

The opinion in support of the decision being entered today was not written for publication and is not binding precedent of the Board.

Paper No. 16

UNITED STATES PATENT AND TRADEMARK OFFICE

**BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES**

Ex parte MICHAEL W. GRADY,
PETER J. DOYLE, and STEPHEN BLOOR

Appeal No. 2001-1499
Application No. 08/957,654

ON BRIEF

Before WINTERS, ADAMS, and GRIMES, Administrative Patent Judges.

ADAMS, Administrative Patent Judge.

GRIMES, Administrative Patent Judge dissenting in part.

DECISION ON APPEAL

This is a decision on the appeal under 35 U.S.C. § 134 from the examiner's final rejection of claims 1-3 and 5-9, which are all the claims pending in the application.

Claims 1 and 7 are illustrative of the subject matter on appeal and are reproduced below:

1. A method of treating a wound comprising applying to the wound, externally, a therapeutically effective amount of alpha-1-antitrypsin, and wherein the wound is selected from the group consisting of venous ulcers, pressure sores, decubitus ulcers and diabetic ulcers.
7. A wound dressing composition for the treatment of wounds selected from the group consisting of venous ulcers, pressure sores, decubitus

ulcers and diabetic ulcers comprising a therapeutically effective amount of alpha-1-antitrypsin.

The references relied upon by the examiner are:

Schwarz et al. (Schwarz)	4,377,572	Mar. 22, 1983
Glover et al. (Glover)	4,829,052	May 9, 1989
Lezdey John et al. (Lezdey)	5,134,119	Jul. 28, 1992
Gillis et al. (Gillis)	5,202,118	Apr. 13, 1993
Clark et al. (Clark)	WO 88/00239	Jan. 14, 1988

Rao et al. (Rao), "α1-Antitrypsin Is Degraded and Non-Functional in Chronic Wounds But Intact and Functional in Acute Wounds: The Inhibitor Protects Fibronectin from Degradation by Chronic Wound Fluid Enzymes," J. Invest. Dermatology, Vol. 105, No. 4, pp. 572-78 (1995)

GROUND OF REJECTION

Claim 7 stands rejected under 35 U.S.C. § 102(b) as anticipated by Schwarz.

Claim 7 stands rejected under 35 U.S.C. § 102(b) as anticipated by Lezdey.

Claim 9 stands rejected under 35 U.S.C. § 103 as being unpatentable over Schwarz in view of Clark.

Claims 1-3, 7 and 8 stand rejected under 35 U.S.C. § 103 as being unpatentable over Gillis.

Claims 1-3, 7 and 8 stand rejected under 35 U.S.C. § 103 as being unpatentable over Gillis in view of Rao.

Claims 5 and 9 stand rejected under 35 U.S.C. § 103 as being unpatentable over Gillis in view of Rao and further in view of Clark.

Claim 6 stands rejected under 35 U.S.C. § 103 as being unpatentable over Gillis in view of Rao and further in view of Glover.

Claim 9 stands rejected under 35 U.S.C. § 103 as being unpatentable over Lezdey in view of Clark.

We affirm the rejections over Schwarz, and Schwarz in view of Clark. We reverse all other rejections.

DISCUSSION

THE REJECTIONS UNDER 35 U.S.C. § 102:

Schwarz:

According to the examiner (Answer, page 3), Schwarz “teach a composition for use in healing wounds in which the composition can comprise 0.5-90 mg/ml alpha-1-antitrypsin [AAT]. ... An intended use limitation does not impart patentability to a composition claim in which the composition is otherwise anticipated by or obvious over the prior art.”

In response, appellants argue (Brief, page 11), “Schwarz merely lists AAT as a potential ‘other’ inhibitor from among a list of plasminogen-activator-inhibitors or plasmin-inhibitors. Absent a clear requirement that AAT is present in a composition, Schwarz cannot be said to anticipate claim 7....” To this the examiner argues (Answer, page 8), Schwarz’s “[e]xample 5 is an actual example limited to the use of AAT (see column 4, lines 36-43, and Table 1) and thus is evidence of anticipation.”

In addition, we note that appellants’ specification (page 2) discloses “the wound dressing composition is a fluid or a gel comprising from 100ng to 10mg/ml, preferably 10 µg to 1mg/ml of AAT....” As set forth in example 5 of

Schwarz, 10 mg/ml of α_1 -antitrypsin is used. Accordingly we affirm the rejection of claim 7 under 35 U.S.C. § 102(b) as anticipated by Schwarz.

