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Datasheet for the decision of 16 October 2014

Case Number: T 0848/10 - 3.3.08

Application Number: 00957502.8

Publication Number: 1210425

IPC: C12N15/12, C07K14/715, A61K38/17, A61K39/395

Language of the proceedings: EN

Title of invention:
BAFF RECEPTOR (BCMA), AN IMMUNOREGULATORY AGENT

Patent Proprietor:
Biogen Idec MA Inc.
Apotech R&D S.A.

Opponent:
Amgen Inc.

Headword:
BAFF receptor BCMA/BIOGEN IDEC, APOTECH

Relevant legal provisions:
EPC Art. 123(2), 84, 83, 54, 56

Keyword:
Admissibility: Main request (yes)
Main request - requirements of the EPC met (yes)

Decisions cited:

Catchword:

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Case Number: T 0848/10 - 3.3.08

D E C I S I O N
of Technical Board of Appeal 3.3.08
of 16 October 2014

Appellant: (Patent Proprietor 1)	Biogen Idec MA Inc. 14 Cambridge Center Cambridge, Massachusetts 02142 (US)
Appellant: (Patent Proprietor 2)	Apotech R&D S.A. 84, rue du Rhône 1204 Genève (CH)
Representative:	Pohlman, Sandra M.and Hilgendorf, Annette df-mp Dörries Frank-Molnia & Pohlman Patentanwälte Rechtsanwälte PartG mbB Theatinerstrasse 16 80333 München (DE)
Appellant: (Opponent)	Amgen Inc. One Amgen Center Drive Thousand Oaks, CA 91320-1799 (US)
Representative:	Care, Alison Kilburn & Strode LLP 20 Red Lion Street London WC1R 4PJ (GB)
Decision under appeal:	**Interlocutory decision of the Opposition Division of the European Patent Office posted on 2 February 2010 concerning maintenance of the European Patent No. 1210425 in amended form.**

Composition of the Board:

Chairman M. Wieser
Members: B. Stolz
D. Rogers

Summary of Facts and Submissions

I. The patent proprietors (collectively referred to as appellant I) and the opponent (appellant II) each lodged an appeal against the interlocutory decision of the opposition division, whereby European patent No. 1 210 425, granted on European patent application No. 00957502.8 (published as international application WO 01/12812) was maintained on the basis of the fifth auxiliary request.

II. Appellant I re-filed the main request and the five auxiliary requests which were the subject of the appealed decision.

III. Each of the appellants replied to the other party's statement of grounds. Appellant II's reply was accompanied by a new document, D22.

IV. The parties were summoned to oral proceedings. A communication pursuant to Article 15(1) of the Rules of Procedure of the Boards of Appeal (RPBA) annexed to the summons, informed them of the preliminary non-binding opinion of the board on some of the issues of the appeal proceedings.

V. Appellant I made additional submissions, and filed a new main request and new auxiliary requests 1 to 39, replacing the requests previously on file.

Appellant II informed the board that it would not attend oral proceedings.

VI. Oral proceedings were held on 16 October 2014, in the absence of appellant II. Appellant I withdrew the main request and made auxiliary request 1 its main request.

VII. The main request consists of 11 claims of which independent claims 1 and 2 read as follows:

"1. (a) A polypeptide which consists of an amino acid sequence which is at least 80% identical to the sequence set forth from amino acid 1 to amino acid 51 of SEQ ID NO:1;

(b) an antibody directed against SEQ ID NO:1, wherein the antibody is an antagonistic monoclonal antibody; or

(c) a polypeptide which is the extracellular domain of BCMA and comprises the amino acid sequence set forth from amino acid 8 to amino acid 41 of SEQ ID NO:1

for use as a medicament.

2.Use of

(a) a polypeptide which consists of an amino acid sequence which is at least 80% identical to the sequence set forth from amino acid 1 to amino acid 51 of SEQ ID NO:1;

(b) an antibody directed against SEQ ID NO: 1, wherein the antibody is an antagonistic monoclonal antibody; or

(c) a polypeptide which is the extracellular domain of BCMA and comprises the amino acid sequence set forth from amino acid 8 to amino acid 41 of SEQ ID NO:1

for the preparation of a pharmaceutical composition for treating an autoimmune disorder, a B-cell lymphoproliferative disorder, or inflammation in a mammal."

