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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

Ex parte CHARLES D. NICHOLS and BANGNING YU [1]

Appeal 2020-001651
Application 15/478,437
Technology Center 1600

Before JEFFREY N. FREDMAN, DEBORAH KATZ, and JOHN G. NEW, *Administrative Patent Judges*.

NEW, *Administrative Patent Judge*.

DECISION ON APPEAL

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[1] We use the word "Appellant" to refer to "applicant" as defined in 37 C.F.R. § 1.42. Appellant identifies Board of Supervisors of Louisiana State University and Agricultural and Mechanical College as the real party-in-interest. App. Br. 3.

SUMMARY

Appellant files this appeal under 35 U.S.C. § 134(a) from the Examiner's Final Rejection of claims 17 and 18 as unpatentable under 35 U.S.C. § 102(b)(pre-AIA) as being anticipated by May et al. (US 6,664,286 B1, December 16, 2003) ("May").[2]

We have jurisdiction under 35 U.S.C. § 6(b).

We AFFIRM.

NATURE OF THE CLAIMED INVENTION

Appellant's claimed invention is directed to a method of treating an inflammatory disorder by administering (R)-1-(2,5-dimethoxy-4-iodophenyl)-2-aminopropane ("(R)-DOI"). Abstr.

REPRESENTATIVE CLAIM

Claim 17 is representative of the claims on appeal and recites:

> 17. A method for the treatment of an inflammatory disorder in a mammal, said method comprising
>
> administering to a mammal in need of such treatment a therapeutically effective amount of (R)-1-(2,5-dimethoxy-4-iodophenyl)-2-aminopropane ((R)-DOI), or a salt thereof,
>
> in a pharmaceutically acceptable carrier.

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[2] The Examiner further rejected claims 17 and 18 as unpatentable under the nonstatutory doctrine of obviousness-type double patenting over claims 1–7 of Nichols et al. (US 9,642,819 B2, May 19, 2007) (the "'819 patent"). Appellant filed a terminal disclaimer that was approved on December 28, 2019. *See* Reply Br. 2. Accordingly, the nonstatutory double patenting rejection is moot and no longer before us on appeal.

App. Br. 7.

ISSUES AND ANALYSIS

We agree with, and expressly adopt, the Examiner's findings, reasoning, and conclusion that the claims are anticipated. We address below the arguments raised by Appellant.

Issue

Appellant argues that May's treatment of glaucoma using (R)-DOI does not necessarily treat an inflammatory disorder and thus May does not inherently anticipate the claims. Reply Br. 4.

Analysis

The Examiner finds that May discloses administering (R)-DOI to treat glaucoma in a person. Ans. 3. The Examiner finds that *Acute Glaucoma Discovered to be an Inflammatory Disease*, UC San Diego Health Newsroom, July 11, 2014, *available at*: https://health.ucsd.edu/news/releases/Pages/2014-07-14-acute-close-angle-glaucoma.aspx (last visited September 18, 2020)("Newsroom") provides extrinsic evidence that glaucoma is an inflammatory disease. *Id.* The Examiner also finds that R. Vohra, et al., *The Role of Inflammation in the Pathogenesis of Glaucoma*, 58 SURVEY OF OPHTHALMOLOGY 311–320 (2013) ("Vohra") provides extrinsic evidence that inflammatory molecules, e.g., tumor necrosis factor-α ("TNF-α"), are up-regulated in glaucoma. *Id.* at 7. Because the extrinsic evidence teaches that glaucoma is an inflammatory response, the Examiner finds that,

when used in the treatment of glaucoma, May's process inherently treats an inflammatory disorder as claimed. *Id.* at 8–9.

Appellant argues that the Examiner misapplies the doctrine of inherency and May does not inherently anticipate the claims. App. Br. 4. Appellant argues that May discloses treating glaucoma generally, and not inflammatory glaucoma specifically. *Id.* Appellant argues that not all types of glaucoma are inflammatory disorders and, therefore, May does not necessarily treat an inflammatory disorder by treating glaucoma. *Id.*

Appellant submits the Declaration of Dr. Angelo Tanna (the "Tanna Declaration") as evidence that treating glaucoma does not necessarily treat an inflammatory disorder. App. Br. 5. Dr. Tanna attests that "[g]laucomas are a group of optic neuropathies" that include both primary and secondary variants of open-angle glaucoma and narrow-angle glaucoma. Tanna Decl. ¶ 4. Dr. Tanna attests that "inflammation is not generally thought to play a role in the etiology of primary open-angle glaucoma" and that primary open-angle glaucoma patients "have elevated intraocular pressure without showing signs of inflammation." *Id.* ¶ 5 (emphasis omitted). In contrast, Dr. Tanna attests that the disclosures of Newsroom[3] are limited to a mouse model of acute-intraocular pressure that is "not representative of the vast majority of cases of human glaucoma." *Id.* ¶ 6 (emphasis omitted). Dr. Tanna attests that "severe IOP elevation, as is observed in the setting of the

