UNIVERSIDAD DE QUINTANA ROO

División de Ciencias Políticas y Humanidades

TRANSLATION FROM ENGLISH INTO SPANISH OF THE ARTICLE "ACQUIRED IMMUNE DEFICIENCY SYNDROME" AND ANALYSIS OF SOME TRANSLATION TECHNIQUES

TRABAJO MONOGRÁFICO
Para obtener Grado de:

LICENCIADO EN LENGUA INGLESA

PRESENTA

Edie Enrique Del Angel Marrufo

Supervisores

M. C. Alessio Zanier Visintin
M. C. Magnolia Negrete Cetina
M. C. Gabriel Lira Gutiérrez

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PRESENTATION

The main goal of translation is to reproduce various kinds of texts, such as religious, literary, scientific and philosophical texts in another language and making them available to readers. The difference between a SL and a TL and the variation in their cultures makes the process of translating a real challenge. We can face problematic factors involved in translation such as form, meaning, style, proverbs, and idioms and so on; for that reason, I have to tackle the basic task of choosing the foreign text to be translated and I will find methods to translate it Bassnet (2002, p.68). As we all know, good translation is not usually just a question of translating each word of the source text into the target language. Nor does it consist, however, of the translator skimming through the source text, putting it aside and then jotting down the general idea of it in his or her own words in the target language. In between the two extremes there is a wide variety of techniques or strategies, many of which translators will use intuitively for any given text. **Translation** is the interpreting of the meaning of a text and the subsequent production of an equivalent text, likewise called a "translation," that communicates the same message in another language. The text to be translated is called the source text, and the language that it is to be translated into is called the target language; the final product is sometimes called the target text.

Translation must take into account constraints that include context, the rules of grammar of the two languages, their writing conventions, and their idioms. A common misconception is that there exists a simple word-for-word correspondence between any two languages, and that translation is a straightforward mechanical process; such a word-for-word translation, however, cannot take into account context, grammar, conventions, and idioms.

Translation, when practiced by relatively bilingual individuals but especially when by persons with limited proficiency in one or both languages, involves a risk of spilling-over of idioms and usages from the source language into the target language. On the other hand, inter-linguistic spillages have also served the useful purpose of importing calques and loanwords from a source language into a target language that had previously lacked a concept or a convenient expression for the concept. Translators and interpreters,

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professional as well as amateur, have thus played an important role in the evolution of languages and cultures.

This work is consisted the translation of a text about Acquired Immune Deficiency Syndrome (AIDS). The text was written by Howard A. Liebman, Timothy P. Cooley and Alexandra M. Levine. The translation of this text will be useful to the people who work in the laboratory of the Hospital General de Chetumal and will provide an important and valuable tool to update AIDS´ tests. Also, this work will show an important and very topical issue about AIDS. The text is scientific and has many technical words but with the help of technical dictionaries and chemists who work in the lab I translated it.

Beutler & Lichtman explained that in 1981, the very first acquired immune deficiency syndrome (AIDS) was discovered and by 1983 the human immune deficiency virus (HIV) was identified as the main agent of this illness. In 1982 AIDS was related to blood transfusions because the majority of cases were identified in patients with hemophilia, but some researches demonstrated that it was not true. By 1984 the responsible of the illness was identified as HIV.

From the very beginning of the epidemic until nowadays, the deceased people were approximately 16 millions and almost 33 millions of people are now infected with the HIV. 90% of those infected by HIV live in the developing countries (CENSIDA, 2001).

Klehment (1993, p. 88) says that by far, the most common is the immune deficiency syndrome (AIDS), followed by the human deficiency virus (HIV). This retrovirus is transmitted by sexual contact, blood and blood product transfusions and by other body fluids. Most patients experience an acute mononucleosis- like syndrome 3 to 6 weeks after the infection. This syndrome is due to acute viremia which is followed by the development of an immune response. The viremia ceases and a period of latency starts which may last for several years.

During the latency period, the patient does not show any symptom, but the infection persists and progresses. However, the patient can infect a sexual partner. Virus can be demonstrated in the patient's lymph nodes and in "T"lymphocytes and macrophages (both are blood's cells) (Ibid).

Lee (2002, p. 104) claimed that the mechanism which the virus uses to exist during the latency period is under intense investigation. There is no doubt that the process involves the progressive destruction of CD4 positive lymphocytes (a main blood antibody) by HIV infection. As the disease progresses, the ratio of CD4 to CD8 T cells (important blood cells which have the function to protect the human body from viruses) decreases.

After a latency period of 5 to 10 years, the patient presents symptoms due to deterioration of the immune system. The diagnosis of AIDS depends on two elements. The first is the demonstration of the antibody to HIV indicating previous infection and the second is the appearance of one or more AIDS defining illnesses, for example: Herpes simplex, Pneumonia, Toxoplasmosis of brain, Encephalopathy, Kaposi's sarcoma and Anemia.

AIDS patients usually show a mix up of anemia, granulocytopenia and thrombocytopenia (all are example of blood abnormalities when the blood rates decrease).

The immune deficiencies can be treated by replacing the missing or dysfunctional component of the immune system. Although it is possible to supply antibodies to replace "B" cell functions, it is not possible to replace defective "T" cell functions except by transplanting a new "T" cell in the system. In addition, the use of gamma globulins (antibodies) could be useful because patients will reinforce their immunological system.

The treatments of AIDS are directed at suppressing the growth of the virus. The drugs which are available inhibit the viral replication and slow the progress of the disease, but they do not result in a cure, only in a treatment. They have a limited duration because of virus-generated drug resistance.

  HIV/AIDS are an increasing challenge to countries all over the world, both directly as a health issue and indirectly through the challenges they pose for development (SIDA, 1999). AIDS is a critical and radical problem for development because of the number of people and sectors affected. By killing large numbers of productive and reproductive adults, AIDS decreases the human development infrastructure and increases health demands. It also increases the total number of children orphaned by death of one or both parents (UNAIDS, 2000). The **World Health Organization** (**WHO**) is a specialized agency of the United Nations (UN) that acts as a coordinating authority on international public health. Established on April 7, 1948, and headquartered in Geneva, Switzerland, the agency inherited the mandate and resources of its predecessor, the Health Organization, which had

been an agency of the League of Nations. As the directing and coordinating authority on international health, the World Health Organization (WHO) takes the lead within the UN system in the global health sector response to HIV/AIDS. The HIV/AIDS Department provides evidence-based, technical support to WHO Member States to help them scale up treatment, care and prevention services as well as drugs and diagnostics supply to ensure a comprehensive and sustainable response to HIV/AIDS. HIV infection in humans is now pandemic. As of January 2006, the Joint United Nations Program on HIV/AIDS (UNAIDS) and the World Health Organization (WHO) estimate that AIDS has killed more than 25 million people since it was first recognized on December 1, 1981. It is estimated that about 0.6 percent of the world's population is infected with HIV. In 2005 alone, AIDS claimed an estimated 2.4–3.3 million lives, of which more than 570,000 were children. A third of these deaths are occurring in sub-Saharan Africa, retarding economic growth and increasing poverty.[4] According to current estimates, HIV is set to infect 90 million people in Africa, resulting in a minimum estimate of 18 million orphans.[1] Antiretroviral treatment reduces both the mortality and the morbidity of HIV infection, but routine access to antiretroviral medication is not available in all countries.

The rate of new HIV infections demonstrated large differences between and within regions. The reasons for these geographical differences are not fully understood, but the main factors are clear and relate to different transmission routes and risk environments, as well as when HIV/AIDS first reached a given region (SIDA, 1999). The first cases of AIDS in Mexico were diagnosed in 1983. From that time through January 1st, 1998 a total of 33,632 cases of AIDS cases have been reported to the Secretary of Health of Mexico. These figures, however, are likely to be underreported. The AIDS surveillance system in Mexico faces complex hurdles with serious problems relating to a lack of diagnosis, delayed diagnoses, and a changing case definition. It is possible that the number of cumulative AIDS cases is actually closer to 53,000 with as many as 200,000 persons infected with HIV. HIV infection is frequently diagnosed quite late in the course of the disease in Mexico. At Anonymous /Confidential AIDS Testing Centers close to 60% of new HIV diagnoses are made when CD4+ T-cell counts are <200 cells/ml with less than 20% of patients being diagnosed with a CD4 cell count >500 cells/ml.

AIDS mortality is increasing among the general population in Mexico with the most dramatic increase found among men who are between the ages of 25 and 44[2]. AIDS is now the fourth leading cause of death nationally for men in this age group; in many states it is the third leading cause of death. In 1996 a total of 4,373 AIDS related deaths were reported in Mexico, a rate of 4.7 per 100,000. No significant drop in AIDS mortality has been seen in Mexico as it has in the United States and other developed countries. This is probably due to the relatively limited availability and use of highly active antiretroviral therapy (HAART) in the general population of infected people.

HIV transmission patterns in Latin American countries are similar to transmission patterns in industrialized countries. While it appears that the current rate of infection is decreasing or at least stabilized, the outlook is not uniform across the Latin American region. In most Latin American countries the epidemic is classified as "concentrated" in specific populations. Seroprevalence rates in these at-risk populations range from 1 to 25%. A seroprevalence rate of fewer than 5% is seen in the total population. However, in recent years some countries in the region such as: Nicaragua, Venezuela, and Peru appear to have rapidly increasing seroprevalence rates.

Mexico ranks 13th globally and third in the Americas in the total number of HIV cases reported. However, when rated on the accumulated number of cases, Mexico ranks 69th globally, 29th in Latin America and the Caribbean, and in 11th place in the Americas, which reflects a comparatively low incidence rate.

Epidemiological analyses of HIV/AIDS in Mexico are made by classifying patients by age, sex, and method of transmission. In males, the primary source of transmission has been sexual (homosexual and heterosexual) and only secondarily through blood transfusions. In women, the initial source of transmission had been by blood transfusion, but now it is primarily through heterosexual contact. The initial cases of pediatric (children under 15) transmission were also by blood transfusion. However, that has now shifted primarily to perinatal transmission with a few incidences of sexual transmission. Although HIV has been transmitted between family members in a household setting, this type of transmission is very rare. These transmissions are believed to have resulted from contact between skin or mucous membranes and infected blood. To prevent even such rare occurrences, precautions, as described in previously published guidelines, should be taken in all settings

"including the home" to prevent exposures to the blood of persons who are HIV infected, at risk for HIV infection, or whose infection and risk status are unknown.

AIDS has been reported in all states of Mexican Republic. 55% of AIDS cases are concentrated in the Federal District, the State of Mexico, and Jalisco. The majority of cases are in the 25 to 44 age group. Thus, AIDS has become the 2nd cause of death in men and the 6th cause of death in women within this age group.

Global patterns of HIV transmission vary by region and country, and are influenced by culture, values, social conditions, sexual dynamics, and socioeconomic situations. In Mexico, two primary patterns have been reported: (1) an urban pattern, observed primarily in the large cities of Mexican Republic and in the northern border with the United States, where there is a larger percentage of males infected and longer incubation periods (18 months); (2) and a rural pattern, with a higher proportion of females infected and shorter incubation (eight months), which is being observed in the central and southern regions of the country.

Because the dynamics and rate of HIV infection in Mexico are changing, it is more difficult to accurately predict the total number of people infected. The estimated number of persons that are infected by HIV in Mexico ranges between 116,000 to 174,000. This estimate is based on HIV incidence in blood donors, sentinel studies in pregnant women, and seroepidemiological studies in specific subpopulations.

There has also been an increase in seroprevalence rates in heterosexuals, particularly in females who are sex workers and/or sexual partners of HIV-infected males. While the current infection rate in heterosexuals is low, risk factors are present which could increase the rate of infection in this group. For example, there is an increasing trend in HIV seroprevalence in tuberculosis patients.

Injection drug use is unusual in Mexico, with the practice more common in northern border States. Thus, the incidence of HIV infection in injection drug users is primarily concentrated there. Seroprevalence in this group is increasing.

Mexico has 98,000 cases of AIDS, 83% are men and 17% are women. Their ages are between 18 and 44. The average age of infected people is 23. The official Saavedra López who is the leader of CENSIDA said that the majority of HIV/AIDS cases by blood transfusions have decreased and were virtually eliminated because of several transfusions

regulations. Public laboratories have the commission to provide an exact diagnosis and in that way all patients will have an opportunity to receive a good medical treatment (SIDA, 2004).

