the claimed genus. To the contrary, Larue 2019 evidences Petitioner’s own extensive work—over a decade after the 2005 priority date sought by Patent Owner—to develop variants with enhanced enzyme activity and temperature stability. Ex. 1052, 2087. As Dr. Hausinger explains, that work “involved mutagenesis, making and testing thousands of amino acid sequences and performing multiple rounds of screening, and generating crystal structures.” Ex. 1070 ¶¶ 62–64; Ex. 1052, 2089–92 (noting, e.g., screening of “approximately 4000 enzyme variants” as part of research project described in Larue 2019); Ex. 1017 ¶¶ 5–74, 81–94). Dr. Hausinger also testifies that the six FT sequences flagged by Patent Owner were earlier published in April 2016 as part of a Monsanto/Bayer patent application (Ex. 1017), predating by about six months Patent Owner’s own filing of any claims to a genus of proteins

defined by an “AAD-1 motif” and the $\geq 85\%$ sequence identity limitation. Ex. 1070 ¶¶ 63–64 (noting the six sequences are among a listing of twenty-two engineered amino acid sequences (Ex. 1017, Figs. 6A–6E, ¶ 20)).⁴²

Unlike Petitioner’s research described in Larue 2019 and Petitioner’s related 2016 patent application, the priority applications do not describe the design and screening of any specific enzyme variants with $\sim 89\text{--}91\%$ sequence identity, much less 85% identity. The three species described in the priority applications, as already explained, differed by no more than one or two amino acids and, thus, were $\geq 99\%$ identical to each other. Patent Owner’s argument is tantamount to treating Petitioner’s extensive testing and publication related to specific RdpA variants between 2016 to 2019 as though that work was part of the priority applications’ disclosure in 2004/2005. Plainly, it was not.

⁴² Petitioner suggests that the impetus for Patent Owner seeking claims with the $\geq 85\%$ sequence identity limitation came from Patent Owner’s study of Petitioner’s enzyme work. Pet. 1–2; Pet. Reply 23. According to Petitioner, it was only after Bayer’s own research published (in April 2016) that Patent Owner began to pursue claims reciting the “AAD-1 motif” and “at least 85% sequence identity” limitations via the October 2016 filing and preliminary amendment. Pet. Reply 23. Between 2004 to 2015 Patent Owner never sought claims with less than 95% identity to RdpA. *Id.* (citing Exs. 1004–1008). The timing is coincidental, and there is some record support to show that Dr. Wright and other of Patent Owner’s scientists were reviewing Petitioner’s sequences in mid-2019 when Patent Owner was [REDACTED]

[REDACTED] . See Ex. 2002, 864–865 [REDACTED]

[REDACTED] ; see also *id.* at 866 ([REDACTED])

[REDACTED] . Ultimately, however, whatever reason Patent Owner had for pursuing a broader set of claims distracts from the key issue, which is the sufficiency of the written description support for those broader claims in the priority applications.

For the reasons discussed above, we find that the three essentially identical AAD-1 species proteins described in the priority applications are insufficiently representative of the much broader genus encompassed by the challenged claims. *Ariad*, 598 F.3d at 1350 (“[M]erely drawing a fence around the outer limits of a purported genus is not an adequate substitute for describing a variety of materials constituting the genus and showing that one has invented a genus and not just a species.”).

(4) *Claim 2*

Written description support for challenged claim 2 in the priority applications is lacking for the reasons above, and more. Claim 2 modifies claim 1’s “AAD-1 motif” by adding an “(X)₂₈₂₋₂₈₄R” suffix. *See supra* Section II.D. The consensus α KGD motif mentioned in the priority applications omits any information pertaining to those added residues. Ex. 1021 ¶ 85; Ex. 1054 ¶¶ 68, 96. Even considering the “mapping” disclosure inserted into the patent’s Specification via amendment in 2019, there is no “mapping” of the α KGD motif to SEQ ID NO: 9 that could “reveal[]” three additional un-specified amino acids (X₂₈₂₋₂₈₄) followed by another arginine residue (“R”) as part of the “AAD-1 motif” that Patent Owner claimed for the first time in 2016 during prosecution of the ’555 patent. *See supra* Section II.E.; Ex. 1008, 5 (amended claim 16 (issued as claim 2 of the ’555 patent)).

