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Patent Foramen Ovale as a Risk Factor for Neurological Decompression Sickness in Recreational Divers: A Narrative Review

Sylwia Pietrajtis, ORCID: https://orcid.org/0009-0009-0104-5896
E-mail: sylwia.pietrajtis@op.pl
Faculty of Medicine, Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland

Angelika Juszczyk, ORCID: https://orcid.org/0009-0007-2378-1363
E-mail: angelika.juszczyk15@gmail.com
St Alexander's Hospital in Kielce, ul. Kościuszki 25, 25-316 Kielce, Poland

Dominika Brożyna, ORCID: https://orcid.org/0009-0002-1542-6904
E-mail: lek.dominikabrozyna@gmail.com
Faculty of Medicine, Jan Kochanowski University in Kielce, al. IX Wieków Kielc 19a, 25-516 Kielce, Poland

Mateusz Wnęk, ORCID: https://orcid.org/0009-0008-1087-973X
E-mail: matweu876@gmail.com
Faculty of Medicine, Jan Kochanowski University in Kielce, al. IX Wieków Kielc 19a, 25-516 Kielce, Poland

Bartłomiej Sałapski, ORCID: https://orcid.org/0009-0008-8366-1272
E-mail: barteksalapski@gmail.com
Faculty of Medicine, Jan Kochanowski University in Kielce, al. IX Wieków Kielc 19a, 25-516 Kielce, Poland

Mateusz Kubicki, ORCID: https://orcid.org/0009-0005-5646-8109
E-mail: mateuszpkubicki@gmail.com
Faculty of Medicine, Jan Kochanowski University in Kielce, al. IX Wieków Kielc 19a, 25-516 Kielce, Poland

Wojciech Kukiełka, ORCID: https://orcid.org/0009-0005-3967-1383
E-mail: w.kukielka2000@gmail.com
Faculty of Medicine, Jan Kochanowski University in Kielce, al. IX Wieków Kielc 19a, 25-516 Kielce, Poland

Michał Łebek, ORCID: https://orcid.org/0009-0006-3331-5262
E-mail: mmichal.lebek@gmail.com
Faculty of Medicine, Jan Kochanowski University in Kielce, al. IX Wieków Kielc 19a, 25-516 Kielce, Poland

Wiktoria Komornik, ORCID: https://orcid.org/0009-0001-0403-0579
E-mail: w4955660@gmail.com
Faculty of Medicine, Medical University of Łódź, al. Kościuszki 4, 90–419 Łódź

Jakub Gałwiaczek, ORCID: https://orcid.org/0009-0005-0912-2594
E-mail: oggalwiaczek@gmail.com
Provincial Integrated Hospital, ul. Grunwaldzka 45, 25-736 Kielce, Poland

Abstract

Background. Decompression sickness (DCS) remains a relevant risk in recreational and professional diving. It results from inert gas (mainly nitrogen) uptake under increased pressure and subsequent bubble formation during ascent. These bubbles may affect tissues and circulation. Neurological DCS is the most severe form and may lead to permanent disability or death.

Patent foramen ovale (PFO), present in approximately 25% of adults, may enable right-to-left shunting and allow venous gas emboli to bypass pulmonary filtration. This mechanism is considered a potential contributor to paradoxical embolization and central nervous system involvement.

Aim. The aim of this review is to evaluate current evidence on the association between PFO and neurological DCS in recreational divers, with emphasis on diagnostic methods and clinical implications.

Materials and methods. A narrative review of English-language publications (2015-2026) was conducted using PubMed and selected peer-reviewed journals in diving and cardiovascular medicine. Original research articles, systematic reviews, meta-analyses, and guidelines were included.

Results. Available evidence suggests that PFO, particularly with large or high-grade right-to-left shunts, is associated with increased risk of neurological DCS. Paradoxical embolization is considered the main underlying mechanism. Diagnostic evaluation is based primarily on contrast echocardiography and transcranial Doppler.

Conclusions. PFO constitutes an important but not exclusive risk factor for neurological DCS. The functional characteristics of the shunt appear to influence this risk. Routine screening is not currently recommended. Targeted evaluation and individualized risk assessment should be considered. Further prospective studies are needed to clarify optimal screening strategies and indications for PFO closure in recreational divers.

