Phytochemicals and HIV Suppression: A Systematic Review
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Abstract
The human immunodeficiency virus (HIV) causes immune suppression known as acquired immunodeficiency syndrome (AIDS) leading to various opportunistic infections and malignancies having high mortality rates. Here we provide a systematic review and discussion of current knowledge on photochemical activities against HIV and AIDS. After several years of research, efficient antiretroviral therapy helps controlling the progression of AIDS. However, due to the overuse of antiretroviral drugs, viral resistance in patients and side effects from long-term use of drug therapy have emerged, which shorten life expectancy of patients. To improve HIV treatment, substances in plants may inhibit the life cycle of HIV through inhibition of the activity of reverse transcriptase, integrase or protease required for processes such as HIV transcription and replication. In addition, phytochemicals regulate the human immune system and thereby suppression of HIV and AIDS development in clinical treatments. Therefore, more experiments are needed to demonstrate the effectiveness and safety of plants for therapeutic AIDS treatment, which may bring forward new HIV and AIDS treatment options.

Keywords: AIDS; Human immune system; Safety, Antiretroviral therapy; Phytostatic therapy

Background
AIDS is a highly harmful epidemic infectious disease caused by human immunodeficiency virus. Since 1981, when the U.S. released reports of five cases of AIDS, the speed of transmission has been accelerating, and the number of AIDS patients continues to increase. Over the past 30 years, AIDS has become one of the most serious and complex public health problems in the world. In 1985, a foreigner died after falling ill in China, kicking off the country's long battle against AIDS. The Global AIDS progress report released by UNAIDS on July 6, 2020, pointed out that currently 38 million are HIV-positive worldwide. At the same time, the report mentioned that if countries do not take action, the gains may be lost and the anti-AIDS work further delayed. The previous goal of global elimination of AIDS by 2020 so far not achieved. The following data are obtained from the relevant notices issued by the Chinese Health Commission, China launched the Human Immunodeficiency Virus Prevention Project in 2003 [1], as of the end of October 2020, the Chinese population with AIDS numbered 1.045 million. HIV infect human immune cells including T helper cells (CD4+T) and macrophages, and by attacking CD4+T cells, the virus accelerates the apoptosis of immune cells and causes the human immunity is gradually compromised by this condition. CD4+T lymphocytes are important subsets of immune cells in the body, HIV persists in a large variety of CD4+T cells [2]. After HIV infection, a large number of

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copies in CD4+T lymphocytes will cause damage to CD4+T lymphocytes, leading to immune function defects. Kaposi Sarcoma, non-Hodgkin's lymphoma, and invasive squamous cell carcinoma of the cervix are malignancies considered to be directly related to immunodeficiency in people with HIV [3,4], and eventually cause the death of the body.

Antiretroviral Therapy, commonly known as "cocktail therapy", antiretroviral therapy with high antiviral activity (HAART), which is currently used as efficient AIDS treatment. In order to achieve a powerful antiviral effect, this therapy requires the combination of at least three antiviral drugs to inhibit HIV-RNA in plasma at low or undetectable levels, HAART therapy can reduce drug resistance caused by a single drug, effectively inhibit HIV replication in the body, and restore the body's damaged immune system, thus greatly reducing the morbidity and mortality of AIDS [5]. HIV patients can live longer with HAART by boosting their CD4+T lymphocyte count, thereby enhancing the body's immunity and reducing infectious complications [6]. However, due to the long-term use of antiretroviral drugs, it will cause serious side effects in the human body; in addition, it will lead to drug resistance of the virus. The toxic side effects of long-term use of antiretroviral drugs will also reduce the patient's compliance, resulting in poor treatment effect. Good compliance is the key factor for successful treatment. Compliance closely related to the degree and duration of viral suppression. Poor patient compliance can reduce the inhibition intensity of drugs on the virus, and then increase the morbidity and mortality of patients [7]. Based on the limitations of antiretroviral therapy in the actual treatment of AIDS, more than ten years ago, the study of Plant-based medicine in the treatment of AIDS, a large number of basic research found that a variety of Plant-based medicine has the effect of inhibiting HIV. Clinical trials have shown that Plant-based medicine has good effects on stabilizing and improving immune function, eliminating and relieving symptoms, and improving quality of life, but its effect on reducing viral load is limited. Plant-based medicine has the characteristics of slow onset, gentle and lasting action, small side effects, good compliance [8], which has great potential in the treatment of AIDS. In addition, some plants in nature contain new anti-AIDS chemical components [9]. By searching the keywords of AIDS suppression in plants, we waited for the hot keywords in the related research fields in Figure 1. Plants contain a variety of phytochemical components, including alkaloids, flavonoids, phenolic compounds, glycosides, tannins, saponins and other substances, so plants may have the effect of enhancing human immunity and preventing HIV replication [10].