Although RdpA (SEQ ID NO: 9) includes the additional arginine residue as it appears in claim 2, that does not constitute a description of a genus of proteins characterized by claim 2’s “AAD-1 motif.” Ex. 1003, 139–141 (sequence showing residue 285 is “Arg”). SEQ ID NO: 9 includes 295 amino acid residues and the arginine at position 285 is not identified as part of the consensus α KGD motif, or any motif for that matter, in the

priority applications. Ex. 1054 ¶ 96. Indeed, it is not identified as significant in the priority applications in any way—it is simply one of hundreds of other residues in SEQ ID NO: 9 to which the disclosure calls no attention. *Id.* And, tellingly, Patent Owner’s own [REDACTED]
[REDACTED]
[REDACTED]” Ex. 2004, 580; Pet. Reply 23–24.

Patent Owner again turns to the “skilled artisan’s knowledge” in an attempt to fill the gap in the priority applications’ description of the claimed AAD-1 motif. PO Resp. 59–60; PO Sur-reply 21–22. Patent Owner identifies a sequence alignment between TfdA and a group of other α KGD enzymes in Schleinitz 2004, as showing that an analogous arginine residue was present in TfdA (Arg278) and conserved in TauD and RdpA. PO Resp. 59 (citing Ex. 2015 ¶¶ 138–142); Ex. 1042, 5360, 5362.

We agree with Dr. Hausinger, however, that “[t]he only motif discussed in Schleinitz 2004 is the generic consensus [α KGD] motif, which does not include an additional arginine” like recited in claim 2. Ex. 1070 ¶ 16 (noting “the 285 arginine in RdpA is highlighted gray, while the consensus motif residues are highlighted black”); Ex. 1042, 5360. Moreover, insofar as the alignment includes arrows pointing to a handful of other non- α KGD motif residues (including Arg278), Schleinitz 2004 states that the black arrows “indicate 2,4-D binding residues in TfdA.” Ex. 1042, 5360 (Fig. 2 (caption)). Schleinitz 2004 does not disclose that the subject arginine performs any particular function in RdpA and, according to Dr. Hausinger, “the most straightforward reading of the caption for the POSA is that there was no known role for R285 in binding 2,4-D.” Ex. 1070 ¶ 17. In contrast, Dr. Bollinger testifies that Schleinitz 2004’s teachings “strongly indicat[e] a similar function for Arg285 in RdpA.” Ex. 2015

¶ 141. That testimony provides, at best, an obviousness rationale for potentially redefining the α KGD consensus motif and then extending such redefinition to RdpA—it does not describe the AAD-1 motif as recited in claim 2. Obviousness is not enough. *PowerOasis, Inc. v. T-Mobile USA, Inc.*, 522 F.3d 1299, 1310 (Fed. Cir. 2008) (“Obviousness simply is not enough; the subject matter must be disclosed to establish possession.”).

The teachings of Hotopp and Elkins do not change the result. Ex. 2015 ¶¶ 140–141 (citing Hotopp (Ex. 1029) and Elkins (Ex. 1023)). Those references evidence that researchers were exploring the potential roles of many conserved amino acid residues among members of the α KGD enzyme family. Neither reference discusses RdpA. Ex. 1070 ¶¶ 18, 21–22 (explaining that those papers related to TfdA and TauD). And neither reference proposed redefining the consensus α KGD motif (or establishing any new motif) to include an additional spacer and terminal arginine residue. *Id.* ¶¶ 16–18. As with Schleinitz 2004, Patent Owner’s reliance on those references’ teachings is unavailing and invokes obviousness, at best. The same is true with the paper co-authored by Dr. Hausinger (Ex. 1035 (Müller)), which *in 2006* predicted and then investigated the possible role for Arg285 in RdpA. PO Sur-reply 21–22 (citing Ex. 1035, 1357–65).

Based on the preponderance of the evidence, we find that the priority applications do not reasonably convey to the POSA that the inventors possessed a genus of proteins defined by an “AAD-1 motif” that includes the subject arginine in a (X)₂₈₂₋₂₈₄R subsequence. *Purdue Pharma L.P. v. Faulding Inc.*, 230 F.3d 1320, 1323 (Fed. Cir. 2000) (“[O]ne skilled in the art, reading the original disclosure, must immediately discern the limitation at issue in the claims.”).

