

F033

Detection of Viral Diseases Causing Calf Diarrhea in Korea

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To evaluate the enteric viruses involved in calf diarrhea in Korea, RT-PCR with primer pairs specific to each groups A, B and C bovine rotaviruses (BRV), bovine coronavirus (BCoV), bovine viral diarrhea virus (BVDV), bovine torovirus (BToV), bovine sapovirus (BSaV), and bovine norovirus (BNoV), respectively, was performed with 153 diarrheic calf fecal samples from 92 farms. Group A BRV was predominantly detected (54/92 farms) in the diarrheic fecal samples. BCoV (25/92), BVDV (24/92), BSaV (22/92), BNoV (15/92), BToV (11/92), group C rotavirus (10/92) and group B rotavirus (6/95) in order were also detected in the diarrheic fecal samples. Of calf farms with diarrhea, 132 fecal samples from 84 farms were positive for at least one of each virus. Among calf farms positive for each enteric virus, 36 farms were infected with only one virus of them. From these results, calf diarrhea in Korea was caused mainly by enteric viruses and considerable numbers of it was concurrently infected, leading the difficulty of vaccine or medical treatments.

F034

Molecular Characterization of HE, M, and E Genes of Winter Dysentery Bovine Coronavirus Circulated in Korea During 2002-2003

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We analyzed the hemagglutinin/esterase (HE) protein, the transmembrane (M) protein and the small membrane (E) protein to characterize ten winter dysentery (WD) bovine coronavirus (BCoV) circulated in Korea during 2002-2003 and compared the nucleotide and deduced amino acid sequences with the other known BCoV. Phylogenetic analysis indicated that the HE gene among BCoV could be divided into three groups. The first group included only respiratory bovine coronavirus (RBCV), while the second group contained calf diarrhea BCoV, RBCV, WD and enteric bovine coronavirus (EBCV), respectively. The third group possessed only all Korean WD strains which were more homologous to each other and were sharply distinct from the other known BCoV, suggesting Korean WD strains had evolutionary distinct pathway. In contrast, the relative conservation of the M and E proteins of BCoV including Korean WD strains and the other coronaviruses suggested that structural constraints on these proteins are rigid, resulting in more limited evolution of these proteins.

F035

Sterile Phenotype in Sumoylation-defective mutants of *Aspergillus nidulans* was Recovered by Overexpression of the GATA factor NsdD.

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SUMO modifies protein function in various ways when attached to lysine residues of the target proteins. The enzymes involved in the sumoylation process are conserved and all components were identified in a filamentous fungus *Aspergillus nidulans*. The *sumO* and *ubcN* genes, encoding homologs of SUMO and Ubc9, respectively, were cloned. Deletion mutants of *sumO* and *ubcN* were not lethal. Mycelial growth was not much affected but conidiation was hardly occurred in both null mutants. $\Delta sumO$ and $\Delta ubcN$ exhibited high sensitivities to various mutagens compared to those for wild-type. Interestingly, cleistothecium, fruit body of *A. nidulans*, was never found in various growth conditions, while Hulle cells were produced in $\Delta sumO$ and $\Delta ubcN$. These results suggested that sumoylation process is required for proper differentiation as well as DNA repair of *A. nidulans*. The conidia production was also not enhanced even in asexual-driven conditions. Over-expression of NsdD, which is the GATA type transcription activator of sexual differentiation, in $\Delta sumO$ mutant forced to produce cleistothecia as many as in wild type, although the diameter of cleistothecia is smaller than wild type. [Supported by KOSEF]

F036

Isolation and Genetic Analysis of Rapamycin-sensitive mutants in *Aspergillus nidulans*.

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The Target of Rapamycin (TOR) signaling is an important conserved signaling pathway which is inhibited by rapamycin. To identify related genes in the TOR pathway of a model filamentous fungus *Aspergillus nidulans*, UV mutagenesis was performed and many mutants having *rapA-F* mutations, which showed rapamycin sensitive phenotype, were isolated. All mutants showed no additional sensitivity against various mutagens other than rapamycin, except *rapA* and *rapE* that showed cyclohexamide resistancy. Among them, *rapA* and *rapB* was chosen for further characterization. In *A. nidulans*, it has been known that the mutants having the *ActA1* allele, originally isolated as actidion (cycloheximide) resistant phenotype, showed rapamycin sensitive phenotype, suggesting that the *ActA1* or genetically linked unknown mutation, named *rapA1*, might be responsible for the rapamycin sensitivity. To identify these mutations, genomic library was introduced into *rapA* and *rapB* mutant and rapamycin resistant transformants were obtained. Sequencing analysis and further characterization of the mutants are in progress. [Supported by KRF]