JUSTIFICATION

Lee (1981, p. 378) explained that in 1981 the first AIDS cases were discovered and in 1983 the human immune deficiency virus was identified as an etiological agent responsible for the illness. HIV is a retrovirus that penetrates the cell's receivers CD4; it is introduced into the blood cells lymphocites T CD4. After being inside the cells, the HIV transfers its ARN to the cells' AND then a new virus is formed, which repeats all the process. Inside, the HIV destroys the cell. As the T CD4 cell has an important function in the immune system, its loss leads to infections which are terrible for humans.

There are other clinical cases, such as pregnant women, and it also represents a challenge to clinical laboratories because a good diagnosis is the only way to identify if the mother and son are infected with AIDS. Not only AIDS is transmitted by sexual relations but also mothers can transmit the virus to their sons (Ibid).

Examination of the blood is central to the diagnosis and management of hematological diseases. In few other cases can the physician make a specific diagnosis and monitor therapy with accessible tissue samples and available methodologies. The blood is examined in order to find abnormalities. Microscopic examination of the blood spread on a glass slide or cover slip yields useful information regarding all the formed elements of the blood (Ibid).

A clinical laboratory is divided into four main areas which are Hematology, Clinical Chemistry, Microbiology and Immunology. Hematology has an important value because it analyses blood abnormalities and blood diseases. With the help of a microscope and other techniques, the people who work in a laboratory can analyze blood cells. They can find if a patient has an anemia and blood abnormalities such as AIDS. Depending on the identification of CD4 T cells, a chemist can interpret and diagnose if the problem exists.

Immunology is the final area which confirms the presence of AIDS but the primary diagnostic tool is detection of antibody via the enzyme-linked immunoessay (ELISA).

It is a test which gives the opportunity to know the presence of HIV by using blood serum collected by standard procedures. Serum must be separated from the red cells of blood promptly to prevent glycolysis. In the majority of South American countries, injecting drug

use and sex between men are the most important routes of HIV transmission. The virus is then passed on to other sexual partners. In Central America, drug use plays a smaller role and most infections appear to be occurring through sexual transmission (both heterosexual and between men).

Unsafe sex among men who have sex with men (MSM) is common across the whole region. HIV prevalence rates among these men are between 10% and 25%. Nearly 60% of the HIV diagnoses to date, in Mexico, can be attributed to unprotected sex between men. Studies in the Andean region have further highlighted the problem. HIV prevalence among MSM in Lima, Peru had been as much as 22%, but now is half that figure. Neighboring Bolivia, however, still has HIV prevalence around 20% in MSM. In Lima, sex between men is widespread; a tenth of men said they had sex with other men and of these, 9 out of 10 said they also had sex with women.

It is estimated that approximately 25% to 33% of men who have sex with men in Central America (not including Panama) also have sex with women. A high proportion of MSM who have sex with women do so without a condom, contributing to the increasing numbers of women becoming infected. A 2006 study showed that condom use among male sex workers in Latin America, particularly in Ecuador and Peru, was not adhered to as consistently as in female sex workers. However, Colombia reports that approximately 90% of sex workers, of both genders, used condoms regularly in 2007.

The spread of HIV through the sharing of drug injecting equipment is still a feature of the epidemic in Latin America, notably in the capitals of Paraguay and Uruguay. However, regionally IDU's appear to account for a smaller number of new infections than shown previously. In Buenos Aires, Argentina, IDUs accounted for only 5% of new infections and this decline is mirrored also in some cities in Brazil.

Hillman (2000, p. 630) says that a chemist has to bring the reagent to room temperature and pipettes into labeled test tubes. Thereafter, he or she has to mix thoroughly and incubate the tube for 10 minutes at room temperature of 37°C. Finally, they have to observe the

presence of agglutination; the lack of it indicates negative presence of HIV. If the test is positive, another test is recommended to confirm the presence of HIV and the laboratory has to use the Western blot test. Data from over 40,000 name-linked sentinel surveillance studies done by CONASIDA throughout Mexico since 1985 reveals seroprevalences from 0.02% (among pregnant women) to 15.6% (among men who have sex with men). Comparisons of seroprevalence data for different risk groups over two five-year periods (1985-1990 and 1991-1995) reveals that HIV incidence in some groups has remained low (eg: female commercial sex workers where seroprevalences range from 0.04% to 2.6%) or even decreased (e.g. blood donors where seroprevalence is now 0.01-0.04%) while it has increased in others (e.g. injectable drug users where limited data from two studies done in Baja California in 1990 and 1995 documented an increase from 1.92% to 9%).

In conclusion, HIV/AIDS is an important health problem in Mexico. The impact of HIV stems both from the total number of infections (5 per 10,000 people) and the fact that it primarily affects those in the most economically productive age group. Mexico has made tremendous strides in controlling the transmission of HIV though blood and blood products but has been far less effective in lowering rates of sexual transmission. New trends in the epidemic that are of particular concern are the increasing number of infections among intravenous drug users and those in rural communities among migrants or sex partners of migrants.

The Hospital General de Chetumal has a clinical laboratory which receives up to 100 patients per day. All of them need blood analysis. The people who work in the laboratory need a valuable tool to do their work and to have updated techniques and texts related to HIV/AIDS.

 Quintana Roo is a state of Mexico, on the eastern part of the Yucatán Peninsula. It borders with the States of Yucatán and Campeche to the north and west, the Caribbean Sea to the east, and the nation of Belize to the south. Quintana Roo also claims territory which gives it a small border with Guatemala in the south west of the state; although this disputed area is also claimed by Campeche.

The capital of Quintana Roo is the city of Chetumal. Quintana Roo also contains the resort city of Cancún, the islands of Cozumel and Isla Mujeres, the towns of Bacalar, Felipe Carrillo Puerto, Playa del Carmen, Puerto Juárez, Akumal, Xcalak, and Puerto Morelos, as well as the ancient Maya ruins of Chacchoben, Chakanbakán, Chamax, Cobá, Dzibanché, El Meco, Ichpaatán, Kohunlich, Muyil, Oxtankah, Tancah, Tulum, Tupak, Xel-Há and Xcaret.

The state covers an area of 50,351 square kilometers (19,440.6 sq mi), and the 2005 census reported a population of 1,135,309. The statewide population is currently expanding at a rapid rate due to the construction of hotels and the demand for workers. Many immigrants to the state come from Yucatán, Campeche, Tabasco, and Veracruz. The state, known as a resort area, is frequently hit by severe hurricanes due to its exposed location.

Chetumal is located in the South-east of the Mexican Republic in the Peninsula of Yucatan. 874,963 people live there and its main economic activity is tourism (CONASIDA, 1999). Every year, thousands of tourists arrive in Quintana Roo; they visit Cancun City, Playa del Carmen, Tulum and Chetumal. According to CONASIDA, the interaction with foreigners is a way to get infected with illnesses, especially AIDS, but it not only happens here in Quintana Roo but also in other cities around the world.

In 1986, México established the National Committee against AIDS. Initially the committee was comprised of professionals who provided their services on a part-time basis to coordinate the fight against AIDS. In August 1988, the National Council for Prevention and Control of AIDS (CONASIDA) was established by presidential decree. CONASIDA became the official government agency charged with the responsibility of meeting the diverse challenges of the HIV/AIDS epidemic in México.

In 1997 an analysis of health-care services and needs was made to help prioritize the primary responsibilities of the Secretary of Health. As a result of this analysis, substantive programs were identified and recommendations were made for new programs at both federal and state levels. Thus, eleven substantive programs were defined, one of which was the Program for HIV/AIDS and other Sexually Transmitted Diseases (STD), which is the direct responsibility of the CONASIDA.

Integration of HIV and STD services was based on recommendations by various federal and state agencies who had been working together to better coordinate activities and services between both programs. CONASIDA is a part of the federal level of the Secretary of Health. Its main function is a normative one of coordination and counseling at national level.

In healthcare institutions for insured populations, persons with AIDS are provided with medical attention, consultations, laboratory, and medications for the prevention and treatment of infections. However, no antiretroviral drugs are provided. The estimated cost of ideal ambulatory care, including triple combination therapy with protease inhibitors for 1997 is $10,197.50 (USD), with 86% of total cost represented by the expense on antiretroviral.

CONASIDA's main activities focus on the following areas:

- Prevention of HIV perinatal transmission, through blood transfusions, injection drug use, and sexual transmission.
- Reduction of the impact of HIV on individuals, families and society.
- Coordination of institutional, interinstitutional, territorial, and intersectorial programs.

CONASIDA's main goals established for the year 2000 were to:

- Reduce by 50% the number of cases of children infected by HIV during pregnancy, delivery, or lactation.
- Reduce HIV transmission through blood transfusion to 0.1%
- Reduce AIDS incidence rate to 2%.
- Increase the use of condoms by 30%.
- Provide timely and appropriate care to 80% of persons infected by HIV and other STDs.
- Eliminate all health sector violations of human rights of persons with HIV.

CONASIDA's recent accomplishments include:

- Creation and/or enhancement of state programs for AIDS prevention and control through the decentralization of the Secretary of Health. Since 1997 the federal entities with programs at the state level were increased by 72%.
- Gradual reduction of HIV transmission through blood transfusion since 1992.

- Implementation of an effective mass media HIV/AIDS educational campaign, with a budget increase of 78% from 1996 to 1997, 56% increase in impact (800,640 vs. 1,247,152), and continuous evaluation of same.

- Introduction of effective HIV/AIDS prevention programs for specific risk groups (migrants, homeless boys/girls, injection drug users, men who have sex with men, pregnant women, adolescents, female sex workers, and long distance drivers).

- Increase in distribution of printed HIV/AIDS educational materials by 157% (309,584 vs. 796,584).

- Coordination of activities with the Public Education Department (SEP).

- Increasing access to antiretroviral drugs to all children under age 15 and HIV-infected pregnant women without social security through FONSIDA A.C. Project.

The Mexican government has to implement new policies in order to promote a good AIDS prevention because it is known that information is the way to decrease AIDS cases but only if the government decides to invest in advertising campaigns. It could work if campaigns start at school by teaching sexual education at first.

My interest in this study emanates from the fact that I work in a clinical laboratory and I take into account the importance of doing a good blood analysis but I have to know updated techniques and texts which are related to this terrible illness.

OBJECTIVE

In Quintana Roo the cases of AIDS have been increasing in the last few years. Consequently, a good diagnosis is needed in order to face this terrible illness. There are texts about AIDS but the majority of these texts are in English. Therefore, the only way to access those texts is by using translation.

This monographic work will try to translate an updated text about AIDS. It will be useful for the people who work in the laboratory of Hospital General de Chetumal and other health-care institutions in order to do a good and optimal diagnosis.

This monographic work pretends to do a good translation of a scientific text which will be useful in the laboratory but also it will be an important chance to use translation techniques. As a matter of fact, translation techniques are important for this work because using only a dictionary and translate word by word is a wrong way to do it; translation techniques are required to do a good work so that they will provide the theory of how to do a translation by avoiding all mistakes which people do without the knowledge of translation techniques. For that reason the second objective of this monographic work is the use of translation techniques to carry out a translation from English into Spanish.

This monographic work pretends to use the techniques of Vinay and Darbelnet (explained in the theoretical framework), for translating texts in a proper way.

Newcomers to translation sometimes proceed as if translation were an exact science — as if consistent, one-to-one correlations existed between the words and phrases of different languages, rendering translations fixed and identically reproducible, much as in cryptography. Such novices may assume that all that is needed to translate a text is to encode and decode equivalents between the two languages, using a translation dictionary as the "codebook".

On the contrary, such a fixed relationship would only exist were a new language synthesized and simultaneously matched to a pre-existing language's scopes of meaning,

etymologies, and lexical niches. If the new language were subsequently to take on a life apart from such cryptographic use, each word would spontaneously begin to assume new shades of meaning and cast off previous associations, thereby vitiating any such artificial synchronization. Henceforth translation would require the disciplines described in this article.

Another common misconception is that anyone who can speak a second language will make a good translator. In the translation community, it is generally accepted that the best translations are produced by persons who are translating into their own native languages as it is rare for someone who has learned a second language to have total fluency in that language. A good translator understands the source language well, has specific experience in the subject matter of the text, and is a good writer in the target language. Moreover, he is not only bilingual but bicultural.

