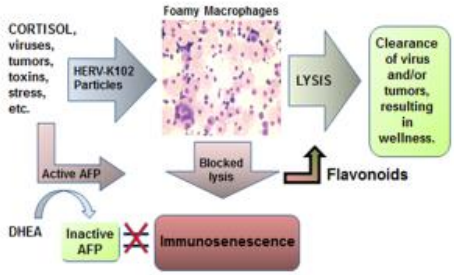


**The HERV-K Way to Keep Coronavirus (COVID-19) at Bay**

**H = HALT the use of anti-inflammatories**
**E = EXERCISE, Sleep Etiquette and EAT well**
**R = REVERSE/prevent immunosenescence**
**V = Institute VIRAL containment protocols to reduce exposures**
**K = Supplement with K (LYSINE)**

# The HERV-K Way to Keep Coronavirus (COVID-19) at Bay

By **Marian Laderoute, Ph.D. Medical Sciences – Immunology**

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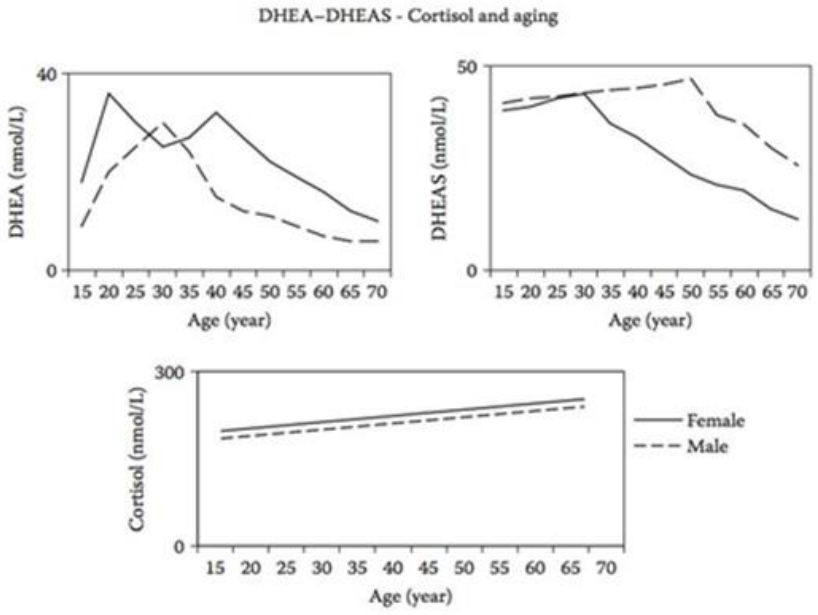
The World Health Organization (WHO) has reported that the fatality rate of COVID-19 is 3.4% but which is likely an overestimation as testing for COVID-19 exposures is not robust. More likely it is slightly elevated over the influenza virus fatality rate because of the lack of pre-existing immunity. Updated information on COVID-19 demographics[1] suggests an increased fatality risk: over the age of 50 (about 1.3 % for 50 - 59 years of age) where risk further increases significantly with age (up to about 18 % for people 80 years of age and older) and that males may be at about 1.66 times the risk of females. The fatality rate for those with cardiovascular risks is the highest (12%), followed by diabetes (8%), and then chronic respiratory disease, hypertension, and cancer (about 7% for each) whereas the fatality rate for those with no pre-existing conditions is about 0.9%. Dr. Anthony Fauci, Director of the National Institute of Allergy and Infectious Diseases (NIAID) at the National Institute of Health (NIH) has announced that the

[1] https://www.worldometers.info/coronavirus/coronavirus-age-sex-demographics/.

COVID-19 vaccine is at a minimum about one to one and a half years away. But what is most troubling is that Health and Human Services Secretary Alex Azar recently suggested that the price of the coronavirus vaccine or other anti-viral treatments may or may not be affordable to most Americans. This raises the important issue as to whether there are other clinically significant ways to help protect oneself against COVID-19 serious or fatal infections.

