

Internal distribution code:

- (A) [-] Publication in OJ
(B) [-] To Chairmen and Members
(C) [-] To Chairmen
(D) [X] No distribution

**Datasheet for the decision
of 4 May 2017**

Case Number: T 0047/12 - 3.3.08

Application Number: 04017077.1

Publication Number: 1529844

IPC: C12N15/62, C12N15/09

Language of the proceedings: EN

Title of invention:

Complex formation for the stabilisation and purification of proteins of interest

Patent Proprietor:

Fraunhofer-Gesellschaft zur Förderung der angewandten Forschung e.V.

Opponent:

Takeda GmbH

Headword:

Protein complex formation/FRAUNHOFER GESELLSCHAFT

Relevant legal provisions:

EPC Art. 123(2), 84, 83, 54, 56

Keyword:

Main request - requirements of the EPC met (yes)

Decisions cited:

G 0003/14

Catchword:



Beschwerdekammern
Boards of Appeal
Chambres de recours

European Patent Office
D-80298 MUNICH
GERMANY
Tel. +49 (0) 89 2399-0
Fax +49 (0) 89 2399-4465

Case Number: T 0047/12 - 3.3.08

D E C I S I O N
of Technical Board of Appeal 3.3.08
of 4 May 2017

Appellant: Fraunhofer-Gesellschaft zur Förderung der
(Patent Proprietor) angewandten Forschung e.V.
Hansastraße 27c
80686 München (DE)

Representative: Weinzierl, Gerhard
Koch, Andreas
Schiweck, Weinzierl & Koch
Patentanwälte Partnerschaft mbB
Landsberger Straße 98
80339 München (DE)

Respondent: Takeda GmbH
(Opponent) Byk-Gulden-Strasse 2
78467 Konstanz (DE)

Representative: Huenges, Martin
Maiwald Patentanwalts GmbH
Elisenhof
Elisenstrasse 3
80335 München (DE)

Decision under appeal: **Decision of the Opposition Division of the
European Patent Office posted on 31 October 2011
revoking European patent No. 1529844 pursuant to
Article 101(3) (b) EPC.**

Composition of the Board:

Chairman P. Julià
Members: B. Stolz
 R. Winkelhofer

Summary of Facts and Submissions

I. The patent proprietor (appellant) filed an appeal against the decision of an opposition division revoking European patent No. 1 529 844, with the title "*Complex formation for the stabilisation and purification of proteins of interest*". The opposition division found that the main request (the claims as granted) lacked novelty and that the auxiliary request lacked an inventive step.

II. Claim 1 as granted reads as follows:

"1. A method for altering the properties and/or generating novel properties of a recombinant target protein, in short the target protein, whereby the properties are selected from the group of accumulation ability, stability and/or integrity, cellular localisation, posttranslational modification, amenability to isolation; purification and phase partitioning, by providing a specific binding partner to the natural or recombinant target protein comprising a tag and/or a targeting sequence, by means of

a) expressing a gene coding for the target protein in a non-human host;

b) expressing a gene encoding the specific binding partner for the target protein or providing the specific binding partner for said target protein in a different manner in said non-human host;

c) interactions between the target protein and the binding partner yielding a complex and thereby linking the properties of the binding partner to said target protein, wherein the complex formation leads to the

accumulation of the target protein, and/or increases the stability and/or integrity of the target protein, and/or affects the subcellular localisation of the target protein, and/or alters the post-translational modification pattern of the target protein and/or the binding partner, and/or enables co-purification of the target protein with its binding partner, and/or affects phase partitioning of the target protein."

- III. With the statement setting out its grounds of appeal, the appellant filed a new main request and new auxiliary requests 1 to 7.
- IV. The opponent (respondent) replied to the statement of grounds of appeal.
- V. 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 provisional, non-binding opinion of the board on some of the issues of the appeal proceedings.
- VI. The appellant replied to the board's communication and submitted a new main request and a new auxiliary request 1.
- VII. The respondent informed the board that it would not attend the oral proceedings.
- VIII. Oral proceedings were held on 4 May 2017 in the absence of the respondent.

