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By: Australian Lifestyle Medicine Association (ALMA)

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- 1. Typically it's thought that the answer to obesity lies in a single key solution (like a single lock and key). This submission suggests that the answer is more complex and is more analogous to a 'barrel lock' where all components have to be lined up for a significant impact on the problem.
- 2. In this light, recent evidence shows that obesity might often be just a marker of lifestyle and environmental factors, rather than a direct cause of chronic diseases.
- **3.** This has been shown recently by the discovery of a form of low-grade systemic inflammation ('metaflammation') which is common to most, if not all, chronic diseases (including cancer), and which may or may not be associated with obesity.¹



4. The real causes of 'metaflammation' are 'upstream' (and coincidentally have similarities with the causes of climate change²)



- 5. Hence treating obesity alone (ie. with diets, exercise programs), while often likely to have a 'partial' (ie. often short-term and limited) effect at the individual level, can be neither *necessary* nor *sufficient* for dealing with the chronic disease epidemic associated with obesity at the population level.
- 6. This requires a major shift in thinking. Firstly towards prevention, and secondly towards total lifestyle including:

Over-eating, poor diet, inactivity, stress, smoking, depression, inadequate sleep, environmental pollution, drug use etc.

... all of which can have a pro-inflammatory effect, sometimes in the presence of, but often in the absence of obesity (See Figure).



- 7. Lifestyle Medicine is a way of dealing with this that encompasses both conventional treatment of risk factors, but more upstream management of causes, including environmental action.³
- 8. The Australian Lifestyle Medicine Association (ALMA) is the first academicallybased non-profit association to do this. Currently it provides training for over 1500 GP's a year and is now targeting the 14 allied health disciplines able to claim Medicare benefits under the Enhanced Primary Care (EPC) System.

References:

- 1. Egger G, Dixon J. Should obesity be the main game? Or do we need a total environmental makeover to manage our inflammatory and chronic disease epidemics. *Obesity Reviews* (in press).
- Egger G. Dousing our inflammatory environments: Is personal carbon trading an option for reducing obesity and climate change? *Obesity Reviews* 2008; 9(5): 456-463
- 3. Egger G. Personal carbon trading: A potential 'stealth intervention' for obesity reduction? *Medical Journal of Australia* 2007; 187: 185-187.

Dousing our inflammatory environment(s): is personal carbon trading an option for reducing obesity – and climate change?

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Summary

Obesity and climate change are two problems currently challenging humanity. Although apparently unrelated, an epidemiological approach to both shows a similar environmental aetiology, based in modern human lifestyles and their driving economic forces. One way of analysing this is through inflammation (defined as '... a disturbance of function following insult or injury') of both the internal (biological) and external (ecological) environments. Chronic, low-grade, systemic inflammation has recently been shown to accompany obesity, as well as a range of biological pathologies associated with obesity (diabetes, heart disease, some cancers, etc.). This is influenced by the body's inability to soak up excess glucose as a result of insulin resistance. In a broader sense, inflammation is a metaphor for ecological 'pathologies', manifest particularly in unnatural disturbances like climate change, ocean acidity, rising temperatures and species extinction, associated with the inability of the world's environmental 'sinks' to soak up carbon dioxide ('carbon resistance'?). The use of such a metaphorical analysis opens the possibilities for dealing with two interdisciplinary problems simultaneously. Strategies for managing climate change, including personal carbon trading, could provide a 'stealth intervention' for reducing population levels of obesity by increasing personal energy expenditure and decreasing energy-dense food intake, as well as reducing the carbon emissions causing climate change.

Keywords: Biology, carbon trading, ecology, economic growth, environment, inflammation.

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Introduction

A robust model for managing epidemics is the epidemiological triad (1), which has been used to deal with a range of health problems from infectious diseases to road traffic injury (2). This suggests that all three corners of a triangle – host, vector and environment – have to be considered if true inroads are to be made in epidemic control. Recently, it has been suggested that the triad could be applied to obesity (3). Perhaps least considered of the three corners of the triad in relation to obesity, however, has been the role of the broad, macro environment. The concept of an 'obesogenic environment' was first proposed over a decade ago (4) to describe the external factors passively influencing energy balance in modern industrialized societies. Developments since then, including the relentless march of the obesity pandemic (5) have confirmed this as a major, albeit 'distal' cause of obesity. However, advances in molecular research, which suggest that obesity is part of a broader inflammatory response in the body (6,7), combined with an increasing world interest in the external environment (particularly climate change), provide an opportunity to expand this to an 'inflammatory' environment, which relates to both the internal (biological) and external (ecological) environments, with wide implications on how both might be managed concurrently.

