

Review

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Veterinary behavioural medicine: a roadmap for the 21st century

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**Abstract**

New areas of endeavour succeed either because they make a compelling case for their worth within an existing paradigm or because a new generation is raised for whom a once heretical paradigm is now the norm. It is difficult to tell which route will lead to the acceptance and growth of veterinary behavioural medicine, but the latter seems likely and, perhaps, necessary. As one of the newest specialities in veterinary medicine, behavioural medicine may suffer more than other specialities from an unclear identity because so many disparate groups who are not rooted in veterinary medicine have participated in its evolution.

There is also the issue of data. Behavioural medicine is often cited as a field for which there are very few hard data; however, it is also a field for which data collection can be difficult and time consuming for non-trivial questions. This is because behaviour is the ultimate integrator of all organ system responses, and as such, is a dynamic outcome resulting from the interactions of complex mechanisms. Understanding such systems is difficult, but progress can occur if an attempt is made to understand all the mechanistic levels that contribute to behavioural patterns and behavioural conditions. For this to happen we need a paradigm shift that moves us away from the medical paradigmatic model and towards a new paradigm that is based on hypothesis formulation and testing between interacting levels of mechanism. Clinical impression and expertise based on outcome must be replaced by a scientific method that provides for phenotypic definitions that are coupled to putative underlying, interacting mechanisms that can be evaluated. Only in this manner can phenotypic description and its clinical application in veterinary behavioural medicine keep pace with advances in molecular biology and genetic epidemiology that deal with genetic liabilities and vulnerabilities inherent in multi-gene and major gene effects.

This paradigmatic shift has the potential to revolutionize the way we view diagnoses in medicine, in general, but has particular implications for the care needed to characterize problematic and truly abnormal behavioural conditions. This approach also has the advantage of alerting us to when we do not know something, and forcing us to consider the needs of our patients on their own terms. A more rigorous scientific approach in this field could also go a long way in making us more humane.

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**1. Introduction**

This paper is the result of an experiment designed to learn whether one can convene a research meeting that can render credible the concept that veterinary behav-

oural medicine is a true scientific discipline. Perhaps it is not surprising that veterinary behavioural medicine is still struggling for professional and academic acceptance: the field is a mere 25 years old. Compared to disciplines like surgery, cardiovascular medicine and oncology, disciplines like psychiatry are still considered poor step-children in human medicine. So, the extension that the analogous veterinary field is somehow also

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‘soft’, ‘touchy-feely’, and heavy on soliloquy and empathy but short on data may be logical.

For those of us who believe that compassion is best based on solid data that allow firm action and redress, the time has come to elevate veterinary behavioural medicine to a cutting-edge discipline. This can only be done by establishing a paradigm founded not on clinical impression, but on the scientific method. Because the field is so new we have the advantage of being unencumbered by hundreds of years of approaches and philosophies that have divided the human medical world into physicians versus scientists. For anyone who disbelieves that this rift exists, the recent focus on ‘evidence-based medicine’ in both the human and veterinary medical fields should be convincing. Veterinary behavioural medicine is uniquely positioned to create a new paradigm where care and treatment is the result of rigour in scientific enquiry and application of the resultant data.

### 1.1. History

Until the early 1980s most of those interested in helping companion animals with undesirable behaviours were not veterinarians. This task fell almost exclusively to dog trainers, those with a background in psychology, those who had studied animal behaviour, ethology, or sociobiology, and those who were good-hearted and generous but without formal training. The only thing that has changed in a quarter of a century is that veterinarians now also participate in this endeavour.

In the early 1990s the American College of Veterinary Behaviorists (ACVB; [www.dacvb.org](http://www.dacvb.org)) began with eight charter diplomates: seven academicians and one research laboratory scientist. In 1995, the first class of candidates sat the exam for board certification, and as of the summer of 2004 there were 36 board-certified specialists members of ACVB, including two Canadians and an Australian. Most diplomates of the ACVB are now private practitioners, a trend that is a concern given the goals stated in this paper.

In Europe in 2003 the first charter diplomates, all of whom come from the practitioner community, were granted permission to create the European College of Veterinary Behavioural Medicine (ECVBM-CA; [www.ecvbm-ca.org](http://www.ecvbm-ca.org)). The Royal College of Veterinary Surgeons (RCVS) recognized one academic veterinary animal behaviourist as a specialist in 2004. In Australia, two veterinary behaviour specialists have been registered in the past 15 years.

