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UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

Ex parte TAREK MOUSTAFA, MICHAEL TRAUNER and PETER FICKERT[1]

Appeal 2019-004162
Application 12/670,639
Technology Center 1600

Before DONALD E. ADAMS, JOHN G. NEW, and RACHEL H. TOWNSEND, *Administrative Patent Judges.*

NEW, *Administrative Patent Judge*.

DECISION ON APPEAL

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[1]We use the word "Appellant" to refer to the "applicant" as defined in 37 C.F.R. § 1.142". Appellant identifies Medizinische Univeristät Graz as the real party-in-interest. App. Br. 1.

SUMMARY

Appellant files this appeal under 35 U.S.C. § 134(a) from the Examiner's Final Rejection of claims 8 and 9 as unpatentable under 35 U.S.C. § 103(a) as being obvious over the combination of Zadini et al. (WO 2007/061820 A2, May 31, 2007) ("Zadini") B.I. Cohen et al., *Differing Effects of Nor-ursodeoxycholic or Ursodeoxycholic Acid on Hepatic Histology and Bile Acid Metabolism in the Rabbit*, 91 GASTROENTEROL. 189–97 (1986) ("Cohen"), and Castagnola et al. (US 4,892,868, January 9, 1990) ("Castagnola").

Claims 8 and 9 stand further rejected as unpatentable under the combination of Trauner et al. (WO 2006/119803 A1, November 16, 2006) ("Trauner"), P. Fickert et al., *Primary Sclerosing Cholangitis – The Arteriosclerosis of the Bile Duct?*, 6:3 LIPIDS IN HEALTH AND DISEASE, 1–8 (January 25, 2007) ("Fickert"), and Sasahara et al. (US 2007/0203115 A1, August 30, 2007) ("Sasahara").

We have jurisdiction under 35 U.S.C. § 6(b).

We AFFIRM.

NATURE OF THE CLAIMED INVENTION

Appellant's invention is directed to the use of nor-bile acids and their pharmaceutically acceptable salts, esters, and/or derivatives in the treatment of arteriosclerosis. Abstr.

REPRESENTATIVE CLAIM

Claim 8 is representative of the claims on appeal and recites:

> 8. A method of treating arteriosclerosis in a human or animal subject comprising the step of administering at least nor-ursodeoxycholic acid and/or at least one pharmaceutically

> acceptable salt and/or ester thereof, wherein the oral dosage form is a controlled release dosage form that releases the at least nor-ursodeoxycholic acid and/or the at least one pharmaceutically acceptable salt and/or ester thereof only after the oral dosage form has reached the stomach or the gastro-intestinal tract.

App. Br. 21.

ISSUES AND ANALYSES

We adopt the Examiner's findings, reasoning, and conclusion that the claims on appeal are *prima facie* obvious over the cited prior art. We address the arguments raised by Appellant below.

A. Rejection of the claims over Zadini, Cohen, and Castagnola

Issue

Appellant argues the Examiner erred because Zadini, Cohen, and/or Castagnola fail to provide, either individually or in combination, any teaching, suggestion, or motivation that would have led a skilled artisan to combine the references to arrive at the claimed method. App. Br. 11.

Analysis

The Examiner finds that Zadini teaches a method of treating atherosclerosis comprising administering a list of biliary compound to a human, in which the compound is orally ingested, and is a slow-release pharmaceutical preparation. Final Act. 4 (citing Zadini claims 1, 3, 11, 6, 12). The Examiner finds that Zadini teaches that the orally-ingested composition is in an oral dosage form, and that the slow release corresponds to claim 8's recitation of "controlled release." *Id.* The Examiner further

finds that Zadini teaches that its orally-ingested dosage form comprises an intestinal absorption enhancer. *Id*. (citing Zadini claim 18). The Examiner therefore finds that Zadini teaches that its slow-release dosage form formulated with an intestinal absorption enhancer would be understood by a skilled artisan to correspond to the controlled-release dosage form that releases the nor-ursodeoxycholic acid ("nor-UDCA") only after the compound has reached the gastro-intestinal tract, as recited in claim 8. *Id.*

The Examiner notes that Appellant's Specification defines controlled-release dosage form as a composition that releases the active agent only after the dosage form has reached a certain site of the body, e.g., the stomach or the gastro-intestinal tract, or, additionally or alternatively, it may designate a dosage form, which releases the active agent over a prolonged period of time. Final Act. 4 (citing Spec. 21).

