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Datasheet for the decision of 13 August 2008

Case Number: T 1127/06 - 3.3.04

Application Number: 94931873.7

Publication Number: 0736106

IPC: C12Q 1/68

Language of the proceedings: EN

Title of invention:
Methods of screening for beta-amyloid peptide production inhibitors

Patentee:
ELAN PHARMACEUTICALS, INC., et al.

Opponent:
Glaxo Group Ltd.

Headword:
Beta-Amyloid Peptide/ELAN

Relevant legal provisions:
EPC Art. 54(3)

Relevant legal provisions (EPC 1973):
EPC Art. 56, 87(4)

Keyword:
"Main request - inventive step (no)"
"First auxiliary request - priority, novelty, inventive step (yes)"

Decisions cited:
-

Catchword:
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Case Number: T 1127/06 **-** 3.3.04

D E C I S I O N
of the Technical Board of Appeal 3.3.04
of 13 August 2008

Appellant: (Opponent)
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Decision under appeal:
Interlocutory decision of the Opposition Division of the European Patent Office posted 17 May 2006 concerning maintenance of European patent No. 0736106 in amended form.

Composition of the Board:

Chair: U. Kinkeldey
Members: M. Wieser
T. Bokor

Summary of Facts and Submissions

I. The appeal was lodged by the Opponent (Appellant) against the decision of the Opposition Division, whereby the European patent No. 736 106, filed on 17 October 1994 and claiming priority from US 143697, filed on 27 October 1993, could be maintained in amended form according to Article 102(3) EPC (1973).

II. The patent had been opposed under Article 100(a) EPC on the grounds of lack of novelty (Article 54 EC) and lack of inventive step (Article 56 EPC).

The Opposition Division had decided that claims 1 to 9 of the Patent Proprietor's (Respondent's) main request, filed during the oral proceedings before the Opposition Division, met all requirements of the EPC.

III. The Board expressed its preliminary opinion in a communication dated 7 February 2008.

Oral proceedings were held on 13 August 2008.

IV. The Appellant requested that the decision under appeal be set aside and that the patent be revoked.

The Respondent requested that the appeal be dismissed (main request) or, alternatively, that the decision under appeal be set aside and that the patent be maintained on the basis of any of the sets of claims filed as first, second, third or fourth auxiliary requests, all filed with letter of 8 February 2007.

V. Claims 1, 2 and 8 of Respondent's main request are those filed during the oral proceedings before the Opposition Division and read as follows:

"1. A method for monitoring β-amyloid precursor protein (βAPP) processing *in vivo*, said method comprising detecting the presence of an amino terminal fragment of βAPP (ATF-βAPP) in a specimen from a non-human animal transformed to express the Swedish mutation of human βAPP, wherein the amino terminal fragment has been cleaved between Leu^{596} and Asp^{597}.

2. A method for identifying β-amyloid production inhibitors, said method comprising:

detecting the amount of an amino terminal fragment of βAPP (ATF-βAPP) cleaved between Leu^{596} and Asp^{597} in a specimen from a non-human animal transformed to express the Swedish mutation of human β-amyloid precursor protein (βAPP) and to which a test compound has been administered; and comparing the detected amount of ATF-βAPP with a control amount of ATF-βAPP produced in the absence of the test compound.

8. A method for screening test compounds for the ability to inhibit or modulate cleavage of βAPP, said method comprising:

administering a test compound to a mouse transformed to express the Swedish mutation of human βAPP, wherein said βAPP is processed to ATF-βAPP in a sufficient amount to be detectable in a brain homogenate of said transformed mouse, and

detecting a change in βAPP processing in said transformed mouse as compared to said processing in the absence of the test compound."

Claims 3 to 7 refer to preferred embodiments of the method of claim 1 and/or claim 2. Claim 9 refers to a preferred embodiment of the method of claim 8.

Claims 1 to 7 of Respondent's first auxiliary request, which consists of claims 1 to 8, are identical to claims 1 to 7 of the main request. Claim 8 reads:

"8. A method for screening test compounds for the ability to inhibit or modulate cleavage of βAPP, said method comprising:

administering a test compound to a mouse transformed to express the Swedish mutation of human βAPP, wherein said βAPP is processed to ATF-βAPP in a sufficient amount to be detectable in a brain homogenate of said transformed mouse, and

detecting a change in βAPP processing in said transformed mouse as compared to said processing in the absence of the test compound, wherein said change in βAPP processing is detected by measuring a change in the amount of ATF-βAPP."

