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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

Ex parte JOHN BONDO HANSEN and MIKAEL S. THOMSEN¹

Appeal 2019-003393
Application 14/937,286
Technology Center 1600

Before TONI R. SCHEINER, ERIC B. GRIMES, and
RACHEL H. TOWNSEND, *Administrative Patent Judges*.

GRIMES, *Administrative Patent Judge*.

DECISION ON APPEAL

This is an appeal under 35 U.S.C. § 134 involving claims to a method of treating certain movement disorders, which have been rejected as obvious, nonenabled, and lacking adequate written description. We have jurisdiction under 35 U.S.C. § 6(b).

We reverse.

STATEMENT OF THE CASE

“Movement disorders are a group of diseases that affect the ability to produce and control body movement, and are often associated with

¹ Appellants identify the Real Party in Interest as Contera Pharma ApS. Appeal Br. 1.

neurological disorders.” Spec. ¶ 3. “Movement disorders are frequently caused by impaired regulation of dopamine neurotransmission.” *Id.* ¶ 11. “Dopamine release and re-uptake is regulated by a number of neurotransmitters, including serotonin (5-HT).” *Id.* ¶ 13. “Serotonin acts by binding to different serotonergic receptors. These include . . . 5-HT1A, 5-HT1B, 5-HT1D, 5-HT1E, 5-HT1F,” etc. *Id.* ¶ 14.

“The present invention relates to use of 5-HT1 agonists . . . in methods for treatment of movement disorders related to neurological dysfunctions.” *Id.* ¶ 2. Buspirone is a 5-HT1A agonist. *Id.* ¶ 30. “[A] subgroup of 5-HT1B/5-HT1D receptor agonists are collectively called ‘the triptans’. The triptans have been developed as medication for treatment of migraine. . . . These compounds include sumatriptan, zolmitriptan,” etc. *Id.* ¶ 81.

Claims 54, 56, and 60 are on appeal. Claim 54 is the only independent claim and reads as follows:

54. A method for treatment or alleviation of a movement disorder selected from the group consisting of: ataxia, dystonia, Huntington’s disease, Rett syndrome, Tourette syndrome, Wilson’s disease, Machado-Joseph disease, restless leg syndrome and spasmodic torticollis, comprising:

one or more steps of administration of a synergistically effective amount of a pharmaceutical composition comprising zolmitriptan or a pharmaceutically acceptable salt thereof, and

one or more steps of administration of a synergistically effective amount of buspirone or a pharmaceutically acceptable salt thereof, to an individual in need thereof.

The claims stand rejected as follows:

Claims 54, 56, and 60 under 35 U.S.C. § 103(a) as obvious based on Green,² Barber,³ Byrne,⁴ and Bonelli⁵ (Ans. 15);

Claims 54, 56, and 60 under 35 U.S.C. § 112, first paragraph, as lacking adequate written description (Ans. 19); and

Claims 54, 56, and 60 under 35 U.S.C. § 112, first paragraph, as nonenabled (Ans. 22).

I

The Examiner has rejected claims 54, 56, and 60 as obvious based on Green, Barber, Byrne, and Bonelli.⁶ The Examiner finds that “Green relates to . . . a method of treating extrapyramidal motor disorders, comprising a 5-HT1A agonist, such as buspirone.” Ans. 16. “Green discloses that 5-HT1 agonists have been used in the treatment of Huntington’s disease.” *Id.* “Example 10 . . . specifically discloses a composition with zolmitriptan as the main ingredient.” *Id.*

The Examiner notes that “Green discloses a number of diseases,” but finds that “Barber is directed to the treatment of neurodegenerative diseases specifically, to include Huntington’s disease specifically,” and discloses

² WO 98/42344, published October 1, 1998.

³ US 2008/0227813 A1, published September 18, 2008.

⁴ Alan Byrne et al., BENEFICIAL EFFECTS OF BUSPIRONE THERAPY IN HUNTINGTON’S DISEASE, 151 *Am. J. of Psychiatry* 1097 (1994).

⁵ Raphael M. Bonelli & Peter Hofmann, A SYSTEMATIC REVIEW OF THE TREATMENT STUDIES IN HUNTINGTON’S DISEASE SINCE 1990, 8 *Expert Opin. Pharmacotherapy* 141–153 (2007).