THEORETICAL FRAMEWORK

Fawcett (1997, p. 45) explained that translation text analysis should not only ensure full comprehension and correct interpretation of the text or explain its linguistic and textual structures and their relationship with the system and norms of the source language (SL).
 It should also provide a reliable foundation for each and every decision which the translator has to make in a particular translation process.

What is needed is a model of source-text analysis which is applicable to all texts types. Such a model should permit translators to understand the function of the elements in the content and structure of the source text. Translation typically has been used to transfer written or spoken SL texts to equivalent written or spoken TL texts. In general, the purpose of translation is to reproduce various kinds of texts—including religious, literary, scientific, and philosophical texts—in another language and thus making them available to a greater number of readers.

If language were just a classification for a set of general or universal concepts, it would be easy to translate from an SL to a TL; furthermore, under these circumstances the process of learning an L2 would be much easier than it actually is. In this regard, Culler (1976, p.103) believes that languages are not nomenclatures and the concepts of one language may differ radically from those of another, since each language articulates or organizes the world differently, and languages do not simply name categories; they articulate their own. The conclusion likely to be drawn from what Culler (1976, p.233) writes is that one of the troublesome problems of translation is the disparity among languages. The bigger the gap between the SL and the TL, the more difficult the transfer of message from the former to the latter will be.

The differences between an SL and a TL and the variation in their cultures make the process of translating a real challenge. Among the problematic factors involved in translation such as form, meaning, style, proverbs, idioms, etc., the present paper is going to concentrate mainly on the procedures of translating culture-specific concepts (CSCs) in

general and on the strategies of rendering allusions in particular. Although some stylists consider translation "sprinkled with footnotes" undesirable, their uses can assist the TT readers to make better judgment of the ST contents. In general, it seems that the procedures 'functional equivalent' and 'notes' would have a higher potential for conveying the concepts underlying the CSCs embedded in a text; moreover, it can be claimed that a combination of these strategies would result in a more accurate understanding of the CSCs than other procedures.

Various strategies opted for by translators in rendering allusions seem to play a crucial role in recognition and perception of connotations carried by them. If a novice translator renders a literary text without paying adequate attention to the allusions, the connotations are likely not to be transferred as a result of the translator's failure to acknowledge them. They will be entirely lost to the majority of the TL readers; consequently, the translation will be ineffective.

It seems necessary for an acceptable translation to produce the same effects on the TT readers as those created by the original work on its readers. This paper may show that a translator does not appear to be successful in his challenging task of efficiently rendering the CSCs and proper names (PNs) when he sacrifices, or at least minimizes, the effect of allusions in favor of preserving graphical or lexical forms of source language PNs. In other words, a competent translator will advise not to deprive the TL reader of enjoying, or even recognizing, the allusions either in the name of fidelity or brevity.

It can be claimed that the best translation method seem to be the one which allows translator to utilize 'notes.' Furthermore, employing 'notes' in the translation, both as a translation strategy and a translation procedure, seems to be indispensable so that the foreign language readership could benefit from the text as much as the ST readers do.

A really troublesome area in the field of translation appears to be the occurrence of allusions, which seem to be culture-specific portions of a SL. All kinds of allusions, especially cultural and historical allusions, bestow a specific density on the original language and need to be explicated in the translation to bring forth the richness of the SL text for the TL audience. What is translating? We define it as the process of reading,

understanding, interpreting, rephrasing and delivering an original message—with all of its subtlety and impact—to a new audience, in its mother tongue.

The model should be general enough to be applicable to any text and specific enough to take account of as many general translation problems as possible.

A translator's basic responsibility is to be true to the original text. Some of the most important theories about translation are repeated hereunder:

Newmark (1988, p. 213) mentions the difference between translation methods and translation procedures.

He writes that while translation methods relate to whole texts, translation procedures are used for sentences and the smaller units of language. He goes on to refer the following methods of translation (Ibid):

- Word for word translation: in which the SL word is preserved and the words translated singly by their most common meanings, out of context.

- Literal translation: in which the SL grammatical constructions are converted to their nearest TL equivalents, but the lexical words are again translated singly, out of context.

- Faithful translation: it attempts to produce the precise contextual meaning of the original within the constraints of the TL grammatical structures.

- Semantic translation: This differs from faithful translation because it must take more into account the aesthetic value of the SL text.

- Adaptation: it is the freest form of translation and it is used mainly for plays and poetry; the themes, characters, plots are usually preserved, the SL culture is converted to the TL culture and the text is rewritten.

- Free translation: it produces the TL text without the style, form, or content of the original.

- Idiomatic translation: it reproduces the message of the original but tends to change meaning by preferring colloquialisms and idioms where these do not exist in the original.

- Communicative translation: it attempts to render the exact contextual meaning of the original in such a way that both content and language are readily acceptable and comprehensible to readers.

Graedler (2000, p. 309) puts forth some procedures for translating culture-specific concepts. He suggests:

- Making up a new word.

- Explaining the meaning of the SL expression in lieu of translating it.

- Preserving the SL term intact.

- Opting for a word in the TL which seems similar to or has the same relevance as the SL term.

And last but not least, Vinay and Darbelnet (1996, p. 49) explained their methods of translation. During my time at school teachers taught me their methods, and so I consider their concepts as interesting to be used in my monographic work.

Vianay and Darbelnet have 7 methods divided into the two main areas: Direct and Oblique translation. The first three are direct and the last four are oblique:

*Borrowing: Vinay and Barbelnet (1996, p. 53) define it as a word which is taken from the SL without translating it. The foreign word remains without any change but it could suffer a phonetic and morphologic adaptation.

Examples Bassnet (2002, p. 101):
-Diskette
Disquete
-Format
Formatear
-Reset
Resetear

* Calque: Bassnet (2002, p. 103) says that it is a kind of borrowing where the syntax is borrowed from the source language but all its elements are translated in a literal way. Calque is a translation procedure where a translator translates an expression or a word literally into the target language, translating the elements of the expression word for word.

Example (Ibid):
-Weekend

Fin de semana

-Science-fiction

Ciencia ficción

Calque is one way to avoid the introduction of foreign words and to build a new word as for example:

- Football

  Fútbol

*Literal translation: Bassnet (2002, p. 210) explained that according to Vinay and Darbelnet, it is the rendering of text from one language to another word for word rather than conveying the sense of the original. Literal translation commonly mis-translates idioms. Sometimes the translator has to do some changes in order to adequate the message from the original expression.

Example (Ibid):

-Plant species

  Especies vegetales

-We can expect to discover

  Cabe esperar que descubramos.

* Transposition: Fawcett (1997, p. 889) explained that it consists of a modification of the grammatical category of a sentence without modifying its general sense. It is the substitution of a message's part but the sense does not change.

Example (Ibid):

-After they left

-Tras su partida

* Modulation: Fawcett (1997, p.67) says that it consists of a message's variation which is obtained by changing the perspective or point of view. Modulation can be obligatory or optional.

Example (Ibid):

-To pull one's leg

Tomar el pelo a alguien

-Raw material

Materias primas.

-He was entrusted.

Se le encomendó.

* Equivalence: Fawcett (1997, p. 739) explained that it tries to transmit a similar situation by using stylistics and structural resources, but all of them are different. Then these two units are considered to be equivalent. The domain of equivalence covers linguistic units such as morphemes, phrases, clauses, idioms and proverbs. It is equal in value, measure, force, effect and significance.

Example (Ibid):

-Excuse me

-Permiso

-You are welcome.

De nada.

-Men at work.

Obras

* Adaptation: Bassnet (2002, p. 99) explained that it is also known as free translation. It is a translation procedure where the translator replaces a social or cultural reality in the source text with a corresponding reality in the TL text; this new reality would be more usual to the people of the TL text. Usually an adaptation tries to find an equivalence of the message in order to find the same meaning.

Example (Ibid):

-The end is at hand.

El final esta cerca.

-He kissed his daughter on the mouth.

Abrazó tiernamente a su hija.

The techniques of Vinay and Darbelnet will be used in this work because they are more flexible than others. How to use their techniques will be explained in the methodology.

METHODOLOGY

As we all know, good translation is not usually just a question of translating each word of the source text into the target language. Nor does it consist, however, of the translator skimming through the source text, putting it aside and then jutting down the general idea of it in his or her own words in the target language (Intercultural Studies, 2007).

The text on Acquired Immune Deficiency Syndrome was chosen because it is a terrible illness. Here in Chetumal our local health system has poor information about AIDS. So, the main reason for translating the text is to provide recent data about AIDS to the Clinical Laboratory of Hospital General.

As we already know, Vinay and Darbelnet have specified translation techniques but this monographic work did not use all of them because only a few of them were useful and applicable in this translation. The most important translation techniques were analyzed and used in order to do this work.

   A good reading of the text was needed before starting a translation, in order to analyze the main ideas and to find the possible problems that the text can bring up.

Through the reading, notes were needed which are an aid to support the work. The main use of note is to relieve memory. Moreover, through notes the translator can reproduce the content and structures of the sentences, stressing the main ideas, the secondary elements and the relations among them.

Other important data are dates and proper names, in this case scientific names.

Thereafter, the text was read again in order to find the scientific or technical words because in order to find the meaning of new words, technical dictionaries were required. I would like to point out that in some cases the researcher needed help from a chemist because sometimes the translation required orientation and only the people specialized in this theme could help me because in scientific texts some words need a specific explanation in order to be translated. A translator's basic responsibility is to be "true" to the original text. If we consider a translation to be a form of intercultural message, then we should evaluate its faithfulness to the original on two counts: how the original message is expressed in the target language, and how it is received by the target audience. The translator is responsible for both of these steps in the communication process.

During the translation I needed books on hematology, clinical hematology, chemistry and all kind of sources related to AIDS.

And finally, by using the techniques of Jean Paul Vinay and Jean Darbelnet, I translated the whole text. I chose their techniques because from my point of view they have one of the most complete techniques in translation and because during my major, I worked with their techniques.

The translation work needed different drafts in order to avoid and correct all possible mistakes and difficult vocabulary.

The first draft was written and it provided the first view of the whole work. With the help of dictionaries and the guide of a chemist the difficult vocabulary was translated (for example vocabulary related to AIDS), and it was necessary to consult specialized dictionaries as well. A second draft was required because during the translation there were vocabulary and sentences which needed more work.

The third draft was more complete; the mistakes were fewer and the translation was done but it demanded a deep research in order to avoid mistakes. And finally, one last draft where the translation took its final shape.

At the end of this monographic work some examples of each technique were cited, analyzed and explained in detail. This work included the analysis of translation techniques and some possible recommendations to translators in order to improve their future translations.

Aproximadamente en la misma época en que el SIDA fue reconocido en 1981, se dieron reportes de un síndrome de inmunodeficiencia similar, caracterizado por infecciones despiadadas y debilitantes, misma que fue descrita en muchas colonias de macacos ubicados en los centros para primates de los Estados Unidos. La enfermedad, conocida como síndrome de inmunodeficiencia primate (SIP), fue asociada con la infección por un retrovirus, denominado virus de inmunodeficiencia primate (VIP). Pruebas subsecuentes revelaron que mas del 20% de los monos verdes africanos sintomáticos silvestres llamados mangabeys tuvieron evidencia serológica de la infección de VIP.

La infección de la cepa viral de VIP de estos monos es relacionada al VIH-2, una cepa menos virulenta que el virus de inmunodeficiencia humana, encontrada primeramente en África del oeste. Un virus de inmunodeficiencia relacionada al VIH-1 que infecta a los chimpancés Africanos fue también identificado. Evidencia reciente sugiere que la subespecie del chimpancé Pan troglodita puede haber sido el huésped original del VIH-1.

Bajo estas observaciones, se ha postulado que originalmente el VIH pudo haber sido transmitido a los humanos por una especie de primate Africano. A mediados de los 60´s, circunstancias políticas y sociales empezaron a cambiar drásticamente de manera que facilitaron la rápida propagación de esta enfermedad hacia los humanos. Los movimientos aislados previos de gente africana de aldeas rurales hacia grandes centros urbanos; cambios en hábitos sexuales, resultado de una exposición generalizada del incremento de parejas sexuales. La epidemia mundial del abuso de drogas, y el advenimiento del comercio aéreo contribuyó a la epidemia actual de la infección por VIH.

