

because you asked

Food and meds

Do certain foods interfere with antiretrovirals?

Nutritionist Michèle Cossette responds:

Consuming any food can affect the way that oral medications are absorbed into the body. Taken on an empty stomach, most medications pass quickly into the intestine, where they're absorbed into the bloodstream. Taking medications with food increases the amount of time the medication stays in the stomach, which delays its passage to the intestine and slows absorption. This doesn't mean that less medication is absorbed, just that it takes more time.

With most medications, these changes are minimal so it doesn't really matter whether you take them with food or not. But for some, even slight increases in the amount of medication in the blood can lead to toxicities; for others, even slight decreases can make the medication ineffective. It's therefore important to follow the pharmacist's recommendations for each medication.

The following medications are more effective when taken with food: etravirine (Intelence™),

atazanavir (Reyataz®), ritonavir (Norvir®), darunavir (Prezista®)/ritonavir combinations, the liquid formulation of lopinavir/ritonavir (Kaletra®), saquinavir (Invirase®)/ritonavir, and tipranavir (Aptivus®)/ritonavir.

On the other hand, didanosine (Videx®) must be taken on an empty stomach (1½ hours before eating or 2 hours after), because foods can change the level of acidity in the stomach and change the way it dissolves. Efavirenz (Sustiva®) can be taken with or without food, but it's advisable to avoid taking it with high-fat foods. Fat increases the availability of this drug and can therefore increase side effects of efavirenz and Atripla® (which contains efavirenz plus tenofovir and FTC).

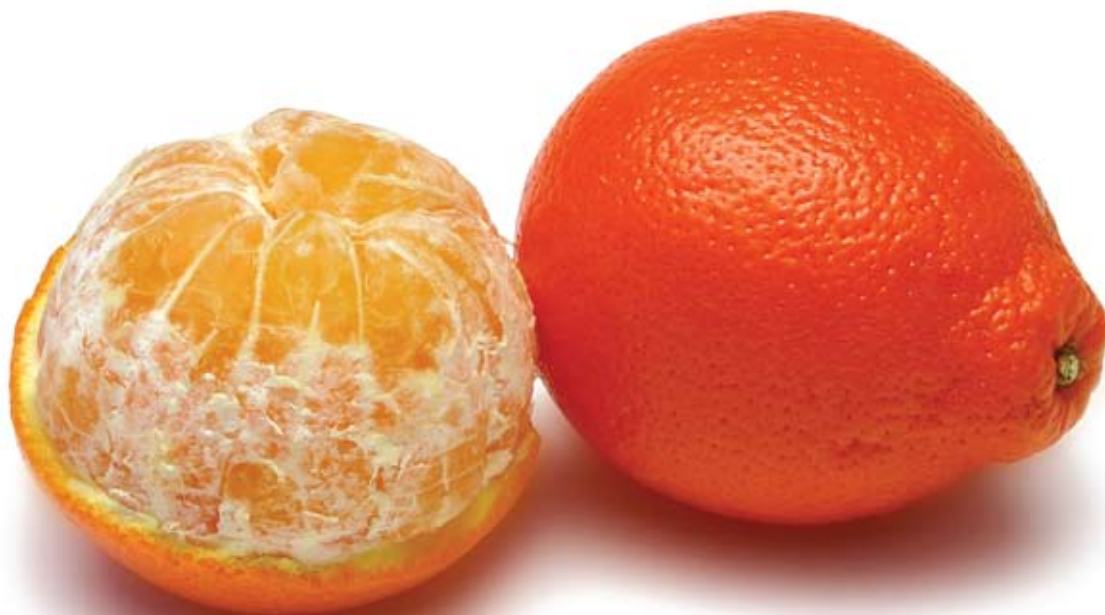
Some particular foods can interfere with medications. Grapefruit juice is known to interfere with many meds, even if consumed long before the drug is taken. The same holds true for Seville oranges (often used in marmalade), tangelos and pomegranate juice. It's better to avoid these fruits if you're taking antiretroviral medications.