The Examiner also finds that Zadini teaches biliary compounds, including nor-bile acid derivatives such as 23-methylursodeoxycholic acid and 24-nor-UDCA. Final Act. 4 (citing Zadini 21, ll. 15–16). The Examiner finds that Zadini teaches biliary compounds include 3-hydroxy-5-cholen-24-oic acid 3-sulfate ester (*see* page 19, line 18) and hyodeoxycholate-6-O-glucuronidel (*see* page 20, line 11).

The Examiner finds that Cohen teaches that, from a therapeutic standpoint, not only is nor-UDCA not hepatotoxic in humans, but also that if administered in its unconjugated form, it will not accumulate in the enterohepatic circulation, unlike ursodeoxycholic acid ("UDCA"). Final Act. 5.

The Examiner finds that Castagnola teaches that nor-UDCA, and its salts, derivatives, and esters, have been shown to have significantly

improved properties as regards to cholesterol-dissolving ability, higher critical micellar concentration and stimulation of the bile flow compared to ursodeoxycholic acid (UDCA). Final Act. 5. The Examiner finds that these properties are combined with an exceptional stability against natural degradation and remarkable hepatic tolerance, enabling these compounds for long term treatment. *Id.* (citing Castagnola col. 2, ll. 1–59). The Examiner further finds that Castagnola teaches that these compounds can be administered *via* oral and parenteral routes. *Id.* (citing Castagnola col. 7, ll. 38–40).

The Examiner concludes that it would have been *prima facie* obvious to a person of ordinary skill in the art to select nor-UDCA, or a salt or ester thereof, because Cohen teaches that nor-UDCA is not hepatotoxic in humans because it does not accumulate in the enterohepatic circulation compared to its amidates (i.e., UDCA), and also because Castagnola teaches that nor-UDCA, salts, derivatives, and esters thereof, have been shown to have significantly improved properties with respect to cholesterol-dissolving ability, higher critical micellar concentration and stimulation of the bile flow compared to UDCA, exceptional stability and low hepatotoxicity. Final Act. 5–6. The Examiner finds that a person of ordinary skill in the art would have been motivated to select nor-UDCA due to its advantages over UDCA. *Id.* at 6.

Appellant argues that Zadini and Cohen each teach away from the claimed invention and that Castagnola does not cure these deficiencies. App. Br. 11. With respect to Zadini, Appellant argues that Zadini teaches that bile acids, including nor-UDCA, should not be administered using the oral-digestive route of administration. According to Appellant, Zadini

further teaches that, if oral-digestive administration is used, nor-UDCA must be chemically modified. *Id.*

By way of example, Appellant contends that Zadini teaches that: "biliary compounds can be administered via many routes, except that they cannot be administered via the oral digestive route, because when ingested they are […] sequestered in the enterohepatic circulation." App. Br. 12 (quoting Zadini 29). Appellant points out that Zadini further teaches that:

> In animals, as in human bodies, bile salts however are confined to the digestive system, in the so called enterohepatic circulation, and do not come in contact with arteries either of the systemic or pulmonary circulation, therefore the biliary salts in nature are prevented from displaying their benefits on atherosclerotic plaques.

Id. (quoting Zadini 5). Appellant notes that Zadini therefore teaches a number of methods for administering bile acids or their salts via means which bypass the enterohepatic circulation, including *via*: "[o]ral mucous membrane, such as sublingual," but not *via* oral ingestion. *Id.* (quoting Zadini 31).

Appellant therefore argues that Zadini neither teaches nor suggests the oral administration of nor-UDCA. App. Br. 13. Rather, Appellant contends, oral-sublingual administration of nor-UDCA, or its pharmaceutically acceptable salts or esters, is expressly excluded by claim 8, which requires that the oral dosage form be released "only after the oral dosage form has reached the stomach or the gastro-intestinal tract." *Id.*

Appellant acknowledges that Zadini teaches that:

> Biliary compounds can also be chemically manipulated and designed in such a way that they are not captured by the liver in any significant amount to be sequestered into the entero-hepatic

> circulation once introduced into the body by any route including the oral-digestive route. The use of these types of compounds makes oral administration possible even with biliary compounds, expanding even further the possibilities of the disclosed treatment of atherosclerosis.

