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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
12/129,935	05/30/2008	Becker Hewes	PC69808A	3004

25291 7590 10/27/2020

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ART UNIT	PAPER NUMBER
1627	

NOTIFICATION DATE	DELIVERY MODE
10/27/2020	ELECTRONIC

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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

Ex parte BECKER HEWES

Appeal 2019-006474
Application 12/129,935
Technology Center 1600

Before DONALD E. ADAMS, ERIC B. GRIMES, and TIMOTHY G. MAJORS, *Administrative Patent Judges*.

MAJORS, *Administrative Patent Judge.*

DECISION ON APPEAL

Appellant[1] submits this appeal[2] under 35 U.S.C. § 134(a) involving claims to a method for treating a BcrAbl positive leukemia in a subject that is resistant to imatinib, which claims have been rejected for obviousness under 35 U.S.C. § 103 and for obviousness-type double patenting. We have jurisdiction under 35 U.S.C. § 6.

We REVERSE.

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[1] We use the word "Appellant" to refer to "applicant" as defined in 37 C.F.R. § 1.42(a). Appellant identifies Wyeth LLC as the real party-in-interest. Appeal Br. 3. We also note that Pfizer Inc., and its Legal Department, is identified as the addressee of record for this application, and Pfizer's counsel appears to be responsible, at least in part, for prosecution of this application. *See, e.g.*, *id.* at 12; Ans. (mailing page).

[2] This appeal is related to Appeal 2014-007550, decided July 22, 2016.

STATEMENT OF THE CASE

Patients with chronic myelogenous leukemia ("CML") are often treated with imatinib, a drug sold under the name *Gleevec* (also known as STI-571). Spec. ¶¶ 2–3. This drug "blocks the tyrosine kinase protein 'BcrAbl,' an abnormal protein driving overproduction of abnormal white blood cells characteristic of leukemia." *Id*. ¶ 2. Some patients, however, develop resistance to imatinib "due to point mutations in the bcr/abl gene." *Id*. ¶ 4.

"[T]he invention is directed to methods of treating imatinib-resistant BcrAbl positive leukemia." *Id.* ¶ 2. According to the Specification, it has "been discovered that a significant number of patients having known point mutations associated with resistance to imatinib respond favorably to treatment with SK-606"—a drug also known as "bosutinib," with the chemical name 4-[(2,4-Dichloro-5-methoxy-phenyl)amino]-6-methoxy-7-[3-(4-methyl-1-piperazinyl)propoxy]-3-quinolinecarbonitrile. *Id*. ¶ 13; *see also id*. ¶ 9. The Specification identifies F317L as such a mutation. *Id.* ¶ 52. The amino acid change of F317L corresponds to mutation of the bcr/abl gene at 949T>C, reflecting the specific nucleotide position and change. *Id.*

Claims 17–28 are on appeal. Appeal Br. 13–14. Claim 17 is illustrative and reads:

> 17. A method for treating a BcrAbl positive leukemia in a subject that is resistant to imatinib which comprises administering to the subject a therapeutically effective amount of 4-[(2,4-Dichloro-5-methoxy-phenyl)amino]-6-methoxy-7-[3-(4-methyl-1-piperazinyl)propoxy]-3-quinolinecarbonitrile, wherein the subject has a mutation in BcrAbl protein selected from F317L.

Id. at 13 (underlining omitted).

Claim 18, the other independent claim on appeal, is substantially identical to claim 17, except the corresponding wherein clause reads: "wherein [the subject[3]] has a resistance-associated nucleic acid mutation in the BcrAbl gene selected from the group consisting of: 949T>C." *Id.*

Appellant seeks review of the following rejections:

I. Claims 17–20, 23, and 24 under 35 U.S.C. § 103 as obvious over Manley;[4]

II. Claims 17–28 under 35 U.S.C. § 103 as obvious over Boschelli '780[5] in view of Shah.[6]

III. Claims 17–28 for obviousness-type double patenting over claims of Boschelli '148[7] in view of Shah.

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[3] This bracketed text reflects what, in context, appears to be the missing language. Claim 23 also depends from "claim 1," which is not pending. Appeal Br. 13. For this appeal, we treat claim 23 as depending from claim 17. If prosecution continues, corrections should be made.