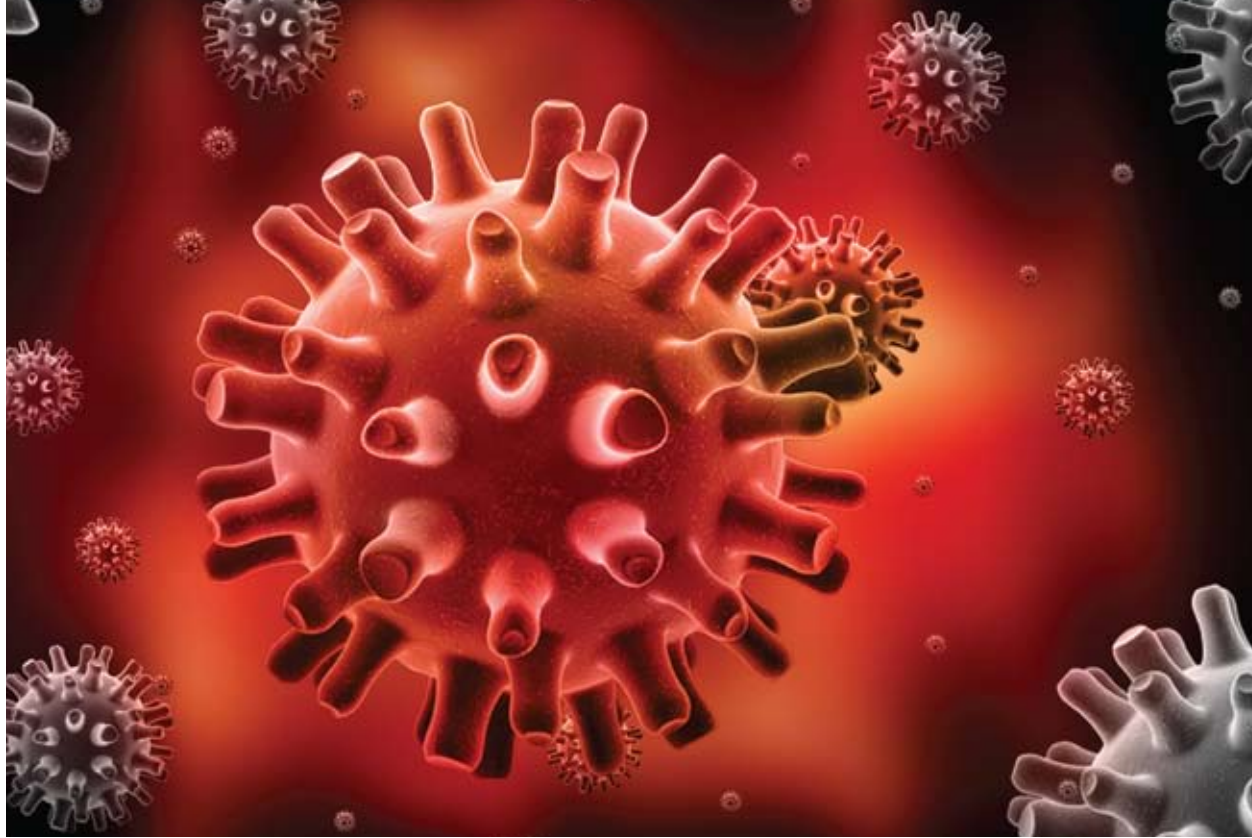
Michèle Cossette is a nutritionist with the Équipe d'intervention, de prévention et de dépistage ITSS (treatment, prevention and screening of STDs) at the Jeanne-Mance CSSS in Montreal

Dr. Darrell Tan is a Clinical fellow in Infectious Diseases at the University of Toronto.

Dr. Jean-Pierre Routy is an associate professor at McGill University and a physician in the Division of Hematology and Immunodeficiency Service at the McGill University Health Centre in Montreal.

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Herpes

I'm worried that I may have herpes. How do I know if I have it, and what does it mean for my HIV?

Dr. Darrell Tan responds: Genital herpes is a common infection caused by the herpes simplex virus type 2 (HSV-2) or, less commonly, type 1 (HSV-1). The typical symptoms of genital herpes are painful blisters or ulcers that occasionally appear in the “boxer short” area, often at times of physical or emotional stress. Both HSV-1 and HSV-2 can also cause sores around the lips or mouth, often called “cold sores” or “fever blisters.” Herpes symptoms can be more severe in people with HIV.

The usual way to diagnose herpes is to have your doctor take a swab of a blister or ulcer. Medications like acyclovir (Zovirax®), valacyclovir (Valtrex®) and famciclovir (Famvir®) can speed the healing of herpes blisters if taken soon after symptoms develop — ideally within 24 hours — and can help prevent herpes flares if taken every day. However, many people with herpes have no symptoms at all, and may not even know they're infected. Sometimes, a person can become infected with herpes at a younger age and not develop symptoms until later in life. Herpes can be transmitted by direct skin contact, even when no blisters are present.

Herpes infection is extremely common; roughly 80% of Canadian adults living with HIV are also infected with HSV-1, and 50% have HSV-2. Among

Canadians without HIV, the prevalence of HSV-1 is around 60%, and HSV-2 around 25%.

Preliminary research suggests that herpes might make HIV infection worse, but studies are still needed to figure out exactly how this happens and what significance it may have for people living with HIV. HSV-2 infection makes it easier to transmit HIV to a sexual partner, as well as to get infected with HIV. So far, studies have been unable to show that taking anti-HSV medications can decrease these risks.

