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SUPERINTENDENCIA DE INDUSTRIA Y COMERCIO

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Doctor

GIANCARLO MARCENARO JIMÉNEZ

Superintendente Delegado Para la Propiedad Industrial

EXPEDIENTE: 03-077.204

**ASUNTO: Solicitud de patente a nombre de
The Procter & Gamble Company**

Estimado Doctor:

En mi calidad de apoderada de la sociedad solicitante de la patente de la referencia manifiesto a usted lo siguiente:

1. Mediante memorial radicado en ese despacho el 28 de junio de 2004, el cual no ha sido resuelto aún, solicité que se realizara el examen de patentabilidad.
2. En vista de que han transcurrido **2 años y 3 meses**, sin que se haya atendido dicha solicitud, ruego a usted se sirva dar las instrucciones del caso para que se proceda de inmediato a resolver este asunto.

Cordialmente,


JIMENA ESCOBAR URIBE
CC 39.779.452 de Usaquén.
T.P. 68228

DIVISION DE NUEVAS CREACIONES

SUPERINTENDENCIA DE INDUSTRIA Y COMERCIO



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RADICACIÓN EXPEDIENTE No 03-77204

EL EXTRACTO DE LA PRESENTE SOLICITUD DE PATENTE DE INVENCION

FUE PUBLICADO EN LA GACETA DE PROPIEDAD INDUSTRIAL No 538 DE

FECHA 31 DE MARZO DE 2004 EL TERMINO HABIL SEÑALADO POR LA

LEY EXPIRA EL 01 DE JULIO DEL 2004

NOTA: Dentro del plazo de los seis (6) meses siguientes a partir de la fecha de publicación en la gaceta de Propiedad Industrial, deberá solicitar y cancelar el examen de patentabilidad so pena de que la solicitud caiga en abandono, de acuerdo con lo establecido en el Artículo 44 de la Decisión 486/2000.

SE PRESENTARON OPOSICIONES POR PARTE DE TERCEROS.

SI _____

NO__ _X _



US005786203A

786

United States Patent [19]
Lovenberg et al.

[11] **Patent Number:** 5,786,203
[45] **Date of Patent:** Jul. 28, 1998

- [54] **ISOLATED NUCLEIC ACID ENCODING CORTICOTROPIN-RELEASING FACTOR₂ RECEPTORS**
- [75] **Inventors:** Timothy W. Lovenberg, Carlsbad; Tilman Oltersdorf, Cardiff; Chen Wang Liaw, San Diego; Dimitri E. Grigoriadis, Carlsbad; Errol DeSouza, Del Mar, all of Calif.
- [73] **Assignee:** Neurocrine Biosciences, Inc., San Diego, Calif.
- [21] **Appl. No.:** 381,433
- [22] **Filed:** Jan. 31, 1995

Related U.S. Application Data

- [63] Continuation-in-part of Ser. No. 259,959, Jun. 14, 1994, abandoned.
- [51] **Int. CL⁶** C07K 14/705; C12N 5/10; C12N 15/12
- [52] **U.S. CL.** 435/252.3; 435/69.1; 435/320.1; 536/23.5; 530/350
- [58] **Field of Search** 435/69.1, 252.3, 435/320.1, 7.1, 7.2; 530/350; 536/23.5; 247/60

[56] **References Cited**

FOREIGN PATENT DOCUMENTS

695 802 A2 2/1996 European Pat. Off. .

OTHER PUBLICATIONS

Chang et al., "Identification of a Seven Transmembrane Helix Receptor for Corticotropin-Releasing Factor and Sauvagine in Mammalian Brain," *Neuron* 11:1187-1195, 1993.

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Primary Examiner—John Ulm
Attorney, Agent, or Firm—Seed & Berry LLP

[57] **ABSTRACT**

The present invention provides isolated nucleic acid molecules encoding CRF₂ receptors, recombinant expression vectors and host cells suitable for expressing such receptors, as well as compositions and methods which utilize such receptors.

13 Claims, 4 Drawing Sheets

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[75] Inventors: Timothy W. Lovenberg, Carlsbad; Tilman Oltersdorf, Cardiff; Chen Wang Liaw, San Diego; Dimitri E. Grigoriadis, Carlsbad; Errol DeSouza, Del Mar, all of Calif.

[73] Assignee: Neurocrine Biosciences, Inc., San Diego, Calif.

[21] Appl. No.: 381,433

[22] Filed: Jan. 31, 1995

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[58] Field of Search 435/69.1, 252.3, 435/320.1, 7.1, 7.2; 530/350; 536/23.5; 247/60

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De Souza, E., "Corticotropin-Releasing Factor Receptors in the Rat Central Nervous System: Characterization and Regional Distribution," *Journal of Neuroscience* 7(1): 88-100, 1987.

Primary Examiner—John Ulm
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[57] ABSTRACT

The present invention provides isolated nucleic acid molecules encoding CRF₂ receptors, recombinant expression vectors and host cells suitable for expressing such receptors, as well as compositions and methods which utilize such receptors.

13 Claims, 4 Drawing Sheets

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ISOLATED NUCLEIC ACID ENCODING CORTICOTROPIN-RELEASING FACTOR₂ RECEPTORS

CROSS-REFERENCE TO RELATED APPLICATIONS

This application is a continuation-in-part of application U.S. Ser. No. 08/259,959, filed Jun. 14, 1994, now abandoned.

TECHNICAL FIELD

The present invention relates generally to cell surface receptors, and more specifically, to Corticotropin-Releasing Factor₂ receptors.

BACKGROUND OF THE INVENTION

Corticotropin-releasing factor ("CRF") is a 41-amino acid peptide originally isolated from the hypothalamus by virtue of its ability to stimulate the production of adrenocorticotrophic hormone ("ACTH") and other proopiomelanocortin ("POMC") products of the anterior pituitary (Vale et al., *Science* 213:1394-1397, 1981). Briefly, CRF is believed to initiate its biological effects by binding to a plasma membrane receptor which is distributed throughout the brain (DeSouza et al., *Science* 224:1449-1451, 1984), pituitary (Wynn et al., *Biochem. Biophys. Res. Comm.* 110:602-608, 1983), adrenals (Udelsman et al., *Nature* 319:147-150, 1986) and spleen (Webster, E. L., and E. B. DeSouza, *Endocrinology* 122:609-617, 1988). This receptor is coupled to a GTP-binding protein (Perrin et al., *Endocrinology* 118:1171-1179, 1986) which mediates CRF-stimulated increase in intracellular cAMP (Bilezikjian, L. M., and W. W. Vale, *Endocrinology* 113:657-662, 1983).