Figure 1: Top keywords on how plants suppress AIDS.
The HIV life cycle and modes of transmission

The HIV life cycle

Immunodeficiency caused by AIDS infection is the main reason for the occurrence and development of AIDS-related diseases. HIV mainly uses CD4+ T lymphocytes as host cells for reproduction, causes CD4+ T lymphopenia and immune dysfunction in the body, making the infected individuals prone to opportunistic infections or other diseases such as tumors, and eventually leading to the death of patients [11]. Studies show that CD4 molecules on mature T helper lymphocytes being the main HIV receptors [12]. In addition, HIV can also infect macrophages and dendritic cells, which in turn causes the human immune system to lose the ability to resist the virus [13]. Figure 2 shows the process of HIV replication in cells roughly divided into 9 steps. These include adsorption, penetration, decapsidation, early protein synthesis, viral genome nucleic acid replication, late protein synthesis, nuclear shell assembly, viral particle maturation and release [14]. HIV has two glycoproteins on the outer membrane, gp120 and gp41. The gp120 subunit on the surface of HIV envelope glycoprotein and the transmembrane subunit gp41 contribute to HIV infection in host cells [14]. After HIV enters the human body, gp120 binds to CD4, the main receptor of the human cell membrane, and then binds to co-receptors such as CCR5 and CXCR4 after conformational changes. When an infection occurs, CCR5 and CXCR4 work in conjunction as co-receptors [15], and their conformational changes further expose gp41. After fusion with the human cell membrane, the viral nucleocapsid penetrates the envelope and then releases viral nucleic acid and reverse transcriptase, integrase, and protease required for viral replication. After entering the human host cell, HIV releases nucleotides into the cytoplasm of patient cells, and nucleotides are reverse-transcribed into viral DNA under the action of reverse transcriptase (RT). Viral DNA fuses with host chromosomal DNA under integrase (IN) action. The integrated viral DNA transcribed into viral gene RNA and messenger RNA, and further viral protein synthesized. The synthesis of viral proteins into mature virus particles and released from host cells under the action of protease (PR) [16]. After a series of repeated processes, the released HIV virus continues to replicate and reproduce through the above life cycle, so that the number of HIV in the human body surges.

Figure 2: HIV life cycle from cell entry to release. (By Figdraw).
Mode of transmission

Among the newly reported AIDS cases in China, 90% transmitted through sexual transmission, and nearly 9% transmitted through drug use. There are three main routes of HIV transmission: sexual transmission, blood transmission and mother-to-child transmission [17,18]. Unprotected sex with AIDS patients can lead to the transmission of AIDS, whether it is homosexual, heterosexual or between the two sexes and male homosexual behavior is more harmful. HIV infection transmitted through blood, semen, vaginal secretions, breast milk, the use of contaminated needles or syringes, and pregnancy or childbirth. It is also possible to spread HIV through incised or damaged mucous membranes [19], giving a person AIDS. Understanding how HIV transmit help to reduce the risk of HIV infection.

