# THERAPEUTIC NOTES

## Problems of Influenza in Pathogenesis and Therapy

DR. MED. JOHANNES JOHN



*Report on address by Dr. med. Johannes John (Baden-Baden) on the occasion of the 8th Congress of the International Society for Homotoxicology and Antihomotoxic Therapy in the course of the Autumn Congress of the Central Association of Physicians for Natural Healing Methods in Freudenstadt, West Germany on the 16th September, 1967.*

Particular reference was made in the introduction to the fact that the finding made two decades ago, based on homotoxin theory, that influenza is always a cellular phase, has been scientifically supported by later and the latest discoveries.[1,2a]

In summary and in addition to the review of influenza and its biotherapeutic treatment in the Homotoxin-Journal, Issue 5/1967, pages 265-272[3], the following was emphasized in particular, also by reference to several impressive pictorial demonstrations which are printed as well for the sake of better understanding apart from the literature references:

As can be seen from Fig. 1 (according to [1]) the influenza virus (diameter: 70-100 mu according to Schafer[1], 80-150 mu according to Rolly [2b]) is a conglomerate both of virus (a, b, c) and of cell (d) specific components, whereby the virus genome becomes the vagabonding genome, i.e. the pathogen only due to the envelope.[1]

Concerning the molecular-biological or biochemical structure of the influenza virus displayed diagrammatically in Figure 1, the following should be observed in the individual case:

a) The RNA has a molecular weight of approximately two million.[1]

b) The nucleic acid thread is connected with a protein to the ribonucleotide antigen (RNP antigen).[1]

c) Hemagglutinin + neuraminidase are enzymes which attack the mucins in particular.[1]

d) The entire structure is held together by lipoproteins, which are not induced by the genome of the virus, but originate from the membrane of the host cell.[1]

Based on a further diagrammatic display of the intracellular multiplication of the influenza virus (Fig. 2, acc. to [1]), the following has been established in particular:

re (2): Pseudo receptors or missing receptors at the cell membrane can obstruct the approach of the virus envelope to the cell membrane and thus prevent the "striptease" of the virus[4] with penetration of the genetic material into the cell.[1]

(3) - (9) show the intracellular phase, i.e. the ecliptic phase (Joklik; 1965[5]) of virus multiplication, in which infectious virus material is no longer detectable.[2a] In this case new RNA is formed firstly at the (3) or in the (4), (5), (6) cell nucleus and subsequently this or the entire RNP antigen complex — i.e. the virus structure protein — is transported into the cytoplasm.[1] The second antigenic component of the virus, namely the hemagglutinin later forming the virus envelope or the virus neuraminidase appears in the cytoplasm (8).[1]

re (9): If sufficient nucleic acids and proteins of the virus are synthesized in a cell, there follows the ripening or maturation process[2a] with shifting of the individual components in the direction of the cell periphery[1], where the same are combined in fine evaginations of the membrane of the cell (cell bays) making use of material of the cell membrane (lipoproteins) (1) to form the new virus particles[1], under segmentation of the cell membrane, and are subsequently continuously discharged (release) (1) (see [1] and [2a]).

The following was also emphasized in particular: the influenza virus causes no inhibition of the normal RNA and protein synthesis,[1] in contrast to the picorna virus (polio virus — diameter 25 mu), which can be referred to as a prototype of the cell killer.[1] The influenza virus also does not stimulate the cell DNA synthesis, it therefore does not accelerate the cell metabolism, and is therefore not a tumor-generating agent such as the polyoma virus, which has a diameter of 45 mu.[1]

Interferon,[6,7] a protein produced by the cells themselves, discovered in 1957 by A. Isaacs and J. Lindenmann, was also discussed and it was established that the therapeutic trials in virus diseases with this cell-derived protein substance have been disappointing.[1,4]

Special reference was also made in this connection to the fact that it has not yet been clarified certainly whether the site of action of interferon lies somewhere before the synthesis of the virus nucleic acids[1] or in the stage of intracellular ripening, i.e. of the intracellular completion of the virus — therefore either before (3) or only at (9)[8,9] in the diagrammatic representation of the multiplication of the influenza virus in Figure 2.

