Isolation and structural characterization of the rat corticotropin releasing factor receptor (CRFR) gene was performed to determine the exon/intron organization of the coding region and the potential for splice variants. The CRFR gene contains 13 exons and 12 introns, and the positions of the exon/intron junctions are similar to those of other Class II G protein-coupled receptor genes including the parathyroid hormone and glucagon receptors. The promoter resides within 593 base pairs of the initiation codon and the major transcriptional start site at nucleotide –238. This domain does not possess a TATA box but contains multiple Sp1 and AP-2 sites upstream and downstream of the major transcriptional start site. Intron junctions were identified in the extracellular, transmembrane (TM), and cytoplasmic (C) domains of the CRFR, giving the potential for differential signal transduction by splice variants. CRFR cDNAs derived from rat Leydig cell mRNA included the pituitary Form A, which spans exons 1–13, and two splice variants with deletion of exon 3 or exons 7, 11, and 12. An evolutionary link between the intronless TM/C module of the glycoprotein hormone receptors and the intron-containing TM/C module of the CRFR is suggested by the common position of the luteinizing hormone receptor Form D alternate acceptor splice site and the CRFR intron 12.

Corticotropin releasing factor (CRF)1 is a principal neuroregulator within the hypothalamo-pituitary-adrenal axis that coordinates endocrine, behavioral, and autonomic responses to stress. CRF is also an important anti-reproductive hormone that inhibits luteinizing hormone/human chorionic gonadotropin (cAMP)-stimulated androgen production in the rat testis through inhibition of the catalytic subunit of adenylyl cyclase (1). High affinity CRF receptors have been characterized in several target tissues including the pituitary (2) and testicular Leydig cells (3). Recent molecular cloning of the pituitary CRF receptor cDNA from human (4), rat (5), and mouse (6) indicate that the expressed protein is a member of the G protein-coupled seven-transmembrane receptor (GPCR) family and has been shown to couple to both adenylate cyclase and phospholipase C (7), stimulating cAMP production and phosphoinositide hydrolysis. The CRF receptor exhibits significant homology to the parathyroid (8) and glucagon (9) receptor family that have been designated as members of Class II (10, 11) in the GPCR superfamily. This class exhibits little primary sequence similarity to the Class I GPCR, and members contain specific conserved amino acid motifs in the extracellular and transmembrane domains (11). In addition, genomic structures of the rat parathyroid (12) and glucagon (13) receptors suggest that some members of the Class II family differ from the catecholamine and the glycoprotein hormone receptor families by the presence of introns in both the NH2-terminal extracellular and the transmembrane/cytoplasmic domains.

Several variant CRF receptor clones have been identified that carry insertions and deletions of both the extracellular and seven-transmembrane module (7, 14). This suggests the presence of multiple introns in the CRF receptor gene and the expression of splice variants that potentially may differ in ligand specificity and modes of signal transduction. This was particularly relevant to our studies in the rat Leydig cell where the CRF receptor did not stimulate cAMP production or appear to be coupled to Gs (1, 3) and suggested either a tissue-specific post-translational modification of the CRFR, different G proteins or inhibitors, or the presence of a splice variant of the CRF receptor in the Leydig cell. The structural organization of the CRF receptor gene was determined for subsequent elucidation of isoforms in order to clarify the nature of the Leydig cell receptor.

MATERIALS AND METHODS

Isolation of the Rat CRF Receptor Gene—A rat liver lambda dash genomic library (Stratagene, La Jolla, CA) was screened with 32P-labeled DNA containing the following segments of the rat CRFR (5) DNA sequence: (–14/+361 bp), (215/1318 bp), (–78/+60 bp), (–78/–49 bp), and (–55/–26 bp). Positive clones were subjected to restriction enzyme mapping, Southern analysis, and DNA sequencing as described previously (15).

Mapping of 5′-flanking Region of Rat CRF Receptor Gene—A 741-bp Alul digestion fragment of done I (see Fig. 1) was used to analyze the 5′-flanking sequence of the CRF receptor. This was subcloned into a Smal site of pGEM4z. 135- and 606-bp PstI restriction fragments were then subcloned into pBluescript (Stratagene, La Jolla, CA). Isolated clones were sequenced by the dideoxynucleotide chain termination method (U. S. Biochemical Corp.).