I think it can be argued that the answer may be yes. The demographics of the fatality risks appear to mirror the natural decline of dehydroepiandrosterone (DHEA) with age, which occurs earlier in males than females (Figure 1). Note that this does not appear to be true for the levels of DHEAS which represents the sulphated and biologically inert version of DHEA. Importantly, according to Figure 1, with age the DHEA/cortisol ratio declines. Of interest, most clinical laboratories do not test for DHEA but test instead for DHEAS, which may less accurately reflect risk of susceptibility to infections which may instead be more accurately determined by the DHEA/cortisol ratio.




## Figure 1. Sex Differences in the Loss of DHEA with Aging

From: **Watson RR (ed). DHEA in Human Health and Aging, June 7, 2017, CRC Press, Boca Raton Florida, pp 472.**

## *The Demographics of COVID-19 Fatality Rates is Consistent with a Decreased **DHEA/Cortisol Ratio** and/or **Immunosenescence** Bestowing Greater COVID-19 Risks*

The age-related decrease in the DHEA/cortisol ratio is a major determinant of immunosenescence observed during aging.[2,3] DHEA counteracts the effects of cortisol[4] and may help to revert immunosenescence.[2-4] In particular, decreased DHEA/cortisol ratios correlate with increased cardiovascular disease and all-cause mortality.[5] Most importantly, however, in age and gender adjusted logistic regression analysis for community-acquired pneumonia, cortisol (OR 2.8, 95% CI: 1.48-5.28) and DHEA (OR 2.62, 95% CI 1.28-5.34) and ratios but not DHEAS predicted all-cause mortality.[6] There is now a growing appreciation that immunosenescence rather than inflammation per se is causally related to age-associated chronic disease.[3, 7]

In terms of immunosenescence,[3] it was recently suggested that the induction of human endogenous retrovirus K102 (HERV-K102) particles which generates lipid body negative foamy macrophages and what happens next (Figure 2),[7] may be critical in determining outcomes, and thus, may impact COVID-19 severity. Depending upon the DHEA/cortisol ratio, once monocytes/macrophages are triggered by a virus, this ratio will determine if immunosenescence occurs or not. According to this definition,[3] immunosenescence then relates to the failed lytic release of the HERV-K102 protective particles and the loss of 'trained (innate) immunity'. Trained (innate) immunity refers to antigen non-specific protective immunity pertaining to the activation of

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[2] Bauer ME. **Stress, glucocorticoids and ageing of the immune system**. Stress. 2005 Mar;8(1):69-83.

[3] Laderoute MP. **A new paradigm about HERV-K102 particle production and blocked release to explain cortisol mediated immunosenescence and age-associated risk of chronic disease.** Discov Med. 2015 Dec;20(112):379-91.

[4] Prall SP, Muehlenbein MP. **DHEA modulates immune function: a review of evidence**. Vitam Horm. 2018;108:125-144.

[5] Rutkowski K, Sowa P, Rutkowska-Talipska J, Kuryliszyn-Moskal A, Rutkowski R. Dehydroepiandrosterone (DHEA): hypes and hopes. Drugs. 2014 Jul;74(11):1195-207. doi: 10.1007/s40265-014-0259-8.

[6] Mueller C, Blum CA, Trummler M, Stolz D, Bingisser R, Mueller C, Tamm M, Mueller B, Schuetz P, Christ-Crain M. Association of adrenal function and disease severity in community-acquired pneumonia. PLoS One. 2014 Jun 9;9(6):e99518. doi: 10.1371/journal.pone.0099518.