Pathogenesis of AIDS

During HIV infection, T cells react and proliferate slowly leading to chronic infection [19]. AIDS causes severe immunodeficiency in humans due to malfunctioning of CD4+ T cells during the late stages of HIV infection [20]. These important immune cells express receptors for antigen recognition (TCR) that bind to MHC class II molecules and participate in the antigen recognition process. HIV causes immunodeficiency by directly killing CD4+T lymphocytes and that hinders the renewal of CD4+T lymphocytes [21]. Because CD4+ T cells are pivotal in cellular immunity and regulation, their destruction inevitably lead to immune deficiency. CD4 is also the main receptor of HIV, and their assessment is therefore a convenient method for clinical evaluation of AIDS progress providing a reference for patient treatment. The human immune system is a complex entity consisting of many cell types interwoven in a structural framework, and the detection of CD4+T lymphocyte levels is only one of the ways to assess the progression of AIDS [22]. The pathogenesis of CD4+ T-cell depletion and AIDS is controversial and is the result of direct cytopathic effects of HIV, nonspecific activation of T-cell apoptosis, dysregulated cytokine production, or autoimmunity. After comparison of hypothesis experiments, it is concluded that HIV may cause immunosuppression, but not through the dreary cytopathic effect, but through the traditional virus-specific cytotoxic T cell-mediated immunopathology [19].

HIV integrates through the genome, the former viral form is present in CD4 cells, and in the early stage of infection, cytokines such as IL-2 are secreted by helper T cell 1. Under the stimulation of IL-2, CD8+T lymphocytes exert a strong immunosuppressive effect on CD4+T cells, thus keeping the virus in a latent state of suppression. In the late stage of infection, the secretion of helper T cell 2 is dominant. By secreting cytokines such as IL-10, CD8+ T cells lose their inhibition on CD4+T cells, the virus proliferates and releases new viral particles to infect more CD4+ T cells, resulting in the death of large amount of CD4+ T cells and eventual depletion and loss of immune function. In this way, the body eventually loses its immune function.

Types of HIV

Two types of HIV are identified (HIV-1 and HIV-2), which have similar viral structure and transmission route, the main difference is the difference in the envelope glycoprotein. HIV-1 is highly prevalent and aggressive, and HIV-2 discovered in West Africa in 1986 [23]. HIV-1 is the pathogen causing the AIDS epidemic worldwide. At present, the international research on AIDS based on HIV-1. Currently, HIV-2 mainly confined to various regions of Africa [24] and some countries and regions.

HIV belongs to the genus Lentivirus in the family Retroviridae in viral taxonomy. HIV RNA contains Gag, Env and Pol genes, as well as tat, vif, vpr, vpx (vpu), nef and rev six regulatory genes. gag gene encodes the core protein of the virus. env gene encodes the viral envelope protein, which is the main antigen for immunodiagnostic of HIV. Replication of viruses requires reverse transcriptase, protease, and integrase encoded by pol. Up to six regulatory genes encode accessory proteins that regulate viral protein synthesis and replication. HIV-1 originated from SIVcpz in chimpanzee [25] and HIV-2 originated from SIVsmm in mangabees [26]. Multiple and different introductions of simian immunodeficiency virus (SIV) into the human population have led to global epidemics of HIV-1 and HIV-2 [27]. SIV only cause immune deficiency when the virus spreads across species [25]. Although the origins of them are different, they are interrelated retroviruses, showing about 55% similarity in Gag and Pol genes, more than 35% similarity in translated proteins, and about 55% overall similarity in nucleotides [28]. In terms of pathogenicity, according to relevant reports, HIV-2 has less pathogenicity [29], HIV-1 is much more virulent and transmissible than HIV-2, and the course of AIDS caused by HIV-2 is slow and mild. HIV-2 patients' CD4+T cell counts decline slowly [30]. In terms of viral load in the patient's blood, HIV-1 infected people have a higher viral load than HIV-2 infected people [31]. According to relevant studies, HIV-1 and HIV-2 infected patients with similar levels of untreated CD4+T cells have similar transcription levels of Gag mRNA, this study shows that despite the generally low viral load of HIV-2 patients, significant viral transcription occurs in such infected patients [32].