Primer Extension and S1 Nuclease Mapping—Rat pituitary polyadenylated RNA (10 μg) was used for primer extension and S1 nuclease mapping studies to determine transcriptional start sites (16). Two synthetic oligonucleotide DNA primers that include the initiation codon, coding region, and 5′-flanking domain (–23/+6), (+33/+59), and a genomic DNA fragment containing the 5′-flanking region (–55/+6), isolated by
The CRF Receptor Gene

### Results and Discussion

**Isolation of the Rat Leydig Cell CRF cDNA Clones by Reverse Transcriptase PCR:**

PCR, were end labeled with [γ-32P]ATP and used for primer extension and S1 nuclease mapping, respectively (16).

Promoter Activity of the CRF Receptor Gene—Two luciferase reporter gene constructs were synthesized for analysis of the promoter activity. A 610-bp PstI restriction fragment of the 5′-flanking region or a 668-bp PvuII restriction fragment of the 5′-flanking region was subcloned into LucLink v2.2 (a gift from Dr. R. Maurer, Oregon Health Sciences University) containing the luciferase reporter gene (17). Transient co-transfection of each of these constructs and the β-galactosidase reporter gene, with a CMV promoter gene construct (Promega) into mouse Leydig tumor cells and Chinese hamster ovary cells was performed as described previously (17). Luciferase activity was measured and normalized with β-galactosidase activity.

Isolation of Rat Leydig Cell CRF cDNA Clones by Reverse Transcriptase PCR—Poly(A) RNA was isolated from purified rat Leydig cell by centrifugal elutriation (Invitrogen, San Diego, CA). Synthetic oligonucleotide primers tailed with XbaI and HindIII restriction enzyme cleavage sites were designed to conform to the DNA sequence of the rat pituitary CRF receptor cDNA. Primer 1, (−27 to −7 bp); Primer 2, (60 to 82 bp); Primer 3, (159 to 215 bp); Primer 4, (340 to 360 bp); and Primer 5, (1298 to 1312 bp). The poly(A) RNA was reverse transcribed using Primer 1 or 2 (Life Technologies Inc.). Primer pairs (1–5, 2–4, and 3–5) were used as templates for two additional rounds of amplification. The products of this reaction were size selected and used as templates for two additional rounds of amplification. These clones were digested with XbaI and HindIII and ligated into pGEM M4z (Promega). Clones were sequenced by the dideoxynucleotide chain termination method. Primer pairs (1–5) yielded the Form A pituitary receptor. Primer pairs (2–4) yielded the Form B receptor with deletion of exon 3, and pair (3–5) yielded the Form C receptor with deletions of exons 7, 11, and 12. A recombinant Form A/C CRFR was constructed by ligating the 5′ 302-bp PvuII fragment of Form A with the 3′ 711-bp PvuII fragment of the Form C receptor and was subcloned into pGEM M4z. Sequence analysis was performed with the program Pile-Up in the Genetics Computer Group (Madison, WI).

| EXON | INTRON | DONOR | ACCEPTOR |
|------|--------|-------|----------|
| 1    |        | GCTGAG (333) | GTCAGA...CCACAG (34) |
| 2    |        | TTTCCTG (121) | GTGATT...TTCCATG (122) |
| 3    |        | CCAACA (124) | GTGAGG...TCTCCAG (124) |
| 4    |        | GAAGGC (327) | GTGAGG...CCCTCAG (328) |
| 5    |        | GCTGAC (343) | GTGAGA...GCCGCC (343) |
| 6    |        | ATATTG (555) | GTACTG...TGGCAG (556) |
| 7    |        | CTCGGA (709) | GTGACC...CCTCCAG (710) |
| 8    |        | TGAAAG (770) | GTGAGC...CCTCTG (771) |
| 9    |        | CTCCTG (843) | GTGAGA...ATGGCG (844) |
| 10   |        | TGACAG (929) | GTGACTG...ACCCTG (930) |
| 11   |        | TGACAG (1065) | GTGACC...CTTCTG (1066) |
| 12   |        | AGTGAA (1107) | GTGAGG...CCACAG (1108) |

The 722 bp of 5′ UTR of the gene is identical to the deduced amino acid sequence of the rat pituitary CRF receptor cDNA (accession number L24096) (5) (Fig. 3, r(A)).

