Researching vs. Reifying Race: The Case of Obesity Research

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ABSTRACT

This paper deals with the reification of the concept of race in biomedical research. It combines philosophical analysis and a quantitative approach to investigate the ways in which the reification fallacy may occur in race research, thereby providing theoretical legitimacy to the misuse of scientific research. It examines the prevalence of obesity in the US and some African countries as an empirical case to guide a conceptual analysis. The paper suggests that, to avoid the reification of race, researchers need to be more aware of the fact that continental genetic clusters do not necessarily correspond to the genotypic partitions of interest in therapeutic reaction or disease etiology, and need to take seriously the phenotypic variability of breeding populations within continents.

I. Introduction

Biomedical race research continues to spark controversies because of its problematic epistemic foundations and potential negative externalities. But because researchers often use racial categories as placeholders for human

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population substructures,[1] race research may, under certain conditions, positively impact population health. Yet even the most optimistic race researcher might still concede that this type of research may lead, at the very least, to racial stereotyping. However, in the case of racial stereotyping for example, it is clear that race researchers do not have control over third-parties' uses of their findings. Nevertheless, they have an obligation to avoid providing inadvertently theoretical legitimacy to the misuse of their research. A race researcher may unwittingly provide theoretical ground for misuses of his/her research results if the study commits, for instance, what is called the reification fallacy. Generally speaking, the fallacy of reification consists of erroneously attributing an objective independent basis to a notion that is merely an abstraction (Whitehead, 1925a; 1925b; 1929). As we shall see later in detail, the fallacy of reification of race is defined as a mistaken attribution of an objective biological basis to race (Gould, 1996; Duster, 2005).

Actually, the reification of race in scientific research has the potential to affect negatively medical practice itself. It may for example prevent a health professional from tailoring treatment to the individual needs of a patient because of racial assumptions. Thus racial assumptions about sickle cell anemia (see section II below) lead to misdiagnoses and various negative therapeutic consequences including «ineffective pain treatment» (Royal et al., 2011, p. 391). There is thus a need to subject race research to stringent methodological and ethical constraints (Maglo, 2010, 2012). To be sure, the reification of race takes many different forms in biomedical research even if all these forms may appear to be underwritten by an implicit belief that race is a natural biological fact in light of human evolutionary history. In fact, the possibility of determining continental ancestry in genomics has recently given a new momentum to biological race realism, or the contention that race is a natural category and therefore is rooted in biological reality. In this view, racial categories pick out distinct evolutionary kinds of humans. Biological race realism has sometimes served as a means to justify theoretically the use of race in biomedical research (Burchard et al., 2003; Risch et al., 2002). We argue that biomedical race research does not require a theoretical grounding in a realist framework and that, to avoid the reification fallacy, researchers should

[1] The term population substructure refers to the underlying genetic variation between subsets or subgroups within a population. The variation may be due to factors such as mating and migration patterns, mutation, etc.

use race, when need be, parsimoniously in an instrumentalist framework merely as a problem-solving conceptual device.

Accordingly, we have chosen to examine in what follows the ways in which the reification fallacy may occur in race research by studying obesity prevalence in various populations. Obesity is a complex trait influenced by multiple interactions between genetic and environmental factors (Martin, Woo, & Morrison, 2010). Yet social attitudes towards body image vary across time and space. But body mass index (BMI) has health implications, and health considerations are paramount in obesity research. Thus, the "obese" person may not only be subjected to negative social attitudes towards body size but s/he also may suffer from adverse clinical effects of the "obese" body. Nonetheless, rather than the esthetical standards associated with body image, it is the scientific measure in terms of BMI and its clinical correlates that concerns us here. Obesity research is particularly suitable for probing the reification fallacy because although it is a common condition, its incidence rates vary significantly among human populations. The question then is how we should go about researching this variation without reifying race.

For example, we may report some of the results of our study about obesity with the following statement: «we found that black women have higher incidence rates of obesity than white women». Statements such as this are typical of scientific race research reports. Yet not only would our statement be misleading but it may, even more so, be fraught with the reification fallacy if the study were conducted only on the US population as is often the case in race research.[2] Indeed, even though obesity prevalence tends to be higher among African American women compared to European American women in our study (see Fig. 1), we also found that obesity rates in many African populations (see Table 1) are far lower than those of European American women and, hence, of European American men. Combining then empirical methods and philosophical analysis,[3] we identified four major potential forms of reification in biomedical race research. These are: reification by mean-thinking, reification by cluster stability rule violation, reification by molecular

[2] Some researchers attempt to alleviate these concerns by specifying in their reports for example "US blacks", "US whites", etc. So we are not arguing for a complete ban on the use of racial categories. We only recommend that researchers be more alert to the potential of the reification fallacy and to the conditions of an appropriate use of these terminologies (for more details, see section IV).

[3] The method adopted in this paper – which consists of combining empirical investigation, normative ethical inquiry and conceptual analysis – has been dubbed elsewhere "axiological empiricism" (Maglo, 2010; 2012).

reductionism and reification by analysis of variance (ANOVA) epistemic naturalization. We explain each of these forms of reification in detail in section IV. Suffice it here to say that by ANOVA epistemic naturalization, we refer to the claim that contemporary statistical and computational methods vindicate race as a biological category having a regular predictive value in biomedicine.

The paper is an interdisciplinary paper articulated in three main parts. We first analyze the historical and theoretical issues surrounding the putative biological reality of the concept of race and its reification in scientific research. We show that even genuine scientific discoveries, when cashed out in questionable theoretical and ethical frameworks, may lead to the reification of race and cause harms. Then, we present empirical evidence about the prevalence of obesity in the US and in some African countries. The results of this empirical investigation, particularly the findings about the variability in obesity incidence rates within and between populations across continental regions, set the stage for the identification of potential forms of reification of the concept of race in biomedical research. Finally, we discuss the issue of reification in connection with the role of continental genetic ancestry in race research. Here, we demonstrate the importance for biomedical researchers to take seriously the philosophical implications of phenotypic plasticity, a phenomenon linked with the discovery that gene expression may depend on environmental conditions.