Key words: patent foramen ovale, decompression sickness, neurological DCS, diving medicine, right-to-left shunt

Content

1. Introduction

Patent foramen ovale (PFO) is a remnant of fetal circulation resulting from incomplete postnatal closure of the foramen ovale. It is present in approximately 25% of the adult population worldwide (1, 2). In fetal life, the foramen ovale enables right-to-left shunting of blood, bypassing the pulmonary circulation. After birth, functional closure normally occurs due to increased left atrial pressure; however, in some individuals, the septum primum and septum secundum do not fuse, leaving a potential interatrial communication. Importantly, PFO is not considered a true atrial septal defect inasmuch as it does not involve a deficiency of septal tissue (3).

Although PFO is usually asymptomatic, it may permit right-to-left shunting under conditions of increased right atrial pressure. This mechanism enables paradoxical embolization, which has been associated with several clinical conditions, including cryptogenic stroke, migraine, platypnea–orthodeoxia syndrome, and decompression sickness (1,2).

Decompression sickness (DCS), together with arterial gas embolism (AGE), constitutes decompression illness (4). These disorders may occur during or after ascent in compressed-gas

diving due to reduced ambient pressure. According to Henry's law, the quantity of gas dissolved in tissues is proportional to its partial pressure; therefore, during ascent, inert gas (mainly nitrogen) may form bubbles if elimination is insufficient. In DCS, bubbles originate from dissolved inert gas within tissues, whereas in AGE they arise from pulmonary barotrauma and enter the arterial circulation directly (4). Neurological DCS represents one of the most severe manifestations, possibly causing stroke-like symptoms, spinal cord injury, paralysis, or death.

Given the high prevalence of PFO in the general population and its possible role in facilitating arterialization of venous gas emboli, PFO remains a clinically relevant risk factor for neurological DCS. Enhancing the understanding of this association may help to improve risk stratification and medical fitness assessment in recreational and professional divers.

2. Research materials and methods

A narrative literature review was conducted using PubMed and selected peer-reviewed journals in diving and cardiovascular medicine. Studies published in English between 2015 and 2026 were analyzed. The following keywords were used: "patent foramen ovale", "decompression sickness", "neurological decompression sickness", "diving", "right-to-left shunt", and "bubble embolism".

Original research articles, systematic reviews, meta-analyses, and consensus guidelines were included. Studies were selected based on relevance, methodological quality, and clinical relevance for diving medicine.

3. Research results

3.1. Pathophysiology of Decompression Sickness and PFO

Bubbles form in tissues or in the venous circulation when, during ascent, the partial pressure of dissolved gas exceeds ambient pressure, resulting in tissue supersaturation. The depth and duration of the dive directly influence inert gas uptake and, consequently, bubble creation.

The resulting gas emboli, most frequently ranging from 19 to 700 μm in diameter, are usually filtered by the pulmonary capillaries. However, in some cases, they may reach the arterial circulation. One possible mechanism is a patent foramen ovale, which allows a right-to-left shunt (5). Although PFO is usually functionally closed due to higher left atrial pressure,

transient increases in right atrial pressure (e.g., during Valsalva maneuvers, coughing, or straining) may result in temporary right-to-left shunting.

A right-to-left shunt allows venous gas emboli to bypass the pulmonary filtration system and enter the systemic circulation, possibly resulting in paradoxical embolization to the central nervous system.

3.2 Epidemiology

As previously stated, PFO occurs in approximately 25% of the adult population worldwide (1). Although it is difficult to estimate the exact percentage of divers with PFO because there are no routine examinations, studies show that, in certain populations, e.g., breath-hold divers, the prevalence is relatively high. The results of a 2022 study show that apnea divers had a significantly higher prevalence of patent foramen ovale (19 of 36, 53%) compared to controls (9 of 36, 25%). The higher prevalence of PFO in apnea divers remains unexplained. Hyperbaric exposure during breath-hold diving redistributes central blood volume and reduces lung volume, while ascent-related alveolar hypoxia induces hypoxic pulmonary vasoconstriction. The resulting increase in pulmonary and right atrial pressure may promote right-to-left shunting through a patent foramen ovale, potentially helping to preserve cardiac output and reduce capillary hydrostatic stress (7).

The presence of a PFO significantly increases the risk of neurological DCS in divers. Cohort and case-control studies indicate that the relative risk of developing neurological DCS in individuals with a PFO is approximately 5 to 13 times higher compared with divers without this cardiac anomaly. The greatest risk is observed in divers with large or high-grade PFO, particularly in cases of unprovoked DCS, in which symptoms occur despite dives within accepted safety limits (8).

