An Overview on Viral Origin of Cancer in Human
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INTRODUCTION
William Coley, M. D., a surgeon of New York in late 19th century, had proposed the parasitic origin of cancer. He, for the first time opined that the cancer might be of microbial origin. This theory in early 20th century faces many hardships as the link between bacteria and human cancers were not established; finally, a strong opposition stood up to pronounce the parasitic theory as dead. Even, in 1911, a biologist of Rockefeller Institute named Peyton Rous when clearly established the virus as a cause of cancer, then unfortunately, no one believed it (Rous 1911) [1]. It was not until 1966 when the same Peyton Rous was awarded a Nobel prize in physiology and medicine for his discovery of Rous chicken sarcoma virus was vindicated as a cause of cancer. It was a milestone contribution in the field of microbial origin of cancer (Weiss and Vogt 2011) [2].

Viral origin of cancer appears to be the second most important risk factor for cancer development in human (Zur Hausen 1991) [3]. Nearly, 20% of all human cancers worldwide may be attributed to viruses (Parkin 2006) [4]. These viruses play an important role in the development of cancer and new evidences link between these two. In fact, the virus by itself does not cause cancer, but it might influence tumor development when the changes occur. The relationship between these two is complicated and not yet very well understood.

An oncovirus is a virus capable of causing cancer having either DNA or RNA genome developing tumor. The possible role of viruses in the development of cancer has been the subject of much intensive studies during the past 50 years. In general, a viral genome, after entering into a living cell, must hijack the cells machinery in such a way to push the cell toward becoming cancer. These viral fragments containing oncogene (v-onc) transform the cells to produce tumors as soon as it expressed. It induces uncontrolled and unlimited growth of cells. Several oncomodulations confirming a strong link between these oncoviruses and the formation of cancer in experimental animals has been established. Perhaps, the most important motto at this juncture has been to work on the protection of tumor suppressor protein so that the tumor is not developed. But unfortunately, it couldn’t happen as several viruses containing oncogenes (v-onc) deregulate the synthesis of tumor suppressor protein via the formation of their own proteins. It causes impairment in signal transduction pathways. At present, the identification of these viral proteins causing cancers are the key point in the understanding of viral origin of cancer (Krzysztof Reiss and Kamil Khalili 2003) [5].
Both DNA and RNA viruses have been shown to be capable of causing cancer in humans. DNA tumor viruses are a group of double stranded DNA viruses that alter the glucose metabolism of normal cells (Mustaq et al., 2016) [6]. There are many DNA tumor viruses such as Human Papilloma viruses (HPVs), Human Polyoma Viruses (HPyVs), Human Herpes Viruses (HHVs) and Adenoviruses. Adenoviruses do not cause cancer in human. Similarly, some RNA viruses causing cancer are Hepatitis viruses, Human T-cell leukemia virus and the viruses associated with AIDS (Gaston 2012, Flora and Maestra 2015) [7, 8]. Further, some of the well noted cancers caused by these oncoviruses are described as under:

1. **HPVs** – Cervical, anal, mouth, throat, vaginal and penile cancers.
2. **HPyVs** – Merkel cell carcinoma of the skin; SV-40 causing primary brain and bone cancers; JCV causing lung, colon and brain tumor cancers and BKV causing nephrotic cancer after kidney transplant.
3. **HHVs** – HSV-1 causes thyroid tumor; HSV-2 causes melanoma, cervical and prostate cancers; Varicella zoster causes leukemia and breast tumor cancers; Epstein-Barr virus causes Burkitts lymphoma, nasopharyngeal and gastric carcinoma; Cytomegalovirus causes glioblastoma and salivary gland cancer; Roseolovirus causes demyelination in brain and tumor and HHV-8 causes Kaposi’s sarcoma.

4. **Hepatitis viruses** – cause hepatocellular carcinoma.
5. **HTLV-1** – causes human adult T-cell leukemia (ATL).
6. **HIV** – causes a variety of cancers in immunocompromised patients due to parallel infections.

The present review is an attempt to deal with the study of viral origin of cancer in the light of recent researches done so far in the same field.