In addition to its role in stimulating the production of ACTH and POMC, CRF is also believed to coordinate many of the endocrine, autonomic, and behavioral responses to stress, and may be involved in the pathophysiology of affective disorders. Moreover, CRF is believed to be a key intermediary in communication between the immune, central nervous, endocrine and cardiovascular systems (Crofford et al., *J. Clin. Invest.* 90:2555-2564, 1992; Sapolsky et al., *Science* 238:522-524, 1987; Tilders et al., *Regul. Peptides* 5:77-84, 1982; Fisher et al., *Reg. Peptide* 5:153-161, 1983).

A receptor for CRF has been cloned from rat (Perrin et al., *Endo* 133(6):3058-3061, 1993), and human brain (Chen et al., *PNAS* 90(19):8967-8971, 1993; Vita et al., *FEBS* 335(1):1-5, 1993). This receptor is a 415 amino acid protein comprising seven membrane spanning domains, and has a predicted molecular weight of 44,000 daltons. A comparison of identity between rat and human sequences shows a high degree of homology (97%) at the amino acid level. In addition, Scatchard analysis of recombinantly produced human receptor demonstrates a single component site, with a high affinity (K_d of 1.6 ± 0.3 nM) for CRF.

The present invention provides new, previously unidentified CRF receptors, designated as the "Corticotropin-Releasing Factor₂" ("CRF₂"), Corticotropin-Releasing Factor receptors. In addition, the present invention provides compositions and methods which utilize such CRF₂ receptors, as well as other, related advantages.

SUMMARY OF THE INVENTION

Briefly stated, the present invention provides compositions and methods which utilize CRF₂ receptors (also termed

"CRF₂ Corticotropin-Releasing Factor receptors" or "CRF₂R"). Within one aspect of the present invention, isolated nucleic acid molecules are provided which encode CRF₂ receptors. Within one embodiment, nucleic acid molecules are provided which encode a CRF₂ receptor such as that disclosed in Sequence ID. No. 4, from amino acid number 1 to amino acid number 411. Within another embodiment, nucleic acid molecules are provided which comprise the sequence of nucleotides in Sequence ID. No. 3, from nucleotide number 216 to nucleotide number 1449. Within another embodiment, nucleic acid molecules are provided which encode a CRF₂ receptor such as that disclosed in Sequence ID No. 2 from amino acid number 1 to amino acid number 431. Within another embodiment, nucleic acid molecules are provided which comprise the sequence of nucleotides in Sequence ID No. 1 from nucleotide number 44 to nucleotide number 1336. Nucleic acid molecules which encode CRF₂ receptors of the present invention may be isolated from virtually any warm-blooded animal, including for example, humans, macaques, horses, cattle, sheep, pigs, dogs, cats, rats and mice.

Within another aspect of the present invention, isolated nucleic acid molecules are provided which encode portions of a CRF₂ receptor, such as the N-terminal extracellular domain. Within one embodiment, isolated nucleic acid molecules are provided comprising the sequence of nucleotides in Sequence ID. No. 3, from nucleotide number 216 to nucleotide number 570. Within another embodiment, isolated nucleic acid molecules are provided which encode a protein having the amino acid sequence of Sequence ID. No. 4, from amino acid number 1 to amino acid number 118. Within another embodiment, isolated nucleic acid molecules are provided comprising the sequence of nucleotides in Sequence ID No. 1 from nucleotide number 44 to nucleotide number 451. Within another embodiment, isolated nucleic acid molecules are provided which encode a protein having the amino acid sequence of Sequence ID No. 2 from amino acid number 1 to amino acid number 138.

Within other aspects of the invention, expression vectors are provided which are capable of expressing the above-described nucleic acid molecules. Within other aspects, recombinant viral vectors are provided which are capable of directing the expression of the above-described nucleic acid molecules. Representative examples of such viral vectors include retroviral vectors, adenoviral vectors, and herpes simplex virus vectors. Also provided by the present invention are host cells which contain the above-described expression vectors, as well as the receptor or portions thereof which are encoded by the above-described nucleic acid molecules. Within other embodiments, isolated portions of CRF₂ receptors are provided, including for example, isolated portions of extracellular domains such as the N-terminal extracellular domain.

Within other aspects of the invention, isolated antibodies are provided which are capable of specifically binding to the above-described CRF₂ receptors. Within one embodiment, the antibodies may be selected from the group consisting of polyclonal antibodies, monoclonal antibodies, and antibody fragments. Within other embodiments, antibodies are provided which are capable of blocking the binding of CRF (or other substrates such as sauvagine or urotensin I) to a CRF₂ receptor. Within preferred embodiments, the antibodies may be selected from the group consisting of murine and human antibodies. Within preferred aspects of the invention, the above-noted antibodies are produced by hybridomas.

Within yet another aspect of the present invention, nucleic acid molecules are provided which are capable of specifi-

cally hybridizing to a nucleic acid molecule encoding any of the CRF₂ receptors described above. Such molecules may be between at least "y" nucleotides long, wherein "y" is any integer between 14 and 1230, and furthermore, may be selected suitable for use as probes or primers described below. Particularly preferred probes of the present invention are at least 18 nucleotides in length.

Within other aspects of the present invention, methods for detecting the presence of a compound which binds to a CRF₂ receptor are provided, comprising the steps of (a) exposing one or more compounds to cells that express CRF₂ receptors under conditions and for a time sufficient to allow binding of the compounds to the receptors, and (b) isolating compounds which bind to the receptors, such that the presence of a compound which binds to a CRF₂ receptor may be detected. Within another aspect, methods for detecting the presence of a compound which binds to a CRF₂ receptor are provided, comprising the steps of (a) exposing one or more compounds to a CRF₂ receptor N-terminal extracellular domain under conditions and for a time sufficient to allow binding of a compound to the N-terminal extracellular domain, and (b) isolating compounds which bind to the CRF₂ receptor N-terminal extracellular domain, such that the presence of a compound which binds to a CRF₂ receptor may be detected. Within one embodiment, the compounds are labeled with an agent selected from the group consisting of fluorescent molecules, enzymes, and radionuclides.

