INTRODUCTION
Lymphomas are malignant neoplasms characterized by the proliferation of cells native to the lymphoid tissues ie, lymphocytes, histiocytes and their precursors and derivatives. The two major variants of malignant lymphoma are non-Hodgkin’s lymphoma (NHL) and Hodgkin’s disease (HD). Although both of these tumors infiltrate reticuloendothelial organs, their biologic and clinical behaviours suggest that they are probably not related.

Hodgkin’s disease, like NHL, is a disorder involving primarily the lymphoid tissues (Portlock, 1984). It arises almost invariably in a single node or chain of nodes and spreads characteristically to the anatomically contiguous nodes. HD is characterized morphologically by the presence of only a small proportion of characteristic atypical cells, the mononuclear Hodgkin cells and the pathognomonic multinucleated Reed-Sternberg cells (H-RS cells). These H-RS cells are embedded in an abundance of reactive cells without cytological atypia comprising lymphocytes, plasma cells, macrophages, neutrophilic and eosinophilic granulocytes, as well as fibroblasts.

History
In 1832 Thomas Hodgkin was the first to propose the concept of a malignancy originating from primary tumors of the lymphatic system and was named Hodgkin’s disease by Wilks in 1865. In 1898 and 1902, Carl Sternberg and Dorothy Reed reported on the typical multinucleated giant cells bearing
their names Reed Sternberg cells and recognized the unique relationship of these cells to Hodgkin's disease. It was not until 1967 that the clonal origin of the malignant Reed-Sternberg cell were confirmed by cytogenetic analysis of cell lines by Sief and Spriggs. Today, the identity of the normal counterpart of the Reed-Sternberg cell and the kinds of molecular events that result in its malignant transformation remain controversial.

Pathological Classification

Hodgkin's disease differs from almost every other known malignant tumor in its unique cellular composition: a minority of putative neoplastic cells (Reed-Sternberg cells and their variants) in an inflammatory background. From case to case, differences in the morphology of the Reed-Sternberg cells and in the composition of the cellular background are associated with differences in patient demographics, clinical sites of disease, and natural history; these features form the basis for pathologic subclassification of Hodgkin's disease.

In the first clinically useful subclassification of Hodgkin's disease developed by Jackson and Parker, cases were divided into three groups: paragranuloma, granuloma and sarcoma (Jackson and Parker, 1944). In 1966, Lukes, Hicks and Butler proposed a new histologic classification that appeared to correlate well with clinical stage and aggressiveness of disease. This scheme was later simplified into the Rye classification, which is now widely employed. In the Rye classification, Hodgkin's disease is divided into four categories: lymphocyte predominant (LP), mixed
cellularity (MC), lymphocyte depleted (LD) and nodular sclerosis (NS).

**Lymphocyte Predominant**

The original Lukes and Butler scheme subdivided lymphocyte-predominant Hodgkin’s disease (LPHD) into nodular and diffuse subtypes. This distinction was obliterated by the Rye modification but appears to be important in the light of new information that consistently links the nodular subtype to the B-cell arm of the immune system. (Coles et al., 1988; Pinkus & Said, 1985; 1988; Poppema, 1980). In LPHD, the lymph node architecture is usually effaced, although a remnant of normal lymph node may remain. The cellular proliferation is composed of benign-appearing lymphocytes with or without benign histiocytes. The growth pattern may be diffuse but is more frequently nodular, and the nodules are considerably larger than those of follicular lymphomas. In nodular lymphocyte predominant Hodgkin’s disease (NLPHD), the Reed-Sternberg cell variants differ from mononuclear and classic Reed-Sternberg cells in that they have vesicular, polylobated nuclei and small, usually peripheral nucleoli; these have been called lymphocytic and histiocytic cells (L & H cells), or “popcorn” cells. In fact, they resemble “exploded” centroblasts. Although popcorn cells may be numerous, usually no classic Reed-Sternberg cells are found. The background is predominantly lymphocytes; clusters of epitheloid histiocytes may be numerous; plasma cells are infrequent; and eosinophils and neutrophils are rarely seen (Burns et al., 1984). Occasional sclerosis may cause lesions to resemble nodular sclerosis. This subtype is more common in male than in female patients and often occurs in the younger age groups (< 35 years of
age). Most patients have clinically localized disease and are asymptomatic, and the prognosis is usually favourable.

