

December 17, 2010

VIA ECF

Elisabeth A. Shumaker
Clerk of Court
United States Court of Appeals for the Tenth Circuit
The Byron White U.S. Courthouse
1823 Stout Street
Denver, CO 80257

Re: *Laboratory Corporation Holdings v. Metabolite Laboratories, Inc.* (Case No. 10-1194)

Dear Ms. Schumaker:

Pursuant to FRAP 28(j), Metabolite Laboratories, Inc. respectfully advises the panel of a decision issued on December 13, 2010 by the Board of Patent Appeals and Interferences (“BPAI”) (enclosed).

LabCorp has suggested that the validity of the ’658 patent is “doubtful.” LC CAFC Br. 4 n.1. The December 13 decision disposes of that suggestion. On reexamination, an examiner had originally determined that claims 13, 15-17 and 33 of the ’658 patent were invalid for obviousness. In a July 30, 2009 decision (also enclosed), the BPAI reversed that determination and confirmed the validity of the patent. On rehearing, the BPAI reaffirmed its previous holding that none of the prior art references rendered the claims obvious.

The BPAI’s decision underscores the error committed by the district court, whose summary judgment ruling was premised on the conclusion that “LabCorp’s material breach ... terminated” the License Agreement. A26. But LabCorp could *lawfully* terminate the Agreement *only* by following the contractually prescribed steps, including a demonstration that its assays do “not infringe a valid enforceable claim of the Licensed Patents.” A8999. The Federal Circuit previously held that LabCorp’s alternative serum homocysteine assays (whether performed in-house or outsourced to Specialty Laboratories) infringe the ’658 patent. *Lab. Corp. of Am. Holdings v. Metabolite Labs., Inc.*, 599 F.3d 1277 (Fed. Cir. 2010). And the BPAI decision confirms that the ’658 patent is a valid and enforceable patent. Read together, these decisions indisputably establish that LabCorp owes know-how royalties on all Net Sales of serum homocysteine assays for the period between the date of the jury verdict in November 2001 and 60 days after LabCorp complied with the contractual termination provision (October 20, 2007). A22009; *see* A20 n.57; A8999.

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Rather than defend the decision below, LabCorp has argued that *Metabolite* “chose” to terminate the License Agreement. That argument fails the test of common sense: *Metabolite* has spent *years* attempting to enforce its intellectual property rights. LabCorp has had the benefits of *Metabolite*’s inventions and should be held to its contractual obligation to pay for those benefits.

Respectfully submitted,

s/ Mark A. Perry

Mark A. Perry

MAP/rjl

cc: (w/enclosures, via ECF and U.S. Mail)
All Counsel of Record, per Court’s Service List

Enclosures

ELECTRONIC SUBMISSION CERTIFICATE

I, the undersigned, hereby certify with regard to the foregoing FRAP 28(j) LETTER OF METABOLITE LABORATORIES, INC., that:

1. all required privacy redactions have been made; and
2. the ECF submission was scanned for viruses with Symantec AntiVirus, Version 10.1 (August 31, 2009 update), and, according to Symantec AntiVirus, is free of viruses

s/ Mark A. Perry

CERTIFICATE OF SERVICE

I hereby certify that a copy of this FRAP 28(j) LETTER OF METABOLITE LABORATORIES, INC., was served on the following persons via the ECF system and U.S. Mail on December 16, 2010:

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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
90/008,305	10/23/2006	4940658	466992801100	9384
22334	7590	12/13/2010	EXAMINER	
PETER F WEINBERG GIBSON DUNN AND CRUTCHER LLP SUITE 4200 1801 CALIFORNIA STREET DENVER, CO 80202			PONNALURI, PADMASHRI	
			ART UNIT	PAPER NUMBER
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			12/13/2010	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte COMPETITIVE TECHNOLOGIES, INC.
and
THE TRUSTEES OF COLUMBIA UNIVERSITY,
Appellants

Appeal 2009-005519
Reexamination Control 90/008,305
Patent 4,940,658¹
Technology Center 3900

Before CAROL A. SPIEGEL, DONALD E. ADAMS, and
ROMULO H. DELMENDO, *Administrative Patent Judges*.

SPIEGEL, *Administrative Patent Judge*.

DECISION ON REQUEST FOR REHEARING²

The Examiner requests rehearing of our 30 July 2009 Decision ("Dec."), in which we reversed her decision to reject claims 13, 15-17, and

¹ Patent 4,940,658, *Assay for Sulfhydryl Amino Acids and Methods for Detecting and Distinguishing Cobalamin and Folic Acid Deficiency*, issued 10 July 1990, to Allen et al. The real parties in interest are COMPETITIVE TECHNOLOGIES, INC. and THE TRUSTEES OF COLUMBIA UNIVERSITY (STATEMENT UNDER 37 CFR 3.73(b) filed 14 December 2006).

