

Medical modelling of obesity: a transition from action to experience in a 20th century American medical textbook

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Abstract Obesity is now the focus of considerable attention in the medical profession, and many have noted that obesity has been progressively *medicalised*. The subjecting of phenomena to medical explanation, however, has been associated with both the potential to relieve and also to exacerbate the attribution of individual responsibility. In order to understand the ways in which a particular phenomenon, obesity, can be variously conceptualised at different time-points within a medical framework, we conducted a content analysis of a series of medical textbook entries. Using the widely-consulted *Cecil Textbook of Medicine*, we reviewed entries on obesity from 1927 to 2000 and found that throughout this period the text consistently maintains that obesity results from a simple excess of caloric intake over expenditure. Despite the unwavering nature of this basic model, an evolving set of causal factors is superimposed. Early models invoke aberrant individual activities, such as habitual overeating, while later editions drop these factors in favour of genetic and, paradoxically, environmental effects. Obesity shifts in ontological status, as it is transformed from being the product of something that individuals *do* to something that they *experience*. Concurrent with these changes, we find a change in the social appraisal of obesity. In each edition there is a narrative regarding the cost/benefit relationship between obese persons and society, as well as a construction of accountability for obesity as an outcome. Obese individuals are progressively held less responsible for their condition in successive editions of the text. Initially cast as societal parasites, they are later transformed into societal victims. Using these texts and obesity

as a case-example, we demonstrate that medical conceptualisation of a presumably cohesive object of knowledge can undergo transformation quite independently of definitive experimental evidence, with a persistent dialectic between etiological configuration and formulations of social culpability and remediation. We situate our findings with respect to ongoing debates concerning the nature and implications of medicalisation. This case effectively highlights a more general epidemiological tension between an individual level of focus on risk behaviours and a population level of focus that contextualises behaviours within a social and material framework.

Keywords: obesity, overweight, medical model, medicalisation, textbooks, responsibility, agency, lifestyle, structure

Introduction

Obesity, now described as 'epidemic', is currently the focus of considerable attention and concern for medical professionals in the US, the UK and other postindustrial nations. It is most widespread in the US, where researchers estimate that more than half the adult population is overweight or obese (Flegal *et al.* 1998). In the UK, the National Audit Office recently reported that obesity levels in England have tripled in the last 20 years, warning that in 10 years levels could reach those of the US unless millions change their lifestyles (Meikle 2001). In consultation with the World Health Organization, an International Obesity Task Force convened in 1997 to discuss the prevention and management of obesity as a global epidemic (WHO 1997). Alongside such developments, many have noted that obesity as a phenomenon has been progressively *medicalised* (Reissman 1983, Conrad and Schneider 1992, Sobal 1995). Medicalisation refers to the process by which certain behaviours or conditions are defined as medical problems (rather than, for example, as moral or legal problems), and medical intervention becomes the focus of remedy and social control (Reissman 1983, Fox 1988, Conrad and Schneider 1992). Sobal (1995) argues that in this century fatness has shifted from a moral conception of fat as a personal wrong (or failing), to the medicalisation of fat as a sickness. The medical profession and others have made powerful claims over the control of fatness, ranging from defining it as a disease to the application of a wide variety of medical treatments (Sobal 1995).

When a socially deviant status is medicalised, there typically occurs a concomitant change in imputed responsibility (Conrad and Schneider 1992). What was once wilful and sinful, or even criminal, behaviour becomes unwilful behaviour secondary to illness. Under a biomedical as compared to

a moral model, individual responsibility is diminished, and the social response is characterised by a therapeutic rather than punitive cast (Conrad and Schneider 1992), rendering the medical model a potentially more humanitarian approach, though the cast of abnormality can be retained (Becker and Nachtigall 1992). The medical model, however, is also criticised for its scientific reductionism and a theoretical commitment to individualism (Crawford 1980, Reissman 1983, Coreil *et al.* 1985, Travers 1995). Problem and disease are situated in the individual, and the individual human body is taken as the locus of explanation, perception, diagnosis, and intervention (Crawford 1980). Structural conditions such as sociocultural, environmental, and material context are often obscured or ignored in favour of isolated individual factors such as lifestyle and personal behaviours. Social problems thus become individualised (Conrad and Schneider 1992). Medical campaigns promoting healthy lifestyles are frequently subtended by the implicit assumption that behaviours are voluntary and independently modifiable, and systemic constraints are underplayed while emphasis is directed toward individual responsibility (Crawford 1980, Coreil *et al.* 1985). Thus, medicalisation, or the invocation of a biomedical model, has been associated with the potential to relieve as well as exacerbate the attribution of individual responsibility. In this sense, medical claims to jurisdiction over a phenomenon, or simply the application of a medical understanding, can bear variable consequences for the framing of accountability (Lowenberg and Davis 1994).

In this study, we conducted a content analysis of a series of medical textbook entries for obesity, covering a time-span of seven decades during the 20th century, a method used in past studies of medical knowledge (Lawrence and Bendixen 1992, Christakis 1997, Carron *et al.* 1999, Christakis 1999). We used the *Cecil Textbook of Medicine*, which dates back to 1927 and is one of the most prominent and widely-consulted (by both students and practitioners of medicine) medical textbooks in the US. We also considered *Harrison's Principles of Internal Medicine*, another popular US text, but chose *Cecil* because it predates *Harrison's* by three decades. Specific sales and usage figures for this individual text were not publicly available¹. However, some sense of the distribution of this textbook is reflected in medical school library holdings. For example, there are 121 medical schools in the US, and an on-line examination of a random sample of 40 revealed that 53 per cent have at least one edition from each decade since 1930. Furthermore, despite the fact that many of these schools were not established until after WWII, 80 per cent have holdings that date back to 1927 or the 1930s. Lastly, 90 per cent have holdings dating back to at least the 1950s, and 100 per cent have holdings from the 1990s to 2000.

