Producing Obesity and Cardiovascular Disease in Preventive Constellations - populations, biosociality, race

Working Paper

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Abstract
On the basis of an analysis of the biomedical literature on obesity and the metabolic syndrome, this paper outlines four discursive formations rooted in distinct epistemic communities, revealing different understandings of obesity and cardiovascular risk. The rationales behind these parallel understandings are briefly sketched from a science studies perspective. The main focus lies on the consequences of these different modes of production for the interactions between medical and social practice, prevention regimes and different forms of corporeality, sociality and selfhood. The conclusions outline an empirical approach combining the perspectives of science and technology studies and social anthropology, which may be suitable to study some of the complexities involved in observing dynamic constellations in a medical context.

Introduction
Picture the 2005 annual meeting of the German Obesity Society ¹, which in many ways epitomises the difficulties today’s medical community has in making sense of overweight: an opening plenary session reminds all participants of the seriousness of the disease, of its epidemic proportions, of the great suffering of those afflicted and of the major threat its co-morbidities pose to individuals as well as the nation’s health and social se-

¹ see www.dag.de for details
curity systems. The large crowd of several hundred participants is enthused and makes its way to four different session rooms offering parallel presentations of the latest research findings. Most scientific conferences probably follow a similar procedure, so there would not be anything interesting about this one if it were not for a remarkable participant distribution. At the risk of simplifying, two types of sessions can be easily distinguished. Type one, some of them sponsored by a pharmaceutical company, attracts a crowd of largely male, senior (clinical) researchers from genetics, molecular and cell biology and biochemistry. The presentations in English deal with the basic research end of obesity. The role of the endocannabinoid system or particular receptor structures take centre stage, while the possibilities for drug based interventions or the genomic perspectives on the disease are debated by a rather elite subgroup of a small audience. Clinical relevance features as a future possibility at the end of a long road of solving complex research questions. For the time being, all data from clinical trials and drug based interventions show more or less the same picture: a rapid decrease in weight for about six months followed by a short period of stagnation that leads into weight gain so that the majority of study participants are at least back to normal weight within two years. Most speakers conclude that drug based therapies, which utilise the latest research findings, are of utmost importance but need to go hand-in-hand with the usual lifestyle interventions. These interventions are taken for granted somewhat unworthy of further attention.

The session rooms are of medium size and only about a third of the seats are taken. One cannot help wondering to which room the majority of the crowd from the opening sessions has ventured. A return to the plenary room answers the question. Two thirds of the overall attendance, mainly women, have gathered in this session. To them, overweight is clearly a lifestyle issue that needs to be tackled head on in the field. The presentations in German are not concerned with the genetics of receptors. Overweight is about people that weigh too much and need to sort their lives out in order to lose some of it. Basic biological research is not treated with disrespect – it is simply irrelevant, in the same way that lifestyle issues were acknowledged amongst the molecular crowd but ignored nevertheless. Most attendees are practitioners with a background in food/nutrition, a few health psychologists or sport scientists are mixed in. The presentations focus on different interventions, dietary and exercise regimes that are being tried and evaluated with medium sized groups often recruited locally. A faint sense of romanticism and social engineering hangs in the air as television and computer games are condemned and the great outdoors is summoned to rebuild a transcendental roof for those destabilised by late
modernity. Nevertheless, the curves illustrating the lifestyle intervention outcomes take similar shapes to the ones shown next door - weight gain after two years at the latest. This is being terribly unfair to what seemed to be a useful gathering of researchers and practitioners, the vast majority of whom understand the multiple facets of obesity better than most. Yet there is some truth in the observation that a complex issue such as obesity and cardiovascular risk lies perpendicular to conventional thinking within the medical community. As a consequence, parallel discursive formations form within relatively distinct epistemic communities, each of which explains the phenomenon differently. Yet the fact that the average weight continues to increase throughout the Western World reveals all too clearly the formations’ most obvious shortcoming.

The following sections briefly outline four of these discursive formations before discussing in detail some of the less obvious effects, which are beginning to emerge from this constellation. The conclusions tentatively sketch an empirical approach suitable to investigate such constellations, combining perspectives from social anthropology and science and technology studies, and argue for a different style of interdisciplinary research so far without an institutional basis at least in Germany.

**Obesity in four discursive formations**

**Overweight**

In general practice, overweight is usually interpreted as the result of a positive energy balance in everyday life. A diet rich in fats and sugars, an overall caloric surplus and a sedentary lifestyle are seen as the primary causes of an energy surplus stored largely in the form of fat. Consequently, treatment focuses on weight reduction through a reduced calorie diet and increased exercise. Drug based interventions or surgery are rarely considered in the absence of further anamnestic information and for those not clinically obese.