(5) *Miscellaneous*

The parties both argue that their opponent is applying the wrong legal standard in assessing whether the priority applications provide sufficient written description support for the claims. PO Resp. 44–48; PO Sur-reply 2–3; Pet. Reply 5–8. According to Patent Owner, all that is required is a “reasonable structure-function correlation” yet Petitioner “essentially seek[s] to elevate the *correlation* standard for common structure to a *causation* standard.” PO Resp. 45 (citing, e.g., testimony from Dr. Hausinger using the phrase “determinative of” in describing the relation between a portion of RdpA’s structure and function (Ex. 1054 ¶¶ 74, 109)); PO Sur-reply 2–3.

We disagree that the parties are invoking an incorrect legal standard in advancing their respective arguments. Rather, the parties are applying the standard as reaffirmed in the Federal Circuit’s *en banc Ariad* decision and its progeny. *See, e.g.*, Pet. 23 (citing *Ariad* as the standard for determining whether a genus is sufficiently described), 30–33 (applying *Ariad* and other precedents including *AbbVie*); PO Resp. 29–31, 44–45 (same). The dispute is principally about the *application* of those precedents on this record. And, as for the alleged structure-function correlation, we agree with Petitioner that “this is not a case of less-than-perfect correspondence or a ‘well-known’ structure-function relationship” because the priority applications and prior art “identified no correlation whatsoever between structure and the claimed (*R*)-enantioselective activity”—particularly, the (*R*)-FOP degrading activity that the Specification asserts was “novel” and the distinguishing characteristic over the prior art. Pet. Reply 6 (citing Ex. 1005 ¶ 18; Ex. 1003, 4:52–58). Indeed, we credit Dr. Hausinger’s testimony that there is no sufficient disclosure of any amino acids among RdpA’s 295 residues that are “important to” the claimed (*R*)-FOP degrading activity. Ex. 1054

¶ 74 (testifying that insofar as structure is identified (accepting, e.g., that an AAD-1 motif is identified), it would be understood as relevant to generic α KG dioxygenase activity because the same motif residues appear in other α KGD enzymes that lack the claimed activity).

Further to its argument about the appropriate legal standard, Patent Owner places significant emphasis on the Federal Circuit’s decision in *Ajinomoto Co. v. ITC*, 932 F.3d 1342, 1360 (Fed. Cir. 2019). PO Resp. *passim*. There are, however, material factual distinctions between the present case and *Ajinomoto*. The structure-function relationship at issue in *Ajinomoto* was shown to be well-understood. *See, e.g., Ajinomoto*, 932 F.3d at 1351 (noting “because *E. coli* promoters were well understood in the art”), 1359 (citing a “well-known link between consensus sequence and promoter strength”). Further, the court pointed out, the invention in *Ajinomoto* related to a discovery that a specific gene conferred microbial resistance to phenylalanine when that gene’s expression was enhanced, *not* to the means of expression enhancement (i.e., replacement of a native promoter sequence with a more potent promoter sequence) on which the written description dispute turned. *Id.* at 1359 (finding the invention was “not the well-known techniques for performing the amplification or expression enhancement”). Indeed, on the disputed issue, the court explained, “the genus of more potent promoters was already well explored in the relevant art by the time of the . . . invention.” *Id.*;⁴³ *see also PureCircle*, 2024 WL 20567 at *4 n.10

⁴³ In addition to the promoters being well understood in the art, the court also noted that the patent expressly included four examples of more potent promoters and cited another article with data on fourteen promoters and instructions for evaluating promoter strength, such that the POSA could

(suggesting that the reasoning of *Ajinomoto* would not apply absent a known or disclosed structure-function correlation, and where substantial experimentation would be involved).

This case presents a very different factual backdrop—the alleged invention is the discovery that AAD-1 proteins degrade herbicides in two different classes, including (*R*)-FOPs. *See, e.g.*, Ex. 1003, 4:15–19, 4:52–62. According to the '745 patent, “[n]o α -ketoglutarate-dependent dioxygenase enzyme has previously been reported to have the ability to degrade herbicides of different chemical classes and modes of action.” *Id.* at 4:55–58. If Patent Owner was first to discover a broad genus of proteins that degrades not only phenoxy auxins but also herbicidally-active (*R*)-FOPs, it is unsurprising that that prior art evidences no well-known correlation between the common structures and function for this newly-defined AAD-1 genus. It was, therefore, incumbent on Patent Owner to supply the necessary disclosures—by describing sufficient representative species or the common structural features defining the newly-claimed genus that correlate to this newly-claimed function. For the reasons explained above, we agree with Petitioner that the priority applications do not provide a written description sufficient to support the claimed genus.

