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Heterogeneity of NeuN Protein Distribution as a Marker of Morphological Personalization of Cerebral Cortex Neurons: an Experimental Study

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Summary

Aim. To identify personalized morphological neuronal phenotypes based on the distribution pattern of the neuronal protein NeuN in the cerebral cortex layers.

Materials and Methods. A histologic study of the cerebral cortex was performed in rats (*N*=10). Tissue sections were stained with hematoxylin and eosin, and the neuronal nuclear protein NeuN was visualized by immunohistochemical staining. Analysis was performed by microscopy and image analysis software.

Results. NeuN immunohistochemical staining revealed distinct localization and intensity patterns within cortical neurons. Contrary to the definition of NeuN as a nuclear neuronal protein, its localization was observed in both the nucleus and cytoplasm in most neurons. The following neuronal phenotypes were identified based on NeuN staining patterns:

1) Neurons with stained nuclei but unstained cytoplasm;
2) Neurons with stained cytoplasm but unstained nuclei;
3) Neurons with stained nuclei and cytoplasm;
4) Fully stained neurons with no visible nuclei;
5) Neurons with stained processes (dendrites/axons).

A significant difference was found between mean intensity of NeuN-positive neurons depending on the localization in the layers of the cerebral cortex.

Conclusion. Given the critical biological role of NeuN, the identified neuronal phenotypes based on NeuN localization warrant further research as they may reflect the functional states of neurons. The interpretation of the absence of NeuN staining as a marker of neuronal damage is not scientifically justified. Future studies using NeuN immunohistochemical staining should consider not only the total number of NeuN-positive neurons, but also their distinct phenotypes.

Keywords: NeuN protein; personalized neuronal phenotyping; neuronal phenotypes; cerebral cortex; immunohistochemistry; morphology; morphometry.

Conflict of interest. The authors declare no conflict of interest.

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Introduction

The NeuN protein was discovered during research on monoclonal antibodies (mAb) targeting brain cell nuclei. One of these antibodies, mAb A60, recognizes a nuclear protein specific to vertebrate neurons. This protein has been named NeuN (neuronal nuclei). Immunohistochemically detectable NeuN first appears when a neuron exits the cell cycle or begins terminal differentiation [1].

For a long time, the protein to which the A60 antibody binds was unknown. NeuN was first identified as Fox-3, also known as Rbfox3 [2], in 2009. The mammalian genome contains three genes: Fox-1, Fox-2, and Fox-3. The Fox mammalian proteins play a role in regulating mRNA splicing. An alternative name for these three proteins is hexaribonucleotide-binding protein 1, 2, and 3 (HRNBP1, HRNBP2, and HRNBP3).

Thus, NeuN (Fox-3, Rbfox3, or hexaribonucleotide-binding protein-3) is a neuronal nuclear antigen that is widely used as a biomarker for neurons.

This neuron-specific nuclear protein is consistently expressed in most postmitotic neurons of the vertebrate nervous system. The absence of NeuN staining has been interpreted as a marker of neuronal damage in several studies [3–6]. Studies using primary antibodies against NeuN have been performed on both experimental and human autopsy

material. Literature data highlight the potential of NeuN in the study of neuronal responses to injury, including in interspecies comparative studies [7].

However, many studies question the role of NeuN as a marker of intact neurons, noting that its staining is variable and may be absent under certain pathological or physiological conditions [8]. It has been shown that the presence or absence of immunohistochemical (IHC) reactions with primary antibodies against NeuN depends on the phosphorylation status of NeuN, which serves as an epitope for Rbfox3, a member of the Rbfox1 splicing factor family [9]. Thus, the absence of NeuN staining may not indicate neuronal death or complete loss of neuronal expression, but rather a reduction in NeuN protein expression or a loss of NeuN antigenicity [10].

Previously, we observed significant variability in NeuN staining in the cortical neurons of patients who died of COVID-19 [11]. However, the factors leading to decreased protein expression or loss of antigenicity remain poorly understood. Furthermore, there is limited information on the relationship between neuronal functional activity and the characteristics of NeuN staining and localization.

The aim of the study was to identify personalized neuronal morphological phenotypes based on the distribution patterns of the neuronal protein NeuN in the cerebral cortex layers.