**Nodular Sclerosis**

Nodular Sclerosis Hodgkin’s disease has at least a partially nodular pattern, with fibrous bands separating the nodules in most cases; diffuse areas are common, as is necrosis. The characteristic cell is the lacunar-type Reed-Sternberg cell, which may be abundant (Anagnostou et al., 1977). These are cells with characteristically multilobated nuclei and small nucleoli, with abundant, pale cytoplasms, which retract in formalin-fixed sections, producing empty lacunae. Diagnostic Reed-Sternberg cells are also present but may be rare. The background usually contains lymphocytes, histiocytes, plasma cells, eosinophils and neutrophils (Coles et al., 1988). Necrosis is common, and necrotic foci are often surrounded by palisaded histiocytes, resembling granulomas. Neoplastic cells are usually most numerous around areas of necrosis.

In some cases with characteristic lacunar cells and a nodular or diffuse pattern, fibrous bands may be absent, and the differential diagnosis with NLPHD may be difficult. These cases have been called cellular phase of NSHD (Burns et al., 1984). The clinical course of these cases was slightly worse than that for typical Hodgkin’s disease (Colby et al., 1981). Another morphologic variant is syncytial NS. Focally, the NS pattern is lost, and large sheets of malignant cells resemble lacunar Reed-Sternberg cell variants (Strickler et al., 1986). Other studies have suggested that NSHD with lymphocyte depletion is associated with large mediastinal masses, advanced
stage, and poor response to radiation therapy alone (Kant et al., 1986; Mauch et al., 1982). Several grading schemes exist (NSI/II) based on the number and atypia and the Reed-Sternberg cells in the nodules (MacLennan et al., 1989; Haybittle et al., 1985).

Nodular sclerosis is the only form of Hodgkin's disease that is more common in female than male patients. It most frequently occurs in adolescents and young adults and is unusual in patients older than 50 years of age. The process has a striking propensity to involve lower cervical, supraclavicular, and mediastinal lymph nodes. It is often curable.

**Mixed Cellularity**

In mixed-cellularity Hodgkin's disease (MCHD), the infiltrate is usually diffuse or at most nodular, without band-forming sclerosis, although fine interstitial fibrosis may be present. Reed-Sternberg cells are of the classic, diagnostic type and are usually easily identified. Many mononuclear variants are also present; rare lacunar cells may be seen. Diagnostic Reed-Sternberg cells are large cells (15-45 μm in diameter) with bilobate, double, or multiple nuclei, with a large, eosinophilic, inclusion-like nucleolus in at least two lobes or nuclei. The infiltrate typically contains lymphocytes, epitheloid histiocytes, eosinophils, neutrophils and plasma cells (Lukes et al., 1966). Lymphocytes may predominate, however, giving rise to a differential diagnosis of lymphocyte predominance Hodgkin's disease (LPHD). If the Reed-Sternberg cells are of the classic type, a diagnosis
of LPHD should not be made.

Mixed cellularity patients are usually adults: males outnumber females, and the stage may be more advanced than NS or LP types, involving lymph nodes, spleen, liver or marrow. The course is moderately aggressive, but often curable.

**Lymphocyte depletion**

In lymphocyte-depleted Hodgkin's disease (LDHD), the infiltrate is diffuse and often appears hypocellular, owing to the presence of diffuse fibrosis and necrosis. There are large numbers of Reed-Sternberg cells, and bizarre sarcomatous variants, with a paucity of other inflammatory cells. Confluent sheets of Reed-Sternberg cells and variants may occur and rarely predominate (called reticular variant or Hodgkin's sarcoma) (Neiman et al., 1973). Before the availability of immunophenotyping studies, many cases diagnosed as LDHD were in reality cases of large B-cell lymphoma or T-cell lymphomas, often of the ALCL type (Kant et al., 1986). The borderline between the reticular variant of LDHD and ALCL is not sharp and may be a matter of definition (Leoncini et al., 1990; Stein et al., 1991).

This is the least common variant of HD and is most common in older people, in human immunodeficiency virus-positive (HIV) individuals and in nonindustrialized countries (Pelstring et al., 1991). It frequently presents with abdominal lymphadenopathy, spleen, liver and bone marrow involvement and without peripheral lymphadenopathy. The stage is usually
advanced at diagnosis: however, response to treatment is reported not to differ from other subtypes.

