

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

PAR PHARMACEUTICAL, INC.,
Petitioner,

v.

JAZZ PHARMACEUTICALS IRELAND LTD. and
JAZZ PHARMACEUTICALS, INC.,
Patent Owner.

Case IPR2016-00002
Patent 8,772,306

Before ERICA A. FRANKLIN, BRIAN P. MURPHY, and
CHRISTOPHER G. PAULRAJ, *Administrative Patent Judges*.

PAULRAJ, *Administrative Patent Judge*.

DECISION
Denying Institution of *Inter Partes* Review
37 C.F.R. § 42.108

I. INTRODUCTION

Par Pharmaceutical, Inc. (“Petitioner”) filed a Petition (Paper 3, “Pet.”), requesting institution of an *inter partes* review of claims 1–34 of U.S. Patent No 8,772,306 (Ex. 1001, “the ’306 patent”). Jazz Pharmaceuticals Ireland Ltd. and Jazz Pharmaceuticals, Inc. (collectively, “Patent Owner”) timely filed a Preliminary Response (Paper 10, “Prelim. Resp.”). We have jurisdiction under 35 U.S.C. § 314, which provides that an *inter partes* review may not be instituted “unless . . . there is a reasonable likelihood that the petitioner would prevail with respect to at least 1 of the claims challenged in the petition.”

Upon consideration of the Petition and the Preliminary Response, and for the reasons explained below, we determine Petitioner has not shown a reasonable likelihood that it would prevail with respect to any of the challenged claims. We, therefore, decline to institute an *inter partes* review of claims 1–34 of the ’306 patent.

A. Related Proceedings

Petitioner has identified a related litigation proceeding in the District of New Jersey: *Jazz Pharmaceuticals, Inc. v. Par Pharmaceutical, Inc.*, C.A. No. 14-6150 (D.N.J.). Pet. 1. Patent Owner has further identified the following related litigation proceedings involving the ’306 patent: *Jazz Pharmaceuticals, Inc. v. Amneal Pharmaceuticals LLC*, 2:13-cv-391 (consolidated) (D.N.J.); *Jazz Pharmaceuticals, Inc. v. Roxane Laboratories, Inc.*, 2:15-cv-1360 (D.N.J.); *Jazz Pharmaceuticals, Inc. v. Wockhardt Bio AG*, 2:15-cv-5619 (D.N.J.); and *Jazz Pharmaceuticals, Inc. v. Lupin Ltd.*, 2:15-cv-6548 (D.N.J.). Paper 8, 1–2. Patent Owner also identified two other cases, *Jazz Pharmaceuticals, Inc. v. Amneal Pharmaceuticals, LLC*,

2:15-cv-6562 (D.N.J.) and *Jazz Pharmaceuticals, Inc. v. Par Pharmaceutical, Inc.*, 2:15-cv-7580 (D.N.J.), concerning a patent related to the '306 patent. *Id.* at 2.

In addition, Ranbaxy Inc. and Amneal Pharmaceuticals each filed separate petitions for *inter partes* review of the '306 patent. *See* IPR2016-00024; IPR2016-00546.

B. The '306 Patent (Ex. 1001)

The '306 patent issued on July 8, 2014, and claims a priority date as early as March 1, 2013. *See* Ex. 1001, Title Page. It names Mark Eller as the sole inventor. *Id.*

The '306 patent relates generally to methods for improving the safety and efficacy of the administration of gamma-hydroxybutyrate (“GHB”) or a salt thereof to a patient. *Id.*, Abstract. More specifically, the '306 patent is concerned with treating patients suffering from certain disorders such as cataplexy or narcolepsy, who are concomitantly receiving treatment with valproate, with a reduced dose of GHB. *Id.* at 1:15–36. The specification states that valproate can increase or prolong the effects of GHB, resulting in unsafe conditions such as excessive daytime sleepiness. *Id.* at 15:19–16:21. In certain embodiments, the reduced amount of GHB ranges from 1% to 50% of the effective dose normally given to the patient. *Id.* at 1:32–36.

C. Illustrative Claims

Petitioner challenges claims 1–34 of the '306 patent. All of the challenged claims are directed to methods of treating certain sleep disorders by orally administering a reduced dosage of GHB to patients who are concomitantly receiving valproate.

