

The opinion in support of the decision being entered today was not written for publication and is not binding precedent of the Board.

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte GABRIEL LOPEZ-BERESTEIN, ANA M. TARI
and RALPH B. ARLINGHAUS

Appeal No. 2005-0740
Application No. 08/679,437

ON BRIEF

Before, ELLIS, SCHEINER and MILLS, Administrative Patent Judges.

ELLIS, Administrative Patent Judge.

DECISION ON APPEAL

This is an appeal pursuant to 35 U.S.C. § 134 from the examiner's final rejection of claims 1-4 and 7-28, all the claims pending in the application.¹

¹ The oral hearing scheduled for May 5, 2005, was waived by the appellants.

We note the appellants' statement on page 4 of the Supplemental Brief (hereinafter, Brief) that the claims do not stand or fall together. In this regard, with respect to Rejection I, the enablement rejection, we find that the appellant argues that there are two (2) groups of claims- Group I consisting of claim 8, and Group II consisting of claims 17-28. Brief, pp. 10-14. Accordingly, we will consider the issues as they apply to claims 8 and 17, which are representative of the subject matter in each group. With respect to Rejection II, the appellants appear to argue that there are three (3) groups of claims- Group III consisting of claims 1, 3, 7, 9, 11, 13 and 15; Group IV consisting of claims 2, 4, 10, 12 and 16; and Group V consisting of claims 13, 14 and 17-20. *Id.*, p. 6, para. 1. Accordingly, we will consider the issues pertaining to the obviousness as they apply to representative claims 1, 2 and 13.

Claims 1, 2, 8, 13 and 17 read as follows:

1. A composition comprising a polynucleotide that hybridizes to the translation initiation site of a Grb2-encoding polynucleotide, wherein said polynucleotide inhibits the expression of Grb2.
2. A composition comprising a polynucleotide that hybridizes to the translation initiation site of a Crk1-encoding polynucleotide, wherein said polynucleotide inhibits expression of Crk-1.
8. The composition of claim 2, wherein the polynucleotide is an oligonucleotide having the sequence GTCGAACCGGCGGAGGA (SEQ ID NO:6).
13. A composition comprising (i) a polynucleotide that hybridizes to the translation initiation site of a Grb2-encoding nucleic acid or (ii) a polynucleotide that hybridizes to the translation initiation site of a Crk1-encoding nucleic acid, wherein said composition inhibits the expression of Grb2 and Crk1 [emphases added].

Appeal No. 2005-0740
Application No. 08/679,437

The claims stand rejected as follows:

- I. Claims 8 and 17-28 stand rejected under 35 U.S.C. § 112, first paragraph, because the specification is enabling only for claims limited to a method of inhibiting proliferation of chronic myelogenous leukemic cells in vitro comprising treating the cells with a composition comprising a polynucleotide consisting of SEQ ID NO. 5.
- II. Claims 1-4, 7 and 9-20 stand rejected under 35 U.S.C. § 103 as being unpatentable over Bonati, Clabretta and Groffen in view of Lowenstein, ten Hoeve and Wang.

We affirm Rejection I with respect to claim 8, but do not reach the merits with respect to claims 17-28. We affirm Rejection II with respect to claims 1-4, 7, 9-12, 15 and 16, but do not reach the merits with respect to claims 13, 14 and 17-20. In addition, we set forth a new ground of rejection pursuant to 37 C.F.R. § 41.50(b) for claims 13, 14 and 17-28.

Background

The present invention involves the treatment of a type of cancer; viz., chronic myelogenous leukemia. For purposes of background, the specification discloses (p. 1, line 15- p. 3, line 11) that

[c]hronic myelogenous leukemia (CML) is a hematologic malignancy in which uncontrolled proliferation of granulocytes occurs. It often is characterized by the reciprocal translocation of chromosomes 9 and 22, which relocates the Abelson (abl) protooncogene onto the 3'-end of the breakpoint cluster region (bcr). This produces a chimeric bcr-abl gene encoding a p210^{bcr-abl} fusion protein, which is tumorigenic and is necessary for the growth of CML cells [citations omitted].