II. Researching Race: The Lingering Ethical And Theoretical Framework Conflict

Concerns about the reification of race in research are not abating in the post genomic era. Critics worry that improvements in human biotechnologies simply increased the risk of fallacious attributions of a biological reality to a problematic concept that has ambiguous meanings. Already in the pre-genomic era, some researchers complained about the ways in which statistical methods such as factor analysis[4] were being used to reify, for example, IQ in race research. The issue was not that factor analysis was impotent in proving the validity of the IQ construct in race research. The point of contention was

[4] Factor analysis refers to a statistical method which takes a number of correlated variables and seeks to discover if the observed variables can be explained in terms of a smaller number of unobserved variables (see Gould, 1996, for an in-depth discussion in connection with the issue of the reification of race).

rather that the mathematical validity of the like constructs were cashed out in an unwarranted realist framework (Gould, 1996). In the post genomic era, criticisms of reification in race research unite disciplines ranging from the biological sciences and the health sciences to the social sciences and the humanities (Gannett, 2004; Duster, 2005; Smedley & Smedley, 2005; Shields et al., 2005; Cooper, 2005; Bibbins-Domingo & Fernandez, 2007; Bolnick, 2008; Oubré, 2011). Post genomic era critics of the reification of race take aim at what the sociologist Troy Duster called the «molecular reinscription of race» (Duster, 2006) which in the main leads to attributing a «misplaced genetic concreteness» (Duster, 2005, p. 1051) to the concept of race.[5]

Yet like their pre-genomic predecessors, post-genomic era critics of the reification of the concept of race do not deny that biotechnological, statistical and computational methods increasingly yield crucial and actionable biomedical information about human population differences. Rather, they challenge potentially harmful policy recommendations of race research and the realist ontological status accorded to race in the biological and biomedical

[5] Duster's notion of "misplaced genetic concreteness" is an appropriation of Whitehead's definition of the fallacy of reification as a "fallacy of misplaced concreteness", with concreteness inferred from abstract logical considerations (Whitehead, 1925b, p. 51). Below, we re-appropriate this Whitehead-Duster's notion of "concreteness vs. abstraction" to redefine the fallacy of the reification of race in general terms. In fact, Whitehead's definition of this fallacy occurred in part in the context of the philosophical revival of criticisms of the conceptual legacy of 17th Century science. Key to the dispute was a charge pressed by the French philosopher Henri Bergson against the philosophy of nature stemming particularly from Newtonian mechanics. Bergson claimed in various essays that the great success of physics was accompanied by an inevitable distorted representation of nature. The distortion consisted of the intellectual "objectivation" or localization of things in space as mere inert material beings. His target was particularly the mechanistic explanation of life, free will and consciousness made possible, according to him, by the triumph of the theoretical framework of classical mechanics. He mounted pointed attacks against the "spatialisation" of time conceived as reversible and a material object (receptacle) divisible into parts (Bergson, 1960, 1998, 2004). The resurgence of this debate over the modern scientific conception of nature led by Bergson and his followers like Édouard Le Roy prompted reactions by scholars such as the French mathematician and physicist Henri Poincaré (Poincaré, 2001, pp. 315–53). It is in this context that Whitehead, for one, wrote: «[...] I agree with Bergson in his protest: but I do not agree that such distortion is a vice necessary to the intellectual apprehension of nature. I shall in subsequent lectures endeavour to show that this spatialisation is the expression of more concrete facts under the guise of very abstract logical constructions. There is an error; but it is merely the accidental error of mistaking the abstract for the concrete. It is an example of what I will call the "Fallacy of Misplaced Concreteness". This fallacy is the occasion of great confusion in philosophy. It is not necessary for the intellect to fall into the trap, though in this example there has been a very general tendency to do so». (Whitehead, 1925b, pp. 50–1; see also Whitehead, 1925a; Whitehead, 1929)

sciences (Stevens, 2003; Duster, 2005; Ossorio & Duster, 2005). Consider for the moment the pre-genomic example of sickle cell anemia. The understanding of the causal mechanism of sickle cell trait, the HbS mutation due to selective pressures created by malaria infected environments, had a major impact on biomedical research and on fundamental implications for evolutionary biology in the mid-twentieth century. Indeed, the publication of "Sickle Cell Anemia, a Molecular Disease" in 1949 by Linus Pauling and his colleagues at Caltech set the stage for molecular medicine (Pauling, Itano,et al.,1949). As Pauling himself later noted, «sickle cell anemia is the first disease to have been called molecular disease» (Pauling, 1968, p. 268). The discovery of the molecular basis of this epidemiological condition raised hopes for the emergence of the era of molecular treatments. The concept of a molecular medicine arose partly in this optimistic climate. «Molecular medicine may, in one sense, be said», Pauling wrote, «to have originated in 1949» (Pauling, 1968, p. 268; see also Braun, 2002; Fullwiley, 2008; Swensen et al., 2010).

Yet Pauling, the chemistry Nobel Prize Award Winner, who clearly knew the difference between sickle cell carrier status and disease status and who also knew that, because sickle cell anemia is an autosome recessive disease, a child of two heterozygote parents has only 25% chance of being homozygote and therefore of developing the disease, wrote:

> I have suggested that there should be tattooed [sic] on the forehead of every young person a symbol showing a possession of sickle cell gene or whatever other similar gene, such as the gene for phenylketonuria, that he has been found to possess in single dose. If this were done, two people carrying the same seriously defective gene in single dose would recognize the situation at first sight, and would refrain from falling in love with one another. It is my opinion that legislation along this line, compulsory testing for defective genes before marriage, and some form of public or semi-public display of this possession, should be adopted (Pauling, 1968, p. 269).