App. Br. 13 (quoting Zadini 34). Appellant contends that Zadini thus teaches that, to the extent oral-digestive administration of nor-UDCA is contemplated, despite Zadini's teachings to the contrary, nor-UDCA must be chemically modified to turn it into another chemical compound that would be suitable for such use. *Id.* Appellant asserts that Zadini teaches the oral-digestive administration of a nor-UDCA derivative, and not the oral-digestive administration of unmodified nor-UDCA, or pharmaceutically acceptable salt or ester thereof, as required by claim 8. *Id.*

With respect to Cohen, Appellant argues that the reference teaches that nor-UDCA taken up via the stomach will not enter systemic blood circulation. App. Br. 13 (citing Cohen Fig. 1). According to Appellant, chronic administration of nor-UDCA causes hepatotoxicity because chronic ingestion of natural bile acids leads to the accumulation of the toxic metabolite, lithocholate, in the liver. *Id.* at 14. Appellant contends that Cohen teaches, however that nor-UDCA does not cause lithocholate accumulation or hepatotoxicity in the liver of rabbits. *Id.* Appellant asserts that Cohen therefore concludes that nor-UDCA is not hepatotoxic, and that the absence of lithocholate accumulation is caused by enzymatic modification of nor-UDCA that occurs during hepatic passage; in short, the nor-UDCA is converted to nor-UDCA-glucuronide and excreted. *Id.* (citing Cohen 194–95).

Therefore, Appellant contends, Cohen teaches that nor-UDCA does not reach systemic blood circulation, and therefore cannot reach the coronary arteries, where atherosclerosis is predominant. App. Br. 15. In view of this, argues Appellant, a person of ordinary skill in the art would have understood that nor-UDCA administered via the oral-digestive route would not be able to be used to treat arteriosclerosis, and would have also understood that Cohen teaches away from the administration of nor-UDCA via an oral-digestive route for the treatment of arteriosclerosis. *Id.*

With respect to Castagnola, Appellant argues that the reference teaches that nor-UDCA has the ability to dissolve cholesterol at a higher critical micellar concentration and stimulation of the bile flow as compared to UDCA. App. Br. 16 (citing Castagnola col. 2, ll. 24–27). Appellant contends that Castagnola does not provide any teaching or suggestion regarding the intestinal absorption and hepatic metabolism of nor-UDCA. *Id.*

Appellant therefore argues that it would not have been obvious to a person of ordinary skill in the art to choose nor-UDCA, or a salt or ester thereof, for use in oral-digestive administration to treat arteriosclerosis. App. Br. 16. Appellant asserts that a person of ordinary skill in the art would not be motivated to administer nor-UDCA via the oral-digestive route in view of the teachings of Zadini and Cohen that doing so would be unsuccessful, since the nor-UDCA would not reach the circulatory system and could not do so without potentially deleterious modifications. *Id.* Furthermore, argues Appellant, there would be no reasonable expectation of successfully administering nor-UDCA, or a salt or ester thereof, *via* an oral-digestive route to treat arteriosclerosis, because, as taught by Zadini and

Cohen, the nor-UDCA would not enter the blood circulatory system and therefore would not be able to affect any arteriosclerotic vessels. *Id.* at 16–17.

We do not find Appellant's arguments persuasive. We acknowledge that Zadini teaches the passages quoted by Appellant. However, Zadini expressly teaches the oral administration of biliary salts, including nor-DCA. *See* Zadini claim 11 ("The treatment of claim 2 wherein said compound is orally ingested"); *see also* Abstr. ("A pharmacological substance namely a biliary salt or acid or precursor or derivative with emulsifying properties … administered into the systemic circulation of a patient via a variety of routes of administration including topical-mucous membrane such as sublingual, topical-dermatological such as via a skin patch, *oral*, [etc.]") (emphasis added).

Furthermore, Zadini teaches that:

> Biliary compounds can also be chemically manipulated and designed in such a way that they are not captured by the liver in any significant amount to be sequestered into the entero-hepatic circulation once introduced into the body by any route including the oral-digestive route. The use of these types of compounds makes oral administration possible even with biliary compounds, expanding even further the possibilities of the disclosed treatment of atherosclerosis.