We comprehensively reviewed entries for obesity beginning with the 1st Edition (1927), and concluding with the 21st Edition (2000). In this analysis we cite solely from five editions: 1927, 1947, 1967, 1985 and 2000. These include the first and most recent editions, as well as allowing for the passage of 15 to 20 years and a change in author between each successive edition. In

all editions, obesity is listed as a topic in the table of contents and is granted its own section in the text. The title is always simply 'Obesity'. We concentrate, in particular, on (1) the proposed explanation, and (2) the framing of social accountability for obesity. We thoroughly reviewed each entry in its entirety, targeting all statements or passages that relate either to explanation or accountability, regardless of where they appear in the entry. We do not take these textbooks to capture notions of obesity for an entire profession, and we do not take results from one source to represent general historical shifts in the medical appraisal of obesity. Such broader study has been undertaken by others and is not the objective of our analysis (Schwartz 1986, Levenstein 1993, Sobal 1995, Stearns 1997). Rather, we present our findings as a detailed case-example and exploration of how the medical construction and designation of objects of knowledge, such as obesity, can change quite independently of definitive experimental evidence, even within the same source. Furthermore, we use this example to highlight the presence of a persistent and consistent relationship between etiological configuration and conceptions of social responsibility and culpability. Our findings highlight a more general tension in epidemiological research, that between an individual level of focus on risk behaviours, and a population or structural level of focus that contextualises risk behaviours within a social and material framework.

Our aim is to focus on one source text and conduct an in-depth investigation of how its conceptualisation of obesity, a presumably unambiguous and cohesive object of knowledge, can undergo considerable transformation. Our intention is to bring to the fore the underdetermined and partial nature of each instantiation, highlighting the discursive work that underlies the representation of phenomena such as obesity. The differences between entries might be summarised by noting that they are written by different authors at separate points in time, but each entry is contingent on the social actions and conditions of its production, contingencies that are only grossly conceptualised as a change in author or progression with time. Although this case-example is from the US, we describe more general problematics that occur in the setting of biomedical representation. Our findings with respect to conceptual tensions between individual and context, the ties between explanation and the configuration of remediation, and the partiality of scientific textual production are not limited to the US context and, furthermore, inform our understanding of health foci other than obesity per se.

In each edition, the texts consistently maintain that obesity fundamentally results from a simple excess of calorie intake over expenditure, *i.e.* they employ a basic input/output model of mechanical/economic function and efficiency. We will show, however, that their understanding of the *cause* of this imbalance changes dramatically, with attention shifting from actions to experiences. We will also argue that the movement of the medical gaze from the individual and his or her behaviour to that which transcends this locus is accompanied by a reformulation of issues of will and culpability, along with

changes in the social appraisal of obesity and obese persons. Initially cast as societal parasite, the patient is later transformed into societal victim.

Explanatory models of obesity

1927: 1st Edition

In the 1st Edition, the obesity section opens with a definition: ‘Obesity is a state in which the amount of fat stored in the body is excessive’ (Means 1927: 593). The causal explanation is also oriented towards this notion of imbalance, but explicitly draws our attention to the necessary role of individual behaviour. The following is taken from the *Etiology* section:

The fundamental cause of obesity is a positive energy balance. . . . A comparatively slight disproportion between fuel intake and combustion may, over a period of years, result in a marked grade of obesity. . . . Most persons preserve a constant and normal weight. . . . This is because the normal appetite ordinarily adjusts intake so accurately that it just meets, but does not exceed, the requirements of energy expenditure. When this adjustment loses its delicacy and eating falls under the rule of habit, obesity may develop (594).

While these statements indicate that obesity is predominantly caused by excess on the ‘fuel intake’ side, the *Morbid Physiology* section considers a deficit in ‘combustion’.

The performance of muscular work and the ingestion of food accelerate metabolism; so do emotional disturbances. . . . It is entirely possible that the individual who gains weight readily reacts less intensively to such stimuli. . . . For the most part clinical experience bears out such a hypothesis. Individuals who gain weight readily . . . are usually phlegmatic; they worry less, sleep either longer or more soundly, and when at rest relax more completely than persons of normal or thin types (594–5).

Following this excursion into the role of metabolism, we are reminded again of the problem of food intake in the *Treatment* section:

It should be remembered that most fat persons, though they may not admit it, take delight in eating. Food intake with them has fallen under the head of habit instead of instinct (596).

Although the primary cause of obesity is at one point attributed to excessive ingestion and at another attributed to an inadequate combustion, one essential factor is common to both approaches: obesity is primarily the result of

aberrant individual activity. Whether one eats too much out of habit or does not burn enough calories from sleeping too much, obesity is the result of particular behaviours, and the basic unit of analysis is the individual.

1947: 7th Edition

The 7th Edition entry opens with the same definition. Obesity is still ‘that physical state in which the amount of fat stored in the body is excessive’ (MacBryde 1947: 719). In the *Etiology* section the basic causal model also persists, with emphasis placed on an imbalance of calories: ‘A plethora of calories is the only explanation of obesity’ (719). The *Pathologic Physiology* section considers potential sources for this calorie accumulation: ‘Aberration . . . may result from either physiologic or psychologic disturbances or both’ (719). Physiological factors include endocrine disorders, such as Cushing’s syndrome and hypothyroidism, as well as inactivity following infections such as tuberculosis and poliomyelitis. Concluding this section is a paragraph on hereditary influence:

Obesity occurs much more frequently among the members of certain families than among others. In animals the *hereditary influence* is clearer than among human beings, but even in the latter, evidence points to the inheritance of the tendency to obesity (720–1).

Overall the explanatory power attributed to *physiological* considerations is highly circumscribed. Endocrine disorders and physical impairment do not assist in the explanation of common obesity, and the new addition of genetic factors is framed quite tentatively. In contrast, the section on *Psychologic Factors* frames explanation with much greater certitude and, moreover, appears to account for general obesity.

Some persons enjoy food more than others because of habit or training, eat more than they require and become fat. Some enjoy exercise less and prefer sedentary occupations and will become obese even with an apparently normal diet. Such . . . if only moderate in degree, can hardly be considered pathological, for pleasure in eating and avoidance of exertion are universal human traits. . . .

When obesity is extreme, psychologic influences beyond such relatively normal limits are suggested. . . . These psychologic sensations may become deranged so that the usually automatic balance of the intake and outflow of energy is upset. Pleasure in eating may become a dominant personality trait. . . . Addiction to food, like alcoholism, is often a symptom of psychologic maladjustment (721).

The individual continues as the base unit of analysis, but there is a detectable change in the precise role of the individual. First, it is acknowledged that persons can enjoy food more than others secondary to *training*, not just

idiosyncratic habit. Second, there is recourse to ‘universal human traits’, suggesting that some degree of obesity-prone behaviour is expected as part of the natural course of human experience. Third, the explanation is now expanded to include psychological pathology such as a dominant personality trait or maladjustment. With the introduction of hereditary factors, the patient is also subject to the effects of his or her genes. Thus, the role of the individual is now conceptualised to incorporate elements of behaviour that are increasingly more involuntary in nature.