The bcr-abl protein can autophosphorylate at the 177 tyrosine amino acid found within the first exon of bcr. When phosphorylated, the bcr domain of the bcr-abl protein binds to the SH2 domain of the growth factor receptor-bound protein 2 (Grb2) adaptor protein. Through the SH3 domain, Grb2 binds to the human Son of sevenless 1 (hSos1) GDP/GTP exchange factor resulting in ras protein activation. . . . It is believed that when the normal bcr protein becomes tyrosine phosphorylated at amino acid 177, it also will complex with Grb2. When the bcr-abl protein is expressed, the p46 and p52 Shc (Puil et al., 1994) proteins become tyrosine phosphorylated as well. These Shc proteins have also been shown to form stable complexes with Grb2. Therefore, Grb2 appears to play a very important role in the tumorigenicity mediated by the bcr-abl protein (Puil et al. 1994; Pendergast et al., 1993).

Another adaptor protein, Crk-like (Crkl), also has been found to bind to bcr-abl. Unlike Grb2, Crkl binds to bcr-abl through the abl domain. Through its SH3 domain, Crkl can also bind to hSos1, which again leads to Ras protein activation (ten Hoeve et al., 1994a and 1994b). Thus, via the Grb2 and Crkl adaptor proteins, the bcr-abl protein has been linked to ras activation, which is known to lead to tumorigenesis. When ras protein expression is inhibited, proliferation of CML cells is also inhibited. Therefore, one of the major pathways in which bcr-abl protein promotes CML proliferation is by activating ras protein (Skorski et al., 1994 and 1995).

Specifically, as indicated by the claims above, the present invention is directed to antisense oligonucleotides which target the translation initiation site of a Grb2-encoding polynucleotide and a Crkl-encoding polypeptide and method of inhibiting cancer cell proliferation using said oligonucleotides.

Discussion

I. Enablement

As discussed above, the appellants have argued that the claims do not stand or fall together. The appellants' grouping of the claims is consistent with the examiner's arguments. That is, we find that the examiner makes two, separate enablement rejections- one with respect to claim 8, and one with respect to claims 17-28. We have addressed the issues accordingly.

A. Claim 8

The examiner argues (Answer, pp. 8-9) that

. . . the specification teaches that antisense oligos of SEQ ID NOS. 5 and 6 were able to inhibit CML cells' proliferation in vitro, however the sequence of SEQ ID NO. 6 does not appear to be complementary to any sequences within the known Crkl cDNA molecule. The specification does not teach through what mechanism the said [sic] antisense molecule was able to cause inhibition of Crkl mRNA since the sequence was not complementary to the known sequence and thereby cannot possibly be causing inhibition through an antisense mechanism. Also, the skilled artisan would not know what other noncomplementary sequences would be able to elicit Crkl specific inhibition in CML without guidance or working examples from the specification.

Here, we find that SEQ ID NO: 3 sets forth a DNA sequence encoding a Crkl polypeptide which includes the structural gene as well as some of the 5' and 3' flanking regions. We further find that the specification discloses that SEQ ID NO:6 targets the translation initiation site of Crkl. Specification, p. 29. We still further find that with the exception of a single nucleotide, the region of SEQ ID NO: 3 which is targeted by

Appeal No. 2005-0740
Application No. 08/679,437

SEQ ID NO:6 includes the last nucleotide of the initial ATG codon and the nucleotides which encode amino acids 2-7.

We share the examiner's concerns with respect to whether the difference in the nucleotide sequence between SEQ ID NO:6 and SEQ ID NO:3 would enable the formation of a stable duplex needed to inhibit the expression of Crk-I. Stable duplex formation is a function of both the nucleotide sequence and the length of the antisense oligonucleotide. See, e.g., Calabretta, col. 16, lines 53-60. We note that Calabretta discloses that "[m]ore than one mismatch will not be tolerated for antisense oligomers of less than about 21 nucleotides" [emphasis added].² *Id.*, lines 61-63. However, SEQ ID NO:6 is seventeen (17) nucleotides in length and there is only a single nucleotide

² We point out that Calabretta also discloses that:

One skilled in the art may readily determine the degree of mismatching which may be tolerated between any given antisense oligomer and the target sequence, based upon the melting point, and therefore the thermal stability, of the resulting duplex [Calabretta, col. 16, lines 63-67].