In 1994, the International Lymphoma Study Group introduced an updated classification of Hodgkin's disease, incorporating new immunologic and molecular data, as part of the Revised European-American Lymphoma (REAL) classification (Harris et al., 1994). This includes the same categories: NS, MC and LD are considered to be subtypes of "classical HD" and to be distinct from LPHD. One additional category, lymphocyte-rich classical HD, has also been included. Anaplastic large-cell lymphoma (ALCL), Hodgkin's-like is included in the REAL Classification as a provisional entity under the heading of non-Hodgkin's lymphoma; however, it has many features of HD and could be considered a subtype of HD.

Cell of Origin of Hodgkin's Disease

Most of the available immunophenotypic and genetic data suggest that the Reed-Sternberg cells, in most cases of classic Hodgkin's disease, are altered B cells of some kind, and they are at least in part clonal (Inghirami et al., 1994). A hypothesis has been proposed that the confusion over the cell of origin of Hodgkin's disease might be explained if the Reed-Sternberg cell represents an in vivo hybridoma between a follicular dendritic reticulum cell (FDRC) and a fused T or B lymphocyte, allowing the expression of a variety of nuclear characteristics of both FDRCs and T or B lymphocytes in different ways in different patients, including variable Epstein-Barr Virus (EBV) positivity (Sinkovics, 1990). A report of a binuclear Reed-Sternberg cell expressing p53 in
only one of its nuclei supports such a contention (Gupta et al., 1992).

Because Hodgkin’s disease is primarily a disease of lymph nodes, Reed-Sternberg cells might also derive from a rare cell that resides primarily in lymphoid tissue (Delsol et al., 1993). Such a cell could be the FDRC. The FDRC is an antigen-presenting cell that has been observed to be binucleate, to express the B7 costimulatory molecule and CD21, the receptor for EBV and CD40. The Reed-Sternberg cell has been shown to be intimately associated with the FDRC network. Hodgkin’s disease cells have been shown to have all the characteristics of major antigen presenting cells, expressing HLA class II molecules, B7 and CD21, and having a unique pattern of expression of the CD40 receptor (Delabie et al., 1995). The ligand for B7, CD28, is expressed on the CD4 positive T lymphocytes that surround both FDRCs and Reed-Sternberg cells. The interaction among CD28, B7, CD40 and CD40L might be expected to set off a cascade of lymphokine excitation that could explain both the histologic picture of Hodgkin’s disease and some of its clinical manifestations. In addition, many known cytokines have been found in the supernatant and in cells from various Hodgkin’s disease-derived cell lines (Diehl et al., 1990; Newcom and Tagus, 1992), including the eosinophil growth factor interleukin-5 only in patients with eosinophilia, and transforming growth factor â in the cells and urine of only patients with the fibrotic variant, NSHD.
The isolation and characterization of a new cell line, L1236, with rearrangement of the same VH and Vα genes, in cells in both the peripheral blood and the tissue in the same patient, again demonstrates the clonal nature of a malignant cell (Wolf et al., 1996).

Single cell analysis of Reed-Sternberg cells shows the expression of oncogenes and suppressor oncogenes in patterns characteristic of hematopoietic cells. Expression of p53 in Reed-Sternberg cells has been found in more than half of the cases (Trumper et al., 1993). Cases that overexpress EBV usually do not have evidence of p53 abnormality.

CLINICAL FEATURES

Clinical findings

(a) Lymphnodes
Painless enlargement of superficial lymphnodes is the most common presentation of Hodgkin’s disease in at least 70% of patients. There is usually a ‘rubbery’ consistency to the nodes which are non-tender; 60-80% of patients have enlarged cervical nodes, 6-20% have axillary and 6-15% inguinal adenopathy. Exclusive infradiaphragmatic lymphadenopathy is seen only in up to 10% of patients. At presentation mediastinal nodes will be involved in upto 60% cases and retroperitoneal nodes in 25% of cases. Occasionally, alcohol-induced lymphnode discomfort ranging to sharp pain may be noted (<10%). Splenomegaly is found in 30% of patients at presentation.

