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Commentary

Can the Definitions of SARS-Cov-2 and Covid-19 Stand Up to Epistemological Scrutiny?

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The understanding and handling of what is known as the Covid-19 pandemic is based on the validity and legitimacy of genomic epidemiology with its taxonomies that include the immense SARS-Cov-2 corona subclass of genomic sequences. For a taxonomy based on genomic sequences to be pertinent to genomic *epidemiology* (as opposed to genomics *tout court*), its classes of sequences would have to correspond clearly to epidemiological data; and yet there is no such correspondence. The reduction of epidemiology (macro-biology) to genomics (nano-biology) is far from trivial and cannot simply be taken for granted. Against this background, we argue that the definitions of SARS-Cov-2 and Covid-19 do not stand up to epistemological scrutiny: these definitions do not hook on to a new natural kind that is pertinent for epidemiology.

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1. *Introduction*

Scientific taxonomy in any field seeks to carve nature at its joints: its aim is to capture natural kinds. These are objects, patterns or processes that distinguish themselves as salient features in nature independently of our theories and our ways of detecting them. Thus, for instance, the various types of elementary particles are natural kinds that particle physics detects. The elements of the periodic table of elements are natural kinds that chemistry discovers. Water is a natural molecular kind. Cats, horses, humans, etc. are natural kinds of living beings. More to the point of this paper, pulmonary tuberculosis, the various types of cancer, etc. are natural kinds in medicine, more precisely natural kinds in nosology: they are salient types of diseases that exist independently of our methods of diagnosis.

In this paper, we examine whether SARS-Cov-2 or Covid-19 are a natural kind. We do not take issue with the purely genomic taxonomy of various types of coronaviruses. Our concern is with genomic epidemiology. If the taxonomy employed by genomic epidemiology is to track natural kinds – genuine nosological entities in this case –, it must rely on epidemiological data. It cannot take the reduction of epidemiology to genomics for granted and derive its taxonomy from the choices that are made in genomics for the purposes of this discipline. However, no epidemiological data exist to justify the conclusion that Covid-19 is a new natural kind of nosology.

In sections 2 and 3 of this paper, we address the reduction of epidemiology to genomics. In sections 4 and 5, we consider the PCR test and how it has been employed to define Covid-19 cases. We argue that this procedure departs in significant ways from established scientific norms in the diagnosis of a disease. We conclude that the PCR test is an unsuitable means to establish Covid-19 as a genuine nosological entity. Section 6 briefly sums up the results of the paper.

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2. *The relationship between genomics and epidemiology*

"Coronavirus disease 2019" or "Covid-19" corresponds to a base-four definition proposed in December 2019 [1]. A genomic sequence counts as a coronavirus sequence if and only if it satisfies the coronavirus definition D, whose extension, at any given time, thus is the class C of coronavirus sequences. Until December 2019, six definitions D_1, ... D_6 existed, identifying six classes of C_1, ... C_6 of coronavirus sequences. Let us call this the "previous taxonomy". Furthermore, unclassified coronavirus sequences existed that satisfied D but none of the definitions D_1, ... D_6. In December 2019, further unclassified sequences were found, which also satisfied D but none of the definitions D_1, ... D_6. A seventh definition D_7 was proposed at the very end of December 2019, with extension C_7 as new subset of C [1]. Let us therefore call C_1, ... C_7 the "current taxonomy". The seventh definition D_7 gave rise to the claim of the existence of a new coronavirus C_7, dubbed SARS-CoV-2, with the declaration of a specific coronavirus pandemic caused by the spread of this virus and all its medical as well as societal and political consequences.

The definition D_7 is now satisfied by millions of genomic sequences; its extension C_7 is immense. Hence the inevitable question arises: what do all these sequences have in common? The new definition D_7, of course; but the question is whether there are any specific macro-biological phenomena to which all and only the genomic sequences that satisfy D_7 give rise. In genomic *epidemiology* – by contrast to genomics *tout court* – it is by no means sufficient for D_7 to have a purely genomic meaning. It must also have an epidemiological significance that must be established by macro-biological, epidemiological data. While the genomics community is certainly entitled to choose its taxonomy for its own internal purposes, we are here faced with a novel form of genomic epidemiology, namely the attempt to define a clinical entity based purely on a genomic taxonomy.