Patent Owner also argues that Petitioner’s analysis imposes too strict a standard because a sufficient written description does not require ruling out all inoperative embodiments. PO Resp. 48–49 (citing the substitution of the R285 residue resulting in inactive enzyme). The priority applications, however, do not describe a single operative embodiment that lacks the R285

make predictable changes to the native promoter to arrive at more potent promoters. *Ajinomoto*, 932 F.3d at 1359, 1361.

residue and that degrades phenoxy auxins and (*R*)-FOPs. There is a complete absence of any structure-function disclosure relative to R285 or the (*X*)₂₈₂₋₂₈₄*R* subsegment in the priority applications, and Patent Owner admits that this structure is “critical” to activity yet [REDACTED] Ex. 1008, 452, 455; Ex. 2004, 580. This only underscores that the disclosure in the priority applications, which allows *any* amino acid to occupy position 285 and provides no description of proteins with the claimed functionality except for three $\geq 99\%$ identical RdpA variants, is insufficient to support the much broader genus of dual-degradation proteins claimed.⁴⁴

Patent Owner also argues that its challenged claims are not purely functional but also include structural features. PO Resp. 47–48. That is true, but the question remains whether those structural features, and a structure-function correlation between them, are sufficiently described in the priority applications or otherwise known. For the reasons discussed above, we find that the answer to that question is “no.”⁴⁵

⁴⁴ Patent Owner’s modification of the AAD-1 motif formula to recite the R285 residue and the (*X*)₂₈₂₋₂₈₄*R* subsegment is addressed above where we discuss claim 2. We also address above the assertion that a POSA would have recognized the significance of R285 from Schleinitz 2004.

⁴⁵ Patent Owner suggests that “antibody” cases like *AbbVie* are “inapposite” because enzymes are different proteins. PO Resp. 47–48, 57. We decline to limit the Federal Circuit’s precedents as applicable to some proteins, but not others. Pet. Reply 18 (noting that the Federal Circuit has applied *AbbVie* in cases involving enzymes). Of course, more may be known about the functions of some proteins than others and that is worthy of consideration as part of the written description analysis. Insofar as Patent Owner cites *Campbell* (a case about antibodies) in support of its argument that the priority applications’ disclosure is sufficient, that decision is factually distinguishable because the specification there described functionally-active embodiments with sequence identities both above and below the claimed

The parties also suggest that we should discount the testimony of their opponent's respective expert based on alleged overreach in prior matters. PO Sur-reply 4 (arguing Dr. Hausinger has a "history of overreaching," citing a result in a 2012 district court litigation between Bayer Cropscience AG and Dow Agrosiences LLC); Pet. Reply 21 ("*PureCircle* held that Dr. Bollinger's proposal of 9,000 enzymes still resulted in too much testing") (citing 2024 WL 20567 at *3). The opinions of those witnesses in prior cases is not a factor in this case. Drs. Hausinger and Bollinger both appear to be respected and qualified enzymologists (*see supra* Section III.B) and their testimony based on the unique record developed in this case has been considered accordingly. Ex. 1072, 41:1–3.

Patent Owner argues that Petitioner erroneously relies on post-filing date evidence. PO Resp. 51–55 (asserting that Petitioner errs by relying on evidence of the state of the art as late as 2019, long after the 2004–2005 filing dates). Both sides here rely on post-filing testing of AAD-1 variants alleged to include the "AAD-1 motif" and $\geq 85\%$ sequence identity compared to SEQ ID NO: 9. As discussed above, what that testing indicates on this record is that sometimes those variants exhibit the claimed functionality (e.g., the cited FT enzymes with $\sim 89\text{--}91\%$ identity developed by Petitioner between 2016 and 2019) and sometimes they do not (e.g., inactive variants with $\sim 94\text{--}95\%$ sequence identity and lacking R285 residue, [REDACTED] Ex. 1052, 2087, 2090; Ex. 2015 ¶¶ 136–137; Ex. 1008, 452, 455, 462–463; Ex. 2002, 433–444; Ex. 2004; Ex. 1070 ¶¶ 65–68. On balance, we find that

$\geq 90\%$ limit. *See Ex parte Campbell*, No. 2021-000865, 2–3, 15 n.3 (PTAB July 20, 2021).

this evidence is consistent with Petitioner’s position and Dr. Hausinger’s testimony that substantial experimentation would have been required in the 2004–2005 timeframe to determine which variants within the scope of the challenged claims would or would not exhibit the functionality claimed.