Lezdey:

According to the examiner (Answer, page 6), Lezdey “teaches topical compositions comprising alpha-1-antitrypsin analogs for use in treatment of inflammatory skin conditions such as burns and atopic dermatitis.”

In response, appellants argue (Brief, page 20), “Lezdey requires, e.g., in Example III the leucine analog of α_1 -antitrypsin in an amount ... which is at least 2 orders of magnitude higher than needed to achieve an effective composition in [a]ppellants’ invention.” In response, the examiner argues (Answer, page 12), “the Brief disregards Lezdey[‘s] ... [e]xamples I and II with an approximately 1% AAT concentration, and disregards the disclosure at page 2, lines 1-3, of [a]ppellants’ specification that AAT concentrations of up to 10 mg/ml, i.e. about 1%, are preferred concentrations.”

The examiner offers no explanation for how he arrived at a value of 1%, from appellants’ disclosure (specification, page 2) of a “wound dressing composition ... from 100ng to 10mg/ml, preferably 10 μ g to 1mg/ml of ATT....” We also find no explanation from the examiner as to how any therapeutically effective amount in Lezdey would correlate to appellants’ claimed invention. Therefore, in our opinion, the examiner failed to provide the evidence necessary to establish that Lezdey anticipates appellants’ claimed invention. In this regard, we remind the examiner “[u]nder 35 U.S.C. § 102, every limitation of a claim must identically appear in a single prior art reference for it to anticipate the

claim.” Gechter v. Davidson, 116 F.3d 1454, 1457, 43 USPQ2d 1030, 1032
(Fed. Cir. 1997).

Accordingly, we reverse the rejection of claim 7 under 35 U.S.C. § 102(b)
as anticipated by Lezdey.

THE REJECTIONS UNDER 35 U.S.C. § 103:

Schwarz in view of Clark:

According to the examiner (Answer, page 3), Schwarz “teach a
composition for use in healing wounds in which the composition can comprise
0.5-90 mg/ml alpha-1-antitrypsin.” The examiner recognizes, however, (Answer,
bridging sentence, pages 3-4) that Schwarz “do not teach the use of transgenic
alpha-1-antitrypsin” as is required by appellants’ claim 9. To make up for this
deficiency the examiner relies on Clark, who teaches the transgenic production
of alpha-1-antitrypsin. Answer, page 3. According to the examiner, a person of
ordinary skill in the art at the time the invention was made would have used
Clark’s transgenic AAT because it would be expected to exhibit the inhibitory
properties desired by Schwarz, and because the transgenic source would have
been expected to be a convenient source of large amounts of AAT.¹ Answer,
page 4.

In response, appellants argue (Brief, page 12) that the examiner’s
rejection is based on hindsight reconstruction of their claimed invention, and that

¹ We note that Clark teaches (page 2) the need for a high yield, low cost process for the
production of biological substances such as correctly modified eukaryotic polypeptides, and that
the first aspect of his invention is to provide a method of producing a substance comprising a
polypeptide transgenically.

(Brief, page 13) Clark adds nothing to the shortcomings of the examiner's position concerning the broad and unfocused disclosure of Schwarz.

In response, the examiner explains (Answer, page 9), that claim 9, as it depends from claim 7, "is a composition claim, and an intended use limitation does not impart patentability to a composition claim in which the composition is otherwise ... obvious over the prior art." We agree.

As explained by the examiner (Answer, page 3), Schwarz "teach a composition for use in healing wounds in which the composition can comprise 0.5-90 mg/ml alpha-1-antitrypsin." Furthermore, example 5 of Schwarz details such a composition comprising 10mg/ml of AAT. Clark discusses a transgenic method to provide a convenient source of large amounts of medically important human proteins, including AAT. Clark, pages 1-3. In our opinion, the evidence of record weighs in favor of the examiner. Accordingly, we affirm the rejection of claim 9 under 35 U.S.C. § 103 as being unpatentable over Schwarz in view of Clark.

Gillis:

According to the examiner (Answer, page 4), Gillis "teaches treating wounds including chronic wounds such as chronic bedsores and ulcerative skin conditions by administering a composition comprising IL-1 and optionally alpha-antitrypsin." The examiner, however, recognizes (Answer, page 5), Gillis "do not teach that alpha-antitrypsin in and of itself has any intrinsic chronic wound-treating properties." Nevertheless, the examiner maintains (Answer, page 4) that

Gillis “disclose that alpha-antitrypsin would be a useful additive for ... IL-1-containing compositions.”