Dependent claims 3 to 11 define specific embodiments of claim 2.

VIII. The following documents are referred to in this decision:

D1: WO 00/40716, 13 July 2000.

D5: CHRISTINE MADRY ET AL.: "The characterization of murine BCMA gene defines it as a new member of the tumor necrosis factor receptor superfamily" INTERNATIONAL IMMUNOLOGY, vol. 10, 1998, pages 1693-1702.

D6: MARIE-PIERRE GRAS ET AL.: "BCMAp: an integral membrane protein in the Golgi apparatus of human mature B lymphocytes" INTERNATIONAL IMMUNOLOGY, vol. 7, no. 7, July 1995 (1995-07), pages 1093-1106.

D14: BARBARA SCHIEMANN ET AL. "An essential role for BAFF in the normal development of B cells through a BCMA-independent pathway" SCIENCE, vol. 293, 14.9.2001, pages 2111-2114.

D18: APRIL CHIU ET AL.: "Hodgkin lymphoma cells express TACI and BCMA receptors and generate survival and proliferation signals in response to BAFF and APRIL" BLOOD, vol. 109, no. 2, 15.1.2007, pages 729-739.

D21: BRIAN P. O'CONNOR ET AL.: "BCMA is essential for the survival of long lived bone marrow plasma cells", J. EXP. MED., vol. 199, no. 1, 5.1.2004, pages 91-97.

D22: WO1999/013078, published 18 March 1999

D24: CHRISTIAN JORGENSEN ET AL.: "Immunological evaluation of cytokine and anticytokine immunotherapy in vivo: what have we learnt?", Ann. Rheum. Dis., vol. 58; March 1999, pages 136-141.

IX. The arguments of appellant I, as far as relevant for the present decision, can be summarized as follows:

Article 83 EPC

Opponent's objections were not based on facts but on speculation. The patent provided a ligand, BAFF, for the BCMA receptor and described that antibodies against BCMA block the action of BCMA. The case law cited by the opponent did not support opponent's position. The present patent clearly suggested the use of anti BCMA antibodies as antagonists which would be useful for treating disorders involving B cell action such as autoimmune disorders, B cell lymphoproliferative disorders or inflammation. Examples 6 and 7 outlined methods for the testing and screening of antagonistic antibodies. The examples with soluble BCMA provided a basis which rendered the use of antagonistic BCMA antibodies plausible. These conclusions were supported by the post published documents on file.

Article 54 EPC

The claims were entitled to priority as could be seen from the paragraphs on pages 2 and 7, claims 85 and 86, Figure 2 and Example 6 of the first priority document. Therefore, documents D1 to D3 were not available for the assessment of novelty and inventive step. The claimed subject matter was new.

Article 56 EPC

Document D5 described the murine BCMA receptor as a membrane protein and a new member of the superfamily of TNF-receptor proteins but did not disclose any possible ligands. Its structure differed from the structure of other TNF receptor family members. At the filing date, well over a dozen various TNF family ligands were known to be involved in an array of diverse physiological processes. Document D5 described the BCMA protein as primarily located in perinuclear structures. Similar information concerning the location of the protein could be found in document D6. Therefore, and in the absence of a known ligand, a role of the receptor in B cell related diseases was not obvious. Document D22 was not at all concerned with BCMA and not a textbook representing general knowledge.

X. The arguments of appellant II, as far as relevant for the present decision, can be summarized as follows:

Article 83 EPC

The medical results obtained with the soluble fragment of the receptor BCMA could not predict the results obtained with an antagonistic antibody against BCMA. BAFF was one of two ligands, the other being APRIL,

binding to the BCMA receptor, and BAFF itself bound to at least three receptors, i.e. BAFF-R, BCMA and TACI. While the soluble BCMA receptor sequestered all ligands binding to the native BCMA receptor, the results of the inhibition of BAFF signalling through the BCMA receptor by anti BCMA antibodies were unpredictable. It was not disputed that the patent recited antibodies as potential antagonists, however, in view of the complex signalling network it was questionable whether such antibodies would in fact be useful for the treatment of B-cell disorders. Testing for an inhibitory activity of antibodies would be futile, since alternative signalling pathways through BAFF-R and TACI existed and hence B-cell proliferation, would not have been inhibited. This conclusion was supported by document D14 which showed that a BCMA knockout mouse had no detrimental phenotype. Therefore, the invention as defined by claims 1(b) and 2(b) could not be performed without an undue burden.

