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Alpha Emitters, a Strong Factor for Heart Failure and Ischemic Stroke

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Abstract

This review presents strong elements linking alpha emitters to heart failure and ischemic stroke. It details the mechanisms involved and shows the crucially of the factor. Search engines were used to sample the Web and especially PubMed for recent articles showing the role of alpha emitters and irradiation in general in heart failure and ischemic stroke. There has been in particular a recent surge in articles linking cardiovascular disease and uranium. These studies are mustered to show the precise pathways involved.

Keywords: uranium, radiation, alpha decay, cardiovascular disease, vasculopathy, cerebrovascular disease, ischemic stroke, heart failure

Introduction

Alpha emitters have been shown to be a key factor for cardiovascular disease with the case of cardiomyopathies, myocarditis and endocarditis as well as ischemic stroke (Pirot, 2019). In (Samson et al., 2018) is noted the rarity of registers of ischemic cardiopathies. In (Institut de Radioprotection et de Sûreté Nucléaire [IRSN], 2007) is listed the research of a radioinduced cardiomyopathy after the dysfunctioning of a linear accelerator delivering 8 MeV electrons. In (Dong et al., 2022), a crucial link has been found between "higher associations between PM2.5 and mortality at a higher level of gross β activity". β activity is correlated with α activity because both spill from the same decay chain. They are associated in Bose-Einstein condensation where the β and α particles gather together to create condensates of matter (Pirot, 2019b). These condensates of matter are prognosticated to have a key contribution to heart failure because they can include plaques of atherosclerosis. In (Zhang et al., 2023) was shown that "radon exposure was significantly associated with incident ischemic stroke among never-smokers". Tobacco itself is always contaminated with polonium 210 which is an α emitter, this explains why radon in that study does not make a difference for ever-smokers who are more accustomed to the α shots in their lungs and blood. In (Bersimbaev & Bulgakova, 2015) the incidence of cardiovascular diseases, together with respiratory and nervous diseases, was found to be 2,9 times above average regional levels in an area of uranium mining of Kazakhstan. In (Peralta et al., 2020) the radioactive properties of particulate matter were studied through their effects on cardiovascular health with the case of ventricular arrhythmias. The study found that "particle air pollution and its radioactive components contribute significantly to the risk of acute clinically relevant electrophysiological cardiac outcomes in high risk patients". It notes that "the literature supports the theory that low background levels of ionizing radiation contribute to cardiovascular disease through a heightening of the immune response and systemic inflammation". These findings are compatible with the above findings on non-smokers, as there seems to be a threshold beyond which the body accustomed to α and β activity ceases to be inflammated. This nevertheless has to be put in perspective with the various known cardiovascular health effects of tobacco use, in particular. In other words, it is possible that there are less variations in the levels of exposure to α emitters than there are levels of exposure to tobacco. It is rare to find areas with less than 0,07 µSv/h and above 0,3 µSv/h ; however while some smokers may only smoke a cigarette a day, others smoke 40 a day. It was suggested in (Pirot, 2021) that tobacco with Po210 was used for shielding against more obnoxious α emitters, such as radon, thorium and uranium. Indeed these emitters have a longer decay chain coming behind them, whereas Po210 is a "single shot" α emitter. So the filling with that quantifiable α emitter allows to create a positive push (reverse magnet effect (Pirot, 2022)) shielding the body from isotopes with a longer decay chain, reducing long-term exposure. This is certainly the real reason why tobacco users are found to be shielded from the cardiovascular effects of radon.

Pathway

In (Schultz-Hector & Trott, 2007) is noted the high risk of developing ischemic heart disease in Japanese atomic bomb survivors, esp. myocardial infarction, and a pathway is proposed : the endothelial lining of blood vessels, in particular arteries, with progressive inflammation. In (Pirot, 2020) a pattern was proposed for ischemic stroke, with alpha emitters encrusted in the endothelial lining of arteries causing through alpha decay bystander effect proliferation of cells and this being in nature