IX. Claim 1 of the new main request reads as follows:

"1. A method for altering cellular localisation, posttranslational modification or amenability to phase partitioning of a recombinant target protein by providing a specific binding partner to the recombinant target protein which binding partner comprises a tag and/or a targeting sequence, by means of

a) expressing a gene coding for the target protein in a non-human host;

b) expressing a gene encoding the specific binding partner for the target protein or providing the specific binding partner for said target protein in a different manner in said non-human host;

c) interactions between the target protein and the binding partner yielding a complex and thereby linking the properties of the binding partner to said target protein, wherein the complex formation affects the subcellular localisation of the target protein, alters the post-translational modification pattern of the target protein and/or the binding partner, or affects phase partitioning of the target protein."

Dependent claims 2 to 17 define specific embodiments of the method of claim 1.

X. The following documents are cited in this decision:

D1: E. Stoger *et al.*, *Molecular Breeding*, 2002, Vol. 9, 149-158;

D2: D.M. Cox *et al.*, *BioTechniques*, 2002, Vol.33,
267-269;

D3: WO 03/100021 (publication date: 4 December 2003);

D4: S. Honey *et al.*, *Nucleic Acids Research*, 2001, Vol.
29, No. 4 e24, 1-9;

D5: S. Bagga *et al.*, *The Plant Cell*, 1997, Vol.9,
1683-1696.

XI. The arguments of the appellant, as far as relevant for this decision, are summarised as follows:

Admission of the main request

The amendments introduced into the main request were a direct reaction to the provisional opinion of the board expressed in its communication. The main request differed from the requests previously on file by the deletion of contentious subject-matter and claims. For clarification, claim 1 was amended to indicate that the binding partner comprised the tag and/or targeting sequence. Claim 10 was amended to align it with claim 1. The amendments raised no new issues and streamlined the procedure.

Article 123(2) EPC

Claim 1 of the main request was entirely based on granted claim 1 which was found by the opposition division not to contravene Article 123(2) EPC. The specification in claim 1 that the binding partner comprised the tag and/or targeting sequence was based on the disclosure on page 7, lines 11 and 12 of the patent application.

Article 54 EPC

Document D1 did not describe a method for altering the subcellular localisation of a protein where the alteration was a consequence of the formation of a complex with a specific binding partner. On page 152, right-hand column, it was disclosed that an antibody and its antigen were co-expressed in a plant apoplast. However, the antibody remained expressed in the apoplast whether expressed alone or together with its antigen. Document D1 contained no further information regarding the nucleic acid constructs used in this experiment. It was therefore not possible to conclude that the subcellular localisation of either of the two proteins was altered by an interaction with the other.

Document D3 described fusion proteins comprising peptide sequences that induced the formation of inclusion bodies. Inclusion bodies were unordered agglomerates of proteins which were not properly folded. There was therefore no complex formation between a target protein and a specific binding partner. The "inclusion body fusion partner" represented merely an unspecific binding partner which was only aggregated to the target protein but it did not form a complex with it. The fusion constructs of document D3 fell thus outside the scope of claim 1.

Documents D2 and D4 were not concerned with the alteration of the subcellular localisation, post-translational modification or amenability to phase partitioning.

Article 56 EPC

D1, disclosing methods for altering the accumulation, stability and integrity of a recombinant antibody in plants, represented the closest state of the art. Starting from this document, the technical problem could be defined as the provision of ways for altering the subcellular localisation, post-translational modification or amenability to phase partitioning of a recombinant target protein. The solution defined by claim 1 involved the formation of a complex between the recombinant target protein and a specific binding partner, thereby linking the properties of the binding partner to the recombinant target protein.

Table 1 of the patent provided suitable targeting sequences which, when linked to a specific binding partner of a recombinant target protein, redirected said recombinant target protein to a different subcellular compartment as a consequence of complex formation between the two proteins. This was confirmed in the example described in the patent. The claimed method was not limited to expression in plants. Complex formation between proteins expressed in prokaryotic, yeast and mammalian host cells was well known in the art, as evidenced, for instance, by documents D1, D2 and D4. Concerning the provision of a specific binding partner in a non-human host in a manner different from co-expression, the specific binding partner could, for instance, be linked to a cell penetrating peptide the use of which was well known, e.g. the use of the HIV tat protein. The specific binding partner could also be introduced into a non-human host cell by viruses or by other well-known methods used for the transformation of cells, such as transformation with PEG or with gold particles. All this information and methods belonged to the common general knowledge of a skilled person.