Article 54 EPC

The subject matter of claims 1 and 2 was not directly and unambiguously disclosed in the first priority document. In particular, there was no basis for the medical use of polypeptides comprising the amino acid sequence set forth from amino acid 8 to amino acid 41 of Seq ID NO:1 (claims 1(c) and 2(c)) and for peptides with at least 80% identity to the sequence set forth from amino acids 1 to 51 of Seq ID NO:1 (claims 1(a) and 2(a)). Documents D1 to D3 therefore represented prior art under Article 54(2) EPC and each anticipated the claimed subject matter.

Article 56 EPC

Document D5 provided detailed structural information about the human and murine BCMA and identified them as members of the TNF receptor superfamily. At the bottom of page 1693, in Figures 3 and 6, and on page 1701, it explicitly described the extracellular region of BCMA, and suggested a possible role of BCMA in the regulation and development of the immune system. It explained that each member of the TNF receptor family had an extracellular ligand binding domain and a cognate ligand. It was not disputed that the ligand to the BCMA receptor was unknown at the filing date but the underlying technical problem did not consist in identifying this ligand. It was not necessary to know the ligand to the receptor in order to develop a soluble receptor suitable for use in treating diseases with an aspect of inappropriate B cell proliferation. The fact that, at the filing date, the skilled person was not aware of the existence of multiple ligands and receptors in the signalling pathway rendered the claimed solutions all the more obvious. Document D22 showed that soluble receptor fragments of anti receptor antibodies to a new member of the TNF receptor superfamily, TRAIN-R, were therapeutically useful, even in the absence of a known ligand. The selection of the particular extracellular domain fragments as defined by the claims, required no inventive skills.

XI. Appellant I requested that the decision under appeal be set aside and that the patent be maintained on the basis of claims 1 - 11 of the Main Request submitted at the oral proceedings before the Board.

XII. Appellant II requested that the decision under appeal be set aside and the patent be revoked.

Reasons for the Decision

1. The main request is based on the second auxiliary request underlying the decision under appeal. Said second auxiliary request was resubmitted with the grounds of appeal and amended in response to the preliminary communication of the board in which it expressed its view on an objection under Article 83 EPC. The amendment limits the antibodies of claims 1(b) and 2(b) to antagonistic monoclonal antibodies.

2. Appellant II did not raise any objections against the amendment which does not add to the complexity of the case. Therefore, the main request is admitted into the proceedings.

Article 123(2) EPC

3. No objections under Article 123(2) and 123(3) EPC were raised against the second auxiliary request underlying the appealed decision.

The additional amendment to part (b) of claims 1 and 2 can be directly and unambiguously derived from the definition of antibodies as specifically covering monoclonal antibodies against the BAFF receptor, including agonist, antagonist and neutralizing antibodies (page 9, lines 5 to 11, of WO 01/12812, the international publication of the patent application, further on referred to as the application as filed), and from the paragraph disclosing the use of mono- and polyclonal antibodies to block the action of the BAFF receptor (page 13, lines 18 to 21, and Example 6).

Compared to the claims as granted, the amendment leads to a limitation of the scope of protection.

The board is therefore satisfied that the requirements of Article 123(2) and 123(3) EPC are met.

Article 84 EPC

4. Appellant II submitted that the term "the extracellular domain of BCMA" rendered part (c) of claims 1 and 2 unclear. It submitted that the lack of clarity resulted from the lack of an indication of the exact length of the extra cellular domain of BCMA.

5. Part (c) of claims 1 and 2 is directed to

"a polypeptide which is the extracellular domain of BCMA and comprises the amino acid sequence set forth from amino acid 8 to amino acid 41 of SEQ ID NO:1".

6. It is not disputed that the skilled person understands the meaning of the term "extracellular domain", however, the boundaries of this domain are not precisely defined. The boundaries may vary by a few amino acids depending on the method used for their determination. In line with the general understanding, the patent states that the term "extracellular domain of BAFF-R (BCMA)" refers to a form of BAFF-R which is essentially free of transmembrane and cytoplasmic domains of BAFF-R, and will ordinarily have less than 1% of such cytoplasmic and transmembrane domains (page 7, lines 16 to 19).