The reported CRF receptor cDNA variants were found to correspond to the deletion of complete exons (Figs. 1 and 3). Exon 3 of the extracellular domain is deleted from a human variant 3 (accession number U16273) (14) (Fig. 3, h(2)). A second variant, the human CRF Type 2 receptor (accession number L23333) (7) contains an 87-nucleotide insertion after exon 5 of the rat CRF receptor gene (Figs. 1 and 3, h(2)). To determine whether this additional exon or a highly similar sequence was contained within the rat intron between exons 5 and 6, PCR and hybridization studies using the oligonucleotide sequence of the 87-bp insert and genomic clone 3 was performed. We were unable to locate this fragment in the noncoding intron 5 of the rat gene, indicating that this specific splice variant may not be expressed in the rat.

5′ End of CRF Receptor Gene—The 722 bp of 5′ noncoding sequence of the CRF gene contains multiple Sp1 and AP-2 sites but no typical TATA box (Fig. 4). Primer extension analysis reveals heterogenous start sites with extension from two different primers (−23 to +6bp) and (−33 to +59bp), where +1
is the A of the initiation codon ATG (Fig. 5, A and B). The nucleotide position $-238 \pm 2$ was identified as the major transcript start site with both primers. The $-233/-59$ primer produced an extension product at 244 bp that corresponds to position $-238$ (Fig. 5A, lane 3), and the primer $+33/+59$ gave an extension product at 297 that also corresponds to position $-238$ (Fig. 5A, lane 1). An additional start site was identified at position $-174$ only with the $(-33/-59)$ primer (Fig. 5A, lane 1, 233 bp). $S_1$ nuclease mapping using the PCR fragment (-59 to 255) as probe showed a 244-bp protected fragment, which demonstrates a start site at $-238$ bp, consistent with primer extension (Fig. 5B). The major initiation site at $-238$ bp is approximately 20 bp downstream of an AP-2 site (18) and 60 bp downstream of an Sp1 consensus element (Fig. 4).
expression of two CRFR/luciferase reporter gene constructs
with −651 or −593 bp (ATG initiation codon is +1) of the CRFR
5′-flanking region into the mouse Leydig tumor cells and Chi-
inese hamster ovary cells confirmed that a promoter domain is
located within 593 bp of the 5′-flanking domain. The addition of
the domain between −593 and −651 increased promoter activ-
ity in both cell types by 1.6-fold, indicating that either an
enhancer or a second promoter is located between −593 and
−651 bp (Fig. 5C).

Similarity of the CRF Receptor Gene to the Glucagon Recep-
tor Gene Family—The CRF receptor gene exhibits substantial
similarity to the glucagon/PTH (12, 13) receptor gene family
that is characterized by the presence of introns within its
transmembrane/cytoplasmic module, highly conserved cyste-
ine in its extracellular domain, and a highly conserved first
intracellular loop (12). The exon/intron junctions of the CRF,
PTH (12), and glucagon (13) receptor genes are remarkably
similar in alignment (Genetics Computer Group; gap weight =
3; gap length weight = 0.1) following the signal peptide in
exons 1 and 2 (Fig. 6). The CRF receptor exon/intron junctions
are aligned to that of the PTH and glucagon receptors after
exons 3, 5, 7, 8, 9, 10, and 12. CRFR is similar to PTHR in that
amino acids 457–509 are divided into exons 11 and 12, whereas
this domain in the glucagon receptor is a single exon (Fig. 6).
All members of this family induce cAMP generation and phos-
phoinositide hydrolysis (7, 19) and thus the similarity of the
TM domains and intracellular loops. With the exception of the
cysteines in extracellular loops 1 and 2, there is no visible
similarity within the extracellular loops among the three re-
ceptors, and these domains may contribute to ligand specificity.
Extracellular loop 3 has been shown to be of importance to PTH

**Fig. 4.** The 5′-flanking region of the rat CRFR gene. The over-
lined regions indicate consensuselements. tss, transcript start sites. N, unde

**Fig. 5.** Primer extension (A) and S1 nuclease mapping (B) using designated oligonucleotide primers and genomic probes. Bands
at 297 (A, lane 1) and 244 (A, lane 3; B, lane 1) represent −238 bp, and the band at 233 (A, lane 1) represents −174 bp. Yeast tRNA controls (A,
lanes 2 and 4; B, lane 2; C, summary of primer extension data and promoter activity relative light unit/unit β-galactosidase activity).
binding to the PTHR (20) and extracellular loop 1 as well as TM3, TM4, and TM6 for glucagon binding to the glucagon receptor (21). The potential for receptor splice variants that exhibit differential modes of signal transduction is indicated by the presence of introns in the TM/cytoplasmic module and has been shown to be physiologically relevant in a member of the glucagon family, the pituitary adenylate cyclase-activating polypeptide receptor (22). Variants containing combinations of two additional exon cassettes in the third intracellular loop have been identified and sequenced, each exhibiting different degrees of Gs and phosphoinositide coupling (23). These pituitary adenylate cyclase-activating polypeptide receptor exons are inserted at a position that aligns with the CRFR, PTHR, and glucagon receptor exon 10-11 junction (Fig. 6) within the third intracellular loop.

