Interactions of the Borna Disease Virus P, N, and X Proteins and Their Functional Implications*

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Borna disease virus (BDV) causes persistent central nervous system infection and behavioral disturbances in warm-blooded animals. Protein interaction studies were pursued to gain insight into the functions of the putative nucleoprotein (N), phosphoprotein (P), atypical glycoprotein (gp18), and X protein (X) of BDV. Coimmunoprecipitation experiments indicated that N and P, and P and X, form complexes in infected cells. Two-hybrid analyses confirmed interactions between P and P, P and X, and P and N, but not between P and gp18, N and gp18, X and gp18, or X and N. Analysis of P truncation mutants identified three nonoverlapping regions important for oligomerization (amino acids (aa) 135–172), and binding to X (aa 33–115) or N (aa 197–201). Coexpression of X stimulated oligomerization of P but decreased N-P complex formation. Immunocytochemistry of transfected noninfected CHO cells demonstrated that the distribution of P is dependent upon the presence of P-X expressed alone was found predominantly in the cytoplasm whereas coexpression of X and P resulted in nuclear localization. Immunocytochemistry of infected cells revealed nuclear colocalization of P and X. Interactions of P, N, and X may have implications for regulation of BDV transcription/replication and ribonucleoprotein assembly.

Bornavirus disease (BDV) is a nonsegmented, negative-strand RNA virus that infects a broad range of warm-blooded animal species to cause disturbances of movement and behavior (1–5). The antigenome of BDV contains at least six major animal species to cause disturbances of movement and behavior (1–5). The antigenome of BDV contains at least six major...
ensure expression of intact protein (16). GAL4 constructs were cloned using primers containing BamHI (5') or Kpn1 sites (3') to facilitate cloning into those sites in plasmid pGAL4. VP16 constructs were cloned using primers containing EcoRI (5') or HindIII (3') sites to facilitate cloning into plasmid pVP16. Complete predicted coding sequence for the ORF was amplified from infected C6 cells by RT-PCR using the primers GAL4-'5'(BamHI) and GAL4-'3'(Kpn1) and cloned into the EcoR V site of pBluescript SKII+ (Stratagene). Thereafter, the X ORF was subcloned into BamHI and Kpn1 sites of pGAL to create pGAL4-X, and into the EcoRI and HindIII sites of pVP16 to create pVP16-X. The X, N, P, and gp18 ORFs were released from the mammalian two-hybrid vectors and subcloned into the yeast two-hybrid vectors pAD424 and pBT9. The plasmid pBlueScript-BDV-X was generated for in vitro translation of X. It contains the X ORF, a ribosomal binding site predicted to enhance X translation relative to wild type sequence, and a mutated BDV-P AUG designed to abrogate expression of the P ORF without disrupting the X ORF. To create pBlueScript-BDV-X, the X ORF was amplified by PCR from pBlueScript-TA-BDV with primers AUG-X(EcoRI) and pBT5(bamHI) and then cloned into pBlueScript SKII+. The ribosomal binding sequence of X (uugugAUGa) was adapted by this PCR step to a more favorable consensus sequence (cgcAUGG) (17). Furthermore, the AUG of the P, which is located within the X ORF, was mutated to ACG in two consecutive PCR reactions using AUG-P-anti and AUG-P-sense primers.

For transient expression of P, N, and X, the corresponding ORFs were subcloned from pBlueScript-BDV-X, pBlueScript-BDV-P, and pBlueScript-BDV-P into PTRE or pcDNA3.1 vectors.

**In Vitro Translation of BDV-X, -N, and -P**—The plasmids pBlueScript-BDV-X, pBlueScript-BDV-P, and pBlueScript-BDV-N were linearized with XbaI and used as template for in vitro transcription of RNAs containing the corresponding ORFs. In *in vitro* translation reactions followed standard protocols (Stratagene) and used 500 ng of RNA in a total volume of 50 μl with 10 μCi of [35S]methionine and [35S]cysteine (Express Labeling, NEN Life Science Products).