(b) Systemic symptoms
B symptoms, in particular low-grade fever, drenching night sweats, unexplained weight loss are present in about 40% of
patients, especially those with more advanced disease. Other non-specific constitutional symptoms include pruritus (10%) fatigue, anorexia and alcohol-induced pain (<10%) at the site of lymphadenopathy.

(c) Mode of spread
Hodgkin's disease appears to begin in an area within the lymphatic system and to spread in an orderly manner to contiguous lymph nodes via lymphatic channels. Non-contiguous spread and haematological distribution is more common with recurrent disease. This is also more common in certain histological types. In advanced Hodgkin's disease enlargement of the spleen/liver is also seen.

Management
Investigations
Initially, a diagnosis of Hodgkin's disease should be established histologically from a biopsy of involved tissue. It arises most often in lymph nodes, usually in the chest or neck. Further evaluation seeks to define the distribution and extent of the disease i.e., staging.

Staging is used to differentiate patients who can benefit from radiation therapy alone from those who require systemic treatment. Staging systems are anatomic descriptions of sites of tumor involvement in relation to the diaphragm. For staging Ann Arbor Classification was reported in 1971 (Carbone et al., 1971) (Table 1).
TABLE - 1

Ann Arbor Staging Classification for Hodgkin's Disease

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>Involvement of a single lymph node region or a lymphoid structure (e.g., spleen, thymus, Waldeyer's ring) or involvement of a single extralymphatic site (IE)</td>
</tr>
<tr>
<td>II</td>
<td>Involvement of two or more lymph node regions on the same side of the diaphragm (hilar nodes, when involved on both sides, constitute stage II disease); localized contiguous involvement of only one extranodal organ or site and lymph node region on the same side of the diaphragm (IIE).</td>
</tr>
<tr>
<td>III</td>
<td>Involvement of lymph node regions on both sides of the diaphragm (III), which may be accompanied by involvement of the spleen (III), or by localized contiguous involvement of only one extranodal organ site (IIIE) or both (IIISE).</td>
</tr>
<tr>
<td>IV</td>
<td>Diffuse or disseminated involvement of one or more extranodal organs or tissues, with or without associated lymph node involvement.</td>
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DESIGNATIONS APPLICABLE TO ANY DISEASE STAGE

<table>
<thead>
<tr>
<th>Designation</th>
<th>Description</th>
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<tbody>
<tr>
<td>A</td>
<td>No symptoms</td>
</tr>
<tr>
<td>B</td>
<td>Fever (temperature &gt;38°C), drenching night sweats, unexplained loss of &gt;10% of body weight within the preceding 6 months.</td>
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Staging starts with a detailed history and physical examination. The history must determine the presence or absence of systemic symptoms. Physical examination should determine the extent of lymphnode involvement. Basic laboratory tests include evaluation of renal and hepatic function, complete blood count, erythrocyte sedimentation rate (ESR), and serum lactate dehydrogenase and alkaline phosphatase levels.

Treatment

Treatment in newly diagnosed patients are based on the disease stage, involved sites, tumour bulk, and the presence or absence of B symptoms. The pathological subtype doesn't
influence management. Treatment i.e., radiotherapy, cyclical combination chemotherapy or both is virtually always given with curative intent (Gilbert, 1925; Easson and Russel, 1963; Pizzolo et al., 1987; Longo et al., 1991).

**Epidemiology**

Lymphomas as a group ranks seventh among all cancers worldwide, and there has been a 33% increase in estimated numbers (Parkin et al., 1993). Hodgkin’s disease accounts for about 1% of malignant diseases diagnosed in countries in the Western world each year. The incidence of Hodgkin’s disease varies widely in different populations (Correa et al., 1973; Macfarlane et al., 1995). The annual incidence rate per 100,000 population in the United States is 3 (Devita et al., 1997), 2.4 in the United Kingdom (Gupta, 1995), and that in Orientals is much lower, being 0.8 in Hong Kong (Ho et al., 1984) 0.6 in Japan (Kadin et al., 1983), 0.8 in Singapore (Waterhouse et al., 1982). The incidence in developing countries is also low (Correa and O’Conor, 1971; Mueller, 1987a; Kaplan, 1980). The data from the National Cancer Registry Programme (NCRP) of India reveals that the incidence in a developing country like India, varies from 1 to 3.1 (Jain, 1992).

