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Datasheet for the decision of 19 November 2009

Case Number: T 0553/08 - 3.3.08

Application Number: 00902354.0

Publication Number: 1141274

IPC: C12N 15/11

Language of the proceedings: EN

Title of invention:
Soluble receptor BR43x2 and methods of using them for therapy

Patentee:
ZymoGenetics, Inc.

Opponents:
Genentech, Inc.
BIOGEN IDEC INC.

Headword:
BR43x2 receptor/ZYMOGENETICS

Relevant legal provisions:
EPC Art. 123(3), 54, 56, 83

Relevant legal provisions (EPC 1973):
-

Keyword:
"Extension of the scope of the claims (no)"
"Novelty, inventive step, sufficiency of disclosure (yes)"

Decisions cited:
T 1329/05

Catchword:
-

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Beschwerdekammern | Boards of Appeal | Chambres de recours

Case Number: T 0553/08 - 3.3.08

D E C I S I O N
of the Technical Board of Appeal 3.3.08
of 19 November 2009

Appellant: (Patent Proprietor)
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Decision under appeal:
Interlocutory decision of the Opposition Division of the European Patent Office posted 30 November 2007 concerning maintenance of European patent No. 1141274 in amended form.

Composition of the Board:

Chairman: L. Galligani
Members: F. Davison-Brunel
C. Rennie-Smith

Summary of Facts and Submissions

I. European patent No. 1 141 274 with the title "Soluble receptor BR43x2 and methods of using them for therapy", was granted with 38 claims, based on European patent application No. 00 902 354.0.

II. Four oppositions were initially filed on the grounds of Article 100(a) to (c) EPC. Opponents 01 and 02 both withdrew their oppositions on 25 September 2007. The opposition division maintained the patent in amended form on the basis of the second auxiliary request (claims 1 to 6) filed during the oral proceedings on 27 September 2007.

Claims 1 and 6 thereof read as follows:

"1. Use of a fusion protein consisting of a first portion and a second portion joined by a peptide bond, wherein said first portion consists of amino acid residues 25-104 of SEQ ID NO: 6 and said second portion is an immunoglobulin heavy chain constant region for the manufacture of a medicament for treating asthma, bronchitis, emphysema, nephritis, pyelonephritis, renal neoplasms, light chain neuropathy, amyloidosis, membranous nephropathy, IgA nephropathy, Berger's Disease, IgM nephropathy, Goodpasture's Disease, post-infectious glomerulonephritis, mesangioproliferative disease, minimal-change nephrotic syndrome, or secondary glomerulonephritis or vasculitis associated with lupus, in a mammal by inhibiting BR43x2, TACI, or BCMA receptor-ztnf4 engagement.

6. Use of an antibody or antibody fragment that specifically binds to a polypeptide of SEQ ID NO: 18, for the manufacture of a medicament for inhibiting antibody production associated with systemic lupus erythematosus in a mammal by inhibiting BR43x2, TACI, or BCMA receptor-zntf4 engagement."

Claims 2 to 5 related to further features of the use of claim 1.

III. The appellant (patentee) filed an appeal and submitted a statement of grounds of appeal together with a new main request.

IV. Respondents I and II (opponents 03 and 04) did not reply to the appellant's statement of grounds of appeal.

V. The board sent a communication pursuant to Article 15(1) of the Rules of Procedure of the Boards of Appeal, indicating its preliminary, non-binding opinion.

VI. By letters respectively dated 17 September 2009 and 19 October 2009, respondents I and II informed the board of their intention not to take part in the oral proceedings.

VII. On 19 October 2009, the appellant replied to the board's communication and filed therewith two declarations and four auxiliary requests.

VIII. At the oral proceedings which took place on 19 November 2009, the appellant replaced the pending requests by a new request which consisted of 32 claims. Claims 1 to 6 were identical to the claims allowed by the opposition

division except for the fact that no mention was made of the medical condition "renal neoplasms" in claim 1.

Claims 7, 12, 17, 27 and 32 read as follows:

"7. An isolated polynucleotide molecule encoding a polypeptide of SEQ ID NO: 2.