While the notion of a link between climate change and obesity is not novel (8,9), the concept of inflammation is used in this paper both literally and as a metaphor to help understand the relationship. The use of metaphor in science in this way has a long history, from the atom to unfolding protein (10). It is used cautiously here to broaden conceptions and stimulate cross-disciplinary interactions. The similarities between biological and ecological environmental influences are discussed first, and an epidemiological approach is used to track both back to a common distal cause. Finally, a system of personal carbon trading (PCT) (11), in combination with already existent corporate trading plans, is proposed as a potential 'stealth intervention' (12) for reducing obesity, while targeting reductions in climate change. It is proposed that this would work by increasing personal energy expenditure, and decreasing energy-dense food consumption, while at the same time reducing carbon and other gas emissions into the atmosphere (13).

Obesity, climate change and 'inflammation'

Obesity and climate change are both current issues of world concern. Around 15% of the world's population are currently overweight or obese (5), leading to a big rise in metabolic diseases associated with this, such as type 2 diabetes, cardiovascular disease, certain types of cancers and other metabolic disorders (14).

In a similar fashion, atmospheric carbon concentrations throughout the world are excessively high, at around 380 ppm, having increased dramatically from 250 ppm just 50 years ago. This has now been implicated in a number of ecological disorders including atmospheric pollution, global warming and climate change (15).

Both obesity and the modern environment are characterized by a form of disturbance that can be described as 'inflammation' (defined by the Macquarie dictionary as '... a disturbance of function following insult or injury'). At the biological level, this refers to an immune reaction to correct a disturbance of physiological homeostasis. Classic inflammation is usually an acute reaction associated with infection or injury. Recently, however, a subclinical form of inflammation has been linked to obesity and a number of other lifestyle-related disorders (16). A distinction between this type of low-grade systemic inflammation and the classic form is its more chronic nature and the comparatively low, two- to fourfold elevation of inflammatory markers, compared with much higher levels during acute infections (17). The term 'metaflammation' (metabolically triggered inflammation) has been used to distinguish it from the more classical, acute phase, inflammatory state (18).

Inflammation, as so defined, could also be applied to the broader ecological environment, particularly that encompassed by Lovelock's (19) concept of Gaia, which invokes the notion of the ecosphere as a living, responsive system. In this context, elevated carbon (and other gas) concentrations in the atmosphere from human development constitute an ecological disturbance with symbolic similarities to the metaflammation observed in humans. Increased ocean acidity, atmospheric pollution, and global warming are the inflammatory markers that signal such an allostatic state of the ecosphere, representing '... a disturbance of function following insult or injury', with climate change a metaphorical 'dis-ease' outcome. Pictures of industrial environments showing pollutant fumes, such as that shown in Fig. 1, support this description of 'inflamed'. Hence, an appropriate term, in line with the 'metaflammation' described above, would be 'ecoflammation'. Both inflammatory forms invite a more detailed understanding before progressing to discuss causality.

'Metaflammation'

The link between low-grade systemic inflammation (metaflammation), obesity and a range of lifestyle-related disorders, including type 2 diabetes (20), heart disease (21), certain cancers (22), erectile dysfunction (23) and even Alzheimer's (24), is demonstrated in humans by the expres-



Figure 1 An inflamed body in an inflamed environment: is there a common aetiology?

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sion of inflammatory cytokines, chemokines and related proteins from different sources in the body. Because many of these are produced in the adipocyte (25), and as the link between the immune and metabolic systems now seems much closer than was once thought (18), obesity has been typically thought to be the causal link. However, some evidence suggests that at least some subcutaneous fat stores (in contrast to visceral stores), while associated with an inflammatory response, may be as much a marker for disease, as a direct cause (26). In any case, increases in adiposity (possibly beyond a genetically predetermined level) have been convincingly linked with increases in inflammatory processes (27), possibly initiated by the infiltration of macrophages from surrounding stromal cell beds (28).