Pauling explained his stance on the issue by appropriating the following words of his colleague Emile Zuckerkandl: «The probability of twenty five percent of giving birth to a grossly defective child is too great to allow a combination of ignorance and free enterprise in love to take care of the matter» (Pauling, 1968, pp. 269–70). The controversy that ensued from Pauling's public policy recommendation contributed very little to ameliorating public perceptions of this disease (Markel, 1997). However, controversial public health policies

need not derive only from genetic models of disease.[6] For instance, it has recently been suggested that children of obese parents be placed under child protective services with the possibility of removal from the home by the government because obesogenic environments such as low physical activity and high calorie diets contribute to obesity. The researchers, at Harvard University and Children's Hospital Boston, justified their recommendation on the ground that severe pediatric obesity may be a form of child abuse and that governmental intervention may be in the child's best interest (Murtagh & Ludwig, 2011).

Beside the continuing ethical and policy considerations, sickle cell anemia has all along generated in race debate great interest for at least three reasons. First, although Mendelian genetics was superseded by molecular genetics, sickle cell anemia is often viewed as having supplied a paradigmatic explanation of disease *tout court*. It provided the so-called genetic model for disease in which environmental factors are negligible in the actual disease causation because disease is reduced to a molecular structure. Second, sickle cell anemia continues to be perceived by many as a racial disease. It seemed to have offered a racial disease model or, for critics, a model for the racialization of diseases. Third, not only are human races believed to have group-specific diseases but they are also considered biologically real. As such, they are divergent evolutionary groups. Thus human races are not construed as evolutionary divergent groups simply because the emergence of diseases such as sickle cell anemia is explicable by evolutionary mechanisms. They are considered evolutionary divergent groups because they are thought to reach a degree of genetic differentiation that appears to represent distinct evolutionary branches on the tree of life. Otherwise put, they are said to be approaching, to borrow Dobzhansky's words (Dobzhansky, 2008, p. 285), a «degree of existential concreteness» such that, in light of evolutionary biology, they behave like «independent actors in the drama of life». In philosophical terms, race is understood as a biological category because it refers to different evolutionary or natural kinds of humans.[7]

[6] Nonetheless, the lingering impact of the reification of race, stemming from the successful scientific elucidation of the molecular mechanisms of sickle cell anemia, still requires scrutiny (Royal, et al., 2011).

[7] For the applicability of the term "natural kind" to biological kinds particularly in the debate over race, see Hacking, 2005; Kitcher, 2007; Root, 2010; Maglo, 2010; Maglo, 2011.

To repeat, in the debate over the existence of biological human races, a human population is not said to be a biological race simply because a genetic mutation or a trait (advantageous or not) distinguishes it from other such human populations. After all, different haplotypes[8] explain sickle cell anemia in different countries such as Benin, Cameroun and Senegal. Moreover, the HbF level associated with the respective genetic mechanisms causing sickle cell anemia varies among these populations (Green et al., 1993). So we can, thanks to molecular genetic laboratory techniques, tell patients in one country from those in another country. Furthermore, while sickle cell anemia is not prevalent in some sub-Saharan African populations, one finds sickle cell anemia in European countries like Greece (see Maglo, 2010). But the reification of race masks these facts in various ways. Accordingly, we here define the fallacy of reification of race in general, by appropriating Whitehead-Duster's and Dobzhansky's[9] conceptions of the objective reality of a notion, as a «misplaced existential concreteness of a biological kind in the evolutionary history of life».

Avoiding race reification in biomedical research requires a determination of the methodological, theoretical and ethical conditions for an appropriate use of the concept of "race". Without such a careful and systematic rethinking of the ontological status of this concept and the adherence to stringent ethical guidelines, race-based biomedical research may not avoid causing harms. Thus some philosophers have recently advanced a non-realist conceptual framework for the putative biomedical functionality of this double-edge-sword concept (Hacking, 2005; Kitcher, 2007; Root, 2010; Maglo, 2011; Maglo, 2010). Roughly speaking, the emerging philosophical trend distinguishes natural or evolutionary kinds from instrumental kinds (also sometimes called interactive or pragmatic kinds). While evolutionary kinds are biological natural taxa that map evolutionary relationships, instrumental kinds are mere pragmatic groupings that reflect human practical interests and serve as tools for solving problems. As practical problem-solving tools, their use depends only on their explanatory value in a study. That is, they do not have *a priori* any epistemic privilege in research compared to other variables like socioeconomic status (SES), occupation, diet, etc. (See section III below for empirical evidence.)

[8] The term haplotype refers to a group of genetic variants (a set or bloc of single-nucleotide polymorphisms) which are typically inherited as a single unit.

[9] For more details about Dobzhansky's views, see Maglo, 2011.

The instrumentalist conceptual framework appears to capture the ways in which race has been used in epidemiological research since, at the very least, Darwin's 1871 essay in the *Descent of Man* (Darwin, 2004) was published. In fact, one explanation of the putative utility of race in biomedical research is that risk factors influencing disease causation are often unmeshed with population history (social and biological). Because race is a handy taxonomical label for population substructure below the species level, racial partitions may sometimes help probe environmental and biological factors that influence subpopulation health.[10] The question that remains to be answered concerns how racial groups are to be determined. In the biological sciences, some experts simply refer to breeding populations as races, the so-called geographic races. But in recent years the focus seems to have shifted to continental genetic clusters or ancestry. In biomedical research studies, the dominant practice is to use categories of self-identified race,[11] although methods such as molecular genetic correlates of race determination are also currently used. In medical settings, others' assessments of a subject's race, his/her mother's race are important as well (Root, 2010). The practice of collecting data along racial lines is encouraged by the National Institutes of Health (NIH) which requires racial categorization for all studies which receive NIH monies (National Institutes of Health, 2001). The contentious issue is that some have argued that self-identified race corresponds to genetic clusters, and thus that race defined at a social level picks out a biological reality (Risch et al., 2002; Burchard et al., 2003). But as suggested above, the possibility of determining continental genetic ancestry does not mean that genetic ancestral groups are divergent evolutionary kinds. Moreover, the determination of continental