Materials and Methods

Ten brain samples were collected from male Wistar rats weighing 200–250 g (N=10) for histologic analysis. Experiments were conducted in accordance with Directive 2010/63/EU of the European Parliament and of the Council of the European Union on the protection of animals used for scientific purposes. Rats were euthanized by cervical dislocation under general anesthesia with intraperitoneal administration of zolazepam (20 mg/kg) and xylazine (5 mg/kg). Brain tissue was fixed in 10% neutral formalin and processed by standard paraffin embedding techniques. Tissue sections of 4 μm thickness were stained with hematoxylin and eosin.

The nuclear neuronal protein NeuN was visualized by immunohistochemical staining. Tissue sections were deparaffinized in xylene and rehydrated in graded ethanol. Heat-induced epitope retrieval was performed in citrate buffer, pH 6.0 (Target Retrieval Solution, DAKO, Glostrup, Denmark). Sections were cooled, washed three times in distilled water, and rinsed three times for 5 minutes each in phosphate-buffered saline (PBS) with Tween (IHC Wash Buffer + Tween, Cell Marque, Rocklin, CA, USA). Endogenous peroxidase activity was blocked by incubating sections in 3% hydrogen peroxide for 10 minutes. To prevent non-specific binding of primary or secondary antibodies to tissue proteins, a protein blocking solution (Protein Block Serum-free, Abcam, Cambridge, UK) was applied for 30 minutes.

Sections were incubated for 1 hour at 37°C with primary antibodies against NeuN (Abcam, Cambridge, UK) diluted in antibody diluent (Abcam, Cambridge, UK). After incubation, sections were washed three times for 5 minutes each in PBS. Antibody-antigen binding was detected using a commercial kit (Diagnostic BioSystems, Netherlands) containing secondary antibodies and chromogenic substrate (DAB). After washing in PBS, the sections were counterstained with hematoxylin, rinsed in running tap water, dehydrated in graded ethanol, and mounted under cover slips.

Samples were analyzed using a Nikon Eclipse Ni-U microscope. The mean staining intensity of NeuN-positive neurons was determined based on the Mean Density parameter using NIS-Elements BR image analysis software.

Statistical analysis was performed with IBM SPSS Statistics 29.0. A nonparametric statistical method using the Kruskal-Wallis test was used for comparative analysis between groups. Post hoc analysis was performed using Dunn's test with Bonferroni correction. Differences were considered statistically significant at $P<0.05$ (two-tailed test). Statistical data were reported as medians with interquartile range.

Results

In the small neurons of the first cortical layer, NeuN was localized to the periphery of the nuclei.

In the nuclei of the second layer, the nuclear periphery was stained, while the central part of the nuclei showed diffuse and less intense staining. Weak and diffuse staining was observed in the cytoplasm of the neurons. In some neurons, the nuclei demonstrated diffuse and intense staining. A positive reaction for NeuN was observed in the nucleoli of the nuclei. In some neurons, both the nucleus and the cytoplasm were very weakly stained or not stained at all.

The nuclei of most pyramidal neurons in the third layer showed intense diffuse staining. Neurons with nuclei of low staining intensity were also observed. Cytoplasmic staining was low intensity and diffuse. Positive staining was detected in the initial segments of neuronal processes, including dendrites and axons.

Small nuclei were observed in the fourth cortical layer with more intense staining at the periphery. The hyaloplasm was uniformly stained with moderate intensity. In some neurons, the nuclei and cytoplasm were either unstained or showed minimal staining intensity.

In the fifth layer, the nuclei of many large pyramidal neurons showed variable staining intensity. In the cytoplasm, reaction products were represented by a small number of fine granules. Diffuse staining, occasionally combined with stained granules, pre-

dominated. In some large neurons, both nuclei and cytoplasm were either unstained or showed low intensity staining. In some neurons, the initial segments of dendrites and axons showed low intensity staining. The nuclei and cytoplasm of smaller neurons displayed variable staining intensities.