The claimed solution was not obvious in the light of D1, either alone or in combination with any of the other documents on file, in particular documents D2, D4 and D5. The section of document D1 concerned with intracellular targeting (page 152, left-hand column) did not mention co-expression to affect the cellular localisation. The paragraph in the right-hand column of page 152 described that the co-expression of a recombinant target protein (antibody) and its specific binding partner (antigen) resulted in the accumulation of the target protein due to a stabilizing effect conferred by the complex between the recombinant target protein and its specific binding partner. However, document D1 was silent about any other effect resulting from the said complex formation. There was thus no hint in document D1 towards the claimed solution.

XII. The arguments of the respondent, submitted in writing, are as follows:

The main request did not fulfill the requirements of Articles 54 and 56 EPC for the reasons given in the opposition brief dated 12 May 2009, in particular pages 26 to 29, the submissions of 16 June 2010, in particular pages 14 to 17, and the submissions made at the oral proceedings on 21 September 2011 as reflected by the minutes, in particular items 7, 19, 21, 24 and 27.

The arguments presented in the opposition brief, in particular pages 8 to 10, and in the submissions of 16 June 2010, in particular pages 3 to 6, with respect to insufficiency of disclosure applied equally to the main request.

- XIII. The appellant requests that the decision under appeal be set aside and a patent be granted on the basis of the main or auxiliary requests filed with its submission dated 25 April 2017.
- XIV. The respondent requests that the appeal be dismissed or that the case be remitted to the opposition division for a decision on the grounds for opposition under Article 100(b) EPC, should the board come to the conclusion that the main request or one of the auxiliary requests fulfil the requirements of Articles 54 and 56 EPC.

Reasons for the Decision

Respondent's submissions

1. In its reply to the appellant's statement of grounds of appeal, the respondent referred only to the submissions made at first instance (cf. point XII, above).
2. When examining the admissibility of an appeal, a statement of grounds of appeal referring generally to submissions made at first instance, as a rule, cannot be considered sufficient (cf. "Case Law of the Boards of Appeal of the EPO", 8th edition 2016, IV.E.2.6.4.a), page 1102). In the board's view, this applies equally to the submissions of other parties to the proceedings even if the decision underlying the appeal was in their favour. It cannot be left to the board or to the other parties to the proceedings to look for arguments, facts and evidence filed before the department of first instance and to speculate and assess which ones are maintained and of relevance for the requests put forward in appeal proceedings.

3. The new main request differs from both, the main and the auxiliary request underlying the decision under appeal. Since the respondent has only generally referred to a lack of novelty and inventive step without substantiating why it considered this to be the case, the board takes the respondent's arguments into account only insofar as it can deduce them from the decision under appeal.

Admission of the main request

4. The main request was filed after the parties had received the summons to oral proceedings and the communication summarising the board's provisional, non-binding opinion on certain issues underlying the appeal. The respondent did not submit any arguments concerning its admission.
5. The main request differs from auxiliary request 2 filed with the statement of grounds of appeal by amendments to claims 1 and 10 and the deletion of previous claims 12 and 19. The amendments and deletions are a direct response to issues mentioned in the board's communication. They neither raise new issues nor create a fresh case, and they streamline the procedure. Exercising the discretion under Article 114(2) EPC and Article 13(1), (3) RPBA, the main request is admitted into the appeal proceedings.

Main request

Articles 123(2), (3) and 84 EPC

6. Compared to the method of granted claim 1, the method of claim 1 of the main request no longer comprises the alteration of the properties of a recombinant target

protein defined as "accumulation ability, stability and/or integrity", "amenability to isolation; purification". Further amendments made to claim 1 are of an editorial nature and the consequence of multiple deletions that limit the scope of this claim in comparison with claim 1 as granted (cf. point I, above). The specification in claim 1 that the binding partner comprises a tag and/or targeting sequence is explicitly disclosed on page 7, lines 10 to 12 of the patent application. Dependent claim 10 has been amended by deletion of a feature so as to bring it in line with claim 1. These amendments neither contravene Article 123(2) EPC nor Article 123(3) EPC. Furthermore, the amendments do not introduce non-compliance with Article 84 EPC (cf. "Catchword" of decision G 3/14, OJ EPO 2015, 102).