Antiretroviral Therapy

As early as in the early 1990s, anti-AIDS drugs as single drug therapy have made great achievements in the treatment of AIDS [10] to cope with the global pandemic of fatal HIV. At present, six categories of anti-HIV drugs improve the pathogenesis of HIV. Non-nucleoside and nucleoside reverse
transcriptase inhibitors (NNRTIs and NRTIs), integrase inhibitors (INSTIs), protease inhibitors (PIs), viral maturation inhibitors (MIs), coreceptor CCR5 inhibitors. Zidovudine (AZT), a nucleoside reverse transcriptase inhibitor, was the first antiretroviral drug approved for use in 1987 and has shown a significant survival advantage compared with placebo in patients with advanced AIDS [33]. These drugs are the first to demonstrate that HIV infection is completely controllable and treatable, which provides new ideas for treating a range of viral targets. Antiretroviral therapy (ART) has reduced the extremely high mortality caused by infection, transforming HIV from a rapidly fatal disease into a chronic disease that can be treated [34]. Although the number of viruses reduce, disease progression delays, which prolong the survival period of patients in the process of NRTI single drug treatment, the use of single drug cannot continuously inhibit virus replication, and too long medication will lead to drug resistance and other problems. In addition, a single drug rarely reverses the immune function, which is not conducive to the recovery of the patient's autoimmune function [35]. In the mid-1990s, three protease inhibitor-based HIV drugs helped to slow down the global AIDS epidemic. Since then, antiretroviral therapy (cART) combining PI and NRTI drugs was developed, has significantly reduced the viral load of patients and improved their own immune function in clinical settings [36]. Combination antiretroviral therapy has resolved many opportunistic infections, such as Kaposi's sarcoma [37] and progressive multifocal leukoencephalopathy [38]; substantially reduced HIV-related mortality and extended the life expectancy of AIDS patients who underwent early intervention [39], which is now close to the life span of the general population. Since then, ART has become the mainstay of clinical HIV treatment. Thanks to antiretroviral therapy, the average life expectancy of people living with HIV-1 has been extended by about 14 years; If this is measured in life years, it is equivalent to the clinical use of antiretroviral drugs, which has saved millions of life years [40,41]. For example, an individual starting combination ART at age 20 expected to live into their 60s, a significant increase since the mid-1990s. Even so, life expectancy remains below that of the general population, calling for improvements. In addition, some antiretroviral drugs have significant side effects, including the risk of coronary heart disease and insulin resistance syndrome, which poses a challenge to the future promotion of antiretroviral drugs [42]. The main challenges of current retroviral therapy are as follows: 1. Drug resistance and increased genetic diversity of HIV-1. Due to the use of retrotherapy drugs, resistance too many drugs has emerged. In addition, the current direction of ART relies heavily on the targets encoded by the viral pol gene. 2. Cardiac and metabolic complications; Infectious disease specialists need to deal with dyslipidemia, insulin abnormalities, abnormal redistribution of body fat, and related diseases, which in turn lead to a significant increase in the risk of heart disease and type 2 diabetes [43-45]. The development and clinical use of antiretroviral drugs have largely changed the face of HIV infection worldwide, transforming AIDS into a chronic disease controlled by drugs [46].

**Plant Suppression AIDS Therapy**

Due to the advantages of safety, no dependence and low cost of natural resources such as plants in the treatment of diseases, some achievements have been made in the research of using plants as drugs to treat various diseases in recent years including HIV, advantages of plant inhibiting AIDS (Figure 3).

Antiretroviral therapy is efficient for clinical treatment of patients with AIDS, characterized by fulminant immunodeficiency and severe infection, and AIDS patients are prone to Kaposi's sarcoma, or other types of tumors that cause rapid death, which also leads to a low survival rate for AIDS patients. The application of antiretroviral therapy and rapid development of medicine increase the survival rate of patients and improve life quality. During the treatment of chronic antiretroviral drugs, patients may experience a number of side effects, including dyslipidemia, insulin abnormalities, abnormal redistribution of body fat, and related diseases, which in turn lead to a significant increase in the risk of heart disease and type 2 diabetes [43-45]. The development and clinical use of antiretroviral drugs have largely changed the face of HIV infection worldwide, transforming AIDS into a chronic disease controlled by drugs [46].