We need not rely on the post-filing publications from Müller (in 2006) and Chekan (in 2019) to resolve the dispute here. Ex. 1035; Ex. 1019. But, even considering those publications, we find that they would on balance lend further support to Petitioner’s challenge—reinforcing that little was known about the structures in RdpA or other AAD proteins that correlate to substrate specificity in 2004/2005. *See, e.g.*, Ex. 1035, 1358 (teaching that “essentially nothing is known about the phenoxypropanoic acid-binding sites of these proteins [(e.g., RdpA)], especially with regard to the structural basis of enantiospecificity”), 1365 (citing high-sequence identity homologs with opposite stereoselectivities and disclosing “if the entire proteins exhibit this level of identity, the enantiospecificity is likely to be conferred by a few key [but then unknown] amino acid residues”); Ex. 1019, 13300 (teaching “little is known about protein-substrate interactions or how these [AAD] enzymes catalyze enantioselective oxidation of herbicides” and further “[t]o date, no crystal structure has been reported for any member of the AAD family, and there is, therefore, an insufficient knowledge base to distinguish bona fide AADs from other enzymes of the Fe(II)/ α KG dioxygenase class.”). We credit Dr. Hausinger’s testimony about the import of those publications over the competing testimony of Dr. Bollinger. *See, e.g.*, Ex. 1054 ¶¶ 57, 88, 105–106; Ex. 1070 ¶ 22; Ex. 2015 ¶¶ 119–121.

Lastly, Patent Owner argues, for the first time in Sur-reply, that Petitioner disregards the claim language based on a “*Sub Silentio* Claim Construction.” PO Sur-reply 3–6 (arguing Petitioner’s written-description

arguments focus on “(R)-enantioselectivity” or “(R)-enantiospecificity,” which are terms that do not appear in the claims).

This argument is untimely and is waived. Paper 13 (“Scheduling Order”), 9 (“Patent Owner is cautioned that any arguments not raised in the response may be deemed waived.”). Failure to timely assert a right or raise an argument constitutes forfeiture. *In re Google Tech. Holdings LLC*, 980 F.3d 858, 862–63 (Fed. Cir. 2020) (quoting *United States v. Olano*, 507 U.S. 725, 733 (1993)). Patent Owner offers no persuasive justification for why this argument was not made in its Patent Owner Response. We doubt such a justification could be shown because the lack of descriptive support for the AAD-1 enzyme’s enantioselective activity was raised throughout the Petition, in further authorized pre-institution briefing, and in the Institution Decision. *See, e.g.*, Pet. 26–29 (arguing no disclosure reveals structural features “necessary for the (R)-enantioselective activity against chiral phenoxy auxins and aryloxyphenoxypropionates covered by the claims”), 37, 49; Paper 9, 2 (“[T]he claims do not recite a generic α KG dioxygenase activity They require *R*-stereospecific activity, i.e., the ability to degrade *R*-FOPs, and phenoxy auxin herbicides, which include *R*-phenoxy auxins (*see* claim 10).”), 5 (“No disclosure here of what structures provide *R*-stereospecificity”); Inst. Dec. 44–45 (noting Petitioner’s argument that “presence of canonical α KGD residues in SEQ ID NO: 9 does not describe the structural basis for the enzyme’s specificity for (*R*)-FOPs”), 48–51.

In any event, even considering Patent Owner’s belated argument about an alleged *sub-silentio* claim construction, it is unpersuasive because there is no question that all the challenged claims require the AAD-1 enzymes be enantioselective for and degrade (*R*)-FOPs. *See* Section II.D. And, the ’745 patent itself highlights that this enantioselectivity is what

distinguishes the invention over other enzymes in the prior art. *See, e.g.*, Ex. 1003, 4:52–58 (describing the “subject invention” as relating to an enzyme that degrades 2,4-D and FOP herbicides “in an enantiospecific manner”—activity that the patent indicates distinguish the invention from other α KGD enzymes), 110:59–60 (disclosing “AAD-1 (v1) . . . is specific for (R)-enantiomers”).

d) Conclusion on Prior Art Status of Corteva ’437

For the reasons explained above, we determine that the challenged claims lack sufficient written description support in the priority applications. The claims are not entitled to a priority date that is earlier than October 7, 2016. Petitioner has shown by a preponderance of the evidence that Corteva ’437 is prior art under 32 U.S.C. § 102(a)(1) based on Corteva ’437’s November 17, 2005, publication date. Ex. 1021, code (43).