[10] This is not scientifically unusual. That population history affects population health is something humans share in common with nonhuman animals. The heated theoretical dispute has however always been about the fact that the degree of genetic variation between subpopulations within the human species is smaller than the degree of subpopulation differentiation in many nonhuman animal species. While this, by no means, negates the use of animal models, it at first glance seems to negate the philosophical wisdom of applying the same concept, namely race, to different degrees of differentiation across species. But the problem of asymmetric application of a notion to deferring degrees of differentiation is not specific to the concept of race. For instance, the concept of species is not squarely applied to the same degree of differentiation across genera. Moreover, there is more than one species concept. This suggests that our philosophy of biological kinds needs to be grounded in a case-based epistemology (Maglo, 2012).

[11] The term self-identified race refers to how an individual perceives and reports his/her own ancestry or membership in a given racial group. In the US for example, one may identify herself or himself as "White", "Black", "Asian", "Native American", "Hispanic", etc.

genetic clusters is hotly disputed both from theoretical and empirical perspectives (Maglo, 2011).

This epistemic issue is compounded by ethical ones. As we move forward to the age of personalized medicine, individual members of subpopulations that are well researched are more likely to find therapies for their health conditions than are individuals in poorly studied subpopulations. Otherwise put, in the age of individualized medicine, health equality will require even more so research equality across subpopulations.[12] Continental ancestry is certainly a useful tool in biomedical research. Paradoxically however, a focus on continental group or race may mask the subtle ways in which gene expression and risk factors affect local populations or actual breeding units (see Table 1, Section III). To alleviate this problem, some researchers have recently advanced the idea of divisionary levels and of ethno-genetic layering to emphasize the necessity to go beyond "continental race" in research study design while at the same time taking into account ancestry (Maglo, 2010; Jackson, 2004). According to this approach, it is legitimate to study and compare populations according to their substructure level.[13] Yet we cannot generalize the result of such a study to a higher level of substructure by mere use of racial terminologies without running the risk of reification and of causing harms (Maglo, 2010). We will return to this issue in more details in Section IV of this paper. It is sufficient to have shown here the extent to which epistemic and ethical considerations are unmeshed in this debate and to have motivated our cross-continental comparative empirical study. Indeed, as we shall now see, this empirical investigation sheds light on problematic realistic assumptions about race in biomedical research particularly when one studies common and complex traits such as obesity.

[12] Yet more than 90% of all genome wide association studies, for example, are currently conducted on populations of European descent. It is already well known that population substructure can be a confounding factor in research.

[13] A continental population, as explained above, is made up with many genetically distinguishable subpopulations. For example, Sicilians may be considered genetically a subset of the Italian population. Thus the LCT gene controlling for lactose metabolism differentiates the Italian population into various regional subsets of populations. Yet the Italian population as a whole is genetically a subset of the Southern European population, and the LCT gene, among many other genetic variants, allows distinguishing for example Southern from Northern Europeans. But European populations, taken all together, constitute just a subset of the continental population called "Eurasian" in genetic studies (for more detail, see Maglo, 2010; 2011).

III. Race, Obesity Research And Continental Populations

Obesity is an interesting example of how "race" may be useful to consider clinically but at the same time is fraught with reification potential. In the United States, it has long been recognized that individuals of African descent have on average greater body mass index (wt (kg)/ht $(m)^2$) than individuals of European descent (Williamson, 1993). In fact, the most recent NHANES survey found that the age adjusted prevalence of obesity in adults was 44.1 and 32.4 in African Americans and European Americans respectively (Flegal et al., 2010). These differences are greatest in females as 49.6% and 33.0% of African American and European American women respectively are obese while 37.3 and 31.9% of African American and European American men respectively are obese. These differences have also been magnified with the rapid escalation of the obesity epidemic. As shown in the figure based on data from the US health trend, differences between African Americans and European Americans were less in the 1970s, but by the 2000s have increased significantly. Furthermore, there is a similar difference between demographic groups and sex in pediatric cohorts (aged 12–19) where the prevalence of obesity (BMI ≥ 95th percentile of the CDC age sex adjusted standard) is 15.4 and 25.4 for European American and African American girls, respectively, and 19.1 and 18.5 for European American and African American boys, respectively (Ogden et al., 2006). As in adult populations that vary along demographic characteristics, the disparity in obesity rates among youths of varying demographic groups and across gender seems to be increasing through time (Molaison et al., 2010; Freedman et al., 2006).

Figure 1: Trends in Obesity (BMI ≥ 30) Prevalence among US Adults by Ancestry (EA – European American, AA, African American) and Sex

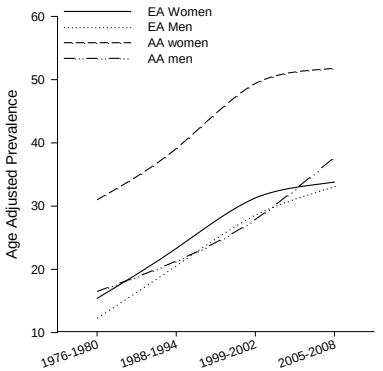
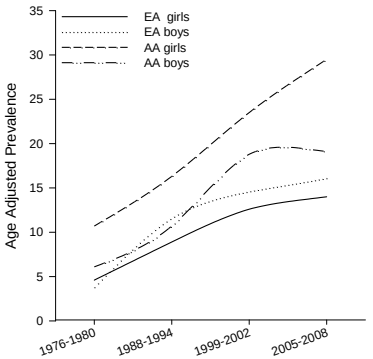


Figure 2: Trends in Obesity (CDC BMI % ≥ 95) among US Children by Ancestry (EA – European American, AA, African American) and Sex



These differences in prevalence are important clinically because obesity is known to be a risk factor for many cardiovascular disease risk factors including hypertension, type 2 diabetes, and dyslipidemia (Cossrow & Falkner, 2004). There are physiologic reasons for this relationship. For example, blood pressure is controlled in large part by the sympathetic nervous system, which when activated raises blood pressure. However, obesity is associated with increased sympathetic nervous system activity (Mancia et al., 2007; Grassi, 2006). For type 2 diabetes and dyslipidemia, it is thought that obesity creates a pro-inflammatory state that reduces insulin sensitivity and free fatty acid metabolism which can ultimately lead to type 2 diabetes and dyslipidemia (Heilbronn & Campbell, 2008; Steinberg, 2007).

