quality of life. Accordingly, there remains a need to identify one or more biological markers, genetic markers, or combinations thereof, that are associated with disorders related to response to a V_{1B} antagonist.

SUMMARY OF THE INVENTION

[0005] In one aspect there is provided a method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist and treating a subject identified as a suitable candidate for said treatment, the method comprising the steps of:

(a) providing a biological sample from the subject; and

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation;

wherein the presence of the marker indicates that the subject is a suitable candidate for treatment with a V_{1B} antagonist; and

(c) treating a subject that is identified as a suitable candidate in step (b) with a V_{1B} antagonist.

[0005a] In another aspect there is provided a method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist and treating a subject identified as a suitable candidate for said treatment, the method comprising the steps of:

(c) providing a biological sample from the subject; and

(d) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation;

wherein if the HPA axis function marker is present at a level above about the 60^{th} percentile of the distribution of the marker in a normal subject sample, the subject is suitable for treatment with a V_{1B} antagonist; and

(c) treating a subject that is identified as a suitable for treatment in step (b) with a V_{1B} antagonist.

[0005b] In another aspect there is provided method for monitoring a subject's response to treatment with a V_{1B} antagonist, comprising

(a) providing a biological sample from the subject receiving treatment with a a V_{1B} antagonist;

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation; and wherein a greater than 25% change in the level of the marker as compared to a baseline, indicates that the V_{1B} antagonist is useful for treating the subject; and

(c) continuing or discontinuing treatment with the V_{1B} antagonist in the subject based on the change in the level of the marker as compared to baseline detected in step (b).

[0005c] In another aspect there is provided a kit for stabilizing AVP in a plasma sample, wherein the kit comprises one or more collection tubes comprising one or more protease inhibitors.

[0005d] In one aspect, the present invention is directed to a method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist. The method may comprise providing a biological sample from a subject and then detecting an HPA axis function marker in the sample. Alternatively, the method comprises detecting an HPA axis function marker in a biological sample obtained from the subject. The presence of the marker indicates that the subject is a suitable candidate for treatment with a V_{1B} antagonist. The HPA axis function marker being present at a level greater than about the 60^{th} percentile, preferably greater than about the 65^{th}, 70^{th}, 75^{th}, 80^{th}, 85^{th}, 90^{th}, or 95^{th} percentile, of the distribution of the marker in a normal subject sample indicates that the subject is a particularly suitable candidate for treatment with a V_{1B} antagonist. The subject may have a disorder characterized by HPA axis dysfunction.

[0006] The HPA axis function marker may be a nucleotide sequence comprising SEQ ID NO:1 (LHPP res7088418) and a nucleotide sequence comprising SEQ ID NO:2 (AKRID1 rs17169521); a nucleotide sequence comprising an NR3C1 genotype, or a combination thereof. The NR3C1 genotype may be SEQ ID NO:3 (rs10482672) or SEQ ID NO:4 (rs17100236). The marker may be detected by genotyping, such as amplifying

the nucleic acid comprising the marker and then detecting the amplified nucleic acids, thereby detecting the marker. The marker may be detected by sequencing.

[0007] In another aspect, the present invention is directed to a method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist. The method may comprise providing a biological sample from a subject and then detecting an HPA axis function marker in the sample. If the HPA axis function marker is present at a level above about the 60^{th} percentile of the distribution of the marker in a normal subject sample, the subject is a suitable candidate for treatment with a V_{1B} antagonist. The subject may have a disorder characterized by HPA axis dysfunction. The HPA axis function marker may be present at a level greater than about the 65^{th}, 70^{th}, 75^{th}, 80^{th}, 85^{th}, 90^{th}, and 95^{th} percentile of the distribution of the HPA axis function marker in a normal subject sample. The marker may be AVP, copeptin, cortisol, cortisone, ACTH, a hepatic metabolite of

We claim:

1. A method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist and treating a subject identified as a suitable candidate for said treatment, the method comprising the steps of:

(a) providing a biological sample from the subject; and

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation;

wherein the presence of the marker indicates that the subject is a suitable candidate for treatment with a V_{1B} antagonist; and

(c) treating a subject that is identified as a suitable candidate in step (b) with a V_{1B} antagonist.