The individual stages of virus multiplication were shown clearly once again in tabular form by means of Table 1 (according to [2a] and [9]) as well as its susceptibility to antibiotics and synthetics, it being especially emphasized that it is here only a question of experimental investigations and therapeutic trials.[9]

Special reference was also made to the following:

All virostatic or virucidal chemotherapeutical agents would also be directed here specifically against one virus

**Fig. 1:** *Molecular biological structure of the influenza virus*

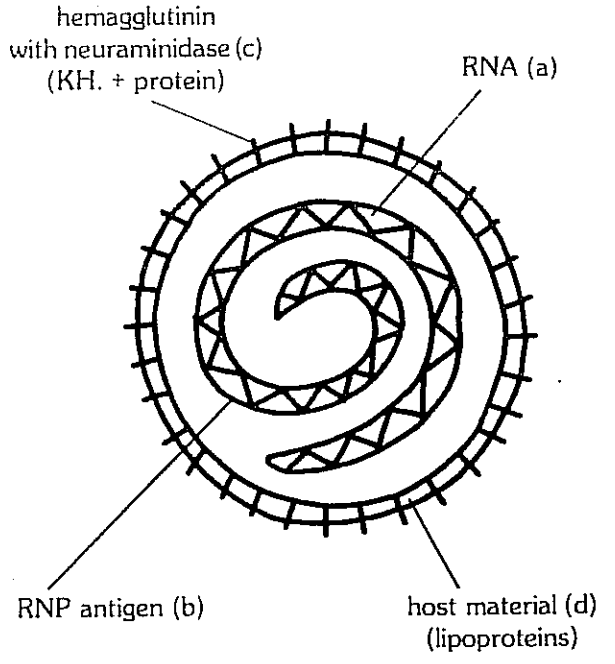
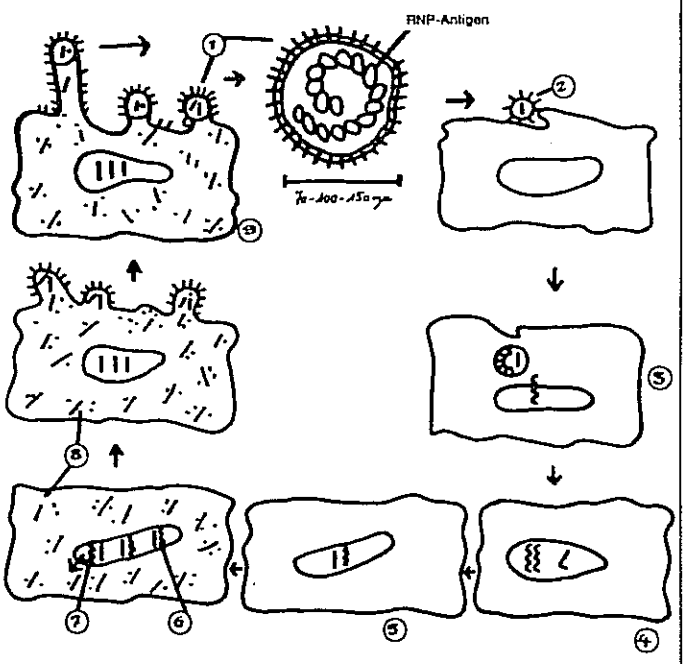



**Fig. 2:** *Multiplication of the influenza virus*




Time taken from 2 to 9 : approximately 6 hours

| = RNP antigen  ( = "early protein"

ξ = RNA  / = hemagglutinin

**Table 1:** *Stages of virus multiplication and their susceptibility to antibiotics and synthetics (experimental and therapeutic trials), according to [2] and [9]*

| Stage of virus multiplication | Influencing by antibiotics and synthetics (experimental and therapeutic trials) |
|---|---|
| Adsorption | Electrostatic: *a*-amino-p-mehtoxy-phenyl-methane-sulfonic acid*<br>Hemagglutinin (neuraminidase): RDE (Receptor destroying enzyme), potassium periodate, tannic acid |
| Penetration | Polylysin peptides (apple pectin, etc.), kethoxal adamantanamine = *B*-ethoxy-*a*-ketobutyraldehydrate) |
| Nucleic acid synthesis | Mitomycin C<br>Acitomycin D<br>5-iodine-desoxuridine (IDU) (= nucleoside) |
| Protein synthesis | Puromycin<br>p-fluophenylalanine<br>DL-methoxinine |
| Enzymes | 2-(*a*-hydroxybenzyl)-benzimidazole |
| Maturation | Isatin-*B*-thiosemicarbazone (Marboran®)<br>interferon |
| Release | *a*-amino-p-methoxy-phenylmethane-sulfonic acid*<br>xerosin |