### Table 1: A List of Viral Origin of Cancer

<table>
<thead>
<tr>
<th>S. N</th>
<th>Name of Oncogenic Viruses</th>
<th>Types of Cancer (Suggestive Link)</th>
<th>References</th>
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<tbody>
<tr>
<td></td>
<td>Merkel cell polyomavirus (MVC, MCPyV, or HPyV5)</td>
<td>Mesotheliomas and brain tumor</td>
<td>Faust et al., 2009 [16]</td>
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<td></td>
<td>Simian virus- 40 (SV-40)</td>
<td>Skin cancer</td>
<td>Carter et al., 2009 [16]</td>
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<td></td>
<td>JC virus (JCV, HPyV2)</td>
<td>Brain and bone cancer</td>
<td>Delianis and Hirch 2013 [10] Faust et al., 2009 [16]</td>
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<tr>
<td></td>
<td></td>
<td>Brain tumor and lung cancer</td>
<td>Toptan et al., 2016 [11]</td>
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<tr>
<td>3.</td>
<td>BK virus (BKV, HPyV1)</td>
<td>Bladder and prostate cancer</td>
<td>Qi et al., 2011 [21]</td>
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</table>

### DISCUSSION

Currently, the viral origin of cancer is well documented by the researches done so far in the past several decades. The present review described different viruses developing the various kinds of cancers comprising of their nature, disease causing ability and the disease complications causing cancer in human. A number of infectious viral agents have been suspected or recognized to be associated with human cancers including human papillomaviruses (HPV), human polyomaviruses (HPyV), human herpes viruses, hepatitis viruses, human T-cell leukemia virus-1 and human immunodeficiency viruses (Table 1). They are being discussed in the light of recent researches as under:
1. Human Papillomaviruses (HPVs)

About a dozen of HPVs have been allocated carcinogenic by the IARC in Group 1. They cause cervical cancer in human, which is the third leading cancer in the world in terms of mortality. In addition to cervix uteri HPV can also target sites in the anogenital regions of both the sexes including upper aerodigestive tract (Flora and Maestra 2015) [7]. They are mainly contaminated in polio vaccine. And, the people who were accidently exposed to this oncogenic virus. Later, it was discovered as a potent carcinogenic bio contaminant in polio vaccine. And, the people who were accidently exposed to this oncogenic virus. Later, it was discovered as a potent carcinogenic bio

- HPV-16 and HPV-18. These two viruses causing cervical cancer were investigated by a Nobel laureate Zur Hausen, 2010. HPVs inhibits the function of tumor suppressor protein p53 of the host cells (Masroor et al., 2020) [9].

2. Human Polyomaviruses (HPyVs)

Human Polyomaviruses are DNA tumor viruses whose infection is widespread but largely asymptomatic causing non-neoplastic diseases in immunocompromised patients. Today, the human polyomavirus (HPyV) family consists of:

1. Merkel Cell Polyomavirus (MCPyV)
2. Simian Virus-40 (SV-40)
3. Trichodysplasia spinulosa Virus (TSV) HPyV8
4. JC Virus (JCV) HPyV2
5. BK Virus (BKV) HPyV1
6. KI Virus (KIpyV) HPyV3
7. WU Virus (WUPyV) HPyV4
8. MW Virus (MWPyV) HPyV10
9. HPyV6
10. HPyV7
11. HPyV8
12. HPyV12
13. HPyV13

These human polyomaviruses (HPyVs) have been linked to a variety of diseases causing cancer (Delianis and Hirch 2013 and Toptan et al., 2016) [10, 11]. Four viruses of polyomaviridae named as SV40, MCPyV, BK and JCV have been reported as cancer causing viruses in human. Simian Virus 40 develops mesothelioma in human and has been allocated by the IARC in Group B carcinogen. Merkel cell virus causes Merkel cell carcinoma and its carcinogenicity is classified in Group 2A; the probably carcinogenic to humans because their carcinogenicity has not yet been proved in experimental animals. Similarly, the other two viruses as BKV and JCV have been classified as still to be proved cancer causing in human (Salim et al., 2020) [12].