According to recent studies, the relative risk of neurological DCS in divers with a PFO is approximately 3–5 times higher than in divers without a PFO, with the greatest risk observed in individuals with a large shunt. In a prospective study by Germonpré et al. (2021), the presence of PFO or another right-to-left shunt was shown to increase the risk of DCS (RR = 3.02 for confirmed cases). Importantly, this risk was strongly associated with diving, particularly dives performed close to the limits of established decompression procedures (9).

In a large cohort study, Honěk et al. (2022) confirmed that high-grade PFO is a major risk factor for unprovoked DCS, particularly its neurological form. The prevalence of PFO in this group

reached 97.2%, compared with 35.5% in the control group. In multivariate analysis, grade 3 PFO emerged as the strongest predictor of unprovoked DCS (10).

Recent data from 2024 provide evidence that the presence of a PFO increases the risk of DCS by approximately fivefold, while echocardiographic features such as a high-grade shunt and atrial septal mobility further elevate the risk of neurological DCS (11).

To summarize, recent cohort and case–control studies consistently indicate that the presence of PFO significantly increases the relative risk of neurological decompression sickness in divers, and that this risk rises with the size and functional significance of the shunt.

3.3 Clinical Presentation

Neurological decompression sickness may present with a wide range of symptoms, including sensory disturbances (such as non-dermatomal hypoesthesia and paresthesias), limb weakness or paresis, ataxia, impaired coordination, and bladder or sphincter dysfunction. Other reported manifestations include girdle-type pain, cognitive impairment, visual disturbances, dizziness, headache, and speech difficulties. In severe cases, seizures or loss of consciousness may occur. Symptoms typically develop within minutes to several hours after surfacing, most commonly within the first six hours (5).

Neurological symptoms and a marked decline in cognitive performance, particularly involving impaired attention, have been described in individuals who have experienced DCS. The prevalence of neurological DCS associated with cognitive and functional impairment ranges from approximately 30% in compressed-air divers to up to 56% among commercial breath-hold divers.

Structural abnormalities affecting both the gray and white matter have been reported not only in symptomatic divers but also in asymptomatic divers and high-altitude pilots. Radiological studies have identified lesions in multiple regions of the central nervous system, including the spinal cord, cerebral cortex, basal ganglia, brainstem, and cerebellum. In some cases, these lesions are assumed to result from arterialized gas bubbles, which may lead to gas embolism (12).

Within the concept of DCS, two main forms can be distinguished. The spinal form is characterized by symptoms related to spinal cord injury. These include limb weakness or paralysis (most commonly of the lower extremities), sensory disturbances, truncal ataxia, and bladder dysfunction, particularly urinary retention. Sphincter dysfunction, girdle-like pain, and

pyramidal signs may also be present. Paraparesis or paraplegia is frequently observed, and the neurological deficits may be asymmetric.

The cerebral form of DCS manifests with central nervous system symptoms. These may include cognitive disturbances (such as difficulties with concentration and memory), visual disturbances (including scotomas and visual field defects), focal motor deficits, ataxia, dizziness, headache, speech disturbances, seizures, and loss of consciousness.

In MRI, typical findings include lesions in the corpus callosum and focal cerebral edema (5).

Differentiation between neurological DCS and arterial gas embolism (AGE) primarily focuses on the timing of symptom onset and the clinical presentation.

AGE typically develops very rapidly, usually during ascent or within minutes after surfacing, and often presents with sudden loss of consciousness, seizures, and focal neurological deficits such as hemiplegia or aphasia. These manifestations may resemble an acute stroke and can be accompanied by pulmonary barotrauma, including chest pain, dyspnea, or hemoptysis (13).

In contrast, neurological DCS generally develops more gradually, most often within 10–60 minutes after surfacing, although symptoms may appear up to several hours later. Clinical manifestations are often less abrupt and may include multifocal neurological deficits, with spinal cord involvement and truncal ataxia being particularly characteristic. Sudden loss of consciousness or seizures are less common in DCS than in AGE (14).

In clinical practice however, distinguishing between these two conditions can be difficult because their symptoms may overlap, and both fall within the broader spectrum of DCI. Importantly, initial management is the same for both conditions: urgent oxygen administration and recompression therapy (15).

3.4 Diagnostic approach

The diagnosis of neurological decompression sickness is primarily clinical, based on the history of recent exposure to pressure changes (e.g., diving) and the presence of compatible neurological symptoms. Imaging and additional tests are primarily used to support the diagnosis, identify complications, and detect predisposing factors such as a right-to-left shunt, most commonly caused by PFO.