Calabretta further discloses a method of determining the thermal stability of hybrids formed by antisense oligonucleotides and their target genes. *Id.*, col. 17, lines 1-21.

Appeal No. 2005-0740
Application No. 08/679,437

mismatch. Thus we find that, on the one hand, there is only one nucleotide mismatch between SEQ ID NO:3 and its reverse complement SEQ ID NO:6, not more than one; and on the other, that SEQ ID NO:6 is less than 21 nucleotides.

We are mindful that “a specification disclosure which contains a teaching of the manner and process of making and using the invention in terms which correspond in scope to those used in describing and defining the subject matter sought to be patented must be taken as in compliance with the enabling requirement of the first paragraph of § 112 unless there is a reason to doubt the objective truth of the statements contained therein which must be relied on for enabling support.” In re Marzocchi, 439 F.2d 220, 223, 169 USPQ 367, 369 (CCPA 1971). However, under the circumstances discussed above with respect to the mismatch between SEQ ID NOS: 3 and 6, we find that there was reason for the examiner to doubt that the specification would not have enabled one skilled in the art to “make and use” the invention as described in claim 8. Having articulated this doubt, on the record, it was reasonable for the examiner to shift the burden to the appellants to establish otherwise.

In this regard, we find that the appellants contend that (1) “the specification provides that levels of complementarity as low as 13 out of 15 bases may function to specifically bind”; and (2) “[c]learly, a 17/18 base match is a level of complementarity higher than 13/15 and sufficient to effect the sequence specific binding common to all

Appeal No. 2005-0740
Application No. 08/679,437

nucleotide sequences.” Brief, p. 14, para. 1. With respect to the first argument, the appellants do not point out, and we do not find, any teachings in the specification that 13 out of 15 nucleotides forms a stable duplex capable of inhibiting the expression of the crk-I gene, and none has been pointed out by the appellants. With respect to the second argument, we find that the appellants have not provided any evidence to support their position. Thus, we find that the appellants’ response to the examiner consists only of attorney argument. We point out that arguments of counsel cannot take the place of objective evidence. In re Payne, 606 F.2d 303, 315, 203 USPQ 245, 256 (CCPA 1979); Meitzner v. Mindick, 549 F.2d 775, 782, 193 USPQ 17, 22, (CCPA) cert. denied 434 U.S. 854 (1977); In re Lindner, 457 F.2d 506, 508, 173 USPQ 356, 238 (CCPA 1972). We accord such arguments little or no evidentiary weight.

Finally, we find that in the Reply Brief, the appellants have acknowledged that the mismatch of a single nucleotide was “introduced into the specification by an inadvertent typographical error at the time of filing.” Reply Brief, p. 15, fn. 1. Thus, as we understand it, SEQ ID NO:6 as set forth in the specification, was never made or used by the appellants. In our view, the appellants’ acknowledgment further supports the examiner’s position that the specification fails to teach one skilled in the art “how to use” SEQ ID NO:6 to inhibit the expression Crk-I as set forth in the claims.

Accordingly, Rejection I is affirmed with respect to claim 8.

B. Claims 17-28

The examiner argues that the claims are directed to unspecific nucleotide sequences “of unproven and unpredictable activity.” Answer, p. 6. The examiner further argues that the claimed methods encompass the administration of antisense oligonucleotides “to a wide variety of cells or tissue types in the treated patient, at any concentration of oligo, by any route, and by any physical means.” Id. The examiner still further argues that the specification only discloses the inhibition of tumor cell proliferation in vitro with CMV cells transfected with liposomes containing an antisense sequences which hybridize to Grb2 or Crkl mRNA. Id. The examiner contends that the in vitro studies disclosed in the specification examples “would not have been accepted . . . as correlative with the successful operation of the claimed invention in vivo.” Id., sentence bridging pp. 6-7. Thus, the examiner concludes that the specification does not enable the use of antisense oligonucleotides for gene therapy. Id., pp. 7-8. The examiner relies on Agrawal, Branch and Gewirtz, for support.

