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Vaccine-Encoded Spike in the Nucleus?

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Abstract

Whether spike protein is present in skin, and where it comes from, has been asked twice: first of infected patients, then of vaccinated ones. Detection in the infection era rested on immunohistochemistry with reagents later shown, by one of the groups that used them, to stain pre-pandemic tissue. Nucleocapsid is actively secreted independently of virion assembly and circulates two orders of magnitude above free spike. Its presence in perfused tissue says nothing about local replication. Endothelial cells lack ACE2 and are not productively infected. This excludes local viral synthesis as a source of endothelial spike. Against this background, several vaccine-era reports share a feature the earlier literature lacks: spike positive with nucleocapsid negative, in patients whose infection has been serologically excluded. The intervals from mRNA injection range from 10 to 1,364 days.

Keywords: SARS-CoV-2; Spike Glycoprotein; COVID-19 Vaccines; Skin Biopsies; Dermatopathology; Endothelial Cells; Immunohistochemistry; Nuclear Localization; Cutaneous Lesions; Nucleocapsid Protein

Abbreviations

SARS-CoV-2: Severe Acute Respiratory Syndrome Coronavirus 2; COVID-19: Coronavirus Disease 2019; ACE2: Angiotensin-Converting Enzyme 2; mRNA: Messenger Ribonucleic Acid; RNA: Ribonucleic Acid; DNA: Deoxyribonucleic Acid; PCR: Polymerase Chain Reaction; ddPCR: Droplet Digital Polymerase Chain Reaction; RT-PCR: Reverse Transcription Polymerase Chain Reaction; TNF-α: Tumor Necrosis Factor Alpha; IL-6: Interleukin-6; ERG: ETS-Related Gene; CD31: Cluster of Differentiation 31; IgG4: Immunoglobulin G4; DAPI: 4′,6-Diamidino-2-Phenylindole; PBMC: Peripheral Blood Mononuclear Cell; ORF: Open Reading Frame; SV40: Simian Virus 40.

Introduction

Dermatologists in Lombardy saw the first wave before most clinicians elsewhere. Six cutaneous phenotypes were described within weeks. The question followed at once: is the virus itself present in the skin? Five years later the answer is less secure than the number of publications suggests. Some of the methods used to reach it have since been shown to be unreliable, in several cases by the groups that first used them. Endothelial cells do not express ACE2, and SARS-CoV-2 does not productively infect them [1-5]. Any spike protein found in an endothelial cell therefore did not arise from viral replication in that cell. The literature now reports spike protein in skin after mRNA vaccination.

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The Infection-era Evidence

Marzano AV, et al. [6] examined 52 patients with COVID-19-associated skin lesions. They used immunohistochemistry for nucleocapsid, spike and ACE2, droplet digital PCR, and RNAscope in situ hybridisation. Twenty of 52 were positive by at least one method. The reagents are given in the supplement. Nucleocapsid: clone B46F, Thermo MA1-7404, 1:100. Spike: GeneTex GTX135356, a rabbit polyclonal, 1:300. ACE2: R&D MAB933. The specificity check consisted of five psoriasis and five basal-cell carcinoma biopsies. All were pandemic-era. No pre-pandemic tissue was examined. The check is described as testing monoclonal antibody specificity, which leaves the polyclonal spike reagent uncovered. Of 22 positive localisations, 15 lie in eccrine glands or their glomeruli, six at small dermal vessels, and one in sebaceous glands. The single in situ hybridisation positive in 52 patients was grade 1+, the lowest positive grade on the authors' own scale, and lay in the upper layer of parakeratosis. Protein and transcript were therefore never found in the same cells. No ddPCR copy numbers are reported.

Some numbers do not correspond. The abstract reports twelve patients positive for nucleocapsid only. The per-patient table supports ten, because patients 3 and 8 are positive for both antigens; ten and ten give the stated total of twenty. The Discussion reports endothelial positivity for SARS-CoV-2 in eight cases. The table records spike at small dermal vessels in three patients and nucleocapsid at the same site in three others.