Physicians, and indeed most untrained eyes as well, will be able to recognise overweight simply by looking at someone, i.e. on the basis of an estimation of anthropometric parameters. Obvious as this may seem, the range of measurements that exists to describe body shape in an accurate and clinically relevant manner is astonishing, has escaped standardisation so far and continues to be the subject of debate. Difficulties range from fundamental conceptual questions over disputes concerning equipment to very real practical issues of measuring very large waist circumferences without undue error or indeed embarrassment for everyone involved. Until recently, many studies have relied on body mass index (BMI in kg/m²), where commonly a BMI >25 is considered overweight, >30 obese and >40 clinically obese. This rather crude estimate of body fat content
suffices with respect to the direct effects of obesity, which relate primarily to mechanical effects, such as an increased wear and tear on joints, tendons and muscles \[8\]. Apart from mechanical problems, the increased burden on physiological parameters over time can lead to an increased risk of organ damage. Further deleterious effects pertain to problems with skin folds \[3\] as well as psychological parameters \[7, 8\].

**Metabolic Syndrome**

Most general practitioners will not consider the direct effects of obesity as the major issue but focus on the increased risk of atherosclerosis, type two diabetes mellitus (NIDDM) and, subsequently, cardiovascular diseases (CVD). Large population studies, such as those on the Framingham cohort \[9\], show obesity to act as an independent risk factor for CVD rivalling age and smoking in importance \[10, 11\]. Yet statistical correlations based on epidemiological data do not reveal causality at the level of physiology. Hence, whether obesity marks a starting point for a deleterious development toward CVD or merely a symptom of an entirely different pathological process remains unclear.

It is largely uncontested that obesity forms part of a complex web of physiological parameters primarily related to the lipid metabolism but increasingly also including (pro)inflammatory, endocrine \[12\] and even viral \[13\] parameters. Already in 1923, Kyrin had speculated about the link between weight, hypertension and lipid metabolism \[14\], while in 1947, Vague began to think about different types of obesity \[15\]. Yet it was not until the 1980s \[ii\] that visceral obesity was discovered. This specific form of obesity is characterised by fat deposits centrally distributed around the trunk rather than subcutaneously \[5, 17, 18\]. Importantly, these visceral fat cells could be shown to be metabolically active primarily via endocrine signals and a range of protein factors, so called adipokines \[19\]. White adipose tissue has since come to be understood as an endocrine organ \[20, 21\].

The metabolic relevance of visceral fat combined with the statistical associations between metabolic parameters and body shape rapidly led to the birth of the **metabolic syndrome**. The metabolic syndrome denotes a set of physiological parameters that have a statistically significant tendency to co-occur in an individual and significantly increase the risk of CVD and NIDDM. Until the summer of 2005, at least five major international organisations have developed their own specific set of criteria and thresholds to diagnose the metabolic syndrome \[22\]. Most commonly used in medical research until 2005 have been the definitions of the World Health Organisation (WHO) \[23\] and the US American Adult Treatment Panel III of the National Cholesterol Education Programme (ATP III) \[24\]. The

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\[ii\] Early hypotheses on the endocrine functions of adipocytes emerge in research on mice in the 1960s with Björntorp’s early work \[16\]. Pinpointing a precise starting point for this line of thinking goes beyond this paper yet would be an interesting investigation from an history of science perspective.
latter defines the metabolic syndrome as involving any three or more of the following criteria:

- Central/abdominal obesity as measured by waist circumference: • > 40 inches (102 cm); • > 35 inches (88 cm)
- Fasting triglycerides: • 150 mg/dL
- HDL cholesterol: • < 40 mg/dL; • < 50 mg/dL
- Blood pressure: • 130/85 mm Hg
- Fasting glucose: • 110 mg/dL

As this paper is being written (2005/6), controversy has arisen over the thresholds for waist circumference, blood pressure and fasting glucose. For all three parameters, the new thresholds have been lowered. In addition, the International Diabetes Foundation has made waist circumference a mandatory criterion and adds two of the other four, reflecting, as they argue, the overwhelming evidence that visceral obesity has the most prominent role in CVD aetiology [22].

Currently, the metabolic syndrome acts as a suite of diagnostic criteria or a “set of risk factors” (American Diabetes Association) largely independent of a unifying physiological basis. It defines subclinical thresholds for a set of parameters, i.e. thresholds that if reached for individual parameters do not necessarily warrant intervention but do so if they co-occur. Similar to the Framingham score, the syndrome also serves as a tool encouraging practitioners to check for a broader set of interconnected risk factors [25].

