In the last chapter of the methodology series we elaborated that, using the replication problem that has afflicted medicine and now psychology, a single study, even if well published, does not make a fact. The data must also be replicable, preferably independently, preferably by other groups using the same or a similar method. Is that enough?

No. I have hinted at it and have already stated it here and there many times: Science is a social process. And an essential part of what is scientifically accepted depends on the consensus of a community of researchers and specialists. Ludwik Fleck was the first to point this out very prominently in the 1930s. He showed how difficult it actually is to determine what a syphilis spirochaete is, i.e. the bacterial pathogen of syphilis. With this example, he was able to demonstrate how crucial social processes are in the formation of scientific consensus.

You can sum up his position in the bon mot: “A scientific fact is the agreement to stop thinking.”

I will illustrate this below with an exciting current example: the “measles virus process” and the question “Does the measles virus really exist?”. Yes, this is a bit like the claim “Bielefeld does not exist”. But that too had its function.

Initial situation

In the so-called “measles virus trial”, it comes down to whether Dr. Stefan Lanka owes Dr. Bardens the prize money of 100,000 euros that was offered. This will be paid out “if a scientific publication is presented in which the existence of the measles virus is not only claimed but also proven and in which, among other things, its diameter is determined.” [1] Dr. Bardens has submitted six studies that he believes meet the criteria. Dr. Lanka has denied that they do. In a civil suit, Dr. Bardens won the award money. The defendant, Dr. Lanka, has appealed.

This is a most interesting situation: an individual who is competent in the matter disputes the consensus of the majority. He does this with the help of a provocation in the form of a prize money; otherwise, after all, no one would presumably care about this provocation. We can already consider at this point: how much sense does this tender – beyond the intended provocation – make at all? Can it even be possible in principle to want to prove any fact with a study? I can abbreviate here for impatient readers: In my view, this is not possible in principle. Nowhere. In any field. The measles virus process is a good example to illustrate this.

So the question to be addressed is *whether the papers presented – see below for details –*

are papers that are scientific and capable of providing evidence of a measles virus. I have taken a look at the six papers for interest’s sake, because I find this discussion exciting, and I want to express a few thoughts on this.

This also addresses what this is *not* about and what (to avoid misunderstandings) is also not the subject of this post:

- It is *not* a matter of clarifying *whether the measles virus exists* or not. This exists as a clinical-pathological entity, of course.
- It is also *not* a matter of clarifying whether measles are triggered and caused by any virus or not,
- and *especially not* whether measles vaccination is effective and useful.

Now what does “scientific” mean in this context?

The notion of scientificity

Scientificity is a complex construct. The fact that a text is published in a scientific journal initially says no more and no less than that competent readers and colleagues understand the text, have found it to be correct and to be good, and that editors and reviewers of the journal find the text interesting for their readership, usually a specialist audience. It says nothing about the truthfulness, nor about the quality of the information published there. In this sense, all but one – see later – of the publications presented in this process are “scientific”. They meet the minimum standard of having been published in a scientific organ.

But the concept of scientificity also includes consensual, social acceptance. This is also not a criterion of truth, but a criterion of indisputability. Controversial opinions are usually not described as “scientifically accepted” or as “scientific”, but are often dubbed “unscientific” by opponents. This usually means “opinion not accepted by the majority of those working in a field”.

If something is generally scientifically accepted, i.e. remains without significant and above all socially high-level opposition, it is usually adopted as “scientific information” in textbooks and public opinion. Then minority opinions are often marginalized and overheard until someone who manages to formulate dissent from a comparatively respected position reopens the debate.

In this sense, the assertion that “*measles are caused by the measles virus*” is undoubtedly a scientifically accepted opinion, which also implies that there is a measles virus and that it has been proven to be the causative agent. Therefore, Dr. Lanka’s position that this

“scientific fact” is due to an error and bad methodology is a minority opinion that would be called “unscientific” by the majority of scientists. This argumentation is implicitly followed by the expert opinion formulated by Prof. Podbielski, the expert appointed in the preliminary proceedings.

Now majority opinion, especially in science, is not a sufficiently good guide, even though it is more appealing to us humans as social beings, and scientists are social beings too, than individually isolated analysis. Many historical examples could be cited where and how majority opinions were wrong. Here are some examples.

As early as 2004, Dean pointed out that actually monocausal thinking is obsolete in medicine, especially in infectious diseases, because the vast majority of infections only develop as an interaction between pathogen and host [2]. But because the focus on the pathogen is easier, complexity is overlooked and monocausality continues to be elevated to the scientific standard in an unaccountable abstraction.

