

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

QIAGEN INC.,
Petitioner,

v.

OXFORD IMMUNOTEC LTD.,
Patent Owner.

Case IPR2016-01575
Patent 8,617,821 B2

Before SHERIDAN K. SNEDDEN, SUSAN L. C. MITCHELL, and
J. JOHN LEE, *Administrative Patent Judges*.

LEE, *Administrative Patent Judge*.

DECISION

Denying Institution of *Inter Partes* Review
35 U.S.C. § 314(a) and 37 C.F.R. § 42.108

INTRODUCTION

On August 9, 2016, Petitioner QIAGEN Inc. filed a Petition (Paper 2, “Pet.”) seeking *inter partes* review of claims 1–9 (“the challenged claims”) of U.S. Patent No. 8,617,821 B2 (Ex. 1001, “the ’821 Patent”). Patent Owner Oxford Immunotec Limited timely filed a Preliminary Response (Paper 6, “Prelim. Resp.”).

We have authority to institute an *inter partes* review only if the information presented in the Petition shows “there is a reasonable likelihood that the petitioner would prevail with respect to at least 1 of the claims challenged in the petition.” 35 U.S.C. § 314; 37 C.F.R. §§ 42.4(a), 42.108. Upon consideration of the Petition and Preliminary Response, we determine that the information presented does not show there is a reasonable likelihood that Petitioner would prevail in establishing the unpatentability of any of the challenged claims. Accordingly, pursuant to § 314, we do not institute an *inter partes* review of the challenged claims of the ’821 Patent, and the Petition is *denied*.

A. *Related Cases*

The parties identify as related to the present case *Oxford Immunotec Ltd. v. Qiagen N.V.*, Case No. 15-cv-13124-NMG (D. Mass.). Pet. 1–2; Paper 5, 2.

B. *The ’821 Patent*

The ’821 Patent relates to an assay for detecting “activated peptide-specific T cells.” Ex. 1001, 1:23–24. The T cells are “pre-sensitised in vivo to a particular peptide.” *Id.* at 1:56–58. These are “unrestimulated T-cells, i.e.,] cells capable of immediate effector function without the need to effect

division/differentiation by in vitro culture.” *Id.* at 2:13–16. The T cells are exposed to the peptide *in vitro* to stimulate the release of cytokines, preferably interferon- γ (IFN- γ), which are detected by the assay. *Id.* at 1:59–63, 2:16–19. After stimulation, the cells are incubated “for a time sufficient to permit CD8+ [T cells] that have been pre-sensitised in vivo to the particular peptide chosen to secrete the IFN- γ ,” but not “so long that quiescent CD8+ cells have time to differentiate and become activated by the peptide and start to secrete cytokines.” *Id.* at 2:53–58.

The specification provides several examples of applications of the claimed technique, including peptides related to antigens secreted by *Mycobacterium tuberculosis*. *Id.* at 6:50–8:35. All of the claims of the ’821 Patent recite “[a]n assay for identifying *Mycobacterium tuberculosis*-specific immediate effector T cells in a subject.” *Id.* at 13:2–3, 14:1–2.

C. Challenged Claims

Petitioner challenges all of the claims of the ’821 Patent. Claims 1 and 6 are the only independent claims. Claim 1 is illustrative and is reproduced below:

1. An assay for identifying *Mycobacterium tuberculosis*-specific immediate effector T cells in a subject, comprising:
 - (a) providing a sample from said subject containing T cells;
 - (b) exposing said T cells to an immunogenic amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope; and
 - (c) prior to the generation of new immediate effector T cells in the sample, determining whether said T cells are activated by said peptide subfragment by measuring secretion of a cytokine from said T cells;

wherein activation of said T cells identifies the presence of *Mycobacterium tuberculosis*-specific immediate effector T cells that were present in the original sample, in said subject.

D. Asserted Grounds of Unpatentability

Petitioner asserts the following grounds of unpatentability (Pet. 4):

References	Basis	Claims Challenged
Brandt-1996 ¹	§§ 102(a), 102(b)	1, 3–8
Pollock-July-1997 ²	§ 102(a)	1–8

¹ Lise Brandt et al., *Key Epitopes on the ESAT-6 Antigen Recognized in Mice During the Recall of Protective Immunity to Mycobacterium tuberculosis*, 157 J. IMMUNOLOGY 3527 (1996) (Ex. 1010, “Brandt-1996”).