Metaflammation is also related to the inability of the body's cells to metabolize glucose as a result of insulin resistance, and it now seems clear that inflammatory mediators can increase insulin resistance in cells with or without obesity (18). An inflammatory reaction has been shown to occur to dietary fats (29) and inactivity (30), as well as to other, lifestyle-related factors; inadequate sleep (31), smoking (32), stress (33), excessive alcohol intake (34) and depression (35). The immune reaction is similar to that to a microbial invader (36) and could be instigated, among other things, by common lipopolysaccharide stimulants (37). Hence, it is feasible to suggest that, in many cases at least, the internal inflammatory environment often ascribed to obesity, may also be related to the actions causing obesity (and other lifestyle factors), which make up part of the modern western lifestyle.

This supports the notion that humans, who have not evolved with a modern industrial lifestyle, have an immune reaction to certain aspects of that lifestyle. The vast proportion of human evolution has been in an active environment, with limited access, for example, to the high energy-dense saturated fats and carbohydrates that are readily available today. And while the acute inflammatory reactions that occur to infrequent meals of this kind might be abated by a compensatory anti-inflammatory response - like a homeopathic dose of a microbiological pathogen - continuous ingestion might result in the chronic proinflammatory state now seen in such common modern maladies as the 'metabolic syndrome'. Similarly, the drastic reductions in ambient activity levels in industrial, compared with pre-industrial environments [estimated to be ~1000 kcal day (38)], disrupt the low inflammatory milieu normally maintained by myokine release from muscular contraction in an active environment (30), thus attenuating the chronic inflammatory state. As unlikely as it may sound, humans may be 'allergic' to the modern industrial environment. It now seems evident that such an environment may also cause an allostatic ecological state, with parallels to metaflammation.

'Ecoflammation'

Paradoxically, the external environment creating the conditions for the metaflammatory state discussed above can also be described as inflammatory ('ecoflammatory'). As suggested above, this is particularly pertinent to Lovelock's (19) concept of the ecosphere, as a living, self-regulating system comprised of physical, chemical, biological and human components. Lovelock's description of himself as '... a planetary physician, whose patient, the living Earth, complains of fever' (39) extends the metaphor. If inflammation is a defensive response to an allostatic load, climate change represents the response to increased atmospheric pollution, just as lifestyle-related diseases represent the response to the allostatic load of energy imbalance and other components of modern lifestyle. Using such an analogy, the warning sign the 'insulin resistance' of the ecosphere - is illustrated in the inability of the planet's sinks to 'soak up' excessive carbon emissions ('carbon resistance'?), just as the body can no longer effectively utilize blood sugars because of the failure of its cellular 'sinks' through insulin resistance. Other symptoms of a disturbance of ecological function are '. . . degradation of land, depletion of resources, accumulation of wastes, pollution of all kinds, climate change, abuses of technology and destruction to biodiversity in all its forms' (40). Perhaps surprisingly, the aetiology (and possibly treatment) of both biological and ecological forms of inflammation may not be that dissimilar.

The aetiology of inflammation

To date, the management of both obesity and climate change has been centred around controlling risk factor markers such as through pharmacotherapy for hypertension or dyslipidaemia, or the development of bio-fuels or geo-sequestration for carbon emissions. However, this is very much a 'finger in the dyke' approach. To ascribe over-eating as the cause of obesity, or gas emissions as the cause of climate change, is like attributing an outbreak of salmonella to 'a restaurant somewhere in London'. A true epidemiological approach would track back the level of causality to its ultimate genesis. As shown in Fig. 2, with the biological and ecological pathologies discussed here, this happens to end in a common causality.

While risk factor markers do need to be managed, there are proximal, medial and more distal causes of these, which are the ultimate causes of the eventual outcome. As shown in Fig. 2, proximal causes are diet and inactivity at the individual level, and temperature increases and ozone depletion in the broader environment, leading to the risk factor markers and dis-ease outcomes that usually attract most popular public attention. At a more medial level, the causes of both forms of 'inflammatory state' begin to merge through the growing misuse of energy; too little personal



Figure 2 The epidemiology of 'inflammation'. A similar causal hierarchy is shown between the route to 'metaflammation', or that associated with individual biological inflammatory states at the top of the figure, and the 'ecoflammation', associated with global ecological pathologies at the bottom. CVD, cardiovascular disease

energy expended and too much high energy-dense food consumed in the case of the individual, and too much fossil fuel energy burned and waste emissions given off in the case of the broader environment. As well as increasing metaflammation in the individual, this has resulted in increasing world levels of atmospheric carbon. The 'tipping point' in being able to reverse this is estimated at around 500 ppm (15), but with a current level of 380 ppm and increases of 2 ppm per year growing exponentially, this is likely to be quickly exceeded.