[4] Manley et al., *Advances in the structural biology, design and clinical development of Bcr-Abl kinase inhibitors for the treatment of chronic myeloid leukaemia*, 1754 BIOCHIMICA ET BIOPHYSICA ACTA 3–13 (2005) ("Manley").

[5] Boschelli et al., US 2005/0101780 A1, publ. May 12, 2005 ("Boschelli").

[6] Shah et al., *Multiple BCR-ABL kinase domain mutations confer polyclonal resistance to the tyrosine kinase inhibitor imatinib (STI571) in chronic phase and blast crisis chronic myeloid leukemia*, 2 CANCER CELL 117–125 (2002) ("Shah").

[7] Boschelli et al., US 7,417,148 B2, issued Aug. 26, 2008 ("Boschelli '148"). Boschelli '148 issued from the application that published as Boschelli '780. Boschelli '148, code (65). The Examiner indicates that the double patenting rejection is over claims 1–10, 21, and 22 of Boschelli '148. Final Act. 3–4. There are, however, no claims numbered 21 or 22 in that patent. Boschelli '148, 18:28–31 (ending at claim 12).

I. *Obviousness Over Manley*

The issue on appeal is whether a preponderance of the evidence supports the Examiner's conclusion that claims 17–20, 23, and 24 would have been obvious over Manley. The Examiner's and Appellant's positions are addressed to the claims as a group. Appeal Br. 4–8; Final Act. 4–6. We focus on claim 17 as illustrative, which claim relates to the treatment of imatinib-resistant leukemia in a subject having a "mutation in BcrAbl protein selected from *F317L*." Appeal Br. 13 (emphasis added).[8] That treatment involves administering a therapeutically effective amount of a drug known as bosutinib (with the chemical name recited in claim 17). *Id.*; Spec. ¶ 9 (describing bosutinib (also known as SKI-606)). The Examiner's determinations and Appellant's arguments are summarized below, followed by our analysis.

The Examiner finds that claim 17 would have been obvious over Manley. Final Act. 4–10. According to the Examiner, Manley teaches "a promising class of new chronic myeloid leukaemia (CML) drugs for patients with imatinib resistance." *Id.* at 5. Also, the Examiner finds, Manley teaches "[t]his resistance is often due to the emergence of clones expressing mutant forms of Bcr-Abl, which exhibit decreased sensitivity towards inhibition by imatinib." *Id.* (citing Manley's Abstract and Introduction). The Examiner interprets Manley as teaching that "[t]he most frequently occurring mutants are F317V>T315A>T315I>F317L." *Id.* (citing Manley 8

[8] As noted above, claim 18 recites the mutation, not as the expressed mutant protein F317L, but based on the corresponding nucleic acid mutation (949T>C). Appeal Br. 13. The other challenged claims depend, directly or indirectly, from either claim 17 or claim 18. *Id.* at 13–14.

(left col., first para.)). Further, the Examiner finds, Manley teaches "[o]ne promising class of CML drugs is the dual Bcr-Abl/Src inhibitor, SKI-606 [i.e., bosutinib]." *Id.* (citing Manley 9).

The Examiner finds that Manley "fail[s] to disclose a single embodiment regarding administration of SKI-606 to a patient suffering from imatinib resistant BCrAbl positive leukemia, which has one of the claimed mutations," but the Examiner nevertheless concludes the method of claim 17 would have been obvious over Manley. *Id.* More specifically, the Examiner determines "it would have been prima facie obvious . . . to administer the dual Bcr-Abl/Src inhibitor, SKI-606, to a patient suffering from imatinib resistant BcrAbl positive leukemia having one of the following mutations, F317V>T315A>T315I>F317L"—the F317L mutation being the one recited in claim 17. *Id.* The Examiner reasons that the ordinarily skilled person would have been motivated to practice the claimed method based on Manley's teaching that bosutinib is one of a class of promising new inhibitor compounds for treating imatinib-resistant CML, that imatinib resistance is due to emergence of clones expressing mutant proteins, and that F317L is alleged to be among the "most frequently occurring mutants." *Id.* at 5–6. Invoking this same reasoning, the Examiner concludes "[t]herefore, one of ordinary skill in the art would have had a reasonable expectation of success in treating imatinib resistant BcrAbl positive leukemia" by administering bosutinib to a subject with the F317L mutation. *Id.*; *see also* Ans. 4–13.