² John M. Pollock & Peter Andersen, *Predominant Recognition of the ESAT-6 Protein in the First Phase of Infection with Mycobacterium bovis in Cattle*, 65 INFECTION & IMMUNITY 2587 (1997) (Ex. 1024, “Pollock-July-1997”).

References	Basis	Claims Challenged
Mukherjee-1995, ³ Brandt-1996, and (Harboe-1996 ⁴ and/or Vordermeier-1995 ⁵ and/or Pollock-May-1997 ⁶)	§ 103	1–9
Rothel-1990, ⁷ Brandt-1996, and (Harboe-1996 and/or Vordermeier-1995 and/or Pollock-May-1997)	§ 103	1–9
Lalvani-1997, ⁸ Brandt-1996, and (Harboe-1996 and/or Vordermeier-1995 and/or Pollock-May-1997)	§ 103	1–9

In addition to the above references, Petitioner also relies on the Declaration of Joel D. Ernst, M.D. (Ex. 1004).

³ Sutapa Mukherjee et al., *Evaluation of Gamma Interferon (IFN- γ) Assay in Human Mycobacterial Infection*, Abstracts Presented at the Ann. Sci. Meeting of the Thoracic Soc’y of Austl. and N. Z., Mar. 1995, at 436 (Ex. 1018, “Mukherjee-1995”).

⁴ Morten Harboe et al., *Evidence for Occurrence of the ESAT-6 Protein in Mycobacterium tuberculosis and Virulent Mycobacterium bovis and for Its Absence in Mycobacterium bovis BCG*, 64 INFECTION & IMMUNITY 16 (1996) (Ex. 1014, “Harboe-1996”).

⁵ H. M. Vordermeier, *T-cell recognition of mycobacterial antigens*, 8 EUR. RESPIRATORY J. 657s (1995) (Ex. 1032, “Vordermeier-1995”).

⁶ John M. Pollock & Peter Andersen, *The Potential of the ESAT-6 Antigen Secreted by Virulent Mycobacteria for Specific Diagnosis of Tuberculosis*, 175 J. INFECTIOUS DISEASES 1251 (1997) (Ex. 1012, “Pollock-May-1997”).

⁷ J. S. Rothel et al., *A sandwich enzyme immunoassay for bovine interferon- γ and its use for the detection of tuberculosis in cattle*, 67 AUSTL. VETERINARY J. 134 (1990) (Ex. 1016, “Rothel-1990”).

⁸ Ajit Lalvani et al., *Rapid Effector Function in CD8+ Memory T Cells*, 186 J. EXPERIMENTAL MED. 859 (1997) (Ex. 1029, “Lalvani-1997”).

ANALYSIS

A. *Claim Construction*

In an *inter partes* review, claim terms in an unexpired patent are given their broadest reasonable construction in light of the specification of the patent in which they appear. 37 C.F.R. § 42.100(b); *see Cuozzo Speed Techs., LLC v. Lee*, 136 S. Ct. 2131, 2144–46 (2016). The parties dispute whether the claim term “contains a CD8+ epitope” in the phrase “exposing said T cells to an immunogenic amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope” is printed matter that should be given no patentable weight. *See* Pet. 15; Prelim. Resp. 38–39. This limitation is recited in both independent claims. For the reasons explained below, we agree with Patent Owner that the term should be given patentable weight. No other claim terms require express construction for purposes of this Decision. *See Vivid Techs., Inc. v. Am. Sci. & Eng'g, Inc.*, 200 F.3d 795, 803 (Fed. Cir. 1999).

A limitation that claims printed matter, which is not functionally or structurally related to the physical substrate holding the printed matter, may not be given patentable weight. *In re DiStefano*, 808 F.3d 845, 848 (Fed. Cir. 2015). “[A] limitation is printed matter only if it claims the content of information.” *Id.* If determined to claim printed matter, the limitation is nonetheless given patentable weight “if the claimed informational content has a functional or structural relation to the substrate.” *Id.* at 851.

According to Petitioner, the “CD8+ epitope” limitation claims printed matter because it is merely the content of information, namely “the string of amino acids in a subfragment of ESAT-6 that includes the amino acids that constitute a CD8+ epitope.” Pet. 15. Petitioner likens the “CD8+ epitope”

to an “instruction limitation” that merely “informs” about the epitope in ESAT-6. *Id.* (citing *In re Kao*, 639 F.3d 1057, 1072–73 (Fed. Cir. 2011)). We disagree. In *Kao*, the relevant limitation claimed “providing information” regarding a particular biological correlation, which the Federal Circuit concluded had no functional relationship to the following step of “providing” a particular drug treatment. *Kao*, 639 F.3d at 1072–73. Here, the relevant limitation is “exposing said T cells to an immunogenic amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope.” The epitope is not “printed” onto a physical substrate (i.e., the peptide); it is *part of* the substrate—the epitope is a sequence of amino acids that is recognized by a CD8+ T cell, and those amino acids are part of the amino acids that make up the peptide itself.

