Datasheet for the decision
of 11 January 2018

Case Number: T 2571/12 - 3.3.01
Application Number: 02799377.3
Publication Number: 1438063
Language of the proceedings: EN

Title of invention:
GLUTATION PRECURSORS FOR THE TREATMENT OF NEUROPSYCHIATRIC DISORDERS

Patent Proprietor:
The Mental Health Research Institute of Victoria

Opponent:
ELKINGTON AND FIFE LLP

Headword:
Treatment of schizophrenia with glutathione/MENTAL HEALTH RESEARCH INSTITUTE

Relevant legal provisions:
RPBA Art. 15(3)
EPC Art. 56, 83
Keyword:

Decisions cited:
T 0789/89, T 0629/90, G 0004/92, G 0002/88, T 0609/02, T 0382/96

Catchword:
Case Number: T 2571/12 - 3.3.01

DE C I S I O N
of Technical Board of Appeal 3.3.01
of 11 January 2018

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Decision under appeal: Decision of the Opposition Division of the
European Patent Office posted on 8 October 2012
revoking European patent No. 1438063 pursuant to
Article 101(3)(b) EPC
Composition of the Board:

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<td>A. Lindner</td>
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<td>Members</td>
<td>T. Sommerfeld</td>
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Summary of Facts and Submissions

I. European patent No. 1438063 is based on European patent application No. 02799377.3, which was filed as an international application and published as WO 2003/026684. The patent has the title "Glutation [sic] precursors for the treatment of neuropsychiatric disorders" and was granted with 10 claims.

II. Notice of opposition was filed against the granted patent, the opponent requesting revocation of the patent in its entirety on the grounds of lack of inventive step (Articles 56 and 100(a) EPC), lack of sufficiency of disclosure (Article 100(b) EPC) and added subject-matter (Article 100(c) EPC).

III. In its decision pronounced at oral proceedings, the opposition division revoked the patent under Article 101(3)(b) EPC.

The opposition division decided that the main request fulfilled the requirements of Articles 123(2) and 83 EPC, but not those of Article 56 EPC, and that none of the further pending requests (auxiliary requests 1 to 3) fulfilled Article 56 EPC either. An amended auxiliary request 1 filed during oral proceedings was not admitted into the proceedings.

IV. The patent proprietor (appellant) lodged an appeal against that decision. In its statement of grounds of appeal, the appellant requested that the opposition division's decision be set aside and that the patent be maintained on the basis of the main request or auxiliary requests 1 to 10, all filed with the grounds of appeal, or on the basis of auxiliary requests 11 to 34, for which "in the interest of conciseness no paper
copies (...) were submitted" but a table describing them was provided instead.

V. Claim 1 of the main request reads as follows:

"1. Use of a glutathione precursor in the manufacture of a medicament for the treatment of a neuropsychiatric disorder in a mammal, wherein said glutathione precursor induces, upregulates or otherwise augments antioxidant functional activity in the brain of said mammal."

Claim 1 of auxiliary requests 1 to 5 differs from claim 1 of the main request by further defining the neuropsychiatric disorder as being "depression" (auxiliary request 1), "bipolar disorder, manic depression or psychotic depression" (auxiliary request 2), "bipolar disorder" (auxiliary request 3), "psychosis, bipolar disorder, manic depression, affective disorder, psychotic depression, drug induced psychosis, delirium, alcohol withdrawal syndrome or dementia induced psychosis" (auxiliary request 4) or "schizophrenia" (auxiliary request 5).

Claim 1 of auxiliary requests 6 to 9 differs from claim 1 of the main request by further defining the glutathione precursor as being: "cysteine or a derivative or precursor thereof" (auxiliary request 6), "cysteine or a derivative or precursor thereof which is acetylated or aminidated on the side chain of cysteine" (auxiliary request 7), "N-acetyl cysteine or a derivative thereof" (auxiliary request 8) or "N-acetyl cysteine" (auxiliary request 9).

Claim 1 of auxiliary request 10 differs from claim 1 of the main request by further defining the neuro-
psychiatric disorder as being "characterised by: (i) aberrant, unwanted or otherwise inappropriate oxidative stress; and/or (ii) inadequate glutathione metabolism, in the brain of a mammal".

