AVIAN INFECTIOUS ANAEMIA
CAUSED BY CHICKEN ANAEMIA AGENT (CAA)

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ABSTRACT

Chicken anaemia agent (CAA) has been studied in vivo and in vitro. Avian infectious anaemia (AIA) was considerably aggravated in chicks dually infected at day-old with CAA and virulent Marek's disease virus (MDV), infectious bursal disease virus (IBDV), or reticuloendotheliosis virus (REV). CAA-induced gross and histological lesions were characterized by panmyelophthisis and generalized lymphoid atrophy, and resembled those of the syndrome of anaemia, panmyelopathy and haemorrhagic diathesis occurring in the field. Circumstantial evidence suggested that CAA is also causing a severe immunodepression in neonatally infected chicks.

CAA replicates in cultured cells of the MDCC-MSB1 lymphoblastoid cell line. Cytopathic effects occurred by 30 hours postinoculation (p.i.) and were characterized by enlargement and subsequent destruction of cells. Infected MSB1 cells have been found to be suitable at 34 to 44 hours p.i. for indirect immunofluorescence tests to detect intranuclear CAA antigens or anti-CAA antibody, respectively.

INTRODUCTION

Chicken anaemia agent (CAA) is the causative agent of avian infectious anaemia and was first isolated by Yuasa et al. (1979). In SPF chicks neonatally infected by intramuscular inoculation, CAA causes an anaemia and pancytopenia between 8 and 20 days p.i. with a maximum between 14 and 16 days. Principal gross lesions consist in a more or less complete atrophy of bone marrow and thymus. Mortality of singly CAA-infected chicks is rather low. Reconvalescence starts as early as 16 to 18 days p.i. and usually is complete by 24 to 32 days p.i. (Yuasa et al., 1979; Taniguchi et al., 1983). Age-resistance against avian infectious anaemia develops during the first 7 days of life and is complete by 14 days. However, if day-old chicks are infected with infectious bursal disease virus (IBDV), age-resistance against anaemia is delayed by 2 to 3 weeks (Yuasa et al., 1980b). Maternal antibodies have been found to confer protection against infectious anaemia (Yuasa et al., 1980b). Single CAA infection of day-old chicks by contact does not cause anaemia. However, dual infection by contact with IBDV and CAA has been found to cause infectious anaemia with high mortality (Yuasa et al.,
1980b). This may have important implications with regard to the pathogenesis of CAA infection in the field. CAA can also be vertically transmitted. Transmission of CAA has been detected by eggs laid between 8 and 14 days after experimental infection of hens (Yuasa and Yoshida, 1983).

Highest titres of CAA have been detected in liver, thymus and spleen of neonatally infected chickens at 7 and 14 days p.i., and viraemia occurs from 1 to 7 days p.i. with a peak at 7 days. The agent persists in several tissues and in intestinal contents for at least 7 weeks but may be eliminated more rapidly in birds infected at 28 or 42 days of age (Yuasa et al., 1983a). Chicken liver is the preferred source of CAA for attempts of agent isolation. According to investigations of Yuasa et al. (1983b) and to studies in our laboratory, CAA is widespread in our chicken populations and may be considered as an ubiquitous infectious agent which may well play an important part in a number of mixed infections occurring in the field.

CAA can be propagated and assayed in cultures of the MDCC-MSB1 lymphoblastoid cell line whereas many other types and lines of cultured avian cells have been found to be unsuitable for CAA replication (Yuasa, 1983). Thus it became possible to employ cell cultures for neutralization tests (Yuasa et al., 1983a) which formerly could only be performed in chickens (Yuasa et al., 1980a).

Physicochemical properties of CAA indicate that this agent is likely to be a small virus like parvoviruses, but the true nature of CAA has not yet been identified. CAA is resistant to ether and chloroform, heat-resistant (1 hour at 70°C), acid-resistant (pH 3 for 3 hours), and it passes filter membranes with a 25 nm pore size (Yuasa et al., 1979).

The purpose of recent studies reported in this paper was to gain further knowledge about properties of CAA in vitro and in vivo with special reference to its possible importance in the field. Experimental methods and results will be described in more detail in two papers which are to be published by the end of this year (Bülow et al., 1985a,b).

EXPERIMENTS AND RESULTS

Growth and assay of CAA in MDCC-MSB1 cell cultures

CAA strains Gifu-1 (Yuasa et al., 1979) and Cux-1 (Bülow et al., 1983) were employed throughout the experiments.

Assay of CAA infectivity in MSB1 cell cultures turned out to be an unusually lengthy procedure, for several reasons. In our hands, CAA titres in culture supernatants increase by only 10-fold or little more within 3 to 4