Claims 1, 11, 19, 30, and 33 are independent. Independent claim 1 is illustrative, and reproduced below:

1. A method for treating a patient who is suffering from excessive daytime sleepiness, cataplexy, sleep paralysis, apnea, narcolepsy, sleep time disturbances, hypnagogic hallucinations, sleep arousal, insomnia, or nocturnal myoclonus with gamma-hydroxybutyrate (GHB) or a salt thereof, said method comprising:

orally administering to the patient in need of treatment at least 5% decrease in an effective dosage amount of the GHB or salt thereof when the patient is receiving a concomitant administration of valproate, an acid, salt, or mixture thereof.

Independent claims 11, 19, 30, and 33 also require either administering or recommending a reduced dose of GHB to a patient who is taking valproate. Petitioner treats all claims similarly under each ground asserted in the Petition. Therefore, we treat all the claims similarly for purposes of our analysis in this Decision.

D. The Asserted Grounds of Unpatentability

Petitioner challenges the patentability of the claims of the '306 patent on the following grounds:

References	Basis	Claims challenged
Xyrem 2005 Label, ¹ Depakote 2011 Label, ² Cagnin, ³ Waszkielewicz, ⁴ and the FDA Guidance ⁵	§ 103(a)	1–34
Xyrem 2005 Label, Depakote 2011 Label, Cagnin, Waszkielewicz, Weiss, ⁶ and the FDA Guidance	§ 103(a)	1–34

II. DISCUSSION

A. Claim Construction

We interpret claims using the “broadest reasonable construction in light of the specification of the patent in which [they] appear[.]” 37 C.F.R. § 42.100(b); *see also In re Cuozzo Speed Techs., LLC*, 793 F.3d 1268, 1278–

¹ Jazz Pharmaceuticals, Inc., Prescribing Information and Medication Guide for XYREM[®] (sodium oxybate) (Nov. 18, 2005) (Ex. 1006).

² Abbvie, Inc., Prescribing Information and Medication Guide for DEPAKOTE (divalproex sodium) (Oct. 7, 2011) (Ex. 1007).

³ Cagnin, A. et al., *γ-Hydroxybutyric Acid-Induced Psychosis and Seizures*, 21(2) *Epilepsy Behav.* 203–05 (2011) (Ex. 1008).

⁴ Waszkielewicz, A. et al., *γ-Hydroxybutyric Acid (GHB) and Its Chemical Modifications: A Review of the GHBergic System*, 56(1) *Pol. J. Pharmacol.* 43–49 (2004) (Ex. 1009).

⁵ FDA’s Center for Drug Evaluation and Research, Guidance for Industry: Drug Interaction Studies—Study Design, Data Analysis, Implications for Dosing, and Labeling Recommendations (Feb. 2012) (Ex. 1011).

⁶ Weiss, T. et al., *Gamma-Hydroxybutyrate (GHB) and Topiramate—Clinically Relevant Drug Interaction Suggested by a Case of Coma and Increased Plasma GHB Concentration*, 69(5) *Eur. J. Clin. Pharmacol.* 1193–94 (2013) (Ex. 1010).

79 (Fed. Cir. 2015) (“Congress implicitly approved the broadest reasonable interpretation standard in enacting the AIA,”⁷ and “the standard was properly adopted by PTO regulation.”), *cert. granted, sub nom. Cuozzo Speed Techs. LLC v. Lee*, 136 S. Ct. 890 (mem.) (2016). Under the broadest reasonable construction standard, claim terms are given their ordinary and customary meaning, as would be understood by one of ordinary skill in the art at the time of the invention. *In re Translogic Tech., Inc.*, 504 F.3d 1249, 1257 (Fed. Cir. 2007). “Absent claim language carrying a narrow meaning, the PTO should only limit the claim based on the specification . . . when [it] expressly disclaim[s] the broader definition.” *In re Bigio*, 381 F.3d 1320, 1325 (Fed Cir. 2004). “Although an inventor is indeed free to define the specific terms used to describe his or her invention, this must be done with reasonable clarity, deliberateness, and precision.” *In re Paulsen*, 30 F.3d 1475, 1480 (Fed. Cir. 1994).