Myriads of studies throughout the 1990s have focused on the aetiology of the metabolic syndrome as well as possible correlations with behavioural patterns and comorbidities. Pre-, peri- and neonatal programming [26-28] and the syndrome in adolescents [29-34], adult risk factors such as diet and inactivity [34-39], psychosocial risk factors [40-44], prevalence in and susceptibility of different (migrant and minority) populations [45-49], the genetics and heritability of the syndrome [50-52], further physiological parameters that may act as diagnostic criteria [10, 53], the implications of the nervous system in food storage and control of appetite [54-57] as well as different treatment options particularly in connection with NIDDM [2, 58-61] have all received significant attention.

**Insulin Resistance**

The metabolic syndrome operates as a diagnostic set of criteria in clinical practice independent of an underlying pathophysiology holding together the different criteria. Yet not least the fact, that its utility compared with conventional risk profiling methods such as Framingham scores [9] is being debated [62, 63], promotes basic research trying to find an underlying mechanism which might be able to explain the statistical pattern.
Currently, insulin resistance and associated compensatory hyperinsulinaemia receives strong backing in the research literature\[6, 52, 60, 62, 64-71\]. The original idea linking insulin resistance with coronary heart disease was published in 1988\[64\]. Up-dated in 2004/5, the basic concept remains the same\[62, 72\]:

“In insulin resistance varies by about 600% in apparently healthy individuals. So there are people who will tend to have resistance to insulin action. They will secrete lots of insulin and they may have some minor degree of glucose intolerance but no frank diabetes but they are likely at increased risk to have essential hypertension and to have a dyslipidaemia characterised by a high triglyceride and a low HDL [high-density lipoprotein cholesterol] concentration. [This cluster represents an] increased risk of coronary heart disease.”

(G. Reaven, NIH lecture, 3rd of November 2004, Bethesda, US)

The disease outcomes associated with insulin resistance largely overlap with the metabolic syndrome, i.e. CVD, type 2 diabetes, essential hypertension, but also include polycystic ovary syndrome\[73\], fatty liver disease\[74\], certain forms of cancer\[75\] and sleep apnea\[76\]. Instead of measuring insulin levels directly, which is a costly procedure, the ratio of triglycerides and high density lipoprotein cholesterol (TG:HDL ratio) has so far proved to be the most practicable, sensitive and specific marker\[69\] and is being suggested as a new diagnostic criterion replacing the ATP III criteria for the metabolic syndrome. As insulin resistance lies on a continuous spectrum, it is epidemiological data suggesting that the 25-30% most insulin resistant people in any given population may carry an additional health risk high enough to warrant intervention. According to Reaven, about half of the 600% variance in insulin resistance is explained by differences in obesity (25%) and physical fitness (25%), the other half appears to be “familial, ethnic and likely genetic”\[72\].
**Allostatic Load**

An area of research which emerged in the early 1990s adds another level of explanation: insulin resistance may be caused by neuroendocrine perturbations\(^7\). Relatively recent work by a number of research groups in the US and Europe suggests that inadequate behavioural and physiologic responses to persistent stressors of different kinds may carry hormonal, neuronal and immune signatures that manifest themselves over time in the form of, *inter alia*, visceral obesity, the metabolic syndrome, insulin resistance and CVD\(^{18,77-91}\). The advancement of this work has been aided by a better understanding of the neuroendocrine system and, methodologically, by the availability of techniques to easily measure one of the signature substances of the stress response, cortisol, in saliva\(^{92}\). This technique has enabled a series of studies determining specific physiological stress responses almost in real time\(^{83,93-95}\). The conceptual basis for this work is depicted in Figure 1 (redrawn from McEwen\(^{96}\)) and can be traced to Walter B. Cannon’s work on stress, which is based on the concept of *homeostasis* defined as “the coordinated physiological process which maintains most of the steady states in the organism.”\(^{97}\)