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

(1) US 07/965,971

(3) WO 93/21 526

(6) Nature, vol.361, January 1993, pages 260 to 263

(7) Nature, vol.360, 1992, pages 672 to 674

(9) WO 91/19 810

(10) Science, vol.253, 1991, pages 323 to 325

(11) Nature, vol.354, 1991, pages 476 to 478

(13) Science, vol.255, 1992, page 1445

(15) Science, vol.255, 1992, pages 1200 to 1202

(16) Annals N.Y. Acad. Sci., vol.695, 1993, pages 224 to 227

(17) Annals of Neurology, vol.35, no.5, 1994, pages 598 to 607

(18) Nature Genetics, vol.1, 1992, pages 345 to 347

(19) Annals N.Y. Acad. Sci., vol.695, 1993, pages 217 to 223

(20) Nature, vol.352, 1991, pages 239 to 241

(21) Declaration of K. Dora Games of 21 December 2005

VII. The submissions by the Appellant, as far as they are relevant to the present decision, may be summarised as follows:

The patent was not entitled to its priority claim as the priority document did not represent a first filing of the claimed subject-matter. The relevant date for defining the state of the art was therefore the filing date. Consequently, document (3), which belonged to the state of the art according to Article 54(3) EPC, anticipated the subject matter of claims 1 to 9 of the main request.

The closest state of the art was represented by document (9), or the corresponding scientific publication, document (20), disclosing a widely accepted and useful animal model for Alzheimer's disease.

No data have been provided that the model used in the presently claimed methods had any advantage over the model of document (9). The problem underlying the claimed invention had therefore to be seen in the provision of an alternative animal model for Alzheimer's disease.

The method of claim 8 of the main request was distinguished from the disclosure in document (9) in so far as the used mice were transformed to express the Swedish mutation of human β-amyloid precursor protein (βAPP), instead of the wild-type form of this protein. It was known from document (7) that the expression of βAPP carrying the Swedish mutation produced 6-7 fold more β-amyloid (βAP), the major constituent of the

amyloid plaques found in brains of Alzheimer's patients, than the expression of wild-type βAPP. The subject-matter of claim 8 of the main request was therefore obvious in the light of the disclosure in document (9) in combination with document (7) and did not meet the requirements of Article 56 EPC.

The methods according to the claims of the first auxiliary request contained as a further distinguishing feature that an amino terminal fragment of βAPP (ATF-βAPP) was used as marker for the *in vivo* processing of βAPP. However, document (7) disclosed that an alternative secretory cleavage of βAPP resulted in a shortened N-terminal form of βAPP and that antibodies were about to be developed that specifically recognized the last few amino acids of this shortened form including the substituted amino acids at positions 595 and 596. Therefore, also the subject-matter of the claims of Respondent's first auxiliary request did not involve an inventive step.

VIII. The submissions by the Respondent, as far as they are relevant to the present decision, may be summarised as follows:

The priority document was the first document disclosing a non-human animal transformed to express the Swedish mutation of βAPP and represented therefore the first filing of the claimed subject-matter. The patent was entitled to its priority claim with the consequence that document (3) did not belong to the state of the art.

Document (9) represented the closest state of the art for the assessment of inventive step. However, the animal model disclosed therein was not useful as model for Alzheimer's disease as it was unable to show *in vivo* processing of human βAPP. The detection of plaques in the brain of the test animals were no proof of βAP formation.

The problem underlying the patent was therefore the provision of a useful animal model for Alzheimer's disease.

Many variations of the disclosure in document (9) were possible in attempting to provide a solution to this problem. Only by hindsight it could be suggested that the skilled person would have immediately latched onto the Swedish mutation. Document (7) was concerned with cultured human kidney cells expressing the Swedish mutation of βAPP and mentioned, without providing data, experiments with CHO cells. It would not have been obvious to mirror results obtained in human cell culture into a non-human animal that does not naturally develop Alzheimer's disease.