12. An isolated polypeptide having the sequence of SEQ ID NO: 2.

17. Use of a compound selected from the group consisting of:
a) a polypeptide comprising the extracellular domain of BR43x2 (SEQ ID NO: 2); and
b) a polypeptide of SEQ ID NO: 4;

for the manufacture of a medicament for treating asthma, bronchitis, emphysema, end stage renal failure, light chain neuropathy, amyloidosis, inflammation, membranous nephropathy, IgA nephropathy, Berger's Disease, IgM nephropathy, Goodpasture's Disease, post-infectious glomerulonephritis, mesangioproliferative disease, minimal-change nephrotic syndrome, or secondary glomerulonephritis or vasculitis associated with lupus, or for inhibiting antibody production associated with an autoimmune disease; in a mammal by inhibiting BR43x2, TACI, or BCMA receptor-ztnf4 engagement.

27. Use of a compound selected from the group consisting of:
a) an antibody or antibody fragment that specifically binds to a polypeptide of SEQ ID NO: 2; and

b) an antibody or antibody fragment that specifically binds to a polypeptide of SEQ ID NO: 4;
for the manufacture of a medicament for treating asthma, bronchitis, emphysema, end stage renal failure, light chain neuropathy, amyloidosis, inflammation, membranous nephropathy, IgA nephropathy, Berger's Disease, IgM nephropathy, Goodpasture's Disease, post-infectious glomerulonephritis, mesangioproliferative disease, minimal-change nephrotic syndrome, or secondary glomerulonephritis or vasculitis associated with lupus, or for inhibiting antibody production associated with an autoimmune disease; in a mammal by inhibiting BR43x2 receptor-ztnf4 engagement.

32. Use of an antibody or antibody fragment that specifically binds to a polypeptide of SEQ ID NO: 6 for the manufacture of a medicament for treating asthma, bronchitis or emphysema in a mammal, by inhibiting TACI receptor-ztnf4 engagement."

Claims 8 to 11 and 13 respectively related to the isolated polynucleotide of SEQ ID NO 1, to an expression vector and a cultured cell expressing/comprising said polypeptide/recombinant vector, to a method of producing the polypeptide and to the polypeptide per se in combination with a pharmaceutically acceptable vehicle. Claims 14 to 16 related to various pharmaceutical compositions comprising antibodies binding to the polypeptide of SEQ ID NO: 2 or of SEQ ID NO: 4. Claims 18 to 26 relate to further features of the use of claim 17, claims 28 to 31 relate to further features of the use of claim 27.

IX. The documents which are referred to in the present decision are the following:

(1) : WO 98/39361 published on 11 September 1998;

(9) : von Bülow, G.U. and Bram, R.J., Science, Vol. 278, pages 138 to 141, 3 October 1997;

(33) : Hymovitz, S.G. et al., The Journal of Biological Chemistry, Vol.280, No.8, pages 7218 to 7227, 25 February 2005;

(38) : Gross, J.A. et al., Nature, Vol.404, pages 995 to 999, 27 April 2000;

(48) : Tak, P.P. et al., Report of experiments carried out as part of a clinical trial of TACI-Ig for the therapy of patients with rheumatoid arthritis (no date);

(49) : Dall'Era, M. et al., Report of experiments carried out as part of a clinical trial of TACI-Ig for the therapy of patients with systemic lupus erythematosus (SLE) (no date);

(50) : Bislborough, J. et al., Report of experiments carried out as part of a clinical trial of TACI-Ig for the treatment of asthma (no date);

(51) : Eun-Yi Moon and Sook-Kyung Ryu, Experimental and Molecular Medicine, Vol.39, No.3, pages 343 to 352, June 2007;

(57) : Pena-Rossi, C. et al; Report of experiments carried out as part of a clinical trial of TACI-Ig for the therapy of patients with systemic lupus erythematosus (SLE) (no date);

X. The appellant's arguments insofar as relevant to the present decision can be summarized as follows:

Article 123(3) EPC; extension of the scope of the claims

Claims 17 and 27 were directed to the use either of a polypeptide comprising SEQ ID NO:2 or of the polypeptide of SEQ ID NO:4 or of antibodies thereto for the manufacture of a medicament against diseases which had not been mentioned in the granted claims 19 or 20. The scope of the claims had nonetheless not been extended as these diseases were specific subsets of the medical conditions recited in the granted claims. Claims 17 and 27, thus, met the requirements of Article 123(3) EPC.

