Expert statement regarding the use of Moderna COVID-19-mRNA-Vaccine in children

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This expert statement was submitted by Italian lawyer Renate Holzeisen in conjunction with a lawsuit that challenges the EU's authorization of the use of Moderna's mRNA vaccine on children of 12 years and older. The Pfizer vaccine was addressed in a previously published expertise. Many of the arguments below—particularly those related to the dangers of expressing the biologically active SARS-CoV-2 spike protein in the human body—apply also to the adenovector-based AstraZeneca and Johnson & Johnson vaccines.

Summary

This expertise on the use of the Moderna COVID-19 vaccine in adolescents is divided into three sections, which will deal with the following questions, in order:

1. Is vaccination of adolescents against COVID-19 necessary?
2. Is the Moderna COVID-19 vaccine effective?
3. Is the Moderna COVID-19 vaccine safe?

The arguments presented in Section 1 pertain to all COVID-19 vaccines, whereas those in Sections 2 and 3 apply specifically to the Moderna vaccine.

Section 1 will show that vaccination of adolescents COVID-19 is unnecessary, because

- in this age group the disease is almost always mild and benign;
- for the rare clinical cases that require it, treatment is readily available;
- immunity to the disease is now widespread, due to prior infection with the virus (SARS-CoV-2) or with other coronavirus strains; and
- asymptomatic adolescents will not transmit the disease to other individuals who might be at greater risk of infection.

Section 2 will demonstrate that the claims of efficacy which Moderna attaches to its vaccine—namely, 94.5% efficacy in adults, and 100% in adolescents—are

- misleading, because these numbers pertain to *relative*, not *absolute* efficacy, the latter being less than 1%;
- specious, because they refer to an arbitrarily defined, clinically meaningless evaluation endpoint, whereas no efficacy at all has been demonstrated against mortality;
- most likely altogether fraudulent.

Section 3 will show that the safety profile of the Moderna vaccine is catastrophically bad. It will be discussed that

- Moderna and the EMA have systematically neglected evidence from preclinical animal trials that clearly pointed to grave dangers of adverse events;
- the Moderna vaccine has caused thousands of deaths within less than a year of its introduction;
- The agencies that granted emergency use authorization for this vaccine committed grave errors and omissions in their assessments of known and possible health risks.

The only possible conclusion from this analysis is that the use of this vaccine in adolescents cannot be permitted, and that its ongoing use in any and all age groups ought to be stopped immediately.

1 Vaccination of adolescents against COVID-19 is unnecessary

1.1 What does the available evidence show? There are several lines of evidence that show vaccination of adolescents against COVID-19 to be unnecessary.

1.1.1 The case fatality rate of COVID-19 in the general population is low. The vast majority of all persons infected with COVID-19 recovers after minor, often uncharacteristic illness. According to world-leading epidemiologist John Ioannidis [1, 2], the infection fatality rate of COVID-19 is on the order of 0.1% to 0.2% across all age groups, with a very strong bias towards old people, particularly those with co-morbidities. This rate does not exceed the range commonly observed with influenza, against which a vaccination of adolescents is not considered urgent or necessary.

1.1.2 COVID-19 has a particularly low prevalence and severity in adolescents. A review by Rajapakse et al. [3] states that, internationally, children and adolescents up to 18 years have accounted for only 1-2% of all confirmed clinical cases of COVID, and that severity was generally low. It has also been reported that children and adolescents only rarely transmit the disease to adults living in the same households; transmission in the opposite direction is far more common [4].

Within this age group, the most severe cases were observed among very young infants [5]. This is consistent with the lack in infants of cross-immunity to COVID-19, which in other age groups is conferred by preceding exposure to regular respiratory human coronaviruses (see Section 1.1.5). Among slightly older children, a peculiar multisystem inflammatory syndrome was observed in early 2020 [6]. Conceivably, these patients, too, were still lacking cross-immunity, although it has also been argued that the syndrome may in fact have post-infectious immune pathogenesis [3].

The basis for the overall very low incidence COVID in children has been elucidated in immunological studies reported by Loske et al. [7]. According to these authors, the mucous membranes of the airways of children exhibit stronger non-specific immunity than those of adults; for example, pattern recognition receptors are more strongly expressed in children. They also exhibit stronger CD8 T-cell responses.

Figure 1 compares the mortality and the infection fatality rates of different age groups. Panel A very clearly shows that adolescents—the age group for which Moderna is seeking use authorization—has vanishingly small mortality; in fact, mortality in this age group is lower than in all others. As of July 2021, the German Robert Koch Institute reported only a total of 11 fatalities in those between 10 and 19 years of age—not even two in a million. To contemplate mass vaccinations with an experimental vaccine in the face of such low overall mortality is not justifiable.

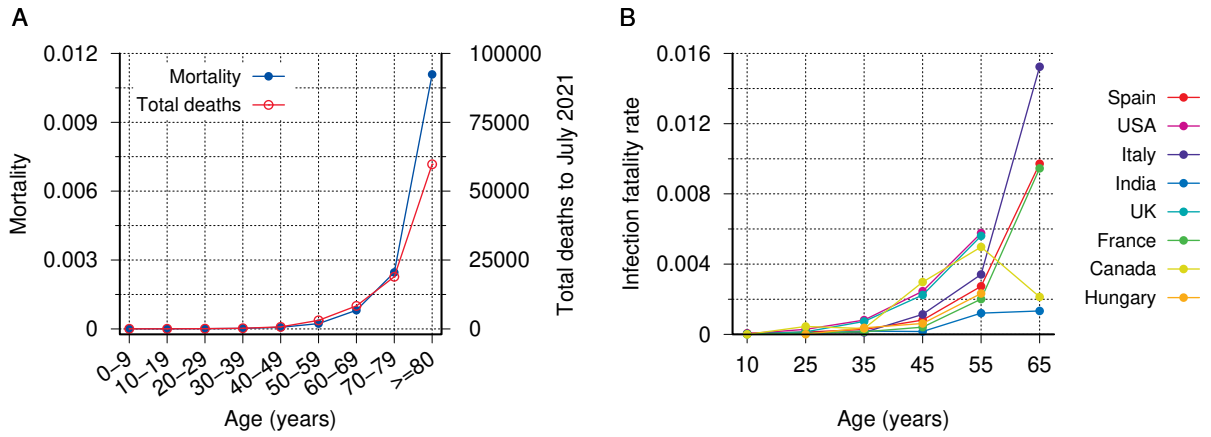

Figure 1 COVID mortality, number of cases, and infection fatality rate by age group. A: Total cases reported to the Robert Koch Institute as of July 13th, 2021, and mortality per age group, based on 2018 census numbers [8]. B: Infection fatality rates by age in various countries. Adapted from Figure 3 in [9].

1.1.3 COVID-19 can be treated. Numerous experienced physicians have collaborated on establishing effective treatment guidelines for clinically manifest COVID-19 [10]. Treatment options are available both for the early stage of the disease, when emphasis is placed on inhibiting viral replication, and for the later stage, at which anti-inflammatory treatment is paramount. Two drugs that have been used successfully at the early stage are hydroxychloroquine and ivermectin. Both drugs have been, and continue to be, in use against a variety of other diseases. Ivermectin, for example, is considered safe enough to be used not only for treating manifest scabies—a parasite infection of the skin that is unpleasant but not severe, and which is quite amenable to local treatment—but even prophylactically in asymptomatic contacts of scabies-infected persons [11].

Ivermectin is also widely used in the treatment of tropical parasitic diseases such as onchocerciasis (river blindness), and for this reason it is on the WHO's list of essential medicines. Yet, with COVID-19, the WHO sees fit to warn against the use of this very same well-known and safe drug outside of clinical trials [12]. This policy cannot be rationally justified, and it has quite appropriately been overridden by national or regional health authorities and ignored by individual physicians worldwide.

The availability of effective treatment voids the rationale for the emergency use of vaccines on any and all age groups, including also adolescents.

1.1.4 Most people, particularly adolescents, are by now immune to SARS-CoV-2. Due to the many inherent flaws and shortcomings of the diagnostic methods in common use (see Section 1.2), it is impossible to accurately determine the proportions of those who have already been infected with SARS-CoV-2 and those who have not. However, there are indications that the proportion of those who have been infected and recovered is high:

- The incidence of multisystem inflammatory syndrome in children (see Section 1.1.2) peaked in early to mid 2020, and then receded, with some slight delay after the initial wave of the COVID-19 respiratory disease itself [13].
- Approximately 60% of randomly selected test persons from British Columbia have detectable antibodies against multiple SARS-CoV-2 proteins (personal communication by Stephen Pelech, University of British Columbia), indicating past infection with the

virus—as opposed to vaccination, which would induce antibodies to only one (the spike) protein.

Past COVID-19 infection has been found to protect very reliably from reinfection [14], and strong specific humoral and cellular immunity is detected in almost all recovered individuals, as well as in those who remained asymptomatic throughout the infection [15]. Thus, a large proportion of individuals in all age groups, including adolescents, already have specific, reliable immunity to COVID-19.

1.1.5 Cross-immunity between SARS-CoV-2 and other coronaviruses. Coronaviruses are a large family of enveloped, single-stranded RNA viruses. In humans and a variety of animal species, they cause respiratory tract infections that can range from mild to lethal in severity. However, the vast majority of coronavirus infections in humans cause mild illness (common cold), although in very young children, who lack immunity from previous exposure, respiratory disease can be more severe. The same clinical picture is also caused by viruses from several other families, predominantly rhinoviruses.

The virus that causes COVID-19 is known as Severe Acute Respiratory Syndrome Corona-Virus 2 (SARS-CoV-2). While it has been maintained that SARS-CoV-2 arose naturally in a species of bats [16], a thorough analysis of the genome sequences of SARS-CoV-2 and of related virus strains indicates unambiguously that the virus is in fact of artificial origin [17–20]. Initially decried as a "conspiracy theory," this explanation has recently and belatedly been gaining acceptance in the mainstream [21].

SARS-CoV-2-reactive T-cells [22–24] and antibodies [25, 26] are widespread even in those who have not been exposed to this virus; this is mostly due to previous infections with coronaviruses. Cross-reactive T-cells, which are likely important for defending against the infection, are found even among those who have no detectable cross-reactive antibodies [27, 28]. The protective effect of such cross-immunity has been documented [29–32].