Antibody Specificity

These reservations are not hypothetical. Gehlhausen JR, et al. [7] stained pre-pandemic autopsy tissue microarrays, confirmed to contain only material from before 2019. Three antibodies were tested: GeneTex GTX632604 and Sino Biological 40150-T62-COV against spike, and Sino Biological 40143-R001 against nucleocapsid. All three gave positive signals in all microarrays, in 6 to 17 per cent of cores. Most positive cells were epithelium; some were macrophages. Isotype controls and COVID-19 lung positive controls were run in parallel. Massoth LR, et al. [8] had reported 53 per cent specificity for SARS-CoV-2 immunohistochemistry against 100 per cent for in situ hybridisation. The paper is a self-correction. Members of the same group had published positive spike staining in the endothelium and eccrine glands of perniotic biopsies, and had proposed deposition of cleaved spike to explain the discordance between protein and RNA [9]. Those three cases were later negative for nucleocapsid and negative for spike mRNA. Among 21 pandemic chilblain patients, 2 (9.5 per cent) showed evidence of prior infection, against a regional seroprevalence of 8.5 per cent.Endothelium and eccrine glands are the two structures Marzano reports. The deposition paper [9] is cited there. The group had also published an earlier negative immunohistochemical series [10].

Nucleocapsid Is Not a Marker of Local Infection

Zheng and colleagues showed that nucleocapsid is actively released from infected cells, independently of virion assembly [11]. Lysis was excluded by supernatant lactate dehydrogenase, cell viability, and the absence of beta-actin from the extracellular fraction. Secreted nucleocapsid is the predominant circulating form. A second group describes a different export route [12]; the route is disputed, the extracellular abundance is not. Released nucleocapsid binds neighbouring cells, including uninfected ones, through heparan sulphate [13].

Coppens L, et al. [14] report the ratio between nucleocapsid and spike in plasma. During acute infection nucleocapsid was quantifiable in 98 of 99 cases, with a median of 253,670 fg/mL and values frequently above one million. Free spike was detectable in 15 of 99. The limits of quantification were 172 fg/mL for nucleocapsid and 350 fg/mL for spike. Pre-pandemic samples were negative for both. Because spike lay below the limit in 85 per cent of samples, the two medians cannot be divided; the asymmetry is at least two orders of magnitude. Dithiothreitol did not unmask antibody-bound spike: it reduced recombinant spike detectability by 90.9 per cent, yet raised the measured signal 2.6-fold in pandemic plasma and 5.5-fold in pre-pandemic plasma.

In acute infection the circulating antigen pool is nucleocapsid. Nucleocapsid is exported rather than virion-bound, and it adheres to cells that were never infected. Nucleocapsid immunoreactivity in a perfused, heparan-sulphate-rich structure carries no information about local replication. Endothelium and eccrine epithelium are such structures.

The Endothelium

Endothelial cells lack ACE2. SARS-CoV-2 does not productively infect them. Both statements rest on primary data from several independent groups. Schimmel L, et al. [1] tested endothelial infection directly. They used umbilical vein and lung microvascular endothelial cells, a three-dimensional vessel under flow, an epithelial–endothelial co-culture, and autopsy lung from ten patients. Endothelial ACE2 and TMPRSS2 protein were low. Entry occurred only at very high inoculum and was abortive. Productive replication

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required ACE2 overexpression. No infection occurred under flow or in co-culture. In the autopsy material no spike mRNA was found in endothelial cells. Subramaniam S, et al. [2] reviewed the field and reached the same conclusion. Their own immunohistochemistry found no nucleocapsid in endothelial cells in animal models or in human post-mortem lung. McCracken IR, et al. [3] found no evidence of ACE2 expression or of replicative infection in human endothelial cells. Muhl L, et al. [4] localised vascular ACE2 to pericytes in mice. Mezache L, et al. [5] reached a comparable conclusion in human myocardium, assigning ACE2 to pericytes and perivascular macrophages. Endothelial spike cannot arise from local viral replication. Deposition from the circulation is a separate question and is limited. Free spike circulates in 15 per cent of acute infections, orders of magnitude below nucleocapsid [14]. In a patient whose infection has been serologically excluded there is no viral source at all. One reagent point belongs here. Gawaz and colleagues identify their antibody only as a GeneTex mouse monoclonal [15]. The common GeneTex mouse monoclonal is clone 1A9 (GTX632604), which binds recombinant S2 ectodomain and is usually treated as S2-directed. If that is the reagent, the detected species contains S2. After cleavage at RRAR, S2 remains membrane-associated, whereas S1 is the shed fragment that most plasma assays measure. Deposition would then require a circulating S2-bearing species: uncleaved trimer, vesicle-associated protein, or S2′. The same catalogue number is one of the three antibodies that stained pre-pandemic tissue [7].