Within other aspects of the present invention, methods for determining whether a selected compound is a CRF₂ receptor agonist or antagonist are provided, comprising the steps of (a) exposing a selected compound to cells which express CRF₂ receptors under conditions and for a time sufficient to allow binding of the compound and an associated response in intracellular levels of cAMP, and (b) detecting either an increase or decrease in the level of intracellular cAMP, and thereby determining whether the selected compound is a CRF₂ receptor agonist or antagonist.

Within other aspects, methods are provided for detecting the presence of a CRF₂ receptor agonist or antagonist in a pool of compounds, comprising the steps of (a) exposing a pool of compounds to cells which express CRF₂ receptors under conditions and for a time sufficient to allow binding of the compound and an associated response in intracellular levels of cAMP, and (b) isolating compounds which either increase or decrease the intracellular level of cAMP, such that the presence of a CRF₂ receptor agonist or antagonist may be detected.

Within another aspect, methods for determining whether a selected compound is a CRF₂ receptor antagonist are provided, comprising the steps of (a) exposing a selected compound in the presence of a CRF₂ receptor agonist to a recombinant CRF₂ receptor coupled to a response pathway under conditions and for a time sufficient to allow binding of the compound to the receptor and an associated response through the pathway, and (b) detecting a reduction in the stimulation of the response pathway resulting from the binding of the compound to the CRF₂ receptor, relative to the stimulation of the response pathway by the CRF₂ receptor agonist alone, and therefrom determining the presence of a CRF₂ antagonist. Within other aspects, methods are provided for determining whether a selected compound is a CRF₂ receptor agonist, comprising the steps of (a) exposing a selected compound to a recombinant CRF₂ receptor coupled to a response pathway under conditions and for a time sufficient to allow binding of the compound to the receptor and an associated response through the pathway, and (b) detecting an increase in stimulation of the response

pathway resulting from the binding of the compound to the CRF₂ receptor, and therefrom determining the presence of a CRF₂ receptor agonist.

These and other aspects of the present invention will become evident upon reference to the following detailed description and attached drawings. In addition, various references are set forth below which describe in more detail certain procedures or compositions (e.g., plasmids, etc.), and are therefore incorporated by reference in their entirety.

BRIEF DESCRIPTION OF THE DRAWINGS

FIG. 1 schematically illustrates the structure of one representative CRF₂ receptor (CRF_{2B}; Sequence ID No. 2).

FIG. 2 schematically illustrates the structure of a second representative CRF₂ receptor (CRF_{2C}; Sequence ID No. 4).

FIG. 3 is a graph which depicts cAMP accumulation in cells transfected with the CRF₁ receptor.

FIG. 4 is a graph which depicts cAMP accumulation in cells transfected with the CRF₂ receptor.

DETAILED DESCRIPTION OF THE INVENTION

Definitions

Prior to setting forth the invention, it may be helpful to an understanding thereof to set forth definitions of certain terms to be used hereinafter.

"CRF₂ receptors" (also termed "CRF₂ Corticotropin-Releasing Factor receptors" or "CRF₂R") as utilized herein refers to receptor proteins which bind corticotropin-releasing factor and other proteins such as urotensin I and sauvagine. CRF₂ receptors may be distinguished from other receptors such as Corticotropin-Releasing Factor receptors based upon criteria such as affinity of substrate binding, tissue distribution, and sequence homology. For example, CRF₂ receptors of the present invention should be greater than 70% homologous, preferably greater than 75% to 80% homologous, more preferably greater than 85% to 90% homologous, and most preferably greater than 92%, 95%, or 97% homologous to the CRF₂ receptors disclosed herein (e.g., Sequence I.D. No. 3). In their native configuration, CRF₂ receptors are believed to exist as membrane bound proteins, consisting of an N-terminal extracellular domain, seven transmembrane domains separated by three intracellular and three extracellular loops, and a C-terminal intracellular domain (see FIGS. 1 and 2). As utilized within the context of the present invention CRF₂ receptors should be understood to include not only the proteins which are disclosed herein (see Sequence I.D. Nos. 2, 4 and 8), but substantially similar derivatives and analogs as discussed below.

"Nucleic acid molecule" refers to a nucleic acid sequence, in the form of a separate fragment or as a component of a larger nucleic acid construct, which has been derived from nucleic acid isolated at least once in substantially pure form, i.e., free of contaminating endogenous materials, and in a quantity or concentration enabling identification, manipulation, and recovery of the sequence and its component nucleotide sequences by standard biochemical methods, for example, using a cloning vector. Such sequences are preferably provided in the form of an open reading frame uninterrupted by internal nontranslated sequences, or introns, which are typically present in eukaryotic genes. Genomic nucleic acid containing the relevant sequences may also be used. Sequences of non-translated nucleic acid may be present 5' or 3' from the open reading

ANTIBODIES TO CRF₂ RECEPTORS

Within one aspect of the present invention, CRF₂ receptors, including derivatives thereof, as well as portions or fragments of these proteins such as the CRF₂ receptor peptides discussed above, may be utilized to prepare antibodies which specifically bind to CRF₂ receptors. Within the context of the present invention the term "antibodies" includes polyclonal antibodies, monoclonal antibodies, fragments thereof such as F(ab')₂ and Fab fragments, as well as recombinantly produced binding partners. These binding partners incorporate the variable regions from a gene which encodes a specifically binding monoclonal antibody. Antibodies are defined to be specifically binding if they bind to the CRF₂ receptor with a K_d of greater than or equal to 10⁷ M⁻¹. The affinity of a monoclonal antibody or binding partner may be readily determined by one of ordinary skill in the art (see Scatchard, *Ann. N.Y. Acad. Sci.* 51:660-672, 1949).