Moreover, the epitope does not merely inform someone of purely informational content. Rather, the epitope has a structural and functional relationship to the claims and to the substrate (i.e., the peptide subfragments). For example, claim 1 recites, “exposing said T cells to an *immunogenic* amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope” (emphasis added). The claim further recites, “measuring secretion of a cytokine from said T cells” to determine “whether said T cells are activated by said peptide subfragment.” The specification describes the activation of CD8+ T cells by peptide subfragments that are CD8+ epitopes. *See* Ex. 1001, 2:30–45. Thus, the CD8+ epitopes are involved in the activation of T cells, which yields immunogenicity (i.e., generation of an immune system response) in the form of cytokine secretion, which is measured as part of the claimed method. The CD8+ epitope limitation is more akin to the attribute data object (ADO) limitations in *In re Lowry*, 32

F.3d 1579, 1583–84 (Fed. Cir. 1994), where the recited ADOs were processed by a computer and affected how data was stored and organized in a memory. Thus, the CD8+ epitope limitation does not constitute printed matter with no patentable weight. *See DiStefano*, 808 F.3d at 851.

B. Priority Date

The '821 Patent claims priority to a PCT application filed on November 25, 1997, which itself claims priority to a British patent application filed one year earlier. Ex. 1001, at [63], [30]. Petitioner alleges the '821 Patent is not entitled to claim priority to the 1996 British application. We need not decide the priority date for the '821 Patent because we determine all the asserted grounds fail for reasons independent of the priority date, as explained below.

C. Alleged Anticipation by Brandt-1996

To anticipate a claim, a single prior art reference must disclose each and every limitation of the claim, either expressly or inherently. *Verdegaal Bros., Inc. v. Union Oil Co. of Cal.*, 814 F.2d 628, 631 (Fed. Cir. 1987). A feature is disclosed inherently if it is *necessarily* present in the single anticipating reference. *Continental Can Co. USA, Inc. v. Monsanto Co.*, 948 F.2d 1264, 1268 (Fed. Cir. 1991).

Petitioner contends claims 1 and 3–8 are anticipated by Brandt-1996. Pet. 22–34. The Petition does not, however, demonstrate sufficiently that Brandt-1996 discloses, “exposing said T cells to an immunogenic amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope,” which is recited in both independent claims.

Brandt-1996 discloses the results of a study in which murine (i.e., mouse) T cells were exposed to whole ESAT-6 protein, as well as nine subfragments of ESAT-6. Ex. 1010, 3528, Fig. 3. Whole ESAT-6 protein, as well as two of the subfragments, yielded a response from the T cells in the form of significant IFN- γ release. *See id.* at 3529–31, Figs. 2, 4.

Petitioner argues that Brandt-1996 discloses exposing T cells to an immunogenic amount of a peptide subfragment of ESAT-6 because it reports a positive response (i.e., heightened IFN- γ release) to the “p1-20” peptide subfragment of ESAT-6 (also referred to as the “p1” peptide). Pet. 22–23 (citing Ex. 1010, 3531, Table 2). The Petition does not explain, however, why exposing the mice in Brandt-1996 to the “p1-20” peptide subfragment of ESAT-6 necessarily exposed the T cells to an immunogenic amount of peptide *containing a CD8+ epitope*. Petitioner’s assertion that a CD8+ epitope is not required by the claims is based on its arguments regarding printed matter, which are unpersuasive as discussed above.

Alternatively, Petitioner argues that whole ESAT-6 protein contains CD8+ epitopes and, thus, the “ESAT-6 fragments disclosed in Brandt-1996 necessarily constitute ESAT-6 subfragments that contain a CD8+ epitope.” Pet. 23. Only two of the subfragments tested in Brandt-1996 were *immunogenic*, however, as required by the challenged claims. *See* Ex. 1010, Fig. 4 (showing responses to the “p1” (amino acids 1-20) and “p6” (amino acids 51-70) subfragments). Petitioner asserts that amino acids 15–29 and 61–82 correspond to murine CD8+ epitopes, but neither of them correspond to the immunogenic subfragments disclosed in Brandt-1996. *See* Pet. 23 (citing Ex. 1008, 3348 (“Bennekov-2006”)). Although Petitioner also asserts that ESAT-6 contains human and bovine CD8+ epitopes (*id.*),

Brandt-1996 only tested mice and, thus, does not disclose immunogenicity for these epitopes specific to other species.