2. Anticipation and Obviousness

Petitioner contends that Corteva ’437 anticipates and/or renders obvious challenged claims 1–29. Pet. 19–20 (arguing that, notwithstanding the lack of written description sufficient to support the breadth of the genus claimed, Corteva ’437 suffices for anticipation because it discloses transgenic maize plants and plant cells that fall within the scope of those claims (citing *Eli Lilly and Co. v. Barr Labs., Inc.*, 251 F.3d 955, 971 (Fed. Cir. 2001)); 41–55 (limitation-by-limitation analysis for claims 1–29); Ex. 1054 ¶¶ 485–487, 493–563 (Dr. Hausinger’s testimony explaining how Corteva ’437 anticipates and renders obvious the challenged claims); *see also id.* ¶¶ 111–209 (analysis for claims of the ’555 patent, which Dr. Hausinger cross-references in his analysis of the ’745 patent’s challenged claims).

In the Patent Owner Response, Corteva does not dispute that claims 1–29 are anticipated by, or would have been obvious over, Corteva ’437. Instead, Patent Owner’s counterargument relates to whether Corteva ’437 qualifies as prior art, which dispute turned on the written description issues discussed above. *See supra* Section III.D.1. PO Resp. 28–29 (noting only that Grounds IA and IB “depend on Petitioners’ written-description-based priority challenge”), 29–60 (addressing written-description issues); Pet. Reply 5 (“Corteva does not dispute that, if claims 1–29 are not entitled to pre-October 2016 priority—*i.e.*, if they are not supported by the 2004/2005 Applications—then the Board should find every claim anticipated and obvious based on Corteva ’437, published in November 2005.”).

We determine that Corteva ’437 is prior art for reasons discussed above. *See supra* Section III.D.1.

We have also considered Petitioner’s unrebutted argument and evidence on the question whether Corteva ’437 discloses at least one species that falls within the broader scope of the challenged claims and, thus, anticipates those claims. For claim 1, for example, Petitioner argues that Corteva ’437 discloses and claims “transgenic soybean plant cells modified to comprise a recombinant polynucleotide that encodes an AAD-1 protein”—specifically including the protein encoded by SEQ ID NO:9. Pet. 42 (citing Ex. 1021 ¶¶ 18–20, 95–105, 149–151, claims 1–4; Ex. 1054 ¶¶ 114–116). Indeed, Corteva ’437 discloses that “transgenic expression of the subject *AAD-1* genes is exemplified in, for example, soybean.” Ex. 1021 ¶ 104 (“soybeans are a preferred crop for transformation according to the subject invention”). Petitioner contends Corteva ’437 discloses that the protein exhibits activity to degrade phenoxy auxin and (*R*)-FOPs herbicides and, when expressed, would render soybean plant cells tolerant to the subject

herbicides as claimed. *Id.* at 42–46 (citing Ex. 1021 ¶¶ 20, 74–76, 86–87, 92–99, 104, 179–215, 260–269, Tbls. 6–8, 16, claims 1–4; Ex. 1054 ¶¶ 496–497). Petitioner argues that “SEQ ID NO:9 is 100% identical to itself” and related AAD-1 (v2, v3) have 99% identity to SEQ ID NO:9, much higher than and, thus, meeting the “at least 85% sequence identity” limitation. *Id.* at 43–44 (citing Ex. 1021 ¶¶ 46, 51, sequence listings for SEQ ID NOS:9, 10, 11; Ex. 1054 ¶ 121–122). Petitioner also contends that Corteva ’437 discloses SEQ ID NOS:9 and 10, which have the residues and residue-spacing that meet the formula of the recited AAD-1 motif. *Id.* at –45 (“Corteva ’437 also discloses and claims proteins having the claim 1 AAD-1 motif” and then citing the residues and their positions in SEQ ID NO:9 as conforming to the motif formula); Ex. 1021 ¶¶ 51–52; Ex. 1054 ¶¶ 122. Dr. Hausinger provides unrefuted testimony that supports Petitioner’s contentions above. Ex. 1054 ¶¶ 111–122, 485–487, 493–498.

We have also considered and we credit Petitioner’s contentions and Dr. Hausinger’s supporting and un rebutted testimony for all the remaining challenged claims. Pet. 46–55; Ex. 1054 ¶¶ 499–563.