Cross-immunity will be particularly effective in healthy adolescents and young adults. Individuals with specific immunity or sufficient cross-immunity cannot possibly derive any benefit from undergoing an experimental vaccination. Rather to the opposite, they are at increased risk of suffering adverse events from it [33].

1.1.6 Asymptomatic transmission of COVID-19 is not real. An oft-cited rationale for vaccinating individuals who are not themselves at risk of severe disease is the need to induce "herd immunity:" the few who are at high risk should be protected by preventing the spread of the virus in the general population.

A subtext of this rationale is the idea of "asymptomatic spread"—persons who have been infected but who show no signs of it other than a positive PCR test are assumed to transmit this infection to other susceptible individuals. If we accept the idea of such asymptomatic spread, then preventative mass vaccination might indeed appear as the only means of reliable protection of those at risk.

It has, however, been unambiguously determined that such asymptomatic transmission does not occur. In a large-scale study which involved almost 10 million Chinese residents, no new infections could be traced to persons that had tested positive for SARS-CoV-2 by PCR, but who did not exhibit any other signs of infection [34]. This agrees with several studies that compared PCR to virus isolation in cell culture among patients with acute COVID-19 disease. In all cases, growth of the virus in cell culture ceased as symptoms subsided, or very shortly thereafter, whereas PCR remained positive for weeks or

months afterwards [35, 36]. It was accordingly proposed to use cell culture rather than PCR to assess infectiousness and to determine the duration of isolation [36].

These findings indicate that restricting contact of persons at risk with those who show, or very recently showed, symptoms of acute respiratory disease would be effective and sufficient as a protective measure. Indiscriminate mass vaccinations of persons who are not themselves at risk of severe disease are therefore not required to achieve such protection.

1.2 Missing evidence: use of inaccurate diagnostic methods. A key element that is lacking in the current discussion of the need for vaccination is a reliable diagnostic tool for determining who is or is not currently infected with SARS-CoV-2. The diagnostic procedure most widely used for this purpose is based on the polymerase chain reaction (PCR). The PCR is a very powerful and versatile method that lends itself to numerous applications in molecular biology, and also in the laboratory diagnosis of viral infections. However, exactly because it is so powerful, PCR is very difficult to get right even at the best of times; it will yield accurate results only in the hands of highly trained and disciplined personnel. The enormous scale on which the method has been deployed during the COVID-19 pandemic has meant that it was entrusted to untrained and insufficiently supervised personnel; in such circumstances, the mass manufacture of false-positive results due to the cross-contamination of samples is a disaster waiting to happen (see for example [37]). While this alone already is reason for grave concern, the problems start even earlier—namely, with the design of the PCR tests and the guidelines used for their interpretation, which would lead to false positive results even in the hands of skilled and diligent workers.

The key conclusion from this section will be that the PCR tests which have been used throughout the pandemic, and which continue to be used, lack accuracy and specificity and cannot be relied on for diagnostic or epidemiological purposes. In order to adequately justify these conclusions, we must first consider the basics of the method in some detail.

1.2.1 The polymerase chain reaction. The polymerase chain reaction (PCR) is a versatile method for the biochemical replication of deoxyribonucleic acid (DNA) in vitro. Immediately after its invention by Kary Mullis in the 1980s, PCR took the world of molecular biology by storm, finding application for creating DNA mutations, DNA sequencing, for shuffling and merging nucleic acids of different origin (recombinant DNA technology), and for the creation of novel nucleic acids or even whole genomes from scratch ("synthetic biology"). PCR also soon found its way into the field of diagnostic medical microbiology [38]. Particularly with respect to viral pathogens, PCR is now one of the mainstay diagnostic methods. Against this background, it is not surprising that PCR methods should also have been adopted in the laboratory diagnostics of SARS-CoV-2.

1.2.1.1 The principle. To understand how PCR works, it is best to start with a piece of double-stranded DNA (the well-known double helix). In such a molecule, each of the paired single strands consists of four different building blocks (nucleotides), which will here be referred to as A, C, G, and T for short. Within each single strand, these building blocks are arranged like pearls on a string; the biological activity and identity of the nucleic acid will be dictated by its characteristic nucleotide sequence.

In a DNA double helix, the two strands are held together by the proper pairing of the nucleotides, such that an A in one strand is always found opposite to a T in the other,

and likewise C is always found opposite G. Thus, the nucleotide sequence of one strand implies that of the other—the two sequences are *complementary*.

The first step in PCR consists in the separation of the two strands, which can be effected by heating the DNA sample past its "melting point." Each strand can now be used as a template for synthesizing a new copy of its opposite strand. To this end, two short, synthetic single-stranded DNA molecules ("primers") are added; their sequences are chosen such that one will bind to each of the DNA template strands, based on sequence complementarity. For this binding to occur, the temperature of the reaction must be lowered.

Once the primers have bound, each is extended by the repeated incorporation of free nucleotide precursors to one of its two free ends. This is accomplished using a thermostable *DNA polymerase*—a bacterial enzyme that synthesizes DNA. The extension is carried out at a temperature which is intermediate between those used for double strand separation and primer binding ("annealing"). After this step has extended each of the primers into a new DNA strand, we will have created two double-stranded DNA molecules from one. We can now repeat the process—separate the two double strands and convert them into four, then eight, and so on. After 10 cycles, the initial amount of double-stranded DNA will have increased by a factor of approximately one thousand, after 20 cycles by a million, and so on—amplification proceeds exponentially with the number of reaction cycles, until the reaction finally runs out of primers and/or nucleotide precursors.

1.2.1.2 PCR and RNA templates. While the above discussion referred to DNA only, PCR can also be used with RNA templates; this is important with SARS-CoV-2, since this virus has RNA rather than DNA as its genetic material. To this end, the RNA is first converted ("reversely transcribed") into DNA, using a reverse transcriptase enzyme. The DNA copy of the viral RNA genome, which is referred to as complementary DNA (cDNA), then serves as the template for the PCR proper.

1.2.2 Potential pitfalls of PCR in diagnostic applications. We just saw that PCR allows us to take a very small sample of DNA and amplify it with extraordinary efficiency. However, this very efficiency of amplification creates a number of problems that must be carefully addressed in order to make the result meaningful, particularly in a diagnostic context.

1. If we use too high a number of repeated reaction cycles, minuscule amounts of nucleic acids will be detected that have no diagnostic significance.
2. The various temperatures used in the reaction must be carefully calibrated, and they must match the length and nucleotide sequence of the two DNA primers. In particular, if the temperature for primer annealing is too low, then the primers may bind to the template DNA in a non-specific manner—in spite of one or more mismatched nucleotides—and DNA molecules other than the intended ones may be amplified. In the context of COVID diagnostics, this could mean that for example the nucleic acids of coronaviruses other than SARS-CoV-2 are amplified and mistaken for the latter.
3. Apart from the temperature, other conditions must likewise be carefully calibrated in order to ensure specificity. These include the concentrations of magnesium ions and of free nucleotides; excessively high concentrations favour non-specific amplification.

There is a further problem that results not from the efficiency of the amplification, but rather from a technical limitation: PCR is most efficient if the amplified DNA molecule is

no more than several hundred nucleotides in length; however, a full-length coronavirus genome is approximately 30,000 nucleotides long. Successful amplification of a segment of several hundred nucleotides only does not prove that the template nucleic acid itself was indeed complete and intact, and therefore that it was part of an infectious virus particle.

1.2.3 Technical precautions in diagnostic PCR. Non-specific or overly sensitive amplification can be guarded against in a number of ways:

1. All primers that are part of the same reaction mixture must be designed in such a manner that they anneal to their template DNA at the same temperature. As may be intuitively clear, a longer primer will begin to anneal to its template at a higher temperature than a shorter one; and since the bond which forms between C and G on opposite strands is tighter than that between A and T, the nucleotide composition of each primer must also be taken into account. If the primers are mismatched in this regard, then the more avidly binding primer will start to bind non-specifically when the temperature is low enough for allowing the other primer to bind specifically. The original Corman-Drosten PCR protocol [39] which was rapidly endorsed by the WHO has been criticized for exactly this mistake [40].
2. Instead of amplifying only a single piece of the template DNA, one can simultaneously amplify several pieces, using the appropriate number of DNA primer pairs, and stipulate that all pieces, or a suitable minimal number, must be successfully amplified for the test to evaluate as positive.
3. One must keep track of the "cycle threshold" or C_t value for short, that is, the number of amplification cycles that were necessary to produce a detectable amount of amplified product: the lower the number of cycles, the greater the initial amount of template nucleic acid that must have been present.

   This aspect is further compounded by the lack of normalization for the amount of genetic material recovered by the swab. It would in principle be possible to achieve such normalization by also amplifying a fragment of host cell DNA. The C_t value of this internal standard could then be subtracted from that of the virus in order to estimate the true abundance of the virus. This, however, is not commonly done.
4. Confirming the identity—the exact nucleotide sequence—of the nucleic acid molecules that were amplified. DNA sequencing has been feasible in diagnostic routine laboratories for a considerable time, and there is no good reason not to use it, particularly when decisions pertaining to public health depend on these laboratory results.

1.2.4 Real-time PCR. The third point above, and to a degree the fourth, can be addressed using real-time PCR. In this method, the accumulation of amplified DNA is monitored as the reaction progresses, in real time, with product quantification after each cycle (quantitative PCR; qPCR for short). Real-time detection can be achieved by the inclusion of a third DNA primer, which binds to either of the template DNA strands, at a location between the two other primers which drive the DNA synthesis. Downstream of the binding of that third primer, a light signal will be emitted, and the intensity of this signal is proportional to the amount of amplified DNA present. Since binding of this primer, too, requires a complementary target sequence on the DNA template, this method does provide some confirmation of the nucleotide sequence of the target DNA.

A second, simpler variety of real-time PCR uses a simple organic dye molecule that binds to double-stranded DNA. The dye displays weak background fluorescence that

increases dramatically upon DNA binding. The measured fluorescence increase is then proportional to the total amount of amplified DNA; but since the dye binds regardless of DNA sequence, in this case the signal does not confirm that the *correct* template DNA has been amplified.