[7] https://www.linkedin.com/feed/update/urn:li:ugcPost:6637771145007505408/

monocytes/macrophages and under certain circumstances (of immunosenescence) is known to mediate atherosclerosis.[8,9]

As of February 26, 2022, we now know that immunosenescence is a key risk factor for COVID-19 severity. In bronchoalveolar (lung) fluids of patients with severe COVID-19 when compared with those with moderate COVID-19, these foamy macrophages are infected with replicating SARS-CoV-2 but only in severe cases.[10] As discussed elsewhere[11] antibodies to spike protein particularly the neutralizing antibodies (as may naturally occur in severe cases of COVID-19 or in response to 2 doses of COVID-19 vaccine) appears to enhance the infection of these foamy macrophages. This antibody dependent enhancement (ADE) of infection then abrogates the critical trained innate immunity mediated by the HERV-K102 system. Most salient is that these SARS-CoV-2 infected macrophages display apoptosis (lysis) resistance[12] and this blocks the lytic release of the protector HERV-K102 particles, thereby inducing immunosenescence. Hence, ADE is associated not only with the loss of critical protection offered by the HERV-K102 trained innate immunity system but induces immunosenescence which increases morbidity and the risk of mortality. That BSG overexpressing, SARS-CoV-2 preferentially infected macrophages[10] mediate cytokine storm associated with COVID-19 mortality was recently shown in a human BSG transgenic mouse model.[13] Moreover, these foamy macrophages are constitutively producing HERV-K102 particles in the sebaceous glands of the mucosa including in the nasopharyngeal passage, where they are called sebocytes. Sebocytes also release their HERV-K102 particles by lysis on day 6 and this waxy substance called 'sebum' is released to the mucosal surface. A more recent paper showed high viral loads in the nasopharyngeal swabs correlated with the presence of SARS-CoV-2 spike protein specific

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[8] van Tuijl J, Joosten LAB, Netea MG, Bekkering S, Riksen NP. Immunometabolism orchestrates training of innate immunity in atherosclerosis. Cardiovasc Res. 2019 Jul 1;115(9):1416-1424. doi: 10.1093/cvr/cvz107.

[9] Laderoute M. The paradigm of immunosenescence in atherosclerosis-cardiovascular disease (ASCVD). Discov Med. 2020 Jan-Feb;29(156):41-51. PMID: 32598862.

[10] Ren X, Wen W, Fan X,et al. COVID-19 immune features revealed by a large-scale single-cell transcriptome atlas. Cell. 2021 Nov 11;184(23):5838. doi: 10.1016/j.cell.2021.10.023.

[11] Laderoute M. A Smarter Pandemic Response. February 26, 2022. hervk102@substack.com, https://hervk102.substack.com/p/a-smarter-pandemic-response?utm_source=url

[12] Liao M, Liu Y, Yuan J, et al. Single-cell landscape of bronchoalveolar immune cells in patients with COVID-19. Nat Med. 2020 Jun;26(6):842-844. doi: 10.1038/s41591-020-0901-9.

[13] Geng J, Chen L, Yuan Y, et al. CD147 antibody specifically and effectively inhibits infection and cytokine storm of SARS-CoV-2 and its variants delta, alpha, beta, and gamma. Signal Transduct Target Ther. 2021 Sep 25;6(1):347. doi: 10.1038/s41392-021-00760-8.

Abs/NAbs in serum while low viral loads were correlated with the absence of antibody.[14] That sebocytes are also infected by SARS-CoV-2 was recently shown.[15] The take-home message here is that whether or not you were naturally previously infected or more recently infected with the omicron variant, or whether or not you have been partially or fully vaccinated with or without boosters, **all steps you take now to prevent or reverse immunosenescence is expected to substantially reduce your risk of SARS-CoV-2 morbidity and mortality.**

In addition, the FLCCC alliance has prepared a protocol for early treatment involving ivermectin which apparently, the main mechanism of action is by blocking SARS-CoV-2 mediated immunosenescence.[16] While USA citizens can locate doctors able to provide this early treatment on the FLCCC alliance website,[17] international patients can order their 'just in case' early treatment kits through 'The Advanced COVID-19 Tele-Health Care Center by booking an appointment at www.drpierrekory.com.