3.4.1. Transthoracic Echocardiography with Contrast (cTTE)

Contrast-enhanced transthoracic echocardiography (cTTE) is commonly used as a first-line screening test for detecting a right-to-left shunt. The test involves intravenous injection of agitated saline ("bubble test"), allowing visualization of microbubbles passing from the right to the left atrium. Detection of bubbles in the left atrium within several cardiac cycles suggests the presence of a PFO or another intracardiac shunt (16).

Although cTTE is widely available and non-invasive, its sensitivity may be lower than that of other techniques, especially for detecting small or intermittent shunts (17).

3.4.2. Transesophageal Echocardiography (TEE)

Transesophageal echocardiography (TEE) is considered the reference standard for anatomical visualization of PFO.

It allows direct visualization of the interatrial septum, assessment of shunt morphology, and evaluation of features associated with a higher risk of paradoxical embolism, such as a large shunt, atrial septal aneurysm, or increased septal mobility.

However, TEE is more invasive and usually reserved for confirmatory evaluation or pre-procedural assessment, particularly when closure of PFO is being considered (18).

3.4.3. Transcranial Doppler Ultrasonography (TCD)

Transcranial Doppler (TCD) with contrast is a highly sensitive non-invasive method used to detect right-to-left shunts by identifying microbubbles entering the cerebral circulation after intravenous injection of agitated saline.

TCD is particularly useful for screening divers, as it can detect even small shunts and grade shunt severity based on the number of detected microbubble signals. Studies have demonstrated that divers with high-grade shunts detected by TCD have a significantly increased risk of neurological DCS (18, 19).

3.4.4. Bubble Test

The bubble test, performed during echocardiography or TCD, involves injecting agitated saline containing microbubbles while the patient performs a Valsalva maneuver, which

transiently increases right atrial pressure and promotes the passage of bubbles through a potential shunt. The appearance of microbubbles in the left atrium or cerebral circulation confirms the presence of a right-to-left shunt (18).

3.4.5. The importance of Detecting a Large Shunt

Identifying the presence and magnitude of a right-to-left shunt is clinically important because the risk of neurological decompression sickness increases with shunt size. Divers with large or high-grade PFOs have a substantially higher risk of neurological DCS and may require risk-reduction strategies, such as conservative diving profiles or consideration of PFO closure in selected cases (20).

The diagnostic methods for DCS can be summarized in the table below.

Table 1. DCS diagnostic methods, along with their advantages and limitations.

Method	**Principle**	**Advantages**	**Limitations**	**Role in Divers with Suspected PFO**
Contrast Transthoracic Echocardiography (cTTE)	Intravenous injection of agitated saline allows visualization of microbubbles passing from the right to the left atrium through a shunt	Non-invasive, widely available, relatively inexpensive	Lower sensitivity for small or intermittent shunts; image quality depends on acoustic window	Common first-line screening test for detecting right-to-left shunt
Transesophageal Echocardiography (TEE)	Ultrasound probe placed in the esophagus enables direct visualization of the interatrial septum and PFO morphology	High spatial resolution; allows assessment of PFO anatomy, shunt size, and associated	Semi-invasive, requires sedation, less suitable for screening	Reference method for anatomical confirmation of PFO and pre-closure evaluation

		features (e.g., atrial septal aneurysm)		
Transcranial Doppler with Contrast (cTCD)	Detection of microbubbles in the cerebral circulation after intravenous contrast injection, indicating right-to-left shunt	Very high sensitivity for detecting shunts; non-invasive; useful for shunt grading	Does not provide anatomical localization of the shunt	Effective screening tool in divers, especially for identifying high-grade shunts
Bubble Test	Agitated saline contrast injected during echocardiography or TCD, often combined with Valsalva maneuver to provoke shunt flow	Simple method that improves detection of right-to-left shunts	Requires correct performance of Valsalva maneuver; may miss very small shunts	Standard provocation technique used during cTTE, TEE, or cTCD
Assessment of Shunt Size	Quantification of microbubble passage or visualization of septal separation	Helps stratify risk of paradoxical embolism and neurological DCS	Grading systems may vary between techniques	Large (high-grade) shunts are associated with higher risk of neurological DCS

3.5 Management and prevention

The management of acute DCS includes the immediate administration of 100% oxygen, immobilization, adequate fluid intake, monitoring of vital signs, and urgent referral for hyperbaric therapy (recompression), which remains the only causal treatment.