While the examiner argues (id.), “the instant claims contain no language which would exclude from their scope the presence of IL-1”, there can be no doubt that the claims require a “therapeutically effective amount of alpha-1-antitrypsin.” Therefore, the examiner’s finding that Gillis “do not teach that alpha-antitrypsin in and of itself has any intrinsic chronic wound-treating properties,” runs counter to finding the claimed invention obvious over Gillis. Obviousness can only be established by combining or modifying the teachings of the prior art to produce the claimed invention where there is some teaching, suggestion, or motivation to do so found either in the references themselves or in the knowledge generally available to one of ordinary skill in the art. See In re Fine, 837 F.2d 1071, 1075, 5 USPQ2d 1596, 1598 (Fed. Cir. 1988).

Accordingly, we reverse the rejection of claims 1-3, 7 and 8 under 35 U.S.C. § 103 as being unpatentable over Gillis.

Gillis in view of Rao:

According to the examiner (Answer, page 5), “[w]hile Gillis et al[.] suggest the use of alpha-antitrypsin in compositions used to treat chronic wounds, Gillis et al. do not teach that alpha-antitrypsin in and of itself has any intrinsic chronic wound-treating properties.” Therefore, the examiner relies (id.) on Rao who suggest “that the topical administration of alpha-1-antitrypsin would be useful in the treatment of chronic wounds.” In support of this position, the examiner

directs our attention to the last paragraph, on page 577 of Rao. According to Rao (page 577, last paragraph):

Intact FN [fibronectin] may be required for the healing of chronic wounds. The observations reported herein suggest that the degradation of AT precedes the degradation of FN in chronic wounds. We show here that AT [α 1-antitrypsin] precedes the degradation of FN in chronic wounds. We show here that AT effectively prevented the degradation of FN by chronic wound fluid serine proteinases. Therefore, topical AT may inhibit FN degrading enzymes and increase the concentration of intact and functional FN in chronic wounds [emphasis added].

According to appellants (Brief, page 15), “[a]t best, Rao postulates that AAT may protect fibronectin in a chronic wound.” We agree. Our dissenting colleague, however, cannot join with us because he believes that Rao’s results illustrated in Figure 6, and explained in the paragraph bridging columns 1 and 2 of page 575 are sufficient to overcome the deficiencies of Gillis. Infra, page 14. The portion of Rao, relied upon by the dissent, however, does nothing more than document the results of an in vitro study to investigate if serine proteinases are responsible for fibronectin degradation in chronic skin wounds, the stated “aim” of Rao’s report. Rao, page 572, column 2. While Rao states (id.) that a further aim of his study is to “examine the status of α 1-antitrypsin...” Rao makes no attempt to correlate his in vitro study, to an in vivo application. Instead, Rao simply suggests (page 577, column 1, last sentence) that AAT regulates fibronectin degradation in chronic wounds. In our opinion, Rao is at best an invitation to explore a promising new field of experimentation. However, as set

forth in In re O'Farrell, 853 F.2d 894, 903, 7 USPQ2d 1673, 1681 (Fed. Cir. 1988):

The admonition that “obvious to try” is not the standard under § 103 has been directed mainly at two kinds of error. In some cases, what would have been “obvious to try” would have been to vary all parameters or try each of numerous possible choices until one possibly arrived at a successful result, where the prior art gave either no indication of which parameters were critical or no direction as to which of many possible choices is likely to be successful. ... In others, what was “obvious to try” was to explore a new technology or general approach that seemed to be a promising field of experimentation, where the prior art gave only general guidance as to the particular form of the claimed invention or how to achieve it.

In our opinion, both the examiner's and the dissent's position fits the second kind of error set forth by O'Farrell, wherein the secondary references relied upon by the examiner suggest, at best, the exploration of a promising field of experimentation.

To establish a prima facie case of obviousness, there must be both some suggestion or motivation to modify the references or combine reference teachings and a reasonable expectation of success. In re Vaeck, 947 F.2d 488, 493, 20 USPQ2d 1438, 1442 (Fed. Cir. 1991). In the absence of a reasonable expectation of success one is left with only an “obvious to try” situation which is not the standard of obviousness under 35 U.S.C. § 103. See O'Farrell, supra.

In our opinion, the evidence relied upon on this record does not provide a person of ordinary skill in the art with a reasonable expectation of success in obtaining the claimed invention. Accordingly, we reverse the rejection of claims 1-3, 7 and 8 under 35 U.S.C. § 103 as being unpatentable over Gillis in view of Rao.