For a long time, the doctrine in astronomy was that planets outside our solar system did not exist. Astronomers had poor chances of advancement if they did not adhere to this dogma. Today, several hundred more planets are known.

During the Nazi regime, a majority opinion of German doctors and geneticists held the racial doctrine, which certified “non-Aryan races” as genetically, medically or otherwise inferior. As is well known, this “scientific” opinion disappeared in the blink of an eye after 1945.

A closer analysis of the nutrition debate, especially in the USA, shows: the claimed danger of saturated fatty acids and superiority of unsaturated fatty acids for disease prevention and maintenance of normal weight, which has been the majority opinion and part of official position papers for decades, is currently collapsing under the weight of contrary data, long known but ignored. The social exclusionary mechanisms of the scientific establishment had meant that minority opinions, however well-founded, were not heard [3].

All in all, it turns out: decisive discoveries come more often from the fringes of the accepted establishment than from its centre [4]. Conversely, it is also well known that economic interests often make use of cleverly positioned outsider opinions to sow doubt and halt changes that are actually sufficiently well established scientifically, as in the question of whether smoking causes cancer or human activity is responsible for the warming of the climate [5].

The same principle naturally applies in the other direction: when, as in the health sector, economic interests are very strong, very obvious facts are often overlooked because all those involved react with a denial of perception. For a different view of things could disrupt

crucial loyalties and interests [6].

In this respect, in all debates about “scientificity”, including this one, one must not forget to look at the social, economic and historical context of the concept of science just used and the values associated with it.

The dependence of methodology on social acceptance and historical circumstances and the need for revision

What is also often overlooked is the following connection: there is not something like *the* scientific method that is fixed once and for all. New methods allow new insights, make old insights obsolete or make them more precise. It is true that the experimental method, which is the main topic here, is a powerful method that has been practised for a long time. But it, too, is becoming more and more refined. For example, a simple comparison used to be sufficient for a scientifically acceptable publication. This is true for the first three of the papers in question here. Now it is standard for comparisons to be generated by chance, and in many subjects blinding is necessary. Blinding, as Sheldrake found in a survey, is not common in biological, medical or physical *basic research* [7].

While in parapsychology, for example, 85% of all experiments were blind, in basic medical research only 6% were. One can see from this that methodological stringency often also depends on how strong the awareness of the acting researchers is that in their subject there is a danger of the results being influenced by the opinion and attitude of the experimenter. Rosenthal has clearly demonstrated this through his experiments for psychology, and by analogy for medicine [8].

This is why blinding is common in clinical medical experiments and in psychological experiments, but hardly ever in basic research. “Why should we?” thinks the researcher involved, “after all, we are measuring objective facts.” But even such measurements and perceptions can be due to a wish, as Fleck was able to show with the example of the serological Aquarius reaction [9].

In the meantime, new standards are proposed, but they only gain acceptance in subdomains because they are complex and costly. This applies, for example, to systematic negative controls. One speaks of a systematic negative control when one repeats an experimental procedure in all details without carrying along the supposed causal agent. If, as we will see in this example, the experiment includes a certain form of preparation and cultivation of the cells into which nutrient solution is then introduced and finally the presumed causal agent, then a systematic negative control would consist of forming a separate group for each individual step. For only in this way would it be possible to see whether it is really only the

causal agent and not possibly artefacts that are responsible for the results obtained.

This form of controls was, to my knowledge, introduced by Jan Walleczek and his group [10]. However, it has not become widely used because it is elaborate. Interestingly, it is mostly used by researchers working in frontier areas [11].

To my knowledge, the most careful research from an experimental medical field with systematic negative controls comes from Garret Yount [12]. Here, Johrei healers, a Japanese tradition, were studied for their alleged ability to use “ki”, an immaterial form of energy, to alter cancer cells. Because initial pilot tests were positive, the researchers decided to conduct careful investigations. Cancer cell cultures were prepared and treated at a distance by Johrei healers, one master in particular. In systematic negative control experiments, the whole setting was now carried along and set up in the same way. Now a person was placed at the same distance and for the same period of time to record any temperature or electromagnetic effects. The same time and place, temperature and humidity were chosen. Systematic negative controls were also performed without a person present, just to capture the time factor and variability of the system. Through all these controls, the initial positive findings were documented as not stable enough and the “healing effect” was exposed as an artefact.

You can see from this example: theoretical models that are less accepted by the community usually have to endure much stronger controls during experimental investigation. The strength of the control usually depends on how strong the a priori certainty derived from theoretical model assumptions and unsystematic experience is that a certain state of affairs is probable. In this respect, this is always also historically conditioned and predetermined by the currently prevailing mainstream. If the general attitude is open-minded and positive, less strong data are sufficient to convince the majority of researchers. Then important controls are often omitted.