Cannon argued that stress above a critical threshold could produce a breaking strain that would result in a failure to maintain homeostasis. After executing a fight or flight response, which would lower the stress level below a critical threshold, the body would return to homeostasis. Hans Selye extended this idea and proposed the ‘general adaptation syndrome’ (GAS), which added the notion of a single, non-specific, stereotypic response to
any kind of demand on the body\textsuperscript{iii}. Stress became “the state that manifests itself by the GAS”. More recent work has added complexity to the notion of a non-specific response suggesting that part of the response pattern is determined by the stimulus as well as the capability to adapt to the situation referred to as coping\textsuperscript{iv}. To do justice to the dynamic nature of homeostasis, the term allostasis was introduced, denoting the body’s ability to increase or decrease vital functions to a new state\textsuperscript{v}. On this basis, McEwen developed the notion of allostatic load, which he defined as the impact of wear and tear on a number of bodily functions that result from “chronic over-activity or under-activity of the allostatic system”\textsuperscript{vi, vii}. Failure to maintain allostasis, i.e. to accommodate chronic stressors, either due to the persistent nature of the stressors or due to an inadequate response, increases allostatic load and, subsequently, increases the risk of a range of chronic diseases, including insulin resistance, metabolic syndrome, atherosclerosis and CVD.

Most of the early work had conceptualised stressors narrowly in terms of physical challenges such as digesting food or standing up. For instance, carefully controlled studies of postprandial disposal of a specified glucose load stratified people according to their digestive system’s ability to deal with the stressor glucose in the bloodstream\textsuperscript{vii}. Yet recent work has begun to link psychosocial stressors to HPA response. On the basis of data from the Whitehall II study, which in a prospective longitudinal design included 10,308 participants sampled across twelve non-industrial grades of the British civil service, significant correlations have been found between control on the job and job strain (high psychological demand and lack of control) and CVD\textsuperscript{44, 103-107}. Though the correlation between socio-economic status and health had been known for some time, the concept of the allostatic load combined with the HPA/cortisol mechanism offered for the first time a convincing pathway\textsuperscript{v} making the effect of psychosocial factors on (patho)physiology plausible within biomedical thinking\textsuperscript{v}.

\textsuperscript{iii} Though Selye cites Cannon has his greatest mentor, Cannon never accepted the idea of an unspecific response. Selye was prepared to add the GAS to a range of specific responses, which Cannon had proposed, but did not change Cannon’s mind on the matter\textsuperscript{vii}.

\textsuperscript{iv} Allan Young, to whom I’m very grateful for pointing out this line of research and sharing his thoughts with me, has introduced the term ‘allostatic body’ to refer to the amalgamation of meta-narrative and mechanism, which enabled an entire line of research. See also his recent work on the modular mind for similar processes in psychiatry\textsuperscript{108}.

\textsuperscript{v} Of course, up until the late 19\textsuperscript{th} century, psychosocial factors, e.g. a man’s constitution, had been central to understanding disease. Yet within biomedical thinking, i.e. a way of mechanistically modelling disease in terms of necessary causes with high specificity at the level of physiology, psychosocial factors have struggled to gain acceptance\textsuperscript{109}.
Discussion

*Understanding Lifestyle*

These four discursive formations are neither clearly separate entities nor do they simply mark a temporal succession. They exist in parallel, with some notions contradicting each other, such as insulin resistance and metabolic syndrome diagnostic criteria, others lending each other support. These different emphases are of interest in themselves and deserving of closer attention from an history of science and a science studies perspective. However, this paper is concerned with some of the effects these different modes of producing obesity may have outside their native epistemic cultures \[110\], i.e. outside their immediate research context.

Producing obesity with tape measures and scales emphasises issues of proportion, mechanical wear and organ damage. Hence it only considers therapeutic interventions for the seriously obese. Those simply a little overweight are largely accepted as *normal*. People of BMI >30 face a range of problems in everyday life from practical issues of capability and fitness to stigmatisation and social exclusion. Many studies have shown that the obese are disadvantaged in many occupational and social settings and can subsequently suffer from reduced self-esteem, depressed states and anxieties. These are effects that occur largely independent of the medical classification as obese, because they are visible to the untrained eye and tangible in everyday practice. Initially, producing obesity as an issue of proportion hence does little to change selfhood and patterns of social interaction. Moral judgments and sanctions are primarily related to aesthetics and a perceived lack of willpower more than a process of medical classification.

However, a medical diagnosis is inextricably linked with the need to lose weight, i.e. exercise more and reduce calorie intake. In many cases, the medical advice to lose weight is matched by a personal wish to do something about the situation to feel better about oneself and escape stigmatisation. Yet the vast majority of patients fails to accomplish any sustained weight loss, initiating a positive feedback loop of increasing frustration, weight gain and social isolation. National statistics from most industrialised countries show a similar pattern underlying the increase in average BMI: those already obese gain weight, while those considered borderline or normal by medical standards lose or hold their weight. While most general practitioners as individuals are aware that their diagnoses and appeals to lifestyle change have at best no effect in the vast majority of cases, the system as a whole, i.e. the apparatus comprising medical and health economic knowledge as a socio-cultural practice as well as subjects in their morality and materiality, does not *understand* individuality and social interaction. The notion of *understanding* as a thoroughly modern concept implies making transparent and being able to change and recon-
Medical practice understands social life as lifestyle, i.e. as additive *homo oeconomicus*, and fails to intervene successfully. This suggests that the medical understanding of social life misses important aspects with consequences for individual patients and patient populations.

