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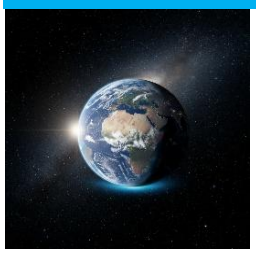

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Anabolic androgenic steroids impact on cardiovascular risk and cardiovascular events. The literature review

Eryk Ubysz
Provincial Polyclinical Hospital in Płock of Marcina Kacprzaka
Medyczna 19, 09 – 400 Płock, Poland
https://orcid.org/0009-0004-9099-7648
eryk.ubysz123@gmail.com

Patrycja Kwitowska
Provincial Hospital in Poznań
Juraszów 7/19, 60-479 Poznań, Poland
https://orcid.org/0009-0006-7297-2871
patrycjakwitowska@gmail.com

Łukasz Muraszewski
University Clinical Hospital in Poznań
Przybyszewskiego 49, 60-355 Poznań, Poland
https://orcid.org/0009-0000-0331-9701
lukaszmuraszewski@outlook.com

Emilia Muraszewska
University Clinical Hospital in Poznań
Przybyszewskiego 49, 60-355 Poznań, Poland
https://orcid.org/0009-0005-4534-1014
muraszewskaemilia@gmail.com

Małgorzata Pyjecka
Provincial Hospital of Ludwik Perzyna in Kalisz
Poznańska 79, 62-800 Kalisz, Poland
https://orcid.org/0009-0002-5061-6359
malgosia.pyjecka@gmail.com

Agata Król
Masovian Bródno Hospital
Kondratowicza 8, 03-242 Warsaw, Poland
https://orcid.org/0009-0001-3461-4786
akrol3088@gmail.com

Edyta Lewandowska
Central Clinical Hospital, University Clinical Center of the Medical University of Warsaw
Banacha 1A, 02-097 Warsaw, Poland
https://orcid.org/0009-0006-1181-727X
e.a.b.lewandowska@gmail.com

Cezary Łuczyński
Multispecialist Provincial Hospital in Gorzów Wielkopolski
Jana Dekerta 1, 66-400 Gorzów Wielkopolski, Poland
https://orcid.org/0009-0002-5128-4712

Maciej Paczkowski
Specialist Hospital Dr. Tytus Chałubiński Lekarska 4, 26-610 Radom, Poland
https://orcid.org/0009-0001-7670-1589
mpaczkowskitbg@gmail.com

Mariusz Wręczycki
St Hedwig of Silesia Hospital in Trzebnica
Prusicka 53/55, 55-100 Trzebnica, Poland
https://orcid.org/0009-0002-1945-4259
mariuszwreczycki85@gmail.com

Corresponding author:
Eryk Ubysz, eryk.ubysz123@gmail.com

Abstract
Background: Anabolic-androgenic steroids are used as a form of doping. Scientific research shows the association of AAS use with multidirectional effects on the cardiovascular system.
Aim: To collect overall information on the effects of AAS on the cardiovascular system based on current scientific studies.
Material and methods: The literature available in PubMed and Google Scholar databases was reviewed using the keywords.
Results: The results of scientific studies have confirmed the effects of AAS on vascular calcification and the development of atherosclerosis. Studies suggest an increased risk of thromboembolic incidents (lack of general consensus). AAS use is associated with the development of hypertension and left ventricular hypertrophy. Their consequences include impaired cardiac systolic and diastolic function, arrhythmias and susceptibility to ischemia. The use of AAS carries the risk of arrhythmias of ventricular and supraventricular origin. AAS increase the risk of myocardial infarction both in the mechanism of coronary artery stenosis and without significant stenosis.
Conclusions: AAS use is associated with increased cardiovascular risk and cardiovascular events. A collective knowledge of the effects of AAS abuse may be useful in the diagnostic and therapeutic process, as well as to develop methods for primary prevention. Some of the effects of AAS use require further research.

Key words: anabolic androgenic steroids, cardiovascular risk, cardiovascular diseases, drug abuse,

1. Introduction and epidemiology: Substances with anabolic effects include testosterone derivatives, growth hormone, and beta-2-sympathomimetics. By introducing modifications to the testosterone molecule, attempts have been made to increase the anabolic effect and minimize the androgenic effect. These substances include Nandrolone, Stanozolol, Oxandrolone, Methandienone, Methandrostenolone, and Trenbolone. The resulting active substances have met expectations to a very limited extent. The side effects of anabolic-androgenic steroids are responsible for their androgenic effect.