1.2.5 Shortcomings of commercial COVID-19 PCR tests. Unfortunately, the number of amplification cycles (the C_t value) needed to find the genetic material in question is rarely included in the results sent to authorities, doctors and those tested. Most commercially available RT-qPCR tests set the limit of amplification cycles up to which an amplification signal should be considered positive at 35 or higher. Multiple studies have indicated that C_t values above 30 have a very low predictive value for positive virus cultures, and thus for infectiousness or the presence of acute disease [35, 41–43]. Considering that in many clinical trials—including the ones conducted by Moderna (see later)—a "COVID-19 case", or an "endpoint" amounts to no more than a positive PCR test, regardless of C_t value, in combination with one or a few non-specific symptoms of respiratory disease, the significance of the use of improperly high C_t cut-off values cannot be overstated. This systematic and widespread error alone has sufficed to gravely distort the diagnoses conferred on individual patients, as well as the epidemiology of the pandemic as a whole.

Further systematic negligence concerns the verification of the identity of the amplified DNA fragments. While Sanger DNA sequencing of such fragments, the gold standard, is feasible on a large scale in principle, it has not been routinely used in the ongoing mass PCR testing campaigns. The error is compounded by the very low number of independent PCR amplifications considered sufficient for a positive test—as few as two, or even only one have been considered sufficient in various jurisdictions—and by various other technical faults in the widely adopted and commercialized Corman-Drosten protocol, which have been discussed in detail elsewhere [40].

In summary, a positive RT-qPCR test result cannot be accepted as proof that the person in question is currently infected and infectious—even if there is reasonable clinical plausibility of actual COVID-19 infection, as well as a significant community prevalence of the disease. Firstly, the RNA material containing the target sequences could very well be from nonviable/inactive virus; this is particularly likely if the patient in question has already recovered from the infection. Secondly, there needs to be a minimum amount of viable virus for onward transmission; but tests carried out with excessively high (yet unreported) C_t values will detect minuscule amounts of genetic material that pose no real risk at all.

2 The Moderna COVID-19 vaccine lacks efficacy

2.1 What does the evidence show? Moderna persistently touts the 94.5% efficacy of its vaccine, based on the clinical trials that formed the basis of the emergency approvals granted by the FDA [44] and the European Union [45]. In a more recent study on adolescents [46], the claimed efficacy has been raised to no less than 100%. However, these claims cannot be taken at face value.

2.1.1 Absolute vs. relative efficacy. In Moderna's first reported clinical trial, the experimental vaccine (mRNA-1273) was administered to 14,134 persons, and 14,073 received placebo. Across both groups, a total of 95 COVID-19 "cases" was recorded, of which 90 occurred in the placebo group, whereas 5 cases were observed in the mRNA-1273 group. Based on these figures—$^5/_{90} = 5.6\%$—Moderna proceeded to claim 94.5% efficacy. Clearly, however, this efficacy is only a *relative* value—in absolute terms, less than 0.6% of the

Table 1 COVID infections detected among vaccinated and unvaccinated persons in Barnstable County, Massachusetts between July 5th and 26th 2021. Data from [47].

	Cases % (n)	Population %	Relative risk
Total vaccinated	74% (346)	69%	1.07
Pfizer vaccine	34% (159)	39%	0.88
Moderna vaccine	28% (131)	26%	1.07
Janssen vaccine	12% (56)	4.8%	2.47
Hospitalization	80% (4)	—	(1.29)

placebo group developed COVID-19, and therefore less than 0.6% of the vaccine group was protected from it.

The situation is similar with the subsequent, smaller test carried out on 12-17 years old adolescents [46]. Here, the vaccine group comprised 2163 individuals, whereas the placebo group included 1073 persons. In the latter group, a grand total of four (4) individuals were subsequently diagnosed with COVID-19, whereas no such cases occurred in the vaccine group. True to form, Moderna converted this absolute efficacy of 0.37% to a relative one of 100%.

2.1.2 Real-world efficacy. Recent data published by the Israeli health ministry indicate that COVID is equally likely to occur in vaccinated and unvaccinated persons, which suggests that the true efficacy is not 95% but rather close to 0%. While Israel mostly used the Pfizer mRNA vaccine rather than the one produced by Moderna, the latter is equally ineffective. This is evident from a CDC report that examines a cluster of COVID infections that occurred among vaccinated and unvaccinated persons in Barnstable County, Massachusetts during July 2021 [47]. The data are summarized in Table 1.

The overall number of cases reported by Brown et al. is five times higher than that reported in all of Moderna's clinical trials [45]. The relative risk of hospitalization stated in the table is based on a total of only 5 cases and therefore is not statistically robust. We note, however, that this low number of hospitalized cases indicates a very low disease severity overall. Of particular interest in this connection is that most of these cases appear to have been due to the so-called Delta variant, which was identified in 89% of those 133 cases in which the viral RNA was characterized by genomic sequencing.

Brown et al. do not state whether the Delta variant was overrepresented among the "break-through" cases in vaccinated persons; thus, the incomplete data provided in this study do not rule out the possibility that the vaccine might have been more effective with the original Wuhan strain of SARS-CoV-2. Be that as it may, however—RNA viruses are always subject to antigenic drift. Even if we assume that the vaccines had been active against the Wuhan strain, their obsolescence due to antigenic drift within mere months after introduction would suffice to make them useless in practice.

Brown et al. [47] state that all of their reported cases were "associated with large public gatherings," which suggests that most of the affected persons were in a reasonable state of health before contracting the infection. Other studies have reported vaccine "breakthrough" cases of infection both among the healthy [48] and among those with pre-existing neurological disease [49]. Overall, it is clear that the vaccines, including Moderna's mRNA-1273 vaccine, are failing.

2.1.3 Negative impact of mRNA-1273 on overall morbidity in adolescents. The cited vaccine study on adolescents [46] states that a "case" of COVID-19 was defined in the same manner as in the previous study on adults [45]. In that study, we read (on page 84):

> *The participant must have experienced at least two of the following systemic symptoms: Fever (≥ 38° C), chills, myalgia, headache, sore throat, new olfactory and taste disorder(s),*
>
> *OR*
>
> *The participant must have experienced at least one of the following respiratory signs/symptoms: cough, shortness of breath or difficulty breathing, OR clinical or radiographical evidence of pneumonia;*
>
> *AND*
>
> *The participant must have at least one NP swab, nasal swab, or saliva sample (or respiratory sample, if hospitalised) positive for SARS-CoV-2 by RT-PCR.*

Thus, one or more symptoms from a laundry list of mostly non-characteristic symptoms, plus a positive finding from an unreliable laboratory test (cf. Section 1.2.5), was deemed sufficient to establish the diagnosis. While the study on adults gives separate criteria for diagnosing "severe" cases of the disease, Ali et al. [46] mention no such diagnostic criteria, and neither do they report any cases that required hospitalization or were considered "severe" for any other reason. Overall, therefore, it is clear that all of the four putative COVID cases in the placebo group were mild.

In stark contrast to these numbers, which pertain to the disease from which the vaccination is supposed to protect, side effects from the vaccination were exceedingly common (92.4%). Apart from injection site pain occurring in a high percentage of the vaccine group (84% after the first dose, and 88% after the second), fatigue (65% after the second dose) and headache (59% after the second dose) abounded. Severe fatigue and headache were reported by several percent of the test persons. Severe headache, in particular, may be associated with underlying thrombotic events (see Section 3.2.6.3). It is therefore clear that, if we consider both COVID-19 and vaccine adverse effects, overall morbidity was far greater in the vaccinated than in the placebo group.

2.1.4 Unlikely claims and contradictions in Moderna's evidence on efficacy. We saw above that the reported efficacy of Moderna's vaccine is very modest when expressed in absolute terms. Even this low efficacy, however, cannot be accepted at face value. This is apparent from the EMA assessment report [45].

2.1.4.1 Contradictory claims about COVID incidence. The text of the EMA document maintains that in all 90 "cases" of COVID occurred in the placebo arm of the study. On the other hand, the study also shows a graph that is said to represent the cumulative incidence of COVID in both the vaccine and the placebo group (Figure 2). At the bottom of this graph, we see the number of individuals at risk of becoming COVID "cases" at various time points after their assignment to either group; from Table 20 in [45], we can infer that this time point coincides with the first injection. The number of those at risk decreases with time; for example, 9911 persons in the vaccine group, and 9736 in the placebo group, were followed during the trial for 80 days or more and were therefore at risk of contracting COVID on day 80. We can estimate that this number would have dropped to 9,000 on or about day 82. Therefore, if all 90 COVID cases had been diagnosed on day 82, then the cumulative incidence should on this day be 1%. However, the dashed arrows drawn atop the graph indicate that the depicted value on this day is approximately 1.6%.

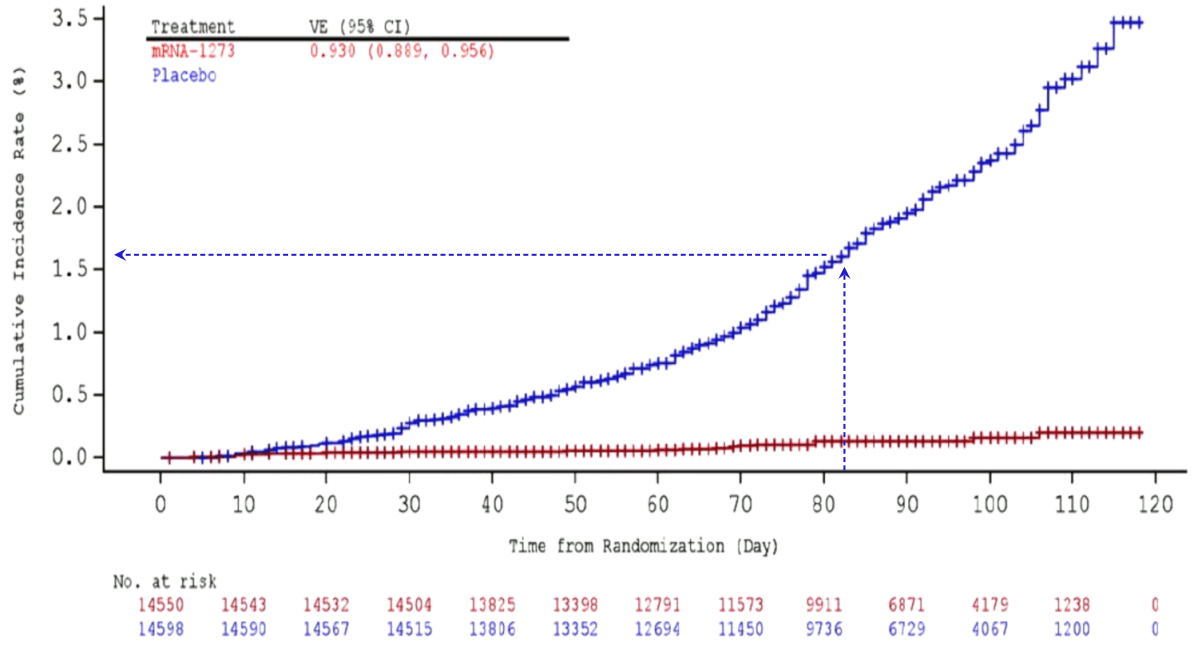

Figure 2 Reproduction of Figure 18 of the EMA assessment report [45]; arrows added by the authors of this document. The figure is said to show the cumulative incidence of COVID-19 cases among vaccinated and placebo groups in the clinical study on the mRNA-1273 vaccine. See text for discussion.