We determine that no explicit construction of any claim term is necessary to determine whether to institute a trial in this case. *See, e.g., Wellman, Inc. v. Eastman Chem. Co.*, 642 F.3d 1355, 1361 (Fed. Cir. 2011) (“[C]laim terms need only be construed ‘to the extent necessary to resolve the controversy.’”) (quoting *Vivid Techs., Inc. v. Am. Sci. & Eng’g, Inc.*, 200 F.3d 795, 803 (Fed. Cir. 1999)).

B. Prior Art Relied Upon

Petitioners rely upon the following prior art in its challenges.

⁷ The Leahy-Smith America Invents Act, Pub. L. No. 112–29, 125 Stat. 284 (2011) (“AIA”).

1. Xyrem 2005 Label (Ex. 1006).

The Xyrem 2005 Label discloses the prescribing information for an oral administration of sodium oxybate (i.e., a GHB salt), a central nervous system depressant for treating excessive daytime sleepiness and cataplexy. Ex. 1006 at 1, 7. According to the Xyrem 2005 Label, the “dose of Xyrem should be titrated to effect.” *Id.* at 22. The Xyrem 2005 Label recommends a starting dose of sodium oxybate of 4.5 g/night, divided into two equal doses of 2.25 g. *Id.* at 22–23. The Xyrem 2005 Label also recommends increasing the dosage to a maximum of 9 g/night in increments of 1.5 g/night (0.75 g per dose), and further discloses that the effective dose range is 6 to 9 g/night. *Id.* at 23.

The Xyrem 2005 Label includes a black box warning on the first page that the drug “[s]hould not be used with alcohol or other CNS depressants.” *Id.* at 1. Additionally, the Xyrem 2005 Label indicates that “[s]odium oxybate is contraindicated in patients with succinic semialdehyde dehydrogenase [SSADH] deficiency.” *Id.* at 8.

2. Depakote 2011 Label (Ex. 1007).

The Depakote 2011 Label discloses the prescribing information for an oral administration of divalproex sodium (valproate), an anti-epileptic drug for treating seizures, migraine headaches, and bipolar disorder. Ex. 1007, 1. The Depakote 2011 Label warns that “[s]ince valproate products may produce CNS depression, especially when combined with another CNS depressant (e.g., alcohol), patients should be advised not to engage in hazardous activities” *Id.* at 48.

3. *Cagnin (Ex. 1008).*

Cagnin is a case report describing a patient affected with bipolar disorder and alcohol dependence who experienced seizures after co-administration of valproate and GHB. Ex. 1008, 203. The patient had been receiving a 500 mg dose of valproate, twice daily, as mood-stabilizing treatment. *Id.* The patient also began receiving a dose of 3500 mg/day of GHB for treatment of alcohol dependence. *Id.* Subsequently, the patient developed psychotic behavior and experienced seizures. *Id.* at 203–204. Cagnin states that “[o]nly GHB discontinuation restored the neurotransmitter balance and caused seizures to cease.” *Id.* at 205.

Cagnin suggests that an interaction between valproate and GHB may have triggered the seizures. *Id.* In particular, Cagnin states that valproate is a potent *in vitro* inhibitor of SSADH, which “catalyzes the production of succinate from succinic semialdehyde [SSA], an intermediate product in the metabolic pathway transforming GHB into GABA and vice versa.” *Id.* Cagnin also discloses that “[a]n inherited deficiency of SSADH activity leads to accumulation of [SSA] and, consequently, a 30-fold increase in GHB level and a 2- to 4-fold increase in GABA in the brains of affected rats,” resulting in tonic-clonic seizures and convulsive status epilepticus. *Id.*

4. *Waszkielewicz (Ex. 1009).*

Waszkielewicz provides a review of the “GHBergic system,” and aims to present the “mechanisms of [GHB] action from pharmacological point of view, which may help to properly treat intoxication and other pathological states caused by GHB ingestion.” Ex. 1009, 43. According to Waszkielewicz, “GHB originates from GABA which is metabolized by a transaminase to succinic semialdehyde and then by a dehydrogenase to