Prior to the creation of these specialty veterinary organizations, even ‘clinical’ behavioural concerns were primarily the purview of non-veterinarians. The Animal Behavior Society (ABS) in the USA has separate certification processes for those holding a PhD and, or a veterinary degree, and those with a Master’s

degree. Because ‘case reports’ describing ‘a rationale for diagnosis and treatment plans’<sup>1</sup> are one requirement for certification, the concern is that such certification procedures have encouraged the practice of medicine without a licence. In the UK, Southampton University has had a certificate programme in applied animal behaviour for a number of years, and both veterinarians and non-veterinarians can earn certificates. Prior to the creation of any veterinary certification groups in the UK, a number of other groups were created to meet the need of pet owners who were distressed about their pets’ behaviours including the Association of Pet Behaviour Counsellors (APBC; [www.apbc.org.uk](http://www.apbc.org.uk)) in the UK and the Association of Pet Dog Trainers (APDT; [www.apdt.org](http://www.apdt.org)) in the USA. While veterinarians were and are members of these groups, the vast majority of the membership is non-veterinary. Many specialists would agree that much of the work done by these individuals is now well within the purview and charter of veterinary medicine, but that the groups can work together (Heath, 2001). As specialty development waxes, the practice of medicine by those not licensed to do so wanes.

Non-specialist veterinarians have also founded their own groups in the USA, Europe, and the UK. The American Veterinary Society of Animal Behavior (AVSAB; [www.avma.org/avsab](http://www.avma.org/avsab)) requires that all voting members be veterinarians, while corresponding members can be licensed veterinary technicians or those with graduate degrees. In the UK, the Companion Animal Behaviour Therapy Study Group (CABSTG; [www.cabstg.org](http://www.cabstg.org)) has a comparably broader membership, as does the European Society for Clinical Veterinary Ethology (ESCVE; [www.escve.org](http://www.escve.org)), with the majority of members now being veterinarians.

Must the academician versus veterinary surgeon/practitioner dichotomy exist within the specialty? Probably not, but we must now consciously consider the effect on the field of such demographics. As discussed, the composition of the American specialty college is now weighted heavily towards those in practice. This change carries with it a proportional decrease in university based residency and post-graduate programmes. In the past 25 years there has been no net growth in full-time veterinary behavioural medicine positions and programmes in American universities, although some have relocated. One wonders where the next generation of veterinary scientists in the field will be trained. This issue is important for one major reason: rigour in the scientific method cannot become a life-long habit without the requisite training commitment and practice. Without this, research cannot advance as the field advances because

<sup>1</sup> Case report Guidelines, 2004, ABS board of professional certification.

there will be gaps in technical and theoretical knowledge that will hamper execution and funding efforts.

In veterinary behavioural medicine, all academic specialists engage in some sort of clinical work. The converse is not true: most practising veterinary surgeons do not engage in research in the USA, although stellar research can be done by such individuals (Ciribassi et al., 2003). Both academicians and practising veterinary surgeons are well aware of the extreme need for state-of-the-art care in veterinary behavioural medicine, and we all agree that patient care is paramount. Yet, in a pattern that appears universal, veterinary schools and colleges in North America have been reluctant to put veterinary behavioural medicine into a full-time curriculum. This is despite the fact that the most common questions from clients involve their pets' behaviours, and veterinarians routinely put behavioural training in the top 3–5 needs that they would like to fulfilled at their alma mater.<sup>2</sup>

Not only are we not training veterinary surgeons to view behavioural medicine as an essential, core facet within veterinary medicine, but if we do not invest in the research and training of more specialists, patient care needs will cease to be met within a generation. Research is a more complex topic, but veterinary schools and colleges would do well to remember that the best, most forward thinking and well-funded research is not done in an environment where one has no colleagues. Accordingly, one of the goals of the Dogs Trust meeting on *Advances in Veterinary Behavioural Medicine* is to demonstrate that not only is this new specialty relevant for veterinary colleges and schools and essential for delivering the best possible patient care, but that it has arrived within the research community, generating publication and funding at rates usually found in much more established fields.