What is “scientifically proven” is thus dictated not only by method, but also by the interaction of a currently valid model with methodological considerations and with a priori considerations of the likelihood of a conjecture based on other evidence, on prevailing views, and on a background theory.

From what has been said it follows that *one* experiment, *one* publication can never give complete certainty at all and certainly cannot establish scientific fact. Rather, this happens in a complex process of exchange in which important publications become the source of discourses: they are replicated, they are criticised, they are commented on. And at the end

of a complex social process of negotiation among experts – much of which takes place not in writing but in discussions – there is then a “scientific fact”.

Therefore, the call for papers can only be seen as a provocation, calling on the scientific community to reconsider. It is interesting to see how a mainstream responder reacts to this provocation by Dr. Lanka. Let’s take a closer look at the studies.

The papers

Study #1

Enders, J.F. & Peebles, T.C. (1954) Propagation in tissue cultures of cytopathogenic agents from patients with measles. Proceedings of the Society for Experimental Biology and Medicine, 86(2): 277-286

This study reports an experimental investigation. Throat swabs, blood samples and stool samples were collected from 7 children who were clinically ill with measles, and biologically processed. These were then treated by suitable procedures in such a way that it could be assumed that bacteria, for example, could no longer be active. The substances were centrifuged and treated with penicillin and streptomycin. In addition, 2 ml of sterile, fat-free milk was added. The solution thus obtained was then introduced into various cell cultures and the changes observed microscopically and compared with untreated cell cultures. The authors found pathological changes that manifest themselves as enlargements of the cells and suggest an “immigration” of foreign substance into the cell nucleus, so that the chromatin, i.e. the chromosomes and the supporting molecules surrounding them, are pushed away. In addition, inhibitory effects are shown when the infectious isolate has been heated, introduced into other cells and then added to infected cells. Cooling, on the other hand, does not change the infectivity. This indirectly suggests that the foreign substance must be a protein.

The authors regard their findings as preliminary: “It is our purpose to describe here these observations in a preliminary manner. Additional evidence … will be sought in future investigations.” (p. 278) At the end of their paper they refer to the data as “indirect

evidence” (p. 286). This indirect evidence would have to be supplemented by two more experiments to be carried out: the direct generation of measles in monkeys and in humans with the material from the tissue cultures.

Methodological remarks and commentary:

The study was done on material from 7 children, all of whom were clinically ill with measles. Infectious material could be isolated from 5 children. The material from one child did not lead to the pathological changes in the throat swab that were observed in the material from the other 4 children. Thus, a 100% infectivity is not given. The authors certainly tried to avoid errors within the framework of the knowledge valid at the time. For example, they tried to treat their solutions by adding antibiotics in such a way that bacterial causation of pathological changes could be excluded as far as possible. However, according to current knowledge, the possibility still remains that resistant strands survived and multiplied during the relatively long incubation period (14-21 days, p. 281). However, the inoculum was filtered through microfilters that can retain Serratia marcescens, a bacterium, and the inoculum, the authors say, was free of bacteria, which they were able to show through negative growth experiments. It is therefore plausible to assume that no bacterium, at least none known at the time, was responsible for the infectious changes.

However, as the authors themselves note, it could be that other infectious agents in the monkey tissues were responsible, because only tissues from monkeys have been consistently shown to be capable of transmitting infectious agents in this and other experiments: “only those in which monkeys were employed as the experimental animal have been consistently confirmed by other workers. Great caution should therefore be exercised in the interpretation of any new claims that the virus has been propagated in other hosts or systems.” (p. 285)

So the authors urge caution, and rightly so. Whether the shortcomings noted by the authors themselves have been corrected in subsequent studies by themselves or by others is not the subject of this review. Apparently, the observations have been confirmed by other authors, as Study 2 reports.

In addition, it should be noted that while the experimental solution was treated with many different substances – sterilized milk, antibiotics, trypsin, etc. – a control solution containing the same substances without the smears or sera from blood or stool was not introduced. In this sense, the comparisons, although very convincing, are not really equivalent. No systematic negative control was included. To be fair, it has to be said that this strict control has only become common practice in recent decades – in this respect, the authors have worked cleanly according to the standards of the time, otherwise the paper would not have been published in a scientific journal – but this study cannot be clear evidence that only the

smear or the sera can be considered for the changes that were observed. At best, it is only a piece of the mosaic in a larger picture.