Although ATF-βAPP had been detected in cell culture, it was neither known nor suspected of being involved in disease pathology nor of being useful as a detection marker for *in vivo* processing of βAPP. Document (7) merely proposed to investigate whether an increase in ATF-βAPP could be detected in cell culture in parallel with an increase in βAP.

The claims of the main request and of the first auxiliary request did therefore involve an inventive step (Article 56 EPC).

Reasons for the decision

Main request

Novelty - Article 54(3) EPC

1. The Appellant argued, that the US 143 697, the priority document of the patent in suit, filing date 27 October 1993, did not represent a first application for the purpose of determining priority, as foreseen in Article 87(4) EPC 1973. The subject-matter of claims 1 to 9 had already been disclosed in document (1), an US application filed on 26 October 1992 by the present inventors, which was the second of two priority documents of document (3). Consequently, document (3), whose content was identical to the content of document (1) and which was published on 28 October 1993, belonged to the state of the art according to Article 54(3) EPC and anticipated the subject-matter of claims 1 to 9.

2. However, document (1) does not disclose a non-human animal transformed to express the Swedish mutation of human βAPP. This is, for the first time, disclosed in the priority document of the patent in suit, which therefore represents a first application in the meaning of Article 87(4) EPC 1973. The patent is therefore entitled to claim priority from US 143 697, 27 October 1993. Consequently document (3) does not belong to the

state of the art according to Article 54(3) EPC and its disclosure is not to be considered for the examination of novelty of the subject-matter of claims 1 to 9.

No other documents have been cited by the Appellant to anticipate the novelty of the claimed subject-matter.

The requirements of Article 54(1) EPC 1973 are met.

Inventive step - Article 56 EPC 1973

3. In accordance with the problem and solution approach, the Boards of Appeal have developed certain criteria for identifying the closest prior art to be treated as a starting point. In selecting the closest prior art, the first consideration is that it must be directed to the same purpose or effect as the invention (cf Case Law of the Boards of Appeal of the EPO, 5th Ed. 2005, Chapters I.D.3.1 and 3.2).

4. The present invention relates to methods for monitoring the *in vivo* processing of βAPP in a non-human animal model for the diagnosis, prognosis and monitoring response to therapy of Alzheimer's disease, and for screening and evaluating of potential drugs for the treatment of Alzheimer's disease (see paragraphs [0001] and [0014] of the patent in suit).

5. Document (9) refers to a transgenic non-human animal model displaying the amyloid-forming pathology of Alzheimer's disease (the same disclosure is contained in document (20), the corresponding scientific publication of document (9)).

It discloses the production of a transgenic mouse over-expressing human wild-type βAPP (page 11, line 33 to page 12, line 4). The transgenic mice are considered to provide both prognostic and diagnostic means for the study of Alzheimer's disease and for determining the efficacy of drugs in treating the disease. Furthermore, it is intended to use the mice as standards to identify one or more candidate compounds capable of metabolizing βAP, or preventing its formation, which is considered to be associated with a predisposition to Alzheimer's disease (see page 12, line 29 to page 13, line 3).

Plaques were found to be formed in the brains of the transgenic animals (page 55, lines 1 to 13; page 64, lines 1 to 6; see also document (20), page 241, last paragraph). By using a polyclonal antibody elevated levels of full length βAPP expression could be detected in Western Blots of total brain homogenates of the transgenic animals (example 11).

Monoclonal antibody 4.1 was used for histological analysis of transgenic mouse brains. This antibody recognized an epitope mapped to the N-terminal 10 residues of βAP and had high affinity and specificity for the neuritic plaques. Samples from the brains of transgenic mice were treated for thirty minutes with 0.3% H_2O_2 and then for two minutes with 80% formic acid before they were contacted with medium from hybridoma secreting 4.1 antibody. An anti-mouse avidin-biotynilated horseradish peroxidase kit was used for visualisation (example 12, page 58).

6. Both parties are of the opinion that document (9) represents the closest state of the art. However, there

are different views concerning the relevance of the experimental data disclosed therein.