### 1.2. *The unmet need and effects for problem pets and those viewed as problems*

In the 1980s orthopedic surgeons taught that 'pain is a great immobilizer'. Two decades later we are appalled by this notion, yet our change in attitude has been accompanied by painstakingly obtained, only moderate progress in understanding and assessing pain (Pascocoe, 2003; Short, 2003), as is also true in human medicine (Scholz and Woolf, 2002).

Although we cannot directly measure pain, we no longer deny its existence or that of the associated nociceptive processes. We should not discount the equally invisible and equally real pain caused to animals and clients, alike, because of behavioural problems. Rather than ask whether we can understand behavioural problems as a form of behavioural pain and suffering, the

vast majority of clients and veterinarians, wittingly or not, engage in a terminology and thought process that is rooted in an adversarial relationship with the animals who share their lives.

Physical pain is deemed as 'real', afflicting innocent patients; behavioural pain is often thought to be someone's fault or the result of a deeply flawed character. We still hear about the need to 'dominate' our dogs, and to 'not give in' to our pets. Both of these admonitions are logical outgrowths of ill-used and misplaced terminology where the failure to distinguish between a pushy or 'dominant' dogs and one afflicted with 'dominance aggression' leads too often to mistreatment for both (Overall, 2004a).

Behavioural problems are still the most common reasons pets are relinquished, abandoned, or euthanased in the USA, Australia, and Canada (Houpt et al., 1996; Marston et al., 2004; New et al., 1999; Salman et al., 1998, 2000; Scarlett et al., 1999, 2002). Such problems can range from serious and pathological aggressions and anxieties to normal, species typical behaviours that the client may find offensive (e.g., pulling when on a lead, scratching to mark, etc.). Even when the problem is relatively minor, if there is an external, non-behavioural justification for relinquishing the pet, the minor behavioural problem becomes the decisive issue (Shore et al., 2003). Retention is most likely when clients feel that the dog has basic manners and when they have realistic expectations for the individual and the breed (Marston and Bennett, 2004). Accordingly, it is incumbent upon us to impose science on a field that has long resisted its advance and to begin to understand the neuro-behavioural pathology of such problems in domestic animals.

## 2. Terminology and diagnosis

What we call something is important in this field as in no other. This is interesting in light of an enthusiastic effort by the International Liver Study Group<sup>3</sup> to formalise and codify the terminology used in their field because circumstances designed to foster the same discussion in veterinary behavioural medicine have languished<sup>4</sup> (Overall, 1994, 1997a,b).

Diagnoses are not diseases; correlation is not causality. Conditions for which there is putative etiologic and pathophysiological heterogeneity (multi-factorial disorders) are complex, and there is nowhere that this is more true than topics of interest in veterinary behavioural medicine. Our problem is, in part, one of scale.

When one diagnoses a problem related to fear, anxiety, or aggression one is doing so at the level of the

<sup>2</sup> *DVM Newsmagazine* in-house survey, 2002.

<sup>3</sup> WSAVA 2002, 2003.

<sup>4</sup> T. De Keuster, ESCVE research meeting, Granada, Spain 2002.

phenotypic diagnosis; when medication is used such conditions are treated primarily at the neurochemical level. There is also secondary treatment at the phenotypic level owing to both the direct behavioural changes medications can cause, and the extent to which these changes enhance the acquisition of learning associated with behavioural modification (King et al., 2000, 2004; Mills and Ledger, 2001).

There are only three reasons for desiring clear definitional criteria that will provide some uniformity in what have been variously called phenotypic/functional/phenomenological diagnoses:

- (1) If the criteria are careful, restrictive but accurate, agreed upon, and clear, people can talk to each other. Good diagnostic criteria mean that multi-centre drug trials are a possibility because the patients will, perforce, be guaranteed to suffer from the same condition. Good diagnostic criteria allow good epidemiological studies that can detect the effects of culture, treatment, and various environmental contributions to the biological conditions. Good diagnostic criteria allow comparison among breeds, individuals of which may manifest the same signs of the same condition, but perhaps not for the same reason (Masuda et al., 2004; Nimi et al., 1999; Parker et al., 2004).
- (2) If one is interested in treatment at the phenotypic level, reliance on an agreed definition allows clients to understand and avoid circumstances that may provoke the problem, even if the animal has never actually experienced the particular provocative stimulus which triggers the behaviour that the client wishes to avoid. Avoidance decreases the number of times that the inappropriate or undesirable behaviour can be reinforced, and hence helps the patient not to learn to become more abnormal (Lueger et al., 2000). In dogs, as in humans, intensity of the problem increases with time (Overall et al., 2001; Overall and Dunham, 2002). When clients understand this scenario compliance increases, and dogs and cats can be safely kept while they undergo treatment and improve.
- (3) If one is interested in mechanism at any reductionist level, one must be using a diagnosis that is based on strict definitional criteria (Overall, 1997a,b). We

now know from human and rodent behavioural studies, and from the logical extension of canine studies, that to obtain a full understanding of a diagnosis, all ‘mechanistic levels’ must be evaluated (see Table 1 and Figs. 1 and 2). The *context* for the evaluation of the relative roles of genes (Fyer and Weissman, 1999; Nijhout, 2003), receptor molecular biology, neurochemistry, and neuroanatomical localization must be the actual behaviour, the phenotypic diagnosis.

This approach will allow us to deal with the paradox that the act of exhibiting the behaviour alters other mechanistic levels that then go on to alter the phenotype (see Fig. 1). Behaviour is complex, and not all mechanistic levels interact on the same time scale. This means that standard medical diagnostic paradigms do not enhance our understanding of behavioural pathology (see Figs. 1 and 3). Clear use of terminology can help to make apparent the parts of these phenotypic diagnoses that display consistent patterns, so that we can understand and separate them from those that are more complex.

Non-specific signs or descriptors are often erroneously or carelessly used as a diagnosis. By viewing a diagnosis mechanistically as a hypothesis to be tested, it is possible to begin to define and understand abnormal behaviours at a variety of levels that include, but are not restricted to, the phenotypic diagnoses that are most commonly employed (see Table 2 and Figs. 1–3). In this example the variants in the condition are due to some difference in environmental response. This could be a purely phenotypic effect (abnormal variant B). Alternatively, the effect could be due to learning and long-term potentiation (in which case the molecular level is affected – abnormal variant A); this molecular effect also affects neurochemistry. The effect could also be one of neurochemistry, without affecting the molecular level (abnormal variant C).

Various approaches to diagnostic criteria include those based on effects of relative developmental stage on behaviour (Pageat, 1998), and those combining ontogeny with ethology and other relevant aspects (Odenaal, 1997). Both of these presuppose mechanism within the classification of phenotype. In contrast, use of the algorithm of necessary and sufficient criteria avoids confounding mechanism with definition, and definition with

Table 1

Understanding patterns of behaviour within levels of a mechanistic approach – note that none of these levels are independent, the first four are very dynamic, an action can originate at any level that then affects the other levels, and the extent to which they interact is a function of the genetic response surface and learning (modified from Overall, 1997a, 1997b; Overall et al., 2004)