It is still important to point out that the authors still use the term “virus” in this text in the old, Latin sense as “infectious agent” or “poison”. For this reason, they also usually speak of “infectious agent” or “etiologic agent”.

Taken together, we can see: the study proves that it is possible to produce cell-altering processes in cell cultures using material from swabs and blood from children who are clinically ill with measles. But on the one hand, this does not happen with all materials. For another, it takes a relatively long time (2 to 3 weeks). In addition, the experimental set-up cannot ensure that only an infectious agent from the material of the sick children is really responsible for the changes and not characteristics that are inherent in the monkey cells examined and have come to light through the treatment. Finally, from today’s perspective, it cannot be ruled out that resistant micro-bacteria have led to the observed changes. The term “virus” is used here in a figurative sense. The study cannot therefore provide proof of the existence of a measles virus, but at best an argumentative building block in a necessarily more complex argumentation.

Study #2

Bech, V. & von Magnus, P. (1958) Studies on measles virus in monkey kidney tissue cultures. Acta Pathologica Microbiologica Scandinavica 42(1):75-85.

This study essentially replicates the findings of Enders & Peebles (1954) and reports two further replications that would have taken place in the intervening period. It is significant for the purposes here that the methodology was essentially replicated. The difference is that the culture media and the suspension media, in which the specimens obtained from smears and blood, respectively, were kept and grown, were different. Again, penicillin and streptomycin were used as antibacterial agents. Isolation of the infectious agent, which is almost universally reifyingly referred to as “virus” in this publication, was by throat swabs or gargling with nutrient solution or from blood. Important for further consideration at this point are the following observations:

- There were 13 patients studied, 5 of these showed positive reactions (“virus recovered”), the other 8 did not.
- Only in one of 11 patients could cultivation from blood be detected.
- The correlation of easier detectability at early collection stages claimed by the authors does not hold: 3 of the 5 agents detected were infections that occurred 24 or 18 hours earlier, and in 2 individuals the time was shorter. This contrasts with

negative findings in the 2 other patients in whom the sampling time was less than 24 hours after the onset of infection.
- The cytophatological changes reported apparently also occur in uninfected tissue of the monkey kidney and can therefore hardly be described as pathognomonic, which incidentally was also described by the other authors: “cytopathic changes similar to those caused by measles virus may be observed also in uninoculated cultures of monkey kidney tissue (Fig. 4-5). These changes are probably caused by virus-like agents, so called ‘foamy agents’, which seem to be frequently present in kidney cells from apparently healthy monkeys” (p. 80).

This latter observation in particular seems to me remarkable, pointing to the non-specificity of the very pathological changes that served as the starting point for the visual evidence of infection in the first publication by Enders & Peebles.

In support of the thesis that it is a “virus”, it is cited that a “complement fixation test” was positive. This was carried out on a total of 4 patients. Under the plausible assumption that the patient numbers reported in Table 2 refer to the patients originally reported in Table 1, there would therefore be two patients among the four in whom no virus could originally be isolated, one in whom this was successful, and the fourth patient is new. It remains unclear in how many patients this fixation test was done with a negative result, or why it was not done in all of them.

Enders & Peebles had cautioned that only experimental causation of the disease by isolate in monkeys or humans could prove causation. Therefore, an infection experiment was carried out on two laboratory-bred rhesus monkeys. One of the two monkeys showed subclinical symptoms. In both of them, a corresponding antibody titre had been detected.

Methodological notes and commentary:

The study suffers in principle from the same weaknesses as the original study by Enders & Peebles:

- There is a possibility that the changes were caused by resistant strains of bacteria not covered by the antibiotics.
- There is a possibility that any substances in the solution media are responsible for the changes.
- There is a possibility that an interaction between solution media and monkey cell leads to the observed change.
- The rate in 5 patients out of 13 is less than 50%, far from Koch’s postulate of 100% infectious causality [13].
- Transfer of the disease to the monkey organism was successful in 1 out of 2 cases. An

> antibody titre was found in both; a previous infection with measles was excluded. However, against the background of the statement that a “foamy agent” in the kidney cells of the monkeys could just as well be decisive for the change, this statement loses convincing power, since it also cannot be ruled out that the same “foamy agent”, which is naturally present in monkeys, could have led to the detected antibody reaction.

Linguistically, it can be noted that in the course of a year and three intervening and cited publications, the opinion that the infectious agent is a “virus” is apparently taken for granted, because practically all that is spoken of is the “virus”. This is an interesting example of how reality is created through concepts instead of reality becoming concept-forming.