strain.[10] A direct site of action on the virus itself would also be coupled constantly with a cytostatic (in particular bone marrow damaging!) or with a mutagenic effect.[11]

A critical opinion was also expressed on general influenza prophylaxis by protective anti-influenza inoculation and it was especially stressed here that this will indeed always remain problematical primarily with regard to the known large number of influenza pathogens, which was shown in a tabular survey (see table 2[12]).

Given the present state of knowledge about influenza, it appeared to the speaker to be tempting to provide a brief historical review of influenza, which is printed here in Table 3.[15]

It was emphasized in particular that in the individual epidemics, not only the clinical picture and serological properies of the pathogen of influenza but even the host as well have changed,[16] as shown by the example of swine influenza (hog flu) in the state of Iowa (USA) in 1919.[17]

The following noteworthy observations were made on the therapy of influenza:

1. The administration of sulfonamides and antibiotics is contra-indicated in the case of uncomplicated viral influenza, since complications are nothing short of provoked with their "prophylactic" application, which changes the normal symbiosis flora of the mucous membranes.[13]
2. The non-indicated use of highly active antibacterial agents also endangers the surroundings of the patient because the resistance of germs is promoted.[13]
3. Convincing proof that influenza can be shortened by using antipyretics or can be prevented at all by quinine has never been supplied.[13]

These three observations do not originate at all from the ranks of doctors practicing natural healing methods, but were made apodictically by the medical superintendent

**Table 2:** *Multiplicity of influenza pathogens*

**I. Adenoviruses**
(called earlier APC viruses = adenoidal-pharyngeal conjunctival)

8 older types, including
- ARD virus (acute respiratory disease)
- Type 8: epidemic keratoconjunctivitis

20 newly discovered types

**II. Common cold** = rhinoviruses

**III. Reoviruses** (= respiratory enteric orphan)
Type 1 = earlier ECHO (enteric cytopathogen human orphan) Type 10
Type 2 = ECHO 28 = RS virus (= respiratory synctial)

**IV. Enteric viruses:**
Coxsackie viruses, esp.
Type A 21 = Coe virus
ECHO viruses (esp. Type 28)

**V. Myxoviruses**
Influenza virus, Type A, B, C
Parainfluenza virus,
Type I (earlier influenza in Japan Type D)
- hemagglutinating virus
- hemadsoption virus

Type II (CA = croup associated)
Type III (= HA virus = hemadsoprtion virus)
Type IV still little researched

**II. and III.** = "Viruses of respiratory infections" (F.O. Horing; 1963[14])

**Table 3:** *Historical review of influenza (according to [15])*

| | |
|---|---|
| 412 B.C. | Hippocrates, first description of an influenza epidemic in this year |
| 1387: | large epidemic in Europe |
| 1733: | Gagliardi: gripper = to grip, possible derivation of the term grippe from this French word. |
| 1741: | adoption of the name "influenza" (or firstly in 1767) |
| 1767:<br>1782:<br>1889-90: | pandemic migrations over all parts of the Earth |
| 1918-20 | "Spanish flu"; "head flu", encephalitis lethargica (v. Economo) |
| 1919: | swine influenza in the state of Iowa (USA) = hog flu |
| 1931: | development of the culturing of viruses in incubated hen's eggs (chicken embryo culture) by Woodruff and Ernest Goodpasture |
| 1933: | discovery of the influenza virus |
| 1935: | improvement to the chicken embryo method by F.M. Burnet (Nobel Prize 1960) |
| 1938: | introduction of electron microscopy in biology and medicine by Helmut Ruska |
| 1947: | discovery of the Coxsackie virus |
| 1957-1958: | A2/Asian 'flu; Type Singapore (with severe central nervous involvement [encephalo-meningitis])<br>Antigen relationship of the virus with that of the pandemic of 1889 to 1890 — not with that of the pandemic of 1918 to 1920! |

of a medical department of a large hospital, namely by Prof. F.O. Horing (Berlin) and are recorded in the known standard work, Clinic of the Present, Issue 6/1958, page 378.[13]