A. Phenotypic diagnoses: the actual behaviours; must meet necessary and sufficient terminological criteria

B. Neuroanatomical diagnoses

C. Neurochemical/neurophysiological diagnoses

D. Molecular diagnoses

E. Genetic/genomic diagnoses — The genetic or genomic response surface sets boundary conditions for what is possible.

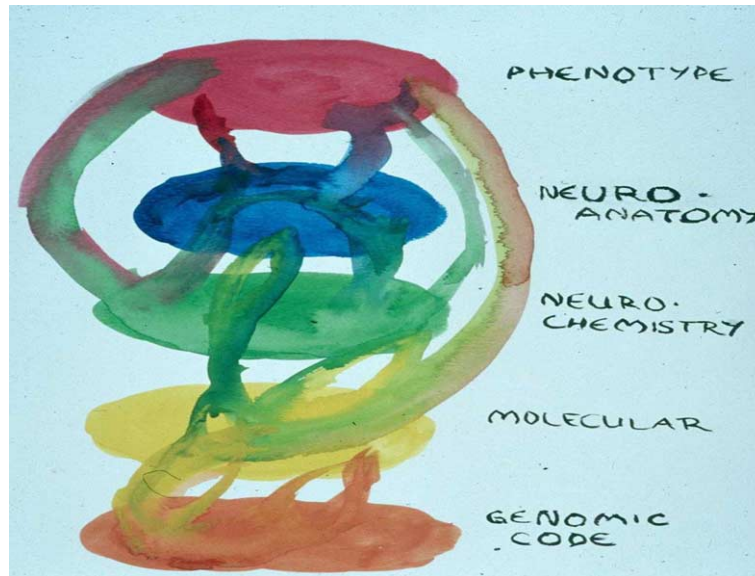


Fig. 1. This graphic illustrates the relative ‘response surfaces’ that exist for each of the mechanistic diagnostic levels discussed in the text. Note that phenotype can be affected directly or indirectly. The extent to which the different levels interact directly or indirectly could be a function of intensity, duration, or type. of stimulus. There is no way to know these effects in absence of specific data collection. Here, the genomic code provides that set of choices that could effect – but not necessarily will effect – molecular and neurochemical expression of behavioural phenotypes. In essence, the genomic response surface acts to define boundary conditions.

MODEL OF VARIOUS RESPONSE SURFACE INTERACTIONS

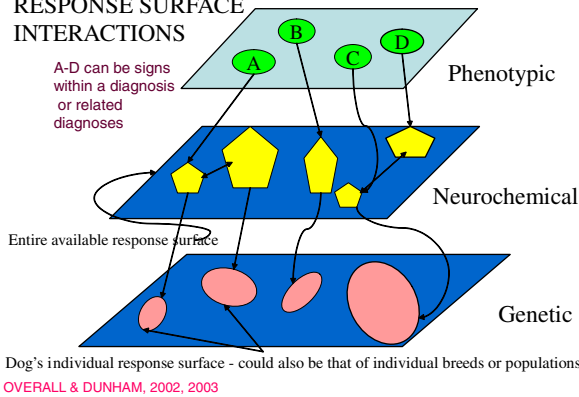


Fig. 2. In this schematic only 3 of 5 discussed mechanistic levels are represented as 3 different response surfaces. Situation B is the one everyone hopes to find: here one gene is responsible for one neurochemical change and that change maps uniquely onto the problem behaviour. The real world is a tad more complex than this. In example D, one set of genes gives rise to one set of neurochemical responses that then alters into another neurochemical response set. Each set of neurochemical responses gives rise to two separate phenotypes ( C and D). So in this case, the same genetic background can produce two diagnostic groups acting through two neurochemical mechanisms. For phenotype A two neurochemical mechanisms are also involved, but they are each the result of two separate genes that then produce two neurochemical responses that interact.

non-specific signs (Overall, 1994, 1997a,b). This approach allows the number and intensity of non-specific signs to be used as a gauge for the severity of the condition, or to act as a flag when there can be variable, non-overlapping presentations of the same condition.

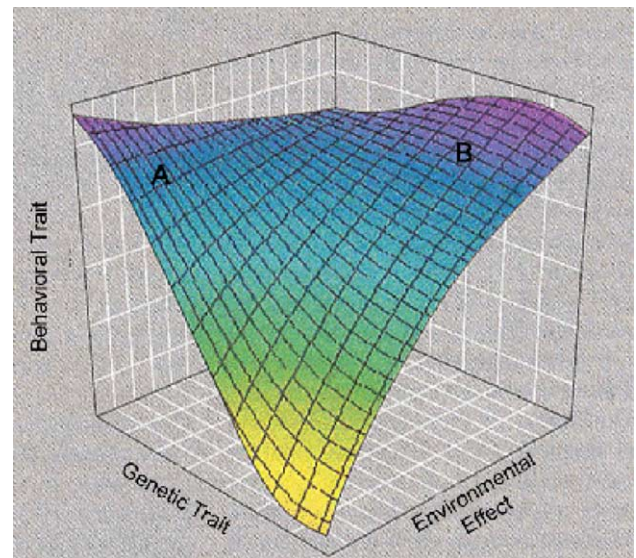


Fig. 3. A model, complex, non-linear response surface that predicts what a trait or phenotype will look like given the effect of a certain gene and the effect of a certain environment. Note that at some points on this response surface the phenotype would be indistinguishable, even given wildly different environment and gene effect, whereas in other regions of the response surface a small environmental or genetic change can, alone, have a huge effect. In this example, A and B are genetically different and have respond differently to environmental factors, but we cannot tell any of this from the phenotype. This is the question we are always asking when we seek to understand temperament in dogs, for example: to what extent does the environment in which the dog lives display any genetic liability for any behaviour? Simple, but specific examples for the outcome of this question are shown in Figs. 4,5 (figure modified from Nijhout (2003) with permission).