Polyclonal antibodies may be readily generated by one of ordinary skill in the art from a variety of warm-blooded animals such as horses, cows, goats, sheep, dogs, chickens, rabbits, mice, or rats. Briefly, the CRF₂ receptor is utilized to immunize the animal through intraperitoneal, intramuscular, intraocular, or subcutaneous injections. The immunogenicity of a CRF₂ receptor or CRF₂ receptor peptide may be increased through the use of an adjuvant such as Freund's complete or incomplete adjuvant. Following several booster immunizations, small samples of serum are collected and tested for reactivity to the CRF₂ receptor. A variety of assays may be utilized in order to detect antibodies which specifically bind to a CRF₂ receptor. Exemplary assays are described in detail in *Antibodies: A Laboratory Manual*, Harlow and Lane (eds.), Cold Spring Harbor Laboratory Press, 1988. Representative examples of such assays include: Countercurrent Immuno-Electrophoresis (CIEP), Radioimmunoassays, Radioimmunoprecipitations, Enzyme-Linked Immuno-Sorbent Assays (ELISA), Dot Blot assays, Inhibition or Competition assays, and sandwich assays (see U.S. Pat. Nos. 4,376,110 and 4,486,530; see also *Antibodies: A Laboratory Manual*, supra). Particularly preferred polyclonal antisera will give a signal that is at least three times greater than background. Once the titer of the animal has reached a plateau in terms of its reactivity to the CRF₂ receptor, larger quantities of polyclonal antisera may be readily obtained either by weekly bleedings, or by exsanguinating the animal.

Monoclonal antibodies may also be readily generated using well-known techniques (see U.S. Pat. Nos. RE 32,011, 4,902,614, 4,543,439, and 4,411,993; see also *Monoclonal Antibodies, Hybridomas: A New Dimension in Biological Analyses*, Plenum Press, Kennett, McKearn, and Bechtol (eds.), 1980, and *Antibodies: A Laboratory Manual*, Harlow and Lane (eds.), Cold Spring Harbor Laboratory Press, 1988). Briefly, within one embodiment a subject animal such as a rat or mouse is injected with a form of CRF₂ receptor suitable for generating an immune response against the CRF₂ receptor. Representative examples of suitable forms include, among others, cells which express the CRF₂ receptor, or peptides which are based upon the CRF₂ receptor sequence. Additionally, many techniques are known in the art for increasing the resultant immune response, for example, by coupling the receptor or receptor peptides to another protein such as ovalbumin or keyhole limpet hemocyanin (KLH), or through the use of adjuvants such as Freund's complete or incomplete adjuvant. The initial immunization may be through intraperitoneal, intramuscular, intraocular, or subcutaneous routes.

Between one and three weeks after the initial immunization the animal may be reimmunized with another booster immunization. The animal may then be test bled and the serum tested for binding to the CRF₂ receptor using assays as described above. Additional immunizations may also be accomplished until the animal has plateaued in its reactivity to the CRF₂ receptor. The animal may then be given a final boost of CRF₂ receptor or CRF₂ receptor peptide, and three to four days later sacrificed. At this time, the spleen and lymph nodes may be harvested and disrupted into a single cell suspension by passing the organs through a mesh screen or by rupturing the spleen or lymph node membranes which encapsidate the cells. Within one embodiment the red cells are subsequently lysed by the addition of a hypotonic solution, followed by immediate return to isotonicity.

Within another embodiment, suitable cells for preparing monoclonal antibodies are obtained through the use of in vitro immunization techniques. Briefly, an animal is sacrificed, and the spleen and lymph node cells are removed as described above. A single cell suspension is prepared, and the cells are placed into a culture containing a form of the CRF₂ receptor that is suitable for generating an immune response as described above. Subsequently, the lymphocytes are harvested and fused as described below.

Cells which are obtained through the use of in vitro immunization or from an immunized animal as described above may be immortalized by transfection with a virus such as the Epstein-Barr virus (EBV) (see Glasky and Reading, *Hybridoma* 8(4):377-389, 1989). Alternatively, within a preferred embodiment, the harvested spleen and/or lymph node cell suspensions are fused with a suitable myeloma cell in order to create a "hybridoma" which secretes monoclonal antibodies. Suitable myeloma lines are preferably defective in the construction or expression of antibodies, and are additionally syngeneic with the cells from the immunized animal. Many such myeloma cell lines are well known in the art and may be obtained from sources such as the American Type Culture Collection (ATCC), Rockville, Md. (see *Catalogue of Cell Lines & Hybridomas*, 6th ed., ATCC, 1988). Representative myeloma lines include: for humans, UC 729-6 (ATCC No. CRL 8061), MC/CAR-Z2 (ATCC No. CRL 8147), and SKO-007 (ATCC No. CRL 8033); for mice, SP2/0-Ag14 (ATCC No. CRL 1581), and P3X63Ag8 (ATCC No. TIB 9); and for rats, Y3-Ag1.2.3 (ATCC No. CRL 1631), and YB2/0 (ATCC No. CRL 1662). Particularly preferred fusion lines include NS-1 (ATCC No. TIB 18) and P3X63-Ag 8.653 (ATCC No. CRL 1580), which may be utilized for fusions with either mouse, rat, or human cell lines. Fusion between the myeloma cell line and the cells from the immunized animal may be accomplished by a variety of methods, including the use of polyethylene glycol (PEG) (see *Antibodies: A Laboratory Manual*, Harlow and Lane (eds.), Cold Spring Harbor Laboratory Press, 1988) or electrofusion (see Zimmerman and Vienken, *J. Membrane Biol.* 67:165-182, 1982).

Following the fusion, the cells are placed into culture plates containing a suitable medium, such as RPMI 1640 or DMEM (Dulbecco's Modified Eagles Medium) (JRH Biosciences, Lenexa, Kans.). The medium may also contain additional ingredients, such as Fetal Bovine Serum ("FBS," i.e., from Hyclone, Logan, Utah, or JRH Biosciences), thymocytes which were harvested from a baby animal of the same species as was used for immunization, or agar to solidify the medium. Additionally, the medium should contain a reagent which selectively allows for the growth of fused spleen and myeloma cells. Particularly preferred is the use of HAT (hypoxanthine, aminopterin, and thymidine)

the level of stimulation of cAMP relative to known agonists (e.g., sauvagine, urotensin I or CRF), and generally involve exposing a preparation of cells which express a biologically active CRF₂ receptor to the selected compound in the presence of radiolabelled ATP.