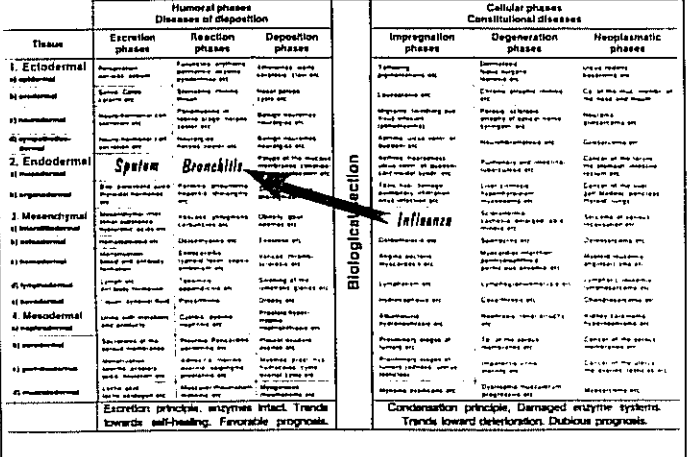
Although some years have passed since these observations from the year 1958,[13] new antibacterial or antiviral aspects of use or even proven in practice for the therapy of influenza have not emerged, as Prof. Horing documents expressly in the Clinic of the Present in 1963.[14]

4. Regarding the antipyretic therapy of influenza, the following should be emphasized according to the latest investigations of the French virologist and Nobel Prize winner A. Lwoff:[18] Even slight temperature increases lead to a considerable reduction in virus multiplication and thus to an easier course of a disease.

   Animal experimental proof: All animals which received fever-suppressing agents (antipyretics) died after infection with a heat resistant virus, while most of the animals in a control group which received no antipyretics survived.

   The saying of Parmenides (approximately 500 B.C.): "Give me the power to generate the fever and I will heal all diseases!"[19] has been supported scientifically in recent times with regard to viral diseases by a precise scientist and Nobel Prize winner.

Fig. 3: *Natural course of influenza in regressive vicariation*




A. Lwoff observed: The non-specific factors in the healing of viral diseases are now known to science and it is now time for them to be mentioned in medical textbooks and followed in practice.[18] Fever is one of the non-specific factors according to A. Lwoff. Acidity and inflammation reactions are the two other non-specific factors in the conquest and healing of virus infections.[18]

With regard to influenza therapy, it could therefore be observed with the words of Prof. W. Schafer (Tubingen) on the occasion of his ceremonial lecture on the opening of the German Therapy Week in 1966 on "Virus research today," that in the case of the influenza viruses the immunoprophylactic, the chemoprophylactic and the chemotherapeutic treatment measures are still very modest[1,4] and that no specific pharmaceutical is yet available for treating virus diseases and thus also influenza.[2,1,4,9] The objective of treatment in the case of influenza was briefly displayed based on an illustration (see Fig. 3 according to [21]), namely the elimination of the influenza virus (intracellular phase; see also Fig. 2) through the reaction phase (feverish influenza stage with bronchitis or enteritis) and through the excretion phase (characterized by expectoration or by diarrhea).[20 21]

Homeotherapy or biotherapy with Biotherapeutika-Antihomotoxika-Heel is, as is known according to homotoxin theory, a stimulation therapy with fine subliminal or slightly supraliminal stimuli, whereby the defence systems (system of major defence) in the fight with poisons (homotoxins) — a process which is generally termed disease — are administered a similar poison in addition. However, since this additional poison is more highly diluted, it represents no additional toxin burden for the organism. It acts solely as a stimulant to mobilize new defence mechanisms still lying in reserve (additional antibodies, for instance), which are directed against this new, therapeutically used toxic substance.