**Immunoprecipitation of in Vivo Labeled BDV Proteins**—Persistently infected and uninfected C6 cells (106) were incubated with 500 μCi of [35S]methionine and [35S]cysteine (Express Labeling, NEN Life Science Products) in Dulbecco’s modified Eagle’s medium containing 5% fetal calf serum overnight. Cells were washed twice with phosphate-buffered saline on ice, and then incubated with 600 μl of lysis buffer (50 mM Tris·HCl, pH 7.2, 1% Triton X-100, 1% deoxycholic acid (Sigma), 0.1% SDS, 150 mM NaCl) for 10 min on ice. After clearing by centrifugation, samples were split into three aliquots containing 300 μl of lysis buffer and 5 μl of rabbit anti-X serum (14), 5 μl of rabbit anti-P serum (15), or 1 μg of monoclonal N serum (15) and incubated at 4 °C overnight. After addition of Protein A-Sepharose (Sigma) for 1 h at 4 °C, beads were washed twice with 0.5 ml of lysis buffer and twice with 1 ml of 50 mM Tris·HCl containing 150 mM NaCl. The bound protein was collected by centrifugation and then released by boiling in Laemmli buffer (18). Proteins were size-fractionated by 15% SDS-PAGE for analysis by autoradiography.

**In Vitro Binding Assays**—Binding assays were performed using Ni-agarose-bound N and in *vitro* translated P and X as described by Waterman and colleagues (19).

**Mammalian Two-hybrid Assays**—COS-7 cells were transfected with reporter and test plasmids (1 μg) using Lipofectin (Life Technologies, Inc.) in Opti-MEM medium (Life Technologies, Inc.). For three protein interaction studies, the plasmid containing an BDV ORF not fused to pVP16 or GAL4 was transfected by the same method except that 5 μg of DNA was used. Forty-eight hours thereafter, cells were lysed in lysis buffer (25 mM Tris·HCl, pH 7.8, 2 mM dithiothreitol, 2 mM EDTA, 10% glycerol, 1% Triton X-100) for 15 min at room temperature. After centrifugation at 15,000 × g for 5 min, the supernatant (cell extract) was collected and the protein concentration was estimated by Bradford protein assay (Bio-Rad). Five hundred nanograms of total protein in each cell extract were assayed for luciferase activity.

**Yeast GAL4 Two-hybrid Assays**—Yeast two-hybrid studies were pursued using two different strains of *Saccharomyces cerevisiae* in accordance with manufacturer protocols (Matchmaker, CLONTECH). Strain CG1945 (MATa ura3–52, his3–200 ade2–101, lys2–801, trp1–901 leu2–3, 112rcan, gal4–542, gal80–538, URA3::GAL1-lacZ) was used in colony lift assays. Strain SFY526 (MATa ura3–52, his3–200 ade2–101, lys2–801, trp1–901 leu2–3, 112rcan, gal4–542, gal80–538, URA3::GAL1-lacZ) was used in β-galactosidase assays to quantify interaction efficiency.

**Paritification of Recombinant N Protein**—Recombinant BDV-N was expressed in *Escherichia coli* transformed with plasmid pET-N (15); the resulting histidine-tagged proteins were purified from the soluble sus-
result in induction of luciferase activity (data not shown). Protein interactions in the yeast two-hybrid system were similar to those observed in mammalian cells. In colony lift assays, there were strong interactions between P and X; weaker interactions between P and N; and no interactions between N and X. A series of amino- and carboxyl-terminal truncation mutants of P fused to VP16 were used to investigate the regions of P critical for interaction with N (Fig. 3A). Western immunoblot analysis was used as a control to ensure that levels of expression were similar with all P constructs (data not shown). Deletion of the first 32 or 93 amino acids (aa) from the amino terminus of P resulted in 5-fold or 3-fold higher binding to GAL4-P. Deletion of 32 aa from the amino terminus of P abrogated its binding to GAL4-P; however, further deletion of 67 aa from the amino terminus of P resulted in less than 50% reduction in binding activity to GAL4-N. In contrast, deletion of 4, 29, 67, or 86 aa from the carboxyl terminus of P abrogated binding to GAL4-N.