We agree that these claims are not patentable; however, because our reasons differ from those of the examiner we have set forth a new ground of rejection under both the first and second paragraphs of § 112, below. Accordingly, we need not address the enablement issue with respect to claims 17-28.

II. Obviousness

The examiner argues that:

A. Bonati discloses the construction of Grb2 sense and antisense

Appeal No. 2005-0740
Application No. 08/679,437

oligonucleotides and the use of said oligonucleotides to inhibit leukemic cell growth.

Answer, p. 4.

B. Calabretta discloses the inhibition of bcr/abl using antisense oligonucleotides directed to “the transcription regions of transcripts and also discloses the use of antisense oligos targeted to multiple transcripts which encode cooperating polypeptides.” Answer, p. 4. Calabretta further discloses the use of liposomes to deliver the antisense constructs. Id.

C. Groffen discloses “the CRKL transcript sequence as well as inhibition of said transcript using antisense oligos.” Answer, p. 4.

D. Loewenstein discloses a cDNA clone encoding GRB2 and that said gene “is a cooperating polypeptide with bcr/abl which is involved in the leukemic pathway.” Answer, p. 4.

E. ten Hoeve discloses that “GRB2 and CRKL are cooperating polypeptides with the bcr/abl polypeptide in the leukemic pathway.” Answer, p. 4.

F. Wang discloses the use of liposomes comprising DOPC to deliver DNA to cells. Answer, p. 5.

The examiner concludes (Answer, p. 5) that

[i]t would have been obvious to one of ordinary skill in the art to target the translation initiation regions of GRB2, CRKL, or bcr/abl with antisense oligos since Bonati et al., Calabretta et al., and Groffen et al. all disclose the use of antisense oligos to inhibit their respective transcripts. It would have been obvious to target the translation initiation region since Calabretta et al. clearly disclose that the region is susceptible to antisense inhibition. Moreover, it would have been further obvious to target multiple transcripts since Calabretta et al. clearly teach that cooperating polypeptides can be targeted simultaneously using [sic, using] antisense oligos. Both Lowenstein et al. and ten Hoeve et al. clearly teach that both GRB2 and CRKL are cooperating polypeptides with bcr/abl. Moreover, one of ordinary skill in the art would have recognized that inhibition of more than one transcript would have resulted in efficient inhibition of leukemia cells as taught by Calabretta et al. Additionally, it would have been further obvious to deliver the antisense oligos using liposomes comprising DOPC, as disclosed by Wang et al., since DOPC was shown to be an efficient transfection agent in the formation of liposomes.

Claim 1

In response, the appellants acknowledge that Calabretta discloses the use of antisense oligonucleotides which bind to the translation start site of genes; however, they contend that the teachings of the patent are limited in that it only discloses the use of antisense oligonucleotides to inhibit the oncogenes and proto-oncogenes disclosed therein. Brief, p. 6. The appellants argue that Calabretta discloses that oncogenes are genes which are responsible in whole, or in part, for the neoplastic transformation of a host cell; whereas, proto-oncogenes are normal cellular genes which when altered become oncogenes. Id., p. 7. According to the appellants, Grb2 and Crk-I were not known in the art as either oncogenes or proto-oncogenes. Id. The appellants rely on

Appeal No. 2005-0740
Application No. 08/679,437

Lowenstein (“page 237 [sic, 437?], last sentence”) which is said to teach that Grb2 does not have oncogenic potential. Id.

The appellants further argue that Senechal³ (p. 23260), “also teaches away from the idea that Grb2 is an oncogene.” Brief, p. 7. In addition, Senechal is said to show that even after the filing date of the present application, Crk-I was not recognized as an oncogene by those having ordinary skill in the art. Id.

We find the appellants’ arguments unpersuasive.