In summary, this study cannot prove that there is “the” measles virus. What the study does show is that there is an infectious agent that can be detected in less than 50% of cases, but that could just as easily have already been present in the cells. It could also, which the authors overlooked, have originated somewhere in the breeding media or in the interaction. This could only have been ruled out by systematic negative controls, which were not common at the time.

Study #3

Nakai, M. & Imagawa, D.T. (1969) Electron microscopy of measles virus replication. Journal of Virology, 3(2): 187-197.

This study provides an electron-microscopic description of the infectious agent in question, here already referred to as “measles virus”. It mentions at the beginning earlier work that was supposed to have described sizes of 100-150 nm or 120-250 nm. Here, the different stages of replication of the virus are to be shown. For this purpose, the so-called “Edmonston strain” of the virus “propagated in HeLa cells [14]” is used. The literature cited refers to the original work by Enders & Peebles (1954), study #1 above. The extraction of the virus is not described; the publication allows two interpretations here: 1) The original isolates of Enders & Peebles, which were introduced into cell cultures by them at the time, were also used here. 2) The methods described by Enders & Peebles for obtaining infectious material were also used here. Which of the two interpretations is correct is difficult to say. These were introduced into new HeLa cells, mixed with various reagents, grown further and purified by means of four increasing length centrifugation steps, apparently with the idea that at the end the lightest particle, the virus, would remain in the filtrate and thus be available for inspection through the microscope. Various differently shaped structures were

found (“the virions are pleomorphic”, p. 189), ranging widely in size from 180 to 600 nm.

The treatment of the controls is not mentioned. The publication only says: “Control preparations of uninoculated HeLa cells were examined in a similar manner” (p. 188). This can be interpreted as meaning that the untreated HeLa cells were also subjected to a similar, stepwise centrifugation and were also examined microscopically. However, this can also be interpreted as meaning that the control cells were provided with the same reagents in the sense of a systematic negative control. Since this is not mentioned, and it can be assumed that it would have been mentioned, since it would have been a complex production step, it cannot be assumed that such systematic negative controls were produced. Nothing is reported about the findings in control cells. The figures show only experimental cells, no controls for comparison.

The authors write that the cytoplasmic inclusion bodies they observed, i.e. inclusions in the cytoplasm of infected cells, could be related to the formation of new virus particles, but describe this as speculation that would need to be confirmed by similar studies with clear immunological labelling. The same applies to inclusion bodies in the cell nucleus. It is unclear how these relate to possible virus replication: “The relationship between the nuclear inclusion body and the replication of measles virus is not clear.” (p. 196)

Methodological remarks and commentary:

The validity of the study is based on three assumptions that are not clear in the context of the publication:

- The study makes the assumption that the method of Enders & Peebles is suitable to isolate an infectious agent; in any case, this study is given as a reference for the isolate. Further details on the extraction were not given. This may be because the recovery method was generally accepted at the time, or because it was simply applied here. It remains unclear whether the agent was newly obtained or has continued to be cultured in cell lines since Enders & Peebles, i.e. for 15 years.
- The study assumes that by re-growing the infectious agent and filtering or centrifugation, only the infectious agent is isolated.
- The study assumes that the reagents that have been added to the HeLa cells to prepare the samples are irrelevant.

It remains unclear, above all, how the supposed virus was grown and propagated in the cells. The authors’ choice of words (“The Edmonston [15] strain of measles virus [6 – this refers to Enders & Peebles 1954; publication 1 above], propagated in HeLa cells, was used in this study.” p. 187) does not contribute to clarification. This is the only indication of how the infectious agent was obtained.

A systematic negative control, i.e. a control condition that was treated in the same way as the experimental cells, including staining, incubation, etc., does not appear to have taken place. Rather, untreated cells were apparently simply inspected. What exactly happened to the control cells is not reported. The publication does not say anything about whether structures of a similar nature were found in the control cells.

In passing, the size variance of the structures found seems remarkable: previous studies report sizes of 100-150 nm, or 120-250 nm. Here, particles of the size range 180-600 nm were found.

In summary, despite the suggestive evidence and images, these studies do not provide evidence in the strict sense. To do so, a systematic negative control would have had to be performed, and it would have had to be clearly reported that no evidence of similar particles was found in these controls. Now, of course, a proponent may say that this was self-evident and therefore not worth mentioning. Although such an argumentation is understandable, in a strict sense, at least one sentence on this statement would have been necessary here. These facts together: namely that it is unclear where the infectious agent came from, or how exactly the controls were handled, and that it is unclear whether anything was visible in the controls and if so what, they render this study useless as an argumentative tool.

