INTRODUCTION
Carcinoma of uterine cervix is one of the major forms of cancer in women. It accounts for about 7% of all the cancer cases among women in the United States of America and other developed countries whereas in the developing world its incidence is 24% and it contributes significantly to cancer related deaths (Parkin et al., 1984). In India it is the most common malignant tumour with the annual incidence constituting about 16% of the world’s annual incidence (WHO, 1986).

A variety of clinical and epidemiological studies suggest that several factors and/or sexually transmitted agent(s) could play role in the etiology of cervical cancer. The incidence of cervical cancer varies among the populations residing in different areas. This may partly be due to the variation in health practices and the efficiency of preliminary screening by Pap smear. Various factors associated with the risk of cervical cancer are the socio-economic variables, sexual behaviour, infection with sexually transmitted agent(s), smoking, diet, number of pregnancies, immunological status and the use of oral contraceptives.

The viral etiology of cervical carcinoma has focused on Herpes simplex virus-2 (HSV-2) and Human papilloma virus (HPV) (Diluca et al., 1987). The evidence in favour of HSV-2 is not compelling and for a long time the etiological importance of HPV could not be evaluated because of the inability to propagate HPVs in cell culture. The molecular
cloning of HPV-DNA from the cervical carcinoma tissue into the plasmid vectors, however, made it possible to study the incidence and oncogenic potential of HPV.

The molecular hybridization studies in relation to cervical, vulvar and penile intraepithelial neoplasia have projected a causal role of HPV infection. Furthermore, using highly sensitive techniques like polymerase chain reaction (PCR), HPV has been linked with as high as 98% of the cervical cancer cases in Indian women (Das et al., 1992) and of the various HPV types, HPV-16 appears to be the most prevalent accounting for about 50% of the genital HPV infections (Zur Hausen, 1989; Das et al., 1992) followed by HPV-18. The increased prevalence of certain HPV types is due to their higher degree of oncogenic potential. Today most of the detection/diagnostic methods are based on nucleic acid hybridizations which in addition to detection also give information regarding the type and physical status of HPV present and hence help in the proper management and prognosis of the case. Other detection methods used are electron microscopy for the presence of viral particles and immunohistochemistry for viral antigen. These methods, however, fail to type the HPV present and give no information about the physical status of the virus in the tissues. Besides, these methods are of little value for the detection of unexpressed virus.

The molecular events leading to the development of tumour or the symptom-free persistence of HPV are not
clearly understood. The long latency period between the symptomatic primary infection and the cancer development implies the involvement of other factors which may result in the breakdown of intracellular surveillance system thus leading to carcinogenesis. HPVs have been found to possess the ability to alter the structure and functions of the cellular protooncogenes (Popescu and DiPaolo, 1989). Viral DNA integration into a specific chromosome region may make the surrounding area of the cellular genome highly unstable, which could be expressed as chromosome break/gap and hence a fragile site. This may provide a medium through which gene mutation can occur ultimately causing cell transformation.

Since the relationship of genomic instability and cancer is well established, cytogenetic parameters like sister chromatid exchanges (SCEs) and chromosomal aberrations can be used as sensitive indices to evaluate the extent of genomic damage. Owing to the difficulties in obtaining primary culture for chromosomal analysis from the surgical specimens of the solid tumours, the assessment of chromosomal damage can be made from the cultured lymphocytes of the patients. The use of lymphocytes is based on the assumption that there should be a correlation between the extent of chromosomal damage in lymphocytes and other somatic cells (Nordenson et al., 1984).

With this background, the present work was undertaken with the objective to study the:
- Prevalence of various risk factors associated with the development of cervical carcinoma in North Indian population.

- Genomic instability in the lymphocytes and the progress of cell cycle in response to cervical carcinoma.

- Incidence of HPV in patients with cervical carcinoma as determined by:
  i) immunohistochemical detection of HPV-antigens.
  ii) Molecular hybridization studies using biotinylated HPV-16 DNA as probe.

- Physical status of HPV in the cervical carcinoma cases and its possible bearing on carcinogenesis.