**Figure 3:** The advantages of plant inhibiting AIDS and some effective components in plant body.
### Table 1: Plants with HIV inhibitory activity and with clinical promise for the treatment of AIDS.

<table>
<thead>
<tr>
<th>Plant species</th>
<th>Active ingredients</th>
<th>Mechanism of action</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aureobasidium Pilatus (Flammulina velutipes)</td>
<td>Lectins, Ribosomal inactivating Protein, Fungal immunomodulatory protein</td>
<td>Inhibition of HIV-RT and HIV activity</td>
<td>Zhou et al. [49]</td>
</tr>
<tr>
<td>Wild paint (Toxicodendron succedaneum)</td>
<td>Bioflavonoids</td>
<td>Inhibition of HIV-RT activity</td>
<td>Lin et al. [50]</td>
</tr>
<tr>
<td>Hypericum perforatum (Hypericum perforatum L.)</td>
<td>St John’s wort, Pseudohypericin</td>
<td>Inhibition of HIV-IN activity</td>
<td>Sanna et al. [51]; Kubin et al. [52]; Birt et al. [53]</td>
</tr>
<tr>
<td>Ghost needle grass (Bidens pilosa L.)</td>
<td>Flavonoids, Polycyelene compounds</td>
<td>Inhibition of HIV-PR activity</td>
<td>Kim et al. [54]; Zeng et al. [55]</td>
</tr>
<tr>
<td>Galangal (Alpinia officinarum Hance)</td>
<td>Acetoxyxipiperacetate</td>
<td>Inhibition of HIV-IN activity</td>
<td>Tamura et al. [56]; Zubair et al. [57]</td>
</tr>
<tr>
<td>Mahogany (Swietenia mahagoni (L.) Jacq.)</td>
<td>Limonin</td>
<td>Inhibition of HIV-PR activity</td>
<td>Parihar et al. [48]; Dong et al. [58]</td>
</tr>
<tr>
<td>Garlic (Allium sativum L.)</td>
<td>Allicin, Flavonoids, Polyphenols</td>
<td>Inhibition of HIV-PR activity</td>
<td>Sabde et al. [8]; Kim et al. [54]; Silprasit et al. [59]</td>
</tr>
<tr>
<td>Red flower Manjusri orchid (Crinum amabile)</td>
<td>Alkaloids such as lycorine and nanchicine</td>
<td>Inhibition of HIV-RT activity</td>
<td>Ali et al. [60]</td>
</tr>
<tr>
<td>Wild grapes (Ampelopsis brevipedunculata M. Trautv.)</td>
<td>Phenolic compounds</td>
<td>Inhibition of HIV activity</td>
<td>Sigidi et al. [61]</td>
</tr>
<tr>
<td>Ox-heart Annona (Annona reticulata Linn.)</td>
<td>Alkaloids, Acetanilide</td>
<td>Inhibition of HIV activity</td>
<td>Hien et al. [62]</td>
</tr>
<tr>
<td>Laura fu wood (Rauvolfia verticillata (Lour.) Baill.)</td>
<td>Monoterpen e in dolo alkaloids papaverine</td>
<td>Inhibition of HIV-RT and HIV activity</td>
<td>Sabde et al. [8]; Stöckigt et al. [63]</td>
</tr>
<tr>
<td>Betel nut (Areca catechu L.)</td>
<td>B1 arecoline, procyanidins</td>
<td>Inhibition of HIV-PR activity</td>
<td>Vermani and Garg [64]</td>
</tr>
<tr>
<td>Fruit of Chinese magnoliavine (S. chinensis (Turcz.) Baill.)</td>
<td>Fructus schisandrae, Tripterpenoids, Nortriterpenoids,</td>
<td>Inhibition of HIV-RT and HIV activity</td>
<td>Szopa et al. [65]; Xiao et al. [66]; Xu et al. [67]</td>
</tr>
<tr>
<td>Kelp (Laminaria japonica)</td>