Within a further embodiment of the invention, the response pathway includes a reporter system, such as luciferase, or β -galactosidase (see Konig et al., *Mol. and Cell. Neuro.* 2:331-337, 1991). For examples, within one embodiment of the invention an expression vector is provided comprising a cyclic AMP response element such as a proenkephalin cyclic AMP response element, operably linked to β -galactosidase or luciferase cDNA. The expression vector is then stably transfected into a host cell, and the host cell then transfected with a second expression vector which expresses a CRF₂ receptor. Upon activation of the response pathway, elevated cAMP levels induce the expression of the reporter product, such as the β -galactosidase or luciferase. If the reporter product is luciferase, it is then exposed to luciferin, and photons which are released during the oxidation of luciferin by the luciferase are measured.

A variety of compounds may be screened utilizing such methods. Representative examples include blocking antibodies discussed above, CRF₂ receptor peptides, and CRF analogs (including both peptide and non-peptide ligands). In addition, large numbers of CRF analogs may be generated by saturation mutagenesis of nucleic acid sequences encoding CRF (e.g., Little, *Gene* 88:113-115, 1990; Hembers et al., *Gene* 88:143-151, 1989), by segment-directed mutagenesis (e.g., Shortle et al., *Proc. Natl Acad. Sci. USA* 77:5375-5379, 1980), by forced nucleotide misincorporation (e.g., Liao and Wise, *Gene* 88:107-111, 1990), or by use of randomly mutagenized oligonucleotides (Hutchison et al., *Proc. Natl. Acad. Sci. USA* 83:710-714, 1986). Individual transformants expressing a CRF₂ analog may then be cloned as discussed above, or pooled.

Other compounds which may also be screened utilizing methods of the present invention, include chemical compounds which are known and either available commercially (e.g., from Sigma Chemical Co., St. Louis, Mo.) or readily synthesized by one of skill in the art. In addition, numerous novel compositions which are provided in combinatorial libraries may also be readily screened utilizing the methods described herein, including for example organic and protein or peptide libraries (see e.g., Gold and Tuerk, U.S. Pat. No. 5,270,163; and Ladner et al., U.S. Pat. Nos. 5,096,815, 5,198,346, and 5,223,409).

Within a preferred aspect of the invention, the screening of chemical libraries (including peptide, small organic molecule or combinatorial chemistry-derived compound libraries) can be assessed in a high-throughput format using the expressed CRF₂ receptors. More specifically, within one embodiment of the invention, a high throughput receptor binding assay may be performed in a 96-well plate and is automated using the BIOMEK 2000 (Beckman, Fullerton Calif.). Briefly, 1×10^5 cells transfected with the CRF₂ receptor are grown in each well. To this, 0.05 ml of assay buffer (Dulbecco's Phosphate Buffered Saline, 10 mM MgCl₂, 20 μ M Bacitracin) with or without unlabeled r/hCRF (final concentration, 1 μ M) is added in duplicate to determine total binding and non-specific binding. In singlets, 0.05 ml of compounds (mixtures from 1 to 30 compounds) are added to each well in addition to 0.05 ml of either [¹²⁵I]-oCRF or [¹²⁵I]-r/hCRF (final concentration ~200 pM). The mixture is incubated for 2 hours at 22° C. Since the transfected cells are adherent, a centrifugation step to separate membrane-bound CRF is not necessary. Next, the fluid is aspirated and the

cells are washed three times with approximately 0.9 ml of PBS. After the third wash and aspiration, 0.2 ml of 4M guanidine thiocyanate is added to each well to solubilize the tissue. An aliquot (0.15 ml) of the solubilized sample is monitored in a gamma counter for radioactivity at approximately 80% efficiency. Compounds which demonstrate $\geq 50\%$ inhibition of [¹²⁵I]CRF binding are tested in a full dose response competition assay to determine the affinity of these compounds (K_i value) at the CRF₂ receptor.

Once purified partially, or to homogeneity, as desired, both CRF₂ receptor agonists and antagonists may be used therapeutically. Generally substantially pure recombinant CRF antagonists of at least about 50% homogeneity are preferred, at least about 70%-80% homogeneity more preferred, and 95%-99% or more homogeneity most preferred, particularly for pharmaceutical uses. In general, the antagonists may be administered intradermally ("i.d."), intracranially ("i.c."), intraperitoneally ("i.p."), intrathecally ("i.t."), intravenously ("i.v."), subcutaneously ("s.c."), intramuscularly ("i.m.") or directly into a tumor. Typically, the antagonists are present as free bases or as acid salts. Suitable salts should be pharmaceutically acceptable. Representative examples include metal salts, alkali and alkaline earth metal salts such as potassium or sodium salts. Other pharmaceutically acceptable salts include citric, succinic, lactic, hydrochloric and hydrobromic acids. Parenteral compositions may be formulated in aqueous isotonic solutions of between pH 5.6 and 7.4. Suitable isotonic solutions may include sodium chloride, dextrose, boric acid sodium tartrate, and propylene glycol solutions.

LABELS

The nucleic acid molecules, antibodies, CRF₂ receptors, and CRF₂ receptor agonists and antagonists of the present invention may be labeled or conjugated (either through covalent or non-covalent means) to a variety of labels or other molecules, including for example, fluorescent markers, enzyme markers, toxic molecules, molecules which are nontoxic but which become toxic upon exposure to a second compound, and radionuclides.

Representative examples of fluorescent labels suitable for use within the present invention include, for example, Fluorescein Isothiocyanate (FITC), Rodamine, Texas Red, Luciferase and Phycoerythrin (PE). Particularly preferred for use in flow cytometry is FITC which may be conjugated to purified antibody according to the method of Keltkamp in "Conjugation of Fluorescein Isothiocyanate to Antibodies. I. Experiments on the Conditions of Conjugation," *Immunology* 18:865-873, 1970. (See also Keltkamp, "Conjugation of Fluorescein Isothiocyanate to Antibodies. II. A Reproducible Method," *Immunology* 18:875-881, 1970; and Goding, "Conjugation of Antibodies with Fluorochromes: Modification to the Standard Methods," *J. Immunol. Methods* 13:215-226, 1970.) For histochemical staining, HRP, which is preferred, may be conjugated to the purified antibody according to the method of Nakane and Kawaoi ("Peroxidase-Labeled Antibody: A New Method of Conjugation," *J. Histochem. Cytochem.* 22:1084-1091, 1974; see also, Tijssen and Kurstak, "Highly Efficient and Simple Methods for Preparation of Peroxidase and Active Peroxidase Antibody Conjugates for Enzyme Immunoassays," *Anal. Biochem.* 136:451-457, 1984).