Cases diagnosed before day 82 would have occurred among a larger number of individuals at risk, which should have lowered the cumulative incidence on day 82. Furthermore, the curve continues to rise after day 82, which implies that some of the altogether 90 cases occurred at a later time. This should further reduce the cumulative incidence observed on day 82. Thus, while the available information does not permit us to quantify the discrepancy exactly, we can say that it is substantial. That the EMA reviewers did not catch this rather obvious problem does not instil confidence in the thoroughness of their assessment process.

2.1.4.2 Early vs. late onset of immunity. According to Figure 2, new COVID cases accumulated at the same pace within the placebo group and the vaccine group until day 12 or 13. Thereafter, they diverge, indicating the onset of immunity in the vaccinated; and the uniformly low increase with time of the cumulative incidence among the vaccinated suggests that the maximum extent of immunity was attained within a very short time period. Such an early onset of immunity is not expected after the first exposure to an antigen; instead, it is typical of a memory reaction. The occurrence of memory reactions would fit with the many observations of cross-immunity reported in other studies (see Section 1.1.5 above). However, Moderna's own data indicate that only some, but not all test persons showed a memory response; on days 15 and 29 after the first injection, the titres of neutralizing antibodies remained low overall, and in what appears to be about half of the individuals below the detection limit (see Figure 3A). Nor can Moderna's reported data on T-cell-mediated immunity account for the rapid onset of clinical immunity: Figure 4 shows that any activation of T-cells is weak and is observed only on day 43, that is, after the second injection. This applies in particular to CD8 cells, which are crucial effectors of cellular antiviral immunity. Thus, an obvious discrepancy exists between the early onset of the claimed clinical immunity and the delayed responses observed in immuno-

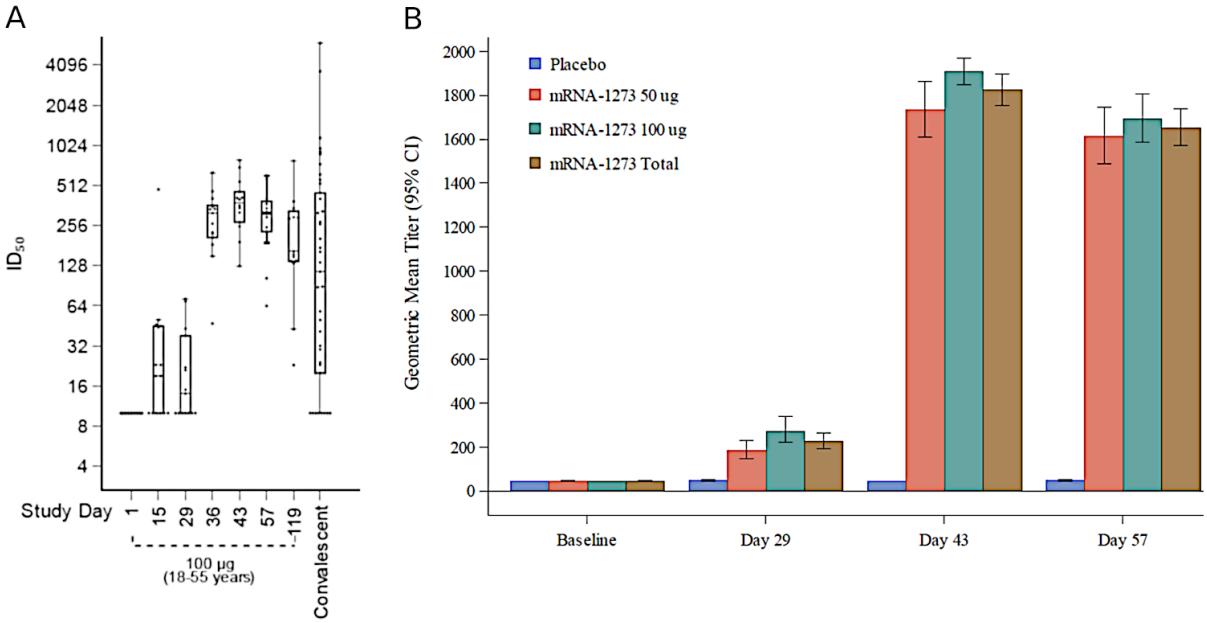

Figure 3 Neutralizing antibodies at various time points after vaccination. A: Box plot of pseudovirus neutralization titres (adapted from Figure 7 of [45]. Note that on days 15 and 29 multiple samples remain below the detection limit; only on day 36 are neutralizing antibodies detected in all samples. Note also multiple negative samples among convalescent individuals. B: Geometric means of neutralizing antibodies. Adapted from Figure 16 of the EMA report. All data pertain to the age group between 18 and 55 years.

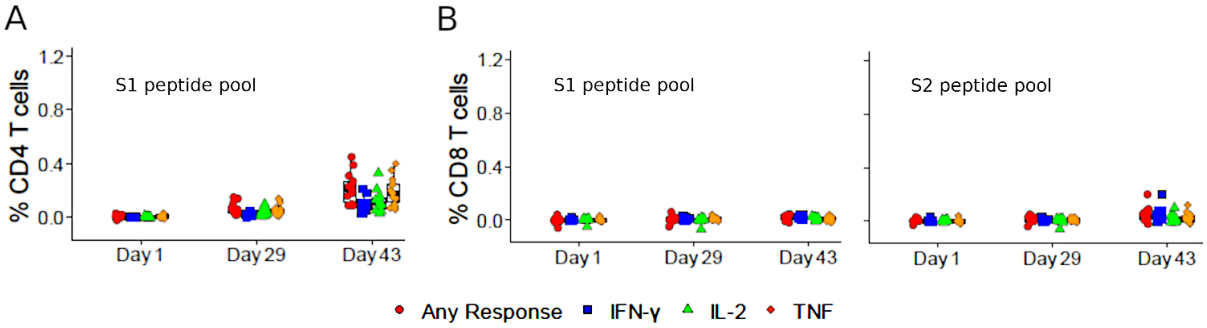

Figure 4 T-cell activation by the mRNA-1273 vaccine. CD4 and CD8 cells were isolated at various time points after the first injection and stimulated in vitro with peptide pools representing the S1 or the S2 fragments of the spike protein and stained for expression of IFN-γ, IL-2, and TNF. A: activation of CD4 cells. Adapted from Figure 10 of [45]. B: activation of CD8 cells. Adapted from Figures 11 and 12 of the EMA report. All data pertain to vaccine doses of 100 µg and the age group between 18 and 55 years.

logical laboratory studies. Had the EMA review been conducted with due diligence, this discrepancy would not simply have been passed over.

2.2 What evidence is lacking to make the case? We have already mentioned the contrived and specious character of the endpoint used in Moderna's clinical trials—namely, the counting of a COVID-19 "case" based on nothing more than a positive PCR result, together with one or more items from a list of mostly uncharacteristic clinical symptoms (see Section 2.1.3). We must therefore ask if the vaccine provides any benefits that are more substantial than the claimed reduction in the count of such trivial "cases."

2.2.1 Prevention of severe disease and mortality.

2.2.1.1 Severe disease and mortality. The study on adults [45] reports that 30 cases of "severe disease" occurred in the placebo group, and none in the vaccine group. The report states that

> *The majority of the severe cases were adjudicated as such based on SpO_2 [blood oxygen saturation] below the defining threshold of 93% for varying duration. Whereas reassuring for efficacy across varying disease severity, the cases overall seem mostly mild, which is a limitation of the dataset.*

The normal range of arterial blood saturation with O_2 is 95 to 100%; therefore, the use of a cut-off as high as 93% to diagnose a "severe" case seems questionable. It is noteworthy that only nine of these "severe" cases were hospitalized, and only two required admission to the intensive care unit (one of these two patients died). Thus, the number of truly severe cases possibly prevented by the vaccine is very small at best. The single fatal case does not suffice to prove efficacy against death.

No fatalities at all occurred in the cited study on adolescents [46]; and we already noted that this study does not report any cases of severe disease either. Therefore, in this specific age group, too, neither a meaningful benefit nor an emergency are in evidence.

We note that these collective findings not only answer the posed question in the negative, but they also dispose of the entire pretext for granting emergency use authorization for this experimental vaccine. If in a study that involves almost 30,000 individuals the number of severe cases or fatal outcomes is too small to permit the detection of any benefit of the vaccine, then surely no "emergency" exists that would justify the very grave risks, and meanwhile manifest harm, associated with the extraordinarily rushed introduction of this and other COVID-19 vaccines.

2.2.2 Effectiveness for those at high-risk of severe COVID-19. The purpose of vaccination is to evoke an immune response; therefore, the success of vaccination tends to be uncertain in those with immunosuppression. The clinical trials in adults on Moderna's mRNA-1273 vaccine don't adequately address efficacy in this group of patients. On page 105 of [45], we read:

> *In addition, no immune-suppressed subjects or subjects with immunodeficiencies were enrolled except for a limited number of HIV-infected individuals.*

And on page 149:

> *An immunogenicity and safety trial in immunosuppressed/immunodeficient people will be conducted post-authorisation as reflected in the RMP.*

With respect to more conventional comorbidities such as chronic lung and heart disease, Table 18 on page 100 claims that the vaccine has 90.9% efficacy.

Note that all of the foregoing comments on comorbidities apply to adults, most of whom are presumably of advanced age. Naturally, Moderna's clinical study on adolescents [46] is completely barren in this regard. Thus, no clinical benefit of Moderna's mRNA-1273 vaccine has been demonstrated in adolescents who may be at higher risk of severe COVID-19 due to underlying illness.

