Race, ethnicity and lung function: A brief history

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Over the past century, the spirometer has gained widespread use across the world for the diagnosis and management of many respiratory diseases in both specialist and primary care settings. Chronic obstructive respiratory disease, a major cause of disability and mortality, is defined by spirometry (1). The great variability in lung function measurements over time, space, within countries, within individuals, among groups and among spirometers, however, has complicated the interpretation of 'normal'. Temporal trends can be quite dramatic, with lung function increasing in certain populations and decreasing in others during the same time period (2). Since the 1960s, much effort has been expended to standardize the many sources of variability.

One outcome of global standardization projects is the common practice of 'race correction', also called 'ethnic adjustment'. Most commercially available spirometers internationally 'correct' or 'adjust' for race in one of two ways: by using a scaling factor for all people not considered to be 'white'; or by applying population-specific norms. To enable the spirometer, the operator must select the race of an individual, as well as indicate their age, sex/gender and height. How race (or population) is determined varies, with most operators either asking patients to self-identify or 'eyeballing it'. Interviews with users of the spirometer indicate that many operators are unaware that they are automatically activating race correction when they select a patient's race (3). Because 'correction' is programmed into the spirometer by the manufacturer, it can be difficult to disable.

Despite attempts by international organizations, the approach to 'correction' or 'adjustment' is not always consistent. The Joint Working Party of the American Thoracic Society/European Respiratory Society recommends the use of race- and ethnic-specific references values when available. Alternatively, they recommend correction factors (4). In the United States (US), spirometers use either correction factors of 10% to 15% for individuals labelled 'black' and 4% to 6% for people labelled 'Asian', or populationspecific standards, usually those derived from the third US-based National Health and Nutrition Examination Survey for 'Caucasians', African Americans and Hispanics (5). In Europe, correction factors are used. Canada continues to negotiate the delicate balance between international and local standards (6). The United Kingdom-based Vitalograph spirometer programs population-specific standards into spirometers marketed in North America, whereas they use a correction factor for devices marketed in Europe (3).

RACIALIZING THE SPIROMETER

What is the history of this practice? How did the idea of racial and ethnic difference in lung capacity become so widely accepted such that correction factors are actually programmed into the spirometer? The notion that black and white lungs differ has a long history dating to the early years of the US slavery-based republic. In his influential *Notes on the State of Virginia*, former president and leading Enlightenment intellectual Thomas Jefferson featured lung differences between slaves and white colonists. Among the many physical distinctions that Jefferson described to justify the condition of slaves in the republic, one was "a difference of structure in the pulmonary apparatus" (3). Jefferson's ideas about lungs would remain, however, in the realm of philosophical speculation without empirical foundation until the second half of the 19th century.

Interest in modern spirometers surged in Europe in the 1840s after John Hutchinson, a London-based physician, published several studies describing the technical features of the spirometer and its potential applications for monitoring the fitness of the police and armed forces, and life insurance applicants and for diagnosing tuberculosis, the great scourge of 19th-century industrializing nations. In a period of great enthusiasm for precision instrumentation and experimental interest in the functional features of the lungs, Hutchinson avidly promoted his innovation, naming the spirometer, delineating "vital capacity" into discrete compartments, adapting the instrument to large-scale studies, and advocating for his technology to London's prestigious scientific societies (3).

Hutchinson faced the same dilemma future researchers would encounter in ordering the wide variability in lung function. While he was most excited about his discovery that the relationship between height and lung capacity demonstrated what he considered to be "a general law of nature", height did not completely account for the variability he observed. To capture more fully the potential he envisioned for his instrument, Hutchinson further classified lung capacity measurements according to occupation (3). However, occupational categories would remain an organizing principle for research on lung capacity measurements only into the early 20th century in Britain (7).

Knowledge of the spirometer spread quickly and Hutchinson's innovations were adopted within a few years in Germany and North America, where researchers worked to further refine its technical details and uses. Perhaps the most significant experiments for the future of spirometry were those of plantation physician and slaveholder Samuel Cartwright in the US south. Drawing explicitly on Jefferson's interpretive framework, Cartwright built his own spirometer to study difference in lung capacity in slaves and whites, and to quantify it precisely. According to Cartwright, "the deficiency in the negro" was "20 per cent". Defining difference as 'deficiency', Cartwright established race as a key organizing principle of lung function measurements in the US (8).