VI. The opponent did not submit any substantive reply to the grounds of appeal. Instead, by letter dated 12 September 2013 it withdrew its opposition.

VII. Oral proceedings before the board took place on 11 January 2018, in the absence of the appellant as announced by fax dated 13 December 2017. At the end of the oral proceedings, the chairman announced the board's decision.

VIII. The documents cited during the proceedings before the opposition division and the board of appeal include the following:

D2 Gillissen and Novak 1998, Respiratory Medicine 92, 609-623
D3 Schulz et al. 2000, Eur.J.Biochem. 267, 4904-4911

IX. The appellant's submissions (in writing) may be summarised as follows:

With regard to inventive step, D3 could be considered as an alternative to D2 as closest prior art. D3 mentioned schizophrenia but merely reported in this context that glutathione (GSH) levels were reduced; however, fluctuations of biomarkers could not be
expected as suitable therapeutic targets, and hence this observation would not motivate the skilled person to investigate glutathione metabolism as a therapeutic target for this disease. In fact D3 itself proposed glutathione precursors for therapy of neurodegenerative diseases but not for schizophrenia. D3 emphasised that it remained to be determined whether or not altered glutathione metabolism played a role in the pathogenesis of the neurodegenerative diseases discussed in D3 and whether raising GSH levels would bring a therapeutic benefit (page 4908, final full paragraph, and page 4909, final paragraph). Moreover, it reported that even accumulation of the GSH precursor procysteine in the cerebrospinal fluid in the ambit of a trial with patients with the neurodegenerative disorder amyotrophic lateral sclerosis did not lead to any increase of GSH in the cerebrospinal fluid (page 4909, penultimate paragraph), illustrating the difficulties associated with trying to raise GSH levels in the central nervous system. Moreover, D3 highlighted that substances that increased brain cysteine were potentially toxic to neurons (page 4909, lines 15 to 18). D4, while concluding that there was a decrease in GSH in schizophrenic patients, did not teach that this decrease was causative of the disease and in fact stated that it could be primary or secondary to another disease (page 3726, left column, second paragraph). Moreover, since D4 indicated that the activity of the glutathione peroxidase enzyme was reduced in schizophrenia (page 3726, paragraph bridging the two columns) or that other enzymes in the GSH cycle could be perturbed (page 3726, right column, lines 9 to 11), the skilled person would not expect that the activity of a defective enzyme could be restored by increasing the levels of its substrate.
With regard to Article 83 EPC, the opposition division had confirmed that there was no evidence to suggest that treatment would not be possible for any neuropsychiatric disorder. Conclusive evidence of the appropriateness of the extrapolations made in the application was provided e.g. by D13, discussing the use of N-acetyl cysteine (NAC) for treatment of depression, and D15, which on page 960, first paragraph, reviewed studies discussing NAC in trichotillomania, obsessive-compulsive disorders, bipolar disorders and schizophrenia.

X.
The arguments of the former opponent, submitted in writing before withdrawal of the opposition and in so far as they are relevant for the present decision, may be summarised as follows:

With regard to Article 56 EPC, D2 could be considered the closest prior art, and the technical problem would be the provision of a further medical indication for NAC. In view of D3 and D4, disclosing that schizophrenia, like the lung diseases of D2, was also characterised by low levels of GSH, and that GSH precursors were capable of penetrating the blood-brain barrier (D3), the skilled person would arrive at the claimed subject-matter without the need for inventive skill.

As for Article 83 EPC, the patent did not contain any evidence that any neuropsychiatric disorder other than schizophrenia was associated with inadequate levels of glutathione. Hence in line with T 609/02, the description of the patent provided no more than a vague indication of a possible medical use.
XI. The appellant requested in writing that the decision under appeal be set aside and that the patent be maintained on the basis of the main request or, alternatively, on the basis of auxiliary requests 1 to 34.

**Reasons for the Decision**

1. The appeal is admissible.

2. Withdrawal of the opposition by the opponent

2.1 During appeal, the sole opponent withdrew its opposition. No issues were on file in relation to which the opponent would have remained a party. Hence the patent proprietor became the sole remaining party to the appeal proceedings.