The Standing of the Case Reports

The evidence that follows consists largely of single patients and small series. The weight such evidence carries is often misjudged. In pharmacology, recognition of serious adverse drug effects has repeatedly begun with one case. McBride WG [16] reported thalidomide embryopathy in a letter of a single paragraph. Herbst AL, Ulfelder H and Poskanzer DC [17] established the association between maternal diethylstilbestrol and clear-cell adenocarcinoma of the vagina from eight cases. The oculomucocutaneous syndrome of practolol, a mucocutaneous reaction, was assembled from scattered clinical reports before any trial examined it [18]. Valvular disease associated with fenfluramine–phentermine was recognised from 24 patients [19]. In each instance the prevailing view at the time was that the association was anecdotal.

Randomised trials are powered for efficacy and for anticipated events. A harm that is rare, delayed in onset, or absent from the study protocol is invisible to them, however large they are. Attentive clinical observation is the instrument that detects such a signal, and often the only one available for years afterwards.

Spike Without Nucleocapsid After Vaccination

Magro CM, et al. [20] reported spike in endothelial cells of deep dermal and subcutaneous microvessels after mRNA vaccination, with caspase-3, TNF-alpha and IL-6, in 10 cases weeks after mRNA injection, and at 7 to 14 days in deltoid biopsies from the authors.

Yamamoto M, et al. [21] described persistent varicella-zoster infection after BNT162b2 in a 64-year-old man. Spike was present in vesicular keratinocytes and in dermal and subcutaneous venular endothelium. Biopsies were taken on days 24 and 88 of disease.

Sano H, et al. [22] described an 86-year-old woman with maculopapular erythema persisting beyond three months after a second dose. Spike S1 was present in vascular endothelium and eccrine glands of the deep dermis at 100 days, confirmed as endothelial by CD31.

In a 53-year-old woman with miliaria-like lesions 15 months after a third dose, spike was found in the acrosyringium and eccrine gland, co-stained with carcinoembryonic antigen; no nucleocapsid staining was obtained [23]. In an 85-year-old woman whose breast carcinoma metastasised to skin within a month of a sixth dose, both antibodies are named: spike GTX135356 and nucleocapsid GTX635679 [24]. The metastatic cells were spike-positive and nucleocapsid-negative. The primary tumour, resected eighteen months earlier from the same patient, was negative for both. That control is unusual and deserves attention. It is autologous. It comes from the same tumour lineage, in the same patient, processed with the same reagents, and taken before the exposure. A non-specific antibody does not distinguish a primary from its own metastasis.

Hulscher N and colleagues followed a 55-year-old man for 3.7 years after three doses [25]. Anti-nucleocapsid antibodies were undetectable at five time points between 809 and 1,433 days, across three independent laboratories, while anti-spike remained elevated. Three serial punch biopsies were taken at 1,160, 1,249 and 1,364 days, all from truncal skin showing clinically active lesions of transient acantholytic dermatosis. All three showed spike protein and no nucleocapsid. The antibodies are named: anti-spike S1 ProSci 9083 and anti-nucleocapsid ProSci 35-720, with transfected cells run as a positive control for spike and a negative control for nucleocapsid.

The molecular findings are compartment-specific, and the distinctions matter. Free spike protein was measured in plasma at 129.0 fg/mL and in plasma-derived exosomes at 11.6 fg/mL at 1,173 days; by 1,284 days it was undetectable

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in both, while spike-specific IgG4 remained elevated. At 1,284 days, RT-PCR of exosomal RNA amplified three separate regions of the spike open reading frame, giving bands of the expected size; PBMC RNA from the same time point showed no spike message. In PBMC DNA at the same time point, the two plasmid replication-origin sequences were amplified and confirmed by Sanger sequencing, while spike DNA and the SV40 enhancer were negative in every PBMC fraction.

The skin finding is a DNA finding. In tissue DNA extracted from the 1,364-day biopsy, PCR detected spike gene fragments across all three amplicons, both origin-of-replication sequences and the SV40 enhancer, with Sanger confirmation of the replication-origin fragments. No spike message was reported in skin. The SV40 enhancer, absent from every blood fraction, was present in the tissue.