Finally, Petitioner argues Brandt-1996 shows that whole ESAT-6 protein is immunogenic, which inherently discloses an immunogenic amount of subfragments containing CD8+ epitopes because ESAT-6 protein necessarily is processed into subfragments. Pet. 24. As Patent Owner argues, however, the record indicates that exposure to ESAT-6 protein does *not* necessarily produce a CD8+ response, particularly when a non-viral vector is used. *See* Prelim. Resp. 55–57. For example, Bennekov-2006 (cited by Petitioner as disclosing murine CD8+ epitopes) reports a response from CD8+ T cells of mice exposed to ESAT-6 protein via an adenovirus vector, but *no* significant CD8+ response when a liposome vector was used. *See* Ex. 1008, 3348, Fig. 2. It is undisputed that the Brandt-1996 study did not use a viral vector. Despite citing Bennekov-2006 to support its inherency arguments, Petitioner does not address or explain these aspects of Bennekov-2006, or their bearing on the disclosures of Brandt-1996.

For the above reasons, Petitioner has not provided sufficient information showing that Brandt-1996 discloses, explicitly or inherently, “exposing said T cells to an immunogenic amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope,” as required by the challenged claims. Thus, Petitioner has not demonstrated a reasonable likelihood of prevailing as to anticipation by Brandt-1996.

D. Alleged Anticipation by Pollock-July-1997

Petitioner contends claims 1–8 are anticipated by Pollock-July-1997. Pet. 34–41. Similar to the anticipation ground based on Brandt-1996, however, the Petition does not demonstrate sufficiently that Pollock-July-

1997 discloses, “exposing said T cells to an immunogenic amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope.”

Pollock-July-1997 discloses the results of a study examining the T cell response of cattle infected with *Mycobacterium bovis*, the cause of bovine tuberculosis. *See* Ex. 1024, 2587. The study involved exposing bovine peripheral blood mononuclear cells (PBMCs), including T cells, to fractions of a mixture of *M. tuberculosis* antigen proteins (“short-term culture filtrate” or “ST-CF”) as well as purified ESAT-6 protein. *Id.* at 2589–90. Based on the results of the study, Pollock-July-1997 concludes that ESAT-6 protein is the dominant antigen causing IFN- γ release during the early stages of tuberculosis infection in cattle. *See id.* at 2589–91. Critically, the Petition does not allege that Pollock-July-1997 explicitly discloses exposing T cells to *subfragments* of ESAT-6 with CD8+ epitopes. Rather, Petitioner asserts that this disclosure is inherent, relying on Vitale-2006⁹ to show that ESAT-6 includes bovine CD8+ epitopes. Pet. 34–35 (citing Ex. 1015, 532).

Vitale-2006, however, does not support a conclusion that the exposure to ESAT-6 protein disclosed in Pollock-July-1997 *necessarily* exposed those T cells to an immunogenic amount of ESAT-6 subfragments with CD8+ epitopes. In Vitale-2006, bovine PMBCs were exposed to a number of ESAT-6 subfragments. Ex. 1015, 530, 532. The results showed that only 10–36% of cows yielded a significant increase in CD8+ T cells releasing IFN- γ . *Id.* at 532, Table 1. Thus, at best, Vitale-2006 appears to indicate

⁹ Fabrizio Vitale et al., *ESAT-6 Peptide Recognition by Bovine CD8+ Lymphocytes of Naturally Infected Cows in Herds from Southern Italy*, 13 CLINICAL & VACCINE IMMUNOLOGY 530 (2006) (Ex. 1015, “Vitale-2006”).

that the ESAT-6 protein in Pollock-July-1997 had a 10–36% chance of exposing T cells to an immunogenic amount of subfragments with CD8+ epitopes, even assuming *arguendo* that exposure to whole ESAT-6 protein necessarily exposed those T cells to the subfragments later studied in Vitale-2006. “Inherency, however, may not be established by probabilities or possibilities.” *Continental Can*, 948 F.2d at 1269. The Petition does not address these aspects of Vitale-2006, or their bearing on the disclosures of Pollock-July-1997.