2.1. Merkel Cell Polyomavirus (MCPyV or MCPyV)

Merkel cell carcinoma is a very aggressive type of skin cancer mostly found in older people who are generally immunocompromised with higher frequency of transplantations kept under the medication of immunosuppressants (Williams et al., 1998, Engels et al., 2002 and Kassem et al., 2008) [13-15]. Currently, no any proper medication or vaccinations are available for the treatment of the same cancer (Carter et al., 2009, Faust et al., 2014) [16, 17].

2.2. Simian Virus – 40

Simian virus-40 was discovered as a contaminant in polio vaccine. And, the people vaccinated during the period 1955 to 1963 for polio were accidently exposed to this oncogenic virus. Later on, it was discovered as a potent carcinogenic bio-organism developing brain and bone cancer, mesothelioma and non-Hodgkin lymphoma in human (Stratton et al., 2003, Regis et al., 2004, Biochuk et al., 2010 and Qui et al., 2011) [18-21].
2.3. JC Virus (JCV, HPyV_2)

The John Cunningham virus or JC virus is only found in humans. The virus produces progressive multifocal encephalopathy (PML) in immunocompromised patients. It crosses the blood-brain barrier to infect the brain causing encephalopathy, demyelination and meningitis. The JC virus is found in huge amount in urban sewage worldwide whose infection took place in a very childhood stage. Nearly, 80% populations are infected with the same virus globally but the virus activated only in those who are immunocompromised. Immunosuppressants arecontradicated in these patients (Del Valley et al., 2001, Khalili et al., 2003, Zheng et al., 2007) [22-24].

2.4. BK Virus (BKV or HPyV_1)

BK virus is developed in those patients who have undergone kidney transplantations. The virus was isolated from urine in the same patient named as BK. It causes graft disorder as BK virus associated nephropathy (BKVN) developing bladder and prostate cancer (Das et al., 2008, Abend et al., 2009, Pino et al., 2013, Syed et al., 2013) [25-28].

3. Human Herpes Viruses (HHVs)

Herpes viruses are the DNA viruses derived from the Greek word herpein (to creep) referring to the latent and recurring infections. Eight viruses have so far been identified belonging to the family herpesviridae infecting humans are categorized in subfamilies as α, β and γ. The α – sub family includes herpes simplex viruses (HSV) type 1 & 2 and Varicella zoster virus (VZV or HSV-3); β- subfamily contains cytomegalovirus (CMV or HHV-5) and roseoloviruses (HHV-6 & HHV-7); similarly, Epstein-Barr virus (EBV or HHV-4) and Kaposi’s sarcoma virus (KSHV or HHV-8) belong to the γ sub family. The genetic material DNA of all 8 viruses replicated well in nucleus in a wide variety of animals including human. Some of the vertebrates they infected are horses, pigs, mice, cattle’s and fishes. It has also been reported that more than 90% of adult humans are at least having one herpesvirus in their lifetime which can remain in a latent form lifelong. They cause either latent or lytic infections. Their latency and reactivation are enough to cause cancer (Grinde 2013) [29]. HSV-2 has also been reported to cause cervical cancer in women. The DNA fragments of genital herpes have been isolated in nearly half the patients suffering from cervical cancer. In addition to cervical cancer, these viruses are also suspected to cause Burkitt’s lymphoma, nasopharyngeal carcinoma and possibly vulvar cancer, Kaposi’s sarcoma occurring primarily in men of homosexual habit and the cancers of prostate and bladder (Kulomaa et al., 1992, Whitley 1996, Haverkos et al., 2000, Davidson 2010, Brown and Newcomb 2011) [30-34].

3.1. Human Herpes Simplex Viruses (HSVs)

There are two types of HSVs as HSV-1 and HSV-2. They cause oral and genital infections. While HSV1 develops herpetic labialis or cold sores, herpetic gingivostomatitis and herpetic gladiatorum, the HSV-2 is usually involved in producing genital warts, melanoma, thyroid, prostate and cervical cancers. These viruses are simply transmitted by close contact with body fluids via kissing and oral sex. Human herpes viruses have developed numerous mechanisms of immuno evasion causing cancer. Further studies are still needed to elaborate the role of these mechanisms in carcinogensis and to apply these knowledge in the development of novel cancer therapies. However, there are some antiviral drugs that are effective against some kinds of herpes viruses (Kulomaa et al., 1992, Gupta et al., 2007, Opstelten et al., 2008, Jensen et al., 2010, Thomas et al., 2011, Mohan et al., 2013, Chi et al., 2015, Anderson et al., 2016) [30, 35-41].

