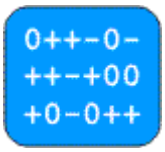

Influenza or corona virus infection: Blocking disease outbreak during the incubation period by short salicylate (ASA, aspirin) pulse therapy in combination with lung hilum hyperthermia *)

Günter Valet *(AI)*

-  *< back* *< health* *(pdf)*

Summary: Acetylsalicylic acid (ASA, aspirin) is **beneficial** for influenza and COVID-19 patients during the **incubation period** as suggested by a small group study (2010- March 2020) where a short **aspirin pulse therapy** (0.4g aspirin at detection of increased body temperature, followed by further 0.4g 12h and 24h later, resulting in a total aspirin dose of 1.2g (**Aspirin pulse therapy**)) in conjunction with temporary lung hilum hyperthermia (500ml hot beverage every 8h for 4 days) **blocked** disease outbreak in influenza or SARS-CoV-2 virus infection, while in independent clinical studies (**U4** (Oct 2020), **U9**, **U10** (Nov/Dec 2021)) the **disease course** of hospitalized COVID-19 patients, infected while under long term daily **low dose** aspirin therapy (50-100mg/d) for cardiovascular protection (risk of myocardial infarction or stroke), was **less severe**. Aspirin pulse therapy has the potential to lower the number of diseased as well as hospitalized COVID-19 patients in a comparatively inoffensive way, allowing patients to maintain their health **themselves** by generally available low cost medication.

- **Attention**: This **therapy** should **not** be used in case of **Aspirin** intolerance, with signs like gastrointestinal pain/bleeding or **Aspirin** allergy, as well as during pregnancy.
- **No** controlled medical study concerning this therapy exists so far, a major reason being the unpredictability of individual flu outbreaks in combination with the necessity for *immediate* therapy onset (15-30min) after detection of body temperature rise.
- **Studies** could, however, be performed on mostly unavailable persons in **flu quarantine**.
- **Aspirin** therapy has prevented *flu outbreak* in the author's environment since **2017**, in contrast to the previously typical 1-3 flu episodes/year.
- **No** sensible **flu infection** occured since **2022**, possibly an *antiviral*

side effect of the since then *regular* 4g/day *attenuated horseradish* intake

Background: SARS-CoV-2 infected patients may suffer from severe respiratory problems and not recover despite high intensive care efforts (**1**). Symptoms like dry cough, altered tracheal and respiratory sensation, temporary anosmia, fatigue, as well as muscle and joint pain or skin and light hypersensitivity are observed during corona or influenza virus incubation periods (37,3-37,9C rectal body temperature (BT)).

Therapy: At the recognition of such symptoms, BT is determined to exclude non infectious (<37.3C) discomfort, followed by a 400mg effervescent aspirin tablet (only adults without salicylate intolerance) and subsequently 2 tablets in 12 hour intervals (0-24h, 0,8g/d), totalling 1.2g aspirin (**aspirin pulse therapy**). Local hyperthermia is applied at the same time to the lung hilus lymphnodes by drinking 4 cups (4x150=600ml) of **55-60C** hot black or herbal tea in quantities between 10-20ml (half/full tablespoon) in *continuous sequence* to generate temporary *retrosternal warmth*. Smaller sips at higher temperature or larger quantities of lower temperature liquid do not generate the intended effect. It is recommended to start around 50C and to stepwise increase temperature according to individual tolerance to avoid thermal irritation of the oesophagus tissues. Tea drinking is continued at the subsequent time points (morning, noon or evening), followed by three hyperthermias per day (m, n, e) for 3-4 further days while the head and neck region is kept warm (cap, shawl). Patients determine ***beverage*** (high temp.range) and ***body temperature*** with an inexpensive hand held infrared thermometer by adding 0,4C to the indicated forhead skin temperature to compare with rectal measurements. They start aspirin pulse therapy at temperatures between 37.3-38C. Early COVID-19 or influenza disease symptoms (see above) are reliably recognized following some training.

This **early symptom therapy** lowers bronchial and pharyngeal symptoms as well as elevated body temperature significantly within 3-4h after the first treatment and influenza disease will typically not break out. Cough decreases and disappears within a few days as well as a certain weakness of the circulatory system during physical work. Once the disease has broken out (typically >38C), salicylate, paracetamol or ibuprofen applications may attenuate symptoms but do *not* substantially influence disease course. The described short intensive aspirin therapy seems of particular interest for SARS-CoV-2 positives under **quarantine** in case of beginning body temperature rise above 37.3C.