CRFR cDNA Isolated from the Rat Leydig Cell—We proceeded to isolate and sequence CRFR PCR products from rat Leydig cell mRNA to determine which CRFR forms are present in this cell type. The assumption is a derivation from the rat CRFR Form A gene described in this study, and several PCR primers were constructed using the sequence of the rat pituitary Form A receptor cDNA (Fig. 3, r(A)) (5). cDNA clones were amplified with select CRF receptor primers by reverse transcription-PCR and sequenced (Fig. 7). Although all Leydig cell CRFR mRNA were expressed at very low levels in comparison with the pituitary, the predominant form of the Leydig cell receptor mRNA was identified as the rat pituitary CRF receptor sequence (Figs. 3, r(A), and 7, FORM A).

Two splice variants (B and C) were also recovered from rat Leydig cell mRNA but at very low expression levels. The first variant Form B that was recovered from the Leydig cell with PCR primers from (160 to 182) in exon 2 to (340–360 bp) within exon 5 amplified a cDNA fragment that contains a deletion of exon 3 (Fig. 7). This deletion is in frame, resulting in the substitution of amino acids 41–71 for an aspartic acid (Fig. 6, crfr(B)). This variant may be of significance, because the exon 3 deletion was also observed in the human Form 3 (Figs. 1 and 3 h(3)) and does not change the reading frame or result in premature termination of the coding sequence. The positions of the initiation and termination codons in CRFR Form B have not been identified. PCR primers from (–27 to –7) did not amplify this clone, indicating that the signal sequence of this

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**Fig. 6. Exon/intron pattern of the rat CRF, PTH, and glucagon receptor family.** Amino acid sequences of rat CRFR (5) (accession number P35353), rat PTHR (8) (accession number P25961), and rat glucagon receptor (9) (accession number P30082). CRFR transmembrane domains are designated as in Ref. 4). Asterisk, intron; dot, gap; dash, identity to CRFR; //, 5’ and 3’ end of sequenced PCR product that corresponds to primer; //, end of cDNA; lowercase letters, novel sequences from exon deletion; +, position of amino acid similarity; O, no visible homology.

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**Fig. 7. CRFR forms identified in the rat Leydig cell by reverse transcription-PCR.** Form A, rat pituitary holoreceptor. Forms B and C, PCR products. Dot, missing exons 3, 7, 11, or 12. No additional 3’ or 5’ end of cDNA; lowercase letters, novel sequences from exon deletion; +, position of amino acid similarity; O, no visible homology.

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**Table 1.**
clone (exon 1) differs from that of the Form A CRFR (Fig. 6). However, Form B does contain the NH\textsubscript{2}-terminal of the mature receptor at leucine 25, because the PCR product begins at valine 21. PCR primers constructed from the CRFR Form A cDNA sequence at the putative second transmembrane domain in exon 6 at 552 or 451 bp or TM3 (731 bp) in exon 7 did not amplify the variant Form B, implying that this cDNA may contain a novel carboxyl-terminal sequence.

Because the exon 5-intron 5 donor splice site is apparently susceptible to alternative splicing (Fig. 3, h(2))(7), it is possible that this is the specific site of divergence of the Form B variant from Form A. With a putative continuation into the intron, a termination codon (TGA) is present at position 2 of the 5’-flanking intron 5 sequence (TGA) (Fig. 2, exon 5), leaving a single transmembrane domain with a single arginine (AGG) in the putative cytoplasmic domain.

A second rat Leydig cell variant CRFR Form C cDNA was amplified with a 5’ primer beginning in exon 3 at position 215 and continues to exon 13 with deletions of exon 7, 11, and 12 (Figs. 1, 3, 6, and 7). PCR with 5’ primers from exons 1 or 2 did not recognize or amplify this variant, implying that this cDNA may contain a novel carboxyl-terminal sequence.