However, if the analogy of managing risk factors as being like 'placing a finger in the dyke' is extended, dealing with proximal and medial causes, still only represents plastering over the cracks in the wall. More upstream (distal) intervention is necessary for long-term treatment, perhaps ultimately by diverting the stream to a more secure course. In the meantime some steps can be taken to reduce the flow, and perhaps even divert this into other channels. As most ecologists and obesity experts would accept, no one technique of changing either energy dependence to reduce climate change, or energy balance to reduce obesity, is likely to be successful alone. A portfolio of approaches may be necessary. And while this might seem a daunting task, an approach that deals with both problems simultaneously could have wide public and cross-disciplinary appeal and add to an arsenal of options available. If such an approach was equitable, relatively painless and easy to instigate, it would also add to its utility and the chances of it being adopted. Just one approach, personal carbon trading (PCT), is considered in this light here.

Dousing the flames

There seems little doubt that environmental correction – a dousing of the ecoflammatory environment – could have

major impacts on human health; the metaflammatory environment. Higher personal physical activity resulting from reduced fossil fuel use from transport alone, for example (41), would help reduce the chronic low-grade systemic inflammation known to be associated with inactivity (30), while reducing the carbon emissions that feed the ecoflammatory state. As fossil fuel use is responsible for over 1/3 of total greenhouse gas emissions (15), reduction in use of this, particularly through transport, seems an unchallengeable goal. A novel 'backcasting' (in contrast to 'forecasting') approach, with potential for obesity reduction, has recently been taken to this (42). It is unlikely, however, that humans will voluntarily give up, or even cut down significantly on, the achievements of modern technology to achieve this [for a more detailed discussion on this (43,44). The well meaning few in developed economies who are prepared to do so are faced with the rising tide of development in countries like China and India, and continuing population growth in some developed countries, which threatens to drown their anti-inflammatory actions. Systems models indicating potential future problems (45), and efforts to ignite a debate about an alternative economic system (46), have also failed to excite political leaders, or the public. Hence, an acceptable means of managing both inflammatory environments, with full support of the public and legislators alike, is needed.

Several possibilities have been proposed, most relying on modified attitudes to the broader environment and climate change. However, attitudes are not necessarily a driver of behaviour. Often it occurs in reverse. Reductions in smoking rates, for example (and many other epidemics), have come from attitude changes following behaviour that is legislated or regulated. Random breath testing, seatbelt use and bicycle safety are just a few examples of attitudes following behaviour that is regulated or legislated. Similarly, the only significant reductions that have occurred in obesity at a population level are in countries like Nauru [as a result of depletion of super-phosphate resources (De Courten M. unpublished WHO data)] and Cuba (as a result of the economic crisis of 1989–2000) (47), where behavioural change has been forced by economic circumstances. In the absence of such 'natural' events, the health promotion axiom: 'Regulate and legislate where you can; educate and motivate where you can't' (48) could apply to correction of both internal and external environments, Carbon emission management offers a potential link.

Carbon trading as a 'non-pharmaceutical anti-inflammatory device'

Carbon emissions from the oxidation of organic fuel sources make up around 70% of the world's greenhouse gases. The reduction of these emissions is now an accepted goal among environmental and health scientists alike. Currently, emissions range from the average levels of around 1 tonne pp/py in Africa to 24 tonnes pp/py in the USA. A goal is to reach a sustainable long-term world average estimated at between 2 and 5 tonnes pp/py (16). Corporate carbon trading, with a 'cap and trade' approach to reducing carbon emissions, is one way of doing this. However, corporate emissions account for only ~50-60% of all gas emissions. The remaining levels are from personal, household and domesticated animal sources, ~70% of which are carbon-related, and the remaining 30% other gases, also related to human development. These can now be accurately measured and attributed to the specific quantity of energy use (i.e. per unit of fuel, electricity, heating, cooling, etc.) on a per capita basis (Table 1). Individual carbon emissions, and hence energy use, are thus able to be given a value that can then be traded on an open market, like any other commodity. Reductions in corporate emissions may help moderate the metaflammatory environment through the possible price increases (and presumably demand decreases) of processed, energy-dense, distantly produced

 Table 1
 Estimates of global warming potential of gases released from combustion of fuels (1 kg of carbon dioxide = 1 carbon unit) [see

 Flemming (52)]

Fuel	Carbon units
Human energy	Long (
Natural gas	0.2 per kWh
Grid electricity (night)	0.6 per kWh
Grid electricity (day)	0.7 per kWh
Petrol	2.3 per litre
Diesel	2.4 per litre
Coal	2.9 per litre

foods, in contrast to those that are natural, less energydense and locally grown. However, corporate trading might be expected to have minimal effect on the other side of the energy equation driving inflammation, i.e. human energy expenditure and fossil fuel use.