Article 54 EPC

7. Claim 1 is directed to a method for altering three different properties of a recombinant target protein, namely i) cellular localisation, ii) post-translational modification, or iii) amenability to phase partitioning.
8. Novelty objections against claim 1 have been raised on the basis of documents D1 to D4.
9. Document D1 relates to the production of antibodies in plants and discusses practical considerations such as yield, storage and other properties for the selection of a particular crop system. It describes various ways of expressing antibodies and mentions in particular that, in rice plants simultaneously expressing both an antigen (carcinoembryonic antigen, CEA) and an antibody (scFv84.66) in the apoplast, the antibody accumulates

to higher levels compared to plants not expressing the antigen (cf. page 152, right-hand column, 3rd but last paragraph).

10. The opposition division came to the conclusion that this teaching of document D1 anticipated the subject-matter of claim 1 as far as it concerned a method for altering the cellular localisation of a recombinant target protein. The opposition division seems to have interpreted the increased accumulation of antibody in the apoplast as an indication of altered subcellular localisation of said antibody (cf. pages 5 and 6, point 4 of the decision under appeal).

11. Claim 1 defines a method for altering cellular localisation of a recombinant target protein (the antibody) by providing a specific binding partner (CEA) which forms a complex with said recombinant target protein and thereby links the properties of the binding partner to said recombinant target protein. It is the formation of the complex which actually affects or alters the subcellular localisation of the recombinant target protein (cf. point IX, above).

12. Document D1 discusses ways of affecting the subcellular localisation of the antibody but only by targeting the scFv84.66 antibody itself to different cellular compartments through the use of targeting signals that were well known in the art (cf. page 152, left-hand column). There is no mention at all of using the specific binding partner (CEA) of the recombinant antibody to form a complex and thereby altering the subcellular localisation of said antibody. Nor is there any mention of altering the post-translational modification pattern or phase partitioning of said antibody.

The above mentioned paragraph on page 152 of document D1 reports the expression of the scFv84.66 antibody in the apoplast of rice plants in the presence and in the absence of co-expression of its specific binding partner (CEA). Contrary to the accumulation and/or stability of the recombinant antibody which is increased and/or enhanced by the formation of a complex with its specific binding partner, the subcellular localisation of the recombinant antibody is not affected or altered by the formation of said complex. In both cases, in the presence and in the absence of co-expression of the specific binding partner, the scFv84.66 antibody is always expressed where it is targeted to, namely in the apoplast.

Document D1 does therefore not disclose a method with all the features of claim 1.

13. Documents D2 and D4 describe that the co-expression of a recombinant target protein with a specific binding partner comprising a tag sequence (D2: MEF2-C with tagged MEF2-A; D4: Cdc28 with tagged Clb2) increases the yield or accumulation of the recombinant target protein. There is however no mention of an alteration of the subcellular localisation, post-translational modification or phase partitioning of the recombinant target protein as a consequence of said co-expression and complex formation. Thus, documents D2 and D4 do not disclose a method with all the features present in claim 1.

14. Document D3 was published on 4 December 2003, i.e. after the priority dates of the contested patent (21 July 2003 and 6 October 2003), and it constitutes thus prior art according to Article 54(3) EPC.

15. Document D3 discloses nucleic acid constructs encoding a fusion polypeptide that comprises a "preselected polypeptide" (a recombinant target protein according to claim 1 of the main request) linked to an "inclusion body fusion partner" (IBFP) and a tag sequence (cf. Figure 18). Fusion of the preselected polypeptide with the IBFP may provide useful characteristics such as isolation enhancement or altered solubility under certain conditions (cf. *inter alia*, page 4, lines 9 to 12, and the paragraph bridging pages 28 and 29). It is well known in the art that the alteration of the solubility of a protein affects its amenability to phase partitioning.

16. Before the opposition division, the opponent/respondent had argued that the expression of nucleic acid constructs, such as those disclosed in document D3, and the subsequent formation of inclusion bodies was comparable to the complex formation of a recombinant target protein and a specific binding partner according to claim 1 of the main request. According to the opponent/respondent, inclusion bodies were aggregates as specifically envisioned in paragraph [0015] of the patent (cf. page 4, last paragraph but one, to page 5, second paragraph of the decision under appeal).

17. In this respect, the board observes that the use of fusion constructs comprising a recombinant target protein and its specific binding partner in a method according to claim 1 is specified in claim 2 of the main request. Thus, the question to be answered with regard to document D3 is whether the IBFP represents a specific binding partner that forms a complex with the recombinant target protein according to claim 1 of the main request.