Stem cells in HIV

I've been hearing a lot about stem cells in the news. Are they being used in HIV research?

Dr. Jean-Pierre Routy responds: Antiretroviral therapy (ART) is becoming a powerful tool to control HIV replication and today's regimens have become much more tolerable. However, ART is expensive and must be taken very carefully to avoid drug resistance and treatment failure. The search is still on for other strategies.

One of the most promising possibilities now at the research stage is to prevent HIV from getting inside human cells by removing the “doorknob” the virus uses to enter. Research efforts combine three of the hottest tools in medicine: stem cells, gene therapy and gene products intervention (otherwise known as RNA interference).

Stem cells circulate in the blood and are found in bone marrow and other organs. They represent

The goal in stem cell therapy for HIV infection is to modify a person's stem cells to make their offspring CD4 T cells resistant to infection

"mother cells" that can develop into several different kinds of cells, such as red and white blood cells, including CD4 T immune cells. These cells are matured and trained in the thymus before making their way back into the blood and lymph nodes. CD4 T cells coordinate the immune fight against HIV but can also become infected by HIV. Following infection with HIV, the virus uses the CD4 cells as a Trojan horse to trick their way into other cells and eventually makes these white cells dysfunctional.

The goal in stem cell therapy for HIV infection is to modify a person's stem cells to make their offspring CD4 T cells resistant to infection. After these stem cells were modified in the laboratory, they would be infused back into the person's bloodstream, where they would mature into CD4 T cells that are able to fight HIV without becoming infected.

Entry denied

Normally, HIV enters a CD4 T cell and directs the cell to make thousands of viruses that will eventually kill that cell and then move on to kill other cells in the body. Scientists are trying to prevent this process by blocking HIV from getting into cells in the first place. To get inside, the virus grabs onto a "doorknob" on the human cell. The concept is to generate human cells that lack the "doorknob," called a CCR5 receptor. To engineer these cells, short pieces of RNA that render cells unable to make the CCR5 receptor are inserted using a technique known as RNA interference.

The inspiration for this research comes from people who never catch HIV, despite having unprotected sex or sharing needles with infected individuals. These healthy people have mutations in the genes that make the CCR5 receptor, so the virus can't get into their cells*. This finding inspired the development of CCR5 entry inhibitors such as maraviroc (Celsentri®). RNA interference offers a

new and precise technique for removing the receptor completely.

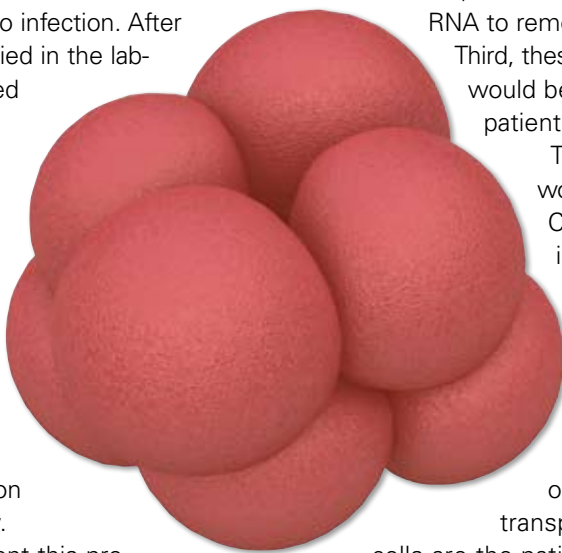
How would it work?

The technique is now being used in "cultured" cells in test tubes. The idea is to tie the knot between RNA interference and stem-cell transplants to create a new therapy for patients already infected with HIV. The treatment scenario would be something like this: first, the doctor would collect stem cells from the patient's blood using a filtering machine called a leukapheresis device.

Second, these cells would be treated with RNA to remove the CCR5 receptor.

Third, these "protected" stem cells would be reintroduced into the patient's bloodstream.

The "protected" cells would mature and become CD4 T cells. These immune cells would be healthy enough to fight infection and survive to create daughter cells that are also resistant to HIV. There would be little chance of the body rejecting the transplant because the stem cells are the patient's own cells. Potentially, the body's immune system could be re-engineered so it was protected for life, as it is following bone marrow transplantation.



Early research

While the research is promising, it's still much too soon to know which people would best respond to this type of therapy. The most effective strategy would both block HIV's entry into the cell and disrupt the virus if it gets inside. This strategy is under development in mice, and will later be tried in the Rhesus monkey, which has the same CCR5 receptor as humans. The recent discovery of the entire human genome and new technologies that allow us to screen thousands of genes at once will certainly speed up our research. **R**

*A small percentage of people with the CCR5 deletion do get HIV through the CXCR4 receptor.

Is there something
you need to know?
Please send your questions to:
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