3.2. Varicella Zoster Virus (VZV, HHV_3)

Varicella zoster virus causes chicken pox and shingles in human. After an infection, the latency of virus is lifelong in human sensory ganglia and is reactivated when a body becomes immunocompromised in future. It produces several lumphoproliferative disorders, leukemia, necrotic skin lesions and breast tumors. (Kurtaran et al., 2009, Gilden et al., 2012, Mina et al., 2012, Chiu et al., 2013, Qian et al., 2019) [42-46].

3.3. Epstein – Barr Virus (EBV or HHV_4)

Epstein-Barr virus is an oncogenic virus already placed in Group 1 carcinogenic agents by the IARC 2011. In 1964, Anthony Epstein, Bert Achong and Yunne Barr identified for the first time a new human oncovirus as Burkitt lymphoma virus (Epstein et al., 1964) [47]. This was a type of herpes virus. This is a DNA tumor virus infecting B-cells of the immune system and epithelial cells. These infections are quite known in the society as “kissing diseases” or mononucleosis or “mono”. The disease is very easily transmitted simply either by kissing or coughing, sneezing, sharing drinking or eating utensils. As with other herpes viruses, though, not everyone develops the symptoms of kissing disease, their infection is lifelong. Similarly, the prominent period of getting infection has been found to be the early teen age. EBV cancers are most common in Africa and Southeast Asia. The EBV in human causes harmless to life threatening infections causing cancer (Masroor 2020) [48]. A list of these cancers caused by the EBV are given as under:

1. Nasopharyngeal carcinoma (Alibek et al., 2013 and Shinokuma et al., 2003) [49,50].
2. Burkitt’s lymphoma (Zhang et al., 2013) [51].
3. Hodgkins lymphoma (Gulfaraz 2005) [52].
5. Gastric carcinoma (Kuzuschima et al., 1999, McLaughlin Drubin and Munger 2008 and Sousa et al., 2008) [58-60].
6. Epithelial malignancies (Tsao et al., 2015) [61].

3.4. Human Cytomegalovirus (HCMV, HHV6)

HCMV may cause serious diseases in people with a weakened immune system. Human cytomegalovirus (HCMV) infects most organs of the human body including brain, breast, colon, eyes, kidney, liver and lungs. However, their most preferable place has been reported to be the salivary glands (Koichi Yamanishi et al., 2007) [62]. HCMV remains latent within the body throughout life but it might be reactivated at any time causing mucocutaneous lesions of salivary gland and other malignancies like prostate cancer (Geder et al., 1977 and Melnick et al., 2011) [63, 64]. HCMV has also been reported to cause breast cancer in women (Georges and Amit 2014) [65] and the glioblastoma, a deadly form of cancer in brain (Price et al., 2013) [66]. Most healthy children and adults infected with CMV will recover within time without any complications and do not require antiviral treatment. However, antiviral treatments may improve the prognosis in some patients. There is no commercially available CMV vaccine. Experimental vaccines are being studied.

3.5. Roseoloviruses (HHV6 & HHV7)

There are two types of roseoloviruses found in nature as HHV-6 and HHV-7 infecting humans. Surprisingly, these viruses are found in all human populations globally. Roseola infantum is characterized by the high fever with rose coloured rashes developed on face, neck, trunk, thighs and legs. This is widespread around the world causing brain cancer in children most commonly involving 6 months to 3 years. Since the salivary glands are the natural reservoir of these viruses, the disease is spread by saliva and not by rashes. The cells infected with the roseoloviruses cause disturbed apoptosis and necrosis developing cancer and death of cells respectively (Kofman et al., 2011, Henri et al., 2015) [67, 68].