2. The method of claim 1, wherein the HPA axis function marker is selected from the group consisting of a nucleotide sequence comprising SEQ ID NO:1 (LHPP rs7088418) and a nucleotide sequence comprising SEQ ID NO:2 (AKRID1 rs17169521); a nucleotide sequence comprising an NR3C1 genotype; and combinations thereof.

3. The method of claim 2, wherein the NR3C1 genotype is selected from the group consisting of SEQ ID NO:3 (rs10482672) and SEQ ID NO:4 (rs17100236).

4. The method of claim 1, wherein the marker is detected by is detected by genotyping.

5. The method of claim 4, wherein the genotyping comprises

(a) amplifying a nucleic acid comprising the marker; and

(b) detecting the amplified nucleic acids, thereby detecting the marker.

6. The method of claim 5, wherein the marker is detected by sequencing.

7. A method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist and treating a subject identified as a suitable candidate for said treatment, the method comprising the steps of:

(a) providing a biological sample from the subject; and

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation;

wherein if the HPA axis function marker is present at a level above about the 60th percentile of the distribution of the marker in a normal subject sample, the subject is suitable for treatment with a V_{1B} antagonist; and

(c) treating a subject that is identified as a suitable for treatment in step (b) with a V_{1B} antagonist

8. The method of claim 7, wherein the HPA axis function marker is present at a level greater than a percentile selected from the group consisting of about the 65th, 70th, 75th, 80th, 85th, 90th, and 95th percentile of the distribution of the HPA axis function marker in a normal subject sample.

9. The method of claim 7, wherein the marker is selected from the group consisting of AVP, copeptin, cortisol, cortisone, ACTH, hepatic metabolite of cortisol, hepatic metabolite of cortisone, CRH, and a combination thereof.

10. The method of claim 9, wherein the copeptin is plasma copeptin.

11. The method of claim 9, wherein the AVP is plasma AVP.

12. The method of claim 9, wherein the hepatic metabolite of cortisol is selected from the group consisting of alpha-tetrahydrocortisol, beta-tetrahydrocortisol, alpha-cortol, beta-cortol, alpha-cortolic acid, beta-cortolic acid, and a combination thereof.

13. The method of claim 9, wherein the hepatic metabolite of cortisone is selected from the group consisting of tetrahydrocortisone, cortolone, cortolonic acid, and a combination thereof.

14. The method of claim 1 or 7, wherein the biological sample is selected from the group consisting of a nucleic acid containing sample, serum, plasma, blood, urine, and saliva.

15. The method of claim 14, wherein the biological sample is urine.

16. The method of claim 15, wherein the marker is the sum of urine amounts of cortisol, cortisone, alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone.

17. The method of claim 15, wherein the marker is the sum of urine amounts of alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone.

18. The method of claim 16 or 17, wherein the sum of the urine amounts is divided by the amount of creatinine in the same urine sample.

19. The method of claim 16, 17 or 18, wherein the urine sample is a 24-hour collection of urine from the subject.

20. The method of claim 16, 17 or 18, wherein the urine sample is an overnight collection of urine from the subject.

21. The method of claim 16, 17 or 18, wherein the urine sample is collected from the subject in a single void.

22. The method of claim 7, wherein the sample is a plasma sample that contains greater than or equal to 8.6 pg/mL AVP as determined by radioimmunoassay.

23. The method of claim 7, wherein the sample is a plasma sample that contains greater than or equal to 2.8 ng/mL of copeptin as determined by enzyme immunoassay.

24. The method of claim 7, wherein the sample is urine sample that contains a sum of cortisol, cortisone, alpha-tetrahydrocortiso, beta-tetrahydrocortisol and tetrahydrocortisone greater than or equal to 3.44 mg per mg of creatinine.

25. The method of claim 7, wherein the marker is detected by an immunoassay.

26. The method of claim 7, wherein the marker is detected by mass spectrometry.

27. The method of claim 1 or 7, wherein the disorder is selected from the group consisting of Cushing's syndrome, dementia, cognitive impairment, mood disorder, anxiety disorder, substance-related disorder, osteoporosis, arthritis, diabetes, dyslipidemia, obesity, hypertension, pain, glaucoma, and combinations thereof.