Anabolic-androgenic steroids are a group of steroids of natural or synthetic origin with a strong anabolic effect consisting in the acceleration of cell division. The anabolic effects of AAS use include increased protein synthesis, an accelerated rate of gain in muscle mass, strength, and endurance, increased appetite, an accelerated rate of bone growth, and increased production of red blood cells [**1**][**2**]. Adverse effects associated with androgenic effects include accelerated aging of certain tissues (especially bones), unnatural breast growth in men and women, sexual problems such as testicular atrophy, impotence, azoospermia, and infertility in men [**3**][**4**][**5**],

as well as voice changes, amenorrhea, uterine atrophy, and clitoral enlargement in women [**6**], and abnormal secretion of endogenous hormones in both sexes. AAS increase the secretion of sebaceous glands, which can cause acne vulgaris, androgenetic alopecia, and excessive hair growth [**4**]. Long-term use of AAS affects mental status, potentially causing headaches, irritability, depression, and AAS addiction syndrome [**7**], and may even lead to suicide [**4**].

Anabolic-androgenic steroids have found numerous medical applications since 1940. AAS are used for recovery from burns, injuries, and surgeries, as well as for conditions associated with aging, such as hypogonadism and osteoporosis. High doses of AAS can treat cachexia, especially that caused by HIV or neoplasms. Numerous scientific studies have shown that androgens can be used to treat leukemia, as well as aplastic anemia, by stimulating bone marrow proliferation and the hematopoietic process. Taking advantage of the fact that AAS stimulate GH synthesis, thereby promoting growth, they can be used to treat diseases that cause short stature (such as Turner's Syndrome). Medical research has also shown that AAS can be used to treat breast cancer [**2**].

AAS are used by athletes as a form of doping, especially in strength sports. Motivations for using AAS include improved athletic performance, muscle gain, better physical appearance, increased libido, and increased self-confidence [**8**]. This is especially true in bodybuilding, weightlifting, and athletic competitions such as running, long jump, cycling, and swimming. AAS abuse and the health effects associated with it are a challenge for scientists. A thorough understanding of the consequences of AAS use is important information for the clinician in the context of the diagnostic and therapeutic process, as well as for AAS users, whose education can contribute to the abandonment of AAS abuse and, consequently, the protection of their own health. One of the most important effects of AAS abuse is the impact of these substances on the cardiovascular system. Numerous scientific publications have increasingly shown a strong link between AAS and increased cardiovascular risk [**9**]. In our review, we attempt to integrate multiple studies and scientific publications in a way that facilitates understanding of the cardiovascular impact of AAS abuse.

Epidemiology The prevalence of AAS abuse has been steadily increasing over the past several years [**10**]. The epidemiology of the abuse of these agents remains important information for healthcare providers, especially in the context of the diagnostic and therapeutic process of a patient with current cardiovascular disease or a history of AAS abuse. According to a meta-analysis of 187 studies, being an athletic male has been shown to be a significant predictor of

AAS abuse [**11**]. According to the 2018 National Institute on Drug Abuse (NIDA), AAS misuse is most prevalent among male weightlifters between the ages of 20 and 30. Research indicates a global lifetime prevalence rate of 3.3% among athletes (6.4% in males and 1.6% in females) [11], highlighting non-medical steroid use as a substantial public health concern. Additionally, 13% of AAS users engage in unsafe practices, including the sharing and reuse of needles or vials [**12**].

1. Research materials and methods: The literature available in PubMed and Google Scholar databases was reviewed using the keywords.

2. Research results:

2.1 Vascular calcification: One primary cardiovascular concern regarding AAS use is its effect on vascular calcification. Given that testosterone contributes to the higher prevalence of cardiovascular disease in males, researchers in 2016 hypothesized that exogenous androgens might promote calcification. Immunohistochemical evidence confirmed androgen receptor (AR) expression within the tunica media of calcified human femoral arteries and heart valves.

Additionally, *in vitro* models showed that testosterone and DHT exposure accelerated calcification in murine vascular smooth muscle cells over a 9-day period [**13**]. This suggests that AR-mediated signaling is a driver of calcification and a possible target for intervention. Notably, vascular calcification is a hallmark of atherosclerosis and chronic kidney disease, predisposing patients to major adverse cardiovascular events [**14**].

A study of athletes using AAS, compared to non-users and those who had stopped using these agents, was conducted, taking into account both sexes as well as the duration of use. One of the parameters studied was the level of coronary artery calcification as determined by angio-CT. The results showed that longer cumulative lifetime AAS use was associated with a greater likelihood of coronary artery calcification and the presence of non-calcified plaques in coronary arteries. AAS use exceeding five years was associated with greater severity of calcification [**15**].