Zadini 34. Zadini further teaches, by way of example, that: "Biliary compounds being designed to enter the systemic circulation through oral-digestive route of administration can be associated with intestinal absorption enhancers so that their bioavailability in the systemic circulation is maximized." *Id.* Appellant's claims do not recite any "chemical manipulation or design" in the administration of nor-UDCA, nor do they

claim association with "intestinal absorption enhancers," however, the claims do recite: "A method of treating arteriosclerosis in a human or animal subject comprising the step of administering at least nor-ursodeoxycholic acid and/or at least one pharmaceutically acceptable salt and/or ester thereof …." The use of the transition term "comprising" does not exclude any additional steps or limitations from the scope of the claim. *See Crystal Semiconductor Corp. v. TriTech Microelectronics Int'l, Inc.*, 246 F.3d 1336, 1348 (Fed. Cir. 2001) ("Use of the transition "comprising" in the language of a claim creates a presumption ... that the claim does not exclude additional, unrecited elements").

Finally, Cohen teaches that nor-UDCA is "glucuronidated during hepatic passage" and suggests that: "it is a reasonable conclusion that the glucuronide was not reabsorbed from the gastrointestinal tract, as other glucuronides are not reabsorbed from the intestine." Cohen 195. Zadini, however, teaches that:

> An interesting compound among the biliary acids is the hyodeoxycholic acid. As reported by Sacquet E. et al.[2] … once it reaches the liver through the portal venous system after absorption by the intestinal mucosa, the hyodeoxycholic acid largely escapes, in healthy humans, the enterohepatic circulation entering the systemic circulation to be excreted through the kidneys in the urine in a very significant amount. It appears that the hyodeoxycholic acid escapes the enterohepatic circulation *after having undergone a process of glucuronidation* by the hepatic cell. The Applicants believe that this peculiarity of the hyodeoxycholic acid to enter the systemic circulation in theory could be exploited to directly emulsify/dissolve the lipid core of atherosclerotic plaques. Another advantage of the

[2] Sacquet E. et al., *Intestinal Absorption, Excretion, and Biotransfonnation of Hyodeoxycholic Acid in Man*, 24 J. LIPID RES. 604–13 (1983).

> hyodeoxycholic acid is that *it can be administered via oral-intestinal route*.

Zadini 12 (emphasis added). Cohen thus teaches that nor-UDCA is converted in the liver to a glucuronide, just as hyodeoxycholic acid is similarly converted (and unlike UDCA; *see* Cohen 194–95; Fig. 1). Cohen hypothesizes that the converted glucuronide is removed from the body via the intestinal system, but does not test this hypothesis, nor does Cohen determine whether nor-UDCA enters systemic circulation. We find that, because both nor-UDCA and hyodeoxycholic acid are capable of glucuronidation, and because, when glucuronidated, hyodeoxycholic acid can pass into systemic circulation, it would not be an unreasonable inference by a person of ordinary skill in the art to conclude that, like hyodeoxycholic acid, nor-UDCA is also able to pass into the systemic circulation.[3]

In summary, we are not persuaded by Appellant that the balance of evidence presented by the cited prior art teaches away from the claims on appeal. A teaching away requires a reference to actually criticize, discredit, or otherwise discourage the claimed solution. *See In re Fulton*, 391 F.3d 1195, 1201 (Fed. Cir. 2004). As we have explained the combination of

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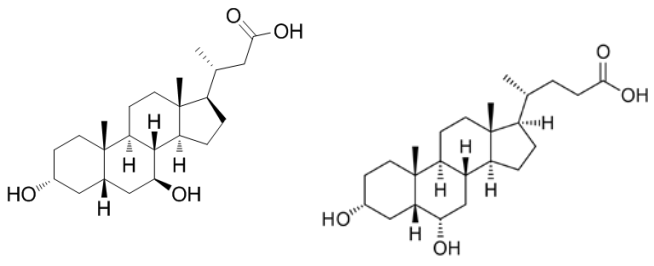
[3] We also note here that hyodeoxycholic acid and nor-UDCA are highly similar structures, as illustrated below:



Structures of nor-UDCA (left) and hyodeoxycholic acid (right)

Zadini and Cohen suggests that oral administration is at least feasible as a means of introducing nor-UDCA into systemic circulation. We consequently are not persuaded by Appellant's arguments, and we affirm the Examiner's rejection.

B. Rejection of the claims over Trauner, Fickert, and Sasahara

Issue 1

Appellant argues that the Examiner erred by failing to articulate any teaching, suggestion, or motivation in the prior art that would have led a person of ordinary skill to combine the references to arrive at Appellant's claimed invention. App. Br. 17.