6.1. The Respondent argued that transgenic mice expressing wild-type βAPP according to document (9) would not have been considered a useful model for Alzheimer's disease. By referring to a declaration of his technical expert (document (21)), he argued that there was no evidence that the wild-type βAPP expressed by the mice was actually processed to release βAP. The amyloid deposits identified only at a low frequency in the transgenic mice of document (9) did not display the pathology of plaques that were present in Alzheimer's disease patients (document (16), page 225). More importantly, it was unclear whether the plaques in the mice of document (9) actually contained βAP, the primary constituent of plaques in Alzheimer's patients. The 4.1 monoclonal antibody used to detect the deposits in the transgenic mice would have reacted with both, βAPP and βAP, since the antibody was raised against the first 28 amino acids of βAPP (see document (20) pages 240 to 241) which were present in both βAPP and βAP. In addition, the staining pattern of the plaques in document (9) with silver, thioflavin S and Congo red is not specific for βAP (document (21), point (9)).

The doubts of a skilled person with regard to the usefulness of the animal model of document (9) would have been even heightened by his/her knowledge that two earlier transgenic models for Alzheimer's disease had been withdrawn. The model originally disclosed in document (10) had to be withdrawn in document (13), following a demonstration that there was no difference between amyloid deposits in the transgenic mice and in

control mice. The other model, originally disclosed in document (11) had to be retracted following allegations of fraud. A summary of the rather unsuccessful and less encouraging story of animal models for Alzheimer's disease was to be found in document (15).

Further doubts were expressed in document (19), which disclosed on page 220, that a much more complete analysis of the transgenic mice of document (9) was necessary for their acceptance as models for Alzheimer's disease, and in post-published document (17) (page 601, right column).

6.2. The Appellant argued that document (15), a review article dealing with Alzheimer's disease animal models, in its subtitle read that "... two of the three published mouse models are now being retracted...", which were the models originally published in documents (10) and (11), and that only the third model, the one disclosed in documents (9) and (20) was holding up (see page 1201, last paragraph). In fact this was the only model wherein the transgenic mice expressed full length βAPP. Despite critical remarks and careful prognosis concerning its reliability the model was not discredited in any of the cited documents.

Even the patent in suit described the animal model of document (9) as useful model for screening test compounds, which had previously been identified by an *in vitro* screen, for their therapeutic effectiveness (see paragraph [0038]).

Contrary to the disclosure in documents (16) and (17), which found that amyloid deposits were identified in

the transgenic mice at a low frequency only, document (20) disclosed in table 1 that all transgenic mice developed deposits.

Before staining of the deposits in the brains of transgenic mice by use of monoclonal antibody 4.1, the samples were treated with 80% formic acid. This treatment has been carried out, as was known by an expert in the field, to ensure that the epitope (the N-terminal 10 residues of βAP) was disclosed and to prevent re-clumping of βAP (example 12 of document (9); page 241, left column first full paragraph and legend to figure 2 in document (20)). This showed, that processed βAP, besides full length βAPP, was present and detected in the plaques found in the transgenic mice according to document (9).

At the priority date of the patent in suit, document (9) was accepted as disclosing a useful animal model for Alzheimer's disease.

7. The issue here for the Board is to decide if a skilled person working in the field of animal models for Alzheimer's disease would pay attention to the teaching of document (9) in the sense that he would consider it as a promising starting point for further investigations.

When considering the disclosure in the documents cited in this context by the parties, the Board is convinced that a skilled person working in the field of the present invention (see point (4) above) would not have ignored the teaching in document (9). This can be seen best from the statement in paragraph [0038] of the

patent itself and from the review of the state of the art given in document (15), which states that the professional circles, after having first been provided with three seemingly useful models, now were left with only one, namely the model of document (9) (see document (15), page 1200, middle column).

The board agrees with both parties and also considers document (9) to represent the closest state of the art for the assessment of inventive step.

The problem underlying the present invention is the provision of an improved method for monitoring the *in vivo* processing of βAPP in a non-human animal model.

8. The Board is convinced that this problem has been solved by the subject-matter of claims 1 to 9, as can be seen in the experimental part of the patent, especially figure 8.

9. The subject-matter of independent claims 1, 2 and 8 is distinguished from the disclosure in document (9) in so far as the used non-human animals have been transformed to express the Swedish mutation of human βAPP.