Another independent study showed a close association between long-term AAS intake and premature calcification in coronary arteries [**16**]. The effect of testosterone on the calcification

process of advanced atherosclerotic lesions in the arterial tree of male and female apolipoprotein E-null mice was also analyzed. Testosterone increased calcification 3- to 4-fold in lesions of the *arteria anonyma* and aortic sinus. This effect was independent of sex [**17**]. The study showed that calcification of advanced atherosclerotic lesions is an androgen-sensitive process.

2.2 Atherosclerosis: atherosclerosis is another AAS-dependent process. AAS use has a negative effect on the lipid profile of users, causing an increase in the LDL fraction and a decrease in the HDL fraction. The negative effects of AAS on the lipid profile have been confirmed by numerous studies [**18**][**19**][**20**]. In addition, strength training alone has been shown to have no effect on the lipid profile [21]. It is noteworthy that the effect of AAS is reversible, and the values of cholesterol fractions normalize within about five months after AAS cessation [**21**].

In 2017, a study was conducted in which AAS use was shown to be associated with coronary atherosclerosis. The study included men, 21 of whom were involved in weightlifting and had taken AAS for at least two years, 20 who also lifted weights but did not take steroids, and 10 who were healthy and had a sedentary lifestyle. The participants underwent angio-CT of the coronary arteries. The study showed that 24% of AAS users had coronary artery atherosclerosis, while none of the people in the other two groups (non-users) did; in addition, AAS users with atherosclerosis also had significantly reduced HDL cholesterol levels and reduced HDL function [**22**].

A cross-sectional cohort study published in 2017 examined 140 athletes reporting ≥2 years of cumulative lifetime AAS use and 54 non-users. Study participants were evaluated for coronary artery atherosclerotic changes, among other factors. AAS users showed greater coronary artery plaque volume compared to non-users of AAS. Lifetime AAS dose was strongly associated with the burden of coronary atherosclerosis [**23**]. Some studies have shown significant differences in the effect on HDL cholesterol depending on the type of AAS; for example, 17-alpha-alkylated testosterone (e.g., Methadienone) resulted in lower HDL levels, while another type of AAS (e.g., Nandrolone) had no effect on HDL cholesterol [**21**].

2.3 Thromboembolic processes: Numerous scientific studies indicate that AAS have a prothrombotic effect on the hemostatic system, thereby increasing the risk of thromboembolic incidents. In 2018, a study of the coagulation system in active AAS users, former AAS users, and a control group of non-users **showed that endogenous** thrombin potential (an indicator of coagulation activity) increased by about 15% in both active and former AAS users compared to non-users. Prothrombin and factor X, on the other hand, increased by about 10% [**24**]. The 2023 HAARLEM study focused on the effect of AAS use on the risk of venous thromboembolism. The study involved 100 athletes who voluntarily started a cycle of AAS, in whom levels of proteins related to clotting and fibrinolysis were measured. The participants used AAS for an average of 13 months (with an average weekly dose of 901 mg). The average levels of many coagulation factors (FII, FIX, and FXI) increased during use compared to the values measured before AAS inclusion, while the levels of FVIII and von Willebrand factor remained unchanged. The largest increases were observed in protein S (by 22%) and D-dimers (1.3-fold). Fibrin cleavage time (CLT) was eight minutes longer [**25**].

Oral AAS users had higher mean levels of FII, FIX, and FXI, as well as longer clot formation times (CFTs) and CLTs, than intramuscular AAS users. Three months after AAS discontinuation, all coagulation parameters were restored to baseline and remained unchanged one year after the discontinuation of these agents. The results of this study did not confirm a clear procoagulant state.

The unclear effect of AAS on thromboembolic processes is also indicated by the results of a study performed in June 2024, in which 37 AAS-using athletes were compared with 17 non-AAS athletes. Platelet aggregation and coagulation parameters were evaluated. There were no significant differences in platelet aggregation between the two groups. Von Willebrand factor was lower among AAS users and P-selectin was slightly higher, while CD40 ligand, β-thromboglobulin, and thrombospondin were not significantly different. There were no differences in the coagulation inhibitors evaluated. AAS users had higher D-dimer levels and lower PAI-1 activity, indicative of increased procoagulant and fibrinolytic activity. These findings suggest that the prothrombotic effects of long-term AAS use may predominantly involve aspects of the hemostatic system other than platelets [**26**]. Despite findings confirming increased activation of coagulation and fibrinolysis in AAS users, the effect of AAS on

thromboembolic incidents cannot be conclusively confirmed. More studies are needed to demonstrate the existence of this relationship, or the lack thereof.