2.2.3 Duration of protection, and prevention of transmission. On page 110, the EMA report states:

It is presently not known if the vaccine protects against asymptomatic infection, or its impact on viral transmission. The duration of protection is not known.

The clinical trials indeed made no provisions for determining any effect on transmission; and the duration of protection (if any) could of course not be ascertained within such a short time. However, as noted above, the failure of the vaccine in the real world has since become apparent (see Section 2.1.2).

2.2.4 Inadequate efforts to determine the optimal dose. Moderna tested several different doses of the mRNA-1273 vaccine (10, 25, 50 100, and 250 µg). The EMA report does not lay out all findings with all dose groups, but it does show that there is very little difference in the levels of neutralizing antibodies with 50 and 100 µg, respectively (see Figure 3B); and the same applies to T-cell activation after doses of 25 or 100 µg (see Figures 10 to 12 in the EMA report). All of these findings suggest that lower dose regimens would provide levels of immunity very similar to that of the 100 µg dose that was ultimately selected. (Of note, a dose of 30 µg was selected for the Pfizer mRNA vaccine, which is very similar in nature to Moderna's vaccine.) Thorough and comprehensive dose-finding studies should therefore have been carried out before selecting the dose of 100 µg. This is of particular significance with children, whose lower body weights should suggest a dose reduction.

Furthermore, the graph shown in Figure 2 shows no decrease in cumulative incidence among the vaccinated after 30 to 50 days. Accordingly, the second injection, which was administered on or about day 28 after the first, has no detectable effect on clinical immunity. This observation should have prompted the evaluation of a single-dose regime, since omitting the second injection could significantly reduce the incidence of adverse events. However, on page 109 of the EMA report, we read:

No definitive conclusion on clinical efficacy after one dose can be drawn based on the very short time window between the two doses and consequently very few cases.

In other words, no separate trial group was established to evaluate the efficacy of a single-dose regimen.

3 The Moderna COVID-19 vaccine lacks safety

3.1 Composition and action mode of the mRNA-1273 vaccine. Moderna's mRNA-1273, like all other gene-based COVID-19 vaccines, causes the expression in vivo of one structural protein of SARS-CoV-2—namely, the so-called spike protein, which naturally occurs on the surface of the virus particle. The spike protein's function is to let the virus particle bind to the host cell and subsequently enter it. The key idea behind the mRNA-1273 vaccine is as follows:

1. a synthetic mRNA that encodes the spike protein is complexed with a mixture of neutral and synthetic lipids, which cluster together in lipid nanoparticles (LNPs);
2. after injection, the LNPs facilitate the uptake of the mRNA into host cells, where the mRNA will cause the expression (synthesis) of the spike protein;
3. the spike protein will appear on the surface of the host cells and induce an immune reaction to itself.

The immune reaction to the spike protein will comprise both antibodies, some of which will be able to neutralize the infectiousness of virus particles, and T-lymphocytes

(T-cells). Some of these T-cells are cytotoxic (also known as T-killer cells); their function is to kill virus-infected body cells.

While this vaccination strategy may look good on paper, it has a number of drawbacks and risks. These arise both from the lipid mixture and from the spike protein, both of which have known toxic activities.

3.2 What does the evidence show? With mRNA-1273 and with all of the other COVID-19 vaccines, clinical trials were rushed through in a very short period of time, which has meant that proper precautions to ensure their safety were not taken. However, animal experiments on mRNA-1273 that were carried out before the start of clinical testing already gave reason to expect severe toxicity, even though they were incomplete and deficient in many ways. Unfortunately, this expectation has been abundantly borne out in practice since the beginning of mass vaccinations.

3.2.1 Toxicity of the spike protein. The SARS-CoV-2 spike protein is a leading cause of the manifestations of severe COVID disease. Some of its harmful effects are mediated by the S1 fragment, a soluble protein molecule that is released from infected cells through proteolytic cleavage of the surface-anchored spike protein. The blood plasma level of S1 correlates with disease severity [50]. S1 can bind to ACE2 receptors on endothelial cells and on thrombocytes (blood platelets), which can promote blood clotting [51, 52]. The spike protein also damages the capillary barriers in the lungs and the brain [53, 54]. In addition to the ACE2 receptor, the protein binds to Toll-like receptor 4 (TLR-4) and to the cell surface protein CD209 (CLEC4M) [55]; these interactions extend the host cell range of the virus. TLR-4 in particular has been implicated in myocarditis, which is associated with the virus infection [56] and also a prominent side effect of the COVID vaccines, particularly in young men.

Against the background of this well-known toxicity, it is very peculiar that all of the current gene-based vaccines, including mRNA-1273, were designed to induce the expression of functionally active spike protein in the cells of our bodies[1] rather than of a "toxoid," that is, an immunogenic but innocuous derivative of the toxic protein. Toxoids can be produced with simple means and have been successfully used as vaccines for a long time, for example with diphtheria and tetanus, whose eponymous toxins can be rendered non-toxic by facile chemical modification. With modern methods of molecular biology, it should have been easy enough to create a non-toxic spike protein derivative for vaccination.

The concerns about vaccine-induced spike protein toxicity are not at all merely hypothetical. Blood plasma levels of S1 detected in vaccinated persons are comparable to those observed in severe cases of the viral infection [50, 57]. Accordingly, similarly grave detrimental effects on vascular integrity had to be expected after vaccination; and this is indeed borne out by a very large number of severe adverse events (see Section 3.2.6).

Aside from the direct toxicity of the spike protein, we must expect additional harm due to immune reactions against it. If the protein is expressed within vascular endothelial cells—the innermost cell layer of the blood vessels—then an immune reaction to it can destroy these cells. The resulting vascular lesion will again activate blood clotting. This immune reaction can involve cytotoxic T-cells, but also antibodies that trigger the complement system and other immune effector mechanisms. Note that this mechanism

[1] The two mRNA vaccines produced by Pfizer and Moderna incorporate two proline substitutions intended to stabilize the spike protein in a "pre-fusion" conformation. However, this does not prevent the proteolytic release of the S1 fragment, which appears to be responsible for much of the direct toxicity.

of cell damage will also operate in other tissues—any body cell that expresses the spike protein will thereby become a target for the immune system.

The immune reaction against cells that produce viral antigens is particularly dangerous in combination with the mRNA vaccine technology, since uniquely in this case *the vaccine particles do not contain any protein antigen, and therefore cannot be recognized by antibodies.* With a conventional live virus vaccine, existing antibodies will intercept the virus particles before they infect a cell, and therefore mitigate the destruction of cells by the immune system. In contrast, mRNA vaccines will "fly under the radar" of the antibody defence and reach the body cells unimpeded. The cells will then produce the spike protein, and subsequently be destroyed and attacked by the killer T-cells. The antibodies, rather than preventing the carnage, will join in by also binding to the cell-associated spike protein, which will mobilize the complement system and other immune effector mechanisms against these cells. In a nutshell, pre-existing immunity mitigates the risk of cell destruction with conventional live vaccines, but it amplifies the risk with gene-based vaccines.

Direct spike protein toxicity is significant because it does not involve an immune reaction and therefore can be triggered right away even in persons without pre-existing immunity. The immune attack mechanism will be particularly dangerous in persons with pre-existing immunity. Such immunity can arise from infection with the SARS-CoV-2 virus or from a previous injection of vaccine. In addition, cross-immunity induced by other coronaviruses (see Section 1.1.5) may also promote cell destruction through immune attack.

3.2.2 Lipid nanoparticle toxicity. In addition to the spike protein that is translated from the mRNA within our body cells, the lipids that mediate the cellular uptake of the mRNA (see Section 3.1) also contribute to vaccine toxicity. This is not too surprising, if we consider that their purpose is to overcome the cell's membrane barriers.

3.2.2.1 Cytotoxic effect of cationic lipids. The first step in process of vaccine particle uptake is *endocytosis*—the particle enters the cell, but it is still trapped within a membrane vesicle that budded of the cell membrane. The crucial step of releasing the mRNA from this vesicle (the endosome) into the cytoplasm is mediated by a synthetic cationic (positively charged) lipid. This compound with the short name SM-102 is likely the most toxic of the four lipid species contained within the lipid nanoparticles. It disrupts the function of the *mitochondria*, which are organelles within our cells which carry out "cell respiration"—they generate hydrogen and react it with molecular oxygen in order to produce ATP, the most important energy-rich metabolite of the cell. Disruption of mitochondrial metabolism will cause *reactive oxygen species* (ROS) to form. These ROS, in turn, can wreak all kinds havoc inside the cell, including genetic damage.

It should be noted that with any agent that causes genetic damage—this includes ionizing radiation, but also cytotoxic anticancer drugs—there is a risk of cancer and leukaemia, and moreover there is a lifetime limit on the overall dose that can be tolerated. Thus, the prospect of frequently repeated COVID "booster shots," and also that of extending mRNA technology to vaccines against other pathogens or non-infectious diseases, conjures up a very grave public health risk.

3.2.2.2 Sensitivity of lymphocytes to cytotoxic agents. Reactive oxygen species also mediate, to a large extent, the cytotoxic effects of ionizing radiation. A cell type that is particularly sensitive to radiation, but also to metabolically inflicted genetic damage,

are the lymphocytes.[2] Since the lymphocytes are the backbone of the adaptive immune system, we must expect that cationic lipid toxicity will cause immunosuppression. This is indeed borne out in clinical observations (see later).

3.2.2.3 The role of apolipoproteins in the organ distribution of lipid nanoparticles. Aside from the differences in susceptibility between cell types and tissues, we must also consider the distribution of the lipid nanoparticles within the body, which tends to resemble that of *lipoproteins*. Lipoprotein particles occur naturally in the bloodstream and within the tissues of our body. They consist of a core of lipids that is surrounded with a shell of proteins called *apolipoproteins*. Their purpose is to transport lipids such as cholesterol and triacylglycerol (regular fat) between organs. For example, a specific type of lipoprotein called *chylomicrons* transports dietary fats after they have been taken up in the small intestine. Other lipoproteins called VLDL and LDL distribute fats that have been synthesized in the liver to other organs and tissues.

The various apolipoproteins that encase the lipoproteins stabilize the particles, and they also serve as "address tags" that bind to receptor molecules on cell surfaces. This interaction will trigger the uptake of the lipoproteins into those cells. Artificial lipid nanoparticles (LNPs) like those used in the COVID mRNA vaccines can acquire a shell—a "corona"—of the body's own apolipoprotein molecules [59]. This corona then enables these vaccines to be taken up into the cells of our body, too.