La Organización Mundial para la Salud es quien ha estimado que más de 30 millones de personas a nivel mundial hayan sido infectadas por el VIH a mediados de 1998, la mayoría infectada por contacto heterosexual, mientras que el contacto homosexual y el uso de drogas son los modos predominantes de transmisión en los Estados Unidos y Europa oriental. La transmisión vertical de madre infectada a hijo ahora está disminuyendo en países desarrollados, aunque la transmisión continua incrementándose en regiones pobres en recursos del mundo.

Etiología y Patogénesis

Virus de Inmunodeficiencia humana-1
El VIH-1 es un integrante de la subfamilia de retrovirus lentivirinae de los primates. El ARN de los virus induce a una infección crónica celular convirtiendo su genoma del ARN en un provirus del ADN que es integrado dentro del genoma de la célula infectada. La infección por estos lentivirus es caracterizada por largos periodos de latencia seguidos por un comienzo gradual de los síntomas relacionados a la enfermedad.

Transmisión del VIH

El VIH puede ser transmitido por contacto sexual con una pareja infectada, por el uso de drogas con agujas contaminadas por exposición a sangre infectada o derivados de sangre, y por exposición perinatal de la madre infectada hacia el recién nacido.

Mecanismos generales de transmisión sexual

El VIH-1 ha sido aislado del semen de hombres infectados por VIH-1 así como también de fluido seminal sin células y podría ser detectado durante las primeras 3 o 4 semanas después de la infección primaria. Factores asociados con el incremento de la carga viral en el semen incluyen un avance en la sintomatologia de la enfermedad por VIH, altos niveles de ARN del VIH en la sangre, conteo de células CD4 menor a 200/mcl, y la presencia de leucocitos en el fluido seminal. La infección por VIH ha sido reportada después de la exposición al semen infectado durante la inseminación artificial.

El VIH ha sido obtenido de secreciones vaginales y cervicales de mujeres infectadas por VIH, y células endoteliales y macrófagos infectados por VIH han sido detectados en biopsias cervicales. Factores que incluyen los niveles de VIH-1 en secreciones del tracto genital femenino incluyen los niveles de la enfermedad por VIH; el estatus menstrual, parámetros hormonales, infecciones vaginales, edad, los niveles de ARN del VIH-1 en plasma, y la terapia antiviral. Aunque también se ha reportado transmisión de VIH de mujer a mujer, esto parece ser relativamente inusual. La transmisión del VIH puede ser facilitada por la presencia de otras enfermedades de transmisión sexual, ambas con o sin ulceración, y el VIH ha sido aislado directamente de las ulceras genitales. La prevención o tratamiento de las enfermedades transmitidas sexualmente han sido asociadas con una disminución en la transmisión de VIH.

Transmisión a través del uso de drogas.

El intercambio de agujas y jeringas es una forma importante de transmisión entre los que usan drogas. El uso de la cocaína ha sido asociada con un alto riesgo de infecciòn particularmente de VIH, presuntamente relacionado a su corta media vida y la consiguiente necesidad de un mayor número de inyecciones. Factores de comportamiento pueden llevar al riesgo de incrementar la transmisión del VIH incluso entre usuarios de drogas no ilícitas.

Transmisión a través de productos de sangre infectada.

El riesgo de infección con VIH después de recibir una unidad de sangre infectada es de aproximadamente un 90 %. La transfusión de productos de sangre derivado de múltiples unidades de sangre combinada puede también transmitir el VIH y fue la causa inicial de un alto nivel de prevalencia de infección por VIH entre pacientes con hemofilia. Examinando toda la sangre donada, empezando en marzo de 1985, y el subsecuente tratamiento rutinario a base de calor o de solvente detergente de concentrados de factores de coagulación, el resultado es una marcada disminución en las nuevas infecciones asociadas al VIH en las transfusiones.

Manuales para una apropiada desactivación del VIH en las concentraciones de los factores de coagulación han sido desarrollados. Actualmente, el riesgo de adquirir VIH a través de

la recepción de una unidad de sangre que se considera como negativa por anticuerpos de VIH-1 es de aproximadamente de 1 entre 493,000.

Transmisión de madre a hijo.

El riesgo de infección de madre a hijo difiere en varias partes del mundo con rangos de aproximadamente 15% en Europa a 15-30% en los Estados Unidos y de 40-50% en África. El VIH-1 podría ser transmitido en el útero, intraparto(al tiempo de nacimiento), o post parto, a través de la ingesta de la leche materna infectada por VIH-1.

Muchos factores pronostican un gran riesgo de transmisión perinatal. En términos de la madre, una enfermedad por VIH más avanzada, altos niveles de VIH-1en las cargas virales del plasma, uso del cigarro, y adictos activos a la inyección de drogas han sido todos asociados con un incremento de contagio. En términos de los detalles del parto, la ruptura prematura de las membranas amnióticas (más de 4 horas), la presencia de chorioamnionitis y parto vaginal, paralelamente a la sección de cesárea, han sido cada una asociada al incremento de las tasas de transmisión. En cuanto al infante, la alimentación por pecho, prematuridad, y bajo tiempo de gestación son reportados como factores de riesgo. La CDC recientemente ha hecho recomendaciones para el óptimo manejo de mujeres embarazadas infectadas por VIH-1.

Estas recomendaciones difieren para ambientes ricos o pobres en recursos.

En los Estados Unidos, el uso de agentes antiretrovirales durante el embarazo y parto, con una subsecuente administración al infante en las primeras 6 semanas de vida, ha dado como resultado una dramática reducción en el rango de transmisión, de aproximadamente de un 25% a 8%. Con la promoción del uso de cesárea optativa y evitando la lactancia por medio de pecho materno, los rangos de transmisión ha descendido hasta aproximadamente un 2%.

Se han demostrado la eficacia de tratamientos cortos con zidovudine o neviripine que es un inhibidor de la transcriptasa reversa no nucleotidasa y podrían ser más factibles en en regiones de escasos recursos del mundo.

La toxicidad a largo plazo en el útero expuesto a agentes antiretrovirales es desconocida. No obstante, su uso durante el embarazo resulta en una disminución de 43% en el número de niños infectados por VIH vía perinatal en los Estados Unidos comparando información de 1992 a 1996.

El ciclo de vida del VIH-1

El gp120 del VIH-1 se enlaza a la superficie protéica de la membrana del CD4, que resulta en otro vínculo de alta afinidad con el receptor quimiotinico CCR5. El inductor ayudante humano, los linfocitos CD4, los monocitos-macrófagos, las células de Langerhans, las células dentríticas foliculares, los megacariocitos, y las células tìmicas expresan el CD4 y las moléculas receptoras de la quimiokina, y son susceptibles a infectarse por el VIH-1. La diversidad estructural del receptor viral gp120 ha resultado en flujos virales con selectivos

o restringidos patrones de infección, como aquellos que fácilmente infectan a los monocitos, mientras otros son trópicos para los linfocitos CD4.

Macrófagos trópicos cepas de VIH usan la quimiotina receptora para infectar a ambos macrófagos y linfocitos CD4. Las cepas T-trópicas usan la quimiotina receptora CXC4 y podrían también usar el receptor CCR5. Quimiotina receptora adicional CCR2 y CCR3 han estado implicados como coreceptoras para la infección de VIH de ciertos tipos celulares.

Sobre la unión a la proteína CD4 de la célula huésped, la envolvente del virus se fusiona con la membrana celular huésped. Esta fusión es mediada por un dominio hidrofóbico en la porción terminal amino del go41. El internalizado nucleocàpsida luego es desestabilizado y disociado después del enlace a la proteína celular cicloferon "A", exponiendo el diploide viral del genoma ARN asociado con la transcriptasa reversa. La transcriptasa reversa es precedida por la síntesis de una sola cadena de ADN, seguido por la degradación del ARN viral y por la ribonucleasa (h) activa de p66. La transcriptasa reversa luego actúa como una polimerasa ADN formando una segunda cadena de ADN. Esta síntesis de doble cadena del pro virus ADN debe proceder rápidamente para prevenir la degradación del ARN viral por enzimas intracelulares. La tasa estimada de errores de la substitución base por la transcriptasa reversa del VIH podría ser tan elevada como de 1 en 1700 o de 1 en 2000. Esto resulta en 5 de 10 mutaciones nucleósidas por virus para cada replicación del ciclo y explica el alto grado de la diversidad genómica observada entre los virus aislados del VIH.

La integración del pro virus es necesaria para la infección estable de la célula. La integrasa viral es capaz de ambas divisiones del ADN huésped y de la integración de una forma lineal del pro virus. Estudios cinéticos de la infección del VIH-1 han detectado ADN viral presente en el citoplasma dentro de 2 a 3 horas de infección mientras el ADN viral nuclear ha sido detectado por 24 horas. El producto genético del gen VPR aparece para asistir en el transporte de la integración del ADN viral dentro del núcleo para la integración subsecuente. Después de la exitosa integración del genoma viral, la célula infectada de VIH-1 podría desarrollar o bien una latente forma de infección u otra persistente.

El mecanismo o mecanismos de la latencia viral yacen pobremente entendidos pero aparece requerir una activación de la célula infectada, ya que el VIH-1 no se replica eficientemente en el resto de los linfocitos o macrófagos. Proteínas celulares transactivadoras como NF-kB, son mas reguladas en células activadas e incrementan la transcripción pro viral del VIH.

Después de la integración, la transcripción pro viral del VIH-1 dirige a la expresión de proteinas reguladoras designadas tat, rev, y nef. Tat es una proteína pequeña nuclear que es esencial para la replicación del VIH y en conjunción con otras proteínas celulares, TAK (Tat-quinasa asociada) y CycT (T ciclina), ayuda al alargamiento del ARN viral, resultando en un incremento de pliegues de 1000 en la expresión del VIH-1 por las células infectadas.

REV es una proteína viral que regula el intercambio nuclear de la desunión del ARN viral. Como el tat, el rev es esencial para la duplicación viral y debe unir a un elemento rev receptivo localizado en el gen ENV. Las otras proteínas del VIH que codifican son

designadas nef y vpv, que tienen una función en la modulación y en bajar la regulaciòn de la célula receptora, CD4.

Las proteínas estructurales de los genes GAG, POL y ENV son expresadas como proteínas precursoras y subsecuentemente divididas por la proteasa viral para proporcionar una maduración a las proteínas virales. La proteólisis de proteínas por la proteasa viral es esencial para la maduración viral y la inefectividad.

Los productos génicos ENV, gp120 y go41, son transportados a la membrana celular. El centro de la ribonucleoproteina se ensambla en el citoplasma de la célula huésped y subsecuentemente se mueve a la superficie de la membrana para su desarrollo.

La envolvente eficiente del ARN viral es dependiente de los signos de la envolvente presente en la región GAG del ARN viral. El desarrollo del virus parece ser dependiente del producto del gen VPU, el cual asiste en el transporte membranal del gen ENV. Por otro lado, la inefectividad viral aparece requerir el producto génico del gen VIF.

Patogénesis de la infección por VIH.

La infección por VIH resulta en una defectuosa regulación inmune y en inmunodeficiencia. Los numerosos defectos in vitro y in vivo en la respuesta inmune celular observada con la infección por VIH incluyen descenso en la proliferación de linfocitos a antígenos solubles, descenso en la respuesta de ayuda de la síntesis de inmunoglobulina (Ig), retrasando la hipersensibilidad defectuosa, descenso en la producción del interferon-v, y descenso de la citotoxicidad mediada por la célula T de las células virales infectadas.

Disminución de las células CD4+.

La infección con VIH-1 da una pérdida progresiva de (CD4+) linfocito T, resultado de un efecto citopatico directo de VIH sobre los linfocitos CD4+. La formación sincitial de células gigantes multinucledas por mecanismos de fusión envolvente por células infectadas que expresan un gp120 viral con linfocitos T-CD4+ no infectados es otro mecanismo de la disminución de CD4+. La tendencia a ciertas manchas virales para formar sincitio parece ser asociada con un cuadro clínico agresivo. Más información experimental reciente sugiere que un interruptor fenotípico de VIH-1 de un virus M-trópico no-sincitial a un T-trópico sincitial podría ser el evento central en la aceleración de la inmunodisminuciòn inducida por VIH.

La respuesta inmunológica del huésped en contra de linfocitos infectados por VIH también podría contribuir a una pérdida progresiva de linfocitos CD4+ por anticuerpos mediáticos y mecanismos citotóxicos de células T mediáticas. Los linfocitos no infectados podrían también convertirse en espectadores inocentes, objetivos de la destrucción inmunológica debido a la conexión del libre gp120 con su superficie proteica CD4.