The authors state their own principal limitation: although the replication-origin fragment matches the vaccine plasmid backbone by sequence, mononuclear cells can internalise extracellular DNA of other origin, so the biological source of that signal cannot be assigned with certainty.

These reports share a property the infection-era studies lack. Spike positive with nucleocapsid negative, in a patient whose infection has been excluded serologically, is not explained by deposition of circulating viral antigen: during infection that pool is nucleocapsid, not spike. Nor is it readily explained by non-specific binding, since the naive-tissue reactivity shown by Gehlhausen appeared with anti-nucleocapsid reagents as well. An antibody panel that binds host antigens indiscriminately should not produce a clean dissociation between the two antigens.

Study	Exposure	Interval to Biopsy	Result
Marzano 2023 [6]	Infection	Median 21.5 d from onset (range 5–46 d)	Antigen in 20/52; transcript in 1/52, not co-localised
Gehlhausen 2022 [7]	Infection (presumed); pandemic chilblains	Not stated as days from onset	No excess of prior infection; pre-pandemic tissue stains with the reagents
Magro 2021 [20] (10 cases plus 2 deltoid biopsies)	Vaccination	Deltoid biopsies 7–14 d; further cases weeks after injection	Spike in deep dermal and subcutaneous microvascular endothelium, with caspase-3, TNF-alpha and IL-6
Yamamoto 2023 [21]	Vaccination (BNT162b2)	Days 24 and 88 of disease	Spike positive in keratinocytes and venular endothelium
Sano 2023 [22]	Vaccination, 2nd dose	100 d	Spike positive in endothelium and eccrine glands; nucleocapsid not tested
Sano 2024 [23]	Vaccination, 3rd dose	About 15 months	Spike positive in acrosyringium and eccrine gland; nucleocapsid negative
Sano 2025 [24]	Vaccination, 6th dose	Metastasis under 1 month; primary resected 18 months earlier	Spike positive, nucleocapsid negative in metastasis; both negative in the autologous primary
Hulscher 2026 [25]	Vaccination, 3 doses; transient acantholytic dermatosis	Skin at 1,160, 1,249 and 1,364 d; serology 809–1,433 d; blood fractions 1,173 and 1,284 d	Spike positive, nucleocapsid negative in all three biopsies; anti-nucleocapsid seronegative throughout; spike message in exosomes at 1,284 d; spike, ori1/ori2 and SV40 enhancer in tissue DNA at 1,364 d
Gawaz 2026 [15], C10	Vaccination; COVID-arm, lesional but not vasculitic	10 d	Spike positive
Gawaz 2026 [15], V2	Vaccination; vasculitic lesion	12 d	Spike positive (cytoplasmic)
Gawaz 2026 [15], V4	Vaccination; vasculitic lesion	540 d	Spike positive
Gawaz 2026 [15], V5	Vaccination; vasculitic lesion	360 d	Results text reports spike; Table I records V5 as negative
Gawaz 2026 [15], V6	Vaccination; vasculitic lesion	450 d	Spike positive
Gawaz 2026 [15], V8	Vaccination; livedo racemosa / cutaneous polyarteritis nodosa	First biopsy 360 d; second biopsy 1,380 d (46 months)	Spike positive; cytoplasmic at the first biopsy, nuclear at the second

Table 1: Interval from exposure to biopsy, and staining result.

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Intervals are given in days (d) from mRNA injection, or from onset of infection where that is the exposure. N denotes nucleocapsid protein. Gawaz A, et al. [15] entries are restricted to vaccinated patients and are listed one patient at a time. First author only, with the reference number.

Spike Protein in the Nucleus

Gawaz and colleagues studied five patients with persistent or delayed vasculitic lesions after vaccination, selected from 175 histologically confirmed cases [15]. Endothelial spike was reported in all five by the Results text, and in four of five by Table I, which records V5 as negative. The controls are set out in the text. Imaging was by Zeiss LSM 800 confocal microscopy on 3-micrometre sections, with ERG and CD14 as counterstains. The positive patients showed cytoplasmic deposits. Two, V8 and V9, showed nuclear deposits as well. V8 is a 19-year-old woman with livedo racemosa and cutaneous polyarteritis nodosa. Two biopsies were available, the first at 12 months (360 days) and the second 46 months (1,380 days) later. Spike was cytoplasmic in the first and nuclear in the second, shown as a line-profile intensity graph through an endothelial cell. A comparable observation appears in the Kochi series. In the breast-carcinoma metastasis, spike was distributed in the cytoplasm and nuclei of the tumour cells, with a different antibody [24].