Publication #4

Lund, G.A., Tyrrell, D.L.J., Bradley, R.D. & Scraba, D.G. (1984) The molecular length of measles virus RNA and the structural organization of measles nucleocapsids. Journal of General Virology, 65: 1535-1542.

In this study, the structure of measles virus RNA was to be investigated by electron microscopy. To do this, a strain of virus was grown and introduced into cells. These were then incubated for 72 hours and, after 90-95% of the cells had shown clearly visible cytopathological effects, subjected to a purification method. From this, the presumptive virus isolate was obtained, which was then further investigated. For this purpose, the supernatant fluid was treated several times and centrifuged so that ideally the virus would remain. The result was examined by electron microscopy to determine the structure, size and shape of the viral RNA.

Part of the study is an electron microscopic image of a representative virus (Figure 3a). The authors note that the diversity of shape and size (“pleomorphic” p. 1537) previously reported by Nakai & Imagawa (1969) was again found here. While Nakai & Imagawa (1969) reported a size range of 180-600 nm, particles between 300 and 1000 nm were found here, i.e. about a factor of 1.5 larger than in the case of Nakai & Imagawa. The virion shown has a

size of 500nm and is thus about in the middle of the scattering range.

Furthermore, structures were visually examined, length measurements were made and the fine structure of the nucleocapsids was recorded, i.e. those protein structures that contain the viral RNA. Calculations are made on their shape, length and quantity within a virion, which are not further relevant to the question at hand here.

Methodological remarks and commentary:

Control experiments are not reported in this publication. At first glance, this does not seem necessary, but it also exposes the potential weakness of the entire chain of argumentation. This publication is based on the assumption that, through infection and cultivation, a virus can indeed be isolated, which can then be characterized and further investigated. If this assumption is correct, then the shape, size and diversity of the measles virus reported here is indeed proven. If it is false, then the characteristics reported here belong to another particle.

This shows that, as is generally the case in science, the publication relies on the cumulative truth in the literature, i.e. on previous experiments and papers. This saves time and makes sense to a certain extent. But it also obviously increases the dependence on error. For if, initially purely hypothetically, cell components had been transported on from cells by the reported procedure, then all analyses would refer to such components, which would then have been (mis)interpreted as virus particles. Such an oversight could only be ruled out if a single, unambiguous systematic negative control, i.e. a control procedure in which all steps (enrichment, incubation, staining, addition of reagents and nutrient solution) had been carried out *without* the original inoculation with presumably infectious material. However, this has not been done, at least in the literature available here.

Therefore, it could be purely theoretical that what is visible here is not a virus from the measles isolate, but e.g. one that has been contained and grown further in the cell lines, or a mixture thereof. Since the observed particles are “pleiomorphic”, i.e. they can have many shapes and also very different sizes, the question of whether a particle can now be assigned to a specific virus population is probably not so easy to answer.

The discussion is reminiscent of that reported by Ludwik Fleck, who, using the Wassermann reaction and various staining techniques, first had the syphilis spirochete produced as a fact

[16]. Fleck came to the view that a scientific fact was an agreement. So similarly, it can be assumed here that it is an agreement to call the particles found a measles virus. An “objective”, methodologically independent fact can hardly be justified by this. For this would require an important requirement to have been fulfilled in this publication – or in predecessor publications on which this publication is based – which cannot be discovered in the texts available here:

Systematic negative controls would have had to have been carried out that could have ruled out the possibility that the propagated, cultivated and multiplied components actually came from the virus isolate and not from the cell cultures themselves. After all, there is the theoretical possibility, which has also been repeatedly advocated by a minority [17], that cancer cells themselves contain infectious agents, such as bacteria or viruses. If this were so, then the culturing and enrichment procedures used here would further cultivate and isolate them in the same way as the introduced inoculum.

That the image shown in Figure 3 in this publication depicts a particle containing RNA that has been measured, characterised and described in detail seems plausible to me. However, whether this particle originates from the measles inoculum or from the cells themselves is not clearly evident. The fact that this is not discussed as a problem may mean two things:

1. There is one publication where this has been done and to which all other publications refer. The ones discussed above are certainly not among them, and no evidence for this claimed possibility is evident in any text so far.
2. It has not yet been identified as a methodological problem.

It seems to me that b) is the most likely variant: if there were methodological awareness of the problem, then every author publishing on the subject would feel compelled to cite the relevant reference or would refer to it with a sentence in the methods or discussion section. Since this is not done, the problem has most likely either not been recognized, or if recognized, then not found relevant.