7. The skilled person therefore understands that the reference to the extracellular domain of BCMA in part (c) of claims 1 and 2 is not a reference to a

polypeptide of a precisely defined length. It is however clear that this term does not include polypeptides comprising transmembrane and intracellular domains. Since methods for predicting the position of the transmembrane domain, which separates the extracellular from the cytoplasmic domain of a BCMA receptor protein, are available (cf. e.g. document D5, Figures 3 and 6), the skilled person can determine whether a polypeptide comprising amino acids 8 to 48 of SEQ ID NO:1 is a polypeptide consisting of the extracellular domain of a BCMA protein.

8. Thus, the requirements of Article 84 EPC are met.

Article 83 EPC

9. According to established case law, if a therapeutic use of a substance is claimed, attaining the claimed therapeutic effect is a functional feature of the claim.

10. For the purpose of Article 83 EPC, a therapeutic effect may well be acknowledged on the basis of any data that show a clear relationship between the physiological activities of this substance and the disease, or a direct effect of this substance on a metabolic mechanism specifically involved in the disease (cf. "Case Law of the Boards of Appeal of the EPO", 7th edition 2013, II.C.6.2, page 318).

11. At the filing date BCMA was known as an orphan receptor without a known ligand and without a known role in physiology (cf. document D5).

12. The patent discloses a ligand for BCMA termed BAFF (Examples 1 to 3) and the generation of soluble

receptor forms comprising the extracellular domain of BCMA (Example 5). Further examples show that the sequestering of BAFF by soluble BCMA blocks B cell proliferation in vitro (Example 9), attenuates auto-immune diseases, slows progression of nephritis and leads to a reduction of DC cells in vivo (Examples 11 to 14).

13. With respect to antagonistic anti-BCMA antibodies the patent discloses that "blocking agents, such as recombinant variants or antibodies specific to the receptor, have immunoregulatory applications as well" (page 3, line 11). Furthermore, the patent discloses "methods of inhibiting inflammation by administering an antibody specific for BCMA or an epitope thereof" (page 4, line 23). Example 6 discloses a screening assay to identify antibodies lacking agonist activity and inhibiting receptor ligand interactions (in the present case BCMA BAFF interactions, since BAFF was the only ligand known and disclosed by the patent). Example 7 describes a cell line suitable for transformation with the BCMA receptor in order to provide a functional screening assay. Example 9 discloses an in vitro assay to study the effect of BAFF on B cell proliferation. Finally animal models are disclosed which were used to assay the effect of soluble BCMA (Examples 12 to 14).

14. Post published documents such as document D18 disclose that not only BAFF but also a further ligand, termed APRIL, bind to and signal through the BCMA receptor. Moreover, BAFF and APRIL bind to and signal not only through the BCMA receptor but through at least two further receptors termed TACI and BAFF-R (Note that the BAFF-R receptor molecule disclosed in document D18 is a different molecule than the molecule of SEQ ID NO: 1

for which the present patent uses the terms BAFF-R and BCMA synonymously).

15. Appellant II did not dispute that the patent disclosed the medical uses of the polypeptides as defined by parts (a) and (c) of claims 1 and 2. It also agreed that the patent sufficiently disclosed the raising of anti BCMA antibodies. It submitted, however, that the medical use of antibodies as defined by part (b) of claims 1 and 2 was not sufficiently disclosed. While the effect of sequestering BAFF and preventing it from signaling through all three receptors was demonstrated in the patent under appeal, the effect of blocking the signalling through the BCMA receptor with an antibody remained speculative. The appellant questioned whether such antibodies would in fact be useful as BCMA antagonists for the treatment of B-cell disorders and submitted that testing for an antagonistic effect on B-cell proliferation would be futile since BAFF could still signal through the alternative receptors.

16. There is no evidence that the presence of multiple ligands and receptors is an indication of complete redundancy of the signalling pathways. Rather this is an indication of only partially overlapping functions. In the present case the sequestering of BAFF affected B cell proliferation despite the fact that the (at the filing date unknown) second ligand, APRIL, could still signal through the BCMA receptor. Document D24 describes the results from several immune therapy trials of different cytokines with overlapping effects. The data show that inhibition of individual cytokines can be therapeutically efficient, despite the fact that the cytokines have overlapping effects. Therefore, there is no reason to assume that the skilled person, purely upon the basis that the BAFF signalling network

is more complex than originally thought, would not be in a position to readily identify antagonistic antibodies.