Article 56 EPC; inventive step
Claims 7 to 31

The closest prior art was document (9), a scientific article which solely related to the isolation of the human tumour necrosis factor receptor TACI. It provided no suggestion that any isoform of TACI, let alone BR43x2, would exist. A fortiori, document (9) failed to suggest any ligand that bound BR43x2. As such, the skilled person reading the document would have had no reasonable expectation of successfully identifying any ligand for this receptor. It was therefore evident that the therapeutic uses of BR43x2 polypeptide and antibodies which were based on inhibiting the engagement of the

ztnf4 ligand with its receptors would not have been obvious in the light of document (9).

The patent in suit provided unambiguous evidence that the BR43x2 polypeptide was a TNF receptor. Its encoding DNA was isolated through the ability of the BR43x2 polypeptide to bind the TNF ligand ztnf4. Furthermore, the % amino acid sequence identity between BR43x2 and the TNF receptor TACI was almost 100% outside of the cysteine-rich repeats region. BR43x2 contained a cysteine-rich domain as did the previously isolated TNF receptors. In fact, as discussed in document (33), BR43x2 was a splice variant of the TACI TNFR.

Claim 32

This claim recited the use of human anti-TACI antibodies for the manufacture of a medicament for treating a number of specific airway disorders in a mammal. Direct evidence had been provided in the patent of the link between the activity of ztnf4 and these disorders (Examples 3, 9 and 11). By inhibiting the engagement of TACI with ztnf4, the recited anti-TACI antibodies inhibited inflammatory activity downstream of TACI and were, therefore, useful for treating inflammatory airway disorders such as asthma, bronchitis and emphysema. This therapeutic effect of the anti-TACI antibodies would not have been obvious in the light of document (9) which failed to suggest (i) treatment of the recited medical conditions and ii) any ligand for TACI, let alone, ztnf4.

The requirement of inventive step (Article 56 EPC) was fulfilled.

Article 83 EPC; sufficiency of disclosure in relation to the subject-matter of:

C*laims 17 to 31*

Example 9 of the patent in suit provided evidence of an in vivo link between over-expression of the ligand ztnf4 and increased levels of peripheral B cells/elevated levels of IgG, IgM or IgE. It also taught amyloid deposition in the kidney. Examples 3 and 11 described the stimulation of B cell proliferation by ztnf4 in vitro. Example 12 reported the detection of elevated levels of Ztnf4 in serum samples of mice having progressed to advanced stages of autoimmune diseases. Example 13 also demonstrated that mouse models of spontaneous systemic lupus erythematosus were characterized by high serum levels of ztnf4. These data in combination with the fact that BR43x2 was able to bind ztnf4 rendered plausible that diseases associated with high levels of, in particular IgE, such as asthma, bronchitis or emphysema, B cells diseases characterized by elevated immunoglobulin levels such as light chain neuropathy, amyloidosis, inflammatory conditions in general and, more specifically those associated with such symptoms as joint pain, swelling or septic shock (claim 37), renal diseases in general and more specifically those mentioned in claims 17 and 30, autoimmune diseases involving antibody production in general, and, more specifically, those mentioned in claims 17, 28 and 29 could be treated with BR43x2 (claim 17) or anti-BR43x2 antibodies (claim 27).

These data had been obtained with the TACI receptor and not with BR43x2 itself. Yet, as BR43x2 shared the

property of TACI to bind ztnf4, and it was this property which enabled TACI to inhibit the undesirable activities of ztnf4 associated with the medical conditions recited in the claims, there was no reason to believe that the same results would not be achieved with BR43x2/anti-BR43x2 antibodies.

The plausibility of the claimed uses was backed up by post-published evidence in the form of documents (50) and (51) (asthma), document (38) (glomerulonephritis, SLE syndrome), document (48) (rheumatoid arthritis) and documents (49) and (57) (SLE).