Therapy Development: The described therapy was developed by observation of the author's flu episodes during the past years (2010-2020) and directed towards the identification of early intervention points during the incubation period of the viral infection to inhibit potential disease progression towards pneumonia or severe acute respiratory distress syndrome (SARS, ARDS). Salicylate (ASA, aspirin) application in conjunction with lung hilum lymph node conditioning by temporary hyperthermia during the incubation period of influenza virus infections reliably stopped typical disease outbreaks in a family environment during the past 4 years (no anti-flu vaccination). Initial symptoms in March 2020 were dry cough and dryness in the upper trachea/larynx areas in two cases as well as locally distant in two other cases temporary loss of taste and smell despite absence of direct contacts for more

than four weeks prior to disease outbreak. Despite the limited number of cases, it seems worth considering this *inoffensive approach* during the actual corona pandemia.

Possible Mechanisms: The initial absence of humoral and cellular immunity (lympho-/monocytes, macrophages) against the SARS-CoV-2 virus in humans, leaves body defense in large parts to granulocytes and tissue macrophages. Granulocytes typically permeate blood capillary walls to phagocytize viruses and bacteria in lung tissue and alveoles where microorganisms are destroyed by diffusible reactive oxygen species like H2O2, molecular oxygen, hypochloric acid or enzymes such as myeloperoxidase or elastase. These effector molecules are likely to destroy bystander lung tissue, thereby preparing the ground for later superinfection by inhaled bacteria, viruses or fungal spores. Salicylates reduce granulocyte extravasation from the blood stream (2), thus lowering the tissue damaging potential. At the same time granulocyte lifetime is shortened by accelerated apoptosis (3) and certain virus infections are inhibited (4) The remaining presence of granulocytes in the blood vessels provides higher intravasal virus phagocytosis capacity, thus potentially decreasing the extent of primary viremia during the virus incubation period. Primary viremia for influenza viruses is observed in mice (5) but not in human blood donors (6).

Salicylates exhibt a certain antiviral activity at reachable serum concentrations (7) and diminish thrombocyte aggregability by irreversible cyclooxygenase blocking, thus counteracting the tendency for increased thrombus formation in COVID-19 patients (8). Hyperthermia in turn leads to lower virus replication in cells. This is partially due to a more efficient cellular antiviral response (9), and fever is accompanied by lower bacteria levels in patient blood (10). So as a tentative conclusion: Repeated temporary hyperthermias provide sufficient microorganism clearance in a structurally largely intact lung, which is less susceptible to bacterial superinfection despite salicylate induced lower granulocyte permeation into lung tissue and reduced granulocyte life span by accelerated apoptosis. Despite partial humoral and cellular immunity, the discussed sequence of events seems to equally prevail during influenza virus infections.

Intensified efforts for individualized disease outcome predictions (11 table 4) might favor the early identification of risk patients, thus permitting timely adaptation of therapy, as long as the body has not crossed the *recovery point*. Patients beyond this point die at a certain degree of bacterial superinfection in case of therapeutic inactivity, but also upon massive application of antibiotics, probably from toxic products of destroyed microorganisms.

Updates: Following perception by webcrawlers (U1) in April 2020, the described COVID-19 suppression therapy is in tendency supported by planned clinical trials (U2, U3), the observation of altered granulocyte (U5), monocyte, macrophage and thrombocyte properties (U6), public information (U8), as well as by clinical studies with patients under ongoing prophylactic low dose aspirin therapy (50-100mg/d) (U4, U9, U10). while patients with preexisting coronary heart disease do, according to present study results, do not profit from ongoing low dose aspirin therapy prior to COVID-19 (U11).

References:

1. *Thomas-Rüddel D, Winning J, Dickmann P, Quart D, Kortgen A, Janssens U, Bauer M.* "Coronavirus disease 2019" (COVID-19): update for anesthesiologists and intensivists March 2020. Der Anaesthesist (2020) **69**:225-235.