Analysis

The Examiner finds that Trauner teaches a method of treating liver diseases, and preferably primary sclerosing cholangitis ("PSC") or primary biliary cirrhosis ("PBC") comprising nor-UDCA, and/or its pharmaceutically acceptable salt and esters, in an amount of 10 to 8000 mg. Final Act. 8 (citing Trauner 5, 6, claims 1, 4, 5). The Examiner finds that Trauner teaches various delivery systems, including sustained release form. *Id.* (citing Trauner 8). The Examiner points to Appellant's Specification as disclosing that sustained release is a form of controlled release and, therefore, Trauner's teaching of sustained release falls within the scope of "controlled release," as recited in the claims. *Id.* at 8–9 (citing Spec. ¶ 116). The Examiner also finds that Trauner teaches that the sustained release dosage form can be administered as a solid oral dosage form. *Id.* at 9 (citing Trauner 6).

The Examiner finds that Trauner does not teach arteriosclerosis or atherosclerosis; however, the Examiner finds that Fickert teaches that arteriosclerosis and PSC may share common disease mechanisms, based on similar pathogenic features and mediators found in both. Final Act. 9 (citing Fickert 1). The Examiner further finds that Fickert teaches that research on PSC may greatly benefit from translating pathogenic concepts from advanced work in arteriosclerosis to the study of cholestasis. *Id.* (citing Fickert 7).

The Examiner finds that Sasahara teaches therapeutic agents, *viz.* novel benzodiazepine compounds which are useful for treating arteriosclerosis, PSC, and PBC. Final Act. 9 (citing Sasahara ¶¶ 4, 9).

The Examiner concludes that it would have been *prima facie* obvious to a person of ordinary skill in the art to employ the method of treatment taught by Trauner to treat arteriosclerosis (which, the Examiner finds, includes atherosclerosis). Final Act. 9. The Examiner finds that a skilled artisan would have been motivated to do so because Fickert teaches that PSC and arteriosclerosis have common disease mechanisms. *Id.* Therefore, the Examiner finds, a person of ordinary skill would have had a reasonable expectation of success in employing the treatments taught by Trauner to treat arteriosclerosis, because Sasahara teaches that treatments for PSC can also be used to treat arteriosclerosis. *Id.*

Appellant argues that, although PSC and arteriosclerosis share some similarities regarding their pathogenesis, they affect different areas of the body: PSC affects the liver, whereas arteriosclerosis occurs predominantly in the coronary arteries. App. Br. 17–18. Therefore, argues Appellant, a drug that is effective in treating PSC must reach the liver, whereas a drug that is

used in the treatment of arteriosclerosis must reach the coronary arteries. *Id.* at 18.

Appellant contends that a person of skill in the art would have known, at the time Appellant's application was filed, that bile acids like nor-UDCA are kept away from systemic blood circulation upon oral-digestive uptake, because it is confined in the enterohepatic circulation. App. Br. 18. As a result, Appellant contends, such acids reach the liver, but not the systemic circulation, and therefore do reach the coronary arteries where arteriosclerotic plaques occur. *Id.* Therefore, Appellant asserts, it would have been reasonable to treat PSC with an oral-digestive dosage form containing nor-UDCA, as disclosed by Trauner, since nor-UDCA can reach the liver and achieve a high concentration in the liver *via* cholohepatic shunting. *Id.* However, Appellant argues, it would not have been reasonable to use nor-UDCA to treat a condition, such as arteriosclerosis, that requires nor-UDCA to enter the systemic circulation and, in particular, sclerotic arteries. *Id.* Appellant asserts that it was well-known that when administered via an oral-digestive dose, nor-UDCA would not reach the blood circulatory system and that bile acids, such as nor-UDCA, accumulate in the liver, resulting in hepatotoxicity. *Id.*

Therefore, Appellant argues, the mechanism by which nor-UDCA is beneficial in the treatment of PSC, would not have suggested that nor-UDCA would be beneficial in, or even capable of, the treatment of arteriosclerosis, which requires that the nor-UDCA accumulate in the system arteries affected by sclerosis. App. Br. 18 (citing Zadini 5).