2.2 As regards the withdrawal of opposition by an opponent who is not the appellant, this does not affect the appeal proceedings, in so far as it is the principal task of the boards of appeal to review the decision under appeal on the basis of the appellant's requests. While the opponent ceases to be party to the appeal proceedings in respect of the substantive issues (e.g. T 789/89, OJ EPO 1994, 482), the board may nevertheless take into account the submissions and evidence filed by the former opponent before the opposition was withdrawn (e.g. T 629/90, OJ EPO 1992, 654).
3. Absence of the appellant at oral proceedings

3.1 The oral proceedings before the board took place in the absence of the appellant, who had been duly summoned but decided not to attend.

3.2 The present decision is based on facts and evidence put forward during the written proceedings, and the appellant has had an opportunity to comment on them. Therefore the conditions set forth in Enlarged Board of Appeal opinion G 4/92, OJ EPO 1994, 149, are met.

3.3 Moreover, as stipulated by Article 15(3) RPBA the board is not obliged to delay any step in the proceedings, including its decision, by reason only of the absence at the oral proceedings of any party duly summoned who may then be treated as relying only on its written case.

4. Main request – Article 56 EPC

4.1 The patent is directed to the treatment of neuropsychiatric disorders by administration of a glutathione precursor. As disclosed in paragraph [0036], "The present invention is predicated, in part, on the determination both that up-regulation of central nervous system glutathione metabolism, and in particular brain glutathione metabolism, improves aberrant oxidative homeostasis and that it can be achieved via administration of a glutathione precursor, in particular N-acetyl cysteine, to a mammal". In the section "Background of the invention", the patent provides an overview of schizophrenia and its causes, in particular those related to "abnormality in oxidation homeostasis systemically and centrally" and
the implications of this sustained oxidative attack on glutathione metabolism (paragraphs [0006] and [0007]). Paragraph [0008] then goes on to discuss the prior-art studies in which therapeutic possibilities based on the observed biochemical changes have been suggested and/or tested.

4.2 The closest prior art for assessing inventive step is normally a prior-art document disclosing subject-matter conceived for the same purpose or aiming at the same objective as the claimed invention and having the most relevant technical features in common, i.e. requiring the minimum of structural modifications (see Case Law of the Boards of Appeal, 8th edition 2016, "CLBA", I.D.3.1). Hence, in the case of claims directed to medical uses, the closest prior art is usually a document disclosing the same therapeutical indication: in the present case, "treatment of a neuropsychiatric disorder in a mammal". Therefore the board disagrees with the opposition division's conclusion that document D2, which is directed to the treatment of lung diseases and does not provide any teaching at all concerning neuropsychiatric disorders, can be considered the closest prior art. Instead, either the prior-art therapy possibilities for the neuropsychiatric disorder schizophrenia, discussed in the patent in paragraph [0008], or document D4, discussing schizophrenia and its biological bases, would be more suitable starting points for the discussion of inventive step. Alternatively, and in agreement with the appellant's submissions, document D3, which discloses the role of glutathione and oxidative stress in neurodegenerative diseases and the corresponding treatment possibilities with glutathione precursors and indicates that the same pathological mechanism of glutathione decrease is also
present in schizophrenia, can be considered the closest prior art.

4.3 Starting from D3, the difference compared to present claim 1 is that D3 does not explicitly teach the use of glutathione precursors for treatment of neuropsychiatric disorders. The technical problem can thus be formulated as the provision of a therapy for the neuropsychiatric disorder schizophrenia, based on the observation made in D3 that glutathione levels are reduced in this disorder. The solution is the use of glutathione precursors as claimed, and the board is satisfied that this plausibly solves the technical problem.

