Genetic Bottleneck Analyses of Kodi Adu Goat Breed Based on Microsatellite Markers

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The genetic characterization and bottleneck analysis in Kodi Adu goat was done using 25 FAO recommended microsatellite markers. The mean observed number of alleles and polymorphism information content (PIC) were estimated to be 11.52 ± 0.95 and 0.817 ± 0.023 respectively. The mean observed and expected heterozygosities were 0.660 ± 0.045 and 0.846 ± 0.018 respectively. The mean expected equilibrium gene diversity across 21 microsatellite loci under TAM, SMM and TPM were 0.793 ± 0.028, 0.854 ± 0.023 and 0.827 ± 0.026 respectively. All the three statistical tests revealed significant deviation of Kodi Adu goats from mutation-drift equilibrium under the IAM and TPM models, however, non-significant deviation under SMM model. The mode shift analysis supported the results under SMM indicating the absence of genetic bottleneck in the recent past in Kodi Adu goats.

Specific detection of peste des petits ruminants virus antibodies in sheep and goat sera by the luciferase

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Peste des petits ruminants (PPR) is a contagious and often fatal transboundary animal disease affecting mostly sheep, goats and wild small ruminants. This disease is endemic in most of Africa, the Middle, Near East, and large parts of Asia. The causal agent is peste des petits ruminants virus (PPRV), which belongs to the genus Morbillivirus in the family Paramyxoviridae. This genus also includes measles virus (MV), canine distemper virus (CDV) and rinderpest virus (RPV). All are closely related viruses with serological cross reactivity. In this study, we have developed a Luciferase Immunoprecipitation System (LIPS) for the rapid detection of antibodies against PPRV in serum samples and for specific differentiation from antibodies against RPV. PPR and rinderpest (RP) serum samples were assayed by PPR-LIPS and two commercially available PPR cELISA tests. The PPR-LIPS showed high sensitivity and specificity for the samples tested and showed no cross reactivity with RPV unlike the commercial PPR cELISA tests which did cross react with RPV. Based on the results shown in this study, PPR-LIPS is presented as a good candidate for the specific serosurveillance of PPR.

Sample preparation for avian and porcine influenza virus cDNA amplification simplified: Boiling vs. conventional RNA extraction

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RNA extraction and purification is a fundamental step that allows for highly sensitive amplification of specific RNA targets in PCR applications. However, commercial extraction kits that are broadly used because of their robustness and high yield of purified RNA are expensive and labor-intensive. In this study, boiling in distilled water or a commercial lysis buffer of different sample matrices containing avian or porcine influenza viruses was tested as an alternative. Real-time PCR (RTqPCR) for nucleoprotein gene fragment was used as read out. Results were compared with freshly extracted RNA by use of a commercial extraction kit. Different batches of virus containing materials, including diluted virus positive allantoic fluid or cell culture supernatant, and avian faecal, cloacal or oropharyngeal swab samples were used in this study. Simple boiling of samples without any additional purification steps can be used as an alternative RNA preparation method to detect influenza A virus nucleoprotein RNA in oropharyngeal swab samples, allantoic fluid or cell-culture supernatant. The boiling method is not applicable for sample matrices containing faecal material.

Detection and genome analysis of a lineage III peste des petits ruminants virus in Kenya in 2011

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In May 2011 in Turkana County, north-western Kenya, tissue samples were collected from goats suspected of having died of peste des petits ruminant (PPR) disease, an acute viral disease of small ruminants. The samples were processed and tested by reverse transcriptase PCR for the presence of PPR viral RNA. The positive samples were sequenced and identified as belonging to peste des petits ruminants virus (PPRV) lineage III. Full-genome analysis of one of the positive samples revealed that the virus causing disease in Kenya in 2011 was 95.7% identical to the full genome of a virus isolated in Uganda in 2012 and that a segment of the viral fusion gene was 100% identical