Appellant raises five responsive arguments. Appeal Br. 5–8. In support, Appellant relies on, among other evidence, testimony from Brion W. Murray, Ph.D., submitted in an affidavit dated October 3, 2016 ("Murray Affidavit"). *Id.*; Murray Affidavit pp. 1–6.

First, Appellant argues "Manley does not suggest that any promising new CML drug, including bosutinib, could treat imatinib-resistant CML associated with F317L." *Id.* at 5. Indeed, Appellant contends, "none of the second generation drugs are described in Manley as effectively treating imatinib-resistant CML associated with F317L." *Id.* (citing Manley's teaching that "[o]f the patients expressing imatinib-resistant mutants, complete haematological remissions have been observed in most cases [treated with another second generation drug, dasatinib], *with the exception of those expressing* T315I . . . *F317L* and D276G." *Id.* (citing Manley 8 (with Appellant's emphasis)).

Appellant's second, third, and fourth arguments relate, in general, to alleged unpredictability in treatment of imatinib-resistant leukemia in subjects with the F317L mutation. *Id.* at 5–7. According to Appellant, subjects expressing mutant proteins with mutations in the Bcr-Abl protein's hinge region, like the F317L and T315I mutants, had been shown to be especially resistant to treatment, even with second generation drugs like dasatinib and nilotinib. *Id.* (citing, among other evidence, Murray Affidavit ¶¶ 8, 9, 11); *see* Murray Affidavit ¶ 11 (testifying that "mutations in the ABL hinge region (T315I, F317L) were shown to confer resistance to both imatinib and dasatinib in clinical models . . . as well as in patients" (citing several references)). Appellant further contends, citing Dr. Murray's testimony and other references, that imatinib resistance arising from mutations of the Bcr-Abl gene was, in fact, poorly understood at the time of the invention. Appeal Br. 6; Murray Affidavit ¶ 12 (explaining that different patients, even with the same mutations, were known to display different clinical responses upon treatment). And, Appellant argues, preclinical

models in this area were poor predictors of clinical efficacy and success. Appeal Br. 6; Murray Affidavit ¶ 13 (testifying that preclinical models may have predicted drug potency even against F317L mutants, but "clinical findings show F317L to be a[n] imatinib/dasatinib-resistant mutation").

Finally, Appellant contends that the F317L is actually a rare mutation, which would have detracted from the motivation of treating subjects with that mutation. Appeal Br. 7 ("[T]he F317L mutation is uncommon [and thus] there would be a diminished motivation to seek a treatment."). Appellant cites evidence that, in one study, "only one of the 20" imatinib-resistant subjects had a F317L point mutation. *Id.* (citing Manley Table 1). Similarly, citing Shah, Appellant contends that in "mutation data for 37 patients having 51 mutations," evidence shows "only three of the 51 mutations being F317L." *Id.*

The Examiner "bears the initial burden . . . of presenting a *prima facie* case of unpatentability." *In re Oetiker*, 977 F.2d 1443, 1445 (Fed. Cir. 1992). "The Patent Office has the initial duty of supplying the factual basis for its rejection. It may not . . . resort to speculation, unfounded assumptions or hindsight reconstruction to supply deficiencies" in the rejection. *In re Warner*, 379 F.2d 1011, 1017 (CCPA 1967). And, "[a]fter evidence or argument is submitted by the applicant in response, patentability is determined on the totality of the record, by a preponderance of evidence with due consideration to persuasiveness of argument." *In re Oetiker*, 977 F.2d at 1445. Upon considering the prior art, and the argument and evidence of record for and against the rejection, we conclude that the preponderance of the evidence on this record does not support the Examiner's rejection for obviousness. We explain below.