1967: 12th Edition

In this edition obesity is introduced with the following:

The differentiation of adipose tissue was a triumph of evolution . . . However, the ability to store fat in compact form, of great survival value when food was scarce, has become a handicap in affluent societies where overnutrition and underactivity have upset the ancient balance between caloric supply and demand. The consequence is obesity of epidemic proportions . . . (Albrink 1967: 1164).

After these initial comments, obesity is again defined as an excess of adipose tissue, and etiology is again attributed to an excess of caloric intake over expenditure. Cultural and genetic factors are individual subsections under *Etiology*:

Cultural Factors. First the agricultural, then the industrial, and finally the computer revolution have all served to make food increasingly available and life livable with a minimum of physical exertion. Perhaps the single most important cause of widespread moderate obesity in the United States is the plethora of purified foods of high caloric content but low nutrient value.

Genetic Causes. Obesity, especially in its extreme form, tends to be familial. Obesity in children is much commoner than when neither parent is obese. Anabolic forces appear to be in operation from birth in very heavy people . . . (1164).

There is also discussion of additional sources of obesity such as endocrine and central nervous system factors, but their contribution in humans is framed with uncertainty. For example, with respect to endocrine factors, the text states: ‘Many endocrine and metabolic abnormalities have been reported in very obese persons. Whether they are cause or effect is unknown’ (1164). Finally, psychological factors are addressed:

A distinctive personality type, if it exists, is more likely the result than the cause of obesity. Like most of man’s ills at one time or another, obesity has been blamed on the world’s domineering mothers. The role

of mothers in causing obesity in children can more likely be attributed to their enforcement in early life of patterns of overeating than to the psychological effects of overprotection (1165).

Thus, in 1967, the locus and scope of analysis has shifted from the individual to that which is beyond (and external to) individual and body. In the previous editions, obesity appears under the macro-heading 'Diseases of Metabolism,' which directs attention immediately to the body and its interior. In this edition and the two that follow, it appears under either 'Diseases of Nutrition' or 'Nutritional Diseases', suggesting greater attention to context. The comforts and conveniences of modern affluent societies, with their attendant technologies and patterns of parenting, are now central foci in the causal explanation of obesity. The ability to store fat, in times past a triumphant effector of survival advantage, is now viewed as a 'handicap' in the context of modern society, where humans are assailed with conditions promoting overnutrition and underactivity. Psychological factors are discounted with the claim that a distinctive personality type is more likely a *result* rather than a *cause* of obesity. Lastly, with the brief consideration of genetic factors, it is clear that, despite new attention to context, the individual body has not been dismissed. However, with genetic factors the movement away from individual agency continues, and obesity, overall, has changed from being the result of something that individuals *do*, to being the result of something that individuals *experience*.

A transition away from the realm of deliberate action was emergent in the 7th Edition (1947), but obesity-prone behaviours were described without reference to social or historical context, and individual bodies functioned as an independent site of production and investigation. Now, the analysis has moved from an explanatory field bounded by the independent behaviours to one focused on social organisation and consumerist culture.

1985: 17th Edition

Here, the causal explanation of obesity is again configured within the plight of modernisation, and obesity is again defined as an excess of adipose tissue. Unique to this edition, however, is a qualifying comment that 'it is still not clear whether obesity represents a "disease" or a common clinical manifestation of a group of disorders like anemia or hypertension' (Bierman 1985: 1191). In addition, obesity is described as 'the most common disorder of metabolism in man', giving it a disease-like character if not, in fact, characterising it as a disease (1191). The consideration of obesity as *disease* is novel. Obesity is no longer defined exclusively as an excess of adipose tissue; it may also be defined as a disease. Previously, despite being listed under macro-headings incorporating the word 'disease', obesity was, in the text itself, merely characterised in relation to *other* diseases such as diabetes and atherosclerosis. For example, in the 7th Edition (1947) obesity was described as a *symptom*. The casting of obesity as disease represents yet another instance

of movement into a realm wherein obesity is constituted as the result of processes that patients passively experience.

Following this discussion there is a relatively short section on *Prevalence and Epidemiology*, which contains the following:

Cultural influences and socioeconomic status have a strong influence on the prevalence of obesity. Every social factor studied has been correlated with obesity, and thus there are many determinants. Socioeconomic status . . . shows a particularly strong inverse correlation with obesity among women . . . (1192).

This invocation of 'culture' and socioeconomic status further expands the salience of external social factors. In addition to the overall effects of modernisation, the influence of cultural differences and stratification within and between such environments must now be considered.

The *Etiology* section includes a discussion of adipose (fat) cell changes, but it appears that knowledge of these processes does not assist in the explanation of obesity itself:

No primary biochemical lesion of adipose tissue has ever been firmly documented as a cause of generalized obesity in man. Also, little is known of the etiologic basis for adipose cell hyperplasia (1193).

Genetic factors are also alluded to in the *Etiology* section, but discussion is limited to one simple statement: 'Genetic factors may play a role, but their mechanism remains unknown' (1193). In summary, the 17th (1985) Edition directs our attention to modern society, culture, socioeconomic status, and genetic factors.

2000: 21st Edition

In this edition, as in all previous versions, it is argued with respect to etiology that 'an obese individual may have increased intake, decreased expenditure, or both' (Pi-Sunyer 2000: 1156). Following this introductory statement, the *Etiology* section contains but one subheading, *Genetics vs. Environment*, clearly signaling a newly prominent status for genetics in determining the 'disease' of obesity.

Recent twin and adoption studies indicate that human fatness is under strong genetic influence. From 25 to 35% of the variance in skinfold thickness, body mass index, and relative weight has been attributed to genetic factors. Obesity is a polygenic disease, and its genetic determinants are complex and not yet well described. . . . The studies that have shown this degree of variance describe the genetic influences found in persons living under particular environmental conditions, namely those of Western society. . . . Environment is also clearly

important. . . . The interrelation of genetics to particular environments needs to be further investigated. The combination of the increased availability of low-cost, very palatable, high-energy-density food and a great decrease in physical activity has caused waist girth and weight to rise dramatically (1156–7).