<td>Lectins, Sulfated polysaccharide</td>
<td>Glycoprotein receptors on T lymphocytes, Inhibition of HIV activity</td>
<td>Nakashima et al. [68]; Singh and Walia [69]</td>
</tr>
<tr>
<td>Bitter melon (Momordica charantia L.)</td>
<td>Ribosome inactivates proteins, Bitter gourd lectin, Bitter gourd anti-HIV protein</td>
<td>Inhibition of HIV-RT activity, viral core protein p24, immune cells</td>
<td>Fang et al. [70]; Lee-Huang et al. [71]; Meng et al. [72]; Puri et al. [73]</td>
</tr>
<tr>
<td>Rhizoma coptidis (Coptis chinensis Franch.)</td>
<td>Limonin, Quaternary ammonium alkaloid</td>
<td>Inhibition of HIV-PR and HIV activity</td>
<td>Dong et al. [58]; Gupta et al. [74]; Qian et al. [75]</td>
</tr>
<tr>
<td>Scutellaria baicalensis georgii</td>
<td>Flavonoids</td>
<td>Inhibition of HIV-PR, HIV-RT and HIV activity</td>
<td>Li-Weber [76]; Zhao et al. [77]</td>
</tr>
<tr>
<td>Herba violae (Viola philippina)</td>
<td>Cyclic peptide compounds</td>
<td>Inhibition of HIV activity</td>
<td>He et al. [78]; Wang et al. [79]</td>
</tr>
<tr>
<td>Chinese wolfberry (Lycium chinense Miller)</td>
<td>LBP, Quaternary ammonium alkaloid</td>
<td>Inhibition of HIV activity</td>
<td>Shah et al. [80]</td>
</tr>
<tr>
<td>Ginseng (Panax ginseng C. A. Meyer)</td>
<td>Ginseng saponin, Ginseng polysaccharide</td>
<td>Inhibition of HIV-RT activity, improve immune cell function</td>
<td>Cho et al. [81]; Kim et al. [82]</td>
</tr>
<tr>
<td>The root of remembranous milk vetch (A. membranaceus (F. Bunge.)</td>
<td>Astragalus saponin, Astragalus polysaccharides</td>
<td>Human immunomodulators, Inhibition of HIV activity</td>
<td>Chen and Huang [83]; Hijrotani et al. [84]; Rios and Waterman [85]</td>
</tr>
<tr>
<td>Artemisia capillaris (Artemisia capillaris Thunb.)</td>
<td>Dicafeoylquinic acid, Coumarin, Flavonoids</td>
<td>Inhibition of HIV-PR activity</td>
<td>Kim et al. [54]; Evers et al. [86]; McDougall et al. [87]; Tan et al. [88]; Zhu et al. [89]</td>
</tr>
<tr>
<td>Creat (Andrographis paniculata (Burm. F.) Nees)</td>
<td>Andrographolide</td>
<td>Inhibition of HIV and HIV-PR activity</td>
<td>Chang et al. [90]; Hossain et al. [91]; Reddy et al. [92]; Xu et al. [93]</td>
</tr>
<tr>
<td>Fructus arctii (Arctium lappa L.)</td>
<td>Wooden fat element, Dicafeoylquinic acid, Flavonoids</td>
<td>Inhibition of HIV-RT and HIV activity</td>
<td>Kim et al. [54]; McDougall et al. [87]; Schröder et al. [94]; Wang et al. [95]</td>
</tr>
<tr>
<td>Licorice (Glycerrhiza uralensis Fisch.)</td>
<td>Glycyrrhizin</td>
<td>Inhibition of HIV-RT activity</td>
<td>Afreen et al. [96]; Fomenko et al. [97]</td>
</tr>
<tr>
<td>Honeysuckle (Lonicera japonica Thunb.)</td>
<td>Ethyl caffeic acid, Caffeic acid</td>
<td>Inhibition of HIV-PR activity</td>
<td>Wang et al. [98]</td>
</tr>
</tbody>
</table>
Plants with Potential Value for HIV Suppression