Claim 32

Example 9 showed that ztnf4 induced increases in the levels of B cells or of IgE such as found in asthma, bronchitis or emphysema. Anti-TACI antibodies would prevent ztnf4 from binding its cellular receptor (TACI) and, therefore, from inducing such increases. Accordingly, it was entirely plausible that anti-TACI-antibodies could be used for the treatment of these medical conditions. Post-published document (50) provided confirmation of that effect.

The requirement of sufficiency of disclosure was fulfilled in relation to the subject-matter of claims 17 to 32.

XI. The appellant requested that the decision under appeal be set aside and that the patent be maintained on the basis of the main request filed during oral proceedings.

Respondent I requested in writing that any claim request having a scope that went beyond the claim request upheld by the opposition division be refused.

Reasons for the decision

Main request filed during oral proceedings
Claims 1 to 6

1. Claims 1 to 6 of this request correspond to the claims of the request accepted by the opposition division with the omission of the medical condition "renal neoplasms" in claim 1. In accordance with the principle of prohibition of reformatio in peius, a claim request which would consist exclusively of claims 1 to 6 accepted by the opposition division could not be challenged on appeal as the respondents (opponents) did not file any appeal. In contrast, the present situation is that claims 1 to 6 (with the deletion of an embodiment in claim 1) are part of a new request comprising 32 claims. Like all new requests, this request is to be considered as a whole, which means that in the framework of the appeal, claims 1 to 6 would also be open to question. However, the respondents did not object to any of these claims during opposition proceedings (see point 16 of the decision under appeal) and they also did not bring forward any arguments on appeal as to why they would not comply with the requirements of the EPC. The board will not reconsider their allowability of its own motion in the absence of any factual basis for doing so.

Article 123(2)(3) EPC
Claims 17 and 27

2. Claims 17 and 27 relate to the use of either BR43x2 (SEQ ID NO:2), its extracellular domain (SEQ ID NO: 4) or of antibodies against SEQ ID NO: 2 or SEQ ID NO: 4, for the manufacture of a medicament against, in particular, membranous nephropathy, IgA nephropathy, Berger's Disease, IgM nephropathy, Goodpasture's Disease, post-infectious glomerulonephritis, mesangioproliferative disease, minimal-change nephrotic syndrome or secondary glomerulonephritis or vasculitis associated with lupus. These specific medical conditions which are disclosed in the passage bridging pages 55 and 56 of the application as filed (Article 123(2) EPC) are not mentioned in the granted claims. However, as submitted by the appellant, they are specific subsets of the medical conditions of glomerulonephritis or vasculitis which were recited in granted claim 20. For this reason, there is no extension of the scope of the claims. The requirements of Article 123(2)(3) EPC are fulfilled.

Article 54 EPC; novelty
Claims 7 to 32

3. The decision of the opposition division to reject the granted claims for reasons of lack of novelty was taken in relation to subject-matter which is no longer claimed. In the board's judgment, none of the prior art documents on file discloses the BR43x2 polypeptide/anti-BR43x2 antibodies nor, a fortiori, their uses for the manufacture of medicaments against specific diseases (claims 7 to 31). In the same manner,

no prior art document discloses the use of antibodies against the tumour necrosis factor receptor (TNFR) TAC1 (SEQ ID NO: 6) for the manufacture of a medicament against asthma, bronchitis or emphysema (claim 32). The requirements of Article 54 EPC are fulfilled.

Articles 56 and 83 EPC; *inventive step, sufficiency of disclosure*
Product claims 7 to 16

4. These claims relate in particular to the BR43x2 polypeptide/DNA encoding it/pharmaceutical composition comprising anti-BR43x2 antibodies. The BR43x2 DNA was isolated in the course of a study of receptors of the tumour necrosis factor (TNF) ztnf4. Two known tumour necrosis factor receptors TACI and BCMA were found to have the same property as BR43x2, namely that of binding ztnf4.