This edition reveals a considerable shift in confidence with respect to the explanatory power of genes. Genetic factors are ascribed strong significance, and it is unnecessary to qualify their contribution. This change in certainty and relative attention is no doubt partially related to the accumulation of experimental results (e.g. the ‘recent twin and adoption studies’). Note, however, that in earlier editions hesitation stemmed from the fact that genetic mechanisms were unknown. In the current edition these mechanisms are presumably still unknown, for there is little discussion of such matters, and it is conceded that ‘. . . genetic determinants are complex and not yet well described’. Curiously, this lack of knowledge no longer impedes confidence in a genetic explanation, consistent with an increasingly hegemonic status for genetics.

Following the *Etiology* section is a new section entitled *The Regulation of Body Weight*. Here, we are appraised of multiple animal models of obesity resulting from single-gene mutations, again reflecting greater investment in genetic investigation. There is an extended discussion of recent developments with respect to the ‘*ob/ob*’ gene and leptin, a protein found both to inhibit food intake and enhance energy expenditure. It is concluded, however, that leptin is of limited explanatory value, for the leptin gene mutation ‘. . . is a very rare condition and is not the cause of obesity in the great majority of people who develop this problem’ (1157). A subsection on *Energy Intake* follows, and it considers factors such as ‘the hedonic aspects of food’ and ‘maladaptive conditioning’, notions which are reminiscent of earlier explanations. They are now, however, listed alongside *cellular* level derangement such as ‘impaired feedback signals registering satiety’ and ‘insensitive brain receptor centers for the feedback signals’. They have attained a scientific behaviourist cast that is devoid of human will and intention.

Thus, the most recent model of obesity accords explanatory power primarily to the impact of heredity and life in Western society. Both of these features demonstrate a basic transformation wherein explanations grounded within the realm of individual action and control are exchanged for those of intersecting forces that an individual experiences without his or her express desire.

Summary

Over seven decades, each edition is initiated with the same basic model to structure the ensuing explanation: obesity is caused by an excess of calories. Despite the unwavering nature of this central notion, in each version a different set of causal factors is superimposed. In building an explanatory framework, the text focuses our attention on, and inspires our confidence in,

a limited yet changing array of causal considerations. The significance of these changes, however, is not limited to the fact that new elements are added and previous elements are deleted. There are shifts in the dimension of explanation. In the ongoing search for etiological factors, the explanatory lens has focused on a host of elements configured quite differently with respect to the obese patient, bearing significant consequences for the ontological status of obesity as these texts shift from individual patient behaviour to that which transcends this behaviour.

In the earliest version it suffices to know simply what an individual does or does not do in order to understand the causes of obesity. In later versions, these behaviours warrant explanation. Stepping back from behaviour to that which precedes or causes behaviour, however, engenders a change in meaning with respect to the object of knowledge; there occurs a necessary reevaluation of the contribution of human agency. Left with a lens focused on the seemingly paradoxical extremes of genetics and the environment, later texts cast both deeper into and further outside of the individual body. We probe deeper into the obese body to look at its genes, while concomitantly looking outside of this body to consider its social environment. In both cases, however, the voluntary is exchanged for the involuntary.

The social appraisal of obesity

1927: 1st Edition

The following is taken from a section labelled *Incidence*:

Obesity is one of the commonest ailments to which the flesh is heir, and is of importance to the individual in proportion to its degree and its association with other diseases. To the community it is of importance in that it may *per se* decrease human efficiency and shorten human life (Means 1927: 593).

Obesity is injurious to persons, but obese persons are in turn detrimental to the community. By decreasing 'human efficiency', obesity presumably interferes with labour productivity. The implied consequence of shortened 'human life' may as well refer to productivity, or it may simply indicate a more nebulous affront to the vitality of social life or the values of the collective. Later in the entry, fat is described as a 'parasite' with respect to the individual, but it is clear that the obese person himself is also considered parasitic with respect to the community.

Aside from the notion that the obese person is accountable to society for *effects* imposed by the body, the obese person is also held responsible for the *status* of the body itself. Personal, individual control is critical to both inappropriate weight gain and successful weight loss. Contrary to later editions where weight loss is viewed as difficult and problematic, in this entry,

treatment is described in terms of a 'reduction cure' (595). Much of the burden for this 'cure', however, rests with the patient:

For success in treatment the patient's co-operation is essential. No one can be satisfactorily reduced who does not wish to be . . . With the physician's help the habit can be gradually broken and the patient be satisfied with an adequate instead of an excessive amount of food (595).

Thus, in 1927, the obese person is not only harmful to society, but also culpable for the state of his or her body.

1947: 7th Edition

It is argued in the *Pathologic Physiology* section that:

When social, business, or sexual desires are unsatisfied, the enjoyment of food often becomes magnified in importance and serves as a substitute. . . . Placid daydreaming, in which desires are imaginatively fulfilled, may take the place of ambitious exertion. . . . In addition, the resultant obesity makes the satisfaction of social and sexual desires less likely and physical exercise more difficult.

. . . the obese state itself . . . may serve as a defense against undesired contacts or activities, for example, to avoid sexual advances of an unloved husband, or to escape work (MacBryde 1947: 721).

These statements expressly locate the obese person within a social context. Moreover, obesity is conceptualised to exist in *tension* with specific elements of social life such as business, sex, marriage, and work. Overall, obesity has negative effects for the social life of the obese person, but the obese person also has undesirable repercussions for society in general. Thus, obese persons continue to be blameworthy for their bodily status, and the obese body is again characterised as an obstacle to social wellbeing and productivity.

1967: 12th Edition

In this edition, modern society enters into the explanatory framework and is ascribed weighty causal significance. While obese persons were previously depicted as a burden on society, society is now depicted as a burden on a great number of its members, causing them to become obese. There is no longer any mention of the loss of human efficiency or productivity, and society does not enter in terms of potential loss or forfeiture. Instead, society re-emerges as a source of harm. The obese individual and society have exchanged places as perpetrator and recipient of harmful effects. Certain aspects of social life are highlighted as problematic sites. The following appears at the end of sections on *Treatment*:

The excess calories that contribute to mass obesity in the Western world are derived mainly from products that the food industry has made too

readily available. Purified fats and carbohydrates, either alone or combined in the form of rich pastries and other delicacies, are the worst offenders (Albrink 1967: 1174).

Central to the problem of purified foods, is the charge that the food industry has made them 'too readily available'. The consumption of purified foods itself can, in principle, be blamed on either the consumer, the producer, or both. Here, blame is directed almost exclusively at the food industry.