3.6. Kaposi’s Sarcoma Herpes Virus (KSHV, HHV8)

Kaposi’s sarcoma (KS), a kind of cancer usually developed in immunocompromised patients including AIDS and is caused by the KSHV or HHV-8. KS is characterized by the purplish red coloured lesions found on nose, around the lips, ears and eyes (Peng et al., 2020) [69]. This is a sexually transmitted virus but it seems that it to be spread by some other ways also as direct contact through blood and saliva as well (Antman and Chang 2000, Pagano et al., 2004) [54, 70] and cancer known as primary effusion lymphoma and in many people the multicentric Castleman disease i.e. cancer of the lymph nodes or lymphoma. Further, studies are still required to better understand the role of HSV- 8 in these cancers. Treatment of Kaposi control of HIV infections using HAART is an integral part of successful kaposi sarcoma therapy. Usual therapy may be combined with not only antiviral drugs but anticancer drugs too to prevent and treat infections. Since the KS grows and spreads slowly, their lesions are more often treated with surgery, radiations and intralesional therapies (Salim 2021) [71].

4. Hepatitis Viruses (HAV, HBV & HCV)

The inflammation of liver is hepatitis caused by the hepatitis viruses like A, B, C, D and E. Hepatitis B and C viruses are rather more potent carcinogenic viruses than hepatitis A virus. HBV was investigated in 1960 by Baruch Blumberg for which a Nobel prize in physiology and medicine awarded to him in 1976. In 1980, the oncogenicity of the same virus as potent hepatocellular carcinoma (HCC) in human was established by Palmer Beasley. HCV was investigated by Michel Houghton and Brady in 1989 and their pathogenicity was established as developing hepatocellular carcinoma worldwide. Though, HAV is not involved in producing liver cancer, it has been linked with some other cancer like non-Hodgkin lymphoma. Similarly, HCV seems to be the more dangerous than HBV as it produces liver cancer asymptomatically. And, a patient dies within a year of infection developing hepatocellular carcinoma if not treated well within time. However, both the viruses as HBV and HCV are quite different in nature. While HBV is DNA virus belonging to the family hepadnaviridae, HCV is an RNA virus of flaviviridae. Both the viruses are classified by the IARC as Group 1 carcinogenesis. Further, both HBV and HCV can be treated with drugs and can be recovered completely within a few months. The effective vaccines are available for HAV and HBV. However, no effective vaccines are available for HCV. The therapies which can arrest the histological progression and the virus replication are followed. Such therapies include α-IFN and lamivudine for hepatitis B and α-IFN and ribavirin for hepatitis C as the drug of choice (Beasley et al., 1981, Choo et al., 1989, Baruch Blumberg 2010) [72-74].

5. Human T-cell Leukemia Virus -1 (HTLV-1)

HTLV-1 was investigated by Mitsuaki Yoshida, Bernard Poiesz and Robert Gallo in 1980. It causes human adult T- cell leukemia (ATL). The incubation period of developing ATL is about 20 years or more with the probability of causing cancer is 5%. It causes disabling and fatal diseases in human with no satisfactory means of treatment available so far. HTLV-1 is a RNA virus belonging to a class of viruses known as retroviruses. The retroviruses for their multiplication must go through an extra step called reverse transcription for the formation of new DNA genes. Now, these genes when integrated with the host cell chromosome and expressed phenotypically it sometimes, lead to cancer. HTLV-1 is just like HIV, which is another human retrovirus but HTLV-1 does not cause AIDS. However, the modes of transmission are exactly the same as HIV. This cancer is mostly found in southern Japan, south America, the Caribbean
6. Human Immuno Virus (HIV)