Table 2

Example emphasising the importance of using all mechanistic levels in diagnosis: simple effects of environmental response (modified from Overall, 1997a, 1997b; Overall et al., 2004)

	Phenotype			
	A (abnormal variant A)	B (abnormal variant B)	C (abnormal variant C)	D (normal)
Neuroanatomical variant	I	I	I	I
Neurochemistry	a	b	a	b
Molecular products	I'	II'	II'	II'
Genotype	a'	b'	b'	b'

In human psychiatry these presentations are often called 'endophenotypes' (Gottesman and Shields, 1972; Smoller and Tsuang, 1998; Tarantino and Buchan, 2000). It seems likely that the set of signs that two phenotypic groups exhibit in common reflect phenotypic manifestations of final common pathways. The more concordant the signs or data from clusters of groups evaluating different mechanistic levels, the more likely the identification of a final common pathway.

Only an integrated diagnostic and experimental approach – as discussed here – can provide the power to suggest and identify these pathways, while identifying problematic phenotypic diagnoses and correcting them. For example, once the definitional criteria are met, condition A could sort into two phenotypic groups based on treatment response. In the most simple scenario group 1 responds only to drug 1 and group 2 responds only to drug 2, although behaviourally the groups are indistinguishable. A pattern like this would hint that 2 underlying mechanisms are functioning (see Fig. 4). These mechanisms may become more apparent when a mutation appears (Fig. 5).

In another variant of this example, the definitional criteria are met (e.g., everyone has separation anxiety), but group 1 most commonly displays signs 1–3 (urination, defecation, and vocalisation) and group 2 displays signs 3–5 (vocalisation, destruction, and salivation). The question now becomes whether shared or separate mechanisms contribute to these clusters. Neuroanatomical and cellular examination of involved brain regions suggest that such clusters reflect different regional mechanisms (Davis, 1997). Alternatively, if these clusters are truly wholly separate at all levels of mechanism, one could rationally argue that these are two truly phenotypically separate diagnostic conditions, and that sign 3 is a truly non-specific, non-informative sign for this level of inquiry.

The implementation of "necessary and sufficient" criteria is a refinement over descriptive definitions of terms, providing qualitative, and potentially quantitative, exclusion criteria. Such criteria allow for uniform and unambiguous assessment of aberrant, abnormal, and undesirable behaviours. Inherent in an assignment of a diagnosis is the consideration that the behaviour is *not*

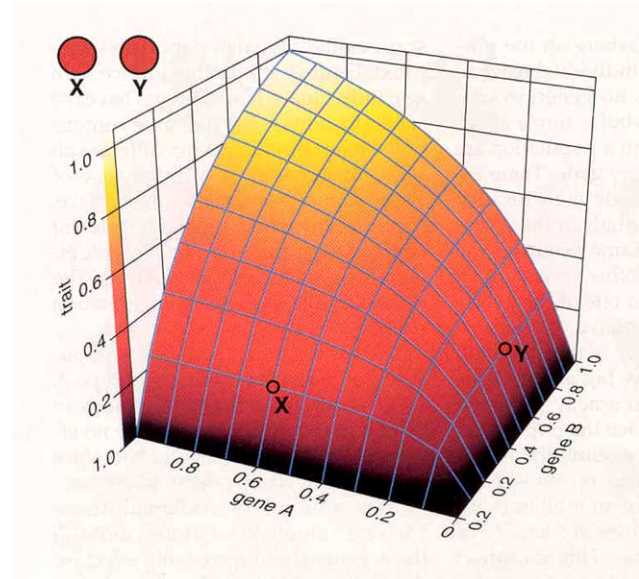


Fig. 4. The specific circumstance where two factors can be very different (in fact, here the effects of each gene on either phenotype are the opposite of the other) but have an equal and indistinguishable phenotypic effect, given the shape of the response surface. Here the phenotypes/diagnoses/behaviors are represented by X and Y (figure from Nijhout (2003) with permission).

a species or breed typical or normal behaviour, that it is *not* a normal behaviour associated with a developmental stage or ontogeny, and that it is *not* a reasonable response to an abhorrent but inescapable environment or situation.