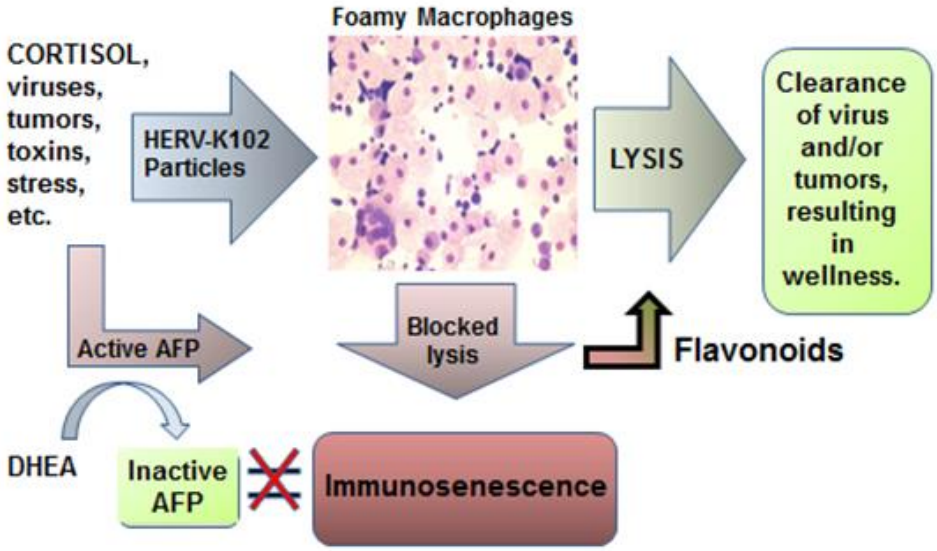
So, lets look at the steps you can take now to prevent and/or reverse immunosenescence yourself.

14 Liu W, Russell RM, Bibollet-Ruche F, et al. Predictors of nonseroconversion after SARS-CoV-2 infection. Emerging Infectious Diseases. 2021;27(9):2454-2458. doi:10.3201/eid2709.211042.
15 Huang N, Pérez P, Kato T, et al. SARS-CoV-2 infection of the oral cavity and saliva. Nat Med. 2021 May;27(5):892-903. doi: 10.1038/s41591-021-01296-8.

16 Laderoute M. Ivermectin May Prevent and Reverse Immunosenescence By Antagonizing Alpha-fetoprotein and Downmodulating PI3K/Akt/mTOR Hyperactivity. Open Heart, April 29, 2021.

**https://openheart.bmj.com/content/8/1/e001655.responses#ivermectin-may-prevent-and-reverse-immunosenescence-by-antagonizing-alpha-fetoprotein-and-downmodulating-pi3k-akt-mtor-hyperactivity.**

17 https://covid19criticalcare.com/ivermectin-in-covid-19/covid-19-care-providers/




## Figure 2. The New Immunosenescence Paradigm, 2015[3]

### *The HERV-K Way to Keep COVID-19 Coronavirus at Bay*

Due to the lack of a previous exposure to COVID-19, and that adaptive immunity requires a full 2 to 3 weeks to become newly established, means a potential way to help protect against COVID-19 severity would be to augment innate immunity, such as the HERV-K102 protector system. As extensively reviewed elsewhere,[3,7,9] HERV-K102 as a non-pathogenic protector foamy retrovirus which maps to chromosome 1 and is unique to humans, likely undergoes lytic infections in virally infected cells (although this clearly needs to be directly examined), while in normal cells or uninfected cells, it merely integrates. It has been suggested that the release of these particles featuring prominent envelope spikes, may trigger innate T and B cell responses with specificity for HERV-K envelope. This is important because, it has been shown that tumors and virally infected cells but not normal cells (reviewed in 3), express HERV-K envelope at their cell surface and serves as a beacon for cell destruction.