28. The method of claim 27, wherein the mood disorder is depression.

29. The method of claim 28, wherein the depression is major depressive disorder.

30. The method of claim 27, wherein the anxiety disorder is selected from the group consisting of post-traumatic stress disorder, generalized anxiety disorder and panic disorder.

31. The method of claim 27, wherein the substance-related disorder is selected from the group consisting of alcohol dependence or abuse, and drug dependence or abuse.

32. The method of claim 27, wherein the dementia is of the Alzheimer's type.

33. The method of claim 27, wherein the cognitive impairment is mild cognitive impairment due to Alzheimer's disease.

34. A method for monitoring a subject's response to treatment with a V_{1B} antagonist, comprising

(a) providing a biological sample from the subject receiving treatment with a a V_{1B} antagonist;

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation; and wherein a greater than 25% change in the level of the marker as compared to a baseline, indicates that the V_{1B} antagonist is useful for treating the subject; and

(c) continuing or discontinuing treatment with the V_{1B} antagonist in the subject based on the change in the level of the marker as compared to baseline detected in step (b).

35. The method of claim 34, wherein the HPA axis function marker is selected from the group consisting of cortisol; cortisone; corticotrophin releasing hormone adrenocorticotrophin hormone (ACTH); hepatic metabolite of cortisol, hepatic metabolite of cortisone, and combinations thereof.

36. The method of claim 35, wherein the hepatic metabolite of cortisone is selected from the group consisting of tetrahydrocortisone, cortolone, cortolonic acid, and a combination thereof.

37. The method of claim 35, wherein the marker is the sum of urine amounts of cortisol, cortisone, alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone.

38. The method of claim 35, wherein the marker is the sum of urine amounts of alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone.

39. The method of claim 37 or 38, wherein the sum of the urine amounts is divided by the amount of creatinine in the same urine sample.

40. The method of claim 34, wherein the baseline is cortisol at a level of between 3 pg/mL and 13 pg/mL.

41. The method of claim 34, wherein the baseline of cortisol is determined using enzyme immunoassay.

42. The method of claim 34, wherein the baseline indicates the level of the HPA axis function marker in a sample taken from the subject prior to beginning V1B antagonist therapy.

43. The method of claim 35, wherein the ACTH is plasma ACTH.

44. The method of claim 34, wherein the biological sample is selected from the group consisting of a nucleic acid containing sample, serum, plasma, blood, urine, and saliva.

45. The method of claim 34, wherein the disorder is selected from the group consisting of Cushing's syndrome, dementia, cognitive impairment, mood disorder, anxiety disorder, substance-related disorder, osteoporosis, arthritis, diabetes, dyslipidemia, obesity, hypertension, pain, glaucoma, and combinations thereof.

46. The method of claim 45, wherein the mood disorder is depression.

47. The method of claim 46, wherein the depression is major depressive disorder.

48. The method of claim 45, wherein the anxiety disorder is selected from the group consisting of post-traumatic stress disorder, generalized anxiety disorder and panic disorder.

49. The method of claim 45, wherein the substance-related disorder is selected from the group consisting of alcohol dependence or abuse, and drug dependence or abuse.

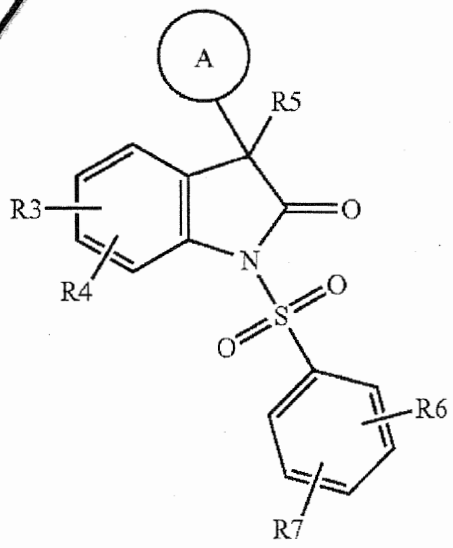
50. The method of claim 45, wherein the dementia is of the Alzheimer's type.

51. The method of claim 45, wherein the cognitive impairment is mild cognitive impairment due to Alzheimer's disease.

52. The method of claim 34, wherein the greater than 25% change in the level of the marker is an increased change compared to the baseline.