The same panel of P mutants (Fig. 3A) was also used to characterize regions of P important for interaction with itself and X. Deletion of 32, 93, or 134 aa from the amino terminus of P did not affect binding to GAL4-P. Deletion of 4 or 29 aa from the carboxyl terminus of P resulted in less than 50% reduction in binding activity to GAL4-P; however, further deletion of 67 or 86 aa from the carboxyl terminus of P abrogated its binding to GAL4-P. Deletion of 32 aa from the amino terminus of P resulted in 3-fold increase in binding to GAL4-X. Further deletion from the amino terminus of P abrogated its binding to GAL4-X. Deletion of 4, 29, 67, or 86 aa from the carboxyl terminus of P resulted in a progressive increase in binding to GAL4-X (from 19% to 834% of wild type values). The three nonoverlapping regions of P critical for oligomerization, and interaction with N or X in two-hybrid studies are illustrated in Fig. 3B.

### Table I

| VP16 | GAL4 | Relative light units |
|------|------|----------------------|
| P    | N    | 104 (±15)            |
| P    | P    | 117 (±14)            |
| P    | gp18 | 132 (±11)            |
| X    | X    | <1                   |
| X    | N    | <1                   |
| X    | gp18 | <1                   |

### Table II

| pAD424 | pGBD9 | Binding efficiency |
|--------|-------|--------------------|
| P      | N     | +                  |
| P      | X     | ++                 |
| P      | gp18  |                  |
| P      | gp18  |                  |
| X      | X     |                  |
| X      | N     |                  |
| X      | gp18  |                  |

**FIG. 2. P but not X binds to His-tagged N in vitro.** In vitro binding experiments were pursued using [35S]methionine labeled P and X (lane 1) and histidine-tagged N coupled to Ni-agarose beads. Agarose-bound proteins were subjected to 15% SDS-PAGE and autoradiography. Lane 1 provides index to relative amounts of P and X employed in these experiments. Lane 2, binding of P to N-Ni-agarose beads. Lane 3, absence of binding of X to N-Ni-agarose beads. Lane 4, absence of binding of P and X to Ni-agarose beads (negative control for specificity of binding to N).

**Binding of X Enhances Oligomerization of P**—To investigate simultaneous interactions between three BDV proteins, two-hybrid analyses were pursued in experiments where a third protein, not fused to either VP-16 or GaL-4, was also present (Table III). Interestingly, coexpression of X markedly enhanced oligomerization of P (4-fold), and had minor impact on VP16-P/GAL4-N interaction (Table III). Coexpression of P or N abrogated binding of VP16-P to GAL4-P, and binding of VP16-P to GAL4-N, respectively (Table III). Binding of VP16-P to GAL4-X was inhibited by coexpression of P, N or X (data not shown).

**P Facilitates Retention of X in the Nucleus**—To assess the effects of coexpression of individual BDV proteins on their subcellular distribution, TCN-repressible stable CHO cell lines expressing P (CHO-P) or N (CHO-N) were transiently transfected with plasmids encoding X. The distribution of relevant BDV proteins were then examined in the presence or absence of TCN. Whereas suppression of P expression (presence of TCN, Fig. 4A) resulted in cytoplasmic distribution of X (Fig. 4C), expression of P (Fig. 4B) resulted in nuclear distribution of X (Fig. 4D). The distribution of X did not vary with the presence or absence of N (data not shown).

The subcellular localization of P, N, and X in infected cells was determined in immunofluorescence experiments using a polyclonal serum to X and monoclonal antibody to P. The specificity of these sera were confirmed on CHO-cells expressing P, X, or N (data not shown). P, N, and X were found to colocalize in the nuclei of infected C6 cells (Fig. 5).
DISCUSSION

Recognition of the association of N and P (soluble antigen complex) dates from the earliest attempts to purify BDV proteins from infected cells and tissues (23–25). Although the presence of the soluble antigen continues to be a mainstay in BDV clinical diagnostics, the basis for complex formation and its significance in the virus life cycle remain poorly understood.