4.4 It remains to be examined whether the claimed solution is inventive.

4.5 D3 reviews the evidence for a disturbance of glutathione homeostasis in neurodegenerative disorders, and teaches that "[b]ecause glutathione does not cross the blood-brain barrier other treatment options to increase brain concentrations of glutathione including glutathione analogs, mimetics or precursors" might be useful (abstract). Hence D3 teaches to use glutathione precursors in order to increase glutathione levels in the central nervous system. This is further developed in the section headed "Therapeutic approaches for neurodegenerative diseases" (page 4908, right column), which reads: "If alterations in glutathione metabolism play an important role in the pathogenesis of the neurodegenerative diseases discussed, treatments that lead to enhanced synthesis of GSH or that inhibit its degradation may result in a slowing of disease progression. Because GSH itself penetrates the blood-brain barrier only poorly and cannot be taken up by
neurons directly, treatments with GSH monoethyl ester, glutathione precursors or other glutathione analogs have been used in patients or animal models". On the next page, treatment with the glutathione precursor N-acetyl-L-cysteine is discussed. Moreover, although D3 is primarily concerned with neurodegenerative disorders such as Parkinson's disease, Alzheimer's disease and amyotrophic lateral sclerosis, it also states that there is a decrease in glutathione in schizophrenia (page 4908, right column, third paragraph: reference [68] is document D4 in the proceedings). Hence, the skilled person would assume that the same conclusions reached for neurodegenerative disorders as regards therapeutic approaches would also apply to schizophrenia. Accordingly, the skilled person would be motivated, from document D3 alone, to investigate glutathione precursors as possible therapeutic agents in diseases of the central nervous system where glutathione is decreased, including schizophrenia.

4.6 In any case, the skilled person would be prompted by document D3 to consider the teachings of document D4. Document D4 reports on the increasing evidence for the existence of "an impaired antioxidant defence and increased oxidative injury in schizophrenia" (page 3721, left column, lines 16 to 18) and, on basis of these observations, sets out to determine the levels of glutathione (GSH), "known as a nucleophilic scavenger and an enzyme-catalysed antioxidant" (page 3721, right column, lines 4 to 5), in both the cerebrospinal fluid and the brain of schizophrenic patients. D4's results show that schizophrenic patients display an important deficit in GSH, "thus supporting a hypothesis involving an impaired antioxidant defence system in the pathophysiology of schizophrenia" (page 3721, right column, lines 9 to 11). From the results obtained, the
authors of D4 propose "a hypothesis that is based on a central role of GSH in the pathophysiology of schizophrenia" (page 3726, right column, lines 24 to 26) and end the paper by stating that: "In conclusion, a deficit in GSH and GSH-related enzymes might play an essential role in the pathophysiology and might constitute a major risk factor of schizophrenia, or some forms of schizophrenia. The proposed hypothesis brings together some elements known about the disease. If it proves to be correct, it could lead to new approaches to its treatment." (page 3727, left column, last paragraph). Hence, by combining the teachings of D4 with those of D3, the skilled person would arrive at the claimed solution in an obvious way.

4.7 The appellant argued that the mere observation in D4 of a decrease in GSH was not indicative of that decrease being causative of schizophrenia and that D4 did not at any point suggest increasing the levels of glutathione as a possible treatment for schizophrenia. The board notes that, while in fact the authors of D4 themselves state (page 3726, left column, beginning of second paragraph) that it is difficult to ascertain whether the glutathione deficit is primary or secondary to another disorder, they nevertheless repeatedly emphasise that the data is consistent with an involvement of GSH metabolism in the pathophysiology of schizophrenia (page 3726, left column, second paragraph, as well as right column, third paragraph, and page 3727, left column). Moreover, while not proposing any specific therapeutic approaches, they foresee in their concluding sentence that, based on the essential role that a deficit in GSH and GSH-related enzymes might play in the pathophysiology of schizophrenia, new approaches to treatment may emerge.
It would thus be straightforward to try this with a reasonable expectation of success.