Representative examples of enzyme markers or labels include alkaline phosphatase, horse radish peroxidase, and β -galactosidase. Representative examples of toxic molecules include ricin, abrin, diphtheria toxin, cholera toxin,

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DIVISIÓN DE NUEVAS CREACIONES

2020
Bogotá, D.C.,

Abogada
Jimena Escobar Uribe

SUPERINTENDENCIA DE INDUSTRIA Y COMERCIO

No. 03-077204- -00006-0000
Fecha: 2008-04-17 13:23:58 Dep. 2020 DIR.NUEVASCR
Tra. 11 PATENTEIYII Eve: 379 FASENACIONALII
Act. 519 TRASLADOINFORME Folios: 1

ASUNTO Radicación 03-77204
 Trámite 002
 Evento 001
 Actuación 430
 Oficio - 5244
 Folios

Patente de Invención ☒ Patente de Modelo de Utilidad ☐
Vía Nacional ☐
Vía PCT (fase nacional) ☒ Cap.I ☐ Cap. II ☒
No. Sol. Internacional PCT/US02/07476
Fecha de Presentación Internal. 06/03/2002

Como resultado del examen de fondo, realizado con fundamento en el artículo 45 de la Decisión 486 de la Comisión de la Comunidad Andina, se le comunica:

1. Documentos estudiados

- 1.1. Descripción: Folios: 16 a 75 como se radicó inicialmente.
- 1.2. Reivindicaciones: Folios: 77 a 81 como se radicó inicialmente.
- 1.3. Figuras: Folios: 82 a 98 como se radicó inicialmente.
- 1.4. Prioridad: 06/03/2001, Estados Unidos, US09/799978.

2. Objeto de la invención

Título de la solicitud: “Métodos para la identificación de compuestos para regular la masa o función muscular utilizando los receptores del factor liberador de corticotropina”.

El objeto de la presente solicitud de patente de invención consiste en métodos de tamizado para identificar compuestos que se unen, activan o prolongan o aumentan la activación de los receptores del factor 2 de liberador de corticotropina (CRF2R) o sus rutas de transducción y para regular o potencialmente regular la masa o función musculoesquelética *in vivo*, composiciones que contienen agonistas del CRF2R, anticuerpos anti-CRF2R y métodos de tratamiento de patologías relacionadas con el mismo.

Reivindicación 1-11 : Un método para identificar compuestos candidatos para regular la masa o función musculoesquelética; el método comprende:

- a) poner en contacto un compuesto de prueba con un CRF2R,
- b) determinar si el compuesto de prueba se une o activa al CRF2R, y
- c) identificar aquellos compuestos de prueba que se unen al CRF2R como compuestos candidato para regular la masa o función musculoesquelética.

Reivindicaciones 13-16: Un método para identificar compuestos candidatos para regular la masa o función musculoesquelética, que comprende, en cualquier orden:

- a) poner en contacto un compuesto de prueba con una célula que expresa un CRF2R funcional y determinar el nivel de activación del CRF2R,
- b) poner en contacto un compuesto de prueba con una célula que expresa un CRF1R funcional y determinar el nivel de activación del CRF2R y determinar si el compuesto de prueba activa al CRF1R,
- c) comparar el nivel de activación del CRF2R con el del CRF1R,
- d) identificar a aquellos compuestos similar actividad hacia el CRF2R y el CFR1R o que muestren selectividad por CFR2R como compuestos candidato para regular la masa o función musculoesquelética.

Reivindicación 17: Un método para identificar compuestos terapéuticos candidatos para regular la masa o función musculoesquelética, que comprende:

- a) administrar el compuesto a un animal no humano, y,
- b) determinar si el compuesto candidato regula la masa o función musculoesquelética en el animal tratado.

Reivindicación 18: Un método para identificar compuestos candidatos que prolongan la activación de CFR2R o de una ruta de transducción del mismo, que comprende, en cualquier orden o en forma concurrente:

- a) poner en contacto un compuesto de prueba con una célula que expresa un CRF2R funcional,
- b) tratar la célula con agonista del CRF2R durante un tiempo y a una concentración suficiente para provocar la desensibilización del CFR2R en células control,
- c) determinar el nivel de activación del CRF2R, y
- d) identificar a aquellos compuestos de prueba que prolongan o aumentan la activación de CRF2R y el CFR1R como compuestos candidato para regular la masa o función musculoesquelética.

Reivindicación 19: Un método para identificar compuestos terapéuticos candidatos para regular la masa o función musculoesquelética de un grupo de compuestos del que previamente se ha identificado que aumenta o prolonga la activación del CRF2R, que comprende:

- a) administrar el compuesto junto con un agonista a un animal, y,
- b) determinar si el compuesto candidato regula la masa o función musculoesquelética en el animal tratado.

Reivindicación 20: Un método para identificar compuestos candidatos para aumentar la expresión de CFR2R o del CFR, que comprende:

- a) poner en contacto un compuesto de prueba con una célula o lisado celular que contiene un gen reportero asociado en forma operativa con un elemento regulador del CFR2R o CFR,
- b) detectar la expresión del gen reportero,
- c) determinar el nivel de activación del CRF2R, y
- d) identificar a aquellos compuestos que aumentan la expresión de gen reportero como compuestos candidato para regular la masa o función musculoesquelética.

Reivindicación 21: Un método para aumentar la masa o función musculoesquelética de un sujeto, que comprende:
a) identificar el sujeto,
b) administrar al sujeto una cantidad segura y efectiva de un agonista del CRF2R.

Reivindicación 22-29: Un método para tratar la atrofia muscular, que comprende:
a) identificar el sujeto,
b) administrar al sujeto una cantidad segura y efectiva de un agonista del CRF2R, un vector de expresión que codifica para CRF2R funcional o constitutivamente activo o un CFR o un compuesto que aumenta la expresión de CRF2R o CRF.

Reivindicaciones 30-32: Un anticuerpo purificado específico para CRF2R que es quimérico o humano.