The experiments reported here define regions along P critical to the N-P interactions, and describe additional interactions between BDV N, P, and X that may provide insight into mechanisms for control of viral transcription/replication and RNP formation.

Co-immunoprecipitation experiments using extracts from infected cells and tissues (23–25). Although the presence of the soluble antigen continues to be a mainstay in BDV clinical diagnostics, the basis for complex formation and its significance in the virus life cycle remain poorly understood. The experiments reported here define regions along P critical to the N-P interactions, and describe additional interactions between BDV N, P, and X that may provide insight into mechanisms for control of viral transcription/replication and RNP formation.

Interactions of BDV Proteins

![Diagram](image)

**Table III**

Interactions between three BDV proteins (N, P, and X) were studied by using a mammalian two-hybrid system modified to include a third protein not fused to either VP16 or GAL4.

The luciferase activity found for interaction of GAL4-N and VP16-P, or GAL4-P and VP16-P, was defined as 100 (arbitrary) units. Values < 1 indicate background levels obtained with transfection of the luciferase plasmid alone. Values are the average of at least three independent experiments and standard deviation is indicated. Parentheses indicate standard deviations.

| GAL4 | VP16 | Third protein | Arbitrary light units |
|------|------|---------------|----------------------|
| P    | P    | –             | 100                  |
| P    | P    | P             | <1                   |
| P    | P    | N             | 100                  |
| P    | N    | P             | 100                  |
| P    | N    | N             | 100                  |

**Figure 3** Three distinct regions of P are critical for its interaction with N, P, and X. A, a series of amino- and carboxyl-terminal truncation mutants of P were fused to VP16 and used for two-hybrid studies in COS-7 cells to characterize the domains of P important for interaction with itself, N, and X. The luciferase activity found for interaction of full-length GAL4-N, GAL4-P, and GAL4-X fusion protein with BDV-P-VP16 was defined as 100 (arbitrary) units. Values < 1 indicate background levels obtained with transfection of the luciferase plasmid alone. Values are the average of at least three independent experiments. Standard deviations are indicated in parentheses. WT, wild type. B, schematic illustrating portions along P of three nonoverlapping regions important for P oligomerization (aa 135–172), binding to X (aa 33–115), and binding to N (aa 197–201).
identification of three nonoverlapping regions important for P oligomerization (aa 135–172), binding to X (aa 33–115), and binding to N (aa 197–201). The aa of P critical for binding to X were mapped to the amino-terminal portion of the protein between aa 33 and 115. Deletion of the first 32 aa of P resulted in increased binding of X, indicating that this region behaves as a negative regulatory domain. The binding of X and P was increased 7-fold in assays using a P mutant in which the oligomerization domain was deleted. This suggests that X binds preferentially to the monomeric form of P. Based on this finding, we anticipated that binding of X to P would inhibit P oligomerization. Intriguingly, the converse was observed; binding of X to P enhanced P oligomerization. In other nonsegmented negative strand RNA viruses, oligomerization of phosphoproteins correlates with viral transcriptional activity (26, 27). Colocalization of X and P in the nucleus of chronically infected cells indicates that X is associated with P at the sites of virus replication and transcription. Taken together, these data suggest the possibility that X may modulate transcriptional activity of BDV via binding to P.

We reported previously that multimerization of P in vitro is dependent upon disulfide bridging of cysteines at aa position 125 in P monomers (15). Although we cannot exclude the possibility that disulfide bridges play a role in P multimerization,

**FIG. 4.** Coexpression of X and P results in a nuclear localization of X. A TCN-repressible CHO cell line was stably transfected with a P-protein expressing construct and subsequently transiently cotransfected with a X-protein expressing plasmid. The distribution(s) of X and P were examined in presence or absence of TCN by indirect double-immunofluorescence using anti-X and anti P-antibodies. A, anti-P antibody; TCN (+), no P expression (control for suppression of P expression in the presence of TCN). B, anti-P antibody; TCN (−), nuclear localization of P (control for expression of P in the absence of TCN). C, anti-X antibody; TCN (+), cytoplasmic localization of X in the absence of P. D, anti-X antibody; TCN (−), redistribution of X to the nucleus in the presence of P.