As an initial matter, we agree with the Examiner that Manley discloses bosutinib as among a promising new class of drugs under investigation for the treatment of imatinib-resistant CML. Manley describes a drug known as "BMS-354825" (also known as dasatinib) and "AMN107" (a drug under development by Novartis) and Manley teaches that "both of these promise a breakthrough in the treatment of imatinib-resistant CML." Manley, Abstr., 4 (depicting structures for selective Abl and dual Src-Abl kinase inhibitors, including dasatinib, AMN107, and SKI-606 (bosutinib), among others). On bosutinib, Manley teaches that it, like dasatinib, is a dual Bcr-Abl/Src inhibitor being studied for treatment of CML. *Id.* at 7, 9. Indeed, Manley teaches that, "[i]n imatinib-resistant K-562 cells . . ., SKI-606 [bosutinib] has been shown to inhibit both Bcr-Abl and Lyn phosphorylation," and had shown "enhanced apoptosis in CD34+ cells isolated from blast crisis CML patients, including those harbouring Y253, E255V, E255K or F359V mutants." *Id.* at 9.

We also agree with the Examiner that Manley teaches imatinib-resistance is "often due to the emergence of clones expressing mutant forms of Bcr-Abl." Manley 3–4 (noting that "over 35 such mutant forms of the enzyme have been observed in CML patients," prompting "a need for improved therapies"). Manley identifies many of those mutants. *See, e.g.*, Manley 6 (Table 1). The Examiner, however, misinterprets Manley in finding that "F317V>T315A>T315I>F317L" are the "most frequently occurring mutants" in imatinib-resistant subjects. Final Act. 5 (citing Manley 8). The Examiner's citation to those four identified mutants arises from a section of Manley describing the most frequent mutants emerging in *dasatinib*-resistant clones—not imatinib-resistant subjects. Manley 8 ("For

BMS-356825 . . ., resistant clones emerged expressing 10 mutant forms of Bcr-Abl, with the most frequently occurring mutants being F317V>T315A>T315I>F317L"). The misinterpretation aside, Manley elsewhere identifies F317L in a listing of "some of the most prevalent imatinib-resistant mutant forms of the enzymes identified in patients." *Id.* at 6 (Table 1); Ans. 9 (citing Table 1).[9]

Although we agree with many of the Examiner's findings, and even if the Examiner's reasoning provided some motivation to attempt treating subjects with the F317L mutation with bosutinib, considering the totality of the evidence, the rejection fails on the issue of reasonable expectation of success. *Intelligent Bio-Systems, Inc. v. Illumina Cambridge Ltd.*, 821 F.3d 1359, 1367 (Fed. Cir. 2016) (explaining that the motivation and reasonable expectation inquiries are different, and the latter refers to likelihood of success in modifying the prior art to reach the claimed invention). As discussed in more detail below, Appellant's argument and supporting evidence shows a lack of predictability in treating imatinib-resistant subjects with the F317L mutation, in particular. *See, e.g.*, Appeal Br. 5–7; Murray Affidavit. In the face of this argument and evidence from Appellant, the Examiner provides no persuasive rebuttal. Ans. 9–13.

[9] According to the Examiner, "[i]n Table 1, BMS-354825 [dasatinib] is shown to have activity for not only the four mentioned mutants, F317V>T315A>T315I>F317L, but also other mutant forms of Bcr-Abl." Ans. 9. Table 1 does not, however, show that dasatinib has therapeutic activity for the T315I mutant, and the T315A mutant is not identified in the table. Manley 6–7 ("In cellular assays, . . . the compound [dasatinib] maintains high potency against a wide range of Bcr-Abl mutants . . ., although, again like AMN107, with the exception of T315I (Table 1)").

Appellant is correct that none of the second generation drugs in Manley are disclosed as effectively treating imatinib-resistant CML associated with the F317L mutation. Appeal Br. 5. Insofar as the Examiner suggests that Manley teaches the second generation drug dasatinib (BMS-354825) is "useful for" treating imatinib-resistant CML in subjects with the F317V, T315A, T315I, *and F317L* mutations, we are unpersuaded. Ans. 9 ("The key part of this argument [of Examiner] is that each of the active agents in Manley go through the same mechanism of action, namely inhibiting Bcr-Abl. For example, in the section drawn to the dual Bcr-Abl kinase inhibitor, BMS-354825, only 4 mutant forms are listed as being useful for, F317V>T315A>T315I>F317L."); *see also id.* (arguing that "[i]n the same manner [as BMS-354825], SKI-606 or bosutinib, is expected to have activity towards the mutant forms listed in that section," including the claimed F317L mutation). To the contrary, Appellant persuades us that Manley teaches that those four cited mutants arose most frequently in *dasatinib-resistant subjects*—suggesting, if anything, that dasatinib would *not be useful* for effective treatment in subjects expressing F317L. Manley 8; *see* Murray Affidavit ¶¶ 11, 13.