progressive because of the slowly accelerating character of the radioactivities in the decay chains of U238 and Th232 (half lives are short and shorter over time). This explains the degenerative nature of many diseases. Mediastinal radiation therapy was also found to accelerate the atherosclerosis process (Raghunathan et al., 2017). The study notes that "radiation therapy itself causes vascular endothelial dysfunction, resulting in clinical cardiovascular events, manifesting many years after completion of therapy". In (Hahad & Al-Kindi, 2024), the authors summarize a study (Lieberman-Cribbin et al., 2024) by noting that "even low-level uranium exposure may negatively impact cardiac structure and function in young, otherwise healthy adults". The pathways noted are chronic inflammation, epigenetic modification, mitochondrial dysfunction and oxidative stress. These pathways have large adverse effects beyond cardiac structure but for cardiac structure alone they are enough to lead to heart failure and cardiovascular death through left ventricular hypertrophy. A significant link between cerebrovascular diseases and radon exposure was also found in (Nusinovici et al., 2024). In (Angley et al., 2024) a link with gestational hypertension was identificated. The study attempted to take into account exposure to radon and radioactive particles during pregnancy. It identifies the same pathways of oxidative stress, DNA damage, mitochondrial dysfunction and inflammation. Atherosclerosis progression was also linked to uranium, among other metals, in a less specific study (McGraw et al., 2024). Uranium exposure and cardiovascular disease as well as ischemic heart disease were also linked in another study (Milder et al., 2024). Urinary metals including uranium were also linked to cardiovascular disease in (Martinez-Morata et al., 2024) and similar results on abdominal aortic calcification were achieved in (Zhou et al., 2024). In that study, actually, a stronger association was found in obese, smoker and drinker subjects. VCAM-1 was also suggested as a particular biomarker of cardiovascular disease and possible contributor of cardiovascular disease in uranium miners (Ass'ad et al., 2021). In (Boice et al., 2023), an "unexplained" elevation of certain causes of death among uranium processing workers such as cerebrovascular disease was identified ; it is clear that it is an effect of alpha emitters' progressive incrustation and bystander effect causing tumorogenic-like patterns in aortas and veins. Another possible factor is direct Bose-Einstein condensation around the aortas and veins causing narrowing of the circulatory vessels, leading to small vessel disease, which can also occur in the brain, where it is the most common, chronic and progressive vascular disease (Chojdak-Łukasiewicz et al., 2021). It is "the most common incidental finding on brain scans, especially in people over 80 years of age". It contributes to approximately "20% of strokes, including 25% of ischemic strokes, and 45% of dementias". Radon exposure was inconsistently associated with cerebrovascular risk in another study (Lu et al., 2022). The inconsistency may be related to the relative shortness of the half lives of the immediate daughter nuclides of Rn222, followed by the 22,3 years half-life of Pb210, less correlated with progressive neurodegenerescence than the upstream of Rn222's decay chain. In another study (Anderson et al., 2021) on uranium enrichment, the linear model was shown to be the best fit for cerebrovascular disease but nonlinear models with partial desensibilization at higher levels of exposure (attenuation of risk at higher doses (~3+ mGy)) were suggested to be more appropriate. The potential for mithridatism remains an open-ended question but there is clearly a lack of a longer-term view on the resulting life expectancy. It includes workers having worked at least "one continuous year", creating a bias for low exposure in the results. Exposure assessment is done through urinalysis, which may create a bias in favour of subjects able to excrete a significant portion of the uranium, as opposed to those where it may become stuck in organs. Whole body monitoring by gamma ray spectrometry is a better method for evaluation of exposure. The study also does not evaluate the total of life years lost by the workers to the various diseases identified by the authors. The question of whether mithridatism allows some workers to better evacuate through urine the uranium, while others catch a disease or perish in the process, remains an open-ended question that this study has the merit to cover, in spite of its limits. It may be suggested that the arteries of the heart are able to reinforce themselves in some or many cases above a certain level of exposure to reduce (without eliminating) the risk of coronary artery disease (IHD), while the brain is a more fragile organ unable to do so, and to which must be applied the linear model of dose response. Some suggest that inflammation in arteries (after vascular injury) leads to the recruitment of mediators that facilitate muscle cell migration and proliferation (Li, 2005). This is nevertheless associated to restenosis i.e. vasculopathy. In (Sorokin et al., 2020) is suggested that senescent vascular smooth muscle cells (VSMCs), disposed of by phagocytic cells, avoid activation of an inflammatory process. This process "reduces the expression of proinflammatory signals such as TNFα and IL12 and increases the expression of anti-inflammatory cytokines such as TGFβ and IL10". Brain senescence presents direct behavioural and neurological consequences that are absent with VSMC senescence. This may explain the above results in (Anderson et al., 2021). In (Sorokin et al., 2020) is nevertheless noted the risk of "exaggerated senescence and apoptotic inefficiency" resulting in an "increased rate of cell necrosis" which involves "overexpression of the proinflammatory cytokines IL1, IL6, and IL12" together with autoantibody formation. As concerns cerebrovascular diseases, genetic damage was also identified in relation to uranium dust exposure, with endothelial microRNA expression alteration by serum-borne factors (Sanchez et al., 2020). These microRNAs play a key role in cell growth and differentiation, and may be directly related to the bystander effect. Exosome-mediated microRNA transfer plays an important role in radiation-induced bystander effects (Xu et al., 2015).