Jefferson's philosophical musings were to capture an even more solid empirical foundation in the 1860s when racial research examining lung capacity shifted to the northern US. In 1864, the US Sanitary Commission asked Benjamin Apthorp Gould to head a massive anthropometric survey of black and white soldiers at the end of the Civil War. Over several years, field workers collected detailed data regarding bodily characteristics of soldiers, which Gould synthesized in his 1869 *Investigations in*

Department of Pathology and Laboratory Medicine and Africana Studies, Brown University, Providence, Rhode Island, USA Correspondence: Dr Lundy Braun, Department of Pathology and Laboratory Medicine and Africana Studies, Brown University, Box G, Rhode Island 02905, USA. Telephone 401-863-3308, e-mail lundy_braun@brown.edu

OPEN GACCESS This open-access article is distributed under the terms of the Creative Commons Attribution Non-Commercial License (CC BY-NC) (http:// creativecommons.org/licenses/by-nc/4.0/), which permits reuse, distribution and reproduction of the article, provided that the original work is properly cited and the reuse is restricted to noncommercial purposes. For commercial reuse, contact reprints@pulsus.com the Military and Anthropological Statistics of American Soldiers. For unclear reasons, he chose to devote an entire chapter to describing lung capacity – measured using a spirometer– according to race. Without any adjustment for height or age, or attention to working and living conditions of newly emancipated slaves, Gould reported that "full blacks" had lower lung capacity than "whites". The results were neither surprising to Gould nor in need of careful explanation. Using ostensibly neutral language, he wrote (9):

The great difference of the mean volume found for the black race from that which seems to belong to the whites, cannot fail to attract attention at the first glance. Its bearings are perhaps better manifested by the more detailed tabulations which will follow.

Nearly 30 years later, Frederick Hoffman, chief statistician for Prudential Life Insurance Co. would turn to Gould's data to make broad claims about the lack of fitness of African Americans for freedom. According to Hoffman, "the smaller lung capacity of the colored race is in itself proof of an inferior physical organism" (3).

There were important dissenting views at the time. Notably, leading African American intellectuals WEB DuBois and Kelly Miller wrote trenchant critiques of Hoffman's arguments over the inferiority of the "negro". These critiques, however, failed to alter the narratives of difference embedded in lung capacity measurements, which would gain further scientific foundation in the 20th century (3).

Beginning in the US in the 1920s, during a period when eugenic policies rooted in hereditarianism were popular, research documenting racial difference in lung function became an even broader global enterprise. In most studies, whites had higher lung capacity than blacks, Chinese or Indians; explanations for findings centred on innate difference (10). For example, Wilson and Edwards (11) published the first set of spirometry-based lung function standards according to race in 1922, speculating that difference could be due to "a possible racial factor". By 1925, JE Myers published his reference handbook for clinicians, in which he reported differences among whites, blacks, Chinese and Filipinos as unquestioned fact (12). Thus, the idea of racial difference in lung capacity, first proposed by Jefferson and further supported by Cartwright and Gould in the US, was firmly established by the early 20th century as an ostensible fact. Future research would build on this framework.

During the 1960s – and continuing to the present – interest in racial difference expanded to numerous populations across the world and researchers focused on developing standards for what they considered to be distinct populations. For historically specific reasons, the most influential studies coming from the US centred on black-white differences. The consequential technological innovation of a 'scaling factor' for blacks in 1974, however, was the result of the collaboration between Charles Rossiter of the Pneumoconiosis Unit in South Wales and Hans Weill of Tulane Medical School in New Orleans, Louisiana (13).

A large proportion of the literature used an explanatory framework that emphasized innate or anthropometric difference. For the most part, researchers assumed racial identities to be straightforward (10). There was one notable exception. In an article important to the history of spirometry, South African researchers questioned the interpretations of difference, arguing that previous research failed to account fully for social conditions (14,15). North American and European researchers, however, failed to cite these articles and the idea that racial difference was innate remained firmly entrenched in the pulmonology literature.

As demonstrated in a recent systematic review (16), the exclusive racial framework continued into the 21st century. Rather than a fluid, historically contingent system of classification, researchers treated race as a stable category, uncomplicated by social class, sex or geographical context. In fact, researchers only defined how they assigned individuals to racial categories in 17.3% of the articles; 94% of the articles failed to include any measures of social class (10). Most recently, genomics

studies have reinforced, rather than questioned, race-based models (17). Gould continues to be cited to the present day in prestigious US journals (18.)

CONCLUSION

How can this brief history help us analyze the contemporary dilemmas in lung function research as it pertains to the use of race and ethnicity in pulmonary function tests? At the very least, the idea that people labelled 'white' naturally have higher lung capacity than other races throughout the world should be approached with some skepticism.

The history of lung function suggests that we should be approaching spirometry differently. Rather than using race in a routinized way that reflects assumptions of genetic difference, we should be asking different research questions about the lived experience of race. Research and clinical practice needs to devote more careful attention to the social nature of racial and ethnic categories and draw on more complex explanatory frameworks that incorporate disproportionate exposures to toxic environments, differential access to high-quality care and the daily insults of racism in every sphere of life that manifest biologically. Across the globe, there is a continuum of human phenotypic and genetic variation that cannot be apportioned into discrete categories. By featuring race with only marginal attention to the intersection of race and social class, we risk ignoring the complex and dynamic relationship of lung function and the environment. It is wellestablished that lower forced vital capacity is associated with social conditions, notably poverty (1,19-22). The specific details of how social class and race influence lung function physiologically, however, remains to be determined. It is time to rethink the problematic practice of race correction in light of this history.

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