were also used. The immunoperoxidase staining was performed according to the manufacturer's recommendations, modified by the substitution of CMV negative human serum for the manufacturer's horse derived blocking serum. This modification was made to eliminate possible non-specific binding of antibodies to the Fc receptor produced by the virus. The in situ hybridisation process was modified by carrying out the denaturation step at 80°C, thereby minimising non-specific background staining due to endogenous biotin.

None of the specimens showed positive staining for components of cytomegalovirus with either technique. We have thus not been able to support our hypothesis. The possibility remains, however, that CMV might act as an initiator in primary biliary cirrhosis, primary sclerosing cholangitis, or autoimmune hepatitis, but by the time the advanced disease process has been recognised the virus has already been eliminated.

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References

AgNORs in diagnosis of serous and mucinous ovarian tumours

In view of the recent interest in AgNORs as a tool in diagnostic histopathology and their recent application to problematic epithelial tumours we examined their potential as a diagnostic aid in serous and mucinous ovarian tumours.

The following tumours were selected from formalin fixed, paraffin wax embedded archival material: mucinous cystadenomas (n = 10); borderline tumours (n = 10); cystadenocarcinomas (n = 7); serous cystadenomas (n = 10); borderline tumours (n = 10); and cystadenocarcinomas (n = 10). Silver staining was performed according to the method of Crocker and Nar. The silver solution was in contact with the slide for one hour. Two hundred nuclei were examined in each tumour. In most preparations the nuclei contained large homogeneously staining structures resembling nucleoli, some of which showed denser areas suggestive of internal structures. We followed a similar policy to Howat et al counting each such structure as one AgNOR, and did not attempt to count intranuclear structures which, in formalin fixed tissue, are difficult to resolve. Tumours with higher AgNOR counts also have variable numbers of small dispersed intranuclear AgNORs which are a feature of malignant cells.

The figure shows the distribution of AgNORs in the six types of tumour. Mucinous cystadenomas had significantly lower counts than borderline tumours and cystadenocarcinomas (p < 0.01), but no significant difference was found between mucinous borderline tumours and cystadenocarcinomas. Serous tumours showed no significant difference between cystadenomas and borderline tumours, but cystadenocarcinomas had significantly higher AgNOR counts than borderline tumours.

AgNOR counts may therefore be useful in distinguishing borderline serous tumours from serous cystadenocarcinomas, but do not seem to be useful in mucinous tumours. The full potential of the AgNOR method may not be achieved in formalin fixed tissue because of the tendency of a variable proportion of the nucleoli to stain homogeneously, obscuring the internal structure of arrays of dot-like NOR-associated proteins seen in air dried and alcohol fixed smears. This raises the question of whether the pathologist should attempt to count partially fused intranucleolar dots, a process which is both tedious and subjective or, as we have done in this study, count only clearly discrete structures. Howat et al have examined this problem in detail. In studies where the nucleolus stains homogeneously and is therefore counted as one AgNOR it may conceal many dot-like NOR-associated proteins. One nucleolus may also be comprised of several loops of chromosomal rDNA (NORs) as several NOR-containing chromosomes may contribute to forming one nucleolus. Thus counting one nucleolus as one AgNOR gives no information about the activity of NORs either at chromosomal or transcriptional levels.

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