The preparation Gripp-Heel (tablets, ampoules) and Engystol, the composition and characteristics of the individual components of which the speaker demonstrated in detail based on the tables printed here once again (see Tables 4, 5, 6, 7, 8, 9, 10), were developed many years ago on the basis of these findings. A detailed presentation of the individual components of Gripp-Heel and of therapeutic experiences with Gripp-Heel (and Engystol) is printed in the Homotoxin-Journal, Issue 5/1967, pages 265 to 272[3], quoting numerous literature references.

**Table 4:** *Gripp-Heel*

(tablets, ampoules)
Polyvalent biotherapeutic for the therapy of real influenza and influenzal infections

| *Composition* | *in 10 g are contained* | |
|---|---|---|
| Aconitum | D 4 | 4g |
| Eupatorium perfoliatum | D 3 | 1g |
| Bryonia | D 4 | 2g |
| Phosphorus | D 5 | 1g |
| Lachesis | D12 | 2g |

**Table 5:** *Aconitum*

blue monkshood = helmet flower
The character of the helmet flower is stormy!
influenza = "lightning catarrh"
Fever agent in all commencing inflammation symptoms which are not yet localized
"A" agent in contrast to "B" agent belladonna, which is indicated in localized reaction phases with reddening.
Subsidence of shivering fits and freezing feeling
antineuralgic ("anesthesia dolorosa")
circulation instability, especially with quick pulse

**Table 6:** *Eupatorium perfoliatum*

water hemp = "leg cure"
pains in the limbs; bone, muscle, soft tissue pains
retrosternal pain with sore feeling
feeling of exhaustion
motor disquiet
lack of perspiration
Constitutional pathology:
"burnt out", "overworked constitutions, old patients, drinkers

**Table 7:** *Bryonia*

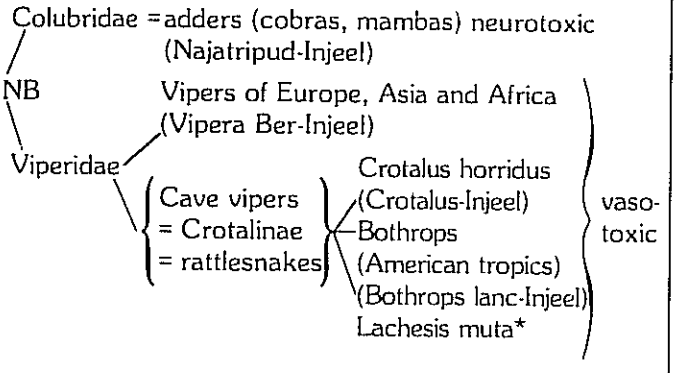
(Bryonia alba = white bryony)
Thirst, for large quantities of cold water ("cow thirst" or also for beer)
Movement impaired
Cough irritation (tracheobronchitis)
Diseases of the serous skin, such as pleuritis, but also meningism and (encephalo-) meningitis with "typhous" conditions and disturbances of the sensorium as well as synovitis ("influenza-rheumatoid")
Retrosternal pain — similar to Eupatorium perfoliatum
Headache, bursting
Constitutional pathology:
powerful, stocky types, cholerics — similar to Nux vomica type, but externally better controlled than this (also thin patients with dark face colour)

**Table 8:** *Phosphorus*

yellow phosphorus
parenchymal agent!
Elective action on the lung parenchyma for prevention and therapy of pneumonia
"Phosphorus is linked with bleeding"
Red hepatisation
Hemorrhagic rhinitis, stomatitis, tracheitis, bronchitis
Petechial skin hemorrhages
Constitutional pathology:
lanky, slim, blond persons with light skin colour, sanguine temperament, youthful

**Table 9:** *Lachesis*

Bushmaster of the South and Central America tropics
Crotalinae (= rattlesnake) group




hemolysis (tendency)
septic conditions
pallor, heaviness and pressure on the crown of the head
dry tongue, sore feeling in mouth and throat with edematous blue-red livid coloration
"everything in throat and larynx too narrow", pseudo croup
deficient leucocyte reaction (influenza from 2nd day leucopenia, therefore risk of septic conditions)
hyperesthesia, weakness, trembling, vasomotor disturbances
(also neurotoxic properties of Lachesis)