[3] Dr. Tanna's Declaration refers to the full article cited by Newsroom: W. Chi et al., *Caspase-8 promotes NLRP1/NLRP3 inflammasome activation and IL-1β production in acute glaucoma*, 111 PNAS 11181–11186 (2014) (submitted September 26, 2018).

rare condition known as acute primary angle closure, is associated with clinical signs of inflammation and breakdown of the blood-aqueous barrier." *Id.* Dr. Tanna concludes by stating that "the inflammatory mechanism described by Chi does not necessarily apply to *any* human glaucoma, much less to *every* type of primary and secondary glaucoma discussed above." *Id.* ¶ 7.

Appellant further argues that Vohra refers to a "possible role of low-grade inflammation as a causal factor in the pathogenesis of glaucoma." App. Br. 6 (emphasis omitted). Appellant argues that Vohra's proposed inflammatory signaling is limited to specific types of glaucoma, and does not establish that all types of glaucoma relate to inflammatory disease. *Id.*

We are not persuaded by Appellant's argument and evidence. "[W]hen considering a prior art method, the anticipation doctrine examines the natural and inherent results in that method without regard to the full recognition of those benefits or characteristics within the art field at the time of the prior art disclosure." *Perricone v. Medicis Pharm. Corp*., 432 F.3d 1368, 1378 (Fed. Cir. 2005). Accordingly, we consider whether the claimed method is the natural and inherent result of the prior art.

The claims recite treating an inflammatory disorder by administering a therapeutically effective amount of (R)-DOI. The Specification defines a "therapeutically effective amount" as an amount that "inhibits or reduces the release of proinflammatory compounds to a clinically significant degree." Spec. ¶ 88. The Specification explains that 5-HT_{2A} receptor agonists, e.g., (R)-DOI, "potently inhibits TNF-α-induced inflammation." *Id.* ¶ 16. Accordingly, the claimed method encompasses treating an inflammatory disorder by inhibiting or reducing TNF-α.

May discloses that "serotonergic compounds which possess agonist activity at $5HT_2$ receptors," e.g., (R)-DOI, unexpectedly "lower and control [intraocular pressure ("IOP")] and are useful for treating glaucoma." May col. 2, l. 66–col. 3, l. 2. May does not explain the underlying mechanism of $5HT_2$ agonists, e.g., (R)-DOI, in lowering IOP and treating glaucoma. *See id.* at col. 2, ll. 53–55. However, May expressly discloses a method of treating glaucoma, specifically reducing acute IOP, by administering a pharmaceutically effective amount of (R)-DOI to a mammal in an actual working example. *See id.* at col. 6, ll. 15–45; col. 8, ll. 61–64 (claim 1); col. 9, ll. 3–5 (claim 4). *See In re Montgomery*, 677 F.3d 1375, 1381 (Fed. Cir. 2012) ("[W]e agree with the Board that even if the claim includes an efficacy requirement, efficacy is inherent in carrying out the claim steps.")

Vohra discloses "several studies have shown that TNF-α induces [retinal ganglion cells ("RGC")] death, suggesting that inflammation in response to ischemia may play a crucial role in the development and progression of glaucoma." Vohra 315–316. Vohra discloses that RGC death "has been shown to be ameliorated by inhibition of TNF-α." *Id.* at 316. Vohra summarizes the literature as indicating "that inflammation in response to either ischemia or increased IOP induces enhanced TNF-α levels, thus promoting RGC death through [tumor necrosis factor receptor 1 ("TNFR1")]. *Id.*

Vohra does not describe inflammation as limited to specific types of glaucomas, contrary to Appellant's argument.[4] Rather, Vohra discloses that

[4] Dr. Tanna does not address Vohra's disclosure. *See generally* Tanna Decl.

the characteristic feature of glaucoma, i.e., progressive loss of RGC (Vohra 311), is associated with elevated TNF-α. Vohra 316. Appellant's Specification acknowledges that (R)-DOI is a potent inhibitor of TNF-α. *See supra.* Accordingly, May's method of treating glaucoma with a pharmaceutically effective amount of (R)-DOI naturally and inherently inhibits or reduces TNF-α, thereby ameliorating RGC death. Because May "discloses the very same methods as claimed," we find that "the particular benefits must naturally flow from those methods even if not recognized as benefits at the time of [May's] disclosure." *Perricone*, 432 F.3d at 1378. Consequently, we sustain the Examiner's rejection based on inherent anticipation.

CONCLUSION

The rejection of claims 17 and 18 as unpatentable under 35 U.S.C. § 102(b), is affirmed.

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a)(1).

AFFIRMED

Claims Rejected	35 U.S.C. §	Reference(s)/Basis	Affirmed	Reversed
17, 18	102(b)	May	17, 18	