GHB.” *Id.* at 44. Waszkielewicz further observes that GHB dehydrogenase inhibitors, including valproate, can intensify the effects observed after administration of GHB. *Id.* at 48. Waszkielewicz states that “[t]he effect is observed due to inhibited GHB metabolism.” *Id.*

5. *The FDA Guidance (Ex. 1011).*

The FDA Guidance “provides recommendations for sponsors of new drug applications (NDAs) and biologics license applications (BLAs) for therapeutic biologics regulated by CDER regarding *in vitro* and *in vivo* studies of drug metabolism, drug transport, and drug-drug or drug-therapeutic protein interactions.” Ex. 1011, 1. The FDA Guidance recommends that “[i]nteractions between an investigational new drug and other drugs should be defined during drug development, as part of an adequate assessment of the drug’s safety and effectiveness.” *Id.* at 2. The FDA Guidance indicates that drug-drug interactions should be studied to determine “the need for dosage adjustments, additional therapeutic monitoring, a contraindication to concomitant use, or other measures to mitigate risk.” *Id.*

6. *Weiss (Ex. 1010).*

Weiss is a letter to the editor describing a 52-year old woman who was hospitalized for worsening chronic cluster headache. Ex. 1010, 1193. The patient had taken Xyrem[®] (sodium oxybate) at a dose of 4.5 grams twice-nightly for the previous six years. *Id.* A single dose of 25 mg of topiramate was added to the patient’s therapeutic regime, and the next morning, the patient developed confusion, as well as intermittent myoclonic jerks, miosis, and a rapid onset of coma. *Id.* Weiss observed that “the GHB concentration 5 h after the second daily dose of GHB was 2.8-fold higher

than without topiramate.” *Id.* Weiss suggests that “the coma was drug induced due to a pharmacokinetic interaction between GHB and topiramate.” *Id.* Weiss further notes that prior “[i]n vitro studies have demonstrated that GHB-dehydrogenase is inhibited by the antiepileptic drugs valproate and ethosuximide . . . , but according to Xyrem®’s product information no interaction studies with antiepileptics have been performed in humans.” *Id.* Until the interactions between GHB and antiepileptic drugs are further evaluated, Weiss recommends “using such combinations only with great care.” *Id.*

C. Analysis of Petitioner’s Patentability Challenges

1. Obviousness of Claims 1–34 Based on the Xyrem 2005 Label, Depakote 2011 Label, Cagnin, Waszkielewicz, and the FDA Guidance

Petitioner contends that claims 1–34 are obvious based on the combination of the Xyrem 2005 Label, the Depakote 2011 Label, Cagnin, Waszkielewicz, and the FDA Guidance. Pet. 18–41. In addition to the teachings of the references, Petitioner relies upon the Declaration of John W. Winkelman, M.D., Ph.D. Ex. 1003.

With respect to each of the challenged claims 1–34, Petitioner asserts that, based on the recognition of both potential pharmacodynamic interactions (by the Xyrem 2005 Label and the Depakote 2011 Label) and pharmacokinetic interactions (by Cagnin and Waszkielewicz), a skilled artisan would have been motivated to combine these references to arrive at the claimed invention. Pet. 22. According to Petitioner, the skilled artisan would subscribe to the principle of “start low and go slow,” reducing the dose beyond what was suggested in the Xyrem 2005 Label and re-titrating

the dose to effect. *Id.* at 25–27. Petitioner argues that the skilled artisan “would have found it obvious to try to reduce the dosage of GHB by **at least** 5%—and likely more—when a patient is concomitantly receiving valproate with a reasonable expectation of success in avoiding toxicity from GHB due to an interaction with valproate.” *Id.* at 27.⁸

Patent Owner argues that Petitioner fails to establish that the skilled artisan would have: (1) been motivated to combine the asserted references; (2) been motivated to administer reduced GHB doses even if the skilled artisan believed that valproate causes negative GHB-related side effects in humans; and (3) reasonably expected that the reduced GHB doses would be effective for treating the claimed sleep disorders. Prelim. Resp. 2. In particular, Patent Owner argues that the prior art would have taught the skilled artisan that the co-administration of GHB and valproate would result in unpredictable effects. *Id.* at 9–21. Patent Owner also argues that the prior art does not disclose or suggest reducing the GHB dose in a patient taking valproate. *Id.* at 21–22. Rather, according to Patent Owner, both the Xyrem 2005 Label and Cagnin teach away from the claimed invention by warning against the co-administration of valproate or other CNS depressants with GHB due to potential side effects. *Id.* at 29–41.

