Feature

Veterinary Record 125 years

COMPANION ANIMAL OBESITY

The big problem: battling companion animal obesity

Obesity in companion animals is not a new problem and research is suggesting that it is a complex physiological issue, with genetic and endocrine causes. Eleanor Raffan discusses why some individuals appear to be predisposed to becoming obese, the far-reaching consequences of obesity and how veterinary clinicians can help owners to help their pets

'I am inclined to resent the wide publicity recently bestowed upon a certain [cat which] has achieved notoriety by measuring 33 inches round the waist and weighing just over two stone. That is a good deal of cat; about three times too much, to be perfectly frank.'

'Far be it from me to sneer; obesity, whether in beasts or baronets, is a matter for pity, not for mirth. When I look at what is styled as a "pet dog", wheezy and corpulent, his capricious appetite tempted with dainty food, his healthy canine instincts destroyed by wicked or unnatural pampering, I wonder that its owner is not ashamed.'

Not my words, but those of a certain K. R. G. Browne writing in the *Daily Mail* in 1934 and 'A lady', upset about pampered pooches, around the time that *Veterinary Record* was first published (A lady 1881). Not much has changed since then; there has been much publicity about pet and human obesity over recent years. But does the reality match the hype? How much of a problem is obesity for our companion animal patients? What is the current state of knowledge about how obesity develops and its impact on disease? And how should we as vets be addressing the problem in our patients?

This article will address those questions and suggest that obesity is perhaps best regarded as a derangement of complex homeostatic mechanisms governing energy balance in the body, rather than a moral failing of pets and humans.

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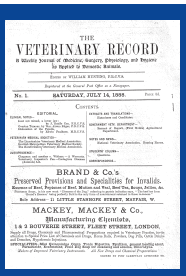
Veterinary Record is 125 years old this year. To celebrate, we are publishing an article each month focusing on a key clinical topic. Each article aims to look at what the challenges have been, how the topic has developed and what the future might hold. Articles published so far are listed in the box on p 291.

The pictures on the right compare the first issue of *Veterinary Record*, published on July 14, 1888, with how it looks today


THE VETERINARY RECORD

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BRAND & Co's Preserved Provisions and Specialities for Invalids.

MACKEY, MACKEY & Co., Manufacturing Chemists, 1 & 2 SOUTHWARK STREET, FLEET STREET, LONDON.

Human obesity pandemic

For the first time in human history, there are more overweight and obese people on the planet than people suffering from malnutrition. The prevalence of obesity has doubled since 1980, with over a quarter of British adults obese and almost two thirds overweight.

Obesity-related disease is common and well understood in people: globally, 44 per cent of diabetes cases, 23 per cent of ischaemic heart disease cases and seven to 41 per cent of certain cancers (endometrial, breast and colon) are attributable to obesity and being overweight. Increasingly, non-alcoholic fatty liver disease (NAFLD), polycystic ovary syndrome and musculoskeletal disorders, such as osteoarthritis, are also recognised as sequelae of obesity.

As well as the huge morbidity cost, there is an enormous financial burden associated with these conditions: an estimated 10 per cent of the NHS annual budget, or £9 billion/year (based on 1997 figures) is spent on obesity-related diabetes care (Diabetes UK 2012, Public Health England 2013, WHO 2013).

With those facts in mind, the hyperbole surrounding obesity as a human health problem seems justified.

Is obesity such a big deal in companion animals?

Earlier this year, the PDSA estimated that 25 per cent of the dogs seen at its hospitals in the UK were overweight. Others have suggested prevalence to be even higher, with at least a third of adult dogs and cats overweight and that figure rising to almost half when only dogs and cats between five and 10 years old are considered (Lund and others 2005, 2006).

Obese dogs die sooner and have a higher incidence of orthopaedic, cardiac, respiratory, urinary, reproductive and dermatological disorders, some cancers and anaesthetic complications. Obesity-related hyperlipidaemia is common and has been linked to the increased incidence of pancreatitis. In cats, the health risks of obesity are also well established; diabetes, hepatic lipidosis, urinary tract disease, lameness and dermatopathies are all more common in obese cats. As such, obesity represents a significant health and welfare problem for pet dogs and cats (Lund and others 2005, German 2006, Lawler and others 2008, Zoran 2010, Yeates and Main 2011).