A PCT system, which was initially proposed by the Global Commons Institute in the UK (49), and elaborated on by others (50-52), would add to the benefits of other proposals to reduce carbon emissions, and might have more of an impact on obesity. A PCT system proposes a workable financial incentive system that provides equity and efficiency in reducing non-renewable energy use and greenhouse gas emissions. The basic concepts of such a system are 'contraction and convergence'; contraction of available carbon allocations to all individuals in a country to a sustainable level over time, and *convergence* towards equal per capita emissions through trade of emission rights between frugal and profligate emitters. The scheme involves the annual setting of tradable energy units per year by a carbon bank, based on about 40% of a total budget (which includes both personal and corporate quotas). These are then distributed equally through a credit card system to all individuals within a country, to be redeemed when paying for non-renewable energy (i.e. fuel, power, etc.). Each unit is equivalent to 1 kg of carbon released through energy use. Allocated and tradable carbon allowances (called TEQs or Tradable Energy Quotas) would be set by an independent carbon bank managed through current market systems, have equity, allow flexible goals to be set into the future and provide a possible 'stealth' intervention' (13) for decreasing metaflammatory states, including obesity.1 Such a scheme, although requiring considerable political and public will (and unlikely to be unchallenged by vested interests), would be relatively easy to administer once accepted.

A PCT scheme has equity in that convergence would occur within countries from rich, high-energy users to poor, frugal users who can actually profit from the scheme. Between countries, trading would take place from rich to poor countries (e.g. the USA, Europe, Australia, etc. to Africa, Central America the Pacific, etc.), serving as a more empowering alternative to aid. Unused units are retired, with a view to contraction of the total energy budget to a sustainable level over time (i.e. ~20 years). Individual country budgets and targets would be determined by that country's current per capita emissions. The PCT idea is currently under discussion in Europe and the UK (43,53), but has received limited attention among some of the biggest polluters, including the USA. As with any major social change, the time phase of the public response to such a proposal might be predicted by

¹For a more detailed discussion of how such a system would work in practice see the study by Fleming (52).

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Mahatma Ghandi's well used revolutionary phrase that: 'First they ignore you, then they laugh at you, then they fight you, then you win' (48).

Effects of personal carbon trading on 'inflammation'

The 'stealth intervention' benefit of a PCT scheme resides in the potential modification of the existing obesogenic environment, through encouraging changing attitudes to energy use, as well as other more direct benefits (Table 2). This would come from greater incentives for the use of personal energy (e.g. walking or cycling) in place of non-renewable energy (e.g. driving fossil-fuel powered vehicles), given the economic and legislative incentive to do so. The demand for infra-structure changes to accommodate personal energy-driven transport (cycleways, walking paths, etc.), and for pedestrians and cyclists to be given right of way at all times, might be expected to follow. Carbon costs of passive inactivity (e.g. TV, video games, etc.) might also be expected to increase activity levels in children through the reduction of inactivity, which has been shown to be more effective in increasing children's personal energy expenditure than increasing their involvement in active sports (54). A desired longterm outcome of all this would be a shift in consumer aspirations from conspicuous consumption, to conspicuous non-consumption, where the lust for non-renewable (and often fattening) consumables is reduced and the importance of health is elevated, almost coincidentally. Indirectly, this could also address concerns about limits to growth in a finite system (45,46). Finally, the system

would help the global disparity in access to resources, which is a chief cause in health differentials (55).

Such a proposal is not meant as a panacea or a single-step approach to either obesity or climate change. Nor is it likely to be as easy to effect as might be interpreted here. Without population control for example, individual carbon emission reductions may still have a positive effect on obesity but are likely only to maintain the status quo with respect to the broader environment. A total world population of nine billion people (as predicted in the next 40 years) emitting an average of 10 tonnes of carbon per person per year would have a similar effect to the current 6 million people emitting 15 tonnes. Yet, discussion of population control remains a highly sensitive political issue, perpetuated by significant vested economic and theological interests. Suggestions of a technological fix within the current economic growth paradigm, also pervade the political scene, despite the now accepted need to vigorously pursue approaches to cut carbon emissions (16), albeit within a cautionary framework (56).