Hodgkin’s disease shows an unusual variation in incidence with age. The variation from country to country is different for the three major groups — childhood, young adult and older adult. MacMahon (1966) reported bimodality in Hodgkin’s disease with peaks in young adults (aged 15 - 34 years) and in older adults (> 50 years). Correa and O’Conor (1971)
reported distinct patterns in the age-specific incidence of Hodgkin's disease which differed according to geographical area and which was based on the level of urbanization and level of economic growth. Three epidemiological patterns of Hodgkin's disease have been established (Correa and O'Conor, 1971; Mueller, 1987a; Kaplan, 1980). Type I, which is observed in developing countries, shows a first peak in childhood (first decade), a low incidence in the third decade and a second peak in older adults (Evans et al., 1978; Albujar, 1973; Yan et al., 1991; Riyat, 1992; Ambinder et al., 1993). Type III is found in developed countries showing the classical bimodal age incidence, characterized by low rates in childhood with rise to a peak in young adults and then a second peak in older adults (MacMahon, 1966; Grufferman and Delzell, 1984). With advancement in socioeconomic conditions, an intermediate pattern, type II, is seen, with the first age peak moving to the second decade. In Israel, Hongkong and rural areas of developed countries, this epidemiological pattern is found (Benharroch et al., 1997; Chan et al., 1995; Munoz et al., 1978; Gutensohn and Cole, 1980). In contrast, the Japanese show a unique pattern of Hodgkin's disease with a single age peak in older adults (Aozasa et al., 1986). A bimodal peak in the age distribution of Hodgkin's disease has been reported from various population based registries of the National Cancer Registry Programme of India (Jain, 1992).

Mixed cellularity is the most common histologic subtype seen in developing countries (Shanta et al., 1982; Glaser, 1990; Riyat, 1992), whereas in developed countries, nodular sclerosis is more common (Mueller, 1987a).
At almost all ages, males have a greater incidence of the disease than females (Cutler and Young, 1975). This excess is most marked during childhood and older ages (Grufferman and Delzel 1984; Ahmed et al., 1992; Erdkamp et al., 1992; MacMahon, 1966). No reasons for these differences have been advanced. As an alternative to a postulated protective role of female sex hormones, other factors such as gender-related differences in exposure and susceptibility to infections (Jarrett, 1993; Jarrett et al., 1996) or occupational exposures may be involved (Franceschi et al., 1991). It has been suggested that childbearing might have a protective effect. (Abramson et al., 1978; Glaser, 1994; Kravdal and Hansen, 1993).

There are racial differences in the incidence of Hodgkin’s disease. Black populations in the United States have a lower incidence but a worse survival rate for the disease than White Americans (Devita et al., 1997). Orientals also have a lower incidence of the disease.

Risk Factors

There have been numerous reports of multiple occurrences of Hodgkin’s disease within families (Grufferman et al., 1977). Siblings of young adult cases are at increased risk, whereas siblings of older adult cases have no increase in risk. There is a threefold risk for first degree relatives and a sevenfold increase for young adults (Razis et al., 1959; Grufferman et al., 1977). Among sibling pairs with Hodgkin’s disease, there is a marked excess of sex-concordant
pairs with a nine fold relative risk compared with fivefold for unlike-sex siblings. This pattern is hard to explain on a simple genetic basis and some component of the increased risk is likely to be due to shared environmental exposures. Families with more than one case of Hodgkin's disease share a human leucocyte antigen (HLA) haplotype between affected members (Robertson et al., 1987). However, it is known that the major histocompatibility complex can influence the manifestation of disease following retroviral infection and these genetic data do not rule out an environmental component. There is some evidence that populations migrating from low risk to high risk areas have an increased risk to developing Hodgkin's disease (Mason and Fraumeni, 1974).

For a short period, there was concern that Hodgkin's disease might be contagious because of reports of clustering of the disease, but that concern has been effectively dispelled (Mueller, 1992). The clustering was first reported by Vianna and associates among high school students exposed to the disease (Vianna et al., 1971; Vianna and Polan, 1973). Cancer registry studies from California and Connecticut (Kryscio et al., 1973) have also argued for cluster grouping of Hodgkin's disease on the basis of chance alone, but these studies have been contradicted by others.

