Science in support of green tea products and EGCg to protect against (prevent) COVID-19 as well as other viral diseases

In parallel, combinations of green tea catechins, and green tea catechins + vanilloids were compared to EGCg for prevention of HIV, (this would likely prevent any virus!). Green tea catechins inhibited infectivity by 98 % at a concentration based on EGCg content of 1,000 nM. Inhibition of virus infectivity by the green tea catechins reached an IC 50 at about 100 nM. In contrast, the catechin–vanilloid mixture inhibited virus infection almost 100 % at a concentration of 100 nM and reached an IC 50 between 0.1 and 1 nM with concentrations based on EGCg content. ***These results demonstrated that the catechin–vanilloid mixture was 100 times more effective on an EGCg basis than the catechins alone.*** EGCg blocks virus infections more effectively in combination with capsicum vanilloids (ElimENOX2-XR) and other green tea catechins (ElimENOX2-XR)

The potent antiviral activity of EGCg and epicatechin gallate against HIV reverse transcriptase at 10–20 ng/mL and against RNA polymerase has been claimed as well. The effect seemed to correlate with competition for the template-primer rather than to an antioxidant action of the EGCg.

Antiviral action was demonstrated in protecting cultured rhesus monkey kidney MA104 cells against rotaviruses and enteroviruses by EGCg. The effect, however, was most pronounced when the virus was treated with the agent before infection of the cells. Both EGCg and thea fl avin digallate inhibited the infectivity of both influenza A and B virus in cultured Madin-Darby canine kidney cells. Electron microscopic inspection revealed virus agglutination and inhibition of virus adsorption to the target cells. Pre- and post-treatment of the cells themselves with EGCg produced significantly weaker effects. Inhibition of the infectivity of influenza virus by tea polyphenols has also been reported as has the inhibition of the Epstein-Barr lytic cycle and a reduction of herpes simplex virus titers all in cultured cells.

EGCG significantly inhibits the RNA synthesis of intracellular replicative intermediates
The associated mechanism may involve EGCG acting as an antagonist of the farnesoid X receptor alpha (FXRα) and the interaction between EGCG and FXRα down regulating the transcriptional activities of the HBV EnhII/core promoter **Fi**

Data also showed that inactivation of the virus occurred because of a direct destructive effect of EGCG on the HSV-1 virions.**g**

An interesting study exploring the reason for the broad-spectrum antiviral activity of EGCG demonstrated that EGCG competitively interacted with virion surface proteins to inhibit the attachment of HSV-1 to heparan sulfate Moreover, in this study, EGCG showed its broad-spectrum antiviral activities on many other viruses, including HCV, IAV, murine cytomegalovirus (mCMV), vaccinia virus (VACV), vesicular stomatitis virus (VSV), reovirus, and adenovirus. This activity was possibly related to a common mechanism: the interaction between the virus and heparan sulfate or sialic acid was inhibited **ure 1**

results showed that the anti-EBV lytic infection mechanisms of EGCG could be associated with inhibition of the MEK/ERK1/2 and PI3-K/Akt signaling pathways ructures of green tea catechins.

EGCG inhibited the attachment of adenovirus by interacting with virion surface proteins . tes the s In 1994, Chang et al. reported for the first time the anti-HIV activities of polyphenolic catechins from Chinese green tea ite

EGCG is an inhibitor of HIV reverse transcriptase. Chang et al. have reported that three catechins, namely, EC, ECG and EGCG, demonstrate strong inhibitory action against HIVRT f GTC f EGCG inhibits HIV entry into target cells. In 2002, Yamaguchi et al. reported that EGCG has a destructive effect on viral particles of HIV-1, causing a decrease in the ability of virions to infect cells and inhibiting viral attachment to cellular surfaces
Besides EGCG, Calland's group demonstrated that ECG and EGC had anti-HCV activities at an early step of the viral life cycle

a new mechanism for the anti-HCV activity of GTCs was reported by Calland and co-workers Their results indicated that after EGCG treatment, an observed bulge was found on the viral particle and this kind of structural alteration did not result in destruction or aggregation of virons. ctions its demonstrated that EGCG interacted with surface proteins of dozens kind of virions, including HCV, and this interaction led to a failure of membrane fusion mediated by heparan sulfate- or sialic acid-containing glycans a

EGCG exerted agglutination effects on virions and prevented the virus from absorbing onto the cell surface. Imanishi et al. further revealed that the anti-IV activity of green tea extracts that included EGCG possibly arose from its inhibitory effects on the acidification of endosomes and lysosomes. was a direct interaction between EGCG and the viral envelope, followed by destruction of the structure of ZIKV virions.
Ebola virus (EBOV) is among the most feared viruses and can cause Ebola hemorrhagic fever, a highly fatal disease. The recent WHO statistics showed that the 2014–2016 EBOV outbreak in West Africa had a high fatality rate of 28~75% . Reid et al. identified a host chaperon protein, HSPS5, as an important target for therapies against EBOV infection and found that EGCG, as an inhibitor of HSPS5, reduced the production of new viruses via its action on HSPS5
From the antiviral effects reported, whether the viral genome is DNA or RNA, GTCs seem to be able to function in different stages of infections of both nuclear viruses (the replication of viral genome occurs in the nucleus, such as all of DNA viruses and some RNA viruses) and cytoplasmic RNA viruses. The inhibitory effects of EGCG on multiple viruses indicate that this compound is a potential alternative agent for viral diseases.
ntiviral effects.