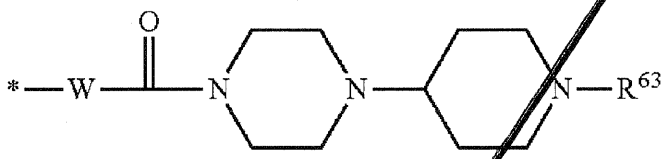
53. The method of claim 34, wherein the greater than 25% change in the level of the marker is a decreased change compared to the baseline.

54. The method of claim 1, 7, or 34, wherein the V_{1B} antagonist is of formula I:



in which A is an aromatic heteromonocyclic ring, where the heterocycles are 5- or 6-membered rings and comprise up to 4 heteroatoms selected from the group consisting of N, O and S, where not more than one of the heteroatoms is an oxygen or sulfur atom, and A may be substituted by radicals R11, R12 and/or R13, where R11, R12 and R13 at each occurrence are selected independently of one another from the group consisting of hydrogen chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, R3 and R4 are selected independently of one another from the group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, or R_3 and R_4 are connected to give -CH=CH-CH=CH-, -$(CH_2)_4$- or -$(CH_2)_3$-,

R5 is

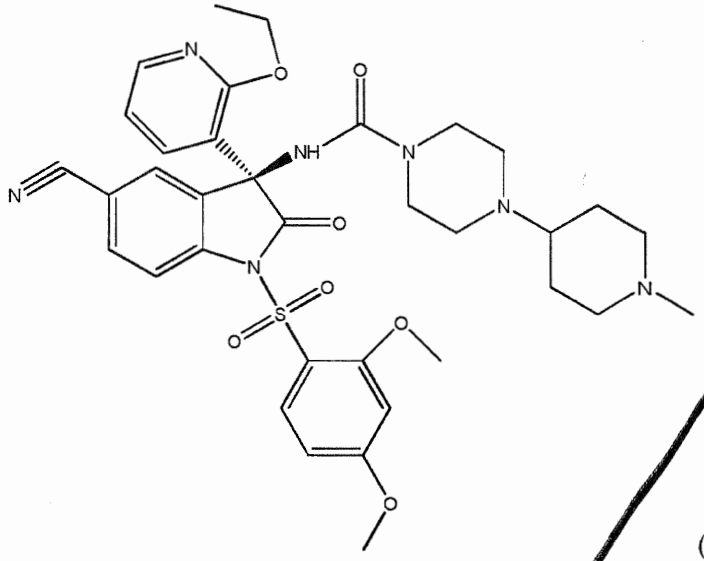


wherein W is selected from the group consisting of NR54, NR54-(C_1-C_4-alkylen) and a bond, R54 is independently selected from the group consisting of hydrogen, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, phenyl and C_1-C_4-alkylen-phenyl, where the phenyl ring may be substituted by up to two radicals R59, R59 is independently selected from the group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, R63 is independently of one another from the group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, R6 and R7 are selected independently of one another from the group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl atoms, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, and their tautomeric forms, enantiomeric and diastereomeric forms thereof.

55. The method of claim 54, wherein A is an aromatic heteromonocyclic system comprising 1 or 2 heteroatoms, wherein one of the 2 heteroatoms is nitrogen.

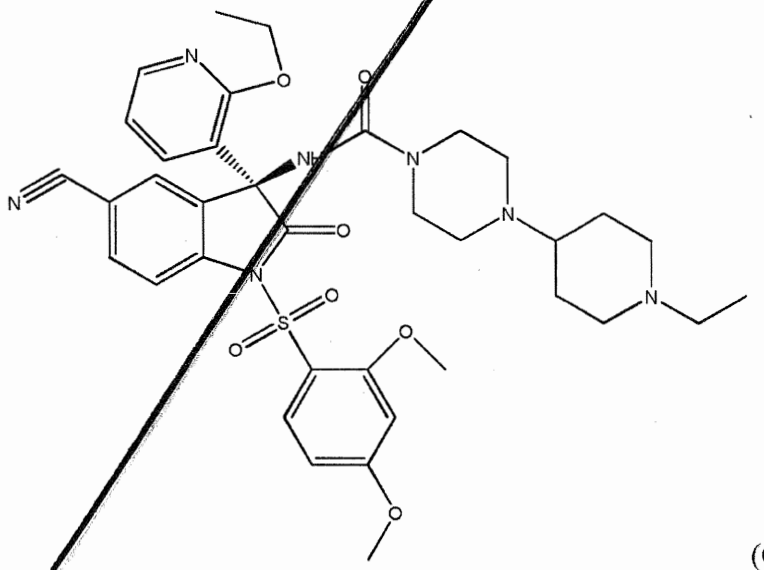
56. The method of claim 54, wherein A is selected from the group consisting of pyrimidine, pyridine, pyridazine, pyrazine, thiazole, imidazole, thiophene- and furan.