**Patient Production in the Age of Biosociality**

The metabolic syndrome produces a different kind of overweight designating it a risk factor for cardiovascular diseases. In the medical sciences, this shift has predominantly been driven by epidemiology and its notion of the *risk score*. Risk score means that particular physiological states lead to particular disease outcomes with a given likelihood for a specified population. Physiological parameters, such as body weight or cholesterol levels, are thus transformed into measures of risk. The metabolic syndrome hence produces patients *at risk*. The epidemiological notion of the risk score implies the existence of a score to which no additional case of cardiovascular disease can be attributed: a physiology carrying zero risk; possible and to a degree sensible within epidemiology but with consequences once taken out of its original context. Zero risk, in the sense of an ideal physiological state, is, of course, a statistical illusion. Yet it implicitly underlies many debates on thresholds and risk reduction. The medical *understanding* of risk reduction as a linear process relates to the reliance on a limited number of input variables into epidemiological modelling. Linear risk reduction towards a safe physiology fails to take into account, that the metabolic syndrome’s way of producing patients causes different effects dependent on the person’s original state. Those seriously overweight are aware of their problems, while those of BMI 25 or 26 do not perceive themselves as at risk, as ill and as patients until diagnosed. It thus makes a significant difference, whether a person with a BMI of 26 or of 33 is diagnosed with the metabolic syndrome – a significant difference, which is not medically understood.

This problematic reveals a more fundamental discrepancy: the metabolic syndrome is valid only as a collective entity not as an individual disease. Only at the level of a population is it possible to assign a certain risk of cardiovascular disease to physiological criteria. While those criteria, applied to the individual, *seem* to enable an assessment of individual risk, they in fact short-circuit physiological thinking with the help of statistics *pretending* that a causal mechanism exists, which could enable the assessment of individual risk on the basis of individual physiology. Despite this worrisome inference, the metabolic syndrome continues to follow its inherent epidemiological logic of linear risk re-

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*Most metabolic parameters start to have pathological impacts when lowered beyond a certain point. Calculating a point of zero additional risk, i.e. the vertex of a graph plotting physiology against risk score, may thus inform debates on these turning points, e.g. in the case of overweight and *anorexia nervosa*.*
duction. As a consequence, the latest criteria from the International Diabetes Foundation applied to a region in Norway diagnose over 90% of those aged 50 and over as having the metabolic syndrome, i.e. at risk from cardiovascular disease, initiating discussions about the purpose of a diagnostic category which pathologises almost the entire population. From an epidemiological and a clinical perspective, using a set of interconnected sub-clinical thresholds makes sense, because, firstly, it encourages physicians to cast the diagnostic net wider than they might have done otherwise; secondly, an early diagnosis, even if not of a disease but of an at risk state, enables more effective pre-symptomatic treatment. The call for lower criteria thresholds hence follows a medical understanding of a healthy population.

Yet trying to explain this drive towards ever tighter criteria with advances in medical knowledge only, would be partly missing the point. A more convincing picture emerges when viewing medical knowledge as part of a broader context including medical research and practice, health economics, social policy and regulation. Economic arguments about the stability of the social welfare state form allegiances with medical knowledge and visions of the good life to construct the emerging apparatus of health promotion and primary prevention with the aim to produce a population, which is able to afford itself. This apparatus employs different strategies reaching from a prevention fee on new televisions and cars, to health insurance bonus schemes and state subsidised entrance fees in fitness centres.