However, there is also population level variation in the risk of these obesity related co-morbidities. As all three co-morbidities are associated with obesity, one might expect that African Americans would have a higher prevalence of these three particular co-morbidities given the increased prevalence of obesity. But, this is not true. While African Americans have higher rates of both hypertension (Dwivedi & Beevers, 2009) and type 2 diabetes (Carter, Pugh, &Monterrosa, 1996) than European Americans, they actually have lower rates of dyslipidemia (Sumner, 2009). There have been some treatment recommendations for hypertension based on race, but these recommendations are controversial (Izzo & Zion, 2011). Indeed, some of the differences in outcomes may be due more to cultural differences than genetic differences (Izzo & Zion, 2011; Scisney-Matlock et al., 2009). On the other hand,

knowledge of population variation may be very important in the treatment of dyslipidemia because dietary interventions to reduce a specific type of lipoprotein are less effective in African Americans than in European Americans (Furtado et al., 2010). Further, the impact of obesity on type 2 diabetes varies according to population. For the same BMI, African Americans actually have a lower risk of type 2 diabetes than do European Americans. This difference has been attributed to differences in fat distribution (Taylor et al., 2010), with European Americans having more central adiposity compared with African Americans (Camhi et al., 2011).

Many factors influence obesity and its related co-morbidities. The rates in obesity have increased over the past three decades due to changes in lifestyle (increased intake of high calorie foods plus decreased physical activity, otherwise known as the obesegenic environment). In the United States, low socioeconomic status is a major predictor of obesity and since African Americans have lower socioeconomic status on average (Kahng, 2010), much of the group differences may be due to cultural/environmental factors. There also may be cultural differences in the perception and recognition of obesity. For example in the United States, African Americans are significantly more likely to self-report obesity than are European Americans (Sivalingam et al., 2011). Yet, African Americans are not a homogeneous group but a subpopulation whose members sometimes show diverse ancestral paths. Indeed, one African American may trace his/her roots to West Africa and to Northern Europe, while another may trace his/her ancestry to West Africa and the Americas, and another to Southern Africa and Asia, etc.

Thus, to understand population level variation in the risk of obesity, it is important to study obesity rates across different continental regions. We have chosen here to focus on the African Continent. To look at the prevalence of obesity in Africa, we performed a PubMed[14] search on the terms "obesity", "prevalence", and "Africa" with publication dates from January 2006 – October 2011 limiting the results to humans. Four hundred and twenty four articles were identified. Table 1 provides an overview of the articles in which obesity prevalence was reported. The table is divided into obesity prevalence rates for adults and children. But in each age group there was a high degree of variability in prevalence estimates. A few studies examined the impact of urban

[14]PubMed (http://www.ncbi.nlm.nih.gov/pubmed) is a free database on life and biomedical sciences. It includes the MEDLINE database and is maintained by the United States Library of Medicine.

versus rural living on the obesity prevalence and these studies found much greater rates of obesity in the urban compared to the rural settings. There was a single study (de Onis, Blossner, &Borghi, 2010) that examined the prevalence regionally (East, West, North, South, and Middle Africa[15]). This study focused on pre-school children and found variability by region, with South Africa exhibiting the highest rates of obesity.

Table 1: Prevalence of Obesity in Some African Populations

Region	Location	Prevalence			Study
		Adults	Children (0–5)	Children (6–18)	
Western Africa	Nigeria	8.8–12.5	5.2	0.3–5.7	(Olatunbosun, Kaufman, &Bella, 2011; Senbanjo & Oshikoya, 2010; Omuemu & Omuemu, 2010; Adegoke et al., 2009; Adedoyin et al., 2009; Senbanjo & Adejuyigbe, 2007; Ben-Bassey, Oduwole, &Ogundipe, 2007)
	Cameroon	11.1			(Kengne et al., 2007)
	Senegal	8.3 –29.0		9.3	(Faye et al., 2011; Fontbonne et al., 2011; Macia, Duboz, &Gueye, 2010; Ziraba, Fotso, &Ochako, 2009)
	Ghana	16.0–35.0			(Ziraba, Fotso, &Ochako, 2009)
	Burkina	4.0–28.0			(Ouedraogo et al., 2008; Ziraba, Fotso, &Ochako, 2009)
	Niger-Urban	7.0–35.0			(Ziraba, Fotso, &Ochako, 2009)
	Benin	18.0			(Sodjinou et al., 2008)
	Regional		6.4		(de Onis, Blossner, &Borghi, 2010)

[15]"Middle Africa" is not a term commonly used in studies on Africa, and "Uganda" (see Table 1) is usually considered an East African country. However, our interest here is in the variability of obesity incidence rates.

