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Bioinformatic analysis of the infectious bursal disease virus

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ABSTRACT

The analysis of amino acid sequences of the Infectious Bursal Disease virus (IBDV) proteins was done to identify their features. Antigenicity plot of the 1011 residue long IBDV sequence revealed 38 potential antigenic sites in viral protein VP2 of the virus. Prosite analysis of the amino acid sequence of IBDV revealed 11 Casein Kinase II Phosphorylation sites, 17 Protein Kinase C Phosphorylation sites, 4 N-Glycosylation sites, 18 N-Myristoylation sites and 2 Tyrosine Kinase Phosphorylation sites, respectively.

KEYWORDS

Infectious bursal disease;
IBD virus;
Bioinformatic analysis;
Host -pathogen interaction.

INTRODUCTION

Infectious Bursal Disease (IBD) is an acute contagious viral disease affecting young chickens up to six weeks of age causing high morbidity but low mortality. IBD Virus (IBDV) selectively affects the B Lymphocytes of chickens. It destroys B cells in the bursa of Fabricius causing significant depression of the humoral immune response. Although VP2 is known to be involved in host cell tropism, the site of predilection for IBDV on chicken B cells is not known yet. Bioinformatic study on the available protein sequence of IBDV was therefore undertaken to explore the molecular basis of host – pathogen interaction in Infectious Bursal Disease of chickens.

MATERIALS AND METHODS

Amino acid sequence of Infectious Bursal Disease virus proteins available in the protein sequence database on web was analyzed using computer programs available in the public domain. The functional sites of the sequence were predicted by PROSITE software. Potential antigenic epitopes of IBDV protein were predicted on the basis of hydrophilicity profile using the ANTIGEN program.

RESULTS AND DISCUSSION

The Infectious Bursal Disease Virus (IBDV) infects the immature B cells in bursa during their differentiation in young chickens. The exact target of IBDV attachment to B cells is not known. The analysis of amino acid sequence of the Infectious Bursal Disease virus VP2 protein was done to identify the features. Antigenicity plot of the 1011 residue long IBDV sequence revealed 38 potential antigenic sites of the virus (TABLE 1).

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The conserved heptapeptide of IBDV VP2 showed similarities to peptide amidase which interacts with chymotrypsin and to an uncharacterized antigen of *Leishmania major* and *Leishmania braziliensis* which infect macrophages. It seems to suggest that IBDV and Leishmania may possibly have similar mechanism of entry into the host cell.

Prosite analysis of the amino acid sequence of IBDV revealed 52 functional sites (11 Casein Kinase II Phosphorylation sites, 17 Protein Kinase C Phosphoryla-

TABLE 1 : Epitopes of IBDV predicted from the available protein sequence

S.no.	Start position	Sequence	End position
1	8	TQQIVPFIRSLLM	20
2	54	SGLIVFFPGFPGSIVGAHYT	73
3	82	FDQMLLTAQNLPASYNYCRLVSRSLTVRS	110
4	113	LPGGVYAL	120
5	124	INAVTFQGSLSELTD	138
6	155	IGNVLVGEGVTVLSLPTSYDLGYVRLGDPIPAIG	188
7	190	DPKMVATC	197
8	201	DRPRVYTI	208
9	212	DDYQFSSQYQAGGVTITLFSAN	233
10	236	AITSLSIGGELVFQTSVQGLILGATIYLIG	265
11	267	DGTAVITRAVAA	278
12	291	PFNIVIPT	298
13	301	ITQPITSIKLEIVT	314
14	329	ASGSLAVTI	337
15	342	YPGALRPVTLVAYER	356
16	358	ATGSVVTVAGVSNF	371
17	379	LAKNLVTE	386
18	425	YFMEVADLNSPLKIAG	440
19	446	DIIRALRRIAVPVVSTLFPPAAPLAH	471
20	473	IGEGVDYLLG	482
21	515	KGYEVVANLFQVPQNPVVDGILASPGILRG	544
22	546	HNLDCVLRE	554
23	556	ATLFPVVITT	565
24	578	KMFAVIE	584
25	604	SGHRVYGYAPDGVLPLET	621
26	623	RVYTVVPID	631
27	638	IMLSKDPIPPIVGS	651
28	653	GNLAIAYMDVFRPKVPIHVAM	673
29	692	KLATAHRLGLKLAG	705
30	733	RLPYLNLPYLP	743
31	748	RQYDLAM	754
32	764	ELESAVRA	771
33	774	AAANVDPLFQSALSVFM	790
34	866	GIYFATPEWVAL	877
35	904	YLDYVHAEK	912
36	917	SEGQILRAATSIYGA	931
37	936	EPPQAFIDEVAKVYEV	951
38	986	PKPNVPT	992