It is well established that the examiner has the initial burden under § 103 to establish a prima facie case of obviousness. In re Oetiker, 977 F.2d 1443, 1445, 24 USPQ2d 1443, 1444 (Fed. Cir. 1992); In re Piasecki, 745 F.2d 1468, 1471-72, 223 USPQ 785, 787-88 (Fed. Cir. 1984). It is the examiner’s responsibility to show that some objective teaching or suggestion in the applied prior art, or knowledge generally available [in the art] would have led one of ordinary skill in the art to combine the references to arrive at the claimed invention. Pro-Mold & Tool Co. v. Great Lakes Plastics, Inc., 745 F.3d 1568, 1573, 37 USPQ2d 1626, 1629 (Fed. Cir. 1996).

With respect to claim 1, we begin our deliberations with the consideration of the primary reference, Bonati. To that end, it cannot be gainsaid that Bonati discloses a

³ Senechal, et al. (Senechal), “The CRKL Adaptor Protein Transforms Fibroblasts and Functions in Transformation by the BCR-ABL Oncogene,” The Journal of Biological Chemistry, vol. 271, pp. 23255-61 (1996).

Appeal No. 2005-0740
Application No. 08/679,437

composition comprising a nucleotide sequence which hybridizes to a Grb-2 encoding polynucleotide and which inhibits the expression of Grb-2. Specifically, Bonati discloses that an anti-sense oligonucleotide specific for the Grb2 gene is able to inhibit 50% of leukemic colony growth. Bonati reports that the “study indicates that the modulation of [the] Grb2 gene inhibits leukemic colonies generated by the BCR-ABL positive K562 cell line.” We agree with the appellants that Bonati does not disclose the portion of the Grb2 gene to which the antisense oligonucleotide hybridizes. Nor does Bonati disclose that Grb2 is an oncogene. However, this does not mean that the examiner has not made a prima facie case of obviousness.

First, it is immaterial that Bonati does not teach the oncogenic potential of the Grb2 gene. We point out that the reason to combine the teachings of the applied prior art does not have to be the same as that of the appellants. In re Kemps, 97 F.3d 1427, 1430, 40 USPQ2d 1309, 1311 (Fed. Cir. 1996); In re Dillon, 919 F.2d 688, 693, 16 USPQ2d 1897, 1901 (Fed. Cir. 1990) (in banc) (“the motivation in the prior art to combine the references does not have to be identical to that of the applicant to establish obviousness”). Here, we find that Bonati’s disclosure that antisense oligonucleotides which inhibit Grb 2 expression and inhibit leukemic colonies suggests that this gene plays a crucial role in chronic myelogenous leukemia and would have motivated one having ordinary skill in the art to make antisense nucleotides which target said gene.

Second, with respect to whether it would have been obvious to one of ordinary skill in the art to synthesize an antisense oligonucleotide which hybridizes to the translation initiation site of Grb2, we direct attention to the teachings of Calabretta. Specifically, we find that Calabretta discloses that “[i]t is believed that translation is most effectively inhibited by blocking the mRNA at a site at or near the initiation codon. Thus, oligonucleotides complementary to the 5'-region of mRNA transcript [sic, transcripts] are preferred.” Calabretta, col. 14, 17-19. We do not agree with the appellants that the teachings of Calabretta are limited to the antisense oligonucleotides described therein which bind to oncogenes or protooncogenes. Rather, in our view, the teachings of Calabretta reflect what was generally known in the field of molecular biology with respect to the construction of antisense oligonucleotides. Thus, we hold that given the teachings of (i) Bonati that antisense oligonucleotides specific for the Grb2 gene are able to inhibit leukemic cell growth; and (ii) Calabretta that the antisense oligonucleotides which are complementary to the translation initiation site of a gene are the most effective at inhibiting mRNA translation, it would have been obvious to one of ordinary skill in the art to construct an antisense oligonucleotide which is complementary to the translation initiation site of a Grb2-encoding polypeptide. Moreover, given the teachings of Bonati with respect to the inhibition of leukemic colonies, said person would have had a reasonable expectation that said antisense oligonucleotide would inhibit Grb2 expression.

Accordingly, we affirm the rejection with respect to claim 1. As discussed above, claims 3, 7, 9, 11, 13 and 15 fall with claim 1.