The sixth layer of polymorphic cells was characterized by variable staining of neuronal nuclei and cytoplasm. Numerous neuronal nuclei showed peripheral localization of NeuN, resulting in ring-like staining patterns along the nuclear periphery after immunohistochemical reaction. Many weakly stained or unstained neuronal nuclei were observed at the border with the white matter of the brain. Moderate or less intense staining was observed in the initial segments of neuronal axons and dendrites.

Based on the staining characteristics of NeuN in cortical neurons, the following phenotypes were identified (Fig. 1):

1) Neurons with stained nuclei but unstained cytoplasm;

2) Neurons with stained cytoplasm but unstained nuclei;

3) Neurons with stained nuclei and cytoplasm;

4) Fully stained neurons with no visible nuclei;

5) Neurons with stained processes (dendrites/axons).

The staining intensity of NeuN-positive neurons varied depending on the cortical layer (Fig. 2, Table). A comparative analysis of the cortical layers based on the mean NeuN staining intensity revealed differences shown in the Table.

Discussion

The cortical neuron phenotypes described above, based on the localization of IHC reactions with NeuN antibodies, have been sporadically men-

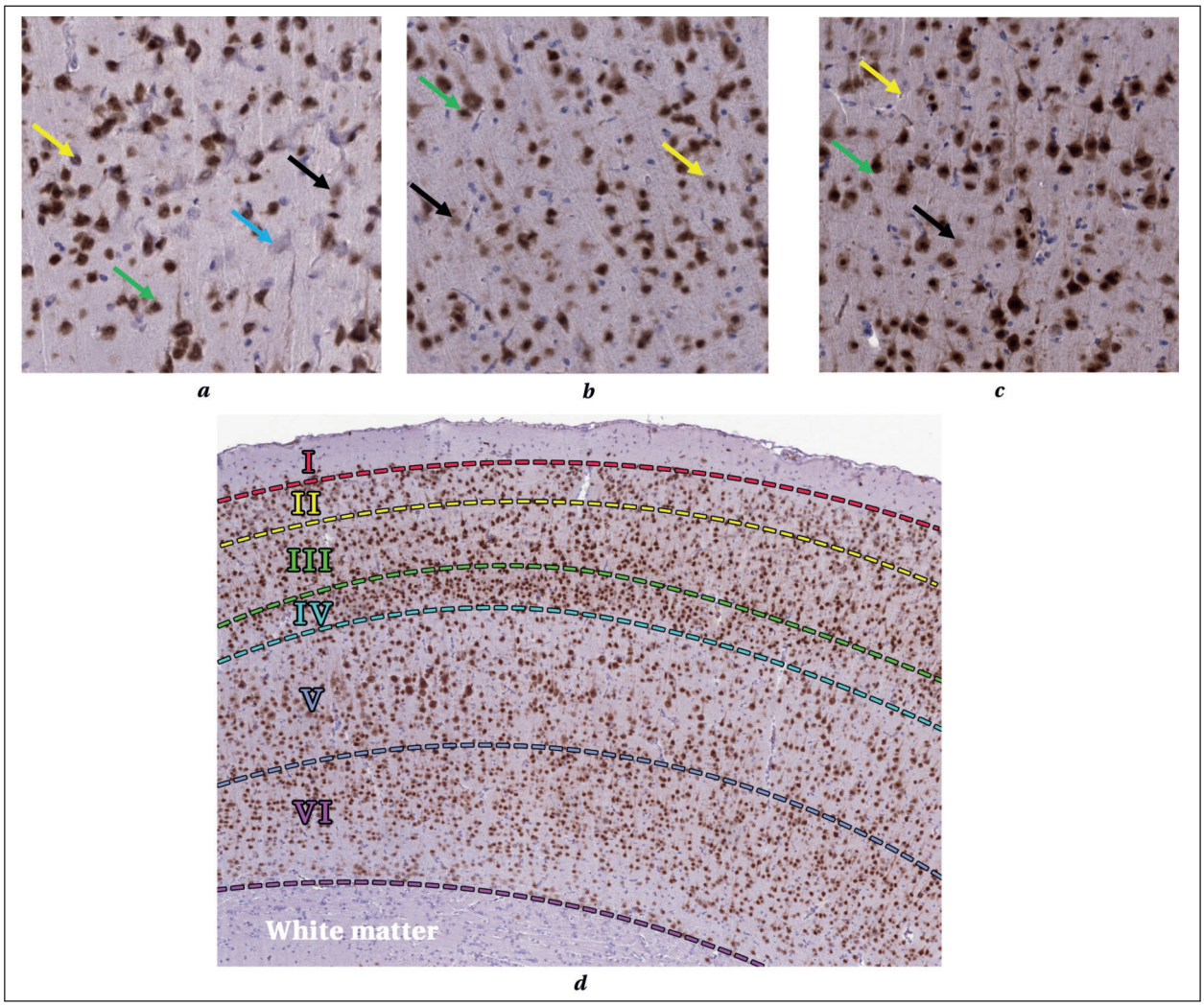

Fig. 1. Localization of NeuN in rat cerebral cortex neurons.
Note. Immunohistochemical (IHC) staining, magnification ×20. Panels *(a–c)* show different staining intensities ranging from weak to strong. *Green arrows*: Neurons with positive IHC staining in both nuclei and cytoplasm. *Yellow arrows*: Neurons with positive IHC staining in nuclei only. *Black arrows*: Neurons with positive IHC staining but without visible nuclear borders. *Blue arrows*: Unstained neurons. Panel *d*: colored lines indicate the boundaries between cortical layers, with Roman numerals indicating the corresponding layers.

tioned in previous studies. In particular, some attempts have been made to explain the variability in NeuN staining and some results have been obtained.