² The two month period for filing an appeal or commencing a civil action, as recited in 37 C.F.R. § 41.52, begins to run from the "MAIL DATE" shown on the PTOL-90A cover letter attached to this decision.

33 under 35 U.S.C. § 103(a) as obvious over Refsum³ in view of Kass,⁴ Wilcken,⁵ and Westhuysen⁶ in the above-identified reexamination⁷ of U.S. Patent 4,940,658 (Request for Rehearing mailed September 29, 2009 ("Req. Reh'g")).

We deny the Examiner's request to modify our original decision.

According to the Examiner, the Board's disagreement with her conclusion of obviousness "is based on the alleged failure by the Examiner to provide a factual basis for extrapolating 'total homocysteine' from one or more forms which comprise it, especially in view of known redistribution between free and bound forms of homocysteine" (Req. Reh'g 1). The Examiner also argues that the secondary references teach a correlation of homocysteine with cobalamin and folate deficiency and that "it is irrelevant if Kass, Wilcken or Westhuyzen are detecting 'less than total' homocysteine" (*id.* 5).

In our original decision, we found that the '658 patent teaches that "both cobalamin (vitamin B₁₂) and folate deficiencies result in an elevated total homocysteine level, but an elevated total homocysteine level cannot

³ Refsum et al., *Radioenzymic Determination of Homocysteine in Plasma and Urine*, 34 CLINICAL CHEMISTRY 624-628 (1985) ("Refsum").

⁴ L. Kass, *Cytochemical Detection of Homocysteine in Pernicious Anemia and in Chronic Erythremic Myelosis*, 67 AM. J. OF CLINICAL PATHOLOGY 53-56 (1977) ("Kass").

⁵ Wilcken et al., *Homocysteinemia, Ischemic Heart Disease, and the Carrier State for Homocystinuria*, 32 METABOLISM 363-370 (1983) ("Wilcken").

⁶ Westhuyzen et al., *Plasma amino acids and tissue methionine levels in fruit bats (*Rousettus aegyptiacus*) with nitrous oxide-induced vitamin B₁₂ deficiency*, 53 BRIT. J. OF NUTRITION 657-662 (1985) ("Westhuyzen").

⁷ Request for Reexamination filed 23 October 2006.

distinguish between a folate and/or cobalamin deficiency" (Dec. at 3, Finding of Fact ("FF") 1). Claim 13 is illustrative and reads

A method for detecting a deficiency of cobalamin or folate in warm-blooded animals comprising the steps of:
 assaying a body fluid for an elevated level of total homocysteine; and
 correlating an elevated of total homocysteine in said body fluid with a deficiency of cobalamin or folate.

In our original decision, we found that total homocysteine consists of four species, i.e., "a combination of about 30% 'free' (homocysteine-cysteine mixed disulfide, homocystine, homocysteine) and about 70% 'bound' (protein-homocysteine mixed disulfide) forms" (Dec. at 3, FF 2, citing Ueland⁸). We further found that none of the applied references disclose any correlation between an elevated total homocysteine level and cobalamin or folate deficiency and that none of Kass, Wilcken, or Westhuyzen measured total homocysteine levels (Dec. at 4, FF 4, and 6-8).

The Examiner argues that "Refsum indicates that there is a linear relationship between free homocysteine and bound homocysteine in a given sample (pg. 625, right column)" (Req. Reh'g 2). However, the Examiner ignores the instability of all forms of homocysteine except for total homocysteine (Dec. at 3, FF 2). To wit, the Examiner has ignored the teachings of Refsum, e.g., "[i]n plasma not supplemented with

⁸ Ueland et al., *Plasma homocysteine, a risk factor for vascular disease: Plasma levels in health, disease, and drug therapy*, 114 J. OF LABORATORY CLINICAL MEDICINE 473-501 (1989) ("Ueland"), cited as relied upon by the Examiner (Examiner's Answer mailed 24 September 2008 ("Ans.") at 3).

dithioerythritol, the value for free homocysteine remained constant for 20 minutes, then declined (Figure 1)" (Refsum at 625, col. 2, ¶ 4) and Ueland (Dec. at 3, FF 2), a reference which the Examiner herself relied upon (see Ans. at 3). Thus, we discern no error in our finding that "homocysteine is only a part of 'total' homocysteine and its increase does not necessarily mean an increase in 'total' homocysteine (FF 2)" (Dec. at 7). In other words, "[t]he Examiner has made no findings which provide a factual basis for extrapolating 'total homocysteine' levels from one or more of the forms which comprise it, especially in view of the known redistribution between free and bound forms of homocysteine (FF 2)" (Dec. at 7-8).

