INTRODUCTION

Foot-and-mouth Disease Virus (FMDV) causes a highly contagious acute vesicular disease affecting a number of economically important animal species. Little is known about the interaction of the virus with cattle dendritic cells (DC). Development of a comprehensive protective T and B cell response requires antigen capture, migration, maturation and antigen presentation by DCs. The capacity to stimulate CD8 and CD4 T cells relies upon the presentation of antigen through MHC class I and II molecules respectively. There is stimulation of specific CD4 and CD8 responses to live and killed FMDV antigen, suggesting presentation occurs through the class I and II pathways. However, the response of CD4 and CD8 T cells isolated from infected cattle are consistently low compared to the response to control antigens, despite the absence of generalised immunosuppression in the FMDV infected cattle. The specific CD4 response to vaccination is variable.

MATERIAL AND METHODS

Bovine dendritic cells generated from CD14+ monocytes were (MoDC) were produced by published methods. Experiments were performed to determine integrin expression on these cells and whether serotype O FMDV could productively infect these cells. In addition, the effect of adding immune complexed virus to the DC was studied. Furthermore, the capacity of DC, pulsed with virus or inactivated antigen, to stimulate specific CD4+ T cell proliferation was determined.

RESULTS

MoDC do not express the integrin used by FMDV for entry and non-structural proteins are detected in 5% of the cells by flow cytometry. In contrast, 70% of the MoDC are infected after the addition of immune complexed virus, causing cell death in 6-8 hours, the production of type-I interferons and decreasing the ability to stimulate specific CD4+ T-cell proliferation. Using immune complexed inactivated virus to target uptake by MoDC, resulted in enhanced CD4+ T-cell proliferation.

DISCUSSION

These in vitro observations improve our understanding of the development of the immune response to FMDV infection in vivo and suggest alternative strategies to improve vaccine efficacy.