Prompt initiation of treatment is recommended, as delays are associated with poorer neurological outcomes. In cases of severe neurological symptoms or arterial gas embolism, treatment should be carried out in a specialized center (21, 22).

Regarding PFO screening and risk stratification, current guidelines do not recommend routine screening for PFO in recreational divers or in most professional divers (21, 22, 24). Screening may be justified in divers with recurrent or severe neurological decompression sickness, particularly when a high-grade shunt is suspected.

In SCUBA divers with prior DCI and without a prior PFO-associated stroke, the SCAI guideline panel suggests against the routine use of PFO closure to prevent DCI (21). Risk stratification is based on the assessment of shunt severity (e.g., using transcranial Doppler or transcranial color-coded sonography and transesophageal echocardiography), as well as on echocardiographic features such as high atrial septal mobility, atrial septal aneurysm, or a large degree of patency. Additional considerations include the diver's profile, for example, professional or tactical divers, and individuals with a positive family history.

Regarding indications and controversies for PFO closure, guidelines from the Society for Cardiovascular Angiography and Interventions (SCAI) and international consensus statements recommend against routine PFO closure after a first episode of DCS. The procedure may be considered in individuals with recurrent or severe neurological DCS, specifically in cases of a high-grade and in professional divers with high occupational risk.

Although PFO closure reduces the risk of recurrent DCS, it does not completely eliminate the risk, and the procedure may be associated with complications such as atrial fibrillation or bleeding. (8, 10, 11, 21, 22).

Several controversies remain concerning the issue, including the lack of randomized controlled trials, the possibility of a residual shunt after closure, and the need for individualized risk–benefit assessment. Therefore, decisions regarding PFO closure should be based on a shared decision-making process between the clinician and the patient.

As for the recommendations from diving organizations and professional societies, the SCAI guidelines and international consensus statements emphasize that the management strategy should be individualized. Recommended approaches include adopting conservative diving protocols, such as limiting dive depth and duration and avoiding decompression dives, as well as considering cessation of diving or PFO closure in selected cases (10, 21-22).

Particular attention is given to professional and tactical divers, in whom the acceptable level of risk is lower due to occupational and operational safety considerations.

After an episode of DCS, divers are advised to adopt more conservative diving practices, consider cessation of diving, or - after PFO closure - return to diving only if no residual shunt is detected and an appropriate risk assessment has been performed (10-11, 21-24).

In older individuals and those with comorbidities, a comprehensive cardiovascular risk stratification is recommended before resuming diving (22). In clinical practice, risk classification systems and fitness-to-dive assessments are commonly used to determine whether a diver can safely return to diving activities.

At present, there is a lack of randomized controlled trials and concise guidelines regarding the optimal strategy for PFO screening and closure. Therefore, decisions should be made on an individual basis, taking into account the diver's risk profile, the characteristics of the PFO, and the diver's preferences.

4. Discussion

4.1. Interpretation of Epidemiological Data

The available epidemiological evidence consistently indicates that PFO is an important risk factor for neurological DCS, particularly in recreational divers exposed to decompression stress. Cohort and case–control studies present a markedly higher prevalence of PFO among divers who develop neurological DCS compared with unaffected controls, as well as elevated relative risk estimates. However, the absolute incidence of neurological DCS remains low even among individuals with PFO, suggesting that PFO alone is not sufficient to cause the disease. Diving behavior, exposure intensity, ascent rate, repetitive dives, dehydration, and individual vulnerability likely interact with the presence of a right-to-left shunt to determine clinical outcome. Variability in study design, diagnostic criteria, and methods for detecting shunts complicates direct comparisons between studies.

4.2. Does every case of PFO increase the risk of neurological DCS?

Not all PFOs pose the same clinical risk. Given that PFO is present in approximately 25% of the general population while only a small proportion of divers develop neurological DCS, the presence of a PFO alone cannot be considered determinative. Small or functionally insignificant shunts may pose little to no additional risk, particularly for divers following conservative dive profiles. Conversely, PFOs that open readily under physiological stress, such as during Valsalva maneuvers performed while equalizing pressure, may facilitate paradoxical

embolization of venous gas bubbles. Therefore, functional characteristics of the shunt are likely more important than simple anatomical presence.