Although having previously cited Manley's teachings about the perceived favorable activity of dasatinib as supporting a conclusion of obviousness, faced with Appellant's rebuttal argument and evidence including the testimony of Dr. Murray, the Examiner responds that "dasatinib is not bosutinib." Ans. 10. That is, according to the Examiner, "nothing can be taken away from the assertion that other next-generation drugs like dasatinib were shown to be ineffective against F317L." *Id.* But it is the Examiner that bears the ultimate burden of showing unpatentability.

There is no actual teaching in Manley that bosutinib has activity or would be therapeutically effective in an imatinib-resistant subject with the F317L mutation. And Appellant's evidence, especially Dr. Murray's testimony, calls into significant doubt whether the ordinarily skilled person would have reasonably expected success with bosutinib when other second-generation, Bcr-Abl/Src inhibitors were known and shown to be *ineffective* in treating subjects with the F317L mutation. Manley 8; Murray Affidavit ¶ 11.

The Examiner also responds to Appellant's arguments and evidence with an assertion that "none of these lines of argument directly relates to bosutinib . . . nor anything specific to the teachings of the cited references." Final Act. 11; Ans. 10. We find, however, that Dr. Murray's testimony about the lack of success with other second generation Bcr-Abl inhibitors is probative of the skilled person's reasonable expectations (or lack thereof) about successful treatment with bosutinib in subjects with the specific mutation claimed.

The documentary evidence cited by Dr. Murray corroborates his testimony. For example, to support his opinion that effectively treating leukemia in subjects expressing F317L was highly unpredictable, Dr. Murray cites, among other references, a publication describing Phase II clinical results on five patients treated with dasatinib where resistance to treatment and relapse coincided with emergence of the F317L mutation. *See* Murray Affidavit ¶¶ 9, 11 (citing Simona Soverini et al., *Presence or the Emergence of a F317L BCR-ABL Mutation May Be Associated With Resistance to Dasatinib in Philadelphia Chromosome-Positive Leukemia*, 24:33 JOURNAL OF CLINICAL ONCOLOGY e51–52 (2006)). The Examiner fails to address directly or persuasively Dr. Murray's opinions or the

literature he cites in support of those opinions. The Examiner's assertions on the reasonable expectation issue, by comparison, are conclusory and lack a persuasive evidentiary basis. Moreover, the Examiner's assertion (Final Act. 11) that Appellant's argument and Dr. Murray's testimony are not specific to the references cited in the rejection is incorrect. The Murray Affidavit repeatedly cites Manley and Shah (among other references) in support of his opinions; both Manley and Shah are asserted by Examiner here. *See, e.g.*, Murray Affidavit ¶¶ 9, 11, 13.

The Examiner also characterizes Appellant's evidence as "merely opinions" on the state of the art, among other issues. Final Act. 10; Ans. 10–11. We are unpersuaded Dr. Murray's "opinions" can be summarily swept aside in this way. As already explained, Dr. Murray cites repeatedly to the scientific literature in support of his opinions, grounding those opinions in an adequate factual basis (e.g., explaining, with citation support, that treatment of subjects expressing the Bcr-Abl protein with hinge-region mutants (like F317L or T315I) have been described in the literature as difficult to treat). *See, e.g.*, Murray Affidavit ¶ 11.[10] The Examiner does not grapple persuasively with Dr. Murray's opinions or the facts on which they are based.

[10] Regarding the Puttini reference, the Examiner responds "Appellant is reminded that that the Puttini reference was not a reference used in the instant rejections." Ans. 11. As we explained in our prior decision, secondary references like Puttini may be considered for their evidentiary value on issues relevant to the obviousness inquiry even if such references are not applied by the Examiner in a rejection. *In re Hewes*, Appeal 2014-007550, 14 n.7 (PTAB July 22, 2016).