Compelling evidence for the potency of this 'trained innate immunity' system is three-fold. Firstly, sex trade workers exposed to HIV-1 but who remain seronegative and thus resistant to HIV-1 acquisition have abnormally elevated levels of HERV-K102

integrated into their genomic DNA as tested in the plasma of blood.[18] Secondly, early evidence suggests Neanderthals and Denisovans appear to have lost HERV-K102 from the orthologous position on Chromosome 1 [19] which may help explain why *Homo sapiens* survived over these other hominins. Thirdly, since we are born with the ability to produce HERV-K102 particles [20] and where about 50% of normal neonates have detectable HERV-K102 particles in their plasma, means it is likely the HERV-K102 system operates from birth to death. In contrast, adaptive immunity in humans develops over the first 5 to 7 years and may not be fully developed until closer to puberty. When taken altogether, it appears that the HERV-K102 HERV-K envelope host defense system may be key to human survival and as such plays an important if not pivotal role in maintaining health.

In theory, there may be ways to help exploit the HERV-K102 based protection system to reduce the risks of COVID-19 and other viruses by supporting the HERV-K102 protector 'angel' virus system,[3] and/or by preventing or reversing the chances of immunosenescence (Figure 2) which is dictated by a poor DHEA/cortisol ratio. To help emphasize potential protective interventions, I have developed an acronym HERV-K to describe the various steps that might be taken.[21]

## H = HALT the use of anti-inflammatories

First you must halt the use the use of anti-inflammatories. All anti-inflammatories are immunosuppressive in one way or another, and thus will interfere with the induction of HERV-K102 particles in macrophages and the generation of non-specific protection (trained immunity). Moreover, as argued elsewhere,[7] it may be a fallacy that inflammation per se causes chronic diseases. This is all more important to understand if you think you might be or have been exposed to COVID-19.

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[18] Laderoute MP, Larocque LJ, Giulivi A, Diaz-Mitoma F. **Further evidence that human endogenous retrovirus K102 is a replication competent foamy virus that may antagonize HIV-1 replication.** Open AIDS J. 2015 Dec 7;9:112-22. doi: 10.2174/1874613601509010112.

[19] Laderoute MP. Potential Importance of Fully Functional HERV-K102 for Potent Innate Immunity and Our Survival as Homo Sapiens Out of Africa, **LinkedIn Post**, February 29, 2016. https://www.linkedin.com/pulse/potential-importance-fully-functional-herv-k102-our-laderoute/

[20] Laderoute MP, Giulivi A, Larocque L, Bellfoy D, Hou Y, Wu HX, Fowke K, Wu J, Diaz-Mitoma F. **The replicative activity of human endogenous retrovirus K102 (HERV-K102) with HIV viremia.** AIDS. 2007 Nov 30;21(18):2417-24.

[21] Please note the disclaimer at the end of the article. This article is not meant to be misconstrued as giving medical advice.

Commonly used over the counter anti-inflammatories include the nonsteroidal anti-inflammatory drugs (NSAIDs). NSAIDs block the COX enzymes which produce prostaglandins which are inflammatory. These include aspirin, celecoxib, ibuprofen, indomethacin and naproxen. While acetaminophen (Tylenol) is not considered an anti-inflammatory but something which reduces pain and fever, it does interfere with sleep and should be best avoided or at least minimized. In addition, glucocorticoids are considered immunosuppressive and should be avoided as they induce stress and would directly contribute to a poorer DHEA/cortisol ratio.

One of the most prescribed anti-inflammatory drugs today are statins.[22] Statins reduce cholesterol and until recently, elevated cholesterol was thought to induce atherosclerosis and heart disease. Accordingly, many healthy adults are prescribed statins to prevent heart attacks. However, the cholesterol hypothesis was debunked late in 2019 (for discussion see references 7 and 9) so there is now, little basis for healthy adults to take statins. There is also some evidence to support the notion that statins may alter the effectiveness of vaccines, such as the annual influenza vaccine.[23] Statins are also known to inhibit the induction of 'trained innate immunity'[24] so it is strongly advisable to not take statins to stay healthy. It should be noted that many drugs on the market today for the treatment of chronic diseases have anti-inflammatory properties by design or as side-effects. Unfortunately, regulators have not required this testing for marketing approval and the anti-inflammatory properties of many drugs in common use remain unknown.