This discussion then continues with some suggestions for combating this problem:

... a simple rule to apply would be to reduce the consumption of food stuffs that require manufacturing at some stage of their preparation. The adoption in American homes of such simple austere diets with reservation of rich treats for special occasions would enhance appreciation of the special occasions and do much to eliminate obesity and its toll of vascular disease (1174).

Immediately following these comments is a concluding section entitled *Prevention*, which provides additional suggestions:

Prevention means even further education of our already diet-conscious civilization. Such education might properly start with mothers who are responsible for establishing in their children life-long dietary patterns. Institutions such as the army, that are responsible for feeding large segments of the population have an opportunity not only to establish restrained eating habits but to prevent weight gain in men during their tour of duty. Most important of all, and perhaps least achievable, would be the restraint by the food industry of its promotion of foodstuffs of high caloric but low nutritive value (1174).

All the above suggestions are pitched to the nation as a whole rather than to specific persons. It is in all of *America's homes* that rich foods should be restricted; it is *mothers in general* who are to educate children in dietary moderation; it is at the level of *national institutions* such as the army that proper habits can be established; and finally, it is incumbent upon the *food industry* to show restraint in its promotion of high caloric foods. These solutions demonstrate that individual behaviours are still at stake, but agency and responsibility have moved from the obese, or potentially obese, to the institutions that have the power to influence individuals.

1985: 17th Edition

In this edition, there is an appeal for sympathy towards obese patients, reflecting a more explicit and direct change in sentiment. The following is taken from the last paragraphs of this entry under the heading of *Prevention*:

As with hypertension, most obesity is 'essential' because definable, preventable, and treatable causes can rarely be identified. . . .

Thus it can no longer be assumed that most obesity is simply the result of overeating and that every fat person is an overfed normal one. . . . the obese individual who has experienced multiple failures in weight reduction needs sympathetic attention rather than admonition (Bierman 1985: 1197).

2000: 21st Edition

On a similar note, the 21st and last edition opens with the following paragraph:

Obesity is a frustrating condition for patient and physician alike. Its underlying cause is rarely clear, and its treatment is fraught with difficulty and failure. Management of obesity therefore requires much understanding and persistence (Pi-Sunyer 2000: 1155).

This is in sharp contrast to the 1st Edition (1927) where treatment is characterised in terms of a 'reduction cure', and success is attained with proper desire and willingness on the part of the patient. Thus, despite many additions in the number and variety of treatment options, treatment expectations change from an expectation of cure to an expectation of failure, and these changes are accompanied by a modulation in patient accountability.

In the last edition there also emerges a new concern, one which depicts society as inimical to obese persons in yet a second manner. A new topic appears on the agenda of relevant considerations:

Psychological manifestations

The psychological toll of severe obesity is large. Poor self-image and impaired social relationships are common. Obese individuals are often discriminated against in educational and professional settings, engendering anxiety, anger, and self-doubt. There is no evidence, however, of any particular neurotic or psychotic character in obese individuals. The depression and anxiety seem to be situational rather than endogenous . . . (1160).

Society is not only marked as a setting in which individuals are subject to overnutrition and underactivity, it is also marked as a setting in which the obese are subject to *discrimination*. In earlier editions it is the obese patient who is held responsible for impaired relationships. Now society is charged with discrimination, and the obese patient is given reprieve with respect to charges of creating social dysfunction. Hence, we have moved from early models, which invoke the psychological *causes* of obesity, to contemporary models, which emphasise the psychological *consequences* of obesity.

Summary

In each entry, some notion of social life appears within the discussion. This notion appears in varying forms, ranging from 'community' in the first entry to 'environment' in the last, and it is deemed relevant to very different aspects of obesity, ranging from the consequences of obesity to its causes. Each time society is invoked, obesity is manifestly situated within a social context, and a narrative is constructed with respect to the relationship of obese persons to the world in which he or she lives. We have shown that this narrative and its implications are not consistent. Over seven decades, it changes from one in which the individual is detrimental to society to one in which society is detrimental to the individual.

In addition to the production of this *cost/benefit* relationship between obese persons and society, there is a related, yet separate, construction of *accountability* for the creation and maintenance of the obese body itself. In the earliest editions, the individual is held fully responsible not only for the effects of his or her body on society, but also for the status of the body itself. In the latter half of this century, the obese person is somewhat exonerated, while the surrounding culture and environment are scrutinised and subject to approbation. Moreover, prescriptive considerations and the burden of preventative measures have shifted from patients to institutions to physicians. Overall, individual behaviour has receded as a site of both explanation and accountability.

Discussion

In this study we trace seven decades of thinking on obesity in a major medical textbook, providing a powerful instantiation of how medical objectivity arbitrates, officiates, and produces knowledge in a realm of considerable scientific and social uncertainty. The causes, consequences, and management of human fatness continue to be a realm of considerable debate in both scientific and lay arenas (Kassirer and Angell 1998, Klein 1996, Taubes 1998, Wickelgren 1998, Austin 1999). Of particular note, we demonstrate that despite a relatively stable overall pathogenic process, there can be important shifts in explanation, shifts that do not follow directly from definitive experimental results, shifts that do not reflect a simple, steady progression towards more 'truthful' beliefs.

Medical modelling and individual culpability

The subjection of a behaviour or condition to medical conceptualisation has been associated with both an increased and a decreased attribution of individual responsibility. In our case-example of the construction of obesity in a fixed source over time, we find elements of both tendencies. The earlier editions tend to target individual behaviours and individual accountability,

while the later editions devote more attention to social and environmental context (as well as genetic components), with a concomitant diminution of individual blame and accountability. These results can be viewed as an instantiation of a more general tension in medical and epidemiological research, a tension between population and individual levels of analysis (Pearce 1996), which bears notable overlap and resemblance to sociological debates concerning structure and agency.

The individual level of analysis focuses on individual lifestyle factors and risk behaviours, while the population perspective takes a more structural approach, contextualising individual factors within a social, economic, cultural, and political framework (Pearce 1996). For example, an excess of calories is associated with obesity, but the thrust of medical interrogation and social policy can converge on overconsumption or overproduction, focusing on individual exposures or the social structures that render individuals susceptible to such exposures. Many have argued that the emphasis on individual behavioural modifications, termed 'healthism' (Crawford 1980) or the lifestyle model, has been the reigning paradigm in medical and public health promotion since the 1970–80s (Crawford 1980, Reissman 1983, Coreil *et al.* 1985, Fitzgerald 1994, Travers 1995, Austin 1999). The medical model is thus reproached for obscuring the social structuration of ill health, preventing critique of the existing social order, and reinforcing the privatisation of potential interventions (Crawford 1980). Our findings in *Cecil* of a redirection from an individual to a population level of focus runs contrary to this broader tendency, however, demonstrating that medical framing itself does not preclude attention to context. Nevertheless, the text does not suggest any form of substantive sociopolitical change or rectification.