A molecular basis exists. The sequence 681-PRRARSV-687 at the S1/S2 junction has been identified as a putative pat7 nuclear localisation signal, lying within the polybasic region that also carries the furin site [26]. Sattar and colleagues tested nuclear localisation experimentally [27]. They used confocal microscopy with z-stacks and orthogonal views, deconvolution, and surface rendering, and confirmed the result by subcellular fractionation and western blotting in transfected A549 cells, with lamin A/C and Cdc42 as markers. In infected primary bronchial epithelial cells from three donors, about 10 per cent of spike was at the nuclear surface or within the nucleus, and about 1 per cent of spike mRNA was within the nucleus.

The motif also spans the furin site. Cleavage separates its halves between S1 and S2. If the motif drives import, the imported species is uncleaved spike or an S1 fragment, not S2. Antibodies against S1 and against S2 should therefore be compared. Discordance between them would itself be informative.

Open questions

No study discussed here has shown spike protein and spike transcript in the same cells of human skin. The single in situ hybridisation positive in the Marzano series lies in parakeratosis, away from the protein signal. Gehlhausen et al. found no spike mRNA in the spike-positive cases. Sano S, Yamamoto M and Gawaz did not perform in situ hybridisation. Sano S [24] calls for it. A separate series has reported spike, without nucleocapsid, in cerebral arterial intima after mRNA vaccination, and did use in situ hybridisation [28].

Reagent reporting is incomplete. GTX135356 appears in Marzano, where endothelial and eccrine staining is attributed to infection, and in Sano, where the same pattern is attributed to vaccination. Gawaz A, et al. [15] identify their antibody only by vendor and species. Hulscher and colleagues name both antibodies and run a transfected-cell positive control, which is the fullest reagent reporting in the group of studies considered here. Whether MA1-7404, used by Marzano et al. at 1:100 and listed by Gehlhausen et al. at 1:400, was among the antibodies applied to the pre-pandemic microarrays cannot be determined from the published text.

The pre-pandemic controls in Gawaz A, et al. [15] are few.

Absence of ACE2 excludes productive infection and receptor-mediated entry. It does not exclude attachment. Heparan sulphate is an established attachment cofactor for spike [29]. The endothelial glycocalyx is rich in it. This is the mechanism invoked above for nucleocapsid and may be available to spike as well. The nuclear observation rests on few patients. In Gawaz A, et al. [15] it rests on single-plane line profiles rather than z-resolved reconstruction. ERG is a nuclear endothelial transcription factor and was co-stained. Concordance of spike with ERG would be stronger than concordance with DAPI, and may be recoverable from the existing images.

Conclusion

The case reports assembled here were made independently. They come from laboratories in Germany, Japan and the United States, from different clinical presentations, using different antibodies against different subunits of the protein. They converge on one observation: spike protein is detectable in skin long after mRNA vaccination, from months to years. In patients in whom infection has been excluded serologically, it is found without the nucleocapsid that infection would deposit alongside it. How often this occurs is unknown, because no one has looked at it systematically. That is normal at this stage of pharmacovigilance: thalidomide embryopathy entered the literature as one brief letter, and the denominator followed later.

The experiments that would settle the matter are not difficult. In situ hybridisation for spike mRNA in the sections

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that show spike protein, in vaccinated and serologically infection-naive patients, with transcript and protein in the same cells. Full reagent disclosure, with pre-pandemic tissue stained under the identical protocol in each laboratory. Anti-nucleocapsid staining in parallel in every case, with a positive control showing that the antibody works in that tissue. Confocal z-stacks with orthogonal reconstruction wherever a nuclear claim is made. Identification of vaccinal spike by immuno-multiple reaction monitoring (immuno-MRM) [30], targeting the double-proline peptide, may be possible in future where material permits. Several of these could be performed on archived material already held by the authors cited here. Until they are, the question in the title remains a question.

Conflicts of Interest

The author declares no conflict of interest.

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