It is noticeable that, although there is much overlap between the disease susceptibilities associated with obesity

Photograph: Zacarias Pereira da Mata/Shutterstock

Picture: The Kennel Club Arts Foundation

Photograph: Tomasz Markowski/Dreamstime

FIG 1: Some breeds, such as labrador retrievers, appear prone to developing obesity. In labradors, a shift from predominantly being used as a working gundog to now being a popular household pet has fuelled the obesity problem in this breed. However, a breed predisposition suggests that genetics are also important in the genesis of obesity in dogs, as in people . . . and owner attitudes play a key role, too

between dogs, cats and people, there are also notable differences. For instance, feline diabetes is predominantly a consequence of obesity-related insulin resistance, as in people. This contrasts with the picture in dogs where, although there is mounting evidence that obesity is associated with significant metabolic derangement, including insulin resistance, the links between obesity and the development of common diabetes are not well established (German and others 2009, Zoran 2010).

Perhaps the most striking interspecies difference concerns cardiovascular disease related to obesity. One of the biggest threats to human health from obesity comes from the high risk of future cardiovascular disease, conferred by the presence of a cluster of risk factors, including insulin resistance, hyperglycaemia, a characteristic dyslipidaemia and elevated blood pressure, collectively known as the 'metabolic syndrome' (Reaven 2007). Although these changes have all been shown to exist in obese dogs and cats, the life-threatening consequences, including atherosclerosis and stroke, are almost unheard of in these species (Tvarijonaviciute and others 2012, Osto and others 2013).

Finally, there are areas ripe for further investigation. For instance, the parallels between feline hepatic lipidosis and human NAFLD have yet to be thoroughly investigated (Verbrugghe and Bakovic 2013), and the reasons why some species or individuals appear protected from particular obesity-associated disease (such as atherosclerosis or diabetes in dogs) are equally fascinating.

Why does obesity cause disease?

The fundamental mechanisms by which obesity causes such a diverse range of diseases have been intensively studied. Consequently, there has been a radical shift from considering obesity as a condition where the mechanical effects of adipose tissue accumulation put a 'strain' on the body, to an understanding that adipose tissue is a metabolically active organ whose malfunction exerts effects by diverse and interlinked mechanisms.

Mechanical effects of fat accumulation certainly account for some obesity-related morbidity: increased wear and tear on joints cause or worsen osteoarthritis; fat deposits around the respiratory tract mechanically impede ventilation; increased pressure due to visceral fat worsens incontinence; and the presence of skin folds predisposes to dermatitis.

However, this simplistic view cannot account for the complex array of obesity-associated disease. Rather, we now understand that adipose tissue, far from being an inert depot where excess energy can be stored as lipid, is actually a dynamic endocrine organ whose function is impaired in obesity such that insulin resistance develops. It is this insulin resistance that is the fundamental pathophysiological disturbance that exerts wide-ranging consequences on different body systems.

Fat tissue secretes a number of important bioactive molecules, collectively known as adipokines. These include the hormones leptin and adiponectin, and numerous cytokines and related proteins.

The role of adipokines in the genesis of insulin resistance and related disease is debated. Both leptin and adiponectin have been proposed to enhance insulin sensitivity. In the case of leptin, it appears that this effect is predominantly via its effect on food intake, with some evidence of an effect on energy expenditure (Farooqi and O'Rahilly 2009). The picture is less clear for adiponectin and the debate continues as to whether failure of the action of adipokines is a cause or consequence of insulin resistance (Cook and Semple 2010). There has been a flurry of papers looking at adipokine levels in dogs over recent years; broadly, levels appear to change as predicted from other species, although the picture is not so clear for adiponectin (Radin and others 2009, Verkest and others 2011, Tvarijonaviciute and others 2012).

A final mention should go to brown fat. Long thought only to play a role in thermogenesis in small mammals and neonates and not to exist or function in adults, there is new evidence that it is not only present in adulthood but that it may also have a role in increasing energy expenditure and combating obesity in human beings and possibly other species (Vosselman and others 2013).

Overall, a picture develops of adipose tissue playing a complex role in normal physiology. When obesity develops, current understanding suggests that insulin resistance is the fundamental pathophysiological insult that has myriad consequences that, together, lead to obesity-related disease.

Development of insulin resistance

The link between obesity and insulin resistance is well established, but the mechanisms by which it develops are not entirely clear, despite intensive research. However, there is accumulating evidence to support the contention that the problem is due to adipose dysfunction, rather than the accumulation of body fat per se.

Specifically, there is evidence to suggest that the degree to which adipose tissue can expand is limited. This goes some way to addressing why some individuals experience obesity-related disease when only

moderately obese, while others can remain healthy despite extreme adiposity.