17. Appellant II also referred to document D14 as evidence that the blocking of BCMA could not have an effect on B cell proliferation, and that therefore no meaningful physiological test could be performed to isolate the claimed antibodies.

18. Document D14, published two years after the filing date, discloses experiments performed in order to establish the role of BAFF in BCMA mediated signalling. The authors of document D14 created mice deficient in either BAFF or BCMA. The $BCMA^{-}/^{-}$ mice are described as "outwardly normal" (page 2112, column 3, second paragraph) up to at least 6 to 8 months of age, as containing normal proportions of B cells in the bone marrow (page 2113, left column, last paragraph), and as having normal appearing B lymphocyte compartments (abstract).

Document D21, on the other hand, published four years after the filing date, provides evidence that BCMA is indeed essential for the survival of long-lived bone marrow plasma cells which are important for sustained humoral immune responses. BCMA knockout mice had drastically reduced numbers of antibody secreting cells (page 95, left column, last paragraph).

19. The board is therefore not convinced by the evidence on file that physiologically meaningful tests, such as the ones disclosed by Examples 9 or 11 to 14, could not be performed at all or only with an undue amount of work.

20. In the absence of convincing arguments and verifiable facts showing that antagonistic antibodies could only be obtained with undue burden and considering that the patent discloses functional in vitro and in vivo assays (cf. point 12 above),the board concludes that the main request meets the requirements of Article 83 EPC.

Article 54 EPC

Priority

21. Appellant II submitted that the first priority document, US 149378P, did not disclose a polypeptide consisting of amino acids 1 to 51 of Seq ID NO: 1, let alone the medical use of a polypeptide according to part (a) of claims 1 and 2.

22. The first priority document contains no explicit disclosure of the subject matter of part (a) of claims 1 and 2. It needs therefore to be established whether the claimed subject matter is implicitly disclosed in a direct and unambiguous way.

23. The first priority document generally describes the disclosed invention as relating to the use of a receptor to BAFF, and its blocking agents to stimulate or inhibit the expression of B cells (page 2, lines 4 to 6). More specifically it states: "the invention provides methods of using BAFF-R" (page 4, line 25), and "included are methods of inhibiting B cell growth, dendritic cell induced B cell growth and maturation of immunoglobulin production in an animal using BAFF-R polypeptide" (page 5, lines 1 and 2). Furthermore it is disclosed that, "the invention provides methods of using BAFF-R in the treatment of autoimmune diseases,

Bcell lymphoproliferative disorders, and inflammation" (page 15, lines 20 to 22).

Thus, the priority document generally discloses the use of "BAFF-R polypeptides" for the medical use of claims 1 and 2.

24. According to the priority document, the term "BAFF-R polypeptide" encompasses "native sequence and BAFF-R variants (which are further defined herein)" (page 7, lines 6 to 7). Furthermore, "BAFF-R variant means an active BAFF-R as defined below having at least about 80% sequence identity with the BAFF-R having the deduced amino acid sequence shown in Seq ID NO:1 for a full-length native sequence BAFF-R or with a BAFF-R ECD sequence" (page 7, line 30 to page 8, line 2). BAFF-R ECD (extracellular domain) "refers to a form of BAFF-R which is essentially free of transmembrane and cytoplasmic domains of BAFF-R" (page 7, lines 19 to 20). "Optionally, BAFF-R ECD will comprise amino acid residues 8 to 41 of SEQ ID NO:1" (page 7, line 23).

Thus, the priority document explicitly discloses the use of the polypeptide defined in part (c) of claims 1 and 2.

25. Figure 2 of the priority document discloses a BAFF-R fragment consisting of amino acids 1 to 51 which represents an extracellular domain fragment within the definition given in the paragraph bridging pages 7 and 8 (cf. point 23, above).

The board concludes therefore, that the subject matter of part (a) of claims 1 and 2 is directly and unambiguously derivable from the first priority document.

26. As for part (b) of claims 1 and 2, the paragraphs of the patent application providing the basis for amended part (b) of claims 1 and 2 (cf. point 3, above) can be literally found on pages 9 (lines 9 to 11) and 13 (lines 13 to 15), and in Example 6 of the first priority document.