The most common phenotype identified in our study includes neurons with both nuclear and cytoplasmic staining. The neuronal nuclear protein NeuN is known to associate with DNA, and according to Y. S. Lin et al. IHC reactions for NeuN are predominantly observed in nuclear regions and to a lesser extent in the perinuclear cytoplasm [12]. This nuclear and cytoplasmic staining may be due to the subcellular localization of different NeuN/Rbfox3 subtypes. Specifically, the 46-kDa subtype is primarily localized in the nucleus, whereas the 48-kDa subtype is predominantly distributed in the cytoplasm [13]. In certain neurons, such as cerebellar granule cells, nuclear staining may be absent in afferent autonomic neurons while the cytoplasm shows a positive IHC reaction [14].

The varying intensity of NeuN staining and the presence of NeuN-negative neurons cannot be interpreted as an indication of neuronal damage alone, but may reflect differences in neuronal functional activity. It has been suggested that the intensity of NeuN staining in the nucleus is related to the chromatin state. Studies have shown that brain cell nuclei in mice expressing high levels of NeuN/FOX3 have decondensed chromatin compared to nuclei with weak or absent staining [15].

The presence of NeuN-negative neurons in the brain, independent of pathological factors, is supported by the findings of F. A. Azevedo et al. who demonstrated that the adult human brain contains 86.1±8.1 billion NeuN-positive cells («neurons») and 84.6±9.8 billion NeuN-negative cells [16].

Further evidence for a stable subpopulation of NeuN-negative neurons in the lateral neocortex was provided by M. L. Hernandez et al. who reported that these neurons may be more susceptible to late cell membrane damage [17].

In a study of substantia nigra neurons in intact laboratory animals (rats) not exposed to pathologic factors, significant variability in NeuN staining was found. This study identified not only NeuN-positive neurons, but also unstained neurons and neurons with weak NeuN staining, as well as different subcellular compartmentalization of NeuN. The authors concluded that morphometric assessment of NeuN expression cannot be reliably used as a neuronal marker in the substantia nigra [18].

A study by I. Unal-Cevik et al. examined NeuN in mouse brains 6 hours after cerebral ischemia, comparing immunohistochemical (IHC) and Western blot analyses. Their results showed that brain samples with no IHC reaction did not show reduced protein levels, suggesting that the decrease in NeuN IHC staining intensity was due to metabolic disturbances rather than neuronal damage or a reduction in neuronal number [19].