The fundamental premise of coronavirus genomics is that such viruses can be defined, told apart and understood purely in terms of genomic sequences. Let us therefore formulate the following genomic completeness assumption: *the genomic sequence is a complete virological description* – with "completeness" understood in an appropriate way that does not refer to anything outside genomics. The idea is that nothing of any nano-biological relevance is missing or would have to be added. The fundamental premise of coronavirus genomic *epidemiology* then is that the corresponding diseases and symptoms and macro-biological manifestations can be understood and managed in terms of genomics alone. This is a bold assumption, which we will scrutinize in this paper.

The genomic sequence is too detailed and fine-grained a description for most purposes. On its own, a single base-four number – out of millions – is practically meaningless; it can only acquire (macro-biological) meaning by belonging to the corresponding class. This is where taxonomy comes in. In order to make sense of the bewildering multiplicity of genomic codes, coarse graining is undertaken, with coronavirus subclasses, picked out by definitions, replacing the sequences themselves as fundamental entities. So far this looks like rather harmless set theory. But what about *macro*-biology? If the first class of base-four numbers caused the common cold, the second class death within a month, the third violent sneezing, the fourth 40+ fever within a day, the fifth diarrhoea, etc. – if it were really as simple as that –, then genomic sequences would naturally represent a legitimate basis for macro-biology, even epidemiology, which could then be understood and managed accordingly; how this *would* work is clear. However, as things stand, there is no established evidence of any such pattern.

To the virologist versed in base-four numerology, certain base-four definitions and classifications could well look nano-biologically more *natural* than others. But even if we grant the idea of *genomic* natural kinds, there is no reason why they should automatically correspond to *epidemiological* natural kinds. In fact, this very correspondence or extension would be one way of understanding the macro-nano reductionism that cannot be taken for granted and indeed has to be established *a posteriori*; or rather, one could either choose to define macro-nano reductionism in terms of this correspondence between natural kinds at such different levels, or even the other way around: one could *define* epidemiological natural kinds by extension from genomics, taking macro-nano reductionism for granted. But again, there is no *a priori* reason why natural kinds from such completely different scales should match. Indeed, this would rather be a way of testing the genomic definitions: do the classes they pick out really correspond to different epidemiological statistics? Such falsifiability would make genomic epidemiology *scientific*. In the Popperian view of science, one demands *falsifiability*, that is to say, precise, unambiguous, nontrivial predictions that are subject, at least in principle, to experimental refutation.

We have no objection to base-four epidemiology as a research programme; but its taxonomies – and notably the coronavirus taxonomy – will clearly evolve if research continues. In a few decades, they may look nothing like the current ones. Even if one endorses the principled reducibility claim of a macro-theory such as epidemiology to a nano-theory such as genomics, this claim does not imply that genomic taxonomy has any significance for current epidemiology. In epidemiology, nano-biology is subordinate to macro-biology. The issue then comes down to the mentioned one: it is – at most – possible to conjecture epidemiological implications from data about genomic sequences. To count as scientific, such conjectures must be falsifiable by means of epidemiological data.

To further elaborate on this crucial issue, consider the general debate about reductionism in the philosophy of science. Let us suppose, for the sake of the argument, that physicalism is true: everything in the natural world is a physical entity. For instance, there is no *élan vital*; everything that there is about living organisms is identical with some physical configuration of matter and its physical properties (token identity). Let us further, for the sake of the argument, assume that the strongest version of reductive physicalism, namely *a priori* reductive physicalism is true: the complete physical description of the natural world *a priori* entails all the other true propositions about the natural world, that is, also the propositions that use macro-biological classifications [2].

The point at issue, then, is that such an *a priori* entailment relation applies only to *final* physics and *final* biology. Obviously, no one would claim that our current physical theories and their taxonomy are the final physics, and no one would make such a claim about our current biology. Hence, even if *a priori* reductionist physicalism is granted, nothing follows about the current micro-taxonomy having any relevance for the current macro-taxonomy. The genomic completeness assumption may well turn out to be vindicated, but only in terms of a final genomics. It cannot be taken for granted as far as our current genomics is concerned. As things now stand, to establish the relevance of genomic taxonomy for epidemiology, one cannot resort to claims about the reducibility of final, ideal theories, but must establish concrete conjectures about matches in taxonomy that are subject to scrutiny by empirical, macro-biological data.

One may object to this demand by invoking functionalism. Macro classifications are coarse grained: they focus on general functional roles such as causal roles characterized by some general macro effects for which data are collected. Any such macro roles can be realized by various micro types (multiple realizability). The objection then is this one: there may be a new micro type, it may give rise to specific macro effects, but these effects do not show up as a new macro classification, because the macro classifications are generic and coarse grained.