We do not find Appellant's argument persuasive. Although Zadini and Cohen are not cited, in this rejection, as bases for the rejection,

Appellant's argument nevertheless explicitly relies upon the arguments presented *supra* with respect to the teachings of Zadini and Cohen, as indicating that the prior art at the time of Appellant's invention taught away from the idea that oral administration of nor-UDCA can reach the systemic circulation.

As an initial matter, Cohen directly contradicts Appellant's contention that "bile acids, such as nor-UDCA, accumulate in the liver, resulting in hepatotoxicity." *See* App. Br. 18. To the contrary, Cohen expressly teaches that nor-UCDA is converted to a glucuronide and consequently does *not* accumulate as the hepatotoxic metabolite, lithocholate, in the liver. *See* Cohen Abstr. ("Ingestion of nor-[UDCA] … did not cause hepatotoxicity, and neither it nor its presumed metabolite, nor-lithocholate, accumulated in biliary bile acids").

Furthermore, we have explained *supra*, why we are not persuaded that the prior art at the time of invention would have taught a person of ordinary skill in the art that oral administration of nor-UDCA could not reach the systemic circulation. To the contrary, we have found that a person of ordinary skill, given the combined teachings of the prior art, and particularly those of Zadini and Cohen, would have believed that there was a reasonable expectation of success that nor-UDCA could be introduced into the systemic circulation, either via chemical manipulation and design in such a way that it would not be captured by the liver, as taught by Zadini (*see* Zadini 34), or *via* hepatic conversion to glucuronide, as suggested by the combined teachings of Zadini and Cohen. *See* Zadini 12, Cohen 194–95. We consequently do not find Appellant's arguments persuasive.

Issue 2

Appellant argues that the Specification discloses the unexpected result that oral-digestive administration of nor-UDCA is effective in the treatment of arteriosclerosis in mice. App. Br. 19 (citing Spec. Ex. 1). Appellant contends that this is a surprising result because Appellant contends that the knowledge in the art at the time of Appellant's invention taught that there is no oral-digestive uptake of nor-UDCA. *Id.*

Additionally, Appellant points to the allegedly unexpected discovery that nor-UDCA has positive effects on the liver, including acting as an anti-inflammatory, when compared to UDCA. App. Br. 20 (citing, e.g., Spec. 27–30, Figs. 7, 8, 9, 12). According to Appellant, treatment in mice with nor-UDCA reduced white adipose tissue as compared to treatment with UDCA. *Id.* (citing, e.g., Spec. 30–33, Fig. 18). Appellant contends that these effects upon treatment with nor-UDCA were unexpected and support the nonobviousness of the instantly claimed invention.

We do not agree. As we have explained, we do not find Appellant's arguments *supra* persuasive of the contention that the prior art at the time of Appellant's invention taught away from the oral administration of nor-UDCA as a means of introducing it into the systemic circulation. Appellant points to no direct evidence in the prior art demonstrating that oral administration of nor-UDCA would not be effective in introducing the composition into systemic circulation, and we have explained how we have found that the prior art suggests that it could be so used.

Furthermore, as found by the Examiner, the combined teachings of the combined cited prior art also directly suggests that nor-UDCA could be effective in the treatment of atherosclerosis.

Finally, the fact that nor-UDCA is beneficial to the liver, as compared to UDCA, which promotes hepatotoxicity via the accumulation of the metabolite lithocholate, is expressly taught by Cohen. *See* Cohen Abstr. 194–95. Moreover, the effect of nor-UDCA in reducing white adipose tissue in mice, as argued by Appellant, is not relevant to the claims and is insufficient to overcome the Examiner's conclusion that the claims are *prima facie* obvious. *See In re Baxter Travenol Labs.*, 952 F.2d 388, 392 (Fed. Cir. 1991) (holding that "[m]ere recognition of latent properties in the prior art does not render nonobvious an otherwise known invention"). We consequently affirm the Examiner's rejection upon this ground.

DECISION

The Examiner's rejection of claims 8 and 9 under 35 U.S.C. § 103(a) is affirmed.

No time period for taking any subsequent action in connection with this appeal may be extended under 37 C.F.R. § 1.136(a)(1)(iv).

AFFIRMED

Claims Rejected	**Basis**	**Affirmed**	**Reversed**
8, 9	§ 103(a) over Zadini, Cohen, Castagnola	8, 9	
8, 9	§ 103(a) over Trauner, Fickert, Sasahara	8, 9	
Overall Outcome		8, 9	