⁶ The Examiner cites Bonelli only in response to Appellants’ declaratory evidence, not as part of the prima facie case of obviousness. *See* Ans. 17–18.

treatment with a specific compound (I) “in combination with additional agents, which include buspirone and zolmitriptan.” *Id.* The Examiner cites Byrne as evidence that “use of buspirone specifically for the treatment of Huntington’s disease specifically, is also known in the art.” *Id.*

The Examiner concludes that the method of claim 54 would have been obvious based on Green, Barber, and Byrne,

because the art clearly discloses both compounds as beneficial for the treatment of neurodegenerative diseases, to include Huntington’s disease specifically. Based on the disclosure of Green alone, or in combination with Barber and Byrne, the skilled artisan would have been motivated to combine the two agents in order to enhance the therapeutic effect of each drug alone.

Id. at 17.

Appellants argue, among other things, that Green discloses “[z]olmitriptan is . . . mentioned in an example separately from buspirone. No teaching or suggestion of a combination of buspirone and zolmitriptan is found in the Green reference.” Appeal Br. 2.

Appellants also argue that Barber does not disclose treatment with its compound (I), combined with both buspirone and zolmitriptan, because “the Barber reference . . . merely provides a ‘laundry list’ of more than 230 compounds. Appellant submits that Barber merely lists ‘buspirone’ and ‘zolmitriptan’ as examples of suitable anxiolytic agents and neurotransmitter agonists, respectively.” *Id.* Appellants argue that “no teaching or suggestion of a combination of buspirone and zolmitriptan is found in the Barber reference.” *Id.* at 4.

Appellants argue that “Byrne only states that buspirone could have an effect on aggression and irritability associated with Huntington’s disease. Byrne makes no reference to effects on abnormal movements.” *Id.* at 4.

Appellant submits that it is incorrect that “[bas]ed on the disclosure of Green alone, or in combination with Barber and Byrne, the skilled artisan would have been motivated to combine the two agents in order to enhance the therapeutic effect of each drug alone.” There simply is no support at all for this assertion and Appellant submits that no prima facie case of obviousness is established.

Id. (quoting Office Action mailed July 2, 2018, alteration in original).

We agree with Appellants that the cited references do not provide “a reason that would have prompted a person of ordinary skill in the relevant field to combine the elements in the way the claimed new invention does.” *KSR Int’l Co. v. Teleflex Inc.*, 550 U.S. 398, 418–19 (2007).

Claim 54 defines a method of treating one of nine specific movement disorders by administering a synergistically effective amount of zolmitriptan and buspirone to an individual in need thereof. Thus, claim 54 requires treating a movement disorder, which is one of “a group of diseases that affect the ability to produce and control body movement, and are often associated with neurological disorders.” Spec. ¶ 3. Claim 54 specifies which neurological disorders affect the treated individual.

As a whole, therefore, claim 54 requires treating an individual whose ability to produce and control body movement has been affected by one of nine specific conditions, with amounts of both zolmitriptan and buspirone that are effective to treat or ameliorate the body movement effects. Claim 54 also requires that zolmitriptan and buspirone are administered in

“synergistically effective” amounts; i.e., amounts that in combination produce an effect that is greater than the additive effect of each drug alone.

Green discloses

a pharmaceutical composition . . . and the use of such a composition for the treatment of anxiety, depression, attention deficit disorder and/or panic disorders, sleep apnoea and/or related respiratory disorders and/or substance addiction, especially alcohol abuse, the treatment and/or prophylaxis of incontinence disorders, inducing immunosuppression and/or treating immune disorders, the alleviation of extrapyramidal motor disorders and/or as a memory enhancer.

Green 1:4–13. More specifically, Green discloses “a pharmaceutical composition for oral administration comprising a carrier and, as an active ingredient, a 5-HT₁ agonist, characterised in that the composition is formulated to reduce pre-systemic metabolism of the 5-HT₁ agonist.” *Id.* at 5:21–25.

Green discloses that “buspirone is a 5-HT₁ agonist.” *Id.* at 1:18–19. “Buspirone is an example from a class of compounds known as the azapirones which have been shown to be effective in the treatment of anxiety.” *Id.* at 2:23–26. Green lists other azapirones. *Id.* at 2:25 to 3:9. “All these compounds act as agonists at 5-HT₁ receptors.” *Id.* at 3:10–11. Green also lists other 5-HT₁ agonists, including zolmitriptan. *Id.* at 3:13 to 5:13; in particular 5:3.