4.8 The appellant further argued that D4 did not in any way suggest that the GSH deficit might be due to a deficit in the rate-limiting building block cysteine, and so the skilled person, even if he would have contemplated addressing the GSH deficit in schizophrenia, would not be motivated to administer a glutathione precursor. Instead, D4 indicated that the activity of the enzyme GSH peroxidase was reduced in schizophrenia (paragraph bridging the right and left columns on page 3726), which would have deterred the skilled person from administering GSH, since he would know that the activity of a defective enzyme could not be restored by increasing the levels of its substrate. Finally, D4 also indicated that other enzymes in the GSH cycle might be involved (page 3726, right column, lines 9 to 11), which again would deter the skilled person from administering GSH. The board notes that, as mentioned above, D4 does not in fact suggest any specific therapeutic approach based on its results, let alone a therapy based on GSH precursors. Such a therapy is however taught in D3, in the context of neurodegenerative diseases for which a similar pathophysiological mechanism involving GSH is disclosed. As regards the involvement of the GSH cycle enzymes, the board notes that D3 discloses that therapy with N-acetyl-L-cysteine resulted in elevated glutathione peroxidase levels (page 4909, left column, lines 4 to 7), hence teaching that, contrary to the appellant's arguments, a therapy with glutathione precursors may also overcome deficits in glutathione peroxidase levels.
4.9 The subject-matter of claim 1 of the main request is thus considered to lack inventive step. Therefore, the main request is not allowable for lack of compliance with Article 56 EPC.

5. **Auxiliary request 1 - Article 83 EPC**

5.1 In claim 1 of auxiliary request 1 the therapeutical indication has been restricted to the neuropsychiatric disorder depression.

5.2 By definition, attaining the claimed therapeutic effect is a functional technical feature of claims directed to medical uses (G 2/88, OJ EPO 1990, 93, reasons 10.3). As a consequence, under Article 83 EPC, unless this is already known to the skilled person at the priority date, the application must disclose the suitability of the product to be used for the claimed therapeutic application (see also T 609/02, reasons 9).

5.3 Although depression is listed in the patent as one of the possible neuropsychiatric disorders that could be treated with glutathione precursors, there is no evidence at all, either in the patent or in the available prior art, that makes it plausible that glutathione precursors may have a therapeutic effect in depression. In fact, there is no hint that the underlying mechanism which is extensively disclosed in the patent for schizophrenia, namely reduction of glutathione levels in the central nervous system and consequent oxidative stress, is also present in depression. Hence the board concludes that the patent does not disclose the suitability of the product to be used for the claimed therapeutic application.
5.4 The board thus disagrees with the conclusions of the opposition division that because "no evidence has been provided by O to show that any neuropsychiatric disorder cannot effectively been [sic] treated using a glutathione precursor such as N-acetyl-cysteine (...) the patent in suit is considered as disclosing the invention in a manner sufficiently clear and complete for it to be carried out by a person skilled in the art" (decision of the opposition division, page 7, second paragraph). It is the patent that has to demonstrate the suitability of the claimed treatment for the claimed therapeutic indication. As explained, for example, in decision T 609/02 supra, a simple verbal statement that compound X may be used to treat disease Y is not enough to ensure sufficiency of disclosure: rather, it is required that the patent provides some information in the form of, for example, experimental tests, to the avail that the claimed compound has a direct effect on a metabolic mechanism specifically involved in the disease, this mechanism being either known from the prior art or demonstrated in the patent per se.

5.5 Decision T 609/02 moreover clarified that post-published evidence may be taken into account for sufficiency of disclosure, but only to back up the findings in the patent application in relation to the use of the ingredient as a pharmaceutical, and not to establish sufficiency of disclosure on its own. Accordingly, post-published document D13, submitted by the appellant with the statement of grounds of appeal as evidence for a role of glutathione precursors in the context of depression, cannot be taken into account in the present context.
5.6 The subject-matter of claim 1 of auxiliary request 1 is thus not sufficiently disclosed in the application. Auxiliary request 1 is not allowable for lack of compliance with Article 83 EPC.

6. Auxiliary requests 2, 3 and 4 - Article 83 EPC

6.1 Claim 1 of these requests has also been restricted to specific neuropsychiatric disorders such as bipolar disorder, manic depression or psychotic depression (auxiliary request 2), bipolar disorder (auxiliary request 3), and psychosis, bipolar disorder, manic depression, affective disorder, psychotic depression, drug induced psychosis, delirium, alcohol withdrawal syndrome or dementia induced psychosis (auxiliary request 4).

6.2 Again, there is no evidence either in the patent or in the available prior art for a therapeutic effect of glutathione precursors for any of the claimed disorders, and hence the post-published document D15, allegedly supporting such an effect inter alia for bipolar disorders, likewise cannot be taken into account for the assessment of sufficiency of disclosure.