Herbal remedies are very popular these days and many are used because of their anti-oxidant and anti-inflammatory properties. If you are confronting COVID-19, halt the use of herbal medicines. One of the most powerful *in vivo* and *in vitro* inhibitors of HERV-K102 particle production is St. John's wort (hypericum)[12] which is used to treat depression and counteract insomnia. Indeed, the induction of HERV-K102 particle production is expected to generate 'flu-like symptoms', poor sleep and may raise cholesterol and/or increase blood pressure temporarily. This is normal, expected and will

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[22] Ridker PM, Danielson E, Fonseca FA, Genest J, Gotto AM Jr, Kastelein JJ, Koenig W, Libby P, Lorenzatti AJ, MacFadyen JG, Nordestgaard BG, Shepherd J, Willerson JT, Glynn RJ; JUPITER Study Group**. Rosuvastatin to prevent vascular events in men and women with elevated C-reactive protein.** N Engl J Med. 2008 Nov 20;359(21):2195-207.

[23] McLean HQ, Chow BD, VanWormer JJ, King JP, Belongia EA. **Effect of statin use on influenza vaccine effectiveness.** J Infect Dis. 2016 Oct 15;214(8):1150-8.

[24] Bekkering S, Arts RJW, Novakovic B, Kourtzelis I, van der Heijden CDCC, Li Y, Popa CD, Ter Horst R, van Tuijl J, Netea-Maier RT, van de Veerdonk FL, Chavakis T, Joosten LAB, van der Meer JWM, Stunnenberg H, Riksen NP, Netea MG. **Metabolic induction of trained immunity through the mevalonate pathway**. Cell. 2018 Jan 11;172(1-2):135-146.

self-resolve. The take-home message here is that you do not want to be taking herbal medicines as they invariably are anti-inflammatory. It should be noted that melatonin over 2 mg per night might also be immunosuppressive (unpublished data) and therefore melatonin over 2 mg should be avoided.

Despite a recent trend towards plant-based nutrition at the expense of animal protein, many people may not be aware that plant-based protein generally has a lysine/arginine ratio less than one[25] and thus may inhibit macrophage activation due to arginine relative excess over lysine (unpublished data). Notable exceptions are potatoes and avocados which have a lysine/arginine ratio but only about 1.5. Nevertheless, people who use plant based protein sources as supplements (including peanut butter in their smoothies), may be putting themselves unwittingly at elevated risk. Instead the best source of an excellent lysine/arginine ratio is whey protein where 23 grams of protein contains about 2133 mg of lysine and about 601 mg of arginine. This represents about a 4.0 ratio and which may be 5 to 6 times better than plant based protein supplements.[17] As well, whey protein has the most tryptophan (W) over plant based protein.[17] This is important to prevent activation of T suppressor cells through the loss of tryptophan when broken down to kynurenine (Kyn).[26] Elevated kyn/W ratios are anti-inflammatory and which would inhibit macrophage activation and the production of the protective HERV-K102 particles. Preferably, one should try to take the whey protein supplement first thing in the morning.

## E = EXERCISE, Sleep Etiquette and EAT well

The benefits of exercise for weight control, and to counteract insulin resistance in part, lies in its ability to counteract stress and restore or maintain a healthy DHEA/cortisol ratio. As mentioned above, the age associated risk of immunosenescence is due to a decreased DHEA/cortisol ratio. So, to keep our DHEA/cortisol ratio optimal for the activation and release of HERV-K102 particles, means we need to exercise on a regular basis. In addition, try taking a brisk walk at noon for about 20 to 30 minutes. The natural Vitamin D from the sun exposure is also good to promote wellness.