Gillis in view of Rao further in view of Clark or Glover:

According to the examiner (Answer, pages 5 and 6) the combination of Gillis in view of Rao is applied as it was against claim 1-3, 7 and 8 above. The examiner relies on Clark (Answer, page 5) to address the limitations of claims 5 and 9 drawn to transgenic alpha-1-antitrypsin. In addition, the examiner relies on Glover (Answer, page 6) to address the limitation of claim 6 which further limits claim 1, by requiring the alpha-1-antitrypsin to inhibit human neutrophil elastase activity.

Neither Clark nor Glover, however, make up for the deficiencies in the combination of Gillis in view of Rao, discussed supra. Accordingly, we reverse the rejection of claims 5 and 9 under 35 U.S.C. § 103 as being unpatentable over Gillis in view of Rao and further in view of Clark; and we reverse the rejection of claim 6 under 35 U.S.C. § 103 as being unpatentable over Gillis in view of Rao and further in view of Glover.

Lezdey in view of Clark:

According to the examiner (Answer, page 6), Lezdey “teaches topical compositions comprising alpha-1-antitrypsin analogs for use in treatment of inflammatory skin conditions such as burns and atopic dermatitis.” However, as discussed supra, the examiner has not established that the claimed therapeutically effective amount taught by Lezdey corresponds to the claimed therapeutically effective amount. Clark, who is relied upon to teach the transgenic production of AAT fails to make up for the deficiency in Lezdey.

Accordingly, we reverse the rejection of claim 9 under 35 U.S.C. § 103 as being unpatentable over Lezdey in view of Clark.

No time period for taking any subsequent action in connection with this appeal may be extended under 37 CFR § 1.136(a).

AFFIRMED-IN-PART

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Sherman D. Winters)	BOARD OF PATENT
Administrative Patent Judge)	
)	APPEALS AND
)	
)	INTERFERENCES
Donald E. Adams)	
Administrative Patent Judge)	

GRIMES, Administrative Patent Judge, dissenting in part.

I agree with the majority's conclusion and reasoning with respect to the rejections based on Schwarz. However, I would affirm the rejections based on Gillis and Rao, in combination and as combined with Clark or Glover. I therefore dissent from the majority's reversal of these rejections.¹

The examiner rejected claims 1-3, 7, and 8 as obvious in view of Gillis and Rao. The examiner cited Gillis as teaching a method of "treating wounds including chronic wounds such as chronic bedsores and ulcerative skin conditions by administering a composition comprising IL-1 and optionally alpha-antitrypsin." Examiner's Answer, page 4. The examiner acknowledged that Gillis did not teach the concentration of alpha-1-antitrypsin to add to the IL-1-containing composition (Examiner's Answer, page 4), nor did Gillis teach that "alpha-antitrypsin in and of itself has any intrinsic chronic wound-treating properties" (Examiner's Answer, page 5). The examiner cited Rao to meet these deficiencies. The examiner characterized Rao as "suggest[ing] that the topical administration of alpha-1-antitrypsin would be useful in the treatment of chronic wounds." Examiner's Answer, page 5. She concluded that it

would have been obvious to one of ordinary skill in the art at the time Applicants' invention was made to include alpha-antitrypsin in the chronic wound-treating compositions of Gillis et al[.] for the additional reason that the Rao et al[.] article suggests that alpha-antitrypsin has intrinsic chronic wound-treating properties and therefore its inclusion would have been expected to increase the potency of the compositions of Gillis et al.

¹ I also do not join the majority's reversal of the rejection based on Gillis alone and the rejections based on Lezdey. These rejections are cumulative in view of the rejections based on Gillis and Rao, and the rejections based on Schwarz. Since it is not necessary to reach any of these rejections, I express no opinion as to their merits.

Id.

I agree with the examiner's characterization of the references and with her conclusion that they render at least claims 1 and 7 prima facie obvious.² Claim 1 is directed to a method of treating a skin ulcer (venous ulcer, pressure sore, decubitus ulcer, or diabetic ulcer) by applying a "therapeutically effective amount" of alpha-1-antitrypsin. Dependent claim 3 clarifies that a therapeutically effective amount of alpha-1-antitrypsin can be 10 to 1000 µg per ml. Claim 7 is directed to a wound dressing composition comprising a "therapeutically effective amount" of alpha-1-antitrypsin, and intended for use in treating the same skin ulcers recited in claim 1.