Claims 1 and 2, in addition, require that the monitoring of βAPP processing comprises the detection of an amino terminal fragment of βAPP (ATF-βAPP).

10. This feature is not contained in the subject-matter of claim 8, which refers to a method for screening test compounds for the ability to inhibit or modulate cleavage of βAPP wherein a change in βAPP processing in

a transformed mouse is detected and compared to said processing in the absence of the test compound.

As this definition of the monitoring step includes the detection via immune-staining disclosed in document (9), the only difference between the subject-matter of claim 8 and the disclosure in document (9) is the use of the Swedish mutation of human βAPP for the development of an animal model for Alzheimer's disease.

11. Document (7) reports that human kidney cells, which express a DNA coding for human βAPP bearing a double mutation (Lys to Asn at residue 595 and Met to Leu at position 596), the so-called "Swedish mutation" (first described in document (18)), produced 6 to 8-fold more βAP than cells expressing normal βAPP. Similar results (for which no data are shown) are reported from cultured CHO cells (see abstract, page 672, left column and page 673, right column).

12. The Respondent argued that the introduction of the Swedish mutation was only one of a number of possibilities a skilled person would have considered in order to solve the problem as defined in point (7) above.

Neither would it have been obvious to the skilled reader that a transgenic animal expressing Swedish βAPP would display any form of βAPP processing and would be useful for detecting modulations in βAP release, nor that a transgenic animal expressing Swedish βAPP would display higher levels of βAPP processing than an animal expressing wild-type βAPP. The increased processing observed in human cell culture in document (7) would

not occur in a non-human animal if the animal's cleavage enzyme did not recognize the Swedish mutation.

Extrapolations from the results of cell cultures to animal models are very tempting for a person not being aware of the complex metabolic situation in a living organism, but mostly very unrealistic for the skilled practitioner. Results obtained in human cell culture might not be mirrored in a non-human animal that does not naturally develop Alzheimer's disease.

13. The Swedish mutation is known to be associated with the early onset of Alzheimer's disease (document (18), abstract). Document (7) equips the reader with the knowledge that that cells expressing βAPP carrying the Swedish mutation produce higher levels of βAP, the major constituent of the amyloid plaques found in brains of Alzheimer's patients, than cells expressing wild-type βAPP.

Document (15), page 1201, last paragraph, discloses, directly after mentioning that the animal model of document (9) is the only one holding up, that "... all researchers involved say that they will continue to work on finding amyloid gene constructs that will yield Alzheimer's pathology. They particularly want to try a mutated form of the gene that was recently linked to the disease."

As both parties confirmed at the oral proceedings, the number of mutations of βAPP known at the priority date of the patent in suit was very limited. One of this few mutations, the Swedish mutation, was known from the

disclosure in document (7) to produce outstandingly high levels of βAP.

Although knowing that *in vitro* experiments do not necessarily mimic the *in vivo* settings and that *in vitro* results are not always confirmed upon *in vivo* testing, the skilled person would have perceived the experiments reported in document (7) which showed a 6 to 8-fold increase of βAP production in cells expressing Swedish βAPP. Thus, in spite of the inherent uncertainties which always characterise biological experiments, the skilled person had no reasons to adopt a sceptical attitude. He or she would have had either some expectations of success or, at worst, no particular expectations of any sort, but a "try and see" attitude, which - as pointed out in decisions T 333/97 of 5 October 2000, and T 1045/98 of 22 October 2001 - does not equate with an absence of a reasonable expectation of success.

Further, the Board holds that in the field of the invention, the motivation of the skilled person to "try and see" will overcome even a non-negligible probability of failure, due to the general recognition of the pressing need for a useful animal model in the research on Alzheimer's disease, as emphasized by the appellant and also illustrated by document (15) (see the expert statement at the end of page 1201).

The Board is therefore convinced, that the skilled person trying to provide an improved method for monitoring the *in vivo* processing of βAPP in a non-human animal model, would have transformed the non-human animals - at the time of priority technically a

feasible enterprise - to express the Swedish mutation of human βAPP instead of wild-type βAPP and thus would have arrived at the subject-matter of claim 8 in an obvious way.

Claim 8 does not involve an inventive step as required by Article 56 EPC 1973.

The main request is therefore not allowable.