La producción defectuosa de citoquimia inmunoestimulatoria, interleucina-2 (IL-2), o la expresión exagerada de inhibidores de la proliferación de linfocitos-T, como el crecimiento transformante del factor-beta (TAG-beta), puede contribuir a la declinación progresiva en

linfocitos CD4. Altos niveles de replica y desarrollo del virus, resultado de una lesión membránica, también ha sido propuesta como un mecanismo para la citotoxicidad de linfocitos.

Avances recientes en la terapia de combinación antirretroviral han resultado en una marcada supresión de replica viral, con marcadas reducciones de reservorios en sangre y tejidos. Una eficiente supresión viral ha resultado en la reconstitución inmunológica prolongada caracterizada por un incremento en el número de linfocitos CD4+, reducción de infecciones temporales y una supervivencia prolongada. No obstante, déficits significantes en el persistente reservorio inmunológico, y la reconstitución inmunológica completa no han sido alcanzados.

Diagnostico de la infección por VIH.

Como las manifestaciones agudas (primaria) de infección por VIH (síndrome retroviral agudo), los resultados de los exámenes de laboratorio no son específicos, con una frecuente elevación de los niveles de transaminasas del hígado y los rangos de sedimentación eritrocitaria. No obstante, la viremia por VIH se presenta durante enfermedades severas y pueden ser detectadas por métodos moleculares como por ejemplo transcripción reversa en cadena de la polimerasa (RT-PCR).

El examen para el diagnostico primario es la detección de anticuerpos vía ensayo inmuno absorbente ligado a la enzima (ELISA). Sin embargo, un resultado de ELISA positivo podría no ser especifico para una infección por VIH-1, todos los resultados testigo del examen de ELISA deben de ser verificados por antígenos de VIH-1 (immunoblotting).

Por las técnicas de ELISA e immunoblot, el tiempo medio de la infección inicial a la primera detección de anticuerpos del VIH ha sido estimado a ser de 2.4 meses, mientras el 95% de los casos son esperados a seroconvertirse entre 5.8 meses. La infección por VIH por más de 6 meses sin presentar anticuerpos detectables es extremadamente rara.

La presencia del antígeno p24 o el ARN del VIH en suero o plasma podrían preceder una seroconversión por muchas semanas. Esta alza inicial en el antígeno p24 presenta una correlación con el arranque de una viremia que ocurre poco después de la infección primaria por VIH. A pesar de estas observaciones, el examen antígeno p24 de unidades de sangre donada parece no proveer beneficio con respecto a las técnicas convencionales de ELISA e immunoblot.

Características de laboratorio en el progreso de la enfermedad.

Con el progreso de la infección inicial aguda hasta el periodo asintomático esperado, varios parámetros de laboratorio podrían ser usados para predecir del desarrollo de enfermedades más avanzadas. La cuantificación de ARN del VIH en plasma (carga viral) y conteo de linfocitos CD4+ decaen durante la infección retroviral aguda y luego se estabiliza durante la primera infección asintomática que podría parecer relativamente normal. El conteo de

CD4+ luego decrece aproximadamente de 40 a 80 mcl/año en la ausencia de medicación retroviral entre los pacientes.

Una medida inicial de carga viral en plasma por los métodos RT-PCR o ADN ramificado (bADN) provee información pronostica importante que puede ser útil para determinar cuándo iniciar las medicaciones con anti retrovirales. La valoración serial de la carga viral de VIH en plasma también permite la valoración rápida y eficaz de medicación anti retroviral. Cambios en la carga viral usualmente preceden alteraciones importantes en el conteo de linfocitos CD4+.

Varios marcadores no específicos de la enfermedad en progreso han sido definidos, incluyendo la microglobulina beta, y la neopterina, cada una de las cuales tienen un valor predictivo independiente en estimar la probabilidad de progresión a SIDA. Sin embrago, cada uno de estos marcadores substitutos han sido largamente remplazados por más exámenes moleculares específicos para cuantificar la carga viral del VIH en plasma.

Anemia por el descenso en la producción de glóbulos rojos.

Un descenso en la producción de glóbulos rojos, podría resultar de factores que disminuyen el CFU-GEMM, como la inflamación de citopenia o por el mismo virus del VIH. Sin embargo, una atenuada producción de eritropoyetina ha sido documentada en pacientes anémicos infectados por VIH, similar a la disminución vista en otros estados de la infección crónica o inflamación.

La infiltración de la medula ósea por tumor, como el linfoma o infección por Micobacteria avium compleja (MAC), podría también encabezar la disminución en la producción de glóbulos rojos. Más aun, MAC podría también ser asociado con citopenia inducida por la supresión de la medula. Envolventes del tracto gastrointestinal (TG) por varias infecciones o tumores podrían encabezar una perdida crónica de glóbulos con una eventual anemia por deficiencia de hierro.

Otra causa prominente de anemia hipoproliferativa en pacientes con infección por VIH es el uso común de medicaciones múltiples, muchas de las cuales podrían causar disminución de glóbulos rojos en la medula.

Zidovudine (AZT), la primera licencia del agente antirretroviral, es uniformemente asociada con el volumen corpuscular medio (MCV mayor a 100) la cual puede ser usada como una indicación objetiva que el paciente ha tomado esta medicación. Es relevante que la anemia dependiente de la transfusión (hemoglobina menor a 8.5 g/dl) ha sido reportada en aproximadamente 30% de los pacientes que han desarrollado la enfermedad del SIDA, que reciben Zidovudine en dosis de 600 mg/día. No obstante, la incidencia de anemia severa es solo el 1% cuando la misma dosis de Zidovudine es usada en pacientes con la enfermedad asintomática de VIH.

La infección de la médula por parvovirus B19 es otra causa de anemia hipo proliferativa en pacientes infectados por VIH, resultando en una infección especifica de los pronormoblastos. Por consiguiente, mientras la falla de la médula que afecta todas las tres

líneas ha sido descrita en asociación con la infección por parvovirus B19, una aplasia pura de glóbulos rojos es la consecuencia usual. La infección por parvovirus es adquirida usualmente durante la niñez, encabezando la "quinta enfermedad", una de las enfermedades exantemáticas propias de la infancia. La exposición al virus encabeza a una infección continua. Aproximadamente el 85% de adultos tienen evidencia serológica de una infección previa al parvovirus. Sin embargo, la sero prevalencia de los anticuerpos entre los pacientes infectados por VIH es solo del 64%. Esto podría sugerir que estos individuos podrían tener una inefectiva respuesta inmune en contra de nuevas infecciones adquiridas. El diagnostico de parvovirus B19 puede ser realizado por exanimación de la médula, revelando pronormoblastos grandes con basofilia difusa y crear vacuolas citoplasmáticas claras; el diagnostico puede ser confirmado por hibridización in situ usando pruebas de secuencia especifica de ADN para parvovirus B19. La terapia para aplasia de parvovirus inducida por glóbulos rojos consiste en inyección de gamma globulina intravenosa (IV) que contiene anticuerpos de plasma de donadores que han sido expuestos al parvovirus. La recaída en aplasia de parvovirus B19 inducida por glóbulos rojos podría ocurrir, necesitando un nuevo tratamiento en estos individuos.

Anemia debido al incremento en la destrucción de glóbulos rojos (Anemia Hemolítica).

El incremento en la destrucción de glóbulos rojos podría ser vista en pacientes infectados por VIH con deficiencia de G-6-PD que son expuestos a drogas oxidantes y en pacientes infectados por VIH con coagulación intravascular diseminada (CID) o Purpura Trombocitopenia trombotica (PTT); la presencia de glóbulos rojos fragmentados y trombocitopenia en citología sanguínea será vista en las últimas dos condiciones, y los cuerpos de Heinz serán vistos asociados con deficiencia de G-6-PD. El síndrome hemofagocítico ha sido también descrito en asociación con la infección por VIH. Una causa adicional de destrucción de glóbulos rojos en pacientes infectados por VIH es el desarrollo de auto anticuerpos, con resultante positivo del análisis de Coombs y la corta vida de los glóbulos rojos. Es interesante notar que un resultado positivo del Coombs directo ha sido reportado en 18 a 77% de los pacientes infectados por VIH, aunque la incidencia de la hemolisis actual es baja. Cuando se presenta anticuerpos anti-i y anticuerpos contra antígenos auto-U han sido descritos ocurriendo en un 64% y 32% en los pacientes infectados por VIH respectivamente. Una alta incidencia en los resultados positivos del análisis en el Coombs directo ha sido también detectada en pacientes con otros estados hipergammaglobulinemico, indicando que el resultado positivo en el análisis del Coombs directo en el VIH podría simplemente ser secundario a la hipergammaglobulimenia policlonal que se sabe que ocurre en el marco de la infección por VIH.

Anemia debido a una inefectiva producción de glóbulos rojos (deficiencia de B12 y Acido fólico).

El acido fólico es absorbido en el yeyuno y es responsable de la transferencia de carbono requerida en la síntesis de ADN. Una deficiencia de acido fólico lleva a una anemia megaloblastica, con ovalocitos en la sangre, neutrofilos hipersegmentados, y un descenso en todas las tres líneas, con anemia resultante, neutropenia y trombocitopenia. Ya que las reservas del tejido de folato son relativamente pequeñas, una deficiencia de folato de tan sólo de 6 a 7 meses podría transformarse en anemia. Es así aparente que pacientes infectados por VIH los cuales estén enfermos y no comen apropiadamente, así como aquellos con una marcada enfermedad del yeyuno, podrían no ser capaces de absorber suficiente acido fólico. Los cambios clásicos de anemia megaloblastica serán detectados con el análisis de la medula ósea, mientras los niveles de folato en suero y glóbulos rojos serán bajos.

Una inefectiva producción de glóbulos rojos, con pancitopenia en la sangre, niveles elevados de bilirrubina indirecta y bajo conteo de reticulocitos podrían también ser vistos en deficiencia de vitamina B12.

La absorción de B12 requiere de una producción inicial de factor intrinsico por células parietales en el estomago, con absorción subsecuente del complejo B12 y el factor intrínseco dentro del Ileon. De este modo, la mal absorción de B12 puede ocurrir en varios desordenes de el estomago (Aclorhidria), por la producción de anticuerpos para el factor intrínseco (anemia perniciosa), o por varios desordenes del intestino delgado y el Ileon (infección o enfermedad de Crohn). Mientras la deficiencia de B12 es altamente imposible en una sola dieta base, pacientes con infección por VIH parecen ser propensos a una mala absorción de B12, presumiblemente debido a un sinnúmero de infecciones y otros desordenes que podrían ocurrir en el intestino delgado.

El balance negativo de vitamina B12 ha sido documentado en aproximadamente un tercio de los pacientes con SIDA, y la mayoría demuestra una defectuosa absorción de la vitamina. Un diagnóstico de deficiencia de B12 puede ser realizado por documentación de niveles bajos de suero B12, mientras la primera indicación del balance negativo de B12 es el descubrimiento de bajos niveles de B12 en sangre los pacientes tratados con transcobalamina II.

La administración mensual de B12 parenteral corregirá la anemia por deficiencia y la pancitopenia en la sangre periférica. Ya que la deficiencia de B12 podría también causar disfunción neurológica (degeneración sub aguda combinada de los tendones), con disfunción motora, sensorial, y cortical alta, la probabilidad de deficiencia de vitamina B12 debe también ser considerada en pacientes infectados por VIH con estos síntomas neurológicos.

Factores de riesgo por infección en pacientes neutropénicos con VIH.

En pacientes con cáncer que recibieron quimioterapia, estudios múltiples han mostrado que el riesgo de infección por bacterias se eleva cuando los conteos absolutos de neutrófilos (CAN) bajan a menos de 1000 células/dl y se elevan otra vez cuando el CAN cae por debajo de 500 células/mcl. Muchos estudios han confirmado la misma relación en pacientes con la infección VIH. De este modo, Moore y colaboradores encontraron que el riesgo de una infección bacterial incrementaba 2.3 veces para pacientes infectados por VIH con menos de 1000 células/mcl y aumentaba 7.9 veces en aquellos con niveles CAN menores a 500 células/mcl. Conteos menores de CAN son asociados con un riesgo incrementado de hospitalización por las serias infecciones en pacientes con VIH, como fue demostrado por un análisis de 2047 pacientes positivos a VIH. En un análisis multivariado, se descubrió que la severidad y duración de la neutropenia eran importantes elementos en la incidencia de la hospitalización por infecciones bacteriales serias.