Relatively little is known about occupational exposures and risk of Hodgkin's disease. The occupation most often considered to increase risk of the disease is wood working (ie, carpenters, sawmill workers, and so on); but this has not been confirmed with case control studies (Grufferman,
Chemists have also been suspected of being at increased risk of Hodgkin's disease. Li and his coworkers reported (1969) that chemists had an excess cancer mortality and that nearly half of the excess deaths were due to malignant lymphomas and cancer of the pancreas.

Vianna and Polan (1979) studied lymphomas and occupational benzene exposure in New York statemen. They found an overall excess of Hodgkin's disease deaths in men employed in jobs entailing exposure to benzene and/or coal tar fractions. There is no increased risk among physicians and nurses who are exposed to patients with the disease (Grufferman, 1982). Two studies have reported that use of the drug dextroamphetamine increases risk of Hodgkin's disease (Newell et al., 1973; Henderson et al., 1979). Studies on rubber workers have shown a slight increase in leukemia and lymphoma mortality for some occupational subgroups with chemical exposures (Monson and Nakanao, 1976; Monson and Fine, 1978; McMichael et al., 1976). It is not possible to say with certainty whether occupational chemical exposures increase risk of the disease.

Miller and Beebe' (1973) were the first to study the risk of Hodgkin's disease in persons with a prior diagnosis of infectious mononucleosis. In several studies that addressed the possibility of an increased risk of Hodgkin's disease associated with infectious mono-nucleosis, there was a modest threefold excess in the incidence of Hodgkin's disease among patients with a prior history of mono-nucleosis over that in controls (Rosdahl et al., 1974; Munoz et al., 1978).
Tonsillectomy and appendectomy has been reported as a possible risk factor for the development of Hodgkin’s disease, but several studies show inconsistencies and the observed associations may be related, at least in part, to socio-economic status (Grufferman and Delzell, 1984; Mueller et al., 1987).

Many studies have shown that the risk of developing Hodgkin’s disease is related to high socioeconomic status, at least in young adults, whilst older patients tend to be from lower socioeconomic class (Gutensohn, 1982). Other studies have suggested correlation with sibship size, housing and parental education, all as independent factors in the risk of developing the disease (Gutensohn and Cole, 1980, 1981). There is an increased risk of Hodgkin’s disease with increasing educational level of the patient (Mueller, 1992).

There have been reports of a seasonal variation in incidence, with a greater number of diagnosis being made in the winter months. However, it is likely that patients would be evaluated more frequently for upper respiratory tract infections more in winter rendering the diagnosis more common during these periods (Crowther et al., 1995).

Mycobacterium tuberculosis was first suspected to be the etiologic organism because of the high incidence of tuberculosis in patients with this disease (Steiner, 1934; L’Esperance, 1929; Van Rooyan 1933, 1934). Recent reports suggest that HIV positive persons are at a slightly increased risk of Hodgkin’s disease (Hessol et al., 1992). Clinical features are distinct and the prognosis is poor compared with
non-AIDS associated Hodgkin’s disease in that patients present with advanced stage disease, B symptoms and unusual sites of involvement such as the liver, central nervous system or skin (Jones et al., 1990; Schoeppel et al., 1986).

Another risk factor for Hodgkin’s disease is related to immunodeficiency status. Patients with primary immunodeficiency syndromes have a markedly increased risk of various lymphoreticular neoplasms (Kersey et al., 1973; Louie and Schwartz, 1978). The only genetic condition that appears to predispose to Hodgkin’s disease is ataxia telangiectasia, which is associated with an immuno deficiency syndrome.

Gutensohn and Cole (1977) pointed out that this model characterizes paralytic poliomyelitis. First, the reciprocal patterns in geographic occurrence for childhood and young adult Hodgkin’s disease in developing and developed countries fit the model well (Correa and O’Conor, 1971). Second, the risk of paralytic poliomyelitis is also related to social class. The higher the social class, the greater the risk. Third, both childhood Hodgkin’s disease and Paralytic poliomyelitis are characterized by a male preponderance (MacMahon, 1966; Melnick, 1976). Fourth, family size seems to be a determinant of risk in both diseases. Fifth, Paffenberger et al (1977) found that men who developed Hodgkin’s disease had a markedly decreased frequency of childhood infectious diseases compared to controls. This would suggest that they might have escaped exposure to a “polio-like” agent in childhood.

These epidemiological features suggest that Hodgkin’s disease may be a heterogeneous condition with different aetiological factors in the different age groups.