2.4 Arterial hypertension: AAS can raise blood pressure. Numerous studies indicate that AAS use is related to increases in blood pressure. According to one study, the increase was primarily in systolic blood pressure (SBP) in a 24-hour measurement (involving both active and former AAS users). The mechanism involved an increase in aortic stiffness and a decrease in MR-proANP (a natriuretic peptide that lowers blood pressure naturally) in both active AAS users and those who had stopped using AAS. Elevated levels of aldosterone and norepinephrine have also been detected in active AAS users, which may also play a role in the rise in blood pressure in this group [**27**].

In the context of increased blood pressure, the effect of AAS on the kidneys is very important. Research indicates that AAS can cause or exacerbate acute kidney injury and chronic kidney disease, and can have toxic effects on the glomeruli. These effects are related to the enhancement of the renin-angiotensin-aldosterone system (RAAS), increased synthesis of endothelin, reactive oxygen radicals, mediators of fibrosis and apoptosis (TGF-β1), and mediators of inflammation (TNF-α, IL-1b, IL-6) [**28**]. A noteworthy effect of AAS is their impact on arterial wall stiffness (a marker of cardiovascular risk) as measured by pulse wave velocity (PWV).

In one study, the PWV of AAS users was compared to the average expected PWV for their biological age. The results indicated that the arterial age of AAS users (as determined by PWV measurements reflecting vascular stiffness) was, on average, seven years higher than the biological age of the subjects [**29**]. The results of this study provide a very important piece of information, as vascular stiffness and hypertension are closely related in a vicious circle: high pressure damages the vascular walls, which leads to stiffening; in turn, stiffer vessels cause a further increase in systolic pressure and vasoconstriction.

One study showed that AAS caused an increase in both systolic and diastolic blood pressure [**30**][**31**], and this increase was correlated with the duration of AAS intake [**30**] (the longer AAS were used, the higher the recorded blood pressure values). The study indicated that 70% of athletes taking AAS suffered from hypertension, compared to 30% of the control group [**30**]. However, not all studies comparing the blood pressure values of AAS users against a control

group agree on the hypertensive effect of AAS. Indeed, there are studies whose results fail to show an association between AAS use and increases in blood pressure [**32**][**33**][**20**][**34**]. The disparity among studies in this regard underscores the need for detailed research that will unambiguously clarify the pathophysiology of the effect of AAS on blood pressure, as well as determine the dose-dependency, duration of use, and reversibility of this effect.

2.5 Myocardial hypertrophy: Numerous studies link left ventricular hypertrophy (LVH) with AAS use. Myocardial hypertrophy has been confirmed in post-mortem studies of men who used AAS compared to the hearts of deceased non-users[**35**]. Myocardial hypertrophy has also been confirmed by imaging methods such as Doppler ultrasound[**36**] and magnetic resonance imaging, showing myocardial hypertrophy, enlargement of the right and left ventricles, increased thickness of the left ventricular wall, and impaired systolic and diastolic function[**37**]. Myocardial hypertrophy in AAS users results from hormonal dysregulation and increased activity of the renin-angiotensin-aldosterone system (RAAS). Increased aldosterone levels cause myocardial hypertrophy and myocardial fibrosis (resulting from increased fibroblast activity and collagen production), and lead to cardiac arrhythmias independent of hypertension[**38**]. Another study found an association between aldosterone levels specifically the responsiveness of aldosterone secretion to increased angiotensin II and increased left ventricular mass[**39**].

A pooled analysis of multiple studies on the cardiovascular effects of AAS showed that the most common macroscopic lesion in AAS users was cardiomegaly (33%), followed by left ventricular hypertrophy (30%) and dilated cardiomyopathy (9%). The most common histological changes were foci of fibrosis (79%) and necrosis of myocardial tissue (52%), with the duration of AAS use ranging from three months to many years [40]. Myocardial hypertrophy carries serious consequences for cardiac function. A study comparing the cardiac function of AAS users with non-users showed that AAS users had impaired left ventricular systolic function (mean ± SD left ventricular ejection fraction = 52 ± 11% versus 63 ± 8%) and diastolic function (early relaxation velocity = 9.3 ± 2.4 cm/s versus 11.1 ± 2.0 cm/s). Current AAS users exhibited significantly lower left ventricular ejection fraction (49 ± 10% vs. 58 ± 10%) and reduced diastolic function, as evidenced by lower early relaxation velocities (8.9 ± 2.4 cm/s vs. 10.1 ± 2.4 cm/s), compared to the control group [**15**].