57. The method of claim 54, wherein the V_{1B} antagonist is:



(Compound A).

58. The method of claim 54, wherein the V_{1B} antagonist is:



(Compound B).

59. The method of claim 1 or 7, wherein the method is used to screen subjects for eligibility for a clinical trial.

60. The method of claim 1 or 7, wherein the method is used to stratify randomization of subjects for a clinical trial.

61. The method of claim 1 or 7, wherein the method is used to stratify analysis of a clinical trial.

62. A kit for stabilizing AVP in a plasma sample, wherein the kit comprises one or more collection tubes comprising one or more protease inhibitors.

63. A kit for assay of AVP in a blood-derived matrix, wherein the kit comprises a collection tube and instructions for stabilizing AVP at room temperature.

We claim:

1. A method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist and treating a subject identified as a suitable candidate for said treatment, the method comprising the steps of:

(a) providing a biological sample from the subject; and

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation;

wherein the presence of the marker indicates that the subject is a suitable candidate for treatment with a V_{1B} antagonist; and

(c) treating a subject that is identified as a suitable candidate in step (b) with a V_{1B} antagonist.

2. A method for determining whether a subject is a suitable candidate for treatment with a V_{1B} antagonist and treating a subject identified as a suitable candidate for said treatment, the method comprising the steps of:

(a) providing a biological sample from the subject; and

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation;

wherein if the HPA axis function marker is present at a level above about the 60^{th} percentile of the distribution of the marker in a normal subject sample, the subject is suitable for treatment with a V_{1B} antagonist; and

(c) treating a subject that is identified as a suitable for treatment in step (b) with a V_{1B} antagonist.

3. The method of claim 2, wherein the HPA axis function marker is present at a level greater than a percentile selected from the group consisting of about the 65^{th}, 70^{th}, 75^{th}, 80^{th}, 85^{th}, 90^{th}, and 95^{th} percentile of the distribution of the HPA axis function marker in a normal subject sample.

4. The method of claim 2, wherein the marker is selected from the group consisting of AVP, copeptin, cortisol, cortisone, ACTH, hepatic metabolite of cortisol, hepatic metabolite of cortisone, CRH, and a combination thereof.

5. The method of claim 4, wherein the copeptin is plasma copeptin; wherein the AVP is plasma AVP; wherein the hepatic metabolite of cortisol is selected from the group

consisting of alpha-tetrahydrocortisol, beta-tetrahydrocortisol, alpha-cortol, beta-cortol, alpha-cortolic acid, beta-cortolic acid, and a combination thereof; and wherein the hepatic metabolite of cortisone is selected from the group consisting of tetrahydrocortisone, cortolone, cortolonic acid, and a combination thereof.

6. The method of claim 1 or 2, wherein the biological sample is selected from the group consisting of a nucleic acid containing sample, serum, plasma, blood, urine, and saliva.

7. The method of claim 6, wherein the biological sample is urine, and wherein the marker is:

- the sum of urine amounts of cortisol, cortisone, alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone, or
- the sum of urine amounts of alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone.

8. The method of claim 2, wherein the sample is selected from:

- a plasma sample that contains greater than or equal to 8.6 pg/mL AVP as determined by radioimmunoassay;
- a plasma sample that contains greater than or equal to 2.8 ng/mL of copeptin as determined by enzyme immunoassay; and
- a urine sample that contains a sum of cortisol, cortisone, alpha-tetrahydrocortiso, beta-tetrahydrocortisol and tetrahydrocortisone greater than or equal to 3.44 mg per mg of creatinine.