Claim 2

With respect to claim 2, we agree with the examiner that Groffen discloses the use of antisense oligonucleotides to “decrease or prevent transcription or translation of CRKL . . . [f]or example, antisense nucleotides . . . specific, i.e. complimentary [sic, complementary], for mRNA encoding CRKL to decrease or prevent the translation of that mRNA into protein.” Groffen, p. 17, lines 20-26. As discussed above, it is immaterial that Groffen does not teach that the Crkl gene is an oncogene. In re Dillon, 919 F.2d at 693, 16 USPQ2d at 1901 (“the motivation in the prior art to combine the references does not have to be identical to that of the applicant to establish obviousness”). We find that Groffen’s disclosure that antisense oligonucleotides which inhibit transcription (Groffen, p. 17, lines 16-29), translation or Crkl protein activity can be employed to treat chronic lymphoblastic leukemia, would have reasonably suggested a composition comprising an antisense oligonucleotide which hybridizes to the Crkl gene to one of ordinary skill in the art. Thus, the issue then becomes whether it would have been obvious to said person to synthesize an antisense oligonucleotide which hybridizes to the translation initiation site of Crkl. To that end, attention is directed to our findings with respect to the teachings of Calabretta. As discussed above, Calabretta exemplifies what was known in the art with respect to the construction of

Appeal No. 2005-0740
Application No. 08/679,437

antisense oligonucleotides. Thus, we hold that given the teachings of (i) Groffen with respect to construction of antisense oligonucleotides specific for the Crkl gene, and the use of said oligonucleotides to treat CML (leukemia); and (ii) Calabretta that antisense oligonucleotides which are complementary to the translation initiation site of a gene are the most effective at inhibiting mRNA translation, it would have been obvious to one of ordinary skill in the art to construct an antisense oligonucleotide which is complementary with the translation initiation site of a Crkl-encoding protein.

Accordingly, we affirm the rejection with respect to claim 2. As discussed above, claims 4, 10, 12 and 16, fall with claim 2.

Claim 13

For the reasons set forth below, we need not reach the merits of claims 13, 14 and 17-20.

New Ground of Rejection

Under the provisions of 37 C.F.R. § 41.50(b), we enter the following new ground of rejection.

Claims 13, 14 and 17-28 are rejected under 35 U.S.C. § 112, first paragraph, written description, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventors had possession of the claimed invention at the time the application was

Appeal No. 2005-0740
Application No. 08/679,437

filed.

We point out that when new matter is added to the claims, the proper course of action is to reject said claims for failing to satisfy the written description requirement of §112, first paragraph. In re Rasmussen, 650 F.2d 1212, 1214, 211 USPQ 323, 326 (CCPA 1981) (“The proper basis for rejection of a claim amended to recite elements thought to be without support in the original disclosure, therefore, is § 112, first paragraph ...”). The purpose of the written description requirement is to “ensure that the scope of the right to exclude, as set forth in the claims does not overreach the scope of the inventor’s contribution to the field as far as described in the patent specification.” Reiffin v. Microsoft Corp., 214 F.3d 1342, 1345, 54 USPQ2d 1915, 1917 (Fed. Cir. 2000). To that end, to satisfy the written description requirement, the inventor “must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention” [first emphasis added]. Vas-Cath Inc. v. Mahurkar, 935 F.2d 1555, 1563-64, 19 USPQ2d 1111, 1117 (Fed. Cir. 1991). “One shows that one is ‘in possession’ of the invention by describing the invention, with all its claimed limitations” [emphases in original]. Lockwood v. American Airlines, 107 F.3d 1565, 1572, 41 USPQ2d 1961, 1966 (Fed. Cir. 1997).