Green discloses the use of its compositions “in the treatment of anxiety” (*id.* at 14:24–25); “in the treatment of depression, attention deficit disorder and panic disorders and as memory enhancers” (*id.* at 14:30–32); “in the treatment and/or prophylaxis of incontinence disorders” (*id.* at 14:33–34); “for inducing immunosuppression and/or treating immune

disorders” (*id.* at 14:37 to 15:1); “in the treatment of sleep apnoea” (*id.* at 15:9–10); and “in the treatment of substance addiction” (*id.* at 15:15).

As relevant specifically to the instant claims, Green discloses that “[a]zapirones, particularly buspirone, are also useful for the alleviation of extrapyramidal motor disorders and can therefore be used to treat such conditions as Parkinson’s disease, neuroleptic-induced extrapyramidal symptoms and tardive dyskinesia.” *Id.* at 15:35 to 16:2. Green discloses that,

[i]n addition to the above conditions, 5-HT₁ agonists have also been used in the treatment of social phobia, obsessive-compulsive disorder, migraine, cerebellar ataxia, levodopa-induced dyskinesias, Huntington’s disease, central and peripheral neurodegenerative disorders, emesis, hypertension, hayfever, asthma and pruritis and to assist smokers in giving up smoking.

Id. at 16:3–10.

Green provides working examples of dosage forms comprising either buspirone or zolmitriptan, but not both. *Id.* at 16:28 to 18:13, 21:1–27 (buspirone); 23:14–25 (zolmitriptan).

Barber discloses methods of treating diseases associated with neurodegeneration in the central nervous system using its compound (I) “alone or in combination with at least one additional therapeutic agent.” Barber ¶ 25. The diseases include ALS, Huntington’s disease, Parkinson’s disease (PD), stroke, and cystic fibrosis. *Id.* ¶ 27. The additional therapeutic agents include anxiolytic agents and neurotransmitter agonists, among others. *Id.* ¶ 81. “Suitable anxiolytic agents include . . . buspirone.” *Id.* ¶ 82. “Suitable neurotransmitter agonists include . . . zolmitriptan.” *Id.*

Byrne “report[s] the successful use of buspirone in the treatment of two aggressive patients with Huntington’s disease.” Byrne 1097. Byrne

concludes that buspirone has a potential role “in the management of aggression and irritability in Huntington’s disease.” *Id.* Byrne does not disclose any effect of buspirone in treating a movement disorder.

We conclude that the above disclosures would not have directed a skilled artisan to the method of claim 54 without the benefit of hindsight gleaned from Appellants’ Specification. Green discloses treating many different disorders, ranging from psychiatric conditions like anxiety, depression, and addiction, to incontinence, immune disorders, and extrapyramidal motor disorders. Green’s only disclosure of treating a condition listed in claim 54 is within a list of thirteen conditions, and even then Green does not specify that its method treats a *movement disorder* associated with Huntington’s disease. *See* Green 16:3–10.

In addition, while Green exemplifies dosage forms comprising either buspirone or zolmitriptan, it does not exemplify or specifically suggest combining either of those active agents with other 5-HT₁ agonists, nor does it suggest combining them with each other.

Barber discloses treating diseases associated with neurodegeneration, rather than movement disorders. Barber’s invention is treatment with its Compound (I), which can be combined with other therapeutic agents within a variety of different classes. Those classes include anxiolytic agents, of which buspirone is one example, and neurotransmitter agonists, of which zolmitriptan is one example, but Barber does not suggest combining both an anxiolytic agent and a neurotransmitter agonist, much less buspirone and zolmitriptan specifically, with its Compound (I) to treat a movement disorder.

Byrne, for its part, discloses treatment of aggression, not a movement disorder, with buspirone alone, and does not suggest combining buspirone with another agent.

“Obviousness requires more than a mere showing that the prior art includes separate references covering each separate limitation in a claim under examination.” *Unigene Labs., Inc. v. Apotex, Inc.*, 655 F.3d 1352, 1360 (Fed. Cir. 2011). “Rather, obviousness requires the additional showing that a person of ordinary skill at the time of the invention would have selected and combined those prior art elements in the normal course of research and development to yield the claimed invention.” *Id.*

In our view, the cited references do not show that a person of ordinary skill in the art would have selected buspirone and zolmitriptan in the normal course of research and development of a treatment for a movement disorder associated with Huntington’s disease (or any of the other disorders listed in claim 54), and therefore the references do not support a prima facie case of obviousness. We reverse the rejection of claim 54, and dependent claims 56 and 60, under 35 U.S.C. § 103(a).