The liver has a central place in lipid and lipoprotein metabolic turnover. Accordingly, liver cells are rich in specific surface receptor molecules which mediate lipoprotein uptake, suggesting that they will efficiently take up LNPs decorated with apolipoproteins also. This is indeed the case. However, other organs have high rates of lipoprotein uptake, too, and they must therefore be expected to accumulate the apolipoprotein-decorated vaccine LNPs as well. According to a study on the closely similar Pfizer mRNA vaccine [60], these organs include the adrenal glands, the ovaries, and the bone marrow. The spleen, too, accumulated large amounts of lipid nanoparticles; and the same must be expected of the placenta and of lactating breast glands, although these organs were not examined in the Pfizer study.

3.2.3 Animal studies on vaccine safety. The EMA report [45] contains only very limited animal data on the mRNA-1273 vaccine; however, some more detail was provided on rat studies which used other experimental mRNA vaccines of similar composition, including one that is directed against cytomegalovirus (CMV). Unless stated otherwise, the studies listed below were carried out with such model vaccines.

3.2.3.1 Distribution and elimination of the vaccine within the body after intramuscular injection. The mRNA component of the model vaccine was detected in multiple organs as soon as two hours after the injection, which indicates rapid onset of uptake into the bloodstream. The organs that accumulated the mRNA at levels higher than those found in the plasma included the eyes and the spleen. The extent of accumulation in the liver is not clearly stated in the EMA report, but the following quote suggests that it was high as well: "Liver distribution of mRNA-1647 is also evident in this study, consistent with the literature reports that liver is a common target organ of LNPs."

With the exception of the kidneys, the mRNA could be detected at lower levels in all other organs examined, albeit at lower levels. In particular, the mRNA was also found

[2] See in particular the example of adenosine deaminase deficiency, a metabolic disease that causes genetic stress to all body cells yet selectively eradicates the lymphocytes, which causes severe combined immunodeficiency (SCID) [58].

inside the brain, indicating that the model vaccine did to some degree overcome the *blood brain barrier*, a special provision found in the capillaries of the brain and the spinal cord which renders these capillaries much less permissive to traversal by blood solutes than the capillaries in most other tissues.

The half-life of the mRNA varied between tissues and ranged from 15 to 63 hours; it was shortest for the tissues at the injection site and longest for the spleen. The long half-life in the spleen, the body's largest lymphatic organ, accentuates the potential for severe toxicity to lymphocytes and thus to the immune system.

3.2.3.2 Mechanism of uptake into the circulation after intramuscular injection. Considering that the complex consisting of mRNA with bound LNPs has a rather large molecular size and therefore cannot cross an intact capillary barrier, we may wonder how the vaccine managed to enter the bloodstream so rapidly. This occurs most likely through lymphatic transport. The fluid within the interstitial space is continuously drained through the lymphatic system; all lymph fluid ultimately enters the bloodstream through the thoracic duct. Particles which are too large for traversing the capillary barrier can ultimately reach the circulation by way of this lymphatic drainage.

The SARS-CoV-2 spike protein, together with activation of the complement system, has been implicated in the causation of injury to small blood vessels in COVID-19 infections [61, 62]. Similar injury must be expected after vaccination near the injection site. The resulting leakiness of the capillaries should accelerate plasma exudation and lymphatic drainage. In addition, it may also permit some of the vaccine particles to enter the bloodstream directly.

3.2.3.3 Organ toxicity. Signs of organ damage were observed in the liver, the spleen, the adrenal glands, and also the bone marrow. It is notable that these organs were previously reported to take up high levels of the Pfizer vaccine [60]. This earlier report also found high uptake in the ovaries, which are not specifically mentioned in the EMA report on Moderna's vaccine.

As reported previously with the Pfizer vaccine, the liver showed hepatocellular vacuolation (a sign of cytotoxicity) and hypertrophy of Kupffer cells (resident macrophages) [45, p. 53]. Both of these effects suggest a high rate of uptake in this organ. The blood and the spleen showed diminished numbers of lymphocytes, whereas the blood and the bone marrow showed elevated numbers of cells of the myeloid lineage.

Cytotoxic effects of the LNPs are also evident from damage to muscle fibres at the injection site [45, p. 49]. We already noted the close similarity of Moderna's mRNA-1273 to the Pfizer mRNA vaccine. With the latter, damage to heart muscle cells was noted in mice after intravenous injection, and to some degree also after intramuscular injection [63]. This must also be expected with the Moderna vaccine.

3.2.3.4 Plasma proteins and blood coagulation. Blood levels of albumin decreased, which suggests either decreased production due to compromised liver function or accelerated loss from the circulation due to capillary leakiness, whereas blood levels of globulins increased. Of great concern is the activation of plasmatic blood coagulation, evidenced by the partial thromboplastin time.

3.2.3.5 Reproduction toxicity. In their study on reproduction toxicity, Moderna used the actual mRNA-1273 vaccine. The vaccine-injected rats showed a decreased fertility index, on which the EMA report comments as follows:

The overall pregnancy index was numerically lower in mRNA-1273- vaccinated female rats (84.1%), compared to control animals (93.2%), but remains within the Test Facility's historical control range (low range being 75%).

The EMA report also notes that an increased proportion of the foetuses showed aberrant numbers of ribs.

3.2.3.6 Genotoxicity. The studies presented by Moderna on this subject, while somewhat preliminary, raise serious concerns. The corresponding section, on page 50 of the EMA report, begins by stating that no evidence of genotoxicity was provided by in vitro studies using the two bacterial species *Escherichia coli* and *Salmonella typhimurium*. While these model organisms are indeed useful for studying the genotoxicity of small, individually soluble molecules such as for example alkyl epoxides or nitrosamines, they lack the machinery for the uptake of lipid nanoparticles into the cells. Therefore, these negative findings had to be expected and must be dismissed as irrelevant.

In Moderna's in vivo experiments with animals, *polychromatic* erythrocytes (red blood cells, RBC) were counted, as well as those with *micronuclei*. Polychromatic RBC are those which have only just finished their differentiation and disposed of their nuclei. At this stage, they still retain their ribosomal RNA, which causes them to appear bluish rather than red in the Giemsa stain. Changes in the percentage of RBC with this characteristic indicate changes in erythrocyte maturation kinetics. Genotoxic agents can cause both decreases [64] and increases [65] in this parameter. Differences between sexes are expected to be small. Using a luciferase-encoding mRNA packaged into a lipid mixture which contained SM-102, Moderna found a significantly decreased level of erythrocyte polychromasia, but only in male rats. This reported gender difference raises questions about the statistical power of this study.

Using another model mRNA and again a lipid mixture containing SM-102, Moderna found "statistically significant increases in micronucleated erythrocytes . . . in both sexes." A so-called micronucleus is a chromosome fragment which was produced by chromosome damage [65, 66] and then left behind in the cytoplasm when the main nucleus was expelled. The micronucleus assay as widely used to assess genotoxicity in vivo [66].

The EMA report quotes a Moderna study to the effect that the increased abundance of micronucleated RBC might have been due not to genotoxicity, but rather to the impeded clearance of these cells from the bloodstream as a consequence of spleen toxicity. However, no proof of this contention is shown; and the EMA report further states that "A strong increase in Molecular initiating event (MIE) was observed 48 hours after the final administration in the highest dose group in male rats." While no details are given as to the exact nature of the MIE, an "increase in molecular initiating events" clearly points to an actual increase in genetic damage rather than merely a decreased clearance of damaged cells.

In conclusion, while once more the data provided by Moderna are inadequate, they strongly suggest that SM-102 is indeed genotoxic. This agrees with prior observations of genotoxicity associated with similar cationic lipids in liposomes, reviewed for example by Inglut et al. [67].

3.2.4 Appraisal of the animal data: potential for grave harm. The findings discussed so far, limited though they are, indicate the potential of grave harm from the vaccine.

3.2.4.1 Manifest risks. The observations provided by the EMA report clearly point to the following risks:

- rapid uptake of the vaccine into the bloodstream implies a risk of blood clotting;
- the depletion of lymphocytes in the spleen and the blood suggests immunosuppression;
- penetration of the blood brain barrier indicates a risk of neurological damage;
- liver damage as well as risks to fertility and to pregnancy are manifest in the animals and must be expected in humans also.

Each of these risks should have been addressed by more thorough animal studies, and they should also have been carefully monitored in the so-called phase II/III clinical trials.

3.2.4.2 Potential risks to fertility and to the breastfed newborn. A high level of expression of spike protein in the ovaries raises the prospect of significant damage to that organ, with possible consequences for female fertility. Uptake of the vaccine by mammary gland cells opens two possible pathways of toxicity to the breastfed child: firstly, the expression of spike protein and its secretion into the breast milk, and secondly, the wholesale transfer of the vaccine into the milk. The mammary glands are *apocrine*, which means that they pinch off and release fragments of their own cytoplasm into the milk; thus, anything that has reached the cytoplasm might also reach the breast milk. In this connection, we note that both the VAERS database and the EU drug adverse events registry (EudraVigilance) report fatalities in breastfed newborns after vaccination of their mothers (see Section 3.2.6.7).

3.2.4.3 Moderna's failure to investigate risks evident from preclinical studies. With the exception of fertility, which can simply not be evaluated within the short period of time for which the vaccines have been in use, all of the risks discussed above have been substantiated since the vaccines have been rolled out—all are manifest in the reports to the various adverse event registries (see Section 3.2.6). We must stress again that each of these risks could readily be inferred from the cited limited preclinical data, but were not followed up with appropriate in-depth investigations. In particular, the clinical trials did not monitor any laboratory parameters that could have provided information on these risks, such as those related to blood coagulation (e.g. D-dimers/thrombocytes), liver damage (e.g. γ-glutamyltransferase), and myocarditis (troponin).

3.2.5 Contaminations arising from the manufacturing process. The commercial scale manufacturing process of mRNA-1273 gives rise to several contaminations that may compromise vaccine safety and effectiveness. For brevity, we will here mention only two such contaminants.

3.2.5.1 Contaminating bacterial DNA. The mRNA is produced in vitro using a DNA template, which in turn is obtained from bacterial cells. They EMA report implies that some of this DNA remains in the final product but gives no indication of the exact level [45, p. 18].