We further point out that it is not necessary for the specification to describe the claimed invention ipsisssimis verbis; all that is required is that it reasonably convey to those skilled in the art that, as of the filing date sought, the inventor was in possession

Appeal No. 2005-0740
Application No. 08/679,437

of the claimed invention. Union Oil of California v. Atlantic Richfield Co., 208 F.3d 989, 997, 54 USPQ2d 1227, 1232 (Fed. Cir. 2000); Vas-Cath Inc. v. Mahurkar, 935 F.2d at 1563-64, 19 USPQ2d at 1117; In re Gosteli, 872 F.2d 1008,1012, 10 USPQ2d 1614, 1618 (Fed. Cir. 1989); In re Edwards, 568 F.2d 1349, 1351-52,196 USPQ 465, 467 (CCPA 1978).

This application is said to be a continuation pursuant to 35 U.S.C. § 120 of Application No. 08/545,473 (filed October 19, 1995), now U.S. Patent No. 6,071,890. As such, no new matter can be added to the specification after the filing date. 37 C.F.R. §1.53(d)(5).

We have carefully reviewed the specification, but do not find any teachings of a composition comprising a polynucleotide which is able to bind to the translation initiation site of either a Grb2-encoding protein or a Crkl-encoding protein wherein said composition inhibits the expression of both proteins as set forth in claims 13 and 17. Rather with respect to claim 13, we find that the appellants acknowledge that it was first presented in its current form as an attachment to the Brief. Brief, p. 2, para. 2. As to claim 17, we find that it was added by amendment along with the Brief filed on March 2, 1998. Accordingly, since the specification does not provide the teachings which “convey with reasonable clarity to those skilled in the art that, as of the filing date sought, .. [the inventors were] .. in possession of the [claimed] invention” [Vas-Cath Inc. v. Mahurkar, 935 F.2d at 1563-64, 19 USPQ2d at 1117], we find that it fails to satisfy the written description requirement of 35 U.S.C. § 112, first paragraph.

Appeal No. 2005-0740
Application No. 08/679,437

Regarding the affirmed rejection(s), 37 CFR § 41.52(a)(1) provides "[a]ppellant may file a single request for rehearing within two months from the date of the original decision of the Board."

In addition to affirming the examiner's rejection(s) of one or more claims, this decision contains a new ground of rejection pursuant to 37 CFR § 41.50(b) (effective September 13, 2004, 69 Fed. Reg. 49960 (August 12, 2004), 1286 Off. Gaz. Pat. Office 21 (September 7, 2004)). 37 CFR § 41.50(b) provides "[a] new ground of rejection pursuant to this paragraph shall not be considered final for judicial review."

37 CFR § 41.50(b) also provides that the appellant, WITHIN TWO MONTHS FROM THE DATE OF THE DECISION, must exercise one of the following two options with respect to the new ground of rejection to avoid termination of the appeal as to the rejected claims:

(1) *Reopen prosecution.* Submit an appropriate amendment of the claims so rejected or new evidence relating to the claims so rejected, or both, and have the matter reconsidered by the examiner, in which event the proceeding will be remanded to the examiner. . . .

(2) *Request rehearing.* Request that the proceeding be reheard under § 41.52 by the Board upon the same record. . . .

Should the appellant elect to prosecute further before the examiner pursuant to 37 CFR § 41.50(b)(1), in order to preserve the right to seek review under 35 U.S.C. §§ 141 or 145 with respect to the affirmed rejection, the effective date of the affirmance is deferred until conclusion of the prosecution before the examiner unless, as a mere

Appeal No. 2005-0740
Application No. 08/679,437

incident to the limited prosecution, the affirmed rejection is overcome.

If the appellant elects prosecution before the examiner and this does not result in allowance of the application, abandonment or a second appeal, this case should be returned to the Board of Patent Appeals and Interferences for final action on the affirmed rejection, including any timely request for rehearing thereof.

AFFIRMED-IN-PART; NEW GROUND OF REJECTION

JOAN ELLIS)	
Administrative Patent Judge)	
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)	BOARD OF PATENT
TONI R. SCHEINER)	APPEALS
Administrative Patent Judge)	AND
)	INTERFERENCES
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DEMETRA J. MILLS)	
Administrative Patent Judge)	

JE/ki

Appeal No. 2005-0740
Application No. 09/679,437

David L. Parker
Fulbright & Jaworski, LLP
600 Congress Avenue
Suite 2400
Austin, TX 78701