The current notion of economic growth based on production and sale of products and services, irrespective of their social or environmental impact, will also have to be re-examined (as was intimated in the 1930s by JM Keynes, the architect of the modern growth system, who stated that as nothing can grow indefinitely, we may have to look to an alternative within 100 years or so). Political will and public acceptance are obviously also major factors, which need to be carefully considered, and it is not intended that these be glossed over here. Such practicalities are for more detailed and practical forums. Other issues such as concessions, such as through national tax systems, would also need to be

Table 2 Potential effects of personal carbon trading on individual health and the broader environment

Potential impacts on obesity and health (metaflammation)	Potential impacts on the environment (ecoflammation)
 Personal carbon quotas lead to frugality of use of non-renewable	 Reduction of carbon emissions into the atmosphere. Reduced
energy	climate change
Conspicuous non-consumption becomes acceptable in contrast to	 Desire for better energy efficiency and development of renewable
over-consumption	energy
 Personal energy use for transport (e.g. walking, cycling) is increased 	 Existing fossil fuel reserves are conserved to last longer
 Infrastructure for personal energy use (walking, cycling, etc.) is more	 Reduced air pollution; improved energy efficiency; altered town
valued, hence encouraging greater personal energy use	planning to increase emphasis on personal mobility
 Increase in cost of passive entertainment (TV, video, etc.), leading to	 Reduced demand on energy supplies leading to less likelihood of
more active leisure in children	outages in peak periods
 Reduced use of air conditioning and non-renewable heating sources	 Improved architecture in homes/hotels, etc. Increased importance o
with their potential effects on body weight	renewable heating/cooling sources
 Decreased demand for high energy-dense (processed) foods due to	 Increased demand for low energy-dense, locally grown, unprocesse
increased price through corporate carbon-trading	food products, hence reducing food transport
 Population levels of personal energy use are increased and levels of	 Through reduction in non-renewable energy 'sources' and less
energy intake are decreased, leading to population reductions in	pressure on 'sinks', a more sustainable economic and ecological
obesity	system is encouraged

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looked at within countries, e.g. for those in essential industries and in remote areas.

Personal carbon trading represents just one arm of a carbon trading armamentaria, which in turn is one part of a required portfolio of approaches for reductions in climate change and increases in public health. While this has been suggested as a means of tackling broad environmental problems, there has been little consideration of its specific benefits for obesity to date. A more detailed analysis and modelling of how this might occur would give greater emphasis to adoption of such a scheme, which is surely worthy of serious consideration if we are to have any hope of permanently dousing our currently pathological inflammatory environment(s). To tackle both environments simultaneously would more than double the benefit.

Conflict of Interest Statement

No conflict of interest was declared.

References

1. Last J. A Dictionary of Epidemiology. Oxford University Press: New York, 1988.

2. Swinburn B, Egger G. Analysing and influencing obesogenic environments. In: Bray G, Bouchard C (eds). *Handbook of Obesity; Clinical Applications*, 3rd edn. Marcel Dekker Inc.: New York, 2008.

Egger G, Swinburn B, Rossner S. Dusting off the epidemiological triad: could it work with obesity. *Obes Rev* 2003; 4: 115–120.
 Egger G, Swinburn B. An ecological model for understanding the obesity pandemic. *BMJ* 1996; 20: 227–231.

5. International Association for the Study of Obesity (website) [WWW document]. URL http://www.iasso.org (accessed November 2007).

6. Mehta S, Farmer JA. Obesity and inflammation: a new look at an old problem. *Curr Atheroscler Rep* 2007; **9**: 134–138.

7. Ferrante AW Jr. Obesity induced inflammation: a metabolic dialogue in the language of inflammation. *J Intern Med* 2007; 262: 408–414.

8. Roberts I. How the obesity epidemic is aggravating global warming, *New Scientist* 2007; 2610: 21.

9. Forsight: Tackling Obesities: Future Choices. [WWW document]. URL http://www.foresight.gov.uk/Obesity/Obesity_final/ Index.html (accessed January 2008).