En un estudio reciente de 62 pacientes infectados por VIH con CAN menores o iguales a 1000 célula/mcl, 24% desarrollaron complicaciones infecciosas, más comúnmente dentro de 24 hrs después del inicio de la neutropenia.

En el análisis multivariado, los 3 factores independientemente asociados con complicaciones infecciosas incluían la presencia de un catéter venoso central, una neutropenia en los primeros 3 meses, y un bajo nivel en el conteo de granulocitos (250 células/mcl en aquellos con infección versus 622 células/mcl sin infección). Entre los pacientes con medicación asociada a la neutropenia, la causa más común fue Zidovudine, seguida por trimetopin-sulfametoxazol, y ganciclovir; la neutropenia fue probablemente menos asociada con la infección en esos pacientes que en individuos quienes eran neutropénicos debido al uso de quimioterapia de cáncer.

Los mecanismos de la trombocitopenia en la púrpura trombocitopenica relacionada con VIH.

Aumento de la destrucción de plaquetas. Como en la purpura trombocitopenica idiomática originaria, (PTI), los pacientes infectados por VIH con PTI también muestran un incremento en la destrucción de plaquetas vía fagocitosis por macrófagos en el bazo. En el VIH relacionado a PTI, sin embargo, muchos mecanismos por plaquetas asociadas a anticuerpos han sido descritos, y usualmente ocurren simultáneamente en un paciente dado. Así, la presencia de anticuerpos específicos de plaquetas, inmunoquimicamente caracterizados como anti-glicoproteina (GP) IIB y/o GPIIIa, han sido detectados en pacientes infectados por VIH con PTI, indicando un mecanismo similar a aquello descrito en la enfermedad originaria.

Sin embargo, han sido también vistos anticuerpos reactivos cruzados entre el VIH GP169/120 y plaquetas GPIIb/IIIa.

Ari, Bettaieb y colegas encontraron que anticuerpos en suero contra el VIH GP160/120 podrían ser eludidos de las plaquetas de pacientes con VIH relacionado a PTC y que estos anticuerpos específicos de VIH compartían un epitope común con los anticuerpos en contra de plaquetas GPIIb/IIIa sobre la superficie plaquetaria. Así es que la aparente imitación molecular entre el VIH GP160/120 y la plaqueta GPIIb/IIIa podría ser operativa en la destrucción inmune de plaquetas en algunos casos de VIH relacionado a PTI.

Un ulterior mecanismo de destrucción de plaquetas inducida por anticuerpos surge de la absorción de complejos inmunes contra el VIH sobre el receptor plaquetario Fc, proveyendo así una porción libre de Fc para un subsecuente vínculo de macrófagos y fagocitosis.

Disminución en la producción de plaquetas.

Estudios cinéticos de la producción y destrucción plaquetaria han sido desarrollados en pacientes con VIH relacionado al PTI, con resultados comparados con un grupo de sujetos de control normal y con pacientes con PTI originario. La supervivencia media plaquetaria fue significativamente menor en pacientes con VIH PTI, lo que ocurre con la misma extensión en pacientes que reciben Zidovudine y aquellos que no fueron tratados. Es interesante remarcar que la supervivencia media plaquetaria fue también significativamente menor en pacientes con VIH y conteos normales de plaquetas. Además de este incremento en la destrucción plaquetaria, la producción media plaquetaria se encontró significativamente menor en pacientes con VIH PTI no tratados, aunque aquellos pacientes que reciben Zidovudine demostraron un subsecuente incremento en la producción plaquetaria, lo que ocurre incluso en el tratamiento con Zidovudine en pacientes infectados por VIH sin trombocitopenia. Así, es aparente que pacientes con VIH PTI, mientras experimentan un moderado incremento en la destrucción plaquetaria, también se enfrentan con un significante descenso en la producción plaquetaria, la cual ocurre incluso en aquellos individuos con conteos normales de plaquetas.

ANALYSIS

As a matter of fact, an analysis of the translated text had to be done. In this analysis the translation strategies are explained in detail, one by one, followed by their definitions and explanations. To make this analysis clearer the translator has decided to make a chart dividing it in two parts, the first one will be the English version taken from the original text, and the second the Spanish translation. After the Spanish version the page number and line were given in order to make it easy for the readers of this monographic work to find the examples in the paper. Finally, after each example, the translator explained how the translation technique was applied.

BORROWING

The author wrote the original text using the more common words to make it easy to read and to understand. Through the original text the author uses a well-established borrowing, which can´t be written differently since it is a proper name.

ENGLISH	SPANISH
The **virus** envelope fuses…	La envolvente del **virus** se fusiona…
An initial measurement of **plasma** viral load…	Una medida inicial de carga viral en **plasma**

LITERAL TRANSLATION

As mentioned before, literal translation technique is divided into word for word translation and literal translation, which is a more flexible one, since it allows minimal adjustments of word order according to the target language.

In the next examples of word for word translation, we can see that the sentence in the target text has the same structure of the sentence in the original text, and the words were translated using their first dictionary definition since the words exist in both languages.

ENGLISH	SPANISH
At approximately the same time that AIDS was first recognized in 1981.	Aproximadamente en la misma época que el SIDA fue reconocido en 1981.
HIV may be transmitted by sexual contact with an infected partner…	El VIH puede ser transmitido por contacto sexual con una pareja infectada…
Transmission of HIV	Transmisión del VIH

CALQUE

We know that the calque is a kind of borrowing where a language borrows an expression from another language. Orthographic calque is used for historical figures and for those that don´t have a traditional, accepted translation, like in the first example.

ENGLISH	SPANISH
Immune thrombocytopenic **purpura**	**Púrpura** trombocitopenica idiopática
Megakaryocyte	**Megacariocito**
The internalized **nucleocapsid**..	El internalizado **nucleocápsida**

MODULATION

This technique involves changing the point of view or perspective of the message. The perspective of a sentence can change a lot just by changing one word. Since the article is extremely technical, obviously the modulation technique was not required.

TRANSPOSITION

As mentioned before, transposition "involves replacing one word class with another without changing the meaning of the message" (Venuti: 1999, p.88). This means the modification of the grammatical category of a part of the sentence without semantic variations

ENGLISH	SPANISH
The SIV **viral strain** infecting these monkeys is related… **Infected partner** **Increased** red cell destruction.	La **infección de la sepa viral** de VIP de estos monos es relacionada… **Pareja que ha sido infectada**. **El incremento en** la destrucción de glóbulos rojos.

EQUIVALENCE

"The notion of equivalence is one of the most problematic and controversial area in the field of translation theory" (Vinay and Dalbernet : 1977). This technique is used when we want to replicate the same situation as in the original text, and is used for proverbs, idioms, clichés and onomatopoeia, as well as in some extremely technical expressions, as was the case in this translation.

ENGLISH	SPANISH
Red blood cells **Immunoassay.** **Large oval cells** **Clumped basophilic chromatin**	**Globulos rojos** **Inmuno absorbente.** **Ovalocitos** **Basofilia difusa**

ADAPTATION

This technique is used when there is no common equivalence for a given expression, or when a situation in the source culture does not exist in the target culture. Since the article is extremely technical, obviously the adaptation technique was not required, being it applied mostly with culture-laden expressions.

EXPANSION

This technique adds some elements to the sentence for greater comprehensibility. In the following examples one or more words were added to give more sense to the sentences.

ENGLISH	SPANISH
Transmission through infected blood products.	Transmisión **a través** de productos de sangre infectada.
The enzyme-linked immunoassay…	Ensayo **inmuno** absorbente ligado a la enzima…

REDUCTION

We use omission when a language is more concise than another because of its structure or grammar.

ENGLISH	SPANISH
Red blood cells.	**Globulos rojos.**
Donated units of blood…	**Sangre donada**…

CONLUSIONS

The main point of this translation work is that of increasing information about a terrible ill which is AIDS. The new media related on AIDS usually are in English. With the help of the translation techniques this translation work was successfully done. Translation techniques are an important tool in translating texts because it is not just a ratter of using a dictionary and translating word by word: the whole thing demands more work to do if you want an excellent translation.

As I said before AIDS is primarily a sexually transmitted disease (STD) intimately related to the sexuality of individuals and populations. In Mexico, as in other countries, AIDS has become a complex health-care challenge, with many psychological, social, ethical, economic, and political dimensions that transcend the usual focus of health-care. This is why it is critical to coordinate an interdisciplinary response from diverse organizations, institutions, and other sectors of society to more effectively meet this challenge.

HIV transmission patterns in Latin American countries are similar to transmission patterns in industrialized countries. While it appears that the current rate of infection is decreasing or at least stabilized, the outlook is not uniform across the Latin American region. In most Latin American countries the epidemic is classified as "concentrated" in specific populations. Seroprevalence rates in these at-risk populations range from one to 25 percent. A seroprevalence rate of fewer than five percent is seen in the total population. However, in recent years some countries in the region -- Nicaragua, Venezuela, and Peru -- appear to have rapidly increasing seroprevalence rates.

Mexico ranks 13th globally and third in the Americas in the total number of HIV cases reported. However, when rated on the accumulated number of cases, Mexico ranks 69th globally, 29th in Latin America and the Caribbean, and in 11th place in the Americas, which reflects a comparatively low incidence rate.

Through this translation I found some problems with technical vocabulary all related to medicine terms so that I needed help from people like doctors and chemists because sometimes there were words which were not easy to find in dictionaries.

However, the original text was difficult to translate. For the same reason, difficult vocabulary was used in this translation.

Nonetheless, it is known that when doing a translation we can find some problems and the translator has to solve them and find the best way to overcome the whole difficult vocabulary.

Most of the text required literal translation and since the article is extremely technical, obviously the adaptation technique was not required, being it applied mostly with culture-laden expressions as is the case with using modulation technique because the text didn´t require too many changes.

In order to find the best examples of each technique, the translator had to read and examine the translation so as to find the best sentences to exemplify the translation strategies.

And last but not least, for coming research for anyone involved in translation, this translation showed some uses of the translation techniques and methods of translating texts because when we are not native speakers of English it could be difficult, but by making our best effort, we can hope to achieve a successful product.

BIBLIOGRAPHY

Alvarez,T. (n.d). *Diccionario English-Spanish*. México: Larousse.

Bassnet, s. (2002). *Translation Studies*. London: Routledge.

Bernard, J. (1998). *Diagnosticos Clinicos por el Laboratorio*. México: Salvat.

Beutler, E & Lichtman, M. (2001). *Hematology*. USA: McGraw Hill.

Collins, W. (1990). *English Language Dictionary*. London: Harper Collins Publisher.

Fawcett, P. (1997). *Translation and Language*. Manchester: St. Jerome.

Hillman R. (2004). *Hematology.* England: Routledge.

Klehment, Y. (1993). *Diccionario Médico Inglés-Español*. México: Editorial Piensa.

Kolmer, J. (1981). *Dignostico Clinico*. México: Inter Americana.

Lee, R. (2002). *Clinical Hematology*. USA: Lippincott Williams Publishers.

Lynch, R. (1990). *Métodos de Laboratorio*. México: Inter Americana.

Mifflin, H. (2006). *English-Spanish Dictionary*. México. Editorial Trillas.

WEB REFERENCES

British Counsil. (n.d). *Literary Translation*. Retrived October 18, 2005 from:
    http://www.literarytranslation.com/art/.

COGUSIDA. (n.d). *Sida en México*. September 18, 2007 from:
        http//www.guanajuato.gob.mx

Intercultural Studies. (n.d). *Translation Techniques*. October, 16 2007 from:

http//isg.urv.es/publicity/masters.