18. According to the section "Brief summary of the invention" of the contested patent, "*complex formation is envisaged to increase the amount of the target protein primarily by decreasing the rate at which it is degraded*" (cf. paragraph [0005], last sentence), possibly because, *inter alia*, the target protein is less subject to partial unfolding while in contact with its binding partner (cf. paragraph [0006], third sentence). In line with the common understanding of its technical meaning, the term "complex formation between a target protein and its specific binding partner" refers thus to discrete, specific inter- or intramolecular interactions between a properly folded target protein and its binding partner (cf. also paragraph [0015], first sentence of the contested patent).

19. An inclusion body, on the other hand, "*is an amorphous deposit in the cytoplasm of a cell; an aggregated protein appropriate to the cell but damaged, improperly folded or liganded, or a similarly inappropriately processed foreign protein ...*" (cf. page 10, lines 26 to 29 of document D3).

20. Document D3 discloses 15 IBFP amino acid sequences (cf. page 50, Table 1) inducing inclusion body formation when linked to a preselected polypeptide (cf. page 10, lines 30 to 33; page 20, lines 25 to 27; page 28, lines 18 to 20). Although the IBFP is physically linked to a preselected polypeptide (recombinant target protein) and induces the formation of inclusion bodies, thereby linking the properties of said IBFP to the preselected polypeptide, the preselected polypeptide and the IBFP linked thereto, as a consequence of the amorphous, unordered and improperly folded nature of the proteins

in an inclusion body, do not form an intramolecular complex. The induction of inclusion body formation in order to alter the solubility of a preselected polypeptide (recombinant target protein) is thus not linked to "interactions between the target protein and the [specific] binding partner yielding a complex" thereby linking the properties of the two proteins, as required by claim 1 of the main request. The 15 IBFP sequences are no specific binding partners of the preselected polypeptides (recombinant target proteins) yielding a complex as defined in the contested patent and as generally understood in the art. Therefore, document D3 does not anticipate the claimed subject-matter.

Article 56 EPC

The closest state of the art

21. The board agrees with the conclusion of the opposition division that document D1 (cf. points 9 and 12, above) represents the closest state of the art (cf. page 7, point 7, page 8, last paragraph but one, and page 9, point 8 of the decision under appeal). Document D1 describes that the targeting of an antibody to different intracellular compartments, such as the ER, apoplast or vacuole, affects the yield of said antibody. Targeting signals which affect the subcellular localisation when linked to the antibody were known in the art (cf. page 152, left-hand column, second but last sentence).

The objective technical problem and the proposed solution

22. Starting from document D1, the objective technical problem to be solved is defined as the provision of

a(n) (alternative) method for altering i) the cellular localisation, ii) the post-translational modification, or iii) the amenability to phase partitioning of a recombinant target protein.

23. As a solution to this technical problem, the patent proposes the method of claim 1.

24. It has not been contested that the claimed method solves this technical problem by providing an alternative way of altering the subcellular localisation of a recombinant target protein and, as a consequence thereof, altering the amenability to phase partitioning of said target protein as well (cf. Tables 2 and 3, paragraphs [0027] to [0035] of the patent). As stated in paragraph [0008] of the contested patent, "intracellular targeting is also required to ensure that recombinant target proteins undergo appropriate post-translational modifications". Therefore, it is plausible that the claimed method may also alter the post-translational modification of a recombinant target protein.

25. In the examples of the contested patent, the recombinant target protein (the scFvT84.66 antibody or its diabody equivalent) and its specific binding partner (CEA, tagged with the KDEL sequence for ER-localisation) are expressed in *Nicotiana* plants (cf. paragraph [0016] of the patent). However, the claimed method is not limited to the expression of proteins in plants (cf. point IX, above). Moreover, claim 1 encompasses the provision of the specific binding partner by either expressing a gene encoding it or "in a different manner" (cf. step b) of claim 1; point IX, above). The question therefore arises whether the

technical problem is solved across the entire breadth of claim 1.

26. In view of the state of the art in general and the prior art documents on file in particular, there is no technical reason why the method of claim 1 should solve the underlying technical problem only in plants. Bacteria, yeast, insect and mammalian cells have been successfully used for the expression and co-expression of recombinant target proteins (cf. paragraph [0002] of the patent summarizing the state of the art; see also documents D2 and D4). In the board's view, no inventive skills are needed to implement the method of claim 1 in any of these non-human host cells.
27. As regards the provision of the specific binding partner in a manner different from recombinant expression, the appellant argues that this could be achieved, for example, by fusing the binding partner with cell penetrating peptides, through the use of viruses or of other techniques used for the transformation of cells, all well known to the person skilled in the art (cf. point XI, above). It is therefore plausible that this particular embodiment of the method of claim 1 also solves the underlying technical problem and that no inventive skills are required to carry it out.
28. Thus, the technical problem is solved across the entire breadth of claim 1.