10. Brown TL. Making Truth. University of Illinois Press: Chicago, IL, 2003.

11. Roberts S, Thurmin J.Centre for Sustainable Energy. A Rough Guide to Individual Carbon Trading: The Issues, Ideas and the Next Steps. Report to DEFRA, November 2006. [WWW document]. URL http://www.defra.gov.uk/environment/climatechange/ uk/individual/pca/pdf/pca-scopingstudy.pdf (accesses November 2007).

12. Robinson TN, Sirard JR. Preventing childhood obesity: a solution oriented research paradigm. *Am J Prev Med* 2005; 28: 194–201.

13. Egger G. Personal carbon trading: a potential 'stealth intervention' for obesity reduction? *Med J Aust* 2007; 187: 185–187. 14. Proietto J. Obesity and disease: insulin resistance, diabetes, metabolic syndrome and polycystic ovary syndrome. In: Kopelman PG, Caterson ID, Dietz WH (eds). Clinical Obesity, 2nd edn. Backwell Publishing: Oxford, 2005.

15. Intergovernmental Panel on Climate Change. Climate change: synthesis report 2001. [WWW document]. URL http://www.ipcc. ch/pub/un/syreng/spm.pdf (accessed November 2007).

16. Shoelson SE, Herrero L, Naaz A. Obesity, inflammation, and insulin resistance. *Gastroenterology* 2007; 132: 2169–2180.

17. Larson GL, Hensen PM. Mediators of inflammation. Ann Rev Immunol 1983; 1: 335-359.

18. Hotamisligil GS. Inflammation and metabolic disease. *Nature* 2006; 444: 860–867.

19. Lovelock J. Gaia: A New Look at Life on Earth. Oxford University Press: London, 1979.

20. Hartge MM, Unger T, Kintscher U. The endothelium and vascular inflammation in diabetes. *Diab Vasc Dis Res* 2007; 4: 84-88.

21. Berg AH, Scherer PE. Adipose tissue, inflammation and cardiovascular disease. *Circ Res* 2005; **96**: 939-949.

22. Tan TT, Coussens LM. Humoral immunity, inflammation and cancer. *Curr Opin Immunol* 2007; **19**: 209–216.

23. Vlachopoulos C, Aznaouridis K, Iokeimidis N, Rokkas K, Vasiliadou C, Alexopolous N, Stefanadi E, Askitis A, Stefanadis C. Unfavourable endothelial and inflammatory state in erectile dysfunction patients with or without coronary heart disease. *Eur Heart J* 2006; 27: 2640–2648.

24. Finch CE, Morgan TE. Systemic inflammation, infection, ApoE Alleles and Alzheimer's disease: a position paper. Curr Alzheimer Res 2007; 4: 185–189.

25. Lago F, Dieguez C, Gomez-Reino J, Gualillo O. The emerging role of adipokines as mediators of inflammation and immune responses. *Cytokine Growth Factor Rev* 2007; **18**: 313–325.

26. Telford R. Low physical activity and obesity: causes of chronic disease or simply predictors? *Med Sci Sports Exerc* 2007; 39: 1233–1240.

27. Arner P. Introduction: the inflammatory orchestra in adipose tissue. J Intern Med 2007; 262: 404–407.

28. Nguyen MTA, Favelyukis S, Nguyen A-K, Reichart D, Scott PA, Jenn A, Liu-Bryan R, Glass CK, Neels JG, Olefsky JM. A sub-population of macrophages infiltrates hypertrophic adipose tissue and is activated by FFAS via TLR2, TLR4 and JNK-dependent pathways. *J Biol Chem* 2007;Oct: doi:10.1074/jbc. M706762200.

29. Shi H, Kokoeva MV, Inouye K, Tzameli I, Yin H, Flier JS. TLR4 links innate immunity and fatty acid-induced insulin resistance. *J Clin Invest* 2006; **116**: 3015–3025.

30. Bruunsgaard H. Physical activity and modulation of systemic low-level inflammation. J Leukocyte Biol 2005; 78: 819-835.

31. Irwin MR, Wang M, Campomayor CO, Collado-Hidalgo A, Cole S. Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. *Arch Intern Med* 2006; 166: 1756–1762.

32. Rohleder N, Kirschbaum C. The hypothalamic-pituitaryadrenal (HPA) axis in habitual smokers. *Int J Psychophysiol* 2006; **59**: 236–243.