The notion of the apparatus enables a Foucauldian perspective on these transformations, which conceptualises power/knowledge as entirely relational, as relying on temporarily stable complementary constellations. Of interest is not so much the fact in itself that the state might be charging a fee for new cars in the not too distant future. Rather, the conditions that allow particular constellations between state, private sector, population and individuals to stabilise take centre stage. Foucault’s distinction between regulatory and disciplinary power, as relying on populations and individuals respectively, needs to be revisited in this context as the preventive constellation is able to draw on both kinds of power at the same time. State sanctions that aim to create a healthier population top-down, such as through changing the quality of meals in work settings or increasing taxes on particular products, co-exist with disciplinary measures, such as health insurance bonus schemes, which rely on changes in selfhood to accomplish a healthier population. It is within this emerging preventive constellation that the metabolic syndrome produces a different kind of overweight. The subtext of moral judgments is changing from aesthet-

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**vii** The different knowledges and implicit notions of subjectivity, sociality and society and how they combine to arrive at such measures have not been investigated in any empirical detail so far.
ics and individual lack of willpower to a responsibility for the common good and the stability of the social welfare state. Again, this transformation relies on a complementary constellation: increasingly somatic forms of individuality [119] create a type of selfhood, which is responsive to corporeal (self)regulation, while at the same time biosociality emerges [120]. Biosociality in this context does not simply refer to people finding to each other because they share being overweight and wanting to get rid of it. Instead, nature being modelled on culture as practice points to changing patterns of solidarity and shifts in moral practice as unintentional side effects of an emerging preventive constellation.

**Genetics in Different Guises**

The discursive formation described above as insulin resistance produces yet another kind of obesity. Already framed in terms of cardiovascular risk rather than a stand-alone health issue, obesity becomes a cause of insulin resistance, which in turn leads to the metabolic syndrome and, ultimately, to cardiovascular and other diseases. According to Reaven, 25% of the variance in insulin resistance in apparently healthy individuals is explained by obesity. In some ways, whether obesity is understood within the metabolic syndrome or insulin resistance does not matter all that much as both lead to cardiovascular disease. Diagnostic tools change but a significant degree of overlap exists between those individuals diagnosed as having metabolic syndrome and those having insulin resistance. Though insulin resistance offers an underlying physiology, it is still a concept based on statistics rather than an understanding of aetiology. Potential pathways exist that lead from insulin resistance to the changes in physiology. Yet a comprehensive aetiological model at the level of physiology remains illusive.

The insulin resistance constellation is of great interest because it explicitly incorporates new factors into the search for a predictive understanding of cardiovascular aetiology. Reaven states that roughly half of the variance in insulin resistance in apparently healthy individuals is caused by “familial, ethnic and likely genetic” factors. Much of basic research in biology and medicine, commonly summarised under the label of genomics, is currently directed at understanding the involvement of genes and gene expression in disease aetiology. It is thus of little surprise that a vast range of mutations and polymorphisms are currently being investigated in the context of obesity in order to understand relevant (patho)physiology and to improve drug targeting. Currently, the obesity gene map includes about 300 genes, markers and chromosomal regions on all chromosomes but the y chromosome, all associated with the human obesity phenotype [121]. So far, this research has acquired little direct clinical relevance not least because findings are often contradictory and cannot be replicated. Diagnosis and treatment in everyday medical
practice operate without this kind of specific genomic knowledge, unless one of a few rare monogenic disorders directly or indirectly associated with obesity is involved. As is the case with all complex diseases, individual genes or particular polymorphisms contribute too little to phenotypic outcome to warrant testing or intervention. Nevertheless, genetics enters research via different routes such as hypothesis building as in the case of the thrifty gene hypothesis: “We have evolved, for some bizarre reason, to maintain glucose homeostasis.” [72] Insulin resistance may have been caused by a set of thrifty genes at one point [26, 122, 123], i.e. those with a genetic make-up leading to insulin resistance may have had a selective advantage in years gone by that made them more likely to survive times of hardship. This selective advantage would have turned into a problem in the context of our present relatively sedentary lifestyle, continuous energy (over)supply and long lives. The search for such thrifty genes has proved futile so far yet the evolutionary discourse continues.

The fact that obesity runs in families offers a second point of entry for genetics. There is now strong evidence to suggest that weight is a highly heritable trait with a heritability of about 40%-70% [124]. Yet these data are based on epidemiology rather than an understanding of physiology. They need to be interpreted with care as developmental programming and epigenetic factors may play an equally important role.

The third point of entry is created by the notion of race/ethnicity. The International Diabetes Foundation supported by a large body of research now advocates ethnically sensitive waist circumference measures, i.e. while a white European male’s risk of cardiovascular disease increases from 94cm waist circumference onwards, a South Asian or Chinese male should be aiming for 90cm and a Japanese male for 85cm [125]. Major international medical journals such as The Lancet or the British Medical Journal are currently publishing papers on ethnically sensitive cut-off points [126, 127]. From a social anthropological perspective, this is highly problematic because it reintroduces biological notions of race into scientific and public discourse.