First auxiliary request

Inventive step - Article 56 EPC 1973

14. Claims 1 to 8 of this request are distinguished from the claims of the main request in so far, as claim 8, like the other independent claims 1 and 2, now also contains the feature, that the monitoring of βAPP processing comprises the detection of an amino terminal fragment of βAPP (ATF-βAPP). Claim 9 has been deleted.

15. The Appellant argued, that document (7) already disclosed an antibody, designated B5, which was raised against the amino terminal shorter form of βAPP, designated APP_s (page 673, left paragraph, figures 1a and 2). The only remaining question was therefore whether it was obvious to generate an antibody specific for ATF-βAPP that could be used to measure production of ATF-βAPP directly. This question was answered in the first sentence on page 674, right column of document (7), where it is stated, that antibodies specifically recognizing the last few residues of the shorter APP_s form including the substituted amino acids at 595 and 596 were currently developed. It had to be established

whether such a secreted form of βAPP increased in parallel with the rise of βAP in the medium of the Swedish tansfectants.

This passage of document (7) moreover contained a reference to document (6), which reported that a substantial portion of βAPP was cleaved precisely at the amino terminus of βAP, at a site designated "β-secretase site" between Met 596 and Asp 597 of wild type βAPP, which site was distinct from the normal processing site of βAPP ("α-secretase site"). Document (6) disclosed an assay for ATF-βAPP and suggested that this secretory pathway was involved in βAP genesis.

In addition, the replacement of βAP as marker for βAPP processing by ATF-βAPP did not bring about any advantage and was therefore merely an obvious equivalent.

16. Although ATF-βAPP has been detected in cell culture, this fragment differed from its co-cleavage product βAP in that it is not involved either in disease pathology or normal physiology. βAP was known to be the principal component of plaques formed in the brain of Alzheimer's patients. Therefore, document (9), representing the closest state of the art, as the other animal models disclosed in documents (10) and (11) and later retracted, was focussed on the detection of βAP as a marker of βAPP processing.

Contrary to the Appellant's argument, ATF-βAPP is not merely an obvious equivalent to βAP in the detection of βAPP processing. While βAP can be used as marker for different processing pathways of βAPP, ATF-βAPP is a

specific marker for β-secretase activity. In addition the Swedish form of ATF-βAPP from human βAPP has an epitope that is not present in mouse ATF-βAPP and can therefore be detected independently of human and mouse wild-type βAPP and of mouse ATF-βAPP.

Both, documents (6) and (7), when referring to the detection of ATF-βAPP do not disclose or suggest to use this fragment as a marker for βAPP processing. Document (7), in the passage bridging the left and right column on page 674, proposes to investigate whether an increase in the ATF-βAPP fragment can be detected in cell culture in parallel with a rise of βAP in the medium. In document (6) the detection of ATF-βAPP fragment is referred to in the context of providing support for a mechanism whereby βAP is generated.

Document (9), representing the closest state of the art, discloses an Alzheimer's disease animal model wherein amyloid plaque formation in transgenic animals expressing human wild type βAPP, is detected by immune-staining of βAP. Neither from the disclosure in document (7) nor from any other document on file would the skilled person, when trying to provide an improved animal model for Alzheimer's disease, get an information that would prompt him to amend the teaching in document (9) and to replace the marker for βAPP processing used in document (9), namely βAP the major component of the amyloid plaques which are the hallmark of Alzheimer's disease, by ATF-βAPP a fragment not known to be involved in disease pathology and whose exact role in the newly detected cleavage pathway of βAPP

still had to be established at the priority date of the patent in suit.

The Board, therefore decides, that the subject-matter of claims 1 to 8 of the first auxiliary request involves an inventive step and meets the requirements of Article 56 EPC 1973.

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 on the basis of the following documents:

Claims:	1 to 8, filed with letter of 8 February 2007 as first auxiliary request;
Description:	pages 2, 3, 5 to 9, 13, filed during the oral proceedings before the Opposition Division on 22 February 2007, page 4 as filed during the oral proceedings before the Board, pages 10 to 12, 14 to 19 of the patent specification;
Figures:	1 - 9 of the patent specification.

Registrar: Chair:

R. Schumacher U. Kinkeldey