9. A method for monitoring a subject's response to treatment with a V_{1B} antagonist, comprising

(a) providing a biological sample from the subject receiving treatment with a a V_{1B} antagonist;

(b) detecting a hypothalamus-pituitary-adrenal ("HPA") axis function marker;

wherein the subject has a disorder characterized by HPA axis dysregulation; and wherein a greater than 25% change in the level of the marker as compared to a baseline, indicates that the V_{1B} antagonist is useful for treating the subject; and

(c) continuing or discontinuing treatment with the V_{1B} antagonist in the subject based on the change in the level of the marker as compared to baseline detected in step (b).

10. The method of claim 9, wherein the HPA axis function marker is selected from the group consisting of cortisol; cortisone; corticotrophin releasing hormone adrenocorticotrophin hormone (ACTH); hepatic metabolite of cortisol, hepatic metabolite of cortisone, and combinations thereof.

11. The method of claim 1, 2 or 10, wherein the marker is :

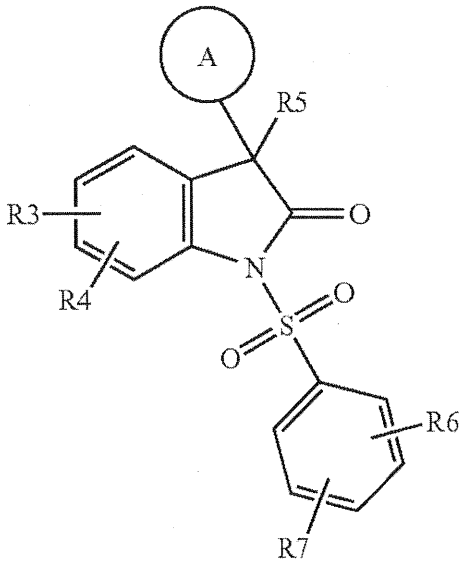
- the sum of urine amounts of cortisol, cortisone, alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone; or
- the sum of urine amounts of alpha-tetrahydrocortisol, beta-tetrahydrocortisol, and tetrahydrocortisone.

12. The method of claim 11, wherein the sum of the urine amounts is divided by the amount of creatinine in the same urine sample.

13. The method of claim 1, 2 or 9, wherein the disorder is selected from the group consisting of Cushing's syndrome, dementia, cognitive impairment, mood disorder, anxiety disorder, substance-related disorder, osteoporosis, arthritis, diabetes, dyslipidemia, obesity, hypertension, pain, glaucoma, and combinations thereof.

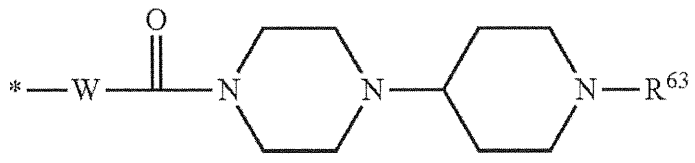
14. The method of claim 13, wherein the mood disorder is depression, in particular major depressive disorder; wherein the anxiety disorder is selected from the group consisting of post-traumatic stress disorder, generalized anxiety disorder and panic disorder; wherein the substance-related disorder is selected from the group consisting of alcohol dependence or abuse, and drug dependence or abuse; wherein the dementia is of the Alzheimer's type; and wherein the cognitive impairment is mild cognitive impairment due to Alzheimer's disease.

15. The method of claim 1, 2 or 9, wherein the V_{1B} antagonist is of formula I:



in which A is an aromatic heteromonocyclic ring, where the heterocycles are 5- or 6-membered rings and comprise up to 4 heteroatoms selected from the group consisting of N, O and S, where not more than one of the heteroatoms is an oxygen or sulfur atom, and A may be substituted by radicals R11, R12 and/or R13, where R11, R12 and R13 at each occurrence are selected independently of one another from the group consisting of hydrogen chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, R3 and R4 are selected independently of one another from the group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, or R_3 and R_4 are connected to give -CH=CH-CH=CH-, -$(CH_2)_4$- or -$(CH_2)_3$-,