Contaminating DNA injected with the vaccine may insert into the genomes of host cells and cause potentially harmful mutations. Bacterial DNA also non-specifically promotes inflammation.

3.2.5.2 Lipid impurities. Lipids are notoriously difficult to purify, particularly on a large scale. Numerous impurities, not all of which were chemically identified, were detected with both of the synthetic lipid components. With regard to PEG2000-DMG, the EMA report states (page 25):

Table 2 COVID-19 vaccine-related adverse events and deaths reported to EudraVigilance, by manufacturer, as of September 11th, 2021 [68]

Manufacturer	Adverse events	Deaths	Deadly events
Moderna	112,252	6,358	5.7%
Pfizer	419,921	11,711	2.8%
AstraZeneca	370,122	5,254	1.4%
Janssen	26,833	1,203	4.5%
Total	929,128	24,526	2.6%

> *Numerical limits for specified and unspecified impurities will be included in the PEG2000-DMG specification post-approval. The current reporting of impurities is not acceptable. Characterisation data for impurities which are reported under 'content of unknown' should be provided post-approval.*

The novel cationic lipid SM-102 contains various contaminants, which the EMA report only summarily describes as follows (page 23):

> *The information provided on potential impurities in SM-102 comprise product related substances and process related impurities (elemental impurities, residuals solvents, peroxides, water content and inorganic impurities).*

Moreover,

> *CQAs [critical quality attributes], CPPs [control process parameters] and critical attributes of the materials used for the manufacture of SM-102 are missing.*

The report also states that "The applicant will provide an evaluation of mutagenic impurities", which implies that no such evaluation had yet been presented when emergency use authorization was granted.

Considering that the synthetic lipids referred to as SM-102 and PEG2000-DMG have never before been used on humans, there is no sound empirical basis for deciding on "acceptable" levels of impurities. Furthermore, it appears that some of the contaminating species have not even been identified. EMA's arbitrary blanket approval of unknown contaminants of unproven vaccine ingredients is completely unacceptable.

3.2.6 Adverse events after the onset of vaccinations. Since the introduction of the vaccines, numerous adverse events have been reported to registries around the world. We will here focus on two registries, namely, the U.S. vaccine adverse events reporting system (VAERS) and the EU monitoring system for drug adverse events (EudraVigilance).

3.2.6.1 Total cases and fatalities reported to EudraVigilance and VAERS. Table 2 summarizes the numbers of adverse events for each of the four COVID vaccines deployed in the countries of the European Union. We see very high numbers of incidents and fatalities across the board. Pfizer has managed to rack up the highest body count because their vaccine is the most widely used. The Moderna vaccine takes the second spot; it is also remarkable for its high percentage of reported events which are fatal.

The totals are somewhat lower but overall still appallingly high in the VAERS database. With VAERS, we can also obtain the case numbers and fatalities by age group. These data are summarized in Table 3, separately for the Moderna vaccine and for all COVID vaccines combined (also including Moderna). In this database, Moderna does not stand out for its proportion of fatal events, which here is slightly below the average. However, a

Table 3 Adverse events (total and deadly) reported to VAERS as of September 17th, 2021, by age group, for the Moderna mRNA-1273 vaccine as well as for all COVID-19 vaccines combined.

	Moderna			All		
Age (years)	Total events	Deaths	Deadly	Total events	Deaths	Deadly
0–10	92	1	**1.09%**	353	3	0.85%
11–20	824	10	**1.21%**	33,270	46	0.14%
21–30	2,290	47	**2.05%**	68,512	136	0.20%
31–40	41,462	98	0.24%	98,338	256	0.26%
41–50	41,705	141	0.34%	97,396	383	0.39%
51–60	43,522	323	0.74%	96,955	799	0.82%
61–70	47,840	661	1.38%	88,872	1,674	1.88%
71–80	33,261	920	2.77%	56,620	2,284	4.03%
>80	15,507	1,373	8.85%	30,213	3,964	13.12%
Total	226,503	3,574	1.58%	570,529	9,545	1.67%

very concerning observation is the high percentage of fatalities in the age groups of up to 30 years (highlighted in the table). As vaccination rates in these age groups increase, adverse events and fatalities must be expected to soar. Should the current trend continue, Moderna seems poised for a very large body count among children, adolescents, and young adults.

It is impossible to know what percentage of all fatalities that occur shortly after vaccination will actually be reported to VAERS or EudraVigilance. However, note that total of the COVID vaccine fatalities in VAERS already exceeds that reported for all other vaccines combined, over the entire 30 year period that this reporting system has been in existence. It is therefore clear that these vaccines are far and away the most deadly ones in history—quite predictably so, and all for a disease whose case fatality rate does not exceed that of influenza and is negligible in otherwise healthy persons (see Section 1.1.1).

3.2.6.2 Heart attacks and myocarditis or pericarditis by age group. It is generally accepted that, in COVID-19 disease, the spike protein of the virus triggers vascular lesions and blood clotting [62, 69, 70]. A prominent clinical manifestation of blood clotting is myocardial infarction (heart attack). Another form of cardiac involvement, also connected to the spike protein but purely inflammatory rather than related to clotting, is myocarditis [56].

Since all of the COVID vaccines induce the production of active spike protein, they, too, must be expected to cause heart attacks and myocarditis; and in fact both VAERS and EudraVigilance document a large number of cases. In Figure 5A, the cases of these diseases reported to VAERS have been grouped by age. The incidence of heart attack rises with age, which is expected. Note, however, that even the youngest age group, there are as many as 213 cases; this is highly irregular. Panel B of the same figure groups the reported heart attacks according to time elapsed since vaccine injection. Of all heart attacks reported, 49% occurred within one day of the vaccination, and 84% within one week. This close correlation in time very strongly points to causation by the vaccine.

From panel A, it is evident that the age distribution of myocarditis/pericarditis is practically a mirror image of that of heart attacks—it is highest in the youngest age group and drops continuously with age. Myocarditis in particular is a very serious condition in

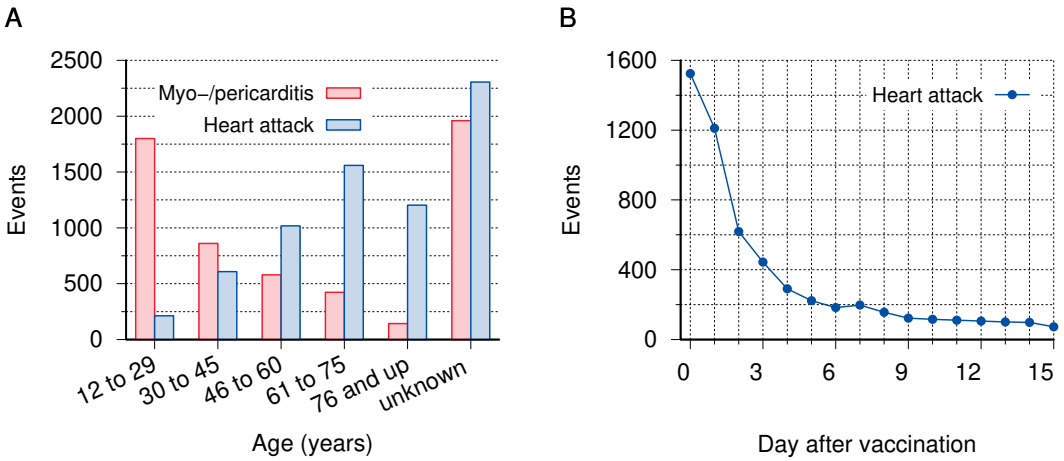

Figure 5 Myocarditis/pericardits and heart attacks reported to VAERS for all COVID vaccines combined, as of September 10th, 2021 [71]. A: Disease cases by age group. B: Reported cases of heart attack by day after vaccination.

its own right; it can be fatal in the acute phase and is likely to leave behind some measure of lifelong functional impairment. Thus, overall, all age groups are at substantial risk to suffer grave harm to their cardiovascular health from the vaccines.

3.2.6.3 Other severe events related to disrupted blood clotting. Aside from myocardial infarctions, the litany of diagnoses in both databases that indicate pathological activation of blood clotting is almost endless—strokes, thromboses in the brain and in other organs, pulmonary embolism; but also thrombocytopenia and bleeding, which result from excessive consumption of thrombocytes and of coagulation factors in disseminated intravascular coagulation. Clotting disorders caused many of the fatalities summarized above; in other cases, they caused severe acute disease, which will in many cases leave behind severe disability.

3.2.6.4 Other severe reactions. Severe reactions also include seizures and other neurological symptoms, particularly related to motor control, and severe systemic inflammation with damage to multiple organs. Again, in many of these patients, long-lasting or even permanent residual damage is highly likely.

3.2.6.5 Severe adverse reactions among adolescents. In the age group of 12-18 years, six deaths related to the Moderna vaccine were already reported to VAERS (see also Table 3). In the same age group, there were 39 cases of seizures, as well as cases of stroke, myocardial infarction, and severe inflammatory disease.

While the numbers of adverse events so far are much lower than those among adults, this must in large measure be ascribed to the hitherto far lower rates of vaccination in this age group. Should systematic vaccination be green-lighted for adolescents, we must expect these numbers to rapidly climb to a level resembling that seen in adults.

3.2.6.6 Miscarriages. As of September 10th, 2021, VAERS contains 1,812 case reports of miscarriage among vaccinated pregnant women. While it is difficult to ascertain what percentage of these miscarriages must be attributed to vaccination—the CDC claimed to have addressed this question [72], but had to admit in an erratum that this study was completely botched [73]—we must note that most of the cases in VAERS and in EudraVigilance were reported by healthcare professionals, who evidently considered a connection to the vaccine at least plausible.

This high number of reports alone would be reason enough to pause the vaccinations and investigate. We must also note that pregnant women had been excluded from the clinical trials on the Moderna vaccine, as well as on the other COVID vaccines. Continuing vaccination without proper investigation in the face of mounting indications of harm is completely irresponsible.

3.2.6.7 Deaths among breastfed infants. Although this issue does not directly relate to the age group which is the focus of this lawsuit and this expert opinion, it bears mention that both VAERS and EudraVigilance contain reports of gastrointestinal bleeding and death among breastfed children shortly after their mothers had received the Pfizer mRNA vaccine. Two similar cases, but without fatal outcome, which involve the Moderna vaccine are on file with VAERS.