In summary, this study does come up with a clear picture, which may well be addressed as a viral particle. However, both the addressed multiformity and the size variance, together with the discussed lack of a systematic negative control in all studies, make it reasonable to doubt that the image offered is indeed an image of a measles virus. Only a morphological analysis of the many figures and unambiguous characterization, e.g. based on immunological methods, and above all robust evidence that they *cannot* be cultivations from the cell cultures would remove any doubt.

Publication #5

Horikami, S.M. & Moyer, S.A.. (1995) Structure, transcription, and replication of measles virus. In: V. ter Meulen & M.A. Billeter (Eds) Measles Virus. Current Topics in Microbiology and Immunology 191 (pp. 35-50). Springer: New York, Heidelberg.

This paper summarizes almost 120 other papers in one review and deals exclusively with the structure of viral RNA, gene coding and corresponding studies. It thus presupposes that the question of interest here has been answered and is not in itself relevant to the question of interest here. However, it does show that a very rich research network has been established by researchers who obviously all work under the consensus that the viruses isolated here are indeed derived from measles. The correctness of this assumption is neither discussed nor problematized, but is obviously assumed. This underpins the factuality. Whether there are indications in the details of the explanations, recognizable to specialists, that gene sequences or the behaviour of RNA is typical for certain viruses, is beyond my competence to determine. However, it is clear that nothing is said in this review about the method of isolating the virus itself and the methodological validity of this very first step. Rather, this is taken for granted as a methodological matter of course. Whether this is the case cannot be determined on the basis of this and the previously discussed publications. Formally, it should perhaps be borne in mind: even if Springer is a very good publisher, such edited works tend to be subjected to a gentle review.

Publication #6

Daikoku, E., Morita, C., Kohno, T. & Sano, K. (2007) Analysis of morphology and infectivity of measles virus particles. Bulletin of the Osaka Medical College, 53(2): 107-114.

This study analysed morphology and infectivity of the measles virus. At the outset, the authors note that several other studies have concluded, including those discussed above, that the infectious agent is polymorphic and has been observed in various sizes between 180 and 600 nm, and 300 and 1000 nm. In addition, the separability into three fractions was reported. This is continued here. The Edmonston strain is also used, with no further details on how it was obtained. Various cells, including those from monkeys, but also human cell lines are infected with it. These are incubated and grown for 7 days before infected cells are obtained via centrifugation and microfiltration. These are subjected to electron microscopy, both conventional and with immunological markers.

As in the other studies, polymorphic particles determined to be measles viruses are revealed. They have sizes ranging from 50 nm to 950 nm. All particles in each size formation are infectious. Most particles have a size of 300 to 500 nm and are thus in the range of sizes observed by others. Particles can be labelled with different immunological methods and thus show different fine structures.

Methodological remarks and commentary:

Formally, it should be noted that the “Bulletin of the Osaka Medical College” is a rather peripheral journal that cannot even boast an impact factor at the moment. Even some journals that publish largely in German, such as “Der Schmerz”, “Der Psychotherapeut” or “Forschende Komplementärmedizin” have impact factors, which shows that their work is cited by other authors. The journal’s self-description on its website suggests that there is no peer review, only internal review. The journal is mainly used by members of Osaka Medical College to communicate their findings. So it is not a “high-ranking” publication, and one might actually have expected a groundbreaking finding such as the clear electron microscopic description to be published in a more widely circulated journal.

This study, like all others, rests on the acceptance and validity of the extraction method. Therefore, we have the same problem as with all other studies: the extraction of the isolate follows the well-known scheme. Here it is presented even more briefly: “MeV, the Edmonston strain, was inoculated.…” (S. 108). With “MeV” short for “measles virus” and “the Edmonston strain”, the discipline-specific research tradition is served.

We saw in publication #3 that the same wording is also present here and a reference to the original study by Enders & Peebles (1954) was used, which is omitted here. One can therefore assume that again either a virus cultivation was carried out according to the same method as in Enders & Peebles, or, more likely, that the infected cell line of that time was used to obtain the isolate. But this again means: everything that has happened and failed to happen since then also happens or fails to happen here. This can be the introduction and further cultivation of another agent, or the further propagation of substances or agents in the cell cultures. Since no systematic negative control has been made, this cannot be decided here either. As convincing as the pictures and analyses are, and as suggestive as the research tradition is: it cannot be ruled out that an infectious agent of a different nature or cellular components were isolated and presented here from the measles culture. The short statement “MeV, the Edmonston strain…” does not allow a decision. Control experiments are not mentioned.

In the end, this study is also not suitable for answering the present question.