The recent emphasis on molecular or genetic factors represents yet another level of distinction. While drawing attention away from social structural context and back to the individual body, it nevertheless accounts for a condition in non-voluntaristic terms. This component of our findings more closely typifies classic depictions of the medicalisation process wherein sickness is substituted for badness with a concomitant change in imputed responsibility (Conrad and Schneider 1992). Hubbard and Wald (1997) have argued that current trends towards the geneticisation of chronic conditions hinges on the assumption that there is a hierarchy of causes presided over by the gene, simultaneously exculpating both individuals and society.

Given recent claims that obesity has been medicalised (Reissman 1983, Conrad and Schneider 1992, Sobal 1995), one might ask whether or not there is evidence of increased medicalisation in these texts. This assessment, however, is largely dependent on how medicalisation itself is conceptualised. Strong (1979) forcefully argued over two decades ago that the thesis of 'medical imperialism', the increased medicalisation of the social world, has been exaggerated by social scientists. One of his objections to this thesis is the empirical finding that patients are often critical of the medical services they receive and, for many problems, highly sceptical of the utility of medical

consultation. More recently, Williams and Calnan (1996) have similarly argued, drawing on Giddens's theorisation of social reflexivity and risk management in 'late' modernity, that medical jurisdiction is now limited by substantial ambivalence, scepticism, and disillusionment with scientific medicine amongst a lay populace of increasingly critical and technically-informed agents.

Strong (1979) also argued that in areas supposedly under threat of medicalisation, such as alcoholism, physicians themselves are often sceptical of the value of medical intervention. In his own work on physicians' attitudes towards alcoholism, Strong (1980) found that physicians generally dislike treating alcoholics, preferring to manage problems that are more straightforwardly 'biological', or easily susceptible to abstraction from social context, problems for which they have clear-cut expertise in etiology, diagnosis, and effective treatment. Given that obesity shares many of these features with alcoholism on the physician side, and given the emergence of resistance in the lay populace against the medical management of obesity on the patient side (Klein 1996, Sobal 1999), one might follow Strong's reasoning on the limits of medical imperialism and hypothesise that the medicalisation of obesity is subject to important doctor and patient constraints. A content analysis of medical texts, however, can neither confirm nor disconfirm such a hypothesis; substantiation would require empirical elaboration of the actual sensibilities and behaviours of doctors and patients.

Our findings, however, do inform this issue in another way. Conrad and Schneider (1980) responded to Strong by arguing that he takes an unnecessarily narrow view of medicalisation by focusing on 'what doctors actually control and do' at the level of doctor-patient interaction. They advocate a broader conceptual frame wherein medicalisation can occur on three levels: the conceptual, the institutional, and the doctor-patient interaction levels (Conrad and Schneider 1980, Conrad 1992). Our findings do operate at the level of the *conceptual*, the level of the formulation of ideas, definitions, and explanations. A content analysis, however, cannot speak to how physicians (or non-physicians) actually conceptualise obesity in everyday practice. Nevertheless, one might still ask if there is evidence of increased *conceptual* medicalisation of obesity *within the confines of this textbook*. Despite such delineation of the question, there is still no clear-cut answer, for even at the conceptual level of medicalisation, the answer is further dependent on which factors are taken to be the most salient indicators of such a process. While some would view the invocation of a lifestyle model as evidence of medicalisation (Crawford 1980), others would argue that the lifestyle model is opposite to medicalisation, because it 'turns health into the moral', while medicalisation is properly conceptualised as that which 'turns the moral into the medical' with the proposition of biomedical causes and interventions (Conrad 1992). The invocation of genetic factors and a disease model would thus constitute evidence of medicalisation for the latter definition of this process. As indicated by these various debates, medicalisation is itself the object of claims-making.

Again, these texts are not generalisable to the medical profession as a whole, and medicalisation is a sociocultural process that is not limited to, or necessarily dependent on, the activities of the medical personnel (Conrad 1992). Sobal (1995) has undertaken such broader analysis and concluded that there has indeed been an overall increase in the medicalisation of fatness over the past century with some recent claims towards demedicalisation. These texts do demonstrate, however, that subjection of a condition to medical conceptualisation can entail attention to individual as well as context, or agent as well as structure. Furthermore, they bring to light the fact that emphasis on the individual, the usual depiction of a medical model, can be associated with individual liability as well as reprieve. This suggests that commonly invoked dichotomies such as individual vs. structure, and medical (organic) vs. social (non-organic), oppositions which are often conceived as overlapping, are actually cross-cutting. Strong (1979) and Conrad/Schneider (1980) also debated the consequences of a social, or non-organic, model of health on individual freedoms. Strong argued that a social model of health could entail closer monitoring and policing of behaviour such as people's nutrition or leisure activities. Conrad and Schneider argued, in response, that the dangers of a social model are contingent on *who* controls that model and the supervision of programmes charged with ameliorating the problem. Drawing on our findings, we can, perhaps, clarify, or at least reframe, this debate by proposing that the implication of a social model of health depends, first and foremost, on whether the 'social' denotes emphasis on (and targeting of) individual behaviours or the structuration of such behaviours by socio-political organisation.

Social and historical contours

The transition in etiological focus from individual behaviours to generic environmental determinants represents a process of positive assertion rather than passive observation, and such change must be situated in the context of broader processes of social and cultural organisation. We suggest some *selected and initial* points of inquiry here, as the avenues of relevant exploration are many in this regard, and a comprehensive analysis is beyond the scope of our current study.