DECISION ON REHEARING

In conclusion, the Examiner's Request for Rehearing is granted to the extent that we have reconsidered our original Decision, but is denied with respect to making any change therein.

DENIED

ack

Patent Owner

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Appeal 2009-005519

Reexamination Control 90/008,305

Patent 4,940,658

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90/008,305	10/23/2006	4940658	466992801100	9384

22334 7590 07/30/2009
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EXAMINER

PONNALURI, PADMASHRI

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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte COMPETITIVE TECHNOLOGIES, INC.
and
THE TRUSTEES OF COLUMBIA UNIVERSITY,
Appellants

Appeal No. 2009-005519
Reexamination Control 90/008,305¹
Patent 4,940,658²
Technology Center 3900

Decided³: July 30, 2009

Before CAROL A. SPIEGEL, DONALD E. ADAMS, and
ROMULO H. DELMENDO, *Administrative Patent Judges*.

SPIEGEL, *Administrative Patent Judge*.

DECISION ON APPEAL

¹ Request for Reexamination filed 23 October 2006.

² Patent 4,940,658 ("the 658 patent" or "Spec."), *Assay for Sulfhydryl Amino Acids and Methods for Detecting and Distinguishing Cobalamin and Folic Acid Deficiency*, issued 10 July 1990, to Allen et al. The real parties in interest are COMPETITIVE TECHNOLOGIES, INC. and THE TRUSTEES OF COLUMBIA UNIVERSITY (STATEMENT UNDER 37 CFR 3.73(b) filed 14 December 2006).

³ The two-month time period for filing an appeal, as recited in 37 C.F.R. § 1.304, begins to run from the decided date shown on this page of the decision. The time period does not run from the Mail Date.

I. Statement of the Case

Appellants appeal under 35 U.S.C. §§ 134 and 306 from an Examiner's final rejection of claims 13, 15-17, and 33 (App. Br. 2; Ans.⁴ 2). We have jurisdiction under 35 U.S.C. §§ 134 and 306. We REVERSE.

Claim 13 is illustrative of the subject matter on appeal and reads (App. Br. Claim App'x.):

A method for detecting a deficiency of cobalamin or folate in warm-blooded animals comprising the steps of:
 assaying a body fluid for an elevated level of total homocysteine; and
 correlating an elevated level of total homocysteine in said body fluid with a deficiency of cobalamin or folate.

The Examiner has rejected claims 13, 15-17, and 33 as unpatentable under 35 U.S.C. § 103(a) over Refsum⁵ in view of Kass,⁶ Wilcken,⁷ and Westhuyzen⁸ (Ans. 3-8).

The dispositive issue is whether the applied prior art teaches, or would have suggested all of the claim limitations to one of ordinary skill in the art.

⁴ Examiner's Answer mailed September 2008 ("Ans.").

⁵ Refsum et al., "Radioenzymic Determination of Homocysteine in Plasma and Urine," 34 *Clinical Chemistry* 624-628 (1985) ("Refsum").

⁶ L. Kass, "Cytochemical Detection of Homocysteine in Pernicious Anemia and in Chronic Erythremic Myelosis," 67 *American Journal of Clinical Pathology* 53-56 (1977) ("Kass").

⁷ Wilcken et al., "Homocysteinemia, Ischemic Heart Disease, and the Carrier State for Homocystinuria," 32 *Metabolism* 363-370 (1983) ("Wilcken").

⁸ Westhuyzen et al., "Plasma amino acids and tissue methionine levels in fruit bats (*Rousettus aegyptiacus*) with nitrous oxide-induced vitamin B₁₂ deficiency," 53 *British Journal of Nutrition* 657-662 (1985) ("Westhuyzen").

II. Findings of Fact ("FF")

- [1] According to the 658 patent, both cobalamin (vitamin B₁₂) and folate deficiencies result in an elevated total homocysteine level, but an elevated total homocysteine level cannot distinguish between a folate and/or cobalamin deficiency (Spec. 4:17-23; 5:40-48, 64-66; 9:26-29).
- [2] Homocysteine in plasma exists as a combination of about 30% "free" (homocysteine-cysteine mixed disulfide, homocystine, homocysteine) and about 70% "bound" (protein-homocysteine mixed disulfide) forms (Ueland⁹ 476-477, 493). Free homocysteine is determined in plasma deproteinized with acid which coprecipitates the protein-bound homocysteine (*id.* 476). In particular, a

marked redistribution between free and bound homocysteine takes place after preparation of plasma. At a high temperature there is a rapid association of free homocysteine with plasma protein, and when stored at -20° C for some weeks, most homocysteine becomes protein-bound and only traces of free can be detected. ... This implies that, without cautious sample handling and storage, total plasma homocysteine should be determined. Free (acid soluble) homocysteine may become artificially low, and the possibility that displacement may affect the distribution between bound and free homocysteine should be considered. *Id.* 477, ¶¶ bridging columns 1 and 2 (footnotes omitted).