II

The Examiner has rejected claims 54, 56, and 60 on the basis that they lack adequate written description in the Specification. The Examiner reasons that, “[t]o provide adequate written description and evidence of possession of a claimed genus, the specification must provide sufficient distinguishing characteristics of the genus,” which “may be achieved by means of a recitation of a representative number of species falling within the scope of the genus or of a recitation of structural features common to the members of the genus.” Ans. 20–21.

The Examiner finds that the Specification

provides no description of the broadly claimed method of treatment of the various movement disorders claimed. To the extent that there is any description of treating, it is for treating Parkinson's disease, LID, akinesia and tardive dyskinesia. There is nothing in the specification to provide a link that treating Parkinson's disease, LID, akinesia and tardive dyskinesia with the claimed combination can also treat vastly different movement disorders as the ones currently claimed.

Id. at 21. The Examiner concludes that “[o]ne of skill in the art would not recognize from the disclosure that the applicant was in possession of the genus.” *Id.*

Appellants argue that “the claims list ataxia, dystonia, Huntington's disease, Rett syndrome, Tourette syndrome, Wilson's disease, Machado-Joseph disease, restless leg syndrome and spasmodic torticollis, all of which are well-known to be ‘movement disorders’ and to be associated with ‘altered dopamine levels.’” Appeal Br. 8–9. Appellants argue that the Examiner's concern about Parkinson's disease versus Huntington's disease is misplaced. *Id.* at 9–10. Appellants cite portions of the Specification to show that “modulators of serotonin receptors can affect dopamine neurotransmitter activity in a complex manner.” *Id.* at 11. Appellants conclude that

one of skill in the art, having reviewed the present specification, would recognize that treatment of the movement disorders listed in claim 1 [sic, 54] would be beneficial and thus, a person skilled in the art at the time the application was filed would have recognized that the inventor was in possession of the invention as claimed.

Id. at 12.

“[T]he examiner bears the initial burden, on review of the prior art or on any other ground, of presenting a prima facie case of unpatentability.” *In re Oetiker*, 977 F.2d 1443, 1445 (Fed. Cir. 1992). “Insofar as the written description requirement is concerned, that burden is discharged by ‘presenting evidence or reasons why persons skilled in the art would not recognize in the disclosure a description of the invention defined by the claims.’” *In re Alton*, 76 F.3d 1168, 1175 (Fed. Cir. 1996) (citation omitted). “If . . . the specification contains a description of the claimed invention, albeit not *in ipsius verbis* (in the identical words), then the examiner . . . must provide reasons why one of ordinary skill in the art would not consider the description sufficient.” *Id.*

Here, the Specification describes the “use of 5-HT1 agonists for the treatment of movement disorders. The combined activation of different serotonergic receptors can lead to a synergic effect which more effectively influences the dopamine levels in the synapse and lead to efficacious treatment of the movement disorders described herein.” Spec. ¶ 22. The Specification exemplifies the combination of zolmitriptan and buspirone to treat a rat model of a movement disorder. *Id.* ¶¶ 239–246. And the Specification states that

[m]ovement disorders according to the present invention may be selected from the group of disorders comprising *ataxia*, *akathisia*, *dystonia*, essential tremor, *Huntington’s disease*, *myoclonus*, *Parkinson’s disease*, *Rett syndrome*, *tardive dyskinesia*, *Tourette syndrome*, *Wilson’s disease*, *dyskinesia*, *chorea*, *Machado-Joseph disease*, *restless leg syndrome*, *spasmodic torticollis*, *geniospasm*, or movement disorders associated therewith.

Id. ¶ 103 (emphases added).

Thus, the Specification describes the invention as a method of treating movement disorders, specifically lists the disorders recited in claim 54 as among the disorders that can be treated, and exemplifies the combination of the two drugs recited in claim 54 in treating a rat model of a movement disorder. We agree with Appellants that the Specification describes the claimed method adequately to demonstrate possession to a person of ordinary skill in the art.

With respect to the Examiner's concern about describing a genus, we note that claim 54 recites treatment of specific disorders with specific compounds. Because claim 54 does not recite a genus, of either disorders or compounds, no genus needs to be described in order to describe the claimed method.