Ortega, S. (n.d). *México, Segundo en América Latina con más enfermos de SIDA.*
September 18, 2007 from:
http//enkidumagazine.com/art/2007/

APPENDICES

At approximately the same time that AIDS was first recognized in 1981, reports of a similar immunodeficiency syndrome, characterized by wasting and opportunistic infections, was described in several colonies of macaques housed at primate centers in the United States.[9,10] The illness, known as *simian immunodeficiency syndrome* (SAIDS), was associated with infection by a retrovirus, termed *simian immunodeficiency virus* (SIV).[11] Subsequent testing revealed that over 20 percent of all tested symptomatic African green monkeys or African mangabeys from the wild had serologic evidence of SIV infection.[12-14] The SIV viral strain infecting these monkeys is related to HIV-2, a less virulent strain of human immunodeficiency virus, found primarily in West Africa.[15] An immunodeficiency virus related to HIV-1 and infecting African chimpanzees was also identified.[16] Recent evidence suggests that the subspecies of chimpanzee *Pan troglodyte* may have been the original reservoir of HIV-1.[17]

Based upon these observations, it is postulated that HIV originally may have been transmitted to humans from an African species of ape.[14,17] By the mid- to late 1960s, political and societal circumstances were beginning to change dramatically in ways that were conducive to the rapid spread of this infection in humans. The movement of previously isolated African peoples from rural villages to large urban centers; a change in sexual habits, resulting in widespread exposure to increasing numbers of sexual partners; the worldwide epidemic of parenteral drug abuse; and the advent of commercial air travel all contributed to the current pandemic of HIV infection.

The WHO has estimated that over 30 million people had been infected by HIV worldwide by mid-1998,[18] the majority infected by heterosexual contact, with homosexual contact and injection drug use the predominant modes of transmission in the United States and western Europe. Vertical transmission from infected mother to child is now decreasing in developed countries, although such transmission continues to increase in resource-poor regions of the world.

ETIOLOGY AND PATHOGENESIS

HUMAN IMMUNODEFICIENCY VIRUS-1

HIV-1 is a member of the primate Lentivirinae subfamily of retroviruses,[19,20] RNA viruses that induce a chronic cellular infection by converting their RNA genome into a DNA provirus that is integrated into the genome of the infected cell. Infection by these lentiviruses is characterized by long periods of clinical latency followed by a gradual onset of disease-related symptoms.[21-23]

TRANSMISSION OF HIV

HIV may be transmitted by sexual contact with an infected partner, by parenteral drug use with a contaminated needle, by exposure to infected blood or blood products, and by perinatal exposure from an infected mother to her infant.

General Mechanisms of Sexual Transmission HIV-1 has been isolated from the semen of HIV-infected men[24] as well as from cell-free seminal fluid[25] and may be detected during the first 3 to 4 weeks after primary infection.[27] Factors associated with increased viral burden in semen include more advanced symptomatic HIV disease, higher levels of HIV-RNA in blood, CD4 cell counts of less than 200/μl, and presence of seminal fluid leukocytosis. HIV infection has been reported after exposure to infected semen during artificial insemination.[27]

HIV has been recovered from cervical and vaginal secretions of HIV-infected women,[28,29] and HIV-infected endothelial cells and macrophages have been detected in cervical biopsies.[30] Factors that influence the levels of HIV-1 in female genital tract secretions include the stage of HIV disease, menstruation status, hormonal parameters, concomitant vaginal infection, age, HIV-1 RNA level in plasma, and antiviral therapy.[31] Although female-to-female transmission of HIV has been reported,[32,33] this appears to be relatively unusual.

HIV transmission may be facilitated by the presence of other sexually transmitted diseases, both with and without ulceration,[34] and HIV has been isolated directly from genital ulcers.[35] Prevention or treatment of sexually transmitted disease has been associated with a decrease in HIV-1 transmission.[36]

Transmission through Parenteral Drug Use Sharing needles and syringes is an important mode of transmission among parenteral drug users.[37] The use of cocaine has been associated with a particularly high risk of HIV infection,[38] presumably related to its short half-life and the resulting need for greater numbers of injections. Behavioral factors may lead to increased risk of HIV-1 transmission even among nonparenteral illicit drug users.

Transmission through Infected Blood Products The risk of infection with HIV after receiving 1 unit of infected blood approximates 90 percent.[39] Transfusion of blood products derived from multiple units of pooled blood can also transmit HIV and accounted for the initially high prevalence of HIV infection among patients with hemophilia. Screening of all donated blood, beginning in March 1985, and the subsequent routine heat or solvent detergent treatment of clotting factor concentrates have resulted in a marked decrease in new transfusion-associated HIV infections. Guidelines for proper inactivation of HIV in clotting factor concentrates have been developed.[40,41] Currently, the risk of acquiring HIV through receipt of a unit of blood that tests negative for antibodies to HIV-1 is approximately 1 in 493,000.[42]

Mother-to-Child Transmission The risk of infection from mother to infant differs in various parts of the world, ranging from approximately 15 percent in Europe to 15–30 percent in the United States and 40–50 percent in Africa.[43-45] HIV-1 may be transmitted in utero,[46,47] intrapartum (at the time of delivery);[48,49] or postpartum, through ingestion of HIV-1 infected mother's milk.[50,51] Several factors predict an increased risk of perinatal transmission. In terms of the mother, more advanced HIV disease,[52,53] higher HIV-1 viral load in the plasma,[54,55] cigarette smoking,[56] and active injection drug use[57] have all been associated with increased risk of transmission. In terms of the details of delivery, premature rupture of the amniotic membranes (over 4 h),[58,59] presence of chorioamnionitis,[57] and vaginal delivery, as opposed to elective cesarean section,[60,61] have each been associated with increased rates of transmission. In terms of the infant, breast-feeding, prematurity, and low gestational age are reported as risk factors.[58,59,62] The CDC has recently made formal recommendations regarding the optimal care for HIV-1 infected pregnant women.[63] These recommendations differ for resource-rich and resource-poor settings. In the United States, the use of antiretroviral agents in pregnancy and delivery, with subsequent administration to the infant for the first 6 weeks of life, has resulted in a dramatic reduction in the rate of transmission, from approximately 25 percent to 8 percent.[64] With the further use of elective cesarean section and avoidance of breast feeding, transmission rates have dropped to approximately 2 percent.[60] The efficacy of shorter courses of zidovudine or neviripine (a non-nucleoside reverse transcriptase inhibitor) have been demonstrated and may be more practically feasible in resource-poor regions of the world.[65,66] The long-term toxicities of in utero exposure to antiretroviral agents are unknown. Nonetheless, their use during pregnancy resulted in a 43 percent decrease in the number of children with perinatally acquired HIV infection in the United States when comparing data from 1992 and 1996.[67]

HIV-1 gp120 binds to the CD4 surface membrane protein, resulting in a further high-affinity binding to the chemokine CCR5 receptor.[70,85] Human helper-inducer (CD4) lymphocytes, monocytes-macrophages, Langerhans' cells, follicular dendritic cells, megakaryocytes, and thymic cells express the CD4 and chemokine receptor molecules and are susceptible to infection by HIV-1. The structural diversity of the gp120 viral receptor has resulted in viral strains with selective or restricted patterns of infection, such as those that readily infect monocytes, while others are tropic for CD4 lymphocytes.[76,86] Macrophage-tropic (M-tropic) strains of HIV use the CCR5 chemokine receptor to infect both macrophages and CD4+ lymphocytes.[87,88] The T-tropic strains use the CXC4 chemokine receptor and may also use the CCR5 receptor.[87,88] Additional chemokine receptors CCR2 and CCR3 have also been implicated as coreceptors for HIV infection of certain cell types.[88,89]

Upon binding to the CD4 protein on the host cell, the virus envelope fuses with the host cell membrane[90] (Fig. 89-3). This fusion is mediated by a hydrophobic domain on the amino terminal portion of gp41.[77] The internalized nucleocapsid then is destabilized and dissociates after binding to the cellular protein cyclophilin A,[91] exposing the diploid viral RNA genome associated with reverse transcriptase.[92] Reverse transcription proceeds by the synthesis of a single cDNA strand, followed by degradation of the viral RNA by the ribonuclease H activity of p66. Reverse transcriptase then acts as a DNA polymerase, forming a second DNA strand. This synthesis of the double-stranded DNA provirus must proceed rapidly to prevent the degradation of viral RNA by intracellular enzymes. The estimated rate of base substitution errors for HIV reverse transcriptase may be as high as 1 in 1700 to 1 in 2000.[93,94] This results in 5 to 10 nucleoside mutations per virus for each replication cycle and explains the high degree of genomic diversity observed between viral isolates of HIV.[95]

The integration of the provirus is necessary for stable infection of the cell. Viral integrase is capable of both cleaving host DNA and integrating a linear form of the provirus.[96] Kinetic studies of HIV-1 infection have detected viral DNA present in the cytoplasm within 2 to 3 h of infection, while nuclear viral DNA has been detected by 24 h.[97] The gene product of the VPR gene appears to assist in the transport of the preintegration viral DNA into the nucleus for subsequent integration.[98,99] After successful integration of the viral genome, the HIV-1–infected cell may develop either a latent or a persistent form of infection.

The mechanism or mechanisms of viral latency remain poorly understood but appear to require activation of the infected cell, since HIV-1 does not replicate efficiently in resting lymphocytes or macrophages.[100,101] Cellular transactivating proteins, such as NF-κB, are upregulated in activated cells and enhance HIV proviral transcription.[102]

After integration, HIV-1 proviral transcription leads to the expression of regulatory proteins designated tat, rev, and nef.[97,98] Tat is a small nuclear protein that is essential for HIV replication and, in conjunction with other cellular proteins, TAK (Tat-associated kinase) and CycT (cyclin T), assists in viral RNA elongation, resulting in a 1000-fold increase in HIV-1 expression by the infected cells.[98,103,104]

Rev is a viral protein that regulates nuclear export of unspliced viral RNA.[98,105,106] Like tat, rev is essential for viral replication and must bind to a rev-responsive element located in the ENV gene. The other HIV-encoded proteins, designated nef and vpu, have a role in the modulation and down-regulation of the cellular receptor, CD4.[98,107-110]

The structural proteins of the GAG, POL, and ENV genes are expressed as precursor proteins and subsequently cleaved by viral protease to yield mature viral proteins. Proteolysis of proteins by the viral protease is essential for viral maturation and infectivity. The products of the ENV gene, gp120 and gp41, are transported to the cell membrane. The ribonucleoprotein core assembles in the cytoplasm of the host cell and subsequently moves to the membrane surface for budding. The efficient packaging of the viral RNA is dependent upon packaging signals present in the Gag region of the viral RNA.[111] The budding of virus appears to be dependent upon the product of the VPU gene, which assists in membrane transport of ENV gene products.[107,108,112] In addition, viral infectivity appears to require the gene product of the VIF gene.[113]

HIV infection results in aberrant immune regulation and immunodeficiency. The numerous in vitro and in vivo defects in cellular immune response observed with HIV infection include decreased lymphocyte proliferation to soluble antigens,[114] decreased helper response in immunoglobulin (Ig) synthesis,[115] impaired delayed hypersensitivity,[1,2] decreased interferon-γ production,[116] and decreased T-cell–mediated cytotoxicity of virally infected cells.[117]

·DEPLETION OF CD4+ T CELLS

Infection with HIV-1 results in a progressive loss of CD4-positive (CD4+) T lymphocytes, resulting from the direct cytopathic effect of HIV on CD4+ lymphocytes. Formation of syncytial multinucleated giant cells by a mechanism involving fusion of infected cells expressing viral gp120 with noninfected CD4+ T lymphocytes is another mechanism of CD4 depletion.[118] The propensity of certain viral stains to form syncytia appears to be associated with an aggressive clinical course.[117,118] More recent experimental data suggest that an HIV-1 phenotypic switch from an M-tropic (nonsyncytial) to a T-tropic (syncytial) virus may be the central event in acceleration of HIV-induced immunodepletion.[119]

The host immunologic response against HIV-infected lymphocytes also may contribute to the progressive loss of CD4+ lymphocytes by antibody-mediated and cytotoxic T-cell–mediated mechanisms.[120,121] Noninfected lymphocytes may also become "innocent bystander" targets for immunologic destruction by binding free gp120 to their surface CD4 protein.

Defective production of immunostimulatory cytokines, such as interleukin-2 (IL-2),[122-124] or exaggerated expression of inhibitors of T-lymphocyte proliferation, such as transforming growth factor-β (TGF-β),[125] can contribute to the progressive decline in CD4 lymphocytes. High-level replication and budding of virus, resulting in membrane injury, has also been proposed as a mechanism for lymphocyte cytotoxicity.