At the turn of the century, Western medicine inherited not only a legacy of secularisation, but also the scientific and bureaucratic rationalisation of society with industrial capitalism. Turner (1996) has argued that the project of modernity was critically linked to an ideology of rational, self-controlled mastery over the desires of the labouring body, with religious moral authority transferred to secular institutions such as the medical profession. Drawing on Foucault's (1977) analysis of the systematisation of rational surveillance over the body and the subordination of desire to reason, Turner regards the growth of nutritional management as a rationalisation of conduct and an important technique aimed at improving labour efficiency. In an extensive cultural history of dieting in the United States, Schwartz (1986) has argued

that in the first decades of the 20th century, impelled by movements ranging from Taylor's scientific management in the workplace to domestic and nutritional science in the home, the body is cast in terms of an economy and efficiency to be centrally and willfully regulated. During WWI, Herbert Hoover, as head of the US Food Administration, mounted a propaganda campaign for domestic food conservation with slogans such as 'Food Will Win the War', equating individual excess with treason (Schwartz 1986). During the Depression, Americans were again asked to show restraint in food consumption, this time by Hoover's presidential administration (Schwartz 1986). These notions resonate with early models in *Cecil* that concurrently frame obesity as a function of individual misconduct and as a deterrent to labour productivity and societal well-being.

Similar to Foucault's (1977) description of the emergence of detailed examinations and taxonomies for discipline of the body, Schwartz (1986) describes an early 20th century progression within multiple sectors of the US, ranging from the insurance industry to criminology, wherein bodies are increasingly weighed and charted, with weight functioning as an index of moral character. Such speculation about the connection between body shape and character has been related to anxiety over the arrival of new immigrants from Southern and Eastern Europe (Saukko 1999). For example, obesity and an attendant portrayal of slack personality were often associated with immigrant groups such as Italians and Jews during this era (Saukko 1999). Again, it is in the context of such broader processes that obesity is attributed to moral failure and personal deficiency in the early editions of *Cecil*.

Armstrong (1983) has argued that in the course of the 20th century the clinical gaze comes to exceed the space bounded by body and clinic, moving into the surrounding social sphere. In the later editions of *Cecil*, individual accountability recedes in favour of vigilance over the surfeit of conveniences found in Western consumer culture. Many have postulated that the modern cult of slimming and health consciousness relates to a moral and aesthetic rebellion against the gross excesses of consumer culture, and point to the contradictions imposed by late capitalism, a political economy that at once requires ever higher levels of consumption and the regulation of desires to cultivate production (Crawford 1984, Schwartz 1986, Bordo 1993, Stearns 1997). In the postmodern economy of advertising, information technology, and service industries, the labouring body has become the desiring body, a body in search of personal satisfaction through an emphasis on leisure and seemingly insatiable consumption practices (Turner 1996). Cultural imaginings and ethical precepts with respect to individual, physical bodies may reflect anxieties concerning the social body (Douglas 1966). Fatness, therefore, may function as a site for social reflection, or social diagnosis, an index of our relation to consumption in advanced consumer capitalism.

Criticism of the food industry emerges in *Cecil* in the 1960s, a time of civic unrest in the US and the rise of postindustrial political sentiments advocating distrust in government, industry and consumerism. Levenstein (1993)

has traced how elements of the New Left, consumers' rights activists and environmentalists seemed joined together in attacking the food industry, particularly with respect to the marketing and proliferation of processed foods. Attacking the food industries was becoming 'a mini-industry in its own right' with a willing media and a receptive middle class audience that was losing faith in the food industries and the government (Levenstein 1993). The postwar period in America was also marked by a psychiatric turn to psychoanalysis and the role of childhood traumas, along with popular and academic anxiety about suburban conformity, with particular attention to the conduct of the mother in middle class families (Saukko 1999). In *Cecil*, it is during this time period that mothers and American homes are charged with enforcing early patterns of overeating.

The representation of obesity also informs, and is informed by, other social tensions. In the US, obesity disproportionately affects women of lower socioeconomic status and certain racial and ethnic groups (Sobal and Stunkard 1989, Kuczmarski *et al.* 1994). The framing of obesity, therefore, does not operate independently of the framings of race, class, and gender. For example, the female body has been symbolically configured as a site of excess and irrationality (Bordo 1993), resonant with a view of obesity as a failure of rational self-control. Obesity is also correlated with poverty, and we have noted the linkage of bodily constitutional types with various immigrant groups earlier this century. More generally, poverty itself is enmeshed within arguments concerning the role of individual responsibility versus victimisation caused by unfavourable social circumstances. The association of diseases with poverty must be placed in the context of multiple long-standing, historical debates on the causal nature of these relationships, the direction of causation, the emphasis on collective versus individual malady, and the efficacy of economic versus medical intervention (Schwartz 1984, Eyler 1992).

One might also consider the influence of shifting theoretical paradigms within biomedicine. World War II has been described as a watershed for the beginning of the chronic disease era for epidemiology (Susser 1985, Susser and Susser 1996). The force of the germ theory paradigm was fading, with infectious disease mortality greatly reduced by higher living standards, vaccines, and chemotherapy. Additionally, the population distribution was shifting toward older age, and chronic diseases, such as coronary heart disease and lung cancer, so-called 'diseases of civilisation', achieved prominence. A new theoretical paradigm and research framework emerged from the standpoint that chronic diseases have environmental causes, many of which are preventable. This post-war reorientation of focus with respect to causative attribution is certainly consistent and coincident with the turn to environment we describe for the explanation of obesity in *Cecil*. Reflecting more recent trends in biomedical research, the turn to genetics in the latest edition signals the increasingly hegemonic status of molecular epidemiology and genetic-based explanations.

The post-war period was also marked by the institutionalisation and rapid growth of federal funding from the National Institutes of Health (NIH), which was internally subdivided into organ- and disease-based categorical institutes. This organisational shift, in part, prompted an increase in the subspecialisation of research and training (Howell 1989). In the early 1970s, the American Board of Internal Medicine acknowledged Endocrinology and Metabolism as a new subspecialty area with obesity falling under its purview. In *Cecil*, authors for the first two entries are affiliated with the general specialty of internal medicine, whereas authors for the later entries are affiliated with the more narrow subspecialty of endocrinology. Advocacy for the delineation and legitimisation of a new subspecialty may, in some instances, motivate a reconfiguration of clinical phenomena into more concrete disease categories and direct research funds towards organic-based entities (Lawrence 1992). Hence, with respect to our findings, the recasting of obesity as disease and the appeal to genetically-mediated, organic causes may be associated with wider professional or organisational transformations.