For the above reasons, Petitioner has not provided sufficient information showing that Pollock-July-1997 discloses, explicitly or inherently, “exposing said T cells to an immunogenic amount of a peptide subfragment of ESAT-6 that contains a CD8+ epitope,” as required by the challenged claims. Thus, Petitioner has not demonstrated a reasonable likelihood of prevailing as to anticipation by Pollock-July-1997.

E. Alleged Unpatentability Under § 103(a)

A claim is unpatentable under § 103(a) if the differences between the claimed subject matter and the prior art are “such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains.” *KSR Int’l Co. v. Teleflex Inc.*, 550 U.S. 398, 406 (2007). The question of obviousness is resolved on the basis of underlying factual determinations, including: (1) the scope and content of the prior art; (2) any differences between the claimed subject matter and the prior art; (3) the level of skill in the art; and (4) objective evidence of nonobviousness, i.e., secondary considerations. *Graham v. John Deere Co.*, 383 U.S. 1, 17–18 (1966).

Additionally, the obviousness inquiry typically requires an analysis of “whether there was an apparent reason to combine the known elements in the fashion claimed by the patent at issue.” *KSR*, 550 U.S. at 418 (citing *In re Kahn*, 441 F.3d 977, 988 (Fed. Cir. 2006) (requiring “articulated reasoning with some rational underpinning to support the legal conclusion of obviousness”)); see *In re Warsaw Orthopedic, Inc.*, 832 F.3d 1327, 1333 (Fed. Cir. 2016) (citing *DyStar Textilfarben GmbH & Co. Deutschland KG v. C. H. Patrick Co.*, 464 F.3d 1356, 1360 (Fed. Cir. 2006)).

1. Obviousness in View of Mukherjee-1995, Brandt-1996, Harboe-1996, Vordermeier-1995, and Pollock-May-1997

Petitioner contends claims 1–9 are obvious in light of the combined teachings of Mukherjee-1995, Brandt-1996, and one or more of Harboe-1996, Vordermeier-1995, and Pollock-May-1997. Pet. 41–55. Based on the evidence and arguments presented in the Petition, however, Petitioner has failed to demonstrate a reasonable likelihood of prevailing on this ground, as explained below.

Mukherjee-1995 is an abstract describing a study in which blood samples from humans previously infected with tuberculosis were exposed to PPD (purified protein derivative) of *M. tuberculosis* to measure IFN- γ release. Ex. 1018, 436. According to Petitioner, a person of ordinary skill would have known that exposure to PPD teaches exposure to ESAT-6 protein, and that exposure to ESAT-6 protein further teaches exposure to ESAT-6 subfragments because a skilled artisan would have understood that ESAT-6 protein is processed into subfragments. Pet. 42. As Petitioner acknowledges, however, Mukherjee-1995 does not teach or suggest subfragments *containing CD8+ epitopes*. See *id.*

For the CD8+ epitope limitation, Petitioner relies principally on Brandt-1996, arguing that it teaches exposure of T cells to subfragments containing CD8+ epitopes because two of the peptides tested in Brandt-1996 (i.e., the “p7” and “p8” peptides) contain human CD8+ epitopes. *Id.* at 43. This argument is unpersuasive for multiple reasons. First, Brandt-1996 does not itself address CD8+ epitopes, or indicate that the “p7” and “p8” peptides contain CD8+ epitopes. Petitioner instead cites a 1998 article (“Lalvani-1998”) as allegedly identifying CD8+ epitopes contained within those peptides (Pet. 43), but that article is *not prior art* and, thus, cannot prove what was taught or suggested at the time of the invention of the challenged claims. The only other evidence cited by the Petition on this point is the testimony of Dr. Ernst, who himself cites only the same 1998 article to support his testimony. *See* Ex. 1004 ¶¶ 99, 101.

Second, the Brandt-1996 experiments with the “p7” and “p8” peptide subfragments of ESAT-6 do not teach an *immunogenic* amount of either peptide because Brandt-1996 did not report any significant T cell response, much less a CD8+ T cell response specifically, for those two peptides. *See* Ex. 1010, Fig. 4 (showing T cell responses to the “p1” and “p6” peptides only). To the extent Petitioner intended to argue that Brandt-1996 teaches the CD8+ epitope limitation because CD8+ epitopes are *inherent* in the “p7” and “p8” peptides (as allegedly shown by Lalvani-1998), such an argument fails for essentially the same reason—Brandt-1996 actually indicates that neither peptide produced a significant T cell response that would indicate any recognized epitope, including any CD8+ epitope. Indeed, this is unsurprising because the alleged CD8+ epitopes in the “p7” and “p8” peptides are *human* epitopes, whereas Brandt-1996 involved tests

exclusively on *mice*. *See* Ex. 1010, 3527–28. Petitioner does not explain sufficiently why a person of ordinary skill would have understood Brandt-1996 to teach exposing T cells to immunogenic amounts of ESAT-6 subfragments with human CD8+ epitopes, when Brandt-1996 did not involve human T cells that would recognize such epitopes and, in any event, did not report that those subfragments were immunogenic.