⁹ Ueland et al., "Plasma homocysteine, a risk factor for vascular disease: Plasma levels in health, disease, and drug therapy," 114 *Journal of Laboratory Clinical Medicine* 473-501 (1989), cited as relied upon by the Examiner (Ans. 3).

- [3] The 658 patent defines "total homocysteine" as the total amount of free and bound forms (Spec. 7:26-29).
- [4] Refsum determines total homocysteine by treating the plasma with dithierythritol to reduce the various homocysteine forms to free homocysteine, converting the free homocysteine to *S*-^{[14]C}adenosylhomocysteine with ^{[14]C}adenosine and *S*-adenosylhomocysteine hydrolase, and quantifying the *S*-^{[14]C}adenosylhomocysteine by HPLC (Refsum 624 ¶7, 627 ¶3).
- [5] Kass reports that red blood cells (erythrocytes or RBCs) and their cellular precursors from bone marrows of patients with untreated pernicious anemia (due to low vitamin B₁₂ levels) showed a yellow colored precipitate when reacted with nickel chloride, which was not seen in folate-deficient anemia (Kass 53 abstract; 55 Table 1).
- [6] According to Kass, "it is not possible to state with certainty that the yellow color observed in... patients who had pernicious anemia...reflects homocysteine alone, since coenzyme A gave a similar...reaction" (Kass 56 ¶3).
- [7] However, Kass states that "[i]f the yellow color...is indeed due to...homocysteine...[t]he test may also provide a rapid preliminary differentiation between vitamin B₁₂ deficiency...and folate deficiency" (Kass 56 ¶5).
- [8] Wilcken describes a pair of twins, each having elevated total homocysteine levels (calculated as twice the homocysteine concentration plus the cysteine-homocysteine concentration) (Wilcken 364 ¶6). The first twin showed normal RBC (340 ng/ml (normal 210-

678 ng/ml)) and serum (3.2 ng/ml (normal > 3.0 ng/ml)) folate levels and a "gray area" subnormal serum B₁₂ (144 pg/ml (normal 190-1000 pg/ml)) level. The second twin showed normal B₁₂ (195 pg/ml) and RBC folate (580 ng/ml) levels and a decreased serum folate (2.0 ng/ml) level. [Wilcken 366 ¶1 - 367 ¶1.]

- [9] While serum folate levels are labile, vary with diet, and are potentially misleading, RBC folate levels provide an accurate index of folate stores and its significance to folate deficiency is more nearly equivalent to that of serum B₁₂ levels in vitamin B₁₂ deficiency (Internal Medicine¹⁰ 1546 "Laboratory Features"; Clinical Hematology¹¹ 587 ¶9; Blood¹² 157 ¶2).
- [10] A serum vitamin B₁₂ level less than 100 pg/ml is indicative of B₁₂ deficiency (Internal Medicine 1544, ¶11; Clinical Hematology 587 ¶8; Blood 157 ¶¶1-2).
- [11] Westhuyzen reports that nitrous oxide-exposed fruit bats show increased plasma homocysteine levels (Westhuyzen 660 Table 2).
- [12] Westhuyzen states that plasma proteins were precipitated with acid prior to determining amino acid levels (Westhuyzen 658 ¶6).

¹⁰ Hershko et al., "Megaloblastic Anemias" in *INTERNAL MEDICINE*, vol. II, Stein et al. eds., Little, Brown and Company, Boston (1983), pp. 1542-1547 (App. Br. Evidence App'x. Ex. I) ("Internal Medicine").

¹¹ "Megaloblastic and Nonmegaloblastic Macrocytic Anemias" in *CLINICAL HEMATOLOGY*, 8th ed., Wintrobe et al., Lea & Febiger, Philadelphia (1981), pp. 559-604 (App. Br. Evidence App'x. Ex. J) ("Clinical Hematology").

¹² "Megaloblastic Anemias" in Jandl's *BLOOD: Textbook of Hematology*, Little, Brown and Company, Boston/Toronto (1987), pp. 153-180 (App. Br. Evidence App'x. Ex. K) ("Blood").