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TABLE 2 : Functional sites of IBD virus proteins

Functional site	Residues	Sequence	Functional site	Residues	Sequence
Casein kinase II	27 - 30	SipD	Protein kinase C	37 - 39	TlR
phosphorylation	48 - 51	TvgD	phosphorylation site	107 - 109	TvR
site	132 - 135	SlsE		200 - 202	SdR
	171 - 174	TsyD		307 - 309	SiK
	209 - 212	TaaD		314-316	TsK
	369 - 372	SnfE		403-405	SeR
	403-406	SerD		491-493	TaR
	564-567	TtvE		496-498	SgK
	565-568	TveD		503-505	SgR
	968-971	TamE		571-573	TpK
	1007-1010	SdeD		594-596	SqR
				621-623	TgR
N-myristoylation site	65-70	GSivGA		687-689	SfR
	69-74	GAhyTL		690-692	StK
	115-120	GGvyAL		828-830	SqR
	122-127	GTinAV		859-861	SkK
	143-148	GLmsAT		992-994	TqR
	224-229	GVtiTL			
	254-259	GLilGA		Tk phosphorylation site	
	281-286	GLtaGT		417-425	ReytDfreY
	318-323	GGqaGD		729-736	Rdw.DrlpY
	340-345	GNypGA		N-glycosylation site	
	360-365	GSvvTV		46-49	NLTV
	490-495	GTarAA	700-705 GLklAG	121-124	NGTI
	540-545	GIlrGA			
	650-655	GSsgNL	714-719 GSnwAT	396-399	NYTK
	675-680	GAlnAY	866-871 GIyfAT	685-688	NVSF

tion sites, 4 N-Glycosylation sites, 18 N-Myristoylation sites and 2 Tyrosine Kinase Phosphorylation sites, respectively) (TABLE 2).

Tissue culture adaptation of infectious bursal disease virus (IBDV) results in alternation of three residues on its major capsid protein VP2 and these residues may engage in receptor binding. In a study by Yip et al (2007)[1], recombinant VP2s of an attenuated strain (D78) and a very virulent strain (HK46) of IBDV tagged with rabbit immunoglobulin G heavy chain were expressed in mammalian cells, generating RAVP2 and RVVP2, respectively, in high purity. Using flow cytometry, both RAVP2 and RVVP2 were demonstrated to bind with Vero cells while these bindings were blocked by D78 viral particles. They suggested that both very virulent IBDVs (vvIBDVs) and attenuated IBDVs bind to Vero cells through the same receptor(s).

The bioinformatic analyses in the present study yielded useful information on the identity, nature and functional aspects of important proteins of IBDV. Thirty eight potential antigenic sites of VP2, and 52 functional sites of IBDV proteins were identified. The results of the present study offer valuable insight into the nature of the viral proteins involved in host – pathogen interaction and may form the basis for useful and confirmatory experimental studies in the future.

REFERENCE

[1] C.W.Yip, Y.S.Yeung, C.M.Ma, P.Y.Lam, C.C.Hon, F.Zeng, F.C.C.Leung; Virus Research, **123**, 50-56 **(2007)**.