33. Black PH. The inflammatory response is an integral part of the stress response: implications for atherosclerosis, insulin resistance, type II diabetes and metabolic syndrome. *Brain Behav Immun* 2003; 17: 350–364.

34. Szabo G, Mandrekar P, Oak S, Mayerie J. Effect of ethanol on inflammatory responses. Implications for pancreatitis. *Pancreatology* 2007; 7: 115–123.

35. Schiepers OJ, Wichers MC, Maes M. Cytokines and major depression. *Prog Neuropsychopharmacol Biol Psychiatry* 2005; 29: 210–217.

© 2008 The Author Journal compilation © 2008 International Association for the Study of Obesity. **obesity** reviews 9, 456–463 36. Tschop M, Thomas G. Fat fuels insulin resistance through toll-like receptors. *Nat Med* 2006; **12**: 1359–1361.

37. Berczi I. Neurohormonal host defense in endotoxin shock. Ann N Y Acad Sci 1998; 840: 787-802.

38. Vogels N, Egger G, Plasqi G, Westerterp KR. Estimating changes in daily physical activity levels over time: implications for health interventions from a novel approach. *Int J Sports Med* 2004; 25: 607–610.

39. Lovelock J. The Revenge of Gaia. Penguin Books: London, 2007.

40. Tickell C. Foreword in Lovelock J. The Revenge of Gaia. Penguin Books: London, 2007.

41. Woodcock J, Banister D, Edwards P, Prentice AM, Roberts I. Energy and transport. *Lancet* 2007; 370: 1078–1088.

42. Hickman R, Bannister D. Towards a 60% Reduction in UK Transport Carbon Dioxide Emissions: A Scenario Building and Backeasting Approach. [WWW document]. URL http://www.ucl. ac.uk/~ucft696/documents/eceee_paper_04.05%20final1.pdf

(accessed January 2008).

43. Hillman M. How We Can Save the Planet. Penguin: London, 2004.

44. Monbiot G. Heat. How to Stop the Planet Burning. Allen Lane: London, 2006.

45. Meadows DH, Randers J, Meadows DL. Limits to Growth: The 30-Year Update. Chelsea Green Publishing: White River Junction, VT, 2004.

46. Daly HE. *Beyond Growth*. Beacon Press: Boston, MA, 1996. 47. Franco M, Ordunez P, Caballero B, Tapia Granado JA, Lazo M, Bernai JL, Guallar E, Cooper RS. Impact of energy intake, physical activity and population-wide weight loss on cardiovascular disease and diabetes mortality in Cuba, 1980–2005. *Am J Epidemiol* 2007;Published in advance Sept 19; doi:10:1093/aje/kwm226. 48. Egger G, Spark R, Donovan R. Health Promotion Strategies and Methods. McGraw-Hill: Sydney, 2005.

49. Global Commons Institute. Contraction and Convergence. A Global Solution to a Global Problem. GCI: London, 2006. [WWW document]. URL http://www.gci.org.uk/contconv/cc.html (accessed November 2007).

50. Starkey R, Anderson K. Domestic Tradable Quotas: A Policy Instrument for Reducing Greenhouse Gas Emissions from Energy Use. Tyndall Centre Technical Report 39, December 2005. [WWW document]. URL http://www.tyndall.ac.uk/research/theme2/ final_reports/t3_22.pdf (accessed 15 September 2007).

51. Roberts S, Thumim J.Centre for Sustainable Energy. A Rough Guide to Individual Carbon Trading: The Issues, Ideas and the Next Steps. Report to DEFRA, November 2006. [WWW document]. URL http://www.defra.gov.uk/environment/climatechange/ uk/individual/pca/pdf/pca-scopingstudy.pdf (accessed November 2007).

52. Fleming D. Energy and the Common Purpose: Descending the Energy Staircase with Tradable Energy Quotas (TEQs). The Lean Economy Connection: London, 2005. [WWW document]. URL http://www.theleaneconsomyconnection.net (accessed January 2008).

53. Stott R. Healthy response to climate change. *BMJ* 2006; 332: 1385–1387.

54. Epstein LH, Roemmich JN, Saad FG, Handley EA. The value of sedentary alternatives influences child physical activity choices. *Int J Behav Med* 2004; **11**: 236–242.

55. Charlton BG, White M, Living on the margin: a salutogenic model for socio-economic differentials in health. *Public Health* 1995; 109: 235-243.

56. Jacobs M. The Green Economy. Pluto Press: London, 1999.