Gillis discloses a method of treating chronic wounds, including "chronic bedsores [and] ulcerative skin conditions" (column 3, lines 24-28; column 4, lines 27-31). Gillis also teaches topical composition for use in treatment (column 3, lines 50-57). Gillis' compositions comprise interleukin-1 (IL-1) as the active ingredient. Gillis teaches that topical IL-1 promotes wound healing (column 2, lines 35-39), and is useful for treating "chronic or intractable wounding conditions" including "chronic bedsores [and] ulcerative skin conditions" (column 3, lines 24-28; column 4, lines 27-31). Gillis also teaches that "[a] variety of additives may be incorporated into the compositions of the present invention,

² Appellants do not separately argue the claims subject to this rejection. Claims 1 and 7 are representative of the claims rejected over Gillis and Rao.

provided that they do not deleteriously affect IL-1 biological activity.” Column 4, lines 52-54. Among the specifically named additives are “[p]rotease inhibitors such as α -antitrypsin inhibitor, . . . [which] may be useful in preventing degradation by proteolytic agents.” Column 4, lines 60-65.

Rao teaches that fibronectin (FN) participates in the repair process in skin wounds and is “proposed to be a beneficial factor in the healing of skin wounds.” Page 572, left-hand column. See also page 577, last paragraph: “Several studies have demonstrated a beneficial effect for topical FN on leg ulcers.” However, “[in] some chronic, non-healing wounds FN is extensively degraded.” Page 572. Rao therefore “investigate[d] if serine proteases are responsible for FN degradation in chronic skin wounds and . . . examine[d] the status of alpha1-antitrypsin (AT) (also called α 1-protease inhibitor), in acute and chronic wounds.” Page 572, right-hand column (citation omitted). Their results “implicate[d] AT as a regulator of FN degradation in chronic wounds. The inhibitor protects FN from degradation by serine proteinases in chronic wounds.” Id.

Rao shows, for example, that alpha-1-antitrypsin prevents degradation of fibronectin in vitro by proteinases from chronic wound fluid. See page 573, paragraph bridging the columns, and the legend to Figure 6. The results showed that alpha-1-antitrypsin protected fibronectin from degradation when added at either 30 μ g per 200 μ l (equivalent to 150 μ g/ml) or 120 μ g per 200 μ l (equivalent to 600 μ g/ml).³ See Figure 6 and page 575, paragraph bridging the columns.

³ When describing the difference between the lanes shown in the Figure, Rao refers to lanes 3 and 4 as showing degradation in the presence of “5 μ g” and “20 μ g,” respectively. These numbers apparently reflect the absolute amount (not concentration) of alpha-1-antitrypsin in the samples; only a portion of each sample was immunoblotted. See the legend to Figure 6.

Rao stated that their “results suggest[ed] that the degradation of AT, by as yet unidentified enzymes, leads to loss of this inhibitor in wounds that eventually feature extensive FN degradation.” Page 576, right-hand column. Finally, in the paragraph cited by the examiner, Rao stated that their results

may have relevance for the treatment of chronic wounds. Several studies demonstrated a beneficial effect for topical FN on leg ulcers and non-healing corneal ulcers. Intact FN may be required for the healing of chronic wounds. The [data] suggest that the degradation of AT precedes the degradation of FN in chronic wounds. . . . AT effectively prevented the degradation of FN by chronic wound fluid serine proteinases. Therefore, topical AT may inhibit FN degrading enzymes and increase the concentration of intact and functional FN in chronic wounds.

Page 577, left-hand column.

I agree with the examiner that the combined disclosures of Gillis and Rao would have rendered claims 1 and 7 prima facie obvious. Specifically, it would have been obvious to a person of ordinary skill in the art to modify Gillis’ IL-1-based method and composition for treating chronic wounds, by adding alpha-1-antitrypsin, in the amount shown by Rao to effectively inhibit proteinase activity in vitro (150-600 µg/ml). Motivation to combine the teachings of the references is provided by Gillis, who specifically suggests including alpha-1-antitrypsin in the IL-1-containing composition (column 4, lines 52-65) and by Rao, who teaches that inhibition of proteinase activity prevents fibronectin degradation, and that fibronectin contributes to wound healing (pages 572 and 577, left-hand columns).

Rao also shows that 150-600 µg/ml alpha-1-antitrypsin effectively prevented fibronectin degradation in vitro by proteinases in chronic wound fluid.