[13] According to Westhuyzen, long exposure to nitrous oxide depletes the vitamin B₁₂ stores in the bat (Westhuyzen 661 ¶5).

III. Discussion

When determining whether a claim is obvious, an Examiner must make "a searching comparison of the claimed invention – *including all its limitations* – with the teaching of the prior art." *In re Ochiai*, 71 F.3d 1565, 1572 (Fed. Cir. 1995) (emphasis added). Thus, "obviousness requires a suggestion of all limitations in a claim." *CFMT, Inc. v. Yieldup Int'l. Corp.*, 349 F.3d 1333, 1342 (Fed. Cir. 2003) (citing *In re Royka*, 490 F.2d 981, 985 (CCPA 1974)). Furthermore, as the Supreme Court recently stated, "*there must be some articulated reasoning with some rational underpinning to support the legal conclusion of obviousness.*" *KSR Int'l v. Teleflex Inc.*, 550 U.S. 398, 418 (2007) (quoting *In re Kahn*, 441 F.3d 977, 988 (Fed. Cir. 2006) (emphasis added)).

Here, the Examiner concluded that it would have been obvious to combine the teachings of Refsum with Kass, Wilcken, and Westhuyzen "[b]ased on the correlation between the elevated levels of homocysteine (free and/or complexed) in blood and urine samples with deficiency of cobalamin or folate taught by Kass, Wilcken ... and Westhuyzen" (Ans. 7-8). We disagree.

The claimed invention requires assaying for "total" homocysteine, which consists of four species (FF 2), and correlating an elevated total homocysteine level with a deficiency of vitamin B₁₂ and/or folate, without distinguishing between either type of deficiency (*see* FF 1). Kass teaches a nickel chloride test (i) which may not be measuring only homocysteine (but

also coenzyme A), let alone "total" homocysteine, and (ii) which, contrary to the claimed method, distinguishes between a vitamin B₁₂ deficiency and a folate deficiency (FFs 5-7). Indeed, the Examiner refuses to acknowledge the possibility that Kass may not have been measuring just homocysteine (Ans. 16). Furthermore, one of ordinary skill in the art would not have reasonably read Wilcken as teaching a correlation between an elevated total homocysteine level and either folate or B₁₂ deficiency. Specifically, one of ordinary skill in the art would not have considered the second twin to be folate deficient based on his serum folate level for two reasons. First, the second twin's RBC folate level was normal and RBC folate levels are art recognized as providing an accurate index of folate stores. Secondly, serum folate levels are labile, vary with diet, and art recognized as potentially misleading. [FFs 8-9.] Similarly, one of ordinary skill in the art would not have considered the first twin to be vitamin B₁₂ deficient based on a B₁₂ level of 144 pg/ml because the art defines a serum vitamin B₁₂ level less than 100 pg/ml as indicative of B₁₂ deficiency (FFs 8 and 10). While Westhuyzen fairly suggests a correlation between an elevated plasma homocysteine level and vitamin B₁₂ deficiency (FF 11 and 13), homocysteine is only a part of "total" homocysteine and its increase does not necessarily mean an increase in "total" homocysteine (FF 2). In particular, since Westhuyzen precipitated the plasma proteins with acid prior to determining the amino acid levels in the plasma (FF 12), Westhuyzen coprecipitated the protein-bound homocysteine (FF 2). Thus, Westhuyzen did not determine total homocysteine levels. The Examiner has made no findings which provide a factual basis for extrapolating "total homocysteine"

levels from one or more of the forms which comprise it, especially in view of the known redistribution between free and bound forms of homocysteine (FF 2). Moreover, none of Kass, Wilcken, or Westhuyzen fairly teach or suggest a correlation between an elevated total homocysteine level and a folate deficiency. In short, the Examiner has failed to provide a sufficient factual basis to support her conclusion of obviousness, i.e., that the applied prior art teaches or suggests all of the claim limitations.

Therefore, we reverse the rejection of claims 13, 15-17, and 33 under § 103(a) over the combined teachings of Refsum, Kass, Wilcken, and Westhuyzen.

IV. Order

Upon consideration of the record, and for the reasons given, it is ORDERED that the decision of the Examiner to reject claims 13, 15-17, and 33 as unpatentable under 35 U.S.C. § 103(a) over Refsum in view of Kass, Wilcken, and Westhuyzen is REVERSED, and

FURTHER ORDERED that requests for extending time for taking any subsequent action in connection with this appeal are governed by 37 C.F.R. § 1.550(c).

REVERSED

sss

Patent Owner

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Appeal 2009-005519

Reexamination Control 90/008,305

Patent 4,940,658

cc: Third Party Requester

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