⁸ Petitioner defines the person of ordinary skill in the art (POSA) as someone “hav[ing] at least a Ph.D., Doctor of Pharmacy degree, or medical degree, and five years of experience of treating patients with neurologic disorders, including at least narcolepsy, cataplexy, and excessive daytime sleepiness in narcolepsy,” and further indicates that “a POSA may also include a clinical pharmacologist with at least three years of experience consulting with physicians on the dosing of drugs in light of potential drug-drug interactions, comorbid conditions, or other factors that could affect dosing.” Pet. 2; Ex. 1003, ¶ 20. We apply that description in our analysis.

We determine that Petitioner has not demonstrated a reasonable likelihood of prevailing with respect to any of the challenged claims. Although the language in each of the independent claims differs somewhat, Petitioner has treated each of these claims as requiring the concomitant administration of GHB and valproate, and asserts that the claims would have been obvious for the same reasons. *See* Pet. 28 (“Claim 11 would have been obvious for the same reasons discussed above for claim 1); *id.* at 29 (“Claim 19 would have been obvious for the same reasons discussed above for claim 1 and 11.”); *id.* at 31 (“Claims 30 and 33 would have been obvious for the same reasons as discussed for claims 1, 11, and 19 above.”). We accordingly treat the independent claims together for our analysis.

As an initial matter, Petitioner does not account for the prior art’s teaching away of the co-administration of GHB and valproate. A reference teaches away from a claimed invention if it “criticizes, discredits, or otherwise discourages” modifying the reference to arrive at the claimed invention. *In re Fulton*, 391 F.3d 1195, 1201 (Fed. Cir. 2004); *see also KSR Int’l Co. v. Teleflex Inc.*, 550 U.S. 398, 416 (2007) (“[W]hen the prior art teaches away from combining certain known elements, discovery of a successful means of combining them is more likely to be nonobvious.”). Here, we find it particularly relevant that Petitioner does not address the explicit black box warning that GHB should not be taken with other CNS depressants, despite Petitioner’s acknowledgement that valproate is a CNS depressant. Ex. 1006, 1; Pet. 5. Nor does Petitioner address the Xyrem Label’s contraindication of GHB in patients with SSADH deficiency, despite Petitioner’s acknowledgement that valproate was known to inhibit SSADH activity. Ex. 1006, 8; Pet. 9–10. Likewise, although Petitioner

relies upon Cagnin's suggestion that an interaction between valproate and GHB may have triggered seizures in a single patient who was not part of a clinical trial or other controlled study (Pet. 20), Petitioner fails to address the ultimate conclusion in Cagnin that "[o]nly GHB *discontinuation* restored the neurotransmitter balance and caused seizures to stop." Ex. 1008, 205 (emphasis added). Both the Xyrem 2005 Label and Cagnin therefore both teach away from the co-administration of *any* amount of GHB with valproate.

Moreover, even if the prior art did not teach away from reducing the effective dosage of GHB by at least 5% when valproate is co-administered, Petitioner has not demonstrated sufficiently that a skilled artisan would have had a reasonable expectation of success in treating the claimed sleep disorders with such a reduced dosage of GHB. Petitioner relies upon an "obvious to try" rationale based on prior art studies showing that valproate can decrease GHB metabolism. Pet. 27. We recognize that the prior art may have suggested to a person of ordinary skill in the art that valproate can increase endogenous GHB levels in the brain, and thereby intensify the effect of GHB. *See, e.g.*, Ex. 1014 (Hechler); Ex. 1015 (Kaufman). Patent Owner, however, points to evidence of record showing that GHB is eliminated from the body through alternate pathways that are not inhibited by valproate, and these alternate elimination pathways may actually decrease GHB levels. Prelim. Resp. 9–12. Petitioner does not account for these other pathways by which GHB may be eliminated. Petitioner, therefore, has not identified a sufficient basis on this record to conclude that any increased brain levels of endogenous GHB caused by valproate could have been predictably compensated for by a corresponding decrease of at least 5% in

the amount of GHB orally administered to patients. *See In re Cyclobenzaprine Hydrochloride Extended-Release Capsule Patent Litigation*, 676 F.3d 1063, 1073 (Fed. Cir. 2012) (“[W]here the prior art, at best gives only general guidance as to the particular form of the claimed invention or how to achieve it, relying on an obvious-to-try theory to support an obviousness finding is impermissible.”) (citations omitted).