But this is not true: if there are specific effects of a new micro type – in the case at hand specific epidemiological effects of a new coronavirus C_7 (SARS-CoV-2) –, then these are detectable on the macro level, and can be classified on that level: in the case under consideration, then, effects exist that can be classified as a new type of respiratory disease. One can then introduce a new functional sub-type of the general functional type of respiratory diseases (see [3] for the general argument). Indeed, Covid-19 is intended to be a new sub-type of the general nosological type of respiratory disease. If this is so, specific macro-biological phenomena must exist that are characteristic of this sub-type. Hence, again, we are back at having to come up with concrete conjectures about micro-macro correlations that must be subject to scrutiny.

3. *The lack of specific epidemiological data for Covid-19*

At first glance, it may seem that this demand is satisfied: after D_7 was introduced in December 2019, the world changed conspicuously. Mass panic, infections, declared Covid-19 deaths, mask mandates, lockdowns, etc. ensued worldwide. That change with all its medical, societal and political consequences, one could contend, is in itself a kind of correlation between the genomic classification given by D_7 and macro-biological data. Hence, D_7 must surely single out a natural kind – for how can one have mass panic, excess deaths and so on without a new natural nosological kind?

However, this reasoning is obviously circular: it presupposes that which needs to be established. Of course, the proclamation of D_7 in the actual context of media attention and reactions by scientists and politicians that provoked mass alarm changed the world profoundly. This context of media attention, and reactions by scientists and politicians *presupposed* that D_7 singled out a new natural kind of respiratory disease – dubbed "Covid-19" – that was dangerous for the general population. But the point at issue is that the correlation between coronavirus sequences of the class C_7 (SARS-CoV-2) and a new natural kind of respiratory

disease (Covid-19) cannot be presupposed. It must be established by macro-biological data; that is, data that do not depend on taking such a correlation for granted. Otherwise, the scientific criterion of falsifiability cannot be satisfied.

The issue of gathering macro-biological data that are correlated with C_7 becomes even more complicated because innumerable sequences satisfying D_7 are known to have been circulating before December 2019, when the definition D_7 was introduced. These sequences are epidemiologically indistinguishable from C_S, the class of seasonal coronaviruses: no macro-biological data available before December 2019 were considered to be specific for a new type of respiratory disease ("Covid-19"). What, then, is the justification of the taxonomy that has been established since December 2019?

There seem to be two replies possible: (1) it took the new virus satisfying D_7 several months to "warm up"; (2) 2020 saw the identification of a deadly new strain of a genomic type of virus satisfying D_7 that had already been in circulation before that time. (1) is far from convincing; (2) would constitute a taxonomic problem that would make one wonder even more about the taxonomy now established: if (2) is correct, then there simply is no new genomic type of coronavirus that gave rise to the Covid-19 epidemic and, hence, "Covid-19" does not designate a new kind of respiratory disease as a separate nosological entity.

Hence, again, the point at issue is a pattern of correlations between genomic sequences satisfying D_7 and appropriate macro-biological data. If there is a pandemic, the most pertinent macro-biological data obviously are excess deaths. However, the total mortality statistics are too ambiguous to suggest a clear pattern. Consider the excess deaths that occurred in Lombardy between March and May 2020, which contributed much to provoking mass alarm in Europe and beyond. We now know that many mistakes in managing the virus outbreak were made; but we have no idea what portion of the problem those mistakes represented. If a ministry of defence adopts wildly counterproductive (strategic, military) measures, many deaths may be expected. The same applies to a ministry of health. If most general practitioners essentially go on strike, important macro-biological numbers are likely to be affected. This is more or less what happened in Italy in early 2020 [4]. However, even if we assume that management and behaviour were perfect and no mistakes were made – all deaths being related to the new definition D_7 –, we still lack a clear pattern: it could be that only a small part of C_7 is deadlier than the seasonal part C_S of C, while the rest of C_7 cannot be told apart from C_S.

Already in March 2020 John Ioannidis warned that panic is not helpful but counterproductive [5]. The extraction of a purely virological mortality signal from a background of mortality noise due to factors such as panic, mismanagement, alarm bordering on hysteria, blunders, counterproductive policies as well as neglected diseases and comorbidities is problematic. How do we distinguish signal from noise? Were the excess deaths of early 2020 due to an emotional over-reaction (which undeniably began in early 2020) or to newly identified pathogens, which are now known to have existed before late 2019?