R5 is



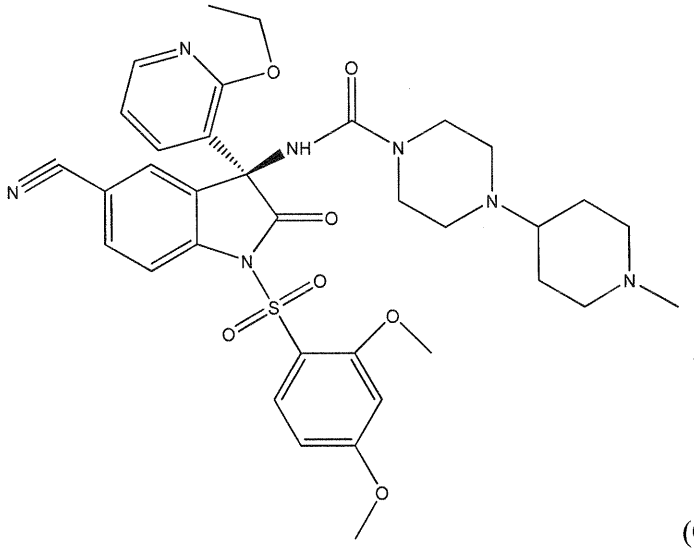
wherein W is selected from the group consisting of NR54, NR54-(C_1-C_4-alkylen) and a bond, R54 is independently selected from the group consisting of hydrogen, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, phenyl and C_1-C_4-alkylen-phenyl, where the phenyl ring may be substituted by up to two radicals R59, R59 is independently selected from the

group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, R63 is independently of one another from the group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, R6 and R7 are selected independently of one another from the group consisting of hydrogen, chlorine, bromine, iodine, fluorine, CN, CF_3, OCF_3, NO_2, OH, O-C_1-C_4-alkyl atoms, O-phenyl, O-C_1-C_4-alkylen-phenyl, phenyl, C_1-C_6-alkyl, C_2-C_6-alkenyl, C_2-C_6-alkynyl, NH_2, NH(C_1-C_4-alkyl) and N(C_1-C_4-alkyl)$_2$, and their tautomeric forms, enantiomeric and diastereomeric forms thereof.

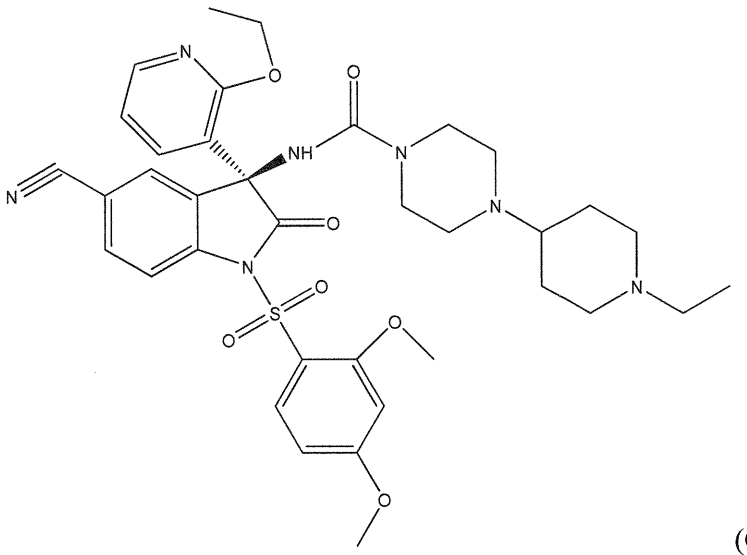
16. The method of claim 15, wherein A is an aromatic heteromonocyclic system comprising 1 or 2 heteroatoms, wherein one of the 2 heteroatoms is nitrogen.

17. The method of claim 15, wherein A is selected from the group consisting of pyrimidine, pyridine, pyridazine, pyrazine, thiazole, imidazole, thiophene- and furan.

18. The method of claim 15, wherein the V_{1B} antagonist is:



(Compound A) or



(Compound B).

19. The method of claim 1 or 2, wherein the method is used to:

- screen subjects for eligibility for a clinical trial;
- stratify randomization of subjects for a clinical trial; or
- stratify analysis of a clinical trial.

20. A kit for stabilizing AVP in a plasma sample, wherein the kit comprises one or more collection tubes comprising one or more protease inhibitors.