Reivindicaciones 33-35: Una composición farmacéutica que comprende:
a) una cantidad segura y eficaz de un agonista de CRF2R, y
b) un portador farmacéuticamente aceptable;

El problema planteado en esta solicitud es la necesidad de identificar agentes terapéuticos que desaceleren los daños en la fibra muscular y retrasen el inicio de la incapacidad en pacientes con distrofias musculares, al tiempo que reduzcan la atrofia producida por la terapias actuales y consecuentemente la necesidad subsecuente de métodos eficientes para identificación y tamizado de compuestos a ser utilizados en el tratamiento de la atrofia musculoesquelética.

Para solucionar este problema técnico la solicitud en estudio proporciona un método de tamizado para identificar compuestos candidato que activan tanto al CRF1R como al CRF2R.

El objeto de la solicitud definida anteriormente se clasificó en la IPC G01N 33/53, A61K 39/00, A61K 39/395.

3. Excepción a la patentabilidad (artículo 20 D486 literal a)-d)) (según causal)

Teniendo en cuenta que según la norma comunitaria los métodos terapéuticos para el tratamiento de humanos o animales se encuentran excluidos de patentabilidad y que las reivindicaciones 13-29, tal como se encuentran reclamadas, corresponden con métodos que incluyen el tratamiento de un animal o un humano, la materia de las citadas cláusulas no podría ser patentada.

De esta manera y de acuerdo a lo establecido en el artículo 20 de la Decisión 486 el contenido de las citadas reivindicaciones deberá excluirse.

4. Determinación del Estado de la Técnica

La fecha que se ha tenido en cuenta para determinar el Estado de la Técnica es:

Fecha de presentación de la solicitud prioritaria – 06/03/2001

Teniendo en cuenta la fecha indicada se realizó la búsqueda en el estado de la técnica de documentos relacionados con la solicitud encontrándose los siguientes documentos más relevantes:

Nº	Documento	Título	Fecha de
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			publicación
D1	US5786203	"Isolated nucleic acid encoding corticotropin-releasing factor2 receptors"	28/07/1998

5. Análisis de patentabilidad (según requisito) (artículo 14 D486)

5.1. Novedad (artículo 16 D486)

La solicitud en estudio reclama:

- Un método para identificar compuestos candidatos para regular la masa o función musculoesquelética; el método comprende: a) poner en contacto un compuesto de prueba con un CRF2R, b) determinar si el compuesto de prueba se une o activa al CRF2R, y c) identificar aquellos compuestos de prueba que se unen al CRF2R como compuestos candidato para regular la masa o función musculoesquelética (cláusulas independientes 1 y 2);
- Un método para identificar compuestos candidatos para regular la masa o función musculoesquelética, que comprende, en cualquier orden: a) poner en contacto un compuesto de prueba con una célula que expresa un CRF2R funcional y determinar el nivel de activación del CRF2R, b) poner en contacto un compuesto de prueba con una célula que expresa un CRF1R funcional y determinar el nivel de activación del CRF2R, c) determinar si el compuesto de prueba activa al CRF1R, d) comparar el nivel de activación del CRF2R con el del CRF1R, y e) identificar a aquellos compuestos similar actividad hacia el CRF2R y el CFR1R o que muestren selectividad por CFR2R como compuestos candidato para regular la masa o función musculoesquelética (cláusula independiente 13);
- Un anticuerpo purificado específico para CRF2R que es quimérico o humano (cláusulas 30-32).
- Una composición farmacéutica que comprende: a) una cantidad segura y eficaz de un agonista de CRF2R, y b) un portador portador farmacéuticamente aceptable; (cláusulas 33-35).

Al comparar la materia reclamada por la solicitud en estudio con el estado de la técnica es evidente que las reclamaciones se encuentran anticipadas por el documento US5786203 (D1), que enseña métodos para detectar compuestos que se unen al receptor CFR2R que comprenden las mismas etapas de los métodos de tamizaje reclamados por la solicitud en estudio (ver *Columna 3: Líneas 8-67*). D1 también exhibe la identificación de actividad de dichos compuestos por medio de moléculas reporteros (ver *Columna 3: Líneas 24-26*) y según los mismos mecanismos y rutas celulares (ver *Columna 3: Líneas 27-30*) y muestra secuencias que corresponden con las presentadas por la solicitud en estudio (p.e. *SEQ No. 8 de D1*).

Igualmente D1 divulga anticuerpos específicos para CRF2R purificados monoclonales o policlonales murinos o humanos (ver *Columna 2: Líneas 54 a 65; Columnas 15-19*) y composiciones farmacéuticas que contienen un agonista de CRF2R junto con portadores farmacéuticamente aceptables (ver *Columna 22: Líneas 10-20*).

En consecuencia ya existían en el estado de la técnica métodos de tamizaje de compuestos para activar o prolongar la actividad del CRF2R o unirse al mismo, anticuerpos específicos contra el este receptor y composiciones farmacéuticas que contienen los compuestos seleccionados, agonistas o antagonistas del CFR2R.

De acuerdo con lo anterior, los métodos, anticuerpos y composiciones reclamados no pueden ser considerados como nuevos y por lo tanto no cumplen con el requisito de novedad establecido en el artículo 16 de la Decisión 486.

6. Conclusión:

Los requisitos de patentabilidad de la presente solicitud se encuentran afectados así:

N°	Documento N°	Reivindicaciones afectadas	Requisito que afecta
D1	US5786203	1-34	Novedad

En cumplimiento del artículo 45 de la Decisión 486 de la Comisión de la Comunidad Andina, se le indica que debe dar respuesta dentro del término de 60 días contados a partir de la fecha de notificación de la presente comunicación. En caso de no dar respuesta al presente requerimiento, o si a pesar de la respuesta subsistieran los impedimentos para la concesión, se denegará la patente.

Notifíquese el presente oficio conforme a lo dispuesto en el numeral 5.2, literal e), del capítulo quinto del título primero de la Circular Única N° 10 de 2001.