*Re poisonous snakes and snake poisons:*
*see also*

*a) Image Roach, Vol. 2/1966, pp. 16 to 21*
*b) Leeser, O.: Texbook of Homeopathy; Special part: Pharmaceutical Theory, Part C: Animal substances (1961), Haug Verlag, pp. 213 to 217, p. 208 and 209 p. 182 ff., pp. 201 and 202*
*c) John, J in* [3]
*d) Freundt, K.J.: FdM Tables for the Practice No. 10 1965, Fortschr. Med. 83, pp. 401 to 403 (1965)*

**cf. Reckeweg, H-H.: Das Homoopathikum: Lachesis: Homotoxin Journal, 6, 223 to 225 (1967)*

**Table 10:** *Engystol*

(ampoules)
Polyvalent biotherapeutic agent for viral diseases

| *In 100 ml are contained:* | | |
|---|---|---|
| a) Vincetoxicum officinale (swallow-wort) | D 8/12/30 | 60g |
| b) vegetable ashes | D 30 | 10g |
| c) sulphur (colloidal) | D 6/12 | 30g |

a) Vincetoxin (steroid glucoside) — stimulation of the body's own defences
a) asclepic acid (constituent of Vincetoxicum off.) — action on vessels and sympathetic nervous system
c) non-specific stimulants — increasing the cell oxidation deblocking disturbed enzymatic functions (sulfide enzymes)

The following was emphasized in particular, in conclusion:

Irrespective of which particular organotropism (organotropy to the upper airways such as nose and throat, pneumotropy, cardiotropy, dermatotropy, enterotropy, neurotropy) or which influenza pathogen is present in the individual case, the potentizing synergism of the individual components of Gripp-Hell and Engystol has the effect according to Burgi's principle[22] that these two preparations, individually or together, must be viewed as basic therapeutics in the treatment of influenza in every stage and at every age. Here it must be stressed in particular that side effects, secondary diseases or therapy damages occur neither after Gripp-Heel nor after Engystol.

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(2) Rolly, H.: Problems and methodical prerequisites of antiviral therapy; Therapie der Gegenwart; 105, (1966) a) Part 1: pp. 1255 to 1267; No. 10, b) Part 2: pp. 1394 to 1402; No. 11, c) Part 3: pp. 1546 to 1555; No. 12, (cf. detailed report on this publication in Homotoxin Journal, No. 4/1967, pp. 248 and 249).

(3) John, J.: The biotherapy of influenza with special reference to experience made with Gripp-Heel; Homotoxin Journal, No. 5/1967, pp. 265 to 272.

(4) ....."Current status of virus research, scientific findings and perspectives"; report on (1) in Tagliche Arztliche Praxis, No. 2 dated 28.6.1966, pp. 38 to 46.

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(7) Schneweis, K.; Interferon; Med. Welt, No. 60, pp. 2665 to 2669 (1962).

(8) quoted in (2a), p 1262.

(9) Bonon, O., Therapeutic trials for virus diseases; Monatskurse fur die arztliche Fortbildung, 16, 467 to 470 (1966) No. 9.

(10) Rolly, H., Selective chemotherapy in virus infections, experience in the treatment of herpes cornea with 5-iodine-desoxyuridine; Munch. med. Wschr., 105, 149 to 151 (1963).

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(14) Horing, F.O., Influenza and grippal diseases; Klinik der Gegenwart, Vol. 6, supplementary issue 1963, pp. E 384 a to E 384 f; p. E 384 d.

(15) somewhat modified after (13), p. 363 (1958) and after (14), p. E 284 a (1963).

(16) quoted in (13), p. 363 (1958) and in (14), p. E 384 a (1963)

(17) Hennessy, A.V., Davenport, F.M., Francis, T., jr.: J. Immunilog., 75, 401 (1955).

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a) Deutches Arzteblatt, 64, 231 (1967), No. 5, under the note "Antibody dogma shaken" and in
b) Medizinischer Monatsspiegel Merck, No. 3/1967, p. 72.

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(21) Reckeweg, H.-H.: Acupuncture, homotoxicosis and vicariation effects; Homotoxin Journal, 1, 85 to 94, (1962) - espec. Fig. 20, p. 91 -.

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