In Section 3.2.2.3, we discussed the possibility of vaccine uptake into the placenta and the breast glands. The reported miscarriages and fatalities in newborns indicate that these risks must be taken very seriously, and that both Pfizer and Moderna acted negligently in not investigating them in any of their reported preclinical and clinical trials.

3.3 Missing evidence. We saw above that significant positive indications of risk were neglected in the clinical trials and in the subsequent rushed emergency approval of the Moderna vaccine, with unfortunate yet predictable outcomes. Equally damning is the list of omissions—potential risks that should have been investigated in preclinical or clinical trials but never were.

3.3.1 Proper pharmacokinetics. Section 3.2.3.1 described some experiments pertaining to the distribution and elimination of a surrogate vaccine. These studies were deficient in several ways:

1. They used mRNA molecules that encoded proteins other than the SARS-CoV-2 spike protein. Since the spike protein increases capillary leakage, including at the blood brain barrier [53, 74, 75], the possibility that expression of the spike protein will change the tissue distribution and penetration of the vaccine particles must be considered and experimentally addressed.
2. No studies were reported on the pharmacokinetics of the cationic lipid (SM-102) which is contained in the mRNA-1273 vaccine. The surrogate studies with "SM-86, a close structural analogue" [45, p. 53] are not an acceptable substitute. Likewise, proper pharmacokinetic studies on the second synthetic lipid component (PEG2000-DMG) are missing.

EMA should have insisted that the distribution and the elimination of both SM-102 and PEG2000-DMG be fully characterized in animal experiments.

3.3.2 Drug interactions. The EMA report states (page 119):

> *Study P301 was not intended to measure drug interactions or the impact of other vaccines being administered in a close temporal relationship to mRNA-1273, based on exclusion criterion 'Has received or plans to receive a non-study vaccine within 28 days prior to or after any dose of IP (except for seasonal influenza vaccine which is not permitted within 14 days before or after any dose of IP).'*

Immunosuppressive effects of mRNA-1273 are apparent from a drop of blood lymphocyte numbers among those vaccinated, as well as from clinical observations of Herpes zoster (shingles), which arises through the reactivation of persistent varicella-zoster

virus [76–78]. This suggests that the desired immune response to other vaccines administered shortly before or after the Moderna vaccine may be impaired. In real life, it is not always feasible to avoid the application of multiple vaccines within a short time frame. Therefore, this potential immunological interaction should have been studied.

Furthermore, studies of interactions should not have been limited to vaccines alone but also included other drugs, since many potential recipients of the vaccine will be on some kind of permanent medication. One area of particular concern is the experimentally apparent liver toxicity of mRNA-1273. The liver has a central place in the metabolic inactivation and disposal of many drugs; any interference with the function of this organ immediately creates numerous possibilities of adverse drug interactions.

3.3.3 Genotoxicity. We had seen in Section 3.2.3.6 that Moderna tried to explain away the observed increase in micronucleated erythrocytes as a consequence of impaired clearance in the spleen (i.e., of spleen toxicity). Aside from the fact that genotoxicity of cationic lipids is in fact well documented, the question whether micronucleated erythrocytes had been elevated due to increased formation or rather by decreased destruction within the spleen could have been answered experimentally by applying the micronucleus assay to cell types that are not subject to spleen clearance, such as for example fibroblasts or buccal epithelial cells [66]. EMA should have demanded that Pfizer provide such experimental answers.

3.3.4 Carcinogenicity. Genotoxicity implies carcinogenicity. Since the limited available evidence clearly points to LNP genotoxicity, experimental studies to determine the carcinogenic potential of the mRNA-1273 vaccine would have been necessary and urgent. However, on this point, the EMA report contains no more than this barren statement:

> *No carcinogenicity studies were submitted. This is scientifically acceptable and in line with relevant guidelines on non-clinical development of vaccine candidates. The components of the vaccine formulation are lipids and natural nucleosides that are not expected to have carcinogenic potential.*

In other words, Moderna was absolved of its responsibility to prove that its vaccine is not carcinogenic simply by using the semantic trick of calling this experimental gene therapy a “vaccine.”

The rationale behind imposing less stringent rules for safety tests on vaccines than on other kinds of drugs is that the chemical ingredients of conventional vaccines are usually well known. However, this is clearly not the case with the mRNA-1273 vaccine, which aside from the synthetic mRNA also contains two novel synthetic lipids (SM-102 and PEG2000-DMG) which have never before been approved as ingredients of clinical drugs or vaccines. In light of the strong indications of genotoxicity, the statement that these compounds “are not expected to have carcinogenic potential” is scientifically untenable.

Considering its mode of action, the mRNA-1273 vaccine should not have been treated like a conventional vaccine, but rather have been held to the same standard that applies to gene therapy products. The vaccine mRNA can undergo reverse transcription to DNA inside the target cell and then integrate into the cellular genome. For such products, the applicable FDA recommendation states that a long term follow-up study (LTFU) of up to 15 years is necessary. This study must include the investigation of new malignancies or haematological disorders, new incidence or exacerbation of a pre-existing neurological disorder, rheumatologic or other autoimmune disorder, or potentially product-related infection. The contrast between these comprehensive and rigorous conventional guide-

lines and the complete lack of long-term safety studies with the Moderna vaccine could not be more stark.

3.3.5 Reproduction toxicity. As noted in Section 3.2.3.5, the fertility index in rats which were injected with mRNA-1273 dropped by 9% (from 93% to 84%). The numbers of animals tested, and therefore the statistical power of this study, are not stated. EMA should have demanded that this study be conducted on a scale sufficient to decide whether or not the observed effect on the fertility index is statistically significant.

The EMA report notes that in these animal trials no vaccine doses were injected during the crucial developmental period of embryonic organogenesis. It is at this stage that teratogenic compounds induce severe malformations. Therefore, the teratogenic effect of the vaccine has really not been evaluated at all.

Moderna also neglected to report on the accumulation of vaccine in the placenta, as well as on the transmission of the vaccine in breast milk. The latter question has been independently studied by Golan et al., who found no vaccine mRNA in the breast milk of women previously vaccinated with the Pfizer or the Moderna vaccine [79], as well as by Low et al., who did find "minimal transfer" of mRNA in the breast milk of mothers after they had received the Pfizer vaccine [80]. Studies on the breast milk level of the spike protein, which might also cause toxic effects in the infant, seem to be lacking entirely. The case reports discussed in Section 3.2.6.7 prove that this risk must be taken very seriously.

3.3.6 Autoimmunity. Exposure to the vaccine will lead to cell damage due to the cationic lipids, and also to immune attack on cells producing the spike protein. From the cells undergoing destruction, proteins and other macromolecules will be released; such material must then be cleared away by macrophages.

When the clearing system is overloaded because of excessive cell damage and apoptosis (cell death), then the accumulation of cellular debris will lead to chronically excessive type I interferon release; this, in turn, will trigger further inflammation. With time, some macromolecules in the debris will become targets for the formation of autoantibodies and the activation of autoreactive cytotoxic T cells—they will begin to function as auto-antigens. This then leads to further tissue damage and the release of more auto-antigens—autoimmune disease will develop. Such an outcome is particularly likely in immunocompromised people or in those who are genetically predisposed to autoimmune disease (e.g. those with the HLA-B27 allele).

The risk of autoimmunity induced by mRNA-1273 could be adequately addressed only in long-term studies; as with fertility or cancer, the very short period of preclinical and clinical testing means that we are flying blind. It should go without saying that all of these risks are particularly grave with children, adolescents, and young adults.

3.3.7 Antibody-dependent enhancement. While antibodies in principle serve to protect us from infections, in some cases they can increase disease severity. This phenomenon is referred to as antibody-dependent enhancement.

3.3.7.1 The principle. Generally speaking, the antibody response to a virus will be composed of many different clonal variants (idiotypes); some, but not all of these idiotypes will neutralize the virus in question. While in most cases non-neutralizing antibodies are not harmful, with some viruses they can actually make matters worse by facilitating entry of these viruses into host cells. This occurs because certain cells of the immune system are supposed to take up antibody-tagged microbes and destroy them. If a virus particle to which antibodies have bound is taken up by such a cell but then manages to

evade destruction, it may instead start to multiply within this cell. Overall, the antibody will then have enhanced the replication of the virus. Clinically, this antibody-dependent enhancement (ADE) can cause a hyperinflammatory response (a "cytokine storm") that will amplify the damage to our lungs, liver and other organs of our body.

ADE can occur both after natural infection and after vaccination; in the latter case, it is sometimes referred to as vaccine-associated enhancement of disease (VAED). The effect has been observed with several virus families, including Dengue virus, Ebola virus, respiratory syncytial virus (RSV), and HIV [81]. Importantly, ADE also occurs with coronaviruses, and it has been documented in particular with SARS, whose causative agent is closely related to SARS-CoV-2. Attempts to develop vaccines to SARS repeatedly failed due to ADE—the vaccines did induce antibodies, but when the vaccinated animals were subsequently challenged with the virus, they became more ill than the unvaccinated controls (see e.g. [82]).

3.3.7.2 SARS-CoV-2 and ADE. The possibility of ADE in the context of natural infection with SARS-CoV-2, as well as of vaccination against it, has been acknowledged [83]. More specifically, ADE due to spike protein antibodies elicited by other coronavirus strains has been invoked to account for the peculiar geographical distribution of COVID clinical disease severity within China [84]. However, the experimental research required to address ADE remains missing, even after more than one year into the pandemic.

With some experimental SARS vaccines, ADE could be mitigated through the use of inulin-based adjuvants [85]. This approach might be feasible for avoiding ADE with COVID-19 vaccines also, but so far this appears not to have been investigated with any of the existing COVID vaccines.

Moderna and the regulatory bodies are well aware of the risk of ADE as well. The EMA report summarizes the information supplied by Moderna as follows (page 126):

> *The potential risk of VAED was assessed in non-clinical animal models in mice and non-human primates and raised no concerns based on a Th1 skewed type of immune response … In the pivotal [clinical] trial, … 30 cases of severe COVID-19 were reported in the placebo group, while 0 case was reported in the vaccine group, providing no signal for a possible disease enhancement after vaccination with mRNA-1273.*

Overall, it is clear that the risk of ADE is recognized in theory but has not been addressed in practice with any degree of rigour; in particular, no animal trials with virus challenge after immunization have been reported. Given the abundant evidence of ADE with experimental SARS vaccines, this is unacceptable.

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