Since the proteinases inhibited in the in vitro experiment were the same proteinases that would be affected in vivo, I find that Rao's results would have provided a reasonable expectation that alpha-1-antitrypsin at 150-600 µg/ml would be effective for treating chronic wounds in vivo. See In re O'Farrell, 853 F.2d 894, 903-04, 7 USPQ2d 1673, 1681 (Fed. Cir. 1988) ("Obviousness does not require absolute predictability of success. . . . For obviousness under § 103, all that is required is a reasonable expectation of success.").

To the extent that the amount of alpha-1-antitrypsin used by Rao might have needed optimization when used therapeutically, it is well-established that it is "ordinarily within the skill of the art" to optimize a variable that is recognized to affect results. See In re Boesch, 617 F.2d 272, 276, 205 USPQ 215, 219 (CCPA 1980). The instant specification does not assert that the amount of alpha-1-antitrypsin used is critical, nor do Appellants argue that their method of using alpha-1-antitrypsin provided unexpectedly superior results.

The examiner also rejected claims 5 and 9 as obvious over Gillis, Rao, and Clark, and she rejected claim 6 as obvious over Gillis, Rao, and Glover. I would affirm these rejections as well.

Claims 5 and 9 are directed to the method and composition of claims 1 and 7, respectively, and add the limitation that the alpha-1-antitrypsin used is "a product of transgenic technology." Clark teaches transgenic production of alpha-1-antitrypsin. See page 2, line 29, to page 3, line 1. Clark also teaches that the disclosed method provides desired proteins at high yield and low cost, and free of infectious agents. Page 2, lines 1-5. I agree with the examiner that Clark's

teachings would have made it prima facie obvious to a person of ordinary skill in the art to use transgenically produced alpha-1-antitrypsin in the method made obvious by Gillis and Rao, because of the advantages disclosed by Clark.

Claim 6 is directed to the method of claim 1, with the added “limitation” that “the alpha-1-antitrypsin inhibits the activity of human neutrophil elastase in the wound.” The examiner cited Glover as evidence that alpha-1-antitrypsin inhibits elastase. Examiner’s Answer, page 6. Thus, claim 6 does no more than recite an inherent property of alpha-1-antitrypsin, as shown by the examiner’s cited references. I agree with the examiner that claim 6 is also prima facie obvious.

Appellants argue that Rao contains “no specific mention of decubitis ulcers, pressure sores, diabetic ulcers or venous ulcers. Furthermore, no in vivo experiments are performed. At best, Rao postulates that AAT may protect fibronecti[n] in a chronic wound.” Brief, page 15.

I do not find these arguments persuasive. First, Rao provides a reasonable basis for concluding that alpha-1-antitrypsin would aid in the healing of chronic wounds. The wound fluid used by Rao was derived from “chronic venous stasis ulcers.” Page 572, right-hand column. At a minimum, therefore, Rao’s teachings would be relevant to the “venous ulcers” recited in the claims. However, Rao disclosed that their findings were applicable to chronic wounds generally, and not limited to venous ulcers. Gillis teaches that chronic wounds generally are amenable to treatment with the disclosed IL-1-containing compositions. Column 3, lines 24-32. Therefore, the skilled artisan would

reasonably have expected that a solution containing IL-1 and alpha-1-antitrypsin would effectively aid healing of chronic wounds in general, including the specific types of chronic wounds recited in the claims.

Second, although Rao does not show in vivo treatment of chronic wounds using alpha-1-antitrypsin, it provides a reasonable basis on which to conclude that the in vitro results they observed would also be seen in vivo. Specifically, Rao discusses the role of fibronectin in wound healing, discloses that fibronectin is degraded in chronic wounds, and discloses that alpha-1-antitrypsin prevents degradation of fibronectin by proteinases in chronic wound fluids. The proteinases inhibited by alpha-1-antitrypsin in vitro in Rao's experiments would be the same as those encountered by alpha-1-antitrypsin in vivo. Thus, those of ordinary skill in the art would reasonably expect to obtain the same results in vivo as those observed in vitro.

Finally, Appellants argue that Rao at best postulates that alpha-1-antitrypsin may protect fibronectin in chronic wounds. The majority agrees with this position, and concludes that the prior art only makes the instant claims "obvious to try." Ante, pages 8-9. For the reasons discussed above, I find that Gillis and Rao would have provided those of skill in the art with a reasonable expectation of success, and I conclude that the references support a prima facie case under § 103. I would affirm all of the rejections based on Gillis and Rao, by themselves or with Clark or Glover, and hold all of the claims unpatentable under 35 U.S.C. § 103.

Eric Grimes
Administrative Patent Judge

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