Once fat tissue has expanded to its maximum, governed by genetic and other factors, adipocytes will no longer take up triglycerides in response to insulin action. Subsequent ectopic lipid accumulation, particularly in the liver and muscle, causes lipotoxic insults, including insulin resistance and apoptosis (programmed cell death). Simultaneously, chronic low-grade inflammation of adipose tissue develops, with associated local and systemic release of proinflammatory cytokines. Accumulation of reactive oxygen species and mitochondrial dysfunction appear to further perpetuate the cycle. Together, these factors converge to activate intracellular signalling molecules that inhibit normal insulin signalling, leading to insulin resistance (Virtue and Vidal-Puig 2010).

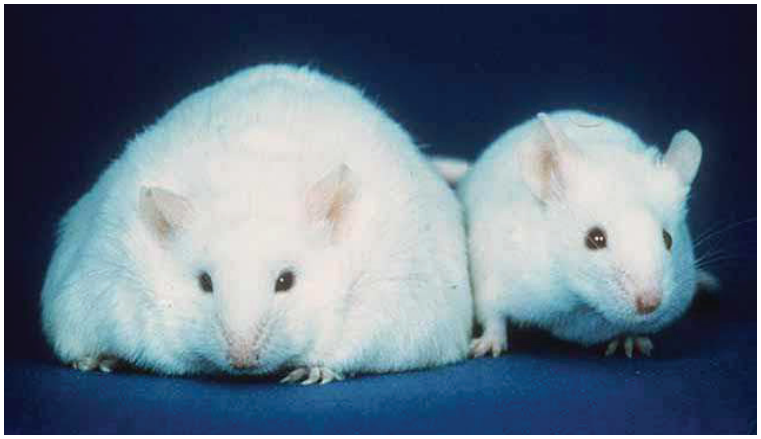

FIG 2: As in people, mice that are unable to produce leptin are hyperphagic (have increased appetite) and become severely obese very young. The mouse on the left is leptin deficient and the mouse on the right has been managed in the same conditions but is wild type

Far-reaching consequences

The complex molecular changes described above have knock-on effects throughout the body. Together, they represent a potent cocktail of endocrine dysfunction, ectopic lipid accumulation and inflammation, which are jointly implicated in leading to the clinical manifestations of insulin resistance.

For instance, consider the development of insulin-resistant diabetes, as seen in cats and obesity-related diabetes in human beings. In the initial stages of disease, in order to compensate for peripheral insulin resistance, there is an increase in insulin production by pancreatic β cells. Amylin, a protein co-secreted with insulin, aggregates causing local disruption of islets. Simultaneously, a combination of ectopic lipid accumulation in, and inflammation of, β cells further disrupts their function and eventually insulin production wanes such that glycaemic control is lost. Hyperglycaemia imposes further oxidative and other molecular stress on β cells, and over time more β cells are lost, insulin production drops further and frank diabetes develops (Prentki and Nolan 2006, Osto and others 2013).

Similarly, increased cytokine release from adipose tissue in obesity leads to a generalised proinflammatory state, probably contributing (along with mechanical effects) to osteoarthritis and inflammatory respiratory disease. The link between obesity and cancer is not well understood. The presence of chronic inflammation probably plays a role, but other important candidate mechanisms include the presence of elevated levels of insulin and other related hormones that act to stimulate cell division and angiogenesis and the presence of hyperglycaemia, which can favour the altered metabolism of neoplastic cells (Ford and others 2013).

Indulgent owners or hard-wired biology?

At a simplistic level, the genesis of obesity as the consequence of calorie intake in excess of calorie expenditure is obvious. And in a society where calorie-dense food – for people and pets – is increasingly affordable, and sedentary lifestyles more common, there are clear reasons why the prevalence of obesity is high. But dismissing human or animal obesity as a straightforward consequence of gluttony and laziness simply does not hold up to scrutiny under the spotlight shone by scientific research.

Rather, there is increasing understanding that appetite, satiety and energy expenditure are closely regulated homeostatic mechanisms, subject to influence by genetic and environmental factors. And that makes sense: since when does nature leave a critical homeostatic process like maintaining energy balance entirely to our conscious thought processes?

Lessons from genetics

Between 40 and 70 per cent of a person's tendency to gain weight is down to heritable factors (Day and Loos 2011). In dogs, strong breed predispositions to obesity suggest genetics are equally important. Labradors, for instance, regularly top the obesity tables (Edney and Smith 1986, Crane 1991). Are we really to believe that labrador owners are so much more exercise-averse and indulgent with food that a high proportion of their dogs develop obesity while owners of borzois and pointers are a virtuous bunch who carefully regulate their dogs' weight? No – it is far more plausible that the tendency to seek out food and eat to excess is hard wired in the biology of some breeds (Fig 1).