HIV causing AIDS does not cause cancer directly. In fact, it weakens the immune system by destroying the helper T-cells of WBCs. A weak immune system might let new cancer cells survive long enough to grow and form life threatening tumor. It increases a persons’ risk of getting several cancers caused by some other viruses too such as Kaposi sarcoma, cervical cancer, Hodgkin disease, non-Hodgkin lymphoma, anal cancer, lung cancer, oral cancer, throat and skin cancer and many other types of cancers. HIV-2 is considered a Group 1 carcinogen by the IARC (Flora and Maestra 2015) and the role of HIV in genesis of many cancers is supported by the findings of several studies (Silverberg et al., 2011 & 2013, Gopal et al., 2014, Siegel et al., 2019 and Geoffrey et al., 2020) [79-83]. Perhaps, the very discovery of HIV virus causing AIDS in human population had surprised us a lot in such a way that we have forgotten the pain of AIDS patients and our whole energy of research has diverted in killing the virus from the society. If we are not exaggerating the fact, no one has died of AIDS virus itself, but the AIDS patients are dying due to para infections caused by several microorganisms causing various ailments, diseases and cancers. And, this is all due to the immunity failure caused by the HIV simultaneously (Yarchoan and Uldrick 2018, Justiz and Naik 2021) [84, 85].

Moreover, HIV has always been an overlooked cause of cancer in AIDS patients. And, the AIDS patients are continuously suffering from cancers and they have never been included in any surveillance programme globally except the programmes framed for the reduction of STDs expected to contribute in controlling the HIV oriented cancers simultaneously. Therefore, currently, a better understanding on cancer incidences of HIV patients is required. Unfortunately, even today, there are no drugs and/or vaccines available to prevent the patients from HIV. However, antiretroviral therapy (ART) or highly active antiretroviral therapy (HAART) have been found to be associated with improved survival among patients with AIDS (Diamond et al., 2006) [86].

Further, generally people with HIV go through the same cancer treatment people without HIV go through. Standard treatments for HIV patients having cancer include chemotherapy, radiation, immunotherapy, targeted therapy and surgery. Unfortunately, all the antiviral drugs which are available in market can kill the human immunoviruses only outside of human body and still we could not make opportunity to kill them inside the human body. Certainly this is due to their nature of viruses as they are non-living outside the body but behaves like a living organism inside the human body. And, they are being treated as the connecting link between non-living and living matters. Lastly, as to be mentioned that despite all the efforts we made in past, we are still struggling to know the complete molecular biology of the viruses to kill them inside the host especially the AIDS viruses acquiring high rate of mutability and adaptability in the host cells. (Coghill et al., 2015; Zucman et al., 2018) [87, 88].

CONCLUSION

The present review deals with the study of a very paradoxial subject of cancer biology as the viral origin of cancer. Currently, the subject is now fully documented in the scientific society and should be acquainted to all of us that microbial origin of cancer exists. In fact, the story began with the award of a Nobel Prize to Peyton Rous in 1966 for the discovery of Rous sarcoma chicken virus as the casual organism in developing the sarcoma cancer in domestic fowl. Since then several viruses have been investigated to cause cancers in human. Some of them are as human papillomaviruses causing cervical, vaginal, penile and anal cancers; human polyomaviruses causing brain and bone cancer, Merkel cell carcinoma developing lung, bladder and prostate cancer; human simplex viruses causing thyroid, prostate, vulvar and cervical cancer and melanoma; herpes zoster virus causing breast, head and neck cancer; Epstein Barr virus causing Burkitts’ lymphoma; human cytomegalovirus causing mucoepidermoid carcinoma of salivary glands, prostate and breast cancer and glioblastoma; human roseoloviruses causing brain tumor and cancer in various body parts; Kaposi’s sarcoma herpes virus causing Kaposi sarcoma; hepatitis viruses causing hepatocellular carcinoma; human T-cell leukemia virus causing T-cell leukemias and human immunodeficiency virus causing AIDS and cancers in human.

ACKNOWLEDGEMENTS

This piece of research work is dedicated to the memory of Late Purva Singh, who died recently in her young age. We are also grateful to the institutions concerned for providing us necessary facilities during the course of this research work.

Ethical Clearance: Since the article is purely a review work hence it does not require an ethical clearance.

Conflict of Interest

The authors have declared that no competing interests exist amongst us. They have approved the final version of the manuscript contributing equally.

Financial support and sponsorship

No financial support was granted during the course of this research work.
Abbreviations

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<thead>
<tr>
<th>Abbreviation</th>
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<tr>
<td>WBCs</td>
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<tr>
<td>DNA</td>
<td>Deoxyribonucleic acid</td>
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REFERENCES