ALIX CARMENZA CÉSPEDES DE VERGEL
Jefe de División de Nuevas Creaciones

El anterior requerimiento fue notificado por fijación en lista, de fecha 16 ABR 2008

CONCEPTO TÉCNICO No. 904	EXAMINADOR TÉCNICO Código No.202081
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AVISO DE NOTIFICACION
CORREO CERTIFICADO



Radicación : 3-77204- -8-0 Fecha: 2008-09-01 08:34:50
Trámite : 11 PATENTEIYII
Evento : 379 FASENACIONALII
Actuación : 432 COMUNICACTOADM
Destinatario : JIMENA ESCOBAR URIBE Folios 1

Señor (a) (es)
JIMENA ESCOBAR URIBE
CRA 11 NO 94-76 (OF. 305)
BOGOTA D.C.

Referencia RESOLUCION : 32929
Fecha : 29/08/2008
Expediente : 3-77204
Trámite : 11 PCT-PATENTE DE INVENCION CAPITULOS I Y II
Evento : 379 FASE NACIONAL INCLUIDO CAPITULO II
Actuación : 432 COMUNICACION ACTO ADMINISTRATIVO

Comendidamente le solicito presentarse a este Despacho ubicado en la Carrera 13 No. 27 - 00 Piso Quinto (5), de la ciudad de Bogotá D.C., dentro del término de cinco (5) días hábiles contados a partir del envío de la presente citación, en horario de 8:00 a.m. a 4:30 p.m. jornada continua, con el fin de notificarle personalmente el contenido de la resolución de la referencia proferida dentro del expediente tramitado con el número de radicación citado en el asunto.

De no hacerse presente en el término indicado anteriormente, la providencia en mención se notificará por edicto, el cual se fijará por el término de diez (10) días en el Centro de Documentación e Información.

Para su comodidad el texto completo de la resolución se encontrará disponible en la página web de la Entidad desde el día hábil siguiente al de su notificación, la cual podrá ser consultada desde un computador con acceso a Internet, en la página www.sic.gov.co, con el número de resolución a través de la opción servicios en línea - caracter general - consulta actos administrativos.

Secretaría General
SUPERINTENDENCIA DE INDUSTRIA Y COMERCIO

Al contestar favor indique el número de radicación consignado en el sticker

Sede Centro: Carrera 13 No. 27-00 Pisos 2, 5, 7 y 10
Sede CAN: Cra 50 No. 26-55 Int 2 PBX: 3820840
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REPUBLICA DE COLOMBIA
SUPERINTENDENCIA DE INDUSTRIA Y COMERCIO

RESOLUCION Nº 3 2 9 2 9

Por la cual se niega una patente de invención

Rad. PCT/03-77204

EL SUPERINTENDENTE DE INDUSTRIA Y COMERCIO
en ejercicio de sus facultades legales, en especial de las que se confirieron en el artículo
4, numeral 5 del decreto 2153 de 1992, y

CONSIDERANDO:

PRIMERO: Que mediante escrito radicado en esta Superintendencia el 04 de septiembre del 2003 bajo el número 03077204, se presentó la solicitud de patente de invención titulada "METODOS PARA LA IDENTIFICACION DE COMPUESTOS PARA REGULAR LA MASA O FUNCION MUSCULAR UTILIZANDO LOS RECEPTORES DEL FACTOR LIBERADOR DE CORTICOTROPINA", para los efectos de que se dé comienzo a la fase nacional, en virtud del Tratado de Cooperación en Materia de Patentes (PCT).

SEGUNDO: Que efectuado el examen de forma en cuanto a los requisitos que debe llenar la solicitud y cumplidas las exigencias legales, se ordenó publicar el extracto sin que se hubieran presentado oposiciones por parte de terceros.

TERCERO: Que el artículo 45 de la Decisión 486 de la Comisión de la Comunidad Andina establece que: *"Si la oficina nacional competente encontrara que la invención no es patentable o que no cumple con alguno de los requisitos establecidos en esta Decisión para la concesión de la patente, lo notificará al solicitante. Este deberá responder a la notificación dentro del plazo de sesenta días contados a partir de la fecha de la notificación. Este plazo podrá ser prorrogado por una sola vez por un período de treinta días adicionales.(...) Si el solicitante no respondiera a la notificación dentro del plazo señalado, o si a pesar de la respuesta subsistieran los impedimentos para la concesión, la oficina nacional competente denegará la patente"*.

CUARTO: Que realizado el examen de fondo, se requirió al solicitante en los términos del artículo 45 de la Decisión 486 de la Comisión de la Comunidad Andina, para que presentara respuesta a las observaciones de carácter técnico visibles a folios 286 a 295 del cuaderno administrativo, relacionadas con la patentabilidad o cumplimiento de los requisitos establecidos por esta Decisión para la concesión de la patente. Frente a dicho requerimiento no hubo pronunciamiento alguno por parte del mismo, por lo cual es procedente negar el privilegio de patente solicitado con fundamento en lo dispuesto en el artículo 45 de la Decisión 486.

RESUELVE:

ARTICULO PRIMERO: Negar el privilegio de patente de invención para la solicitud

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Industria y Comercio
SUPERINTENDENCIA

REPUBLICA DE COLOMBIA
SUPERINTENDENCIA DE INDUSTRIA Y COMERCIO

RESOLUCION **Nº 3 2 9 2 9**

Por la cual se niega una patente de invención

Rad. PCT/03-77204

correspondiente a "METODOS PARA LA IDENTIFICACION DE COMPUESTOS PARA REGULAR LA MASA O FUNCION MUSCULAR UTILIZANDO LOS RECEPTORES DEL FACTOR LIBERADOR DE CORTICOTROPINA", que entró en fase nacional en virtud del Tratado de Cooperación en Materia de Patentes (PCT).

ARTICULO SEGUNDO: Notificar personalmente el contenido de la presente resolución a la doctora Jimena Escobar Uribe, o a quien haga sus veces, en su calidad de apoderada de The Procter & Gamble Company., entregándole copia de la misma, advirtiéndole que contra ella procede el recurso de reposición interpuesto ante el Superintendente de Industria y Comercio, al momento de la notificación o dentro de los 5 días hábiles siguientes a ella.

NOTIFÍQUESE Y CUMPLASE

Dada en Bogotá, D.C., a los **2 9** AGO. 2008

EL SUPERINTENDENTE DE INDUSTRIA Y COMERCIO,

GUSTAVO VALBUENA QUIÑONES

Doctora

JIMENA ESCOBAR URIBE

Carrera 11 A N° 94 – 76, Of. 305.

Bogotá D.C.

202021

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