A *necessary* criterion is one that must be present for the listed diagnosis to be made. A *sufficient* criterion is one that will stand alone to singularly identify the condition (Table 3). Sufficiency, like accuracy, is an outcome of knowledge: the more we learn about the genetics, molecular response, neurochemistry, and neuroanatomy of any condition and its behavioural correlates, the more succinctly and accurately we will be able to refine the sufficient criteria. The purpose of a diagnosis is not so that we have a concept etched in stone; the purpose is to have a hypothesis that can be tested mechanistically and that can be altered or refined as a function of increased knowledge associated with

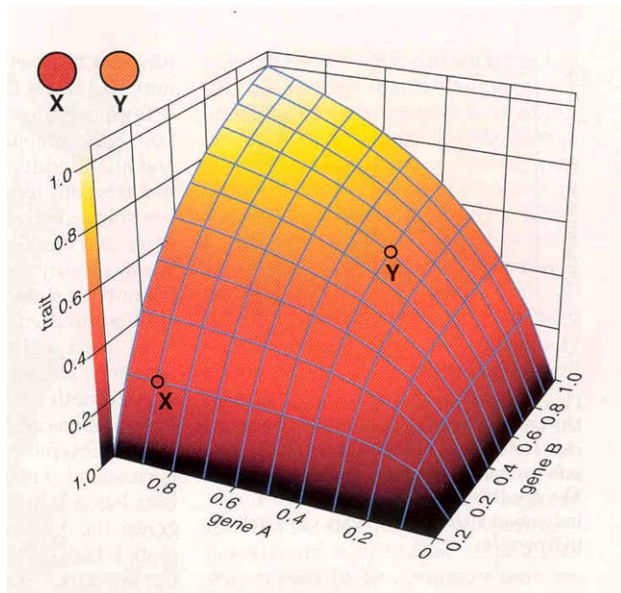


Fig. 5. The specific circumstance where one factor changes, but the other remains constant. Here only the effects of gene A are changed. A change in three units for gene A does not change phenotype X from Fig. 4, above, but the effect of a three unit change on phenotype Y is profound. This is the effect of the complex genetic interactions that define the non-linear response surface. (figure from Nijhout (2003) with permission).

data collection. Simply, we need to frequently re-visit what we know or think we know about diagnoses.

Although criteria are not synonymous with a compendium of signs associated with the condition, as discussed, the pattern by which the signs cluster will help in defining heterogeneity of the underlying afflicted population, will identify potentially important endophenotypes, will permit epidemiological studies to be executed, and will allow tests of multiple causality of underlying mechanism to be conducted.

Implicit in this approach is that there is no known underlying physical or physiological reason for the behavioural problem *and* that physical and physiological “causes” have been ruled out. In other words, the way we recognize all illness in animals involves a change in behaviour: the cat either vomits or she does not. However, behaviours that have proximal physical ‘causes’ (e.g., the cat was poisoned) may act as non-specific signs (e.g., vomiting) and should be recognized as such rather than being forced into an interpretation that requires a non-proximal ‘cause’ (e.g., feline bulimia) when this is not the most parsimonious explanation. It is also important to remember that classifications as discussed here represent true behavioural diagnoses, not just descriptions of a behavioural event (i.e., impulse control aggression can *only* be a diagnosis for an abnormal behaviour, but many behaviours can be ‘impulsive’; inter-dog aggression can be both a diagnosis and description).

This approach is similar but not identical to that taken by the American Psychiatric Association for the Diagnostic and Statistical Manual, the WHO diagnostic guidelines, etc. Required diagnostic criteria are imbedded within descriptions of human conditions, but because numbers of patients examined are huge in human psychiatry, subgroups of patients can be characterized by non-specific signs, demography, treatment responses, etc. Unfortunately, many diagnoses in human psychiatry are now actually based on non-specific signs, accruing a label that may not reflect the biological reality. This failