

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

Paper No. 26

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte CARL H. JUNE

Appeal No. 1999-1245
Application No. 08/245,282

ON BRIEF

Before WILLIAM F. SMITH, MILLS, and GRIMES Administrative Patent Judges.

MILLS, Administrative Patent Judge.

DECISION ON APPEAL

This is a decision on appeal under 35 U.S.C. §134 from the examiner's final rejection of claims 46-60, 85 and 86, which are all of the claims pending in this application.

We reverse.

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Claim 46 is illustrative of the claims on appeal and reads as follow:

46. A method for modulating a response by a T cell expressing a CD28 cell surface receptor which binds a costimulatory molecule, comprising contacting the T cell with an agent which acts intracellularly to modulate production of D-3 phosphoinositides in the T cell.

The prior art references relied upon by the examiner are:

Ward et al. (Ward 1993), "Ligation of CD28 receptor by B7 induces formation of D-3 phosphoinositides in T lymphocytes independently of T cell receptor/CD3 activation," European J. of Immunology, Vol. 23, pp. 2572-2577 (1993)

Vandenberghe et al. (Vandenberghe), "Antibody and B7/BB1-mediated Ligation of the CD28 Receptor Induces Tyrosine Phosphorylation in Human T Cells," The Journal of Experimental Medicine, Vol. 175, pp. 951-960 (1992)

Ward et al. (Ward 1992), "Regulation of D-3 Phosphoinositides during T Cell activation via the T cell antigen receptor/CD3 complex and CD2 antigens," European Journal of Immunology, Vol. 22, pp. 45-49 (1992)

Okada et al (Okada), "Essential Role of Phosphatidylinositol 3-Kinase in Insulin-Induced Glucose Transport and Antilipolysis in Rat Adipocytes," The Journal of Biological Chemistry, Vol. 269, No. 5, pp. 3568-3573 (1994)

Reference relied on by appellant:

Sato et al. (Sato), "Effects of Wortmannin Analogs on Bone in Vitro and in Vivo," The Journal of Pharmacology and Experimental Therapeutics, Vol. 277, pp. 543-550 (1996)

References cited by the Merits Panel:

Bonjouklian et al (Bonjouklian 1)	5,378,725	Jan. 3, 1995 (Filed Jul. 19, 1993)
Bonjouklian et al (Bonjouklian 2)	5,507,103	April 2, 1996 (Filed Aug. 25, 1993)

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Creemer et al (Creemer)

5,480,906

Jan. 2, 1996
(Filed July 1, 1994)

Weisinger et al (Weisinger), "Antiinflammatory Activity of the New Mould Metabolite 11-Desacetoxy-Wortmannin and of Some of its Derivatives," Experientia, Vol. 30, pp. 135-136 (1974)

Grounds of Rejection

Claims 46-60, 85 and 86 stand rejected under 35 U.S.C. § 112, first paragraph as unpatentable for lack of enablement as to how to make and use the claimed invention.

Claims 46-48, 50-60, 85 and 86 stand rejected under 35 U.S.C. § 103 as unpatentable for obviousness over Ward 1993 in view of Vandenberghe and Ward 1992.

Claim 49 stand rejected under 35 U.S.C. § 103 as unpatentable for obviousness over Ward 1993 in view of Vandenberghe, Ward 1992 and Okada. We reverse each of the pending rejections.

DISCUSSION

In reaching our decision in this appeal, we have given careful consideration to the appellant's specification and claims, to the applied prior art references, and to the respective positions articulated by the appellant and the examiner.

Rather than reiterate the conflicting viewpoints advanced by the examiner and the appellant regarding the noted rejections, we make reference to the examiner's Answer for the examiner's reasoning in support of the rejection, and to the appellant's Brief and Reply Brief for the appellant's arguments thereagainst. As a consequence of our review, we make the determinations which follow.

Background

According to the specification, the method of the invention is based, at least in part, "on the discovery that stimulation of a T cell through the CD28 surface receptor leads to the production of D-3 phosphoinositides in a T cell and that an inhibitor of phosphatidylinositol 3-kinase (also referred to herein as PI3K) inhibits production of D-3 phosphoinositides in the T cell upon CD28 ligation. The invention is further based, at least in part, on the discovery that inhibition of PI3K activity in a T cell inhibits T cell responses, such as cytokine production and cellular proliferation." Specification, page 5. "Accordingly, one aspect of the invention pertains to methods for inhibiting a response by a T cell by interfering with intracellular signal transduction associated with signal transduction. ... [A]n intracellular signal is inhibited by contacting a T cell expressing a cell surface receptor that binds a costimulatory molecule with an agent which inhibits production of D-3 phosphoinositides in the T cell." Id. D-3 phosphoinositides are generated intracellularly by the activity of a phosphatidylinositol 3

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kinase (PI3K). A preferred agent which inhibits PI3K activity in a T cell is the fungal metabolite wortmannin. Other agents include the bioflavonoid, quercetin, and the compound LY294002. Specification, page 6.

Alternatively, a T cell may be contacted both with an agent which inhibits PI3K activity and with an agent which inhibits protein tyrosine kinase activity, such as herbimycin A, or a derivative or analogue thereof. Id. The method of the invention is applicable to the treatment of autoimmune disease and other disorders associated with an abnormal immune response. Specification, page 7.

35 U.S.C. § 112, first paragraph

Claims 46-60, 85 and 86 stand rejected under 35 U.S.C. § 112, first paragraph as unpatentable for lack of enablement as to how to make and use the claimed invention. We note that the term “modulate” as it appears in the claims is interpreted by the examiner to mean “inhibit”, in accordance with a restriction requirement and subsequent election made on September 14, 1995 (Paper No. 6) and December 8, 1995 (Paper No. 8). Answer, pages 5-6. For the purposes of this appeal, we adhere to the examiner’s interpretation of the term “modulate.”

In order to establish a prima facie case of non-enablement, the examiner must provide a reasonable explanation as to why the scope of protection provided by a claim is not adequately enabled by the disclosure. See In re Wright, 999 F.2d 1557,

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1561-62, 27 USPQ2d 1510, 1513 (Fed. Cir. 1993). The threshold step in resolving this issue is to determine whether the examiner has met his burden of proof by advancing acceptable reasoning inconsistent with enablement.

Factors to be considered by the examiner in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman, 230 USPQ 546, 547 (Bd. Pat. App. & Int. 1986). They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims. In re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988). We note that all of the factors need not be reviewed when determining whether a disclosure is enabling. Amgen, Inc. v. Chugai Pharm. Co., 927 F.2d 1200, 1213, 18 USPQ2d 1016, 1027 (Fed. Cir. 1991) (noting that the Wands factors "are illustrative, not mandatory. What is relevant depends on the facts.").

In this regard, the following passage from PPG Indus. Inc. v. Guardian Indus. Corp., 75 F.3d 1558, 1564, 37 USPQ2d 1618, 1623 (Fed. Cir. 1996) is instructive here.

In unpredictable art areas, this court has refused to find broad generic claims enabled by specifications that demonstrate the enablement of only one or a few embodiments and do not demonstrate with reasonable specificity how to make and use other potential embodiments across the full scope of the claim. See, e.g., In re Goodman, 11 F.3d 1046, 1050-52, 29 USPQ2d 2010, 2013-15 (Fed. Cir. 1993); Amgen, Inc. v. Chugai

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Pharmaceutical Co., 927 F.2d 1200, 1212-14, 18 USPQ2d 1016, 1026-28 (Fed. Cir.), cert. denied, 502 U.S. 856 (1991); In re Vaeck, 947 F.2d at 496, 20 USPQ2d at 1445. Enablement is lacking in those cases, the court has explained, because the undescribed embodiments cannot be made, based on the disclosure in the specification, without undue experimentation. But the question of undue experimentation is a matter of degree. The fact that some experimentation is necessary does not preclude enablement; what is required is that the amount of experimentation “must not be unduly extensive.” Atlas Powder Co., v. E.I. DuPont De Nemours & Co., 750 F.2d 1569, 1576, 224 USPQ 409, 413 (Fed. Cir. 1984). The Patent and Trademark Office Board of Appeals summarized the point well when it stated:

The test is not merely quantitative, since a considerable amount of experimentation is permissible, if it is merely routine, or if the specification in question provides a reasonable amount of guidance with respect to the direction in which the experimentation should proceed to enable the determination of how to practice a desired embodiment of the invention claimed.

Ex parte Jackson, 217 USPQ 804, 807 (1982).

In the present case, it is the examiner’s position that the specification, while being enabling for in vitro methods of inhibiting D-3 phosphoinositides in T-cells, does not reasonably provide enablement for in vivo methods of inhibiting D-3 phosphoinositides. The examiner argues that the specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims. Answer, page 6.

The examiner finds that claim 46, when read in light of the specification, clearly reads on in vitro, ex vivo and in vivo methods of modulation (see specification page 7,

lines 23-35, for example). Answer, page 5. According to the examiner (Answer, page 6):

Appellant's claimed invention recites the "contacting of the T cell with an agent which acts intracellularly" to inhibit the production of D-3 phosphoinositides. Thus, the claimed invention encompasses any and all agents which can act intracellularly to inhibit production of D-3 phosphoinositides. These agents include wortmannin, quercetin, and herbimycin A (recited in the specification), for example, but also include antisense to D-3 phosphoinositides, or vectors encoding antisense transcripts to D-3 phosphoinositides. Appellant's specification provides absolutely no guidance to the skilled artisan on appropriate DNA or RNA sequences which may even be potentially used as the latter agents.

Thus, the examiner concludes that (Answer, page 7):

... the specification fails to provide a single working example for the numerous claim *in vivo* embodiments of the claimed invention. Therefore, in view of the quantity of experimentation necessary to determine the parameters listed above, the lack of direction or guidance provided by the specification, the absence of working examples for *in vivo* intracellular T cell modulation, the breadth of the claims, and the unpredictable and undeveloped state of the art with respect to *in vivo* cell intracellular transformation with any and all agents, it would have required undue experimentation for one skilled in the art to practice the claimed invention.

In our view the examiner has failed to provide sufficient evidence to support a finding of lack of enablement of the claimed invention. In re Mills, 470 F.2d 649, 651, 176 USPQ 196, 198 (CCPA 1972) holds that "[a]ll the disclosures in a reference must be evaluated . . . a reference is not limited to the disclosure of specific working examples." For example, the specification, pages 11-13, provides detailed disclosure regarding screening assays for identifying inhibitors and activators of PI3K which can

then be used to inhibit or stimulate T cell responses. Such screening assays, once identified, can be routinely used by those of ordinary skill in the art to screen compounds for activity. The examiner has advanced no specific reasoning as to why the compound screening assays outlined in the specification would be unsuccessful in identifying other similar agents, which can act as inhibitors of PI3K.

In addition, appellant argues, citing examples 2 and 5 of the specification, that appellant's specification shows that treatment of T-cells according to the methods of the invention (e.g., using a phosphatidylinositol 3-kinase inhibitor) inhibits T-cell responses such as the production of D-3 phosphatidylinositides or cytokine product, e.g., interleukin-2, and cell proliferation induced by CD28 ligation in vitro. Brief, page 9. More specifically, the specification teaches methods for modulating T-cell response in, for example, a subject suffering from an autoimmune disease or other disorder associated with an abnormal immune response, or a transplant recipient. Such methods as taught in the specification starting on page 8, line 31, include the administration of an agent which modulates production of D-3 phosphoinositides at a dose for a period of time sufficient to induce T-cell unresponsiveness. Brief, page 9.

It is appellant's position that successful in vitro testing for a particular inhibitory/therapeutic activity in an accepted in vivo model establishes a significant probability that in vivo testing for this particular inhibitory and/or therapeutic activity will be successful. Appellant offers Sato, a publication after his filing date, as evidence that

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wortmannin can inhibit PI3K in vivo. Brief, pages 9-10. However, patents are written to enable those skilled in the art to practice the invention, and Section 112 speaks as of the application filing date. W.L. Gore & Assocs. v. Garlock, Inc., 721 F.2d 1540, 1558, 220 USPQ 303, 316 (Fed. Cir. 1983). Moreover, the Court stated in In re Glass¹,

If a disclosure is insufficient as of the time it is filed, can it be made sufficient, while the application is still pending, by later publications which add to the knowledge of the art so that the disclosure, supplemented by such publications, would suffice to enable the practice of the invention? We think it cannot....that application sufficiency under § 112, first paragraph, must be judged as of its filing date. It is an applicant's obligation to supply enabling disclosure without reliance on what others may publish after he has filed an application on what is supposed to be a completed invention.