In addition, AAS users showed greater coronary artery atherosclerotic plaque volume than non-users [**23**]. In a study of rats receiving AAS, it was noted that these agents increased susceptibility to myocardial ischemia, infarct size, and myocardial reperfusion injury, while further impairing cardiac function after an ischemic episode [**41**]. The results of these studies suggest that AAS use causes myocardial hypertrophy and increases the risk of serious cardiovascular complications. According to some studies, AAS use may also be associated with dilated cardiomyopathy [**42**][**43**][**44**]. This association remains controversial, as many studies do not list AAS use as a primary cause of dilated cardiomyopathy, focusing instead on genetic factors independent of the use of these agents [**45**].

2.6 Cardiac arrhythmias: Studies indicate that the use of AAS increases the risk of arrhythmias of supraventricular and ventricular origin, while also increasing the risk of sudden cardiac death (SCD). AAS use can cause cardiac dysautonomia involving increased sympathetic nervous system activity and decreased parasympathetic nervous system activity (resulting from impaired vagus nerve modulation), which increases susceptibility toward arrhythmias, vasovagal syncope and even SCD **[46][47][48]**. One effect of the dysautonomia cited is the lack of the typical post-exercise blood pressure reduction observed in non-AAS users **[3]**. The chronic predominance of the sympathetic nervous system facilitates evoked excitation and also the circulation of the excitation wave in the reentry mechanism (this is due to increased dispersion of repolarization and increased intraventricular calcium current), which increases the risk of subsequent depolarizations **[49]**. In another study, long-term AAS use was found to be associated with AEMD (atrial electromechanical delay) in young healthy bodybuilders, raising the conclusion that AAS use may be related to the development of atrial fibrillation and arrhythmias in the circulating waveform mechanism of the reentrant beat **[50]**. AAS users had a longer QT interval, which also reflects a proarrhythmic state in AAS users **[51]**. A study in mice showed that combining AAS (Nandrolone) use with moderate intensity exercise increased the risk of dangerous arrhythmias in the form of ventricular fibrillation (VF) **[52]**. Another study in mice showed changes in the ventricle repolarization process (action potential and extracellular potassium current were studied) of mice treated with nandrolone. The changes mainly affected the left ventricle **[53]**. Myocardial hypertrophy and fibrosis are independent risk factors for cardiac arrhythmias through impaired conduction of impulses in the cardiac pacing system **[54]**.

2.7 Myocardial ischemia and myocardial infarction: AAS users (both those currently taking AAS and those who have taken AASs in the past and ceased using it) have been shown to have lower coronary reserve values compared to non-users, suggesting deterioration of coronary microcirculatory function by anabolic steroid substances **[55]**. In addition, supra-physiological doses of testosterone acting on calcium channels have been shown to cause coronary vasospasm **[56]**. AAS can also cause coronary vasoconstriction indirectly through other mediators. The study in rabbits showed that nandrolone therapy decreased cGMP levels, which translated into impaired vascular muscle relaxation **[57]**. The study in guinea pigs administered testosterone showed increased sensitivity of the thromboxane A2 (TXA2) receptor relative to a control sample, which is important because TXA2 is associated with increased cardiovascular risk and myocardial infarction **[58]**. In a case report of a 32-year-old patient with myocardial infarction, the researchers linked the fact of AAS intake to infarction without significant coronary artery stenosis on angio-CT. They concluded that the AAS may have affected coronary artery spasm and myocardial ischemia. Thus, acute coronary syndrome can be expected even in a young person without significant coronary artery stenosis **[59]**. In the study in mice, testosterone was shown to reduce prostacyclin production in arterial smooth muscle, thereby promoting plaque and thrombus formation **[60]**. AAS use can lead to coronary artery endothelial hyperplasia, plaque erosion, and thrombus formation potentially leading to sudden cardiac death **[61]**. The cited studies indicate that because of the multidirectional effects of AASs on the coronary vasculature, myocardial infarction can be expected in AAS users both in the mechanism of coronary artery stenosis and without significant coronary artery stenosis. This provides clinically important information for a physician initiating the diagnosis of acute coronary syndrome in a patient taking AAS.