Finally, the Examiner points to the Board's prior decision in the related appeal, where we affirmed the rejection of the then-pending claims for obviousness over the same art asserted by Examiner here. Final Act. 12. But the circumstances and record have changed. The claims have been amended and materially narrowed since the earlier appeal. Claim 17 previously recited a list of twelve mutations,[11] now only one mutation is recited. New evidence is also before the Examiner and the Board, including testimonial evidence that persuades us of an art-recognized difficulty in effectively treating leukemia in subjects with the F317L mutation now recited in claim 17. In our prior decision, we agreed with the Examiner's prima facie case of obviousness, including on the reasonable expectation of success issue, "absent persuasive evidence to the contrary" from Appellant. *In re Hewes*, Appeal 2014-007550, 16 (PTAB July 22, 2016). Appellant has since submitted the sort of persuasive rebuttal argument and evidence that we remarked was missing previously.

For the reasons above, we determine that the preponderance of the evidence does not support the Examiner's conclusion that claim 17 (or claims 18–20, 23, and 24) would have been obvious over Manley.

II. *Obviousness Over Boschelli '780 and Shah*

The Examiner concludes that claims 17–28 would have been obvious over Boschelli '780 and Shah. Final Act. 8–12. This rejection is similar to

[11] As we noted during the prior appeal, some of the evidence/references actually indicated that bosutinib was likely to be efficacious in treating leukemia in subjects with mutations expressly claimed at that time. *In re Hewes*, Appeal 2014-007550, 14 (PTAB July 22, 2016) (pointing out that Puttini disclosed a significant rate of decrease in tumor growth and prolonged survival related to the then-claimed Y253F mutant form).

the rejection over Manley, except the Examiner relies on Boschelli '780's teachings about treating CML with inhibitor compounds, including the Brc-Abl/Src inhibitor compound with the chemical name recited in claim 17. Final Act. 8 (citing Boschelli '780, claims 1–10, 21–22).[12] The Examiner, acknowledging that Boschelli '780 does not disclose treating BcrAbl positive leukemia in an imatinib-resistant subject due to particular mutations, much less the F317L mutation, turns to Shah. *Id.* at 9. The Examiner finds that Shah describes common mutations that arise in subjects with imatinib resistance, including F317L, T315I, M244V, and several others. *Id.* The Examiner describes the art's teachings as a "typical genus/species situation" and concludes it would have been obvious to provide effective CML treatment by administering SKI-606/bosutinib to imatinib-resistant subjects with the F317L mutation. *Id.* at 9–10.

This rejection fails for substantially the same reasons as discussed above. Appellant has presented persuasive evidence showing that treatment of leukemia in resistant subjects with the F317L mutation with second generation Abl-Brc/Src inhibitors (like dasatinib and others) was, for several reasons, known to be especially difficult and unpredictable. Appeal Br. 8–11; Murray Affidavit ¶¶ 9–14. Examiner provides no persuasive rebuttal. On this record, we are unpersuaded the ordinarily skilled person would have had a reasonable expectation of success in practicing the method claimed. Accordingly, the preponderance of the evidence does not support the Examiner's conclusion of obviousness.

[12] Claim 1 of Boschelli '780 recites a method of inhibiting CML comprising administering a class of compounds, and dependent claim 6 recites the compound with the chemical name recited in Appellant's claim 17.

III. *Obviousness-Type Double Patenting Over Boschelli '148 and Shah*

Both Examiner and Appellant treat the double-patenting rejection as rising or falling with the obviousness rejection based on Boschelli '780 and Shah. Final Act. 4 ("[T]his [double-patenting rejection] is the same rejection as the 103(a) rejection below, therefore please see below for the full body of the rejection"); Appeal Br. 12 ("In view of the above, reconsideration and withdrawal of the 35 USC § 103 rejection (and corresponding obviousness-type double patenting rejection) based on Boschelli and Shaw is therefore respectfully requested."). Because we conclude that the preponderance of the evidence does not support the conclusion that claims 17–28 would have been obvious over Boschelli '780 and Shah, the obviousness-type double patenting rejection fails for the same reasons. *See supra* (Sections I and II).

CONCLUSION

In summary:

Claims Rejected	35 U.S.C. §	Reference(s)/Basis	Affirmed	Reversed
17–20, 23, 24	103	Manley		17–20, 23, 24
17–28	103	Boschelli '780, Shah		17–28
17–28	obviousness-type double patenting	Boschelli '148, Shah		17–28
Overall Outcome				17–28

REVERSED