Because Petitioner has not demonstrated a reasonable likelihood of establishing the obviousness of any of the independent claims, we also determine that Petitioner has not made a sufficient showing with respect to the corresponding dependent claims. *See In re Fine*, 837 F.2d 1071, 1076 (Fed. Cir. 1988) (“Dependent claims are nonobvious under section 103 if the independent claims from which they depend are nonobvious.”).

We, therefore, decline to institute an *inter partes* review based on Petitioner’s first obviousness challenge for any of claims 1–34.

2. Obviousness of Claims 1–34 Based on the Xyrem 2005 Label, Depakote 2011 Label, Cagnin, Waszkielewicz, Weiss, and the FDA Guidance

Petitioner contends that claims 1–34 are obvious based on the combination of the Xyrem 2005 Label, the Depakote 2011 Label, Cagnin, Waszkielewicz, Weiss, and the FDA Guidance. Pet. 41–54. Petitioner also relies upon Dr. Winkelman’s Declaration in support of this challenge. Ex. 1003.

For this challenge, Petitioner additionally relies upon the teachings of Weiss. Petitioner argues, based on Weiss’s warning that the combination of GHB with antiepileptic drugs should be used with “great care,” that the skilled artisan would have been motivated to lower the dose of GHB when

co-administering valproate. Pet. 43–44. Petitioner further argues that, in combination with Cagnin, Weiss would have created a pattern showing that serious adverse events may result from co-administration of GHB and antiepileptic agents such as valproate due to a pharmacokinetic interaction. *Id.* at 43. Based on Weiss’s suggestion that “possible interactions should be evaluated in formal pharmacokinetic studies,” Petitioner asserts that the skilled artisan would have looked to the FDA Guidance, which provides instructions on how to do so. *Id.*

We agree with Patent Owner’s argument that Petitioner’s reliance upon Weiss for this challenge does not cure the deficiencies of the other references. Prelim. Resp. 45–50.

Although Weiss cautions that combinations of GHB with antiepileptic drugs, such as topiramate and valproate, should be used “only with great care,” Weiss does not purport to predict the efficacy or safety of using such a combination, even at reduced dosage amounts, in patients being treated for sleep disorders. Ex. 10101, 1193. Weiss’s statement that “possible [drug-drug] interactions should be evaluated in formal pharmacokinetic studies” is, at best, an invitation to conduct further experiments to evaluate the co-administration of GHB with antiepileptic drugs. We find this insufficient to show a likelihood of obviousness of the claimed invention. *See In re O’Farrell*, 853 F.2d 894, 903 (Fed. Cir. 1988) (obviousness not established where “what was ‘obvious to try’ was to explore a new technology or general approach that seemed to be a promising field of experimentation, where the prior art gave only general guidance as to the particular form of the claimed invention or how to achieve it”).

Weiss, moreover, only discloses discontinuing co-administration of GHB and topiramate, not reducing the GHB dose. Weiss also does not equate the effects of valproate and topiramate, or suggest a class effect that would teach one of ordinary skill in the art to equate the drug-to-drug interactions of valproate and topiramate. Petitioner does not provide sufficient evidence for us to draw the conclusion that the interaction of GHB and topirimate, as reported in Weiss, would suggest reducing effective GHB dosing when concomitantly administering valproate to a patient.

We, therefore, decline to institute an *inter partes* review based on this obviousness challenge.

III. CONCLUSION

For the foregoing reasons, we determine that Petitioner has not demonstrated that the information presented shows that there is a reasonable likelihood that it would prevail in proving the unpatentability of claims 1–34 of the '306 patent.

IV. ORDER

In consideration of the foregoing, it is hereby:

ORDERED that, pursuant to 35 U.S.C. § 314(a), the Petition for *inter partes* review is denied as to all challenged claims of the '306 patent.

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Patent 8,772,306

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