Based on the argument and evidence noted above, we find that Petitioner has shown by a preponderance of the evidence that Corteva ’437 anticipates claims 1–29. We further conclude that claims 1–29 would have been obvious over Corteva ’437. Pet. 55 (arguing Corteva ’437 renders obvious all challenged claims because “anticipation is the epitome of obviousness” (*Realtime Data*, 912 F.3d at 1373)); Ex. 1054 ¶¶ 209, 564.

Patent Owner, for the first time in Sur-reply, argues that Petitioner’s anticipation and written description positions are incompatible and, thus, urges us to reject the anticipation challenge. PO Sur-reply 22–24 (arguing “[f]or anticipation, Petitioners contend that the 2005 Priority Application,

Corteva '437 discloses the AAD-1 motif because SEQ ID NOs: 9 and 10 have the required residues and their required spacing. Pet. 43–44. For written description, however, Petitioners contend the opposite, denying those same disclosures in the same Corteva '437 application” (citing alleged inconsistencies between the Petition and Hausinger’s deposition testimony (e.g., Pet. 43–44 versus Ex. 2066, 71:12–18))). This argument is untimely and Patent Owner does not explain why it was not raised in the Patent Owner Response.⁴⁶ In any event, the argument is unpersuasive because anticipation and written description are different inquiries. Tr. 13:3–15 (noting the Sur-reply argument about alleged inconsistencies but explaining “the answer is very simple. This is in the nature of species genus anticipation”). *Chester v. Miller*, 906 F.2d 1574, 1577 (Fed. Cir. 1990) (holding “no impermissible anomaly or logical inconsistency” in a parent application serving as invalidating prior art against “broader claims” that application did not adequately describe); *Eli Lilly*, 251 F.3d at 971 (explaining that “a later genus claim limitation is anticipated by, and therefore not patentably distinct from, an earlier species claim.”).

3. Conclusion

Considering the full record developed through trial and for the reasons explained above, we determine that the challenged claims are entitled to priority no earlier than October 7, 2016. Corteva '437 therefore qualifies as prior art and, as discussed above, we determine that the challenged claims are anticipated by and would have been obvious over Corteva '437.

⁴⁶ To the extent Patent Owner cites testimony of Dr. Hausinger purporting to deny disclosure of the claimed “AAD-1 motif” in Corteva '437, we note that Dr. Hausinger gave similar testimony during his first deposition and prior to the Patent Owner Response due date. *See, e.g.*, Ex. 2016, 39:12–40:2.

E. Obviousness over Saari Combinations (Grounds IIA and IIB)

Petitioner contends that claims 1–6, 9–12, and 29 would have been obvious over Saari 1998 or Saari 1999, in combination with Schleinitz and Streber (Ground IIA). Pet. 55–60. Petitioner contends dependent claims 7, 8, and 13–28 would have been obvious over the above combination with the further addition of Jepson (Ground IIB). *Id.* at 60–64. For Grounds IIA and IIB, Petitioner assumes the challenged claims are entitled to priority to April 30, 2004. *Id.* at 16–17; Ex. 1003, code (60).

Because we determine that Petitioner carried its burden to show unpatentability of all challenged claims under Grounds IA and IB (as discussed above), we need not reach Grounds IIA and IIB. *SAS*, 138 S. Ct. at 1359 (holding petitioner is entitled to a final written decision that covers all challenged claims); *Bos. Sci. Scimed, Inc. v. Cook Grp. Inc.*, 809 F. App’x 984, 990 (Fed. Cir. 2020) (nonprecedential) (explaining that “the Board has discretion to decline to decide additional instituted grounds once petitioner has prevailed on all its challenged claims.”); *Beloit Corp. v. Valmet Oy*, 742 F.2d 1421, 1423 (Fed. Cir. 1984) (holding once a dispositive issue is decided by an agency, there is no need to decide other potentially dispositive issues).

IV. MOTIONS

Three motions to seal remain pending (Papers 28, 33, and 38). Patent Owner moves to seal Petitioner’s Reply (Paper 27) as well as the Reply Declaration of Dr. Hausinger (Ex. 1070). Paper 28. Patent Owner moves to seal Dr. Hausinger’s Deposition Transcript dated August 1, 2024 (Ex. 2066). Paper 33. And, lastly, Patent Owner moves to seal Petitioner’s oral hearing demonstratives (Paper 40). Paper 38. None of the motions is opposed.