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[25] Liu J, Klebach M, Visser M, Hofman Z. **Amino acid availability of a dairy and vegetable protein blend compared to single casein, whey, soy, and pea proteins: a double-blind, cross-over trial**. Nutrients. 2019 Nov 1;11(11). pii: E2613. doi: 10.3390/nu11112613.

[26] Sorgdrager FJH, Naudé PJW, Kema IP, Nollen EA, Deyn PP**. Tryptophan metabolism in inflammaging: from biomarker to therapeutic target.** Front Immunol. 2019 Oct 30;10:2565. doi: 10.3389/fimmu.2019.02565.

Sleep etiquette such as going to bed at a regular time and getting 8 hours of good sleep each night helps to prevent adverse effects of poor sleep on innate immunity.[27] Tryptophan or more commonly, 5-HTP which is better absorbed, also helps to enhance sleep in part because it is considered an anti-depressant. One should also refrain from eating after supper, especially carbohydrates because this will interfere with sleep. Don't exercise too close to bedtime as well. A good guide on sleep dos and don'ts is a book by Matthew Walker.[28]

Eating well means to ensure an adequate intake of fruits and vegetables, and vitamins (to enhance flavonoid intake), for the prevention and/or reversal of immunosenescence (Figure 2). One should also be careful to avoid alcohol, nicotine, carbohydrates and even cannabis which may be anti-inflammatory[29] and which disturb sleep.[20]

It is important to avoid stress (psychological and physiological) and to use various ways to mitigate stress such as socialization, relaxation, yoga and meditation.

## R = REVERSE/prevent Immunosenescence

From the immunosenescence paradigm 2015,[3] a major way to prevent and reverse immunosenescence is through flavonoids. This is because flavonoids help reverse a poor DHEA/cortisol ratio[30] the latter which may favor immunosenescence. About 40 to 60 mg/day of combined isoflavone aglycone equivalents that are mainly genistein and daidzein might be optimal. However, it should be appreciated that flavonoids at higher levels may be immunosuppressive, so it is important to ensure one is taking adequate but not too high amounts. It may take up to 2 weeks to reach adequate tissue concentrations

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[27] Besedovsky L, Lange T, Haack M. **The sleep-immune crosstalk in health and disease.** Physiol Rev. 2019 Jul 1;99(3):1325-1380. doi: 10.1152/physrev.00010.2018.

[28] Walker Matthew. **Why We Sleep: Unlocking the Power of Sleep and Dreams**. Scribner, NY, 2017, pp360.

[29] Grotenhermen F, Müller-Vahl K. **The therapeutic potential of cannabis and cannabinoids.** Dtsch Arztebl Int. 2012;109:495–501.

[30] Bouic PJ, Lamprecht JH. **Plant sterols and sterolins: a review of their immune-modulating properties.** Altern Med Rev. 1999 Jun;4(3):170-7.

to see its beneficial effects. It is probably not too early to start supplementing with isoflavonoids to stay ahead of the COVID-19 coronavirus epidemic.

In the United States but not Canada, dehydroepiandrosterone (DHEA) or 7-keto DHEA (the latter which is not converted to sex hormones unlike DHEA), is available as a nutritional (food) supplement. About 100 mg daily has been shown to improve the DHEA/cortisol ratio in older individuals with no adverse effects over 6 months.[31] The problem however, is that what is stated as the concentration on the label does not always match with what is in the capsule. Choose reliable suppliers that can provide a certificate of analysis for the batch you have purchased and/or send a sample to a credited laboratory such as Endoceutics in Quebec City, Quebec, Canada for analysis.[32]

Zinc which may also help prevent or reverse immunosenescence, is needed for anti-viral immunity, is marketed as a way to promote/rebalance the immune system, and may directly be an anti-viral against some viruses such as influenza.[33] Optimal levels may be at about 30 mg per day of zinc citrate.

## V = Institute VIRAL containment protocols to reduce exposures

We all know the drill about frequent handwashing and coughing into our sleeves instead of our hands to reduce the risks of respiratory infections. If you are feeling sick, stay home from work until you are better. Similarly, try to avoid crowds if possible. Also avoid situations if you can where air recirculates in a confined space such as on airplanes or potentially ships.