Eastern	Kenya	5.1–38.0	3.8		(Mathenge, Foster, &Kuper, 2010; Gewa, 2010; Ziraba, Fotso, &Ochako, 2009; Christensen et al., 2008)
	Tanzania	12.0–32.0		5.9	(Mosha & Fungo, 2010; Ziraba, Fotso, &Ochako, 2009)
	Malawi	12.0–23.0			(Ziraba, Fotso, &Ochako, 2009)
	Regional		6.7		(de Onis, Blossner, &Borghi, 2010)
Northern	Sudan			9.7–10.5	(Nagwa et al., 2011; Salman, Kirk, &Deboer, 2010)
	Tunisia	12.2		4.3–5.7	(Boukthir et al., 2011; Aounallah-Skhiri et al., 2011; Kamoun et al., 2008; Blouza-Chabchoub et al., 2006)
	Morocco	29.9			(El Rhazi et al., 2011)
	Regional		17.0		(de Onis, Blossner, &Borghi, 2010)
Southern	Botswana	9.5			(Letamo, 2011)
	Mozambique	6.8			(Gomes et al., 2010)
	Regional		7.6	3.3–4.0	(de Onis, Blossner, &Borghi, 2010; Armstrong, Lambert, &Lambert, 2011; Reddy et al., 2009; Armstrong et al., 2006)
Middle	Uganda	10.4			(Baalwa et al., 2010)
	Regional		8.7		(de Onis, Blossner, &Borghi, 2010)

These empirical results have various theoretical implications for race research. They shed light on the limitations of the continental race concept in biomedicine and on the necessity to provide conceptual, methodological and ethical guidelines for race research. Though one cannot rule out genetic factors in the understanding of common and complex phenotypic traits such as obesity, the apparent dependency of the latter on environmental conditions and the population variation that ensues deserve serious theoretical scrutiny.

IV. Obesity, Ancestry And The Philosophical Implications Of Phenotypic Plasticity

Our empirical findings reveal a great variability in obesity incidence rates when one considers for example age and sex categories (see Fig. 1 & 2). But they also show the same variability between subpopulations regardless of continent of origin. Briefly, in spite of the difference between groups, there is 1) a significant variation around the population mean; and 2) risk factors associated with obesity vary even among populations sharing the same continental ancestry. These results indeed have many theoretical implications ranging from the stability of continental clusters to the ephemeral nature of developmental kinds. The variability observed here highlights the necessity to reengineer constantly population-thinking in biomedicine in order to avoid erroneous attribution of reality to mean differences in race research. We call here this potential form of erroneous attribution of reality to race "reification by mean-thinking".

a) Individual Variation and Reification by Mean-Thinking

As mentioned above, our empirical study shows for example that although obesity is more prevalent among African Americans than European Americans, many African populations have lower incidence rates than European Americans (see Table 1). Moreover, among African populations themselves, obesity incidence varies according to regions and occupations, with higher rates observed in urban areas as compared to rural areas. The latter information suggests that higher economic status may be a predictor of obesity in some African countries while lower economic status may best predict obesity in the US. But leaving aside for now the issue of explanatory variables, other studies of obesity co-morbidities such as hypertension have reached results similar to ours. For instance, while African Americans have higher hypertension rates than European Americans, comparative studies between populations of African descent and of European descent showed that the lowest incidence rates of hypertension are found in Africa and the highest in Europe (Cooper et al., 2005). Other studies suggested that hypertension incidence varies among African immigrants with length of stay in the US (Borrell et al., 2008). Thus environmental factors and population history play an important role in disease onsets and health outcomes.

Nevertheless, taking into account population history in biomedical research does not imply that all individual members of a population equally share risk factors. Group differences are usually differences in the mean. As Ernest Mayr recently put it, «In a Darwinian population, there is a great variation around the mean. This variation has reality, while the mean value is simply an abstraction» (Mayr, 2002, p. 91). Mayr's observation is relevant not only to systematic zoology but to biomedical research as well. To be sure, the "mean" is a crucial statistic that informs and helps us collect valuable information about populations under study. However, the mean as such lacks concrete reality. Reification occurs when we lose sight of this phenomenon and attribute an objective independent reality to race. What we call here reification by mean-thinking consists of mistakenly attributing, in race research, a concrete natural basis to mean differences while disregarding individual variation. Otherwise put, this form of reification refers to a process whereby one substitutes mean-thinking to population-thinking in biological and biomedical research. Reification by mean-thinking, as an implicit negation of individual variation, has the potential to adversely affect medical practice and to undermine our endeavor to achieve personalized medicine. That said, our study also sheds light on an interconnected form of reification that pertains to the stability of continental clusters.

b) Reification by Cluster Stability Rule Violation

In section III, we showed that continental subpopulations do not necessarily share the same clinical priorities with respect to obesity and its co-morbidities (see Table 1). This raises questions about the explanatory value of the continental race concept in biomedical research since a subpopulation from one continent may cluster at the phenotypic level with subpopulations on other continents. That is, membership in continental racial groups is not necessarily stable with respect to epidemiological conditions (Cooper et al., 2005). Otherwise put, the continental race concept cannot be considered a regular predictive tool. Yet researchers frequently label biomedical differences among Americans as "racial" differences even if they have not compared the American populations to populations in continents associated with their primary geographic regions of origin. To account for findings similar to the differences in obesity rates we observed here between African Americans and European Americans, some researchers may simply state that there are "racial"

differences between "blacks" and "whites". But if the terms "blacks" and "whites" refer to the populations categorized in the US census as "blacks" and "whites" or to continental populations defined in genetic studies, then that interpretation of the results will be, if not simply false, at the very least misleading. In effect, unless there are obviously objective scientific reasons to extrapolate from a subpopulation study to a continental population, researchers violate, by their use of racial terminologies what has been called the cluster stability rule. This rule states the following:

> It is legitimate in rational scientific practice to target a subset of a given continental population in research and clinical trials, but researchers who aim to generalize their findings (or those of other studies) to all the members of the continental cluster are obliged, by the membership stability burden of proof, to provide in their study designs tests for the stability of the cluster (Maglo, 2010, p. 366–7).

Cluster instability derives in part from the fact that a continental genetic grouping may not necessarily be the genotypic kind of interest in drug response or disease etiology of a given subpopulation and from the fact that phenotypic kinds do not necessarily correspond to genotypic kinds. Actually, with recent advances in biotechnology, we can now meaningfully subdivide our species into various genetic groups. Yet risk factors are not necessarily shared equally by members of even relatively "homogeneous" groups, if there is any such group. The point is even more obvious with so-called continental groups or races which encompass numerous breeding populations. Because gene expression may differ among subpopulations even when a causal variant, that is a variant that directly impacts a phenotype of interest, is common among them, the utility of genetic ancestry, rather than licensing sweeping inference about continental race, requires that particular attention be paid to smaller breeding units (see Table 1). Indeed, we may liken the condition of human breeding populations to that of many nonhuman animals. Take for example the field of mouse genetics. There are currently over 13,700 strains of mice (www.informatics.jax.org). Mice could be viewed as having many breeding populations. However, when genes have been knocked out of mice to determine the effect of a gene on a phenotype, the result can vary dramatically from no effect to lethal, depending on the background strain. These strains are housed in similar environmental conditions, suggesting that the difference in phenotype is likely due to underlying genetic differences between the strains.

Yet phenotypic variation may also occur despite genotypic similarity. Bluntly put, genotypic and phenotypic partitions do not always correspond for the same population.

The point we are trying to drive home is that the membership stability burden of proof requires that observations of a breeding population within one continent not be generalized to the whole continental group unless the researcher provides appropriate scientific supporting evidence for the generalizability of the observations. In fact, reification by cluster stability rule violation is the process whereby observed characteristics of an insufficient number of breeding populations within one continent are implicitly generalized without scientific warrant to the whole continental population by mere use of racial labels. Be that as it may, the phenomenon of cluster instability points itself to another potential form of reification which concerns the epistemic status of environmental and molecular factors in biomedical explanatory models.

c) Reification by Molecular Reductionism

We emphasized above the fact that differences in genetic endowment may phenotypically differentiate breeding populations living under the same environmental conditions. Yet, we have also suggested in section III that a phenotypic kind, say the kind "obese" or the kind "hypertensive," may be ephemeral and that gene expression may be conditioned by environmental factors. Phenotypic plasticity, or the fact that the effect of a gene may vary with changes in the environment, may result in developmental kinds that do not necessarily match continental genetic kinds. Thus genotypic kinds and phenotypic kinds may cross classify the same population or individual. On the one hand, genetic ancestry may be a useful tool with which to probe environmental factors. On the other hand, differences in environmental conditions may create epidemiological and clinical disparities even among genetically similar populations.[16] There is in fact a scientific hypothesis known as the "thrifty phenotype hypothesis" which posits that early maternal nutrition of fetuses influences the occurrence of chronic diseases in later life (Hales &

[16] It is worth noting that since Darwin human developmental kinds are not considered good candidates for biological human races either because the traits used for the taxonomy are deemed trivial (Darwin, 2004) or because the groups are not discrete (Keita & Kittles, 1997) or because the differences are ephemeral (Kitcher, 2007; Gannett, 2010).

Barker, 1992; Barker, 1997; Wells, 2007). Otherwise put, many chronic diseases are not reducible to molecular structures. Whatever may become of the fate of the thrifty phenotype hypothesis, it is clear that not all diseases are explainable in the same manner that sickle cell disease can be explained (see section II above).

In fact, aside from some Mendelian disorders caused by highly penetrant single genes,[17] causal genetic models taken alone appear incomplete just as indeterminist environmentalist accounts seem insufficient.[18] The challenge then is how to avoid molecular reductionism by incorporating non-genetic factors in biomedical explanatory models. There is no agreement among philosophers about what the term reductionism means in general.[19] So we here simply define molecular reductionism, at least in so far as race research is concerned, as the view that behavioral patterns, disease states and therapeutic responses are amenable to molecular structures in such a way that molecular genetic factors ultimately provide the only relevant explanation of the variation in trait or treatment outcome associated with groups. Reification by molecular reductionism then consists of reductively positing molecular genetics, in Richard Cooper's words, as the *Deus ex Machina* in race research (Cooper, 2005; see also Oubré, 2011). Put differently, reification by molecular reductionism in race research is the process whereby a researcher supplies a causal genetic explanation of group phenotypic differences that does not integrate environmental effects affecting the process of differentiation.

Nevertheless, we are not suggesting that continental genetic ancestry is useless in biomedical research. The membership instability evidenced by the findings of our obesity study does not exclude molecular mechanisms as

[17]An example of such a disorder caused by a single gene with high penetrance is Huntington disease. It is an autosome dominant disease. The offspring has a 50% chance of inheriting the gene if one of the parents posses the gene. The individual has a 100% chance of developing the disease in adulthood.

[18]For more information about gene-centered and pluralistic explanations in philosophy of biology, see (Giere, 2006; Longino, 2006; Waters, 2006).

[19] Philosophers continue to debate various forms of reduction including inter-theoretical reduction (Nagel, 1961), functional reduction (Kim, 2005) and mechanistic reduction (Bickle, 2003; Bechtel, 2009). It is not our goal here to review and discuss the philosophical literature about reductionism. For our purpose, it is enough to note that the claim that biological and biomedical research aims at discovering the molecular basis of traits and the molecular mechanism of treatment response is unproblematic. In fact, it is a mere restatement of a standard and major scientific goal. What we are objecting to here is the exclusivism of the reductionist thesis, or what has become the dogmatization of molecular explanation in race research.

relevant explanatory factors. Hence, it does not preclude differential effects of genetic ancestry. But that constitutes the membership stability burden of proof, that is, the scientific proof that continental genetic ancestry explains obesity prevalence among heterogeneous developmental kinds within a continental group whose members are exposed to differing environmental pressures. In a word, the requirement to incorporate non-genetic factors into the explanation of complex traits does not diminish appreciation of the importance of molecular factors in biomedical and biological explanation in race research. Furthermore, genomic and epidemiological studies have shown that molecular mechanisms affecting group differences – creating thus population substructures within our species – either are rare and limited to some populations, or are common and usually cut across continental clusters and social groups called human races (Maglo, 2010). That is, 1) continental or racial groups are not discrete, and 2) statistically significant group differences in the occurrence or effects of molecular mechanisms may not necessarily justify the attribution of reality to race in biomedicine.