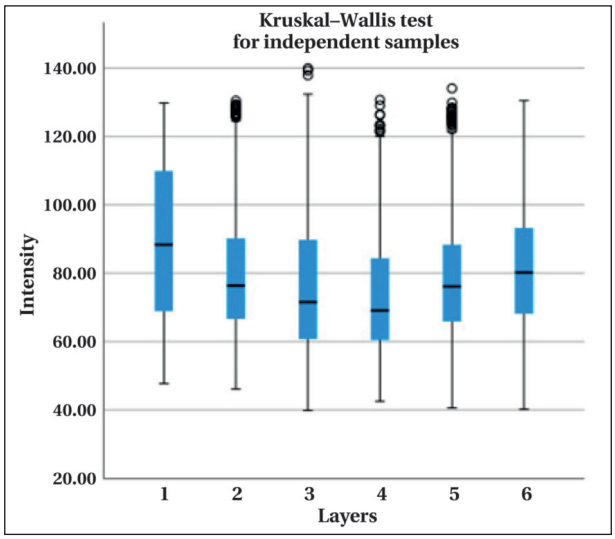

Fig. 2. Staining intensity values of NeuN-positive neurons in the layers of the rat cerebral cortex.

Table. Average staining intensity of NeuN-positive cells in different layers of rat cerebral cortex (median, *Me [Q1–Q3]*).

Layers compared	Average staining intensity		*P*-value
4–3	69 (60–84)	71 (60–89)	0.033*
4–5	69 (60–84)	76 (65–88)	<0.001*
4–2	69 (60–84)	76 (66–90)	<0.000*
4–6	69 (60–84)	68 (80–93)	<0.000*
4–1	69 (60–84)	88 (68–110)	<0.000*
3–5	71 (60–89)	76 (65–88)	<0.000*
3–2	71 (60–89)	76 (66–90)	<0.000*
3–6	71 (60–89)	68 (80–93)	<0.000*
3–1	71 (60–89)	88 (68–110)	<0.000*
5–2	76 (65–88)	76 (66–90)	1.000
5–6	76 (65–88)	68 (80–93)	<0.000*
5–1	76 (65–88)	88 (68–110)	<0.000*
2–6	76 (66–90)	68 (80–93)	0.015*
2–1	76 (66–90)	88 (68–110)	<0.001*
6–1	68 (80–93)	88 (68–110)	0.123

Note. * — significant differences.

Research has suggested that the number of NeuN-positive neurons may be influenced by age and sex [20–22]. For example, A. Sugiura et al. observed a decrease in NeuN-containing hypothalamic neurons with advancing age [22]. However, our examination of the cerebral cortex in COVID-19 non-survivors did not show any effect of sex or age on NeuN staining [11].

L. Luijerink et al. observed weaker IHC reactions in paraffin-embedded human brain sections compared to those from experimental animals. They attributed this difference to the methods of fixation and histological preparation [23]. However, other studies using experimental material have shown significant variability in neuronal staining regardless of the fixation technique employed [18].

Studies have examined NeuN expression in various pathological conditions and injuries. L. T. McPhail et al. found that transection of peripheral nerves in rats and mice resulted in almost complete loss of NeuN in facial motor neurons

within three days of injury. This depletion persisted for up to 28 days, although the neurons remained viable [24]. In cases of sudden perinatal death, NeuN staining was significantly reduced or absent. Notably, neurons with reduced NeuN labeling showed no evidence of apoptosis. Furthermore, a strong association between NeuN depletion in the fetal brain neurons and maternal smoking was observed [25].

In another study, Anderson et al. observed an increase in NeuN expression in the nuclei and cytoplasm of neurons in the dorsal root ganglia on an experimental model of adjuvant-induced arthritis [26]. This finding led the investigators to suggest that NeuN may be involved in the activation of nociceptive neurons and contribute to pain signaling.

Conclusion

Given the important biological role of the NeuN protein, researchers should closely examine the phenotypes of neurons characterized by different NeuN localizations, as these patterns may reflect the functional state of the neurons. The absence of NeuN staining should not be interpreted as evidence of neuronal damage, as there is no scientific justification for this assumption. The studies using immunohistochemical staining and NeuN antibodies should consider and analyze not only the total number of NeuN-positive neurons, but also their distinct phenotypic variations. Based on the results showing significant differences in NeuN staining intensity across cortical layers, future studies should explore the identified neuronal phenotypes as a potential criterion for personalized profiling of cortical layers in the brain.

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