Biological notions of race have been scientifically and politically en vogue at different times throughout the last century and rarely have these been particular happy times. The reification of social differences via scientific practices has been revealed by many, most forcefully in the context of race and medicine probably by Troy Duster [128-130]. Until recently, the fact that intra-group variance exceeds inter-group variance and that polygenic diseases are badly understood, meant that genetic notions of race simply did not make sense. Today, however, the term ethnicity not only reinvigorates the idea of relevant biological differences. It also mixes traditional notions of race with ethnicity and culture to the extent that papers speak of “race-ethnicity-specific[ity]” [131] and the necessity for cul-
culturally sensitive strategies \[^{132}\]. It is important to note that, while ethnicity implies a heritable difference in physiology, all studies on ethnicity rely exclusively on statistical analysis. Based on self-reporting in questionnaires, samples are stratified into various groups, typically including black/African-American, Hispanic/Mexican-American and White.\[^{viii}\] Reaven speaks of Caucasian and non-Caucasian.

Amongst social and cultural anthropologists, ethnicity commonly refers to a sense of socio-cultural belonging. Self-reporting oneself as of a certain ethnicity generally means that one feels as belonging to a particular set of cultural beliefs and practices, as being part of a certain tradition and as contributing to and benefiting from shared social capital. Hence claiming that Caucasians carry the lowest risk of insulin resistance, to many means that those people who partake in the socio-cultural capital of the people of Chechnya and neighbouring countries and regions might be of a particularly robust constitution \[^{72}\].

There is nothing wrong with acknowledging biological differences in a medical context. Skin colour, for instance, tells us something about the risk of malignant melanoma from UV-light. The preventive constellation, however, steers into difficult territory for two reasons: (1) Proclaiming a large, very heterogeneous group of people\[^{ix}\] to be at increased risk from weight gain and CVD lacks medical and ethical justification. An entire group of people is classified at increased risk, i.e. pathologised to a degree, without a convincing concept that might explain the statistical correlations. There is a significant chance that the statistical findings can be explained by biological, psychological or socio-cultural factors that are independent or only indirectly related to a top-down concept of an ethnic group. Yet the experimental and study designs are set up in a way to either show an ethnic correlation or no correlation at all – they reify socially constructed ethnic classifications and, furthermore, employ the same invalid inference as the metabolic syndrome (see pg. 10 above). What is set in motion here, without any real understanding of the consequences, has been termed *looping* by Ian Hacking \[^{133}\]. People become aware of the *at risk* label and start to respond. This in turn will not only affect selfhood and social interaction but is also likely to manifest physiologically, either via behavioural change or pathways such as stress. The changes in physiology will necessitate an adjustment of the original classification, closing Hacking’s loop and further reifying what is clearly negotiable. (2) Not only race-based indicators but most genetic information used in risk communication and prevention widens the gap between diagnosis and prevention. Even if sensible genetic indicators did exist, it would still be unclear how people handle this infor-

\[^{viii}\] The International Diabetes Foundation has introduced Europids, South Asians, Chinese, Japanese, Ethnic South and Central Americans, Sub-Saharan Africans, Eastern Mediterranean and Middle Easter (Arab).

\[^{ix}\] ...a group of people, which defines itself on the basis of shared socio-cultural capital...
formation. Studies in social psychology using hypothetical scenarios have shown that the information, that one’s obesity may have a genetic cause, may well lead to apathy, reduced self-efficacy and disempowerment.

A last point to be discussed in the context of genetics is the somewhat residual category of epigenetics. The intergenerational transfer of genetic information not based on DNA sequence alterations but processes such as methylation and histone modification [134] has begun to destabilise Weismannism, i.e. ‘the doctrine of the continuity of germ and the discontinuity of soma’ [135-97] thus introducing a new kind of genomic plasticity [136] which blurs the boundaries between inheritance and development. The genome may be able to “learn” from its own experiences.

The relevance of these developments for the production of obesity are illustrated by a Swedish study on malnutrition [137] that relates medical data on the health of 287 people, born and grown up in a specific district in Northern Sweden in 1890, 1905 and 1920, with social historical data on the living conditions and the nutritional status of their grandparents’ and parents’ generations. The findings indicate that for the early 19th century male population periods of food scarcity significantly increased the risk of their grandchildren to suffer from diabetes mellitus type two, while periods of surplus during maternal pre-puberty led to protective effects against heart disease. What is beginning to emerge here is an understanding of physiological historicity which extends biographies beyond individual birth and death [138]. For the preventive constellation, this means that intergenerational responsibilities, so far restricted to socio-cultural capital, may extend to include biological substrate – a very different reading of biosocial sustainability. This also raises questions about intergenerational prevention adding another layer of complexity to the interplay between state regulation and selfhood.