Lastly, one might consider how knowledge and representation of an object is refashioned as the object itself changes. The proportion and absolute number of overweight Americans has increased dramatically over the time period covered by these textbooks. Recent studies show that in the last decade alone obesity increased from 12.0 per cent to 17.9 per cent (Mokdad *et al.* 1999), and that over half the adult population is now overweight or obese (Flegal *et al.* 1998). Perhaps an increase in numbers afflicted can itself render a phenomenon less deviant, less a matter of individual particularities, and precipitate recourse to more systemic effects. Indeed, obesity is now framed in the discourse of public health and cast in the terminology of 'epidemics' and even 'pandemics' (Egger and Swinburn 1997, Mokdad *et al.* 1999). Recently, authors in the *British Medical Journal* proposed an 'ecological' approach to obesity wherein obesity is regarded as a normal response to an abnormal environment (Egger and Swinburn 1997), and others in *Science* proposed that we 'cure' the environment to reverse the obesity epidemic (Hill and Peters 1998), echoing the findings of our study.

We do not wish to suggest that individuals are no longer held accountable for their own weight status. The diet, fitness and beauty industries are certainly subtended by, and dependent on, a discourse of individual agency, and obesity continues to be a highly stigmatised state with prejudice and reprobation against obese persons widespread within medicine and society at large. Obesity is often described as the last realm of socially permissible discrimination, and there continues to be enormous social and medical pressure to lose weight (Kassirer and Angell 1998). Moreover, dietary modification is but one component of a modern medical regimen promoting healthy lifestyles as a solution to problems ranging from cancer to sexually transmitted diseases (Fitzgerald 1994). As Foucault (1988) has suggested, the site of disciplinary power has moved, in recent times, from institutionalised surveillance to 'technologies of the self'. On the other hand, the rubric of *victimisation*

has been mobilised in identity politics by activist groups seeking non-discriminatory rights, and groups in the US such as NAAFA (National Association to Advance Fat Acceptance) have recently employed such a platform for political discourse. Sobal (1995) has argued that such developments can be viewed as a demedicalisation of obesity and the deployment of a political model in place of a medical model; the pattern of medicalisation and demedicalisation has much precedent (Fox 1988).

Textual representation and science

Lastly, we conclude by reflecting on the act of writing, an act which is a decidedly social practice. Over the last two decades, critical theoretical insight from multifarious disciplinary commitments has revealed the limits of representation, problematising various traditional assumptions subtending the production of knowledge. Points of contention are many, but examples include: the relation of the subject to his or her object of research (Bourdieu 1977, Clifford 1986, De Certeau 1986); the ideal of an Archimedean point of value-free epistemological privilege (Smith 1987, Haraway 1991); and the objective and subjective dichotomy between scientific and literary textual practice (Clifford 1986, De Certeau 1986, Latour 1987, Agger 2000). The entries from *Cecil* are written in accordance with rhetorical conventions typical, and indeed expected, of scientific texts; the discursive posture signals de-authored transparent representation, or mere reflection, of an external world of accumulated facts. This mode of representation, however, obscures the actions and conditions of its production. Scientific texts are the product of deliberate authorial choices; findings are narrated, and particular problematics are selected over others, while assumptions and *ellipses* are deferred (Agger 2000).

Our analysis of *Cecil* effectively illustrates this process, with each permutation on obesity highlighting the *authoriality* (Agger 2000) and argumentation that determines representation, as well as the underdetermined, contingent nature of all versions². The editions we present are indeed written by five different authors, and their perspectives are shown to be situated and partial (Haraway 1991). Authorial choices are embedded in broader social and historical contexts, and we have offered some initial speculation in this regard. Detailed and substantive inquiry into the local, individual circumstances of each author is beyond the scope of this project, but we do note their institutional and specialty affiliations. The question arises as to whether the changes we find represent change with time or simply differences between authors. Our answer is that they are undoubtedly secondary to both, and one cannot be unequivocally privileged over the other. Moreover, the relatively monotonic nature of the changes suggests that it is not merely author idiosyncrasy that drives the changes, but rather that there is also a real shift in the perception of obesity that the authors are capturing³.

Using *Cecil* and obesity as a specific case-example, our work demonstrates that despite a relatively stable overall physiological process, substantive

changes can occur between levels of explanatory analysis with an ongoing dialectic between these explanations and constructions of culpability and remediation. Given that each level of analysis and focal point for explanation has distinctive ideological and public health implications, we find that the *intelligible* body, a body explained, is always the *useful* body, a body subject to sociopolitical regulation (Foucault 1977).

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Notes

- 1 Although specific sales figures are not available, a variety of sources affirm *Cecil's* standing and popularity in the field. According to MD Consult, a popular Internet clinical information service which includes 35 leading medical texts, *Cecil* is among the top three books searched by physicians (Business Wire 1998). *Cecil* is also a bestseller on YBP Library Services lists (Simba 2000), and it is the oldest of comprehensive internal medicine textbooks recommended by the American College of Physicians in its 'Library for Internists' (Frisse and Valerie 1997). In a study of end-of-life care content in medical texts, Rabow *et al.* (2000), using a previously available published report, listed *Cecil* among the five best-selling textbooks and manuals for internal medicine. Lastly, in a recent *JAMA* review, *Cecil* is referred to as 'the granddaddy of general internal medicine texts' (Bernicker 1996).
- 2 We have focused our discussion on writing, but we would like to address the process of reading as well. Many have argued that reading is ill-conceived as passive, unmediated reception; rather, reading is an active, consumptive practice that partakes in writing a text by interpolating, interpreting, and filling in the gaps (De Certeau 1984, Agger 2000). For any text, meaning is engendered only in the process of particular readings and in the translation of its contents into particular practices. We would extend these arguments on reading, as well those on writing, to our own reading and writing of these textbooks; we, obviously, engage in interpretation and argumentation as well. We emphasise, therefore, that our work does not speak to the specific relationship of these texts to the readings and practices of its intended audiences. Such effects can only be determined through archival and ethnographic work on the dynamics of its usage in particular instances and concrete practices.

- 3 Perhaps we could also answer by rethinking the question itself. What does it mean to distinguish between persons and time here? Suppose that all editions of *Cecil* were authored by the same person. Could we then make the more restricted conclusion that all changes stem purely from factors external to, and apart from, the author? Our position is that we could not, for we would then need to assume that an author can be viewed as self-identical through time, univocal and fixed, passively reflecting an external world that changes. Persons change from moment to moment as well, and identities do not cohere in a fashion that renders them separable from social context and praxis. In demonstrating the continuities and discontinuities of these texts, our intention was to make manifest the partiality of each instantiation, that each is processual in nature, contingent on innumerable factors that are only crudely captured by notions such as person or time.

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