Obviousness

29. In the decision under appeal (cf. page 10, second and third paragraphs), the opposition division concluded that document D1 anticipated the claimed method for

altering the subcellular localisation of a recombinant target protein. The opposition division considered it obvious to the person skilled in the art that, depending on the subcellular localisation, said recombinant target protein had to be isolated from an aqueous or a non-aqueous phase. Therefore, no inventive skills were considered to be required for solving the technical problem of providing a method for altering phase partitioning of a recombinant target protein.

30. As explained in point 12 above, the paragraph in the right-hand column of page 152 describes a stabilising effect of the co-expression of the antigen CEA on the scFv84.66 antibody. It does however not describe an alteration of the subcellular localisation of this antibody as a consequence of said co-expression. There is also no other paragraph in document D1 mentioning or suggesting that the cellular localisation of the antibody could be altered by co-expression of its antigen and complex formation of the two. The only method discussed in document D1 for altering the subcellular localisation of the antibody is by altering the signal sequence linked thereto.
31. Therefore, based on document D1 alone, the solution to the technical problem of providing a method for altering the subcellular localisation of a recombinant target protein as defined in claim 1 of the main request is not obvious.
32. The further documents on file (documents D2, D4 and D5) address the problem of improving the stability, accumulation, or purification of a recombinant target protein by co-expression with a specific binding protein. None of them, however, addresses the problem of altering the subcellular localisation, the post-

translational modification pattern, or the phase partitioning of a recombinant target protein. The solution to the technical problem, as defined in claim 1 of the main request, is therefore not obvious in view of document D1 in combination with any of these prior art documents.

33. It may be that, as stated by the opposition division (cf. point 29, above), the skilled person was aware of the fact that the subcellular localisation of a protein has an effect on its post-translational modification pattern and/or on its solubility. Since it was however not obvious to alter the subcellular localisation of a recombinant target protein by the method of claim 1, it was also not obvious to alter the post-translational modification pattern or the solubility of said recombinant target protein by altering its subcellular localisation through the method of claim 1.
34. Thus, the subject-matter of claim 1 involves an inventive step.

Article 83 EPC and respondent's request for remittal

35. In the decision under appeal, the opposition division did not decide on the merits of the opponent's ground for opposition under Article 100(b) EPC.
36. In its reply to the appellant's statement of grounds of appeal, the respondent requested the board to remit the case to the opposition division for a decision on the ground for opposition under Article 100(b) EPC, should the board come to the conclusion that the main request fulfils the requirements of Articles 54 and 56 EPC. The respondent further referred to its submissions made during the opposition procedure (cf. point XII, above).

37. The respondent's objections raised under Article 83 EPC are essentially that the patent does not enable the person skilled in the art to i) perform the claimed method in host cells other than plant cells, and ii) provide the specific binding partner in a manner different from co-expression (cf. points 1 to 3, above).
38. In the present case, these objections and the above mentioned question whether the technical problem is plausibly solved across the entire scope of claim 1 (cf. points 25 to 27, above) are tightly connected. In fact, since no inventive skills are needed to perform the invention across the entire scope of claim 1 because the required means and tools were readily available to the person skilled in the art, it follows that the method of claim 1 is also disclosed in a manner sufficiently clear and complete across its entire scope. The requirements of Article 83 EPC are thus fulfilled.
39. Under these circumstances, the board does not see any reason for a remittal of the case to the opposition division and, exercising its discretion under Article 111(1) EPC, decides against the respondent's request to remit the case to the opposition division for examination of the objections raised on the ground for opposition under Article 100(b) EPC.

Order

For these reasons it is decided that:

1. The decision under appeal is set aside.
2. The case is remitted to the opposition division with the order to maintain the patent on the basis of claims 1 to 17 of the main request as filed with submission of 25 April 2017, and a description to be adapted.

The Registrar:

The Chairman:



A. Wolinski

P. Julià

Decision electronically authenticated