We reverse the rejection of claims 54, 56, and 60 under 35 U.S.C. § 112, first paragraph, based on lack of adequate written description.

III

The Examiner has rejected claims 54, 56, and 60 as nonenabled, on the basis that, "although the specification is enabling for treating of Parkinson's disease, LID, akinesia and tardive dyskinesia, it is not broadly enabling for treating the broad number of other movement disorders claimed." Ans. 22.

The Examiner cites Bonelli as evidence of "the state of the prior art as it relates to movements disorders as currently claimed, to take Huntington's disease as one non-limiting example." *Id.* at 24. The Examiner finds that Bonelli discloses that one class of drugs used to treat Huntington's disease is antidopaminergics, and "this strategy of using antidopaminergics is the exact opposite of what needs to be achieved with respect to Parkinson's disease

and the related diseases- i.e. enhancement of dopamine levels or activity, for which Applicant has support in the specification.” *Id.* at 25.

The Examiner cites Kandel & Schwartz⁷ as evidence that, although “Parkinson’s disease and Huntington’s disease are both movement disorders, and . . . dopamine has been implicated in both, [that] in no way makes them akin in pathology, and treatment means.” *Id.* at 26. That is, “Parkinson’s disease is a *hypokinetic disorder* characterized by *akinesia* and *bradykinesia*, whereas Huntington’s disease, on the other end of the spectrum, is [a] *hyperkinetic disorder*, characterized by *dyskinesia* and *hypotonia*.” *Id.* The Examiner finds that Kandel & Schwartz also discloses that “the circuitry involved in both [Parkinson’s disease and Huntington’s disease] in the basal ganglia is balanced in a completely different manner,” with “frequently completely opposite activity of increased v. decreased signaling in Parkinson’s disease, as compared to Huntington’s chorea,” relative to a normal brain. *Id.*

The Examiner finds that “the only Examples in Applicant’s specification pertain to testing in only Parkinson’s disease, LID, akinesia and tardive dyskinesia.” *Id.* at 29. The Examiner also finds that “a broad search of the art indicates that there is no silver bullet for any and every movement disorder across the spectrum,” with “different treatment methods from one movement disorder to another,” and “there is no existing prevention regimen for movement disorders.” *Id.*

⁷ Mahlon R. DeLong, “The Basal Ganglia,” in PRINCIPLES OF NEURAL SCIENCE, 4th Edition, Kandel et al. (eds.), McGraw-Hill Health Profs. Div., New York (2000), pp. 853–867.

The Examiner concludes that, even though the “skill of one in the art is expected to be high,” “the specification does not provide support for treatment of all movement disorders as currently claimed, with the claimed combination of buspirone and zolmitriptan” and “[o]ne of ordinary skill in the art would be burdened with undue experimentation to practice the invention commensurate in the scope of the claims.” *Id.* at 30.

Appellants argue that, “as noted in the specification, the listed disorders are all movement disorders and are all associated with altered synaptic dopamine levels, commonalities shared with L-DOPA induced dyskinesia.” Appeal Br. 5.⁸ With respect to the Examiner’s statement that Parkinson’s disease and Huntington’s disease are treated with opposite strategies, Appellants argue that “the idea that Parkinson’s disease is treated directly with dopamine receptor agonists only and that Huntington’s disease is treated directly only with dopamine receptor antagonists is inappropriately reductive.” *Id.* at 6. Appellants point to the discussion in the Specification of treating different movement disorders with serotonin receptor modulators and conclude that “the specification indicates that modulators of serotonin receptors can affect dopamine neurotransmitter activity in a complex manner and that effects on both increased and decreased dopamine neurotransmitter activity are possible.” *Id.* at 6–8.

⁸ Appellants’ arguments regarding the nonenablement rejection refer to “the remarks above.” Appeal Br. 12. Appellants, earlier in the brief, argued that evidence presented in the Specification and in two declarations demonstrated unexpected results with respect to the claimed invention. *Id.* at 4–8. Accordingly, we have considered those arguments and that evidence as they pertain to the issue of enablement.

“[T]o be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without ‘undue experimentation.’” *In re Wright*, 999 F.2d 1557, 1561 (Fed. Cir. 1993).