6.3 The subject-matter of claim 1 of auxiliary requests 2 to 4 is thus considered to be insufficiently disclosed in the application. Auxiliary requests 2 to 4 are hence not allowable for lack of compliance with Article 83 EPC.

7. Auxiliary request 5 - Article 56 EPC

7.1 Claim 1 of auxiliary request 5 has been restricted to schizophrenia as medical indication.
7.2 Both documents D3 and D4 specifically refer to schizophrenia, and the conclusions reached for the main request as regards inventive step were based on schizophrenia as being one of the possible neuropsychiatric disorders to be treated. Hence for the same reasons as discussed above for the main request, the subject-matter of claim 1 of auxiliary request 5 also lacks inventive step.

7.3 Auxiliary request 5 is not allowable for lack of compliance with Article 56 EPC.

8. Auxiliary requests 6 to 9 - Article 56 EPC

8.1 In claim 1 of auxiliary requests 6 to 9 the glutathione precursor is further defined as being: "cysteine or a derivative or precursor thereof" (auxiliary request 6), "cysteine or a derivative or precursor thereof which is acetylated or aminated on the side chain of cysteine" (auxiliary request 7), "N-acetyl cysteine or a derivative thereof" (auxiliary request 8) or "N-acetyl cysteine" (auxiliary request 9).

8.2 As is evident e.g. from dependent claim 5 in auxiliary requests 6 to 8, all these further definitions of the glutathione precursor encompass N-acetyl cysteine. N-acetyl cysteine is also specifically taught in D3 (page 4909, left column, line 5). Hence, for the same reasons as for the main request, the subject-matter of claim 1 of auxiliary requests 6 to 9 also lacks inventive step.

8.3 Auxiliary requests 6 to 9 are thus not allowable for lack of compliance with Article 56 EPC.
9. **Auxiliary request 10 - Article 56 EPC**

9.1 In claim 1 of auxiliary request 10 the neuropsychiatric disorder is further defined as being "characterised by: (i) aberrant, unwanted or otherwise inappropriate oxidative stress; and/or (ii) inadequate glutathione metabolism, in the brain of a mammal".

9.2 According to dependent claim 4, schizophrenia is encompassed in this functional definition of the neuropsychiatric disorder. Hence, for the same reasons as given above for the main request and for auxiliary request 5, the subject-matter of claim 1 of auxiliary request 10 also lacks inventive step.

9.3 Auxiliary request 10 is thus not allowable for lack of compliance with Article 56 EPC.

10. **Auxiliary requests 11 to 34 and further auxiliary requests**

10.1 For none of these auxiliary requests amended versions of the patent, or at least amended sets of claims, have been submitted by the appellant. For auxiliary requests 11 to 34 only a table was provided in the statement of grounds of appeal summarily indicating how each of auxiliary requests 11 to 34 was to be derived from the main and auxiliary requests 1 to 9. The appellant further stated "It will be appreciated that any of these Requests may also be combined with the feature of Auxiliary Request 10. In the interest of conciseness these further combinations are not listed below, but it is hereby expressly confirmed that this combination is contemplated, if required."
10.2 Pursuant to Article 113(2) EPC, the European Patent Office shall examine, and decide upon, the European patent application or the European patent only in the text submitted to it, or agreed, by the applicant or the proprietor of the patent. It derives therefrom that it is the responsibility of the applicant or patent proprietor to define the claimed subject-matter and to submit the text in which the patent should be granted or maintained.

10.3 It may sometimes be useful, especially in order to avoid an unnecessary inflation of the appeal file, that an applicant or patent proprietor indicates on an abstract level and early in the appeal proceedings what will be the fall-back positions. This cannot, however, replace the filing of the actual text.

10.4 Accordingly, in the present case, other than the main and auxiliary requests 1 to 10, there is no further text of an amended patent on file – or at least of amended sets of claims – which could be considered by the board.
Order

For these reasons it is decided that:

The appeal is dismissed.

The Registrar: The Chairman:

M. Schalow A. Lindner

Decision electronically authenticated