The Board recognizes “a strong public policy for making all information filed in a quasi-judicial administrative proceeding open to the public, especially in an *inter partes* review which determines the patentability of claims in an issued patent and therefore affects the rights of the public.” *Garmin Int’l v. Cuozzo Speed Tech., LLC*, IPR2012-00001, Paper 34, 1–2 (PTAB Mar. 14, 2013). Except as otherwise ordered by the Board, the record of an *inter partes* review shall be made available to the public. 35 U.S.C. § 316(a)(1); 37 C.F.R. § 42.14.

The standard for granting a motion to seal is “for good cause.” 37 C.F.R. §§ 42.20(c), 42.54(a). In determining whether good cause has been shown, the Board considers, among other things, whether the information sought to be sealed is truly confidential and whether the expressed interest in maintaining confidentiality outweighs the strong public interest in an open record. *Argentum Pharm. LLC v. Alcon Rsch., Ltd.*, IPR2017-01053, Paper 27 at 2–3 (PTAB Jan. 19, 2018) (informative).

Each of Patent Owner’s three motions to seal is premised on essentially the same rationale. Specifically, Patent Owner contends that the Reply (Paper 27), the Hausinger Reply Declaration (Ex. 1070), Hausinger’s deposition transcript (Ex. 2066), and Petitioner’s demonstratives (Paper 40) include Patent Owner’s confidential information—proprietary protein research data. *See* Paper 28, 1; Paper 33, 1; Paper 38, 1. Indeed, Patent Owner contends, the materials it asks be sealed quote, discuss, or refer to Patent Owner’s protein research data in Exhibits 2002 and/or 2004, which exhibits the Board has already sealed in this proceeding. *See, e.g.*, Paper 38, 1, 4; Paper 16 (Order granting motion to seal and entering protective order); Paper 7, App’x A (Protective Order).

Patent Owner has shown good cause for granting the relief sought in its three pending motions. Papers 28, 33, 38. Our reasoning is the same as stated in our prior order related to the sealing of Exhibits 2002 and 2004. Paper 16, 3–5. Additionally, supporting the showing of good cause, Patent Owner (or Petitioner, as applicable) has filed minimally-redacted, publicly-available versions of the papers and exhibits that are the subject of the three pending motions. *See* Paper 28, App’x A (redacted version of Pet. Reply), App’x B (redacted version of Ex. 1070); Ex. 2066 (public version filed Aug. 16, 2024); Paper 41 (public version of Petitioner’s demonstratives). The public’s interest in an open and understandable record is not materially impaired by the minimal redactions at issue here.

V. CONCLUSION⁴⁷

Petitioner has shown by a preponderance of the evidence that the challenged claims are unpatentable as summarized below.

Claims	35 U.S.C. §	Reference(s)/Basis	Claims Shown Unpatentable	Claims Not shown Unpatentable
1–29	102	Corteva ’437	1–29	
1–29	103	Corteva ’437	1–29	
1–6, 9– 12, 29	103	Saari 1998, Saari 1999, Schleinitz, Streber ⁴⁸		
7, 8, 13– 28	103	Saari 1998, Saari 1999, Schleinitz, Streber, Jepson ⁴⁹		
Overall Outcome			1–29	

⁴⁷ Should Patent Owner wish to pursue amendment of the challenged claims in a reissue or reexamination proceeding subsequent to the issuance of this decision, we draw Patent Owner’s attention to the April 2019 *Notice Regarding Options for Amendments by Patent Owner Through Reissue or Reexamination During a Pending AIA Trial Proceeding*. See 84 Fed. Reg. 16,654 (Apr. 22, 2019). If Patent Owner chooses to file a reissue application or a request for reexamination of the challenged patent, we remind Patent Owner of its continuing obligation to notify the Board of any such related matters in updated mandatory notices. See 37 C.F.R. § 42.8(a)(3), (b)(2).

⁴⁸ Because all challenged claims are determined to be unpatentable in view of Corteva ’437, we decline to further address this additional ground.

⁴⁹ Because all challenged claims are determined to be unpatentable in view of Corteva ’437, we decline to further address this additional ground.

VI. ORDER

In consideration of the foregoing, it is hereby:

ORDERED that Petitioner has proved by a preponderance of the evidence that claims 1–29 are unpatentable;

FURTHER ORDERED that Patent Owner’s Motions to Seal (Papers 28, 33, and 38) are *granted* as provided above;

FURTHER ORDERED that within ten (10) business days, the parties will jointly provide a minimally redacted version of this decision that is available to the public; and

FURTHER ORDERED that, because this is a Final Written Decision, parties to the proceeding seeking judicial review of the decision must comply with the notice and service requirements of 37 C.F.R. § 90.2.

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