In addition in Enzo Biochem, Inc. v. Calgene, Inc., 188 F.3d 1362, 1376, 52 USPQ2d 1129, 1139 (Fed. Cir. 1999), the Court held that the fact that persons skilled in the art are able to practice the invention by the exercise of substantial experimentation well beyond the broad concepts that appear in the specifications is not probative of enablement. In the present case, appellant has failed to show that the alleged post-filing success of Sato were accomplished by following the teachings of the specification. Accordingly, in light of Glass and Enzo, we believe that appellant's reliance on the Sato publication is misplaced.

In spite of the appellant's misplaced reliance on the evidentiary value on Sato, we are mindful that in vitro results with respect to the particular pharmacological activity

¹ In re Glass, 492 F.2d 1228, 1232, 181 USPQ 31, 34 (CCPA 1974).

can be predictive of in vivo test results, if there is a reasonable correlation therebetween. Were this not so, the testing procedures of the pharmaceutical industry would not be as they are. It is not urged, that there is an invariable exact correlation between in vitro test results and in vivo test results. Cross v. Iizuka, 753 F.2d 1040, 224 USPQ 739 (Fed. Cir. 1985); Nelson v. Bowler, 626 F.2d 853, 856, 206 USPQ 881 (1980).

Although not explicitly stated in section 112, to be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without "undue experimentation." In re Vaeck, 947 F.2d 488, 495, 20 USPQ2d 1438, 1444 (Fed. Cir. 1991); In re Wands, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404, (Fed. Cir. 1988); In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970) (the first paragraph of section 112 requires that the scope of protection sought in a claim bear a reasonable correlation to the scope of enablement provided by the specification). Nothing more than objective enablement is required, and therefore it is irrelevant whether this teaching is provided through broad terminology or illustrative examples. In re Marzocchi, 439 F.2d 220, 223, 169 USPQ 367, 369 (CCPA 1971).

In our view, the examiner's analysis of the Wands factors on the record before us is incomplete and has focused almost exclusively on, and given undue weight to, statements of unpredictability in the art, to support the inability of the in vitro model proposed by appellant to be correlated to in vivo results. It would appear that the

examiner has failed to properly consider the state of the art in considering the enablement issue. For example, Bonjouklian 1 and 2², contemporaneous with appellant's invention, reflect that the art was aware of the ability to inhibit PI 3-kinase using wortmannin, in vivo. Bonjouklian 1, column 13, lines 42-47; column 15, lines 9-14. Weisinger describes the administration of wortmannin to rats inhibits paw edema and shows a strong antiinflammatory effect. Weisinger, pages 135-136.

While the factors relied on by the examiner are relevant in determining whether the claimed invention is enabled by the specification, we hold that, on balance, they are insufficient, in view of the state of the art, to establish a reasonable basis to doubt the objective truth of statements, screening assays and examples provided in the specification. In view of the above, the rejection of the claims for lack of enablement is reversed.

35 U.S.C. § 103

Claims 46-48, 50-60, 85 and 86 stand rejected under 35 U.S.C. § 103 as unpatentable for obviousness over Ward 1993 in view of Vandenberghe and Ward 1992. Claim 49 stands rejected under 35 U.S.C. § 103 as unpatentable for obviousness over Ward 1993 in view of Vandenberghe, Ward 1992 and Okada.

² These patents are discussed further in the section of this opinion entitled "Other Issues" herein.

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In rejecting claims under 35 U.S.C. § 103, the examiner bears the initial burden of presenting a prima facie case of obviousness. See In re Rijckaert, 9 F.3d 1531, 1532, 28 USPQ2d 1955, 1956 (Fed. Cir. 1993). It is well-established that the conclusion that the claimed subject matter is prima facie obvious must be supported by evidence, as shown by some objective teaching in the prior art or by knowledge generally available to one of ordinary skill in the art that would have led that individual to combine the relevant teachings of the references to arrive at the claimed invention. See In re Fine, 837 F.2d 1071, 1074, 5 USPQ2d 1596, 1598 (Fed. Cir. 1988). With this as background, we analyze the prior art applied by the examiner in the rejection of the claims on appeal.

According to the examiner, Ward 1993 discloses that the ligation of the CD28 receptor by B7 results in modulation of the D-3 phosphoinositides and states that the data suggest that activation of PI 3-kinase and subsequent D-3 phosphoinositides metabolism may be important signaling events in CD28 mediated costimulation and T cell activation following ligation by B7. Answer, page 8. Ward 1993 further discloses that the CD28 receptor modulates the cellular activity of PI-3 kinase (the enzyme responsible for D-3 phosphoinositides formation) and therefore D-3 phosphoinositides (see page 2573); "that the PI 3-kinase is a protein tyrosine kinase substrate and it is proposed that this tyrosine phosphorylation may regulate the activity of the catalytic

subunit of the PI 3-kinase; that tyrosine phosphorylation of numerous substrates occurs rapidly following CD28 receptor ligation.” Id.

Vandenberghe is relied on for the disclosure that when CD28+ cells are activated with antigen, for example, costimulation with CD28 monoclonal antibodies leads to enhanced production of several lymphokines such as IL-2 and increased cellular proliferation and further discloses that use of the B7/BB1 ligands gives results similar to those above obtained with CD28 monoclonal antibodies. Id. The examiner finds that the results presented in Vandenberghe teach that stimulation of Jurkat T cells and normal T cells with anti-CD28 mAb induces tyrosine phosphorylation of several substrates, and that the intracellular action of herbamycin A inhibits the CD28 induced proliferation and cytokine production by T-cells. Id.

Finally, Ward 1992 discloses that PI 3-kinase produces the D-3 phospho-
inositides and that the D-3 phosphoinositides and the PI 3-kinase may play a role in cell activation. Answer, page 9.

The examiner summarizes (Answer, page 10):

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to modify the methods of Ward (EJI, 1993) by using specific intracellular inhibitors of PI 3-kinase, such as herbimycin A as taught by Vandenberghe, in view of the teachings of both Vandenberghe and Ward (EJI, 1992) (page 45, column 2, top paragraph) that the lipid products of PI 3-kinase may play an important role in cell activation and that the D-3 phosphoinositides and PI 3-kinase have recently been discovered in activated T cells (page 45, column 1, first paragraph). One of ordinary skill in the art would have had a reasonable

expectation of success in view of the teachings of Ward et al. (EJI, 1993), using known chemical compounds, for their known intended use.

Accordingly, the modification of Ward (1993) by modulating the production of D-3 phosphoinositides by the addition of chemical agents as suggested by Vandenberghe and Ward (1992) in order to obtain a method for modulating the response of T cells expressing a CD28 cell surface receptor was within the ordinary skill in the art at the time the claimed invention was made. From the teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole is prima facie obvious, as evidenced by the references, especially in the absence of evidence to the contrary.

As to claim 49, the examiner further finds that

Okada discloses that wortmannin inhibits PI 3-kinases activity and that PI 3-kinase activity is involved in the regulation of cell proliferation. In view of the teachings of Okada regarding the ability of wortmannin to inhibit PI 3-kinases, it would have been obvious to one of ordinary skill in the art to modify the method of Ward (EJI, 1993) and Vandenberghe by the addition of wortmannin since one of ordinary skill would have the reasonable expectation that wortmannin would also inhibit PI 3-kinase activity in activated T cells, lacking evidence to the contrary.

Accordingly, the modification of the method of Ward et al. (EJI, 1993) and Vandenberghe by the addition of wortmannin as suggested by Okada in order to obtain a method of modulating the response of T cells expressing a cell surface receptor was within the ordinary skill in the art at the time the claimed invention was made. From the teachings of the references it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole is prima facie obvious.

We find the examiner has not established a prima facie case of obviousness on the record before us, or that the cited references both suggest the claimed subject

matter and reveal a reasonable expectation of success to one reasonably skilled in the art.

The examiner argues in the Final Rejection (Paper No.13, page 4), that “Vandenberghe teaches that herbimycin A inhibits a protein tyrosine kinase and one of skill would expect that inhibition of protein kinase would affect the PI 3-kinase and ultimately the D3 phosphoinositide production.” If this is the examiner’s position, it remains that the examiner establish, with appropriate evidence, that the inhibitor of tyrosine kinase described by Vandenberghe would have the same specificity, and also act on PI 3-kinase. Appellant’s specification, page 6, lines 34-35 would appear to distinguish between inhibitors of PI 3-kinase and inhibitors of tyrosine kinase activity, such as herbimycin A. However, if the examiner’s supposition is correct regarding the ability of herbimycin A to inhibit PI 3-kinase, Vandenberghe would appear to anticipate claim 1.³

At best, the statement of the rejection establishes that individual parts of the claimed invention were known in the prior art. Okada describe the role of PI3K in insulin induced glucose transport and antilipolysis in rat adipocytes but does not address T-cell activation. Ward 1993 would appear to suggest that activation of PI3K and subsequent D-3 lipid metabolism may be important signaling events in CD-28 mediated

³ Note that the specification, page 9, line 34, indicates that a phorbol ester (PMA) stimulates a T cell. Vandenberghe, page 953, column 1, would appear to suggest that herbimycin A inhibits PMA stimulation.

co-stimulation and T-cell activation following ligation by B7, but provides no reason, suggestion or motivation to inhibit such a metabolic pathway. Ward 1993 concludes that, "The possible activation of PKC ζ in T cells ... by D-3 phosphoinositides following CD28 receptor ligation and its relevance to T cell activation and co-stimulation remains to be established." Ward 1993, pages 2576-2577. Vandenberghe would appear to suggest that CD28-induced tyrosine phosphorylation can be prevented by CD45 and herbimycin A, and that herbimycin A prevents CD28-stimulated IL-2 production. Vandenberghe is particularly deficient in the discussion of any metabolic or enzymatic pathway involved in such inhibition, but would only speculate that tyrosine kinase be somehow involved in signal transduction. Vandenberghe, page 951. What is missing from the examiner's analysis is appropriate evidence establishing that the inhibitor of tyrosine kinase described by Vandenberghe would have the same specificity, and also act on PI 3-kinase. Again, a determination that claimed subject matter is prima facie obvious must be supported by evidence, as shown by some objective teaching in the prior art or by knowledge generally available to one of ordinary skill in the art that would have led that individual to combine the relevant teachings of the references to arrive at the claimed invention. See In re Fine, 837 F.2d 1071, 1074, 5 USPQ2d 1596, 1598 (Fed. Cir. 1988). In our view, the cited references do not reasonably appear to provide evidence of inhibiting a response by a T cell expressing a CD28 cell surface receptor which binds a costimulatory molecule, by contacting the T cell with an agent which acts

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intracellularly to modulate production of D-3 phosphoinositides in the T cell, e.g., such as through inhibition of PI3K.

The rejection of the claims for obviousness of the claimed invention is reversed.

Other Issues

Upon return of the application to the examiner, the Merits Panel recommends that the examiner consider the relevance of the attached patents and/or publication, Boujouklian 1 and 2, Weisinger, and Creemer, to the pending claims. In interpreting claim 1, it would appear that the claim requires a single step of contacting a T cell with an agent which acts intracellularly to modulate production of D-3 phosphoinositides in the T cell, such as wortmannin or quercetin.

Creemer, claim 17, describes a method of inhibiting PI-3 kinase in mammals comprising administering to a mammal an effective amount of a wortmannin derivative. Boujouklian 1 and 2 also describe a method of inhibiting PI-3 kinase in a mammal in need of treatment by administering wortmannin or analogs thereof. Weisinger describes that administration of wortmannin to rats inhibits an inflammatory response.

It is a general rule that merely discovering and claiming a new benefit of an old process cannot render the process again patentable. In re Woodruff, 919 F.2d 1575, 1577-78, 16 USPQ2d 1934, 1936-37 (Fed. Cir. (1990)); Bird Provision Co. v. Owens Country Sausage, Inc. 568 F.2d 369, 375, 197 USPQ 134, 139 (5th Cir. 1978); In re

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Swinehart, 439 F.2d 210, 213, 169 USPQ 226, 229 (CCPA 1971); and Ex Parte Novitski, 26 USPQ2d 1389, 1391 (Bd. Pat. App. & Int. 1993). The examiner should determine whether the methods of administering wortmannin analogs of the cited patents and/or publication describe the claimed method. The examiner should consider the relevance of the references cited herein, including the potential for interfering subject matter, to the pending claims, in view of Woodruff, etc.

In addition, the examiner should clarify and provide appropriate support for his interpretation of the term “modulate” in claim 1.

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