5. Documents (1) or (9) may be taken as closest prior art, document (9) being the scientific publication corresponding to the patent document (1). In what follows, reference will be made to document (1). In this document, TACI is characterized by its sequence (SEQ ID NO: 2). It is described on page 18 as a cell surface protein comprising a C-terminal cytoplasmic end, a membrane spanning segment and an N-terminal extracellular domain with two cysteine-rich regions. Because of its structural features, it is acknowledged on page 19 as a member of the TNFR super family. Document (1), eg page 18 and Example, also teaches that after the cross-linking of its N-terminal end with its ligand, TACI binds at its C-terminal end with the N-terminal end of the CAML protein, which binding, in

turn, induces activation of the Ca^{+}-dependent transcription factor NF-KB. This factor is known to play an important role in the activation of lymphocytes (document (1), page 2, lines 28 to 30). Therapeutic uses are envisaged for TACI/anti-TACI antibodies/TACI ligand based on the link presumed to exist between TACI activation by ligand and B cells activation. It is suggested from pages 56 to 58, that TACI agonists such as TACI ligand could be administered for treatment of a subject in whom immune stimulation, in particular of B cells, is desired; alternatively, that suppression of TACI activity may be useful for treating undesirable immune responses such as autoimmune and inflammatory diseases including vasculitis, glomerulonephritis, systemic lupus erythromatosus (SLE) and rheumatoid arthritis (page 58, first full paragraph). The TACI ligand is, however, not identified and no data are provided which could render plausible any of the hypothesized uses.

6. Starting from the closest prior art, the problem to be solved may be defined as providing another member of the TNFR family.

7. The solution proposed is the BR43x2 polypeptide and the polynucleotide encoding it.

8. The patent in suit describes the isolation of BR43x2 DNA via the ability of the BR43x2 polypeptide - resulting from the transcription and translation of that DNA - to bind to the TNF ztnf4 (example 1). Furthermore, the sequence of BR43x2 is shown to be about 100% identical to that of the TNFR TACI in the region between amino acid 69 and amino acid 292. In

fact, the difference between the two resides in that, in the TACI region 1 to 68, BR43x2 lacks amino acids 21 to 47, ie. the first cysteine-rich region of TAC1 has been replaced by a tryptophan residue.

9. Thus, the patent in suit provides evidence that BR43x2 functions as a receptor by binding a TNF and that it shares significant homology to a member of the TNFR family. Accordingly, it is concluded that BR43x2 is itself a member of this family and, therefore, a bona fide solution to the above mentioned problem.

10. This point is indeed confirmed in post-published document (33) where BR43x2 - under the name of "ShortTACI"- is defined on page 7220, right-hand column, as "an alternative spliced form of TACI containing a single CRD" (CRD: cysteine-rich region).

11. The prior art does not in any way suggest that a shorter version of TACI could act as a TNFR; in fact the existence of such a polypeptide is not even hinted at. If, for the sake of argument, one would consider it likely, then it remains quite unexpected that, although retaining the ability to bind ztnf4, the shorter TACI version has not retained the most conserved of the TACI two cysteine-rich regions but rather the least conserved one. For this reason, the subject-matter of product claims 1 to 7 is inventive (Article 56 EPC).

12. The skilled person would have no difficulty in reproducing the claimed polypeptide on the basis of its sequence or of that of the DNA encoding it. Preparing antibodies or a pharmaceutical composition is well

within the ability of the skilled person. The requirements of Article 83 EPC are also fulfilled.

Articles 56 and 83 EPC; inventive step, sufficiency of disclosure
Use claims 17 to 31

13. It has just been established that product claims 7 to 16 fulfil the requirements for patentability. This implies that the patentability of a generic use of the claimed molecules would not need to be investigated. Yet, while being use claims in relation to BR43x2/portion thereof/ anti-BR43x2 antibodies, claims 17 to 31 have a distinctly different content from that of a "straightforward" generic use claim. Indeed, their subject-matter is rather the "therapeutic link" which may exist between the above mentioned molecules and **specific** medical conditions. For this reason, the patentability of the uses needs be assessed on its own. In accordance with the case law (T 1329/05 of 28 June 2005), a subject-matter may be regarded as a contribution to the art, ie. as an invention susceptible of being patented, if it solves the problem it is intended to solve and this should be made at least plausible by the disclosure in the patent in suit. Post-published evidence may in the proper circumstances also be taken into consideration.