27. The subject-matter of the claims of the main request is directly and unambiguously derivable from the first priority document. Hence, the relevant date for the assessment of novelty is the filing date of the first priority document, 17 August 1999, which has the consequence that documents D1 to D3 do not belong to the state of the art according to Article 54(2) EPC.

28. None of the prior art documents on file discloses the subject matter of the main request, which therefore meets the requirements of Article 54 EPC.

Article 56 EPC

29. Document D5 represents the closest prior art. It discloses the characterization of the murine BCMA gene. By sequence comparison, the gene was identified as a new member of the tumor necrosis factor receptor (TNF-R) superfamily. Its mRNA was primarily found in lymphoid tissues.

30. In the light of this disclosure, the technical problem underlying the patent in suit is the provision of means for treating B cell related disorders, in particular autoimmune disorders and inflammation.

31. As a solution to this problem, the patent proposes the use of the polypeptides and antibodies according to

claims 1 and 2. The patent discloses BAFF as a ligand for BCMA and the experimental evidence provided by the examples shows that the inhibition of BAFF signalling by a soluble BCMA-Ig fusion protein indeed antagonized the effect of BAFF in a mouse model of an autoimmune disease (Example 11), slowed progression of an inflammatory disease (Example 13) and had an effect on B cell proliferation (Examples 9 and 14).

32. The board is therefore satisfied that the proposed solution indeed solves the underlying technical problem.

33. It remains to be examined whether the claimed solution involves an inventive step.

34. Appellant II submitted that the claimed solution was obvious on the basis of document D5 in combination with the general knowledge of the skilled person. In particular, it was argued that the authors of document D5 stated that the expression pattern of BCMA suggested a role of the protein in the development and regulation of the immune system and that the identification of its cognate ligand would greatly improve the understanding of the physiological role of BCMA (final paragraph). The steps of identifying the ligand and testing ligand receptor signalling for physiological effects on B cells were therefore obvious. This sequence of events was obvious as could be seen from document D22 disclosing a new member of the TNF receptor superfamily without a known ligand. Although no ligand to the receptor was identified, a patent was granted with claims to the therapeutical use of antibodies against the receptor.

35. The board agrees that the general sequence of steps on the way to analysing the physiological role of a receptor molecule includes identification of a ligand and in vitro or in vivo assays.

However, as agreed by appellant II (cf. page 5 of the letter of 7 June 2010), document D5 does not suggest at all that BAFF or any other known molecule might be acting as a ligand for BCMA, nor does any other document on file. Moreover, by reference to document D6, the authors of document D5 describe the BCMA receptor as a protein which was found in a perinuclear structure which partially overlapped the Golgi apparatus (page 1694, middle paragraph). The amino acid sequence showed no immediate similarities with other members of the TNF receptor family (page 1698, right column, second paragraph). Only a comparison by a helical cluster analysis revealed the presence of certain conserved cysteine residues and certain conserved amino acids in the cysteine rich extracellular domain. The authors of D5 stated that the cysteine rich domain of the BCMA receptor does not exactly match the signature sequence of the TNFR/NGFR family of proteins page 1699, left column, 2nd paragraph).

36. The disclosure of document D5, in particular the reference to an intracellular location of BCMA and the quite distant relatedness to the TNF receptor superfamily, would have created strong doubts whether the BCMA receptor represented a suitable candidate for treating B cell related disorders. While the skilled person, based on the disclosure of document D5 and the general knowledge, could have tried whether BCMA represented a candidate molecule, he would not have done so with a reasonable expectation of success.

37. The board concludes therefore, that the skilled person trying to solve the above mentioned technical problem and starting from the disclosure in document D5, would not have arrived at the claimed solution in an obvious way. The subject matter of the main request therefore involves an inventive step.

Adaptation of the description

38. At the oral proceedings, the appellant submitted amended pages 3 to 23 of the description to bring it in line with the main request. The board is satisfied that this has been done in agreement with 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 department of first instance with the order to maintain the patent as amended in the following version:

   Description: pages 3 - 23 received at the oral proceedings of 16 October 2014

   Claims: No. 1 - 11 of the Main Request received at the oral proceedings of 16 October 2014

   Figures: No. 1 - 16 of the patent as granted.

The Registrar: The Chairman:

A. Wolinski M. Wieser

Decision electronically authenticated