3. Discussion

The results of the presented scientific studies clearly suggest that AAS increases cardiovascular risk and the risk of cardiovascular events. However, not all studies agree with the nature of this effect and its reversibility. According to one study the effect of AAS on the lipid profile is reversible and cholesterol levels normalize within approximately 5 months after discontinuing AAS use. In addition, the effect on HDL cholesterol levels depends on the type of AAS used**[21]**.

The use of AAS is associated with increased procoagulant and fibrinolytic activity but the effect of AAS on thromboembolic events cannot be clearly confirmed**[26]**. According to some studies, no association between AAS use and increased blood pressure has been demonstrated**[32][33][20][34]**. According to some studies, the use of AAS is associated with dilated cardiomyopathy**[42][43][44]**. This association is controversial as many studies contradict it**[45]**. These discrepancies highlight the need for further research on the effects of AAS on the cardiovascular system and to prove its effects in order to better understand the risk associated with the use of these agents. It is also important to use information of the impact of AAS on cardiovascular system to develop effective primary prevention methods at the population level. For example, WADA (World Anti-Doping Agency) attempts to convey important information to young people via social media about the harmful effects of AAS. Many similar anti-doping campaigns around the world are based on knowledge about the effects of these substances derived from scientific publications which confirms the need to develop knowledge on this subject.

4. Conclusions

The results of scientific studies confirm the effect of AAS on vascular calcification. Longer cumulative lifetime AAS use, especially exceeding five years, is associated with a greater likelihood of calcification. AAS use adversely affects the lipid profile by causing an increase in the LDL cholesterol fraction and a decrease in HDL cholesterol (a reversible effect), thereby increasing the risk of atherosclerosis, including in the coronary arteries. Studies suggest that AAS use increases the risk of thromboembolic incidents, as evidenced by an increase in clotting factors; however, not all studies agree on the definitive association between AAS and thromboembolic episodes.

AAS use has been linked to the development of hypertension (primarily systolic), in part through the stimulation of the renin-angiotensin-aldosterone (RAA) system and increased arterial stiffness. Left ventricular hypertrophy (LVH) also shows a close relationship with AAS use. Myocardial hypertrophy in AAS users results from hormonal dysregulation and increased activity of the RAA system, leading to impaired cardiac systolic and diastolic function, arrhythmias, and increased susceptibility to ischemia. While some studies have attempted to show a link between AAS intake and dilated cardiomyopathy, their results remain inconclusive.

AAS use carries a risk of arrhythmias of both ventricular and supraventricular origin. This is a result of dysautonomia and myocardial fibrosis, among other factors, and is associated with an increased risk of sudden cardiac death. AAS increase the risk of myocardial infarction through both coronary artery stenosis and mechanisms without significant stenosis, such as the disruption of coronary microcirculation and the promotion of coronary vasoconstriction.

The results of the presented studies confirm the negative effects of AAS use on cardiovascular health and indicate an increased risk of adverse cardiovascular events, including cardiovascular death. In view of the epidemiological data and the multidirectional negative health effects of AAS abuse, it is concluded that knowledge of the harmfulness of these substances should be disseminated. Furthermore, appropriate primary prevention should be developed as early as at the school level to mitigate the danger posed by this phenomenon at the population level.

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Author's contributions

Conceptualization: Eryk Ubysz, Patrycja Kwitowska

Methodology: Eryk Ubysz, Patrycja Kwitowska, Emilia Muraszewska, Małgorzata Pyjecka, Łukasz Muraszewski, Agata Król, Edyta Lewandowska, Cezary Łuczyński, Maciej Paczkowski, Mariusz Wręczycki

Resources: Agata Król, Edyta Lewandowska, Cezary Łuczyński, Maciej Paczkowski

Data curation: Mariusz Wręczycki, Cezary Łuczyński, Maciej Paczkowski

Formal analysis: Eryk Ubysz, Patrycja Kwitowska, Łukasz Muraszewski, Mariusz Wręczycki

Investigation: Patrycja Kwitowska, Małgorzata Pyjecka, Łukasz Muraszewski, Edyta Lewandowska

Supervision: Emilia Muraszewska, Małgorzata Pyjecka, Agata Król

Writing–rough preparation: Eryk Ubysz, Patrycja Kwitowska, Emilia Muraszewska, Łukasz Muraszewski, Edyta Lewandowska, Mariusz Wręczycki

Writing-review and editing: Eryk Ubysz, Patrycja Kwitowska, Małgorzata Pyjecka, Agata Król, Cezary Łuczyński, Maciej Paczkowski

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