If all countries showed a pattern of excess deaths in 2020, one could use the excess deaths as the basis for a conjecture linking a new type of coronavirus (C_7) with a new nosological entity (Covid-19). However, excess mortality occurred only in some countries. For instance, there was no significant excess mortality in Germany and Sweden [6], although Sweden resorted only to mild political measures and Germany to harsh ones. In short, excess deaths were registered in some countries, while others showed normal or even reduced mortality; and even where excess deaths occurred, there is no convincing way of extracting a signal associated with D_7 from the noise due to emotional over-reaction and counterproductive measures or behaviour such as neglect of life-threatening conditions. If anything, excess deaths seem to be positively correlated to over-reactions bordering on hysteria, containment measures and possibly also the adverse effects of the vaccines. Moreover, we now know that the introduction or severity of containment measures such as lockdowns, school and business closures, and masks did not correlate with epidemiological data such as total mortality, hospitalisations and infections [7][8][9].

In general, to obtain the conjecture that would be needed to establish Covid-19 as a natural kind, one would have to organise the existing data by looking for a correlation pattern between micro-biological (genomic) and macro-biological (epidemiological) data. One could use a computer to find correlations between sequences satisfying D_7 and appropriate macro-biological data such as symptoms of respiratory diseases, hospitalisations, and deaths. The emergence of a clear pattern of correlations would support the conjecture of a new coronavirus taxonomy that could be subjected to the test of falsification based on macro-biological data. However, the evaluation of the existing

data fails in every respect when it comes to establishing such a correlation.

We conjecture that, if a clear pattern did indeed emerge, the resulting taxonomy would look nothing like the current one, on which the whole declaration of a pandemic was predicated. We may have to wait decades for genomic epidemiology to become a mature science. Indeed, the connection between the genetic sequence and the macro level is so subject to modulation by environmental and stochastic factors including individual immune reactions that genomic epidemiology may never be realized. It is also likely that computers that are programmed in non-identical fashion based on the differing assumptions of their programmers would detect different patterns and thus derive a variety of taxonomies.

In sum, as things stand, the taxonomy of genomic epidemiology in classifying Covid-19 as a new type of respiratory disease looks extremely arbitrary. We fall back to the PCR test as the *only* reliable evidence.

4. *The Corman-Drosten PCR test and the issue of "false positive" results*

Covid-19 cases were *defined* as the presence of a positive result using a variant of the PCR-test described by Corman, Drosten and others in a paper published on 23 January 2020 [10]. Surprisingly, on 17 January 2020, four days prior to the submission of this paper to the journal *Eurosurveillance*, the protocol for their test had already been published and recommended on the website of the World Health Organization [11].

A novel feature of the Corman-Drosten test is this one: it is not based on a sample of virus isolated in a laboratory, but rather on computer-generated assumptions resulting from a single putative viral sequence identified in a 41-year-old man who fell ill on 20 December 2019 and was admitted to the Central Hospital of Wuhan six days later [1]. The putative viral sequence was also not a result of viral isolation, but was generated using an algorithmic trawling approach known as "metagenomic RNA sequencing". This turned up a "high-abundance" contiguous sequence or "contig" of 30'474 nucleotides in length sharing a nucleoside identity of 89.1% with a bat SARS-like coronavirus that had previously been identified in China. In their paper, Corman et al. showed that the PCR targets of their test were identical in sequence to five other samples isolated in Wuhan on 24 December 2019, 30 December 2019 (three samples), and 1 January 2020 [10]. However, these sequences did not affect their test design.

For this reason alone, the link between the Corman-Drosten PCR test and clinical disease data is already tenuous, based as it is on two superimposed layers of computer-generated assumptions. Furthermore, its technical design has also been the subject of detailed criticism regarding primer concentration and design; the siting of all primer pairs towards the 3' end of the putative viral sequence, thus potentiating the intrinsic inability of PCR to distinguish between viral fragments and intact virus; an overly high cycle threshold value of 45; lack of validation of the PCR products by sizing and sequencing; the lack in the original formulation of integrated positive and negative controls and the lack of a standardized operating procedure (ICSLS 2020 [12]).

From the start, a major criticism of the Corman-Drosten PCR test has been its tendency to produce so-called false positive results. This debate has been characterized by a surprising degree of confusion concerning what is meant by the term "false positive". Let us therefore attempt to unpack this term first at a technical level and then at a more fundamental epistemological level.