Discussion and consequences

What do we learn from this situation? I summarize: none of the studies perform a truly robust negative control in which it is ensured that the potentially infectious agent is not already present in the starting material, the monkey kidney cells or the HeLa cells. Either the introduced agents themselves, or these interacting with the cell material, or this alone, or all together with the isolate from the diseased tissue could be responsible for the observed changes.

In this sense, it seems to me that the challenger, Dr. Lanka, is right: a single study will not prove that the measles virus exists, and certainly not one of those presented here.

But why then the consensus in science, which obviously feels disturbed in its business by such a troublemaker as Lanka? This can be seen in the expert opinion of Prof. Podbielski, who points out that the picture only emerges from the overall view of *all* the findings, including the studies not discussed in this trial.

Science is always a cumulative-social process. In the course of all infectiological theorizing, the consensus emerged that measles must be an infectious process. Somehow, everyone expected that it would be possible to isolate something like a virus. So the a priori expectation was high that a study would have to have such a result at some point. And so the totality of researchers looks somewhat benevolently at the methodological weaknesses of the first studies, even if their authors urge caution. The citation tradition suddenly creates factuality, which even – if they were available – later negative studies would not be able to revise so easily.

This was recently demonstrated very vividly in an example where a false theory was supported for years despite sufficient negative findings, simply because the most powerful authors supported the false theory and systematically suppressed negative findings. It was the theory that a certain form of myositis was caused by amyloid deposits. It was only many publications later and after a great deal of effort that it became apparent, on the one hand, that the theory was wrong, and, on the other hand, that this false opinion had come about because facts had been created through citation networks [18].

This factuality becomes all the more difficult to doubt the longer it is handed down and the longer it is accepted by everyone. Yes, but: “there are all these genetic studies, all these electron microscopic studies!” the proponent will say. Right. The question Lanka has raised, which seems perfectly valid, is: were the *very first* data, to which all later studies refer, really collected in such a way as to isolate beyond doubt only the presumed causal agent? As we have seen, this is not the case. In the first studies – and none of the other studies

presented have remedied the shortcoming – no negative controls were included. So agents already present in the monkey cells, the famous “foamy agent”, agents created by interaction, agents introduced by the additives, or agents created by interaction with the HeLa cells, or a mixture of these, could be responsible for the subsequent changes observed. Since all subsequent methods and studies appear to be based on these initial studies, the argument does not appear to have been dispelled.

It would be dispelled by presenting a study that eliminates the problem. Either that does not exist, or the plaintiff has not found it and has not submitted it.

This is an interesting situation. I am curious to see how the court decides. Actually, from my point of view, what should happen now is this:

An excellent laboratory would have to do the isolation of the suspected measles virus all over again and, using systematic negative controls, do a cultivation showing that the accompanying procedures – nutrient solution, cell introduction, transfer to a cell strain – do not lead to infectivity and the observed changes, and then subsequently characterize the virus electron microscopically and biochemically. This study would need to be pre-registered and pre-arranged with a top-tier journal for publication, regardless of the outcome.

Or else, a knowledgeable researcher should pull the publication from the files where this happened. The publications provided do not do the job. More likely, everyone will go back to business as usual, because challenging a consensus that has lasted almost half a century is quite costly.

The measles virus trial may perhaps provide a little food for thought. The discourse will only be really kick-started when a really well-heeled virologist takes up the challenge. Perhaps Mr. Lanka should take his money and go to a good laboratory and arrange for such a study? Maybe that would help. But here, too, I am sceptical. Because: science is socially conditioned and subject to the same weaknesses as all other social interactions. And here, too, with enough chutzpah and tenacity, the majority opinion can be challenged if one is prepared to take the beating that is to be expected at first. Whether change subsequently occurs depends on two factors:

- Whether one is actually right, and it turns out that the majority has been wrong so far, and
- whether one manages to get a spokesperson to speak this truth that finds a sufficient amount of ears.

We can be excited. We are presently witnessing a historic process in which truth is being

negotiated. Lanka’s challenge has pointed out that consensual truth is less certain than it seems. Bardens has attempted to meet the challenge with his response. The studies presented, as the analyses above show, are less strong than one might think. The fact that this does not call into question that measles can be dangerous, that vaccinations may help, etc., is not addressed at all. What is being discussed is the majority consensus that what has happened in science so far is sufficient to prove the factuality of the measles virus. This seems doubtful to me after all I have seen so far. Given the major replication problem in medicine [19], and the doubt it threatens in society, it would probably be wise for a few competent researchers to set out to dispel these doubts through careful replications. Once and for all. Or, alternatively, to reopen the books. At the moment, it seems to me that both are possible, but nothing is definitively proven.

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