Petitioner's remaining arguments as to the CD8+ limitation of the challenged claims are also unavailing. As already discussed, Petitioner's argument that the CD8+ limitation is entitled to no patentable weight as printed matter is unpersuasive. *See* Pet. 43. Additionally, Petitioner's argument that Mukherjee-1995 itself "inherently" teaches exposing T cells to subfragments with CD8+ epitopes is unpersuasive because it relies on the same reasoning as for its anticipation grounds, i.e., that exposing T cells to whole ESAT-6 protein necessarily exposes those T cells to subfragments containing CD8+ epitopes. *See id.* As discussed above, the evidence of record appears to undermine such a conclusion, and Petitioner does not explain sufficiently why a person of ordinary skill nonetheless would have understood Mukherjee-1995 to supply such a teaching. Further, Petitioner's reliance on statements made by a named inventor of the '821 Patent (Pet. 48–49) is unpersuasive in light of the deficiencies discussed above, particularly because, as Patent Owner notes, record evidence (including Petitioner's asserted prior art itself) belies those statements. *See* Prelim. Resp. 36 (citing, e.g., Ex. 1010, 3532).

The Petition does not cite Harboe-1996, Vordermeier-1995, or Pollock-May-1997 as teaching the CD8+ epitope limitation of the challenged claims. *See id.* at 45–48. Therefore, Petitioner has not

sufficiently demonstrated that the asserted prior art teaches or suggests each limitation of the challenged claims. Consequently, Petitioner has not shown a reasonable likelihood of prevailing as to this ground of unpatentability.

2. Remaining Obviousness Grounds

Petitioner's remaining obviousness grounds are similar to its asserted ground based on Mukherjee-1995, Brandt-1996, and one or more of Harboe-1996, Vordermeier-1995, and Pollock-May-1997. Specifically, Petitioner asserts two other obviousness grounds which replace Mukherjee-1995 with Rothel-1990 or Lalvani-1997, respectively. *See* Pet. 55–73. Similar to Mukherjee-1995, Rothel-1990 describes a study in which blood samples of subjects (cattle instead of humans) were exposed to PPD to detect IFN- γ release. *See* Ex. 1016, 134. Lalvani-1997 describes a study in which CD8+ T cells sensitive to influenza virus infection were identified using certain peptides related to the influenza virus. *See* Ex. 1029, 859–60.

Like Mukherjee-1995, neither Rothel-1990 nor Lalvani-1997 teach exposing T cells to immunogenic amounts of ESAT-6 subfragments containing CD8+ epitopes. Petitioner relies on essentially the same arguments regarding that limitation, including those based on Brandt-1996, for all of its asserted obviousness grounds. *See* Pet. 42–49, 56–58, 66–67. For similar reasons as those discussed above for the asserted ground based on Mukherjee-1995, these arguments also are unpersuasive for Petitioner's remaining obviousness grounds. Therefore, Petitioner has not sufficiently demonstrated that the asserted prior art teaches or suggests each limitation of the challenged claims. Consequently, Petitioner has not shown a reasonable likelihood of prevailing as to these grounds of unpatentability.

CONCLUSION

For the foregoing reasons and on the present record, we determine that the information presented in the Petition does not demonstrate there is a reasonable likelihood that Petitioner would prevail in establishing the unpatentability of claims 1–9 of the '821 Patent.

ORDER

It is

ORDERED that the Petition is *denied*.

IPR2016-01575
Patent 8,617,821 B2

PETITIONER:

Judy M. Mohr
Bhanu K. Sadasivan
MCDERMOTT WILL & EMERY LLP
jmohr@mwe.com
bsadasivan@mwe.com

PATENT OWNER:

Steven M. Bauer
Siegmond Y. Gutman
Gerald Worth
PROSKAUER ROSE LLP
ptabmatterboston@proskauer.com
bi-uspto-comm@proskauer.com
gworth@proskauer.com