14. The technical content of the patent in suit which is relevant to claiming medical uses for BR43x2/ portion thereof/anti-BR43x2 antibodies is as follows:

a)- BR43x2 is able to bind ztnf4 (Example 1);

b)- Overexpression of ztnf4 induces increases in B cells and Ig levels and is associated with the appearance of symptoms characteristic of eg. systemic lupus erythematosus, glomerulonephritis and autoimmune diseases (Examples 9 and 12, in vivo data, Example 3, in vitro data);

c)- By binding to ztnf4, TACI-Fg or -Ig inhibits its engagement with its cellular receptor and consequently, induces a decrease in B cells and Ig levels (Example 11, in vitro data, Examples 13 and 14, in vivo data).

15. In the board's judgment, this overall teaching makes it plausible that BR43x2 - which binds to ztnf4 - may be effective against medical conditions involving an increase in B cells and/or Ig levels. Here, it must be noted that the relevant data in the patent in suit regarding point c) were obtained with TACI rather than with BR43x2. However, inasmuch as, as already mentioned, both of them bind ztnf4 and this binding is responsible for ztnf4 inhibition, the board is prepared to accept the plausibility of BR43x2 being suitable for treating the specific diseases mentioned in the claims insofar as they are associated with high levels of B cells or immunoglobulins.

16. Plausibility is confirmed in post-published documents or by experimental data on file. Thus, document (50), page 19, discloses that delivery of TACI-Ig to mouse models of asthma inhibits binding of ztnf4 (identified as BLys) to its cellular receptor and results in B cell depletion and therefore, in a decrease of IgE production. The equivalent teaching is found in

document (51). Documents (49) and (57) confirm that delivery of TACI-Ig to SLE patients induces a reduction in B cells and Ig levels. Prior art document (38), passage bridging pages 995 and 996, discloses that mice which overexpress ztnf4 have elevated levels of B cells, IgM, IgG and IgE and develop symptoms characteristic of SLE such as glomerulonephritis and proteinuria. As for document (48), it teaches that delivery of TACI-Ig to patients suffering from rheumatoid arthritis results in a decrease in IgM and in mature B cells.

17. There is, thus, no doubt as regards the patent specification providing a contribution to the problem of treating the diseases mentioned.

18. For the skilled person, it would have been fully unexpected that a tumour necrosis factor receptor derived from TACI could be put to the claimed uses even taking into account the closest prior art document (1). Indeed, as mentioned above, document (1) is wholly silent as to which ligand will bind TACI and whereas it mentions therapeutic uses, it is on the basis of mere assumptions. In the board's judgment, without the knowledge established in the patent in suit that the TACI ligand is ztnf4 and that the effect of ztnf4 is to increase levels of B cells and immunoglobulins, these assumptions are devoid of substance and, thus, unlikely to affect inventive step. There is no other document on file which, when combined with document (1), would render the claimed subject-matter obvious. Thus, the specific uses of the inventive BR43x2 polypeptide are themselves inventive.

19. For the same reasons, sufficiency of disclosure is achieved for the claimed uses above the obvious sufficiency of disclosure of manufacturing a medicament comprising a reproducible compound (BR43x2,...), that is, it is achieved in relation to the treatment of the specifically mentioned diseases.

20. Claims 17 to 31 fulfil the requirements of the EPC.

Use claim 32

21. The reasoning developed under Articles 56 and 83 EPC in relation to the use of BR43x2/anti-BR43x2 antibodies for the manufacture of a medicament for the treatment of specific diseases applies all the more to the use of anti-TACI (SEQ ID NO: 6) antibodies for the manufacture of a medicament against asthma, bronchitis or emphysema because, as already mentioned above, the experiments relevant in this context were performed with TACI itself. Post-published document (50) provides confirmation of the fact that asthma is linked to an increase in IgE levels which can be treated with TACI-Ig. The claim fulfils the requirements of the EPC.

Order

For these reasons it is decided that:

1. The decision under appeal is set aside.

2. The case is remitted to the first instance with the order to maintain the patent on the basis of claims 1 to 32 of the main request filed during the oral proceedings and a description and figures to be adapted thereto.

The Registrar | The Chairman

A. Wolinski | L. Galligani