Recent advances in combination antiretroviral therapy have resulted in marked suppression of viral replication, with resulting reductions of blood and tissue viral reservoirs.[126,127] Efficient viral suppression has resulted in significant and prolonged immunologic reconstitution characterized by increased CD4+-lymphocyte numbers, reduced opportunistic infections, and prolonged survival.[128,129] However, significant deficits in the immunologic repertoire persist, and complete immunologic reconstitution has not yet been attained.[130,131]

DIAGNOSIS OF HIV INFECTION

Like the clinical manifestations of acute (primary) HIV infection ("acute retroviral syndrome"), the laboratory markers are nonspecific, with frequent elevation of liver transaminase levels and erythrocyte sedimentation rate. However, HIV viremia is present during the acute illness and can be detected by molecular methods such as reverse transcription polymerase chain reaction (RT-PCR).

The primary diagnostic screening tool is detection of antibody via the enzyme-linked immunoassay (ELISA). However, since a positive ELISA result may not be specific for HIV-1 infection, all positive ELISA screening test results should be verified by immunoblotting HIV-1 antigens (see Fig. 89-2).

By ELISA and immunoblot techniques, the median time from initial infection to first detection of HIV antibody has been estimated to be 2.4 months, while 95 percent of cases are expected to seroconvert within 5.8 months (see Fig. 89-4).[150] HIV infection for longer than 6 months without detectable antibody is extremely uncommon.[151-153]

The presence of the p24 antigen or HIV RNA in serum or plasma may precede seroconversion by several weeks.[154] This initial rise in p24 antigen correlates with the burst of viremia that occurs shortly after primary HIV infection.[155] Despite these observations, p24 antigen screening of donated units of blood appears to provide no benefit over conventional ELISA and immunoblot techniques.[156]

- LABORATORY FEATURES OF DISEASE PROGRESSION

With progression from the initial acute infection to the expected asymptomatic period, various laboratory parameters may be used to predict development of more advanced disease.[7,8,157] Quantitation of plasma HIV RNA (viral load) and CD4+ lymphocyte count are the most useful parameters. The CD4+ lymphocyte count falls during the acute retroviral infection and then stabilizes during early asymptomatic infection and may appear relatively normal. The CD4+ count then decreases by approximately 40 to 80 μl/year in the absence of anti-retroviral medications,[158] although there is significant variability among patients.[159]

An initial measurement of plasma viral load by RT-PCR or branched-DNA (bDNA) methods provides important prognostic information that can be useful in determining when to start antiretroviral medications.[7,8] The serial assessment of plasma HIV viral load also allows for rapid assessment of efficacy of antiretroviral medications. Changes in viral load usually precede significant alterations in CD4+ lymphocyte counts.[8,128]

Several nonspecific markers of disease progression have been defined, including β_2-microglobulin[160] and neopterin,[161] each of which has independent predictive value in estimating the probability of progression to AIDS. However, each of these surrogate markers has been largely replaced by the more specific molecular assays to quantify plasma HIV viral load.

ANEMIA DUE TO DECREASED PRODUCTION OF RED BLOOD CELLS

A decrease in production of red blood cells may result from factors suppressing the CFU-GEMM, such as inflammatory cytokines or the HIV virus itself.[174,175] In addition, a blunted production of erythropoietin has been documented in anemic HIV-infected patients, similar to the suppression seen in other states of chronic infection or inflammation.[177] Infiltration of the marrow by tumor, such as lymphoma,[178] or infection, such as *Mycobacterium avium* complex (MAC), may also lead to the decreased production of red cells. In addition, MAC may also be associated with cytokine-induced marrow suppression. Involvement of the gastrointestinal (GI) tract by various infections or tumors may lead to chronic blood loss, with eventual iron deficiency anemia. Another prominent cause of hypoproliferative anemia in patients with HIV infection is the common use of multiple medications, many of which may cause marrow and/or red cell suppression. Zidovudine (AZT), the first licensed antiretroviral agent, is uniformly associated with macrocytosis mean cell volume (MCV >100), which can be used as an objective indication that the patient has been compliant with this medication.[179] It noteworthy that transfusion-dependent anemia (hemoglobin < 8.5 g/dl) has been reported in approximately 30 percent of patients with full-blown AIDS, receiving zidovudine at doses of 600 mg/day. However, the incidence of severe anemia is only 1 percent when the same dose of zidovudine is used in patients with asymptomatic HIV disease.[180]

Infection of the marrow by parvovirus B19 is another cause of hypoproliferative anemia in HIV-infected patients, resulting in specific infection of the pronormoblast.[181,182] Thus, while marrow failure affecting all three lines has been described in association with parvovirus B19 infection, a pure red cell aplasia is the usual consequence. Parvovirus infection is usually acquired during childhood, leading to "fifth disease," one of the common childhood exanthums. Exposure to the virus leads to an antibody response, with subsequent resistance to further infection. Approximately 85 percent of adults have serologic evidence of prior parvovirus infection. However, the seroprevalence of such antibodies among HIV-infected patients is only 64 percent. This would suggest that these individuals may have an ineffective immune response against newly acquired infection. The diagnosis of parvovirus B19 can be made on marrow examination, revealing giant pronormoblasts with clumped basophilic chromatin and clear cytoplasmic vacuoles; diagnosis can be confirmed by in situ hybridization using sequence-specific DNA probes for parvovirus B19. Therapy for parvovirus-induced red cell aplasia consists of infusions of intravenous (IV) gamma globulin that contain antibodies from plasma donors most of whom have been exposed to parvovirus. Relapse of parvovirus B19–induced red cell aplasia may occur, necessitating retreatment in these individuals.[181,182]

ANEMIA DUE TO INCREASED RED CELL DESTRUCTION

Increased red cell destruction may be seen in HIV-infected patients with G-6-PD deficiency who are exposed to oxidant drugs and in HIV-infected patients with disseminated intravascular coagulation (DIC) or thrombotic thrombocytopenic purpura (TTP);[183] presence of fragmented red cells and thrombocytopenia on blood smear will be seen in the latter two conditions, and Heinz bodies will be seen in association with G-6-PD deficiency. Hemophagocytic syndrome has also been described in association with HIV infection. An additional cause of red cell destruction in HIV-infected patients is the development of autoantibodies, with resultant positive Coombs' test result and shortened red cell survival. It is interesting to note that a positive direct Coombs' test result has been reported in as many as 18 to 77 percent of HIV-infected patients, although the incidence of actual hemolysis is quite low. When present, anti-i antibody and antibody against auto-U antigens have been described, occurring in 64 percent and 32 percent of HIV-infected patients, respectively.[184-186] A high incidence of positive direct Coombs' test results has also been detected in patients with other hypergammaglobulinemic states, indicating that the positive Coombs' test results in HIV may simply be secondary to the polyclonal hypergammaglobulinemia that is known to occur in the setting of HIV infection.[187]

Folic acid is absorbed in the jejunum and is responsible for one carbon transfer required in the synthesis of DNA. A deficiency of folic acid leads to a megaloblastic anemia, with large oval red cells in the blood, hypersegmented neutrophils, and a decrease in all three lines, with resultant anemia, neutropenia, and thrombocytopenia. Since tissue stores of folate are relatively small, a deficiency of folate in the diet lasting as little as 6 to 7 months may lead to anemia. It is thus apparent that HIV-infected patients who are ill and not eating properly, as well as those with underlying disease of the jejunum, may be unable to absorb sufficient folic acid. The classic changes of megaloblastic anemia will be detected upon examination of the bone marrow, while serum and red cell folate levels will be low.

Ineffective production of red cells, with pancytopenia in the blood, elevated indirect bilirubin level, and low reticulocyte count may also be seen in vitamin B_{12} deficiency. The absorption of B_{12} requires initial production of intrinsic factor by parietal cells in the stomach, with subsequent absorption of the complex of B_{12} and intrinsic factor within the ileum. Thus, malabsorption of B_{12} can occur in various disorders of the stomach (achlorhydria), by production of antibodies to intrinsic factor ("pernicious anemia"), or by various disorders of the small bowel and ileum (infection or Crohn's disease). While B_{12} deficiency is highly unlikely on a dietary basis alone, patients with HIV infection appear to be prone to B_{12} malabsorption, presumably due to the myriad infections and other disorders that may occur in the small intestine. Negative vitamin B_{12} balance has been documented in approximately one-third of patients with AIDS, the majority demonstrating defective absorption of the vitamin.[188] Diagnosis of B_{12} deficiency can be made by documenting low serum B_{12} levels, while the earliest indication of negative B_{12} balance is the finding of low B_{12} levels in blood in patients taking transcobalamin II.[189] Monthly administration of parenteral B_{12} will correct the deficiency and the resultant anemia and pancytopenia in the peripheral blood. Since B_{12} deficiency may also cause neurologic dysfunction (subacute combined degeneration of the cord), with motor, sensory, and higher cortical dysfunction, the possibility of vitamin B_{12} deficiency should also be considered in HIV-infected patients with these neurologic symptoms.

RISK FACTORS FOR INFECTION IN NEUTROPENIC PATIENTS WITH HIV

In patients with cancer who receive chemotherapy, multiple studies have shown that the risk of bacterial infection rises when the absolute neutrophil count (ANC) falls below 1000 cells/dl and increases again when the ANC falls below 500 cells/μl.[199] Several studies have confirmed the same relationships in patients with HIV infection. Thus, Moore and colleagues found that the risk of bacterial infection increased 2.3-fold for HIV-infected individuals with less than 1000 cells/μl and rose by 7.9-fold in those with ANC levels less than 500 cells/μl.[200] Lower ANC counts are associated with increased risk of hospitalization for serious infection among HIV-infected patients, as shown by a review of 2047 HIV-positive patients. On multivariate analysis, the severity and duration of neutropenia were found to be significant predictors of the incidence of hospitalization for serious bacterial infections.[201]

In a recent study of 62 HIV-infected patients with ANCs less than or equal to 1000 cells/μl, 24 percent developed infectious complications, most commonly within 24 h after the onset of neutropenia.[202] On multivariate analysis, the three factors independently associated with infectious complications included presence of a central venous catheter, neutropenia in the previous 3 months, and a lower nadir of granulocyte count (250 cells/μl in those with infections versus 622 cells/μl in those without). Among patients with medication-associated neutropenia, the most common cause was zidovudine, followed by trimethoprim-sulfamethoxazole, and ganciclovir; neutropenia was less likely to be associated with infection in these patients than in individuals who were neutropenic due to the use of cancer chemotherapy.[202]

MECHANISMS OF THROMBOCYTOPENIA IN HIV-RELATED THROMBOCYTOPENIC PURPURA

Increased Platelet Destruction As in "de novo" immune thrombocytopenic purpura (ITP), HIV-infected patients with ITP also demonstrate increased platelet destruction via phagocytosis by macrophages in the spleen.[210] In HIV-related ITP, however, several mechanisms for platelet-associated antibody have been described, often occurring simultaneously in a given patient. Thus, presence of platelet-specific antibodies, immunochemically characterized as anti-glycoprotein (GP)IIb and/or GPIIIa, have been detected in HIV-infected patients with ITP, indicating a mechanism similar to that described in "de novo" disease.[211] However, cross-reactive antibody between HIV GP160/120 and platelet GPIIb/IIIa has also been demonstrated.[212] Thus, Bettaieb and colleagues found that serum antibodies against HIV GP160/120 could be eluted from platelets of patients with HIV-related ITP and that these HIV-specific antibodies shared a common epitope with antibodies against platelet GPIIb/IIIa on the platelet surface. It is thus apparent that molecular mimicry between HIV GP160/120 and platelet GPIIb/IIIa may be operative in the immune destruction of platelets in some cases of HIV-related ITP. A further mechanism of antibody-induced destruction of platelets arises from the absorption of immune complexes against HIV onto the platelet Fc receptor, thus providing a "free" Fc portion for subsequent macrophage binding and phagocytosis.[211]

Decreased Platelet Production Kinetic studies of platelet production and destruction have been performed in patients with HIV-related ITP, with results compared to a group of normal control subjects and to a group of patients with "de novo" ITP.[210] Mean platelet survival was found to be significantly decreased in patients with HIV ITP, occurring to the same extent in patients receiving zidovudine and in those who were untreated. It is interesting to note that the mean platelet survival was also significantly decreased in HIV-infected patients with normal platelet counts. In addition to this increased destruction of platelets, mean platelet production was found to be significantly decreased in patients with untreated HIV ITP, although those patients receiving zidovudine demonstrated a subsequent increase in platelet production, occurring even in zidovudine-treated HIV-infected individuals without thrombocytopenia. Thus, it is apparent that patients with HIV ITP, while experiencing a moderate increase in platelet destruction, are also faced with significant decreases in platelet production, which occur even in those individuals with normal platelet counts.[210]