On the other hand, visiting grandchildren with their runny noses, may not be a bad idea, as it may help induce HERV-K102 particle production such as to common cold viruses. Just be sure that you are preventing/reversing immunosenescence (see R above) before you do this.

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[31] Morales AJ, Haubrich RH, Hwang JY, Asakura H, Yen SS. **The effect of six months treatment with a 100 mg daily dose of dehydroepiandrosterone (DHEA) on circulating sex steroids, body composition and muscle strength in age-advanced men and women.** Clin Endocrinol (Oxf). 1998 Oct;49(4):421-32.

[32] https://endoceutics.com

[33] Read SA, Obeid S, Ahlenstiel C, Ahlenstiel G. **The role of zinc in antiviral immunity.** Advances in Nutrition, 2019 July 10(4): 696–710.

## K = Supplement with K (LYSINE)

Lysine (K) is frequently sold as an antiviral, along with zinc and Vitamin C. However, lysine per se does not intrinsically have anti-viral activity. Instead, lysine supports the nutritional requirements of the protector HERV-K102 foamy retrovirus, as follows.

The K in HERV-K102 means the virus uses lysine transfer RNA in order to prime for reverse transcription of RNA into double-stranded DNA for integration into the genome. All retroviruses by definition, use reverse transcription and must integrate for replication to occur. Unlike disease causing retroviruses (the orthoretroviruses) which reverse transcribe upon entry into cells, the non-pathogenic foamy retroviruses like HERV-K102 reverse transcribe at the time of release from cells and their particles contain cDNA rather than RNA. This means each particle may contain hundreds of free molecules of lysine. If we consider that HERV-K102 particle levels in plasma can go from zero particles per ml of plasma to 2.55 x $10^{11}$ particles in 84 hours,[12,34] means a substantial amount of daily lysine is required to support HERV-K102 particle production once it is induced. Note that the rapid induction of HERV-K102 particles in the blood, is consistent with its putative potency. It is recommended that between 1000 to 3000 mg of lysine is needed to support HERV-K102 particle production in the adult. If whey supplements are taken in the morning, this would provide about 2000 mg of lysine. In this case only 1000 mg of lysine would need to be added to the whey powder.

## Summary

In summary, 'The HERV-K Way to Keep Coronavirus (COVID-19) at Bay' is an affordable strategy for persons at increased risk, to boost resistance to COVID-19 by promoting and/or supporting the ability to safely activate the HERV-K102 protector virus in macrophages. This system as described elsewhere[3,7] appears to comprise a viral-anti-viral response (HERV-K102 is thought to undergo lytic infections in cells infected by viruses), and an innate endogenous vaccination, the latter which results in innate T and B cell responses. These responses recognize HERV-K envelope on virally infected cells, but which is not expressed on normal cells. This allows the antibodies and innate T cells to HERV-K envelope to eliminate virus infected cells. This putative 'trained (innate) immunity' system may non-specifically protect the host for up to 6 months once HERV-K102 particles are released. It may cost around $70 per month for the whey protein

[34] Laderoute MP. **Clues to finding correlates of risk/protection for HIV-1 vaccines.** F1000 Research, January 29, 2018, 6, 868.

supplement, the combination lysine/zinc and Vitamin C, the 7 -keto-DHEA or DHEA, the one-a-day vitamins and for the isoflavones. Savings might be had by restricting the use of alcohol, nicotine, cannabis and anti-inflammatories during this time of the COVID-19 epidemic. While this approach may help protect against COVID-19 infection or severe cases of infection, it may also protect against other seasonal viruses. As well the HERV-K way may protect against or mitigate the onset of chronic diseases through the reversion or prevention of immunosenescence associated with viral infections. The latter could represent a significant cost savings to health care insurance providers and to hospitals. As such it may help release valuable resources needed for combating the coronavirus epidemic.

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