d) Reification by Analysis of Variance (ANOVA) Epistemic Naturalization

The dispute in race research is not only over the within-continent variability of phenotypes and the ephemeral nature of human developmental kinds. As mentioned in Section II, post-genomic criticisms of the reification of race are not limited to the health sciences, the social sciences and the humanities only. Concerns about race reification are also raised about phylogenomic classifications which seek to map evolutionary relationships between groups based on the study of their genomes. Partly because genetic profiles within our species show smooth gradients across continental regions, critics of the continental race concept argue that continental genetic partitions, though potentially useful in research, reflect sampling and statistical artifacts rather than evolutionary breaks between human populations. According to this line of argumentation, race reification consists of interpreting continental ancestry (a merely useful phylogenomic artifact) as evidence for natural evolutionary groupings having regular predictive values in biomedicine. Concerns about the reification of continental genetic ancestry continue to spark thorny disputes over the interpretations of the results of computational and statistical methods such as "Structure" and "analysis of variance" (Long & Kittles, 2003; Bolnick,

2008; Maglo, 2011). The implication of the non-existence of biologically real human races from a phylogenomic perspective for our discussion is that statistical and computational differences associated with continental genetic ancestry in biomedical research do not license the attribution of a biological basis to race. The biomedical utility of continental genetic ancestry cannot be construed as a proof for the biological reality of race simply because utility or statistical significance by itself alone does not entail reality (Maglo, 2010). Thus we dubbed here the erroneous inference from utility to reality based merely on the notion of statistical significance "reification by analysis of variance (ANOVA) epistemic naturalization."

The identification of this form of race reification fallacy suggests that some of the arguments in this debate implicitly originate in the philosophy of statistics. Yet from the philosophy of statistics perspective, the theoretical quarrel over the interpretation of the results of statistical methods is anything but new. Roughly, the dispute in the philosophy of statistics is whether conclusions obtained by statistical methods which require the specification of a significance level[20] yield, philosophically speaking, "truth" or simply useful and actionable instrumental information about populations under study. In this respect, Neyman and Pearson, the founders of the statistical method of hypothesis testing, wrote:

> We are inclined to think that as far as a particular hypothesis is concerned, no test based upon the theory of probability can by itself provide any valuable evidence of truth or falsehood of a hypothesis [...] But we may look at the purpose of tests from another viewpoint. Without hoping to know whether each separate hypothesis is true or false, we may search for rules to govern our behavior with regard to them, in following which we insure that, in the long run of experience, we shall not often be wrong (Neyman & Perarson, 1933, pp. 290–291).

Thus an instrumentalist conception of scientific research findings does not seem to be foreign to the philosophy of mathematical statistics. In the instrumentalist view, the epistemic aim of statistical methods is less to discover the "truth" of the natural world than to provide actionable information and rules that can govern our decision making.[21] That is, we may well be justified in

[20] The challenge with statistics is that it is based on probability, such that the conclusions are based on whether the data could have resulted from chance (α).

[21] According to Steven Goodman (Goodman, 1999, p. 998), what Neyman and Pearson were suggesting was that «We must abandon our ability to measure evidence, or judge truth, in an

holding that a) statically accurate observations about human populations reflect not necessarily "truths" about the observed populations and that b) statically accurate substructure partitions do not necessarily correspond to naturally independent realities in the actual world. Put in the context of our discussion, we may thus accept that continental genetic ancestry has the potential to yield actionable scientific information about human populations without having to accept that human races are biologically natural realities. Reification of race thus occurs when we naturalize population substructures within our species on the ground that our results are statistically valid and clinically useful. It is this naturalistic interpretation of statistical outcomes in the study of human population substructure that we call "epistemic naturalization" by means of analysis of variance, or simply "ANOVA epistemic naturalization". The fallacy of reification stems from the unfounded belief that validity (or utility) necessarily entails reality. We use the term ANOVA as a catching term for formal approaches to human population substructure. So reification by ANOVA epistemic naturalization refers to a process whereby statistically actionable information about continental genetic ancestry is predicated on natural processes of differentiation between groups construed as divergent evolutionary kinds of humans.

V. Conclusion

We identified and discussed above four potential forms of reification of the concept of race in research including mean-thinking, cluster stability rule violation, molecular reductionism, and ANOVA epistemic naturalization. We illustrated the processes by which these types of reification can occur with an empirical study of obesity prevalence in various continental subpopulations. We did not find in our study evidence of continent-based clinical priorities shared exclusively by subpopulations in one continent with respect to obesity incidence. Our results are similar to findings about obesity co-morbidities such as hypertension. Moreover, we showed that neither the evolutionary mechanism involved in the molecular account of sickle cell trait nor the

individual experiment. In practice, this meant reporting only whether or not the results were statistically significant and acting in accordance with that verdict ... Hypothesis tests are equivalent to a system of justice that is not concerned with which individual defendant is guilty or innocent ... but tries to control the overall number of incorrect verdicts».

phenotypic variability of complex diseases such as obesity and its co-morbidities licenses the attribution of a biological reality to race in biomedicine. Consequently, we suggested that, in order to avoid the fallacy of the reification of race, researchers need to pay particular attention, in their study designs and scientific publications or reports, to the methodological and philosophical implications of phenotypic plasticity, a phenomenon which in turn reflects variability in gene expression in response to differing environmental conditions. But we stressed equally the imperative of being methodologically alert to the fact that, for therapeutic response and disease causation, the genotypic partition of interest may not necessarily be continental "phylogenomic" clusters.

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