Hypericum perforatum, also known as St John’s wort, is a perennial herb of the Hypericum genus in the Gercyaceae family. It is considered as an important medicinal plant and is often used to improve depression and other related symptoms in clinical practice. Hypericin isolated from Hypericum perforatum and pseudohypericin confirmed to inhibit HIV reverse transcriptase and viral integrase, which has great potential in the clinical treatment of AIDS [51-53]. Garlic is an underground bulb of Allium species in the Liliaceae family. Garlic is a famous edible and medicinal plant with biological activities such as prevention and treatment of cardiovascular diseases and anti-tumor. Garlic contains many active components, among which allicin has broad-spectrum antibacterial, sterilization and anti-inflammatory effects, and has strong antiviral ability. In studies, allicin, flavonoids and polyphenolic compounds extracted from garlic have been found to have strong effects on inhibiting HIV reverse transcriptase and protease [8,54,55,104]. Schisandra is a genus of Schisandra in the Magnolia family. Its fruit contains Schisandrin, vitamin C, resin, tannin and a small amount of sugars. Schisandra chinensis extract confirmed to inhibit HIV reverse transcriptase activity [67]. The triterpenoids isolated from the leaves and stems of Schisandra chinensis chinensis show anti-HIV-1 activity [66]. As a famous Plant-based medicine, the anti-HIV effect of Schisandra chinensis is worth further exploration. Bitter gourd is a bitter gourd plant in Cucurbitaceae. Bitter gourd is rich in protein, sugar, minerals and vitamins. In addition, the ribosome inactivation protein, bitter gourd lectin and bitter gourd anti-HIV protein 30 kD extracted from bitter gourd, etc. It has been confirmed that bitter gourd can selectively kill HIV-infected lymphocytes and macrophages, inhibit HIV-RT activity and inhibit viral core protein p24 [70-73]. Bitter gourd shows significant anti-HIV efficacy. Coptis is a genus of buttercups, and as a commonly used medicinal plant, it has antibacterial, anti-inflammatory and antiviral effects, used in clinical practice to clear heat and dampness, and to relieve fire and detoxify the body. The lemon bitter extract from Coptis has the effect of inhibiting HIV-PR activity [58,75], quaternary ammonium alkaloids isolated from Coptis, with significant inhibitory effect on HIV activity [74]. Coptis has HIV inhibiting properties. Scutellaria Baicalensis is a plant of the genus Scutellaria in the Labiaceae family. The root of Scutellaria Baicalensis used as a medicine. Plant-based medicine used to treat upper respiratory tract infection including cough with lung heat, yellow gallbladder with damp heat and pneumonia. Baicalein, baicalin, baicalein and other flavonoids found in the roots of Baicalin [77]. Flavonoids extracted from Scutellaria baicalensis can inhibit HIV-PR and HIV-RT activity [76]. The low drug resistance and high efficiency of inhibiting HIV in Scutellaria Baicalensis are the natural plants for us to explore anti-AIDS. The whole plant used as a medicine, which has the functions of clearing heat and detoxing, cooling blood and reducing swelling. The cyclic peptide isolated from Zi-hua, shown to have anti-HIV effect (He et al., 2011). A positive correlation between the hydrophobicity and anti-HIV activity of cyclic peptides isolated from Zi-hua was also found, and the anti-HIV activity. Moreover, this trend related to their ability to destroy cell membranes [78]. In the future, Zihua has great research value in the treatment of AIDS. Arctium burdock is a burdock plant in Asteraceae family. Arctium contains lignans such as arctin and arctigenin, and arctin strongly inhibit the efficacy of HIV replication [94]. Dicaffeoylquinic acid and flavonoids isolated from arctium burdock have been proved to have the effect of inhibiting HIV [95]. Arctium burdock has great potential in inhibiting HIV and need to be explored.

Conclusion

Drug resistance and side effects that reduce life expectancy of patients hamper AIDS treatment. A variety of phytochemicals not only inhibit the replication of HIV, but also repair the immune system damaged by the virus. A number of plants may therefore be used as new efficient drugs for the treatment of AIDS. For this purpose, it is necessary to understand the mechanism of effective chemical components of plants and conduct cytotoxicity tests on corresponding extracts to ensure the safety and effectiveness in clinical application. This requires a number of chemicals analyses, preclinical and clinical trials before that could be in place 10-20 years from now.

References