Incorporations

The discursive formation allostatic load suggests that it may not only be times of extreme hardship which produce somatic memo effects. Rather, this kind of imprinting may occur from all kinds of different chronic stressors. Using Whitehall II and other data sets (see pg. 7 above), many studies have shown correlations between CVD and social position, stress and inequality [104-107, 139-143], eating and drinking behaviour [56, 91], neighbourhood characteristics such as community structure or community atmosphere [144, 145], dietary restraint [57, 93, 146], social phobia [94], burn out [95] or marital quality [147-152]. While this is not a new phenomenon, it is worth investigating how social and cultural phenomena are incorporated into observational routines, how methods and theoretical perspectives are adapted and how changes in the meta-narratives of biomedical research are produced.
and how they manifest themselves. From a distance, the emerging formation of allostatic load and a growing interest more generally in the integration of different levels of analysis, from molecules via organs to behaviour and the environment [153, 154], rings of the kind of holism advocated by constitutionalists during the early 20th century. Aronowitz’ careful tracing of coronary heart disease illustrates that attempts to incorporate ‘the social’ come up throughout the history of the medical sciences with a certain regularity, yet on the basis of very different theories and methods [109]. Today, many suggest that the self in its social context is set to disappear from medical research as most are moving to a genetic and molecular level to find the causes of disease. A closer look at recent work on CVD suggests that, to the contrary, notions of the self, of behaviour and of social interaction, are firmly built into research on the aetiology of obesity. Theories about genetic, neuroendocrine or cellular causes change rapidly, yet, implicitly or explicitly, all of them depend on notions of the self in its social context; via an evolutionary discourse on social life during the Pleistocene, via allostatic load and coping or via intergenerational imprinting and developmental programming. Rather than vanishing, the self in its social context has become a necessary condition for much of medical research in that it informs hypotheses and theory building.

Conclusions
The opening anecdote from the annual meeting illustrated the difficulties an issue such as obesity can cause, when it proves not to be amenable to being thought and understood along existing medical disciplinary lines. This trend is exacerbated by an increasing disciplinary specialisation, which has as much to do with the progression of theory and method as it has with science management, funding structures and policy. While the preceding paragraphs have tried to show the implicit dependence of specialised medical research on different levels of understanding self in social context, in everyday science as practice, the interdisciplinary transfer of knowledge has become a rare event - certainly between the natural sciences/medicine and the humanities/social sciences. This paper points to two strategies, which might help to ameliorate current conceptual difficulties.

(1) The analysis of an emerging biosociality, of the reification of race and of biolooping, demonstrates a need to relate the study of science as practice closely to its interaction with policy and social practice. Studying dynamic constellations such as the emerging prevention and health promotion regime, needs an empirical approach which symmetrically analyses domains as different as science, policy, regulation and society. We suggest to combine STS and social anthropology as practice-oriented, empirical disciplines and connect the different domains as epistemic
cultures at the level of practice. Not least Hacking’s concept of *biolooping* further suggests the need to base these analyses on a notion of embodied practice, which is able to integrate discourse and materiality. The conceptual and empirical basis is weak for understanding and relating to each other different forms of manipulating soma in selfhood, science and regulation. We have introduced the notion of *somatography* elsewhere [¹³⁸], combining soma and ethnography, to suggest a symmetrical empirical analysis at the interface of STS and social anthropology. This analysis of overweight and cardiovascular risk illustrates a need to employ such approaches to better understand emerging constellations of prevention and health promotion in Europe and North America.

(2) Getting such important studies off the ground, at least in Germany encounters institutional barriers. A lack of truly interdisciplinary teaching means that disciplinary outlooks on fields of research persist in the medical sciences as much as in the social sciences and humanities. Cross-listing courses, co-teaching, placements and other mechanisms designed to develop an inside understanding of different practices and perspectives are virtually non-existent at German universities – certainly not between natural and social sciences. The few postgraduate training courses, such as in bioethics, tend to remain at the level of textual analysis and discourse and largely fail to offer an opportunity to develop truly interdisciplinary research questions, which may lead to a stream of innovative research. The same holds true for postdoctoral research. Outside an institution such as Berlin’s Centre for Advances Studies, everyday commitments as well as departmental and funding barriers keep most scientists away from interdisciplinary research. Mode II science, advocated as a problem-oriented, temporary research community incorporating different perspectives, presents a real opportunity for the interface between medical research, clinical practice, social science and humanities, yet has so far failed to acquire institutional support.
Bibliography