**FIG. 5.** Colocalization of X, N and P in BDV-infected cells. Confocal, microscopic images of infected C6 cells stained for detection of X and N, or X and P. A, X (green fluorescence). C, N (red fluorescence). B, overlay of images in panels A and C, D, X (green fluorescence). F, P (red fluorescence). E, overlay of images in panels D and F. Primary antibodies employed were rabbit anti-X and mouse monoclonal antibodies to N and P.
in some cellular compartments in vivo, it is unlikely that this mechanism applies in the highly reducing environment of the cytoplasm (28, 29). In the two-hybrid system, the critical region for interaction of P with itself was not found at Cys-125 but was instead localized toward the carboxyl-terminal portion of the protein. Deletion of 4 or 26 aa from the carboxyl terminus resulted in a less than 50% decrease in binding. A complete abrogation of binding was observed in P mutants lacking 67 or 86 aa at the carboxyl terminus.

It is possible that phosphorylation is involved in BDV-P homomultimer formation as has been suggested for the phosphoprotein of vesicular stomatitis virus (27, 30). P is predominantly phosphorylated in vitro and in vivo at serine residues in the amino-terminal portion of the protein (31, 32). It is intriguing to speculate that phosphorylation of serine residues within the negative regulatory domain of P (first 32 aa of P) (32) may be responsible for modulating binding of P to X and N. Accordingly, deletion of this region may mimic nonphosphorylated P, which exhibits a higher binding efficiency to X and N. Whether lack of phosphorylation in the yeast two-hybrid system causes the 100-fold higher binding efficiency of P-X complexes compared with P-N complexes remains to be shown. The presence of the negative regulatory domain in the mammalian two-hybrid system had no substantive influence on the capacity of P to bind to itself. If the function of this domain is dependent upon phosphorylation, our model would predict that phosphorylation should not significantly influence oligomerization of BDV-P. Indeed, recent studies indicate that nonphosphorylated P expressed in E. coli is competent for oligomer formation (15).²

The last 4 carboxyl-terminal aa of P were found to be critical for its interaction with N (deletion eliminated binding to N). In this respect BDV is similar to respiratory syncytial virus, where the last 20 carboxyl-terminal aa of the phosphoprotein are essential for interaction with the nucleoprotein (33). No similar terminal region is defined in the phosphoprotein of vesicular stomatitis virus, although the last 5 carboxyl-terminal aa of the nucleoprotein are essential for binding to the phosphoprotein (22).

The nuclear localization of BDV replication and transcription requires provisions for bidirectional transport of viral proteins and RNAs across the nuclear membrane. Although it is formally possible that each of the BDV proteins has its own signals for bidirectional transport, an alternative system would allow proteins to traffic together in complexes. Recent work has shown the presence of a functional nuclear export sequence in X (34) and suggests the speculation that X may facilitate nuclear export of ribonucleoprotein complexes in a manner similar to the NS2 protein of influenza virus (35). Support for this notion is found in the communoprecipitation and colocalization experiments, which indicated an association between P and N, proteins predicted to be components of the RNP, and X. Efforts to find in vitro evidence for association of N and X were not successful. X did not bind to N in the two-hybrid systems or the in vitro binding assay using N-Ni-agarose alone or N-Ni-agarose in the presence of P. It is conceivable that higher

² M. Schwemmle and W. I. Lipkin, unpublished data.

affinities of P for X and N for P obscure detection of complexes comprising P, N, and X. Indeed, the affinity of the three protein complex may be lower in vitro because additional components (for example genomic RNA) are absent. This hypothesis has not been tested because methods are not established for purification of BDV RNPs. However, two-hybrid studies identified three distinct regions of P that could allow simultaneous interaction of P with itself, N, and X (Fig. 3B), suggesting a mechanism whereby P could bridge N and X to facilitate a complex of the three proteins.

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