4.3. Importance of shunt size

The magnitude of the right-to-left shunt appears to be the most important predictor of neurological DCS among individuals with PFO. Large or high-grade PFOs allow a greater volume of venous gas emboli to bypass pulmonary filtration and enter the arterial circulation, heightening the likelihood of embolization to the brain or spinal cord. Multiple studies have shown that divers with high-grade shunts are disproportionately represented among cases of unprovoked or severe neurological DCS. Echocardiographic features such as atrial septal aneurysm, increased septal mobility, and large patency further intensify the risk. Consequently, quantitative assessment of shunt severity plays a central role in clinical risk stratification and decision-making regarding preventive strategies.

4.4. Should all divers be screened?

Routine screening of all divers for PFO is not currently recommended by most professional societies. Inasmuch as PFO is common, whereas neurological DCS is relatively rare, universal screening would identify many individuals who may never experience decompression illness, possibly causing unwarranted anxiety, restrictions, or invasive procedures. Moreover, there is insufficient evidence that screening asymptomatic divers improves outcomes. Targeted screening appears more appropriate, particularly in divers with unexplained, recurrent, or severe neurological DCS, or in those whose professional duties involve high exposure to decompression stress.

4.5. Risk–benefit balance of PFO closure

Percutaneous PFO closure has been shown to reduce the recurrence of neurological DCS in selected individuals, especially those with large shunts and prior events. Nevertheless, the procedure is not without risk. Potential complications include atrial fibrillation, device-related thrombosis, vascular complications, and bleeding associated with antithrombotic therapy. Importantly, closure does not eliminate the risk of decompression illness entirely, as alternative mechanisms, such as pulmonary shunting or in situ bubble formation, may still occur. Therefore, closure should be considered only after careful individualized assessment,

particularly in divers who wish to continue high-risk diving activities or whose occupation depends on diving.

5. Limitations of the Review

This review has several limitations. The available evidence is derived predominantly from observational studies, retrospective analyses, and mixed populations, with a lack of randomized controlled trials specifically addressing divers with PFO. Differences in diagnostic techniques, shunt grading systems, and outcome definitions limit comparability across studies. Publication bias and underreporting of mild or asymptomatic cases may similarly influence the perceived strength of associations. Furthermore, rapid progress in imaging techniques and changes in diving practices may affect the applicability of older data to current recreational diving populations.

6. Conclusions

Patent foramen ovale constitutes a significant risk factor for neurological decompression sickness in divers, particularly when other predisposing conditions are present. The risk is not uniform across all individuals with PFO and appears to depend primarily on the functional characteristics of the shunt.

The most clinically relevant determinant is the presence of a large right-to-left shunt, which facilitates the passage of venous gas emboli into the arterial circulation and increases the likelihood of central nervous system involvement.

Diagnostic evaluation for PFO in divers should therefore be selective rather than routine, focusing on individuals with unexplained, recurrent, or severe neurological DCS or those with high exposure to decompression stress.

Decisions regarding percutaneous PFO closure entail careful individualization, taking into account shunt size, clinical history, diving profile, occupational considerations, procedural risks, and the diver's preferences.

Disclosure.

Authors' contribution:

Conceptualization: Sylwia Pietrajtis, Angelika Juszczyk

Methodology: Sylwia Pietrajtis, Angelika Juszczyk, Dominika Brożyna, Mateusz Wnęk, Bartłomiej Sałapski, Mateusz Kubicki, Wojciech Kukiełka, Michał Łebek, Wiktoria Komornik, Jakub Gałwiaczek

Formal Analysis: Dominika Brożyna, Bartłomiej Sałapski

Investigation: Mateusz Kubicki, Wojciech Kukiełka, Michał Łebek

Writing-rough preparation: Sylwia Pietrajtis, Angelika Juszczyk, Dominika Brożyna, Mateusz Wnęk, Bartłomiej Sałapski, Mateusz Kubicki, Wojciech Kukiełka, Michał Łebek, Wiktoria Komornik, Jakub Gałwiaczek

Writing-review and edition: Sylwia Pietrajtis, Angelika Juszczyk, Dominika Brożyna, Mateusz Wnęk, Bartłomiej Sałapski, Mateusz Kubicki, Wojciech Kukiełka, Michał Łebek, Wiktoria Komornik, Jakub Gałwiaczek

Visualization: Mateusz Kubicki, Wojciech Kukiełka

Supervision: Sylwia Pietrajtis, Wiktoria Komornik, Mateusz Wnęk

Project Administration: Angelika Juszczyk, Jakub Gałwiaczek

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