2. *Spagnuolo PJ, Ellner JJ.* Salicylate blockade of granulocyte adherence and the inflammatory response to experimental peritonitis. Blood (1979) **53**:1018-22 .
3. *Milot E, Filep JG.* Regulation of neutrophil survival/apoptosis by mcl-1. TSWIJ (2011) **11**:1948-62.
4. *CJ Chen, SL Raung, MD Kuo, YM Wang.* Suppression of Japanese encephalitis virus infection by non-steroidal anti inflammatory drugs. J Gen Virol (2002) **83**:1897-1905.
5. *Mori I, Komatsu T, Takeuchi K, Nakakuki K, Sudo M, Kimura Y.* Viremia induced by influenza virus. Microb Pathol (1995) **19**:237-44.
6. *Stramer SL, Collins C, Nugent Th, Wang X, Fuschino M et al.* Sensitive detection assays for influenza RNA do not reveal viremia in US blood donors. JID (2012) **205**:886-94.
7. *Müller C, Karl N, Ziebuhr J, Pleschka S.* D, L-lysine acetylsalicylate + glycine Impairs Coronavirus Replication. J Antivir Antiretrovir (2016) **8**:142-150.
8. *Klok FA, Kruip MJHA, van der Meer NJM, Arbous MS, Gommerse DJ, Kant KM, Kaptein FHJ, van Paassen J, Stalsa MAM, Huisman MV, Endeman H.* Incidence of thrombotic complications in critically ill ICU patients with COVID-19. Thromb Res 2020 in press
9. *Foxman EF, Storer JA, Fitzgerald ME, Wasik BR, Hou L, Hongyu Zh., Tirner PE, Pyle AM, Iwasaki A.* Temperature-dependent innate defense against the common cold virus limits viral replication at warm temperature in mouse airway cells. PNAS (2015) **112**:827-32.
10. *Weinstein MP, Towns ML, Quartey SM, Mirrett S, Reimer LG, Parmigiani G, Rell LB.* The clinical significance of positive blood cultures in the 1990s: A prospective comprehensive evaluation of the microbiology, epidemiology, and outcome of bacteremia and fungemia in adults. CID (1997) **24**:584-602.
11. *Valet GK, Roth G, Kellermann* Risk assessment for intensive care patients by automated classification of flow cytometric data. In: Phagocyte Function, Eds. JP Robinson, GF Babcock, Wiley-Liss Inc, New York 1998, p 289-306.

Updates:

U1. *Valet G* Priority corona3.html Apr 02, 2020 by webcrawler pickup (Apr.02,03,12/2020)
U2. NIH clinical trial NCT04363840 (Apr.27,2020).
U3. NIH clinical trial NCT04365309 (Apr.28,2020).
U4. *Chow JH et al.* Aspirin Use is Associated with Decreased Mechanical Ventilation, ICU Admission, and In-Hospital Mortality in Hospitalized Patients with COVID-19. Anesth Analg. (2020) doi: 10.1213/ANE.0000000000005292
U5. *Vitte J et al.* A Granulocytic Signature Identifies COVID-19 and Its Severity. JID (2020) **222**:1985-1996.
U6. *Stephenson E.* Single-cell multi-omics analysis of the immune response in COVID-19. Nat Med (2021) https://doi.org/10.1038/s41591-021-01329-2
U8. Comment Shisan(Bob) Bao, Dep.Pathology, Univ.Sydney (Australia, Dec.28,2020).
U9. *Sisinni A, et al.* Pre-admission acetylsalicylic acid therapy and impact on in-hospital outcome in COVID-19 patients: The ASA-CARE study. Int.J.Cardiol. (2021) **344** :240-245. (doi: 10.1016/j.ijcard.2021.09.058. Epub 2021 Oct 4.)
U10. *Tantry US, Bliden KP, Gurbel PA.* Further evidence for the use of aspirin in COVID-19. (2021) Int.J.Cardiol Nov 11;S0167-5273(21)01790-3
U11. *Yuan S, Chen P, Li H, Chen C, Wang F, Wang DW.* Mortality and pre-hospitalization use of low-dose aspirin in COVID-19 patients with coronary artery disease J Cell Mol Med. (2021) **25**: 1263–1273.

***) PS:** The above therapy concept was developed for family use. As a consequence of the increasing severity of the current corona pandemia, it was made available on the Internet on March 30, 2020 to hopefully decrease the number of diseased as well as of intensive care patients at a more widspread use.

- ***< Cell Biochemistry*** ***< Predictive Medicine*** ***< Concepts*** ***(pdf)***

Internet: https://www.classimed.de/corona3.html

Last update: Feb 15,2026
First display: March 31,2020