But how do genes influence weight gain? Genome-wide studies in thousands of people show common obesity is the product of many small incremental effects of different genetic variations (Day and Loos 2011). The situation is likely to be broadly similar in dogs, although the unusual way in which dogs have been selectively bred means that, within a single breed, obesity is likely to be governed by relatively fewer genetic variants of larger effect (Karlsson and Lindblad-Toh 2008).

Greater insight into the mechanisms of bodyweight regulation has come from the study of people with severe, early-onset obesity. Although very rare, understanding how physiology is perturbed when a monogenic disorder ablates the function of key players in a biological system is very informative.

The first such syndrome to be discovered was in patients carrying mutations in the leptin gene who had early-onset obesity, predominantly due to increased food intake (Fig 2). Remarkably, treatment with exogenous leptin rendered patients slim, with normal appetites. Similarly, patients with mutations in other genes involved in coordinating the hypothalamic response to energy balance display obesity as prominent components of complex phenotypes (Farooqi 2011).

A recurring message from genetics is that failure of one component of the networks that govern food intake and energy expenditure often results in a compelling behavioural phenotype, with increased food-seeking behaviour and decreased satiety responses to food. This provides evidence that even very complex behaviours can have a genetic influence, and undermines those who claim that food-seeking behaviours (or greed, as it is often pejoratively termed) are simply evidence of social conditioning or weak morals.

Controlling energy balance

Current understanding is that neurohormonal control centres in the hypothalamus and brainstem are master regulators of appetite, satiety and energy regulation (Fig 3). Signals generated peripherally in response to short-term changes in circulating nutrients or the presence of food in the gut, and long-term changes in energy stores, are integrated in the hypothalamus, that in turn transmits neurohormonally to the brain and body. Complex responses result, which can either be emotional/behavioural (satiety, hunger,

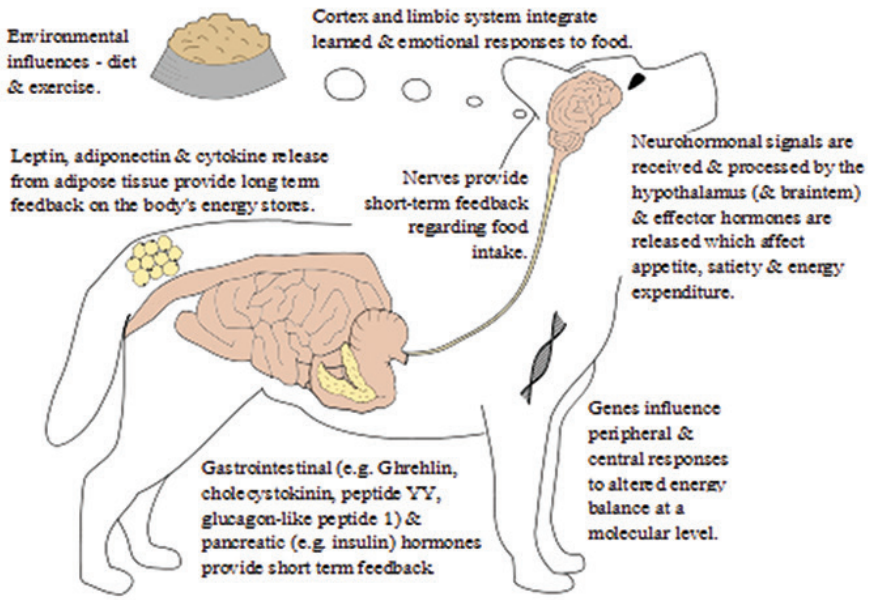

FIG 3: Key players in the regulation of energy balance, appetite and obesity

food-seeking behaviour) or physiological (altering energy expenditure).

Within the hypothalamus, neurons sense peripheral signals and release peptides that act on melanocortin receptors, exerting powerful stimulatory or inhibitory effects on appetite and energy expenditure. Multiple ligand and receptor combinations are involved in coordinating the hypothalamic response, with serotonin and gamma-aminobutyric acid signalling important in generating a reward response to food (Yeo and Heisler 2012).

What merit is there in understanding this system? I think the answer to that question lies in the fact that acknowledging its complexity should lead us away from considering obesity in our patients as some kind of failing on the part of their owners, and towards a more thoughtful and effective way to address obesity as a clinical problem.

Tailored treatment strategies

Perhaps the best example of how applying basic biology can aid clinical outcomes comes from the pet food manufacturers. They have long integrated knowledge about how signals are generated in response to gut fill and nutrient release to develop foods to aid weight reduction (German 2006). Similarly, specialist diets for diabetic cats reflect an understanding of the pathophysiology of insulin resistance and glucose homeostasis (Zoran and Rand 2013).