When rejecting a claim under the enablement requirement of section 112, the PTO bears an initial burden of setting forth a reasonable explanation as to why it believes that the scope of protection provided by that claim is not adequately enabled by the description of the invention provided in the specification of the application; this includes, of course, providing sufficient reasons for doubting any assertions in the specification as to the scope of enablement.

Id. at 1561–62.

In this case, the Examiner has not established that the Specification does not enable practicing the claimed method without undue experimentation. The Specification describes the invention as “relat[ing] to use of 5-HT1 agonists . . . in methods for treatment of movement disorders related to neurological dysfunctions.” Spec. ¶ 2. The Specification states that one “example of a movement disorder is dyskinesia which [is] characterized by various involuntary movements, which can affect discrete body parts or can become generalized and severely disabling. Tardive dyskinesia is one example of dyskinesia.” *Id.* ¶ 4.

The Specification also states that the treatment of PD [Parkinson’s disease] with L-DOPA often gives rise to dyskinesia (diminished voluntary movements and presence of involuntary movements) in advanced PD patients with impaired regulations of [dopamine] levels. This specific type of dyskinesia is called L-DOPA Induced Dyskinesia (LID) and is caused by excessive dopamine levels in the synapses.

Id. ¶ 7.

The Specification provides working examples of “the evaluation of zolmitriptan and buspirone in the 6-OHDA rat model.” *Id.* ¶ 239. “The results . . . showed that buspirone (1 mg/kg/day i.p.) in combination with zolmitriptan (3 mg/kg/day i.p. or 10 mg/kg/day) significantly reduced L-DOPA-induced dyskinesia.” *Id.* ¶ 246.

The Examiner concludes that the Specification is enabling for treatment of LID, among other movement disorders. Ans. 22. LID is a “dyskinesia . . . caused by excessive dopamine levels in the synapses.” Spec. ¶ 7. The movement disorder caused by Huntington’s disease (a.k.a. Huntington’s chorea⁹) is also a dyskinesia. Ans. 26 (citing Kandel & Schwartz at 861).

The Examiner also cites the disclosure in Kandel & Schwartz that: “Drug-induced dyskinesias [e.g., LID], which closely resemble chorea, are a side effect of dopamine replacement therapy for Parkinson disease. The pathophysiology of those pharmacologically induced dyskinesias may be in part similar to that of chorea in Huntington disease.” *Id.* at 28 (quoting Kandel & Schwartz at 865).

Thus, both LID and the movement disorder of Huntington’s disease are dyskinesias; i.e., movement disorders characterized by abnormal involuntary movement. Spec. ¶¶ 4, 7; Ans. 26, 28. LID “is caused by excessive dopamine levels in the synapses.” Spec. ¶ 7. The movement disorder of Huntington’s disease has been treated with, among other things, “dopamine blocking, or dopamine depleting” agents. Ans. 29. Thus, the

⁹ See Ans. 35 (“(Huntington’s) chorea (i.e. Huntington’s disease)”).

movement disorder of Huntington's disease is also characterized by excessive dopamine.

The Specification's working examples use a rat model of LID, and the Examiner has concluded that the Specification is enabling for treatment of both Parkinson's disease and LID, among other movement disorders. The Examiner concludes that the Specification does *not* enable treating the movement disorder of Huntington's disease because the underlying causes of Parkinson's disease and Huntington's disease differ. However, those differences also apply to LID; both LID and the movement disorder of Huntington's disease are caused by excessive dopamine.

In our view, the Examiner has not persuasively explained why the Specification's evidence showing treatment of an animal model of LID enables treatment of Parkinson's disease, but does not enable treatment of the movement disorder of Huntington's disease. The Examiner cites Kandel & Schwartz as evidence that Parkinson's disease and Huntington's disease ("chorea") have different effects on "the circuitry involved in . . . the basal ganglia." Ans. 26. However, the reproduced text and figure do not adequately explain why the Specification's results using an animal model of LID—a movement disorder caused by excessive dopamine—would not be enabling for Huntington's disease.

In summary, the Examiner has not presented sufficient evidence or technical reasoning to support a conclusion that the Specification is not enabling for treatment of Huntington's disease. The Examiner does not specifically address any of the other movement disorders recited in claim 54. We therefore reverse the rejection of claims 54, 56, and 60 under 35 U.S.C. § 112, first paragraph, for lack of enablement.

Appeal 2019-003393
Application 14/937,286

SUMMARY

We reverse all of the rejections on appeal.

REVERSED