Because the exon 5-intron 5 donor splice site is apparently susceptible to alternative splicing (Fig. 3, h(2))(7), it is possible that this is the specific site of divergence of the Form B variant from Form A. With a putative continuation into the intron, a termination codon (TGA) is present at position 2 of the 5’-flanking intron 5 sequence (TGA) (Fig. 2, exon 5), leaving a single transmembrane domain with a single arginine (AGG) in the putative cytoplasmic domain.

A second rat Leydig cell variant CRFR Form C cDNA was amplified with a 5’ primer beginning in exon 3 at position 215 and continues to exon 13 with deletions of exon 7, 11, and 12 (Figs. 1, 3, 6, and 7). PCR with 5’ primers from exons 1 or 2 did not recognize or amplify this variant, implying that this cDNA may contain a novel carboxyl-terminal sequence. The CRFR Form C PCR product begins with amino acid 73 in exon 3. Deletion of exon 7 results in a frameshift with premature termination after a unique 39-amino acid tail that leaves only two hydrophobic domains (Fig. 6). It remains to be proven whether this form of the CRFR is responsible for the unique properties of the Leydig cell CRFR (1, 3). The absence of initiating methionines in the PCR products Form B and C CRFR did not permit expression studies with these clones.

A comparison of splice variants CRFR Form B and Form C and the human variant 3 (14) suggests that exon 3 may be of particular importance to receptor function, because two of the PCR products exclude exon 3, and the third appears to begin with exon 3. Exon 3 contains three of the five extracellular cysteines that are also conserved in the PTH and glucagon receptors (Fig. 6). Functional studies that did not address changes in receptor number or expression levels of the CRFR variant 3 (Fig. 3, h(3))(14) indicate a significant decrease in CRF binding activity (14). However, specific mutations of the conserved cysteines in exon 3 of the PTH receptor indicate that these cysteines are essential for receptor processing and expression on the cell surface (20), and mutant PTHR binding activity was commensurate with lower expression levels. It is interesting to note that the sequence differences between the pituitary Form A and several of the reported human and rat variants (NH\textsubscript{2}-terminal signal and exon 3) appear to impact on surface expression.

The genomic structure of the mammalian GPCFR are diverse and vary from intronless seven-transmembrane/cytoplasmic (TM/C) modules without an NH\textsubscript{2}-terminal extracellular domain (24) (Fig. 8, row 21 to intron containing NH\textsubscript{2}-terminal protein binding modules conjugated to intronless TM/C domains (16, 25) (Fig. 8, row 2) to the intron containing EC/TM/C...
11, and 12 terminate in the 3'-end of the CRFR, immediately following TMVII (Figs. 8, row 2, and 6). The alternative splice sites in the LHR coding region substitute for the 3'-end of the intron within the spliceosome (15), serve as a cleavage site and possibly as a site of intron insertion in evolution (Fig. 8, row 3). Studies with the Tetrahymena Group I intron show intron insertion at RNA spliceosome (15), serve as a cleavage site and possibly as a site of intron insertion as intron 12 of the CRFR, immediately following TMVII (Figs. 8, row 2, and 6). The alternative splice sites in the LHR coding region substitute for the 3'-end of the intron within the spliceosome (15), serve as a cleavage site and possibly as a site of intron insertion in evolution (Fig. 8, row 3). Studies with the Tetrahymena Group I intron show intron insertion at RNA splice sites via RNA intermediates, and reverse transcription resulting in incorporation of the intron into the gene is proposed (27). It is interesting to note that CRFR exons 1, 4, 5, 10, 11, and 12 terminate in the 3' intron acceptor consensus element AG, (Fig. 2, column 1 versus column 3) indicating that these positions may have served as alternate acceptor (or intron insertion points) within an intronless coding region. The 3' nucleotides of CRFR exons 11 and 12 are not unique to the CRFR. These are conserved in the PTHR (12) and glucagon (13) receptor genes and are also present in the secretin receptor at this aligned position (28) (Fig. 8, bottom). The 3' element of CRFR exon 11 conforms to the LHR TTXCAG 3'-intron J (between 959 and 960 of the LHR) splice element and the alternative splice sites that produce LHR forms B and C (15). This position is not an intronic site in the glucagon receptor (13) but is in the PTHR (12) and CRFR (Figs. 6 and 8, rows 5 and 6). Thus, the glucagon receptor may be an intermediate form between the LHR and CRFR.

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The Genomic Structure of the Rat Corticotropin Releasing Factor Receptor: A MEMBER OF THE CLASS II G PROTEIN-COUPLED RECEPTORS
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