In contrast, most drug interventions in obesity have tended to address the energy in/energy out equation rather crudely. Historically, human obesity treatments, such as thyroid hormone, aimed to increase energy expenditure, but use fell out of favour given their side effects. Later drugs aimed to reduce the absorption of lipids from the gut, as do the currently available drugs for obesity treatment in dogs, and the one available for use in people. However, the canine drugs appear to have a parallel effect of reducing appetite, although the precise mechanism by which that occurs is unknown. Drugs in development, however, apply a more sophisticated understanding of energy homeostasis. For instance, glucagon-like peptide 1 receptor agonists mimic the action of a gut hormone that is released after a meal and reduces appetite as well as improving insulin sensitivity (Gossellin and others 2007).

Helping owners help their pets

Understanding that the drive to eat to excess is part of hard-wired biology may enable us as clinicians to counsel owners of obese pets more effectively. I have found owners are encouraged to work harder at making the necessary diet and lifestyle interventions by being told that their pets' drive to eat to excess is real and physiological.

Ideally, we would prevent obesity developing in our patients in the first place. There is widespread acceptance that neutering is associated with obesity, but many vets and most owners appear to treat this as an unavoidable association; perhaps it would help both groups to know that neutering can reduce energy expenditure by up to a third (Fettman and others 1997, Jeusette and others 2004). It isn't neutering that makes the animal fat – but feeding it the same amount of food as it had before surgery.

Once obesity has developed, how can we encourage effective and sustained weight loss? In busy clinics, I've certainly been a culprit of doling out sensible but blunt advice to 'just feed less and exercise more'. Most readers will be pet owners and well aware of how powerful the 'big pleading eyes treatment' is in soliciting treats, and how busy lifestyles can mean dog walking comes second to other tasks. Changing those habits is hard and it is therefore perhaps no surprise that instructions based on making their pets hungrier are going to be hard to follow.

Instead, it's worthwhile spending a few minutes explaining that a drive to eat to excess is a recognised challenge, worse in some individuals, that there are real risks to obesity and benefits in weight loss, and how your recommendations can help their pet lose weight. Specifically, explaining that specialist weight reduction diets are formulated to improve satiety despite decreasing calorie intake (while encouraging loss of fat over lean tissue and providing adequate nutrients) may encourage their use. Similarly, pointing out that after a period of calorie restriction, metabolic rate slows and the maintenance requirement therefore drops is also of merit if weight loss is to be maintained in the long term (German and others 2012).

Exercise has been shown to have multiple benefits to obese patients. Not only does it increase energy expenditure during the exercise, but also it is maintained afterwards, and independent of weight loss there are beneficial effects on insulin sensitivity (Roberts and others 2013) that may confer particular benefits to prediabetic cats. Once again, explaining to owners that the effort to increase their pet's exercise has more profound effects than a few short-term calories burned can motivate them to follow your advice.

Over to you!

Pet owners have been in receipt of sensible advice that feeding 'plain, wholesome food will be sufficient to keep [them] in the best of health, plus sufficient exercise' (Tailwagger 1935) since *Veterinary Record* was in its youth, but despite that, there has been evidence of mounting concern in the veterinary profession about pet obesity since related articles first started to appear in the journal in the 1960s.

As the journal celebrates its 125th year of publication, I hope a shift from regarding pet obesity as an unappealing consequence of lax animal management to a recognition that it represents a complex physiological derangement with genetic and endocrine causes will help us as clinicians deliver that old message more effectively and so improve the health and welfare of our patients.

■ Eleanor Raffan is currently collecting samples for a research project that aims to find out why labradors are so food motivated. Any vets interested in getting involved are asked to visit www.GOdogs.org.uk

GOdogs

Investigating the Genetics of Obesity in Dogs

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Articles published in *Veterinary Record*'s 125th anniversary series so far have considered:

- Cattle lameness (*VR*, January 26, 2013, vol 172, pp 92-95)
- Dermatology in companion animals (*VR*, February 23, 2013, vol 172, pp 201-207)
- Articular disorders in dogs (*VR*, March 30, 2013, vol 172, pp 332-335)
- Equine surgery (*VR*, April 27, 2013, vol 172, pp 442-446)
- Imaging (*VR*, May 25, 2013, vol 172, pp 546-551)
- Cardiology (*VR,* June 29, 2013, vol 172, pp 678-682)
- Parasitology (*VR,* July 27, 2013, vol 173, pp 89-91)
- Equine infectious diseases (*VR,* August 31, 2013, vol 173, pp 186-189)

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