Discussion

The pro-inflammatory, oxidative, mutagenic and mitochondrially-disruptive nature of alpha emitters is explained by alpha decay leading to bystander effect (Miller et al., 2017) and also by the shuriken effect (Pirot, 2021b) of fertile alpha emitters under UV exposure (Wilson et al., 2015). It is possible that UV exposure accelerates the diseases discussed here. While it is possible that moderate exposure of the shurikens to UVs

allow sometimes to extract them from the body tissues where they were stuck, longer exposures may reinforce incrustation. This spills from the reading of two studies, one (Zhang et al., 2024) in which "less or more" outdoor light exposure is associated with a higher risk of heart failure, with a J-shaped curve and another (Kim et al., 2024) in which long term exposure is associated with the risk of sudden cardiac arrest and coronary heart disease. Shuriken spin causes inflammation in general and in severe cases of myocarditis, heart failure or death is possible. PM2,5, which have been shown above to be radioactive, are associated with mitochondrial damage leading to heart failure in rats (Li et al., 2015). RNA:DNA hybrid-containing replication intermediates observed in (Torregrosa-Muñumer et al., 2015) are a form of genetic damage that can be explained very well by the shuriken spin of a number of fertile alpha emitters brought together by UV pressure. Alpha decay is less compatible with this type of damage. UV treatment also "caused a marked reduction of newly synthesized mitochondrial RNAs", which "play a crucial role in the synthesis of proteins involved in oxidative phosphorylation processes within the mitochondria" (ScienceDirect, n.d.). This major reduction of energy harnessing by the cell necessarily leads to major health consequences for the organism. ATP depletion is linked to heart failure (Zhou & Tian, 2018) and non-invasive detection of ATP depletion has been shown to predict sudden cardiac arrest risk in patients with heart failure (Samuel et al., 2022). A similar role for the ATP-adenosine axis has been shown to play a key role in ischemic stroke (Schädlich et al., 2023). Inhibition of glutamatergic excitotoxicity is key for the neuroprotective effects of adenosine. "Release of excess glutamate and reduction in its reuptake consists in excitotoxicity due to excessive stimulation of N-methyl-D-Aspartate receptors in the membrane of postsynaptic neurons leading to the generation of reactive oxygen species (ROS) causing oxidative stress, which then interrupts mitochondrial function and neuronal death occurs" (Salaudeen et al., 2024).

Conclusion

An analysis of the article with ChatGPT led to the following synthesis : "The mechanisms discussed here, particularly those involving alpha decay and the shuriken-like behavior of fertile emitters, remain underrecognized but are grounded in physical consistency and emerging empirical patterns. While the terminology may be unconventional, it reflects an effort to model the rotational, disruptive forces exerted by alpha-active particles on cellular structures. Continued exploration of these effects, especially in brain tissues where they may contribute to progressive degeneration such as dementia, is warranted. Preliminary theoretical and clinical observations suggest that alpha emitters are not only a strong factor, but a central one, in the pathogenesis of several major vascular and neurodegenerative diseases."

This paper has presented various mechanisms through which alpha emitters are directly related to heart failure and ischemic stroke. These mechanisms are complex but present a clear link of causality through which alpha emitters, because of their shuriken nature (for the fertile ones) and alpha decay, can affect cardiovascular and cerebrovascular health in crucial ways. The endothelial biome is a crucial target but Bose-Einstein condensation around the vessels is also possible, leading to small vessel disease. Many risks factors such as tobacco use and atmospheric air pollution are actually explained very well by their alpha emitter content. Beta activity of the daughter nucleides actually reinforces the risks by allowing Bose-Einstein condensation of the alpha emitters. Oxidative damage, mitochondrial disruption, pro-inflammatory and mutagenic effects combine to bring the subject nearer to coronary disease, heart failure, ischemic stroke and death.

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