The authors of the Corman-Drosten review report allude to the problem of false-positive results. They define them as "a negative sample, which initially scores positive, but which is negative after retesting with the same test", citing that this applied to four of 310 samples in the paper by Corman et al. (2020) (ICSLS 2020, section 7 [12]). This is a self-referential and therefore unsatisfactory definition of the term "false-positive", for how can one know if the fault lies with the initial or with the repeated result?

A second, slightly better approach to come to terms with false positive results concerns the issue of the number of cycles that the polymerase chain reaction goes through before the amount of PCR-product exceeds the threshold for detection (cycle threshold, or ct value). With each PCR cycle, the amount of PCR-product is almost doubled and therefore accumulates exponentially. If the cycle number is sufficiently high, single molecules of viral material may be detected (the calculated limit for detection in the paper Corman et al. using 45 PCR cycles was given as 3.8 copies for the putative ribonucleic acid-dependent ribonucleic acid polymerase gene and 5.2 copies for the putative envelope gene [10]). Apart from the problem of contamination at such extreme degrees of amplification, serious questions surround the pathogenicity of such low amounts of virus. It is often

forgotten that we swim in an ocean of viral and bacterial microbes, most of which are harmless and from the remainder of which we are generally protected by our immune systems. A single species PCR will only detect its target, but not other simultaneously present pathological viruses or microorganisms that may actually be the cause of the patient's symptoms. In addition, such ultrasensitive testing is likely to detect viral fragments which are inert and do not pose a risk for infection.

When the Corman-Drosten PCR test was rolled out, ct cut-offs for deciding "positive" test results were set at 45 as in the paper by Corman et al. [10] or at 40, as in the test produced by Roche Diagnostics, which rapidly became the de facto industry standard worldwide (see [13] for details). This led to an inflation of clinically irrelevant "positive" PCR results and "cases" of Covid-19. In a household survey performed by the United Kingdom Office for National Statistics, it was found that only test results with a ct value below 25 (indicating a high viral load) were likely to be infectious [14]. Yet an analysis of 162,457 individuals investigated using the Roche test in the German city of Münster showed that only 40.6% of "positive" tests had a ct value below 25 [13]. That is to say, assuming that the samples were collected appropriately, only 40.6% of "positive" tests indicated a likelihood of being "true positive" in terms of contributing to the spread of the infection.

5. *Why the Corman-Drosten PCR test does not discern a natural kind*

A central purpose of any laboratory test is to contribute to the establishment of a diagnosis, a word derived from the Greek "dia-gnosis" which literally means "to know apart from another", that is, to discern or distinguish. But the Corman-Drosten PCR test can neither distinguish "Covid-19" from other entities showing up as acute respiratory disease, nor can it identify a particular genetic entity, based as it is on two putative gene fragments clustered at one end of the viral genome. The sole function of the test is to identify the presence of RNA sequences complementary to the sequences contained in the primers of the PCR test. As pointed out by the Corman-Drosten review report [12], it is not even the case that all PCR tests use the same sets of primers or probes for detection of PCR product.

The Nobel laureate and inventor of the PCR method Kary Mullis stated that PCR tests should not be used for diagnostic purposes [15]. What he meant by this is that, for the technical reasons listed above, the PCR test alone is not suited for making a diagnosis in the sense of distinguishing one natural kind – namely a natural nosological entity in medicine – from all others.

Moreover, in the case at hand we have the additional and even more fundamental problem that the term "Covid-19" does not define a natural kind that exists independently of a "positive" PCR-test result. To illustrate this issue, let us consider another infectious respiratory disease, namely pulmonary tuberculosis. If a 45-year-old male smoker reports to his physician that he has a cough, feels ill, has been losing weight and has coughed up some blood, two differential diagnoses that might spring to the physician's mind are pulmonary tuberculosis and lung cancer. Let us assume that the patient undergoes a chest X-ray that shows no solid mass, but rather enlarged lymph nodes at the lung root and patchy areas of consolidation within the lungs. This picture is more consistent with tuberculosis than with cancer. Let us further assume that on examination under the microscope, a sample of sputum shows the presence of acid-fast bacilli, and that a culture of sputum on Löffler medium reveals the presence of bacterial colonies typical of *Mycobacterium tuberculosis*, which under the microscope consist of the same acid-fast bacilli previously revealed on the sputum sample. The diagnosis for this patient then is unequivocally pulmonary tuberculosis. He may have other conditions we are unaware of, but of his tuberculosis we can be certain.

Now let us assume that we have before us 100 such cases, and an additional 100 control cases in which pulmonary tuberculosis has been excluded with equal exhaustiveness. We then examine sputum from these 200 cases using a PCR-test for tuberculosis (such a test actually exists). Let us assume that of the 100 tuberculosis cases, 96 show a "positive" and four a "negative" PCR result, while among the 100 controls without tuberculosis, three show a "positive" and 97 a "negative" PCR result. Based on these results, we can now proceed to calculate the performance of our tuberculosis PCR-test in terms of false-positive and false-negative rates (three and four percent, respectively). Furthermore, if we know the prevalence of pulmonary tuberculosis in our population, we can go on to calculate the positive predictive value of a "positive" PCR result, that is, the likelihood of someone with a "positive" PCR result actually having pulmonary tuberculosis.

However, none of these conditions is fulfilled in the case of the entity "Covid-19". The symptoms of Covid-19 are so diffuse, non-specific, and wide-ranging that the diagnosis depends *entirely* on the presence of a

"positive" PCR test. A "positive" PCR test is a "case" of Covid-19, and "cases" of Covid-19 are persons with a "positive" PCR test. This circularity also gave rise to the concept of the "asymptomatic cases" that were used as justification for severe restrictions on basic freedoms and social interactions. The point at issue hence is that a "positive" PCR test is employed as *definition* for having "Covid-19". But this, then, implies that "Covid-19" cannot be a natural kind, namely a natural nosological entity, as pulmonary tuberculosis is a natural nosological entity. For this to be the case, there would have to be methods that are independent of a "positive" PCR test and that confirm that the patient has "Covid-19" in distinction to another disease, or no disease at all. In short, as it stands, "Covid-19" as a nosological entity is an artefact of a "positive" PCR test.

In terms of the science of medical diagnostics, this point is so obvious as to be almost trivial. Yet it has hardly been addressed in the debate surrounding "Covid-19". To be sure, the authors of the Corman-Drosten review report (ICSLS 2020, section 1d) refer to it obliquely and cite a literature source in this regard. However, this source also refers to the issue only in an indirect fashion:

As with all laboratory testing, micro-biological laboratory results are never definitive, and the clinical significance of the test result should always be placed in the context of the patient's clinical presentation. Molecular diagnostic techniques are no exception to this rule [16].

Indeed, they are not and must be complemented by other elements to establish the diagnosis of an infection or disease.

6. *Conclusion*

In sum, when one analyses the causal chain "novel respiratory disease" -> "identification of a novel coronavirus" -> "positive" PCR test -> "diagnosis" of "Covid-19" (including "asymptomatic cases") -> "pandemic" -> "pandemic response", one discovers a lack of firm foundation at all levels. This lack is not due to a weakness in our PCR test. Even if we had a perfect PCR test, the epistemological issue that such a test on its own cannot establish anything as a natural kind (nosological entity) would remain.

Again, this is not to deny that there is a causal chain that originates in a specific, individual event of a virus outbreak at a particular moment in Wuhan in 2019 and that subsequently spread around the world. Our claim is that as long as the PCR test is the only means that is intended to hook on that causal chain, (i) it is not sufficient to detect an infection or a disease and (ii) it cannot establish Covid-19 as a new natural kind or specific nosological entity, namely as a specific, new kind of a respiratory disease.

Furthermore, even if one grants that SARS-Cov-2 is a valid classification in genomics, one cannot take the reduction of genomic epidemiology to genomics for granted. One must establish a specific pattern in macro-biological data that can count as the manifestation of the genomic entity SARS-Cov-2. However, there is no such pattern that could warrant the classification of what is known as Covid-19 as a new kind of respiratory disease and thus as a natural kind in nosology. In short, the Covid-19 disease is an artefact of the PCR test instead of a new kind of respiratory disease.

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15. ^*"Corporate Greed and AIDS" Conference, Santa Moni ca, California, USA 12 July 1997 with Kary Mullis, Sean Current, Paul Philpott and Christine Maggiore. In resp onse to an audience question about how PCR tests can be misused, Mullis said the test itself cannot be misuse d, but the interpretations of it can, because it creates "a whole lot of something from something.... with the PC R, if you do it well, you can find almost anything in an ybody". See https://www.youtube.com/watch?v=wT3I qZjT_9A*
16. ^*Kurkela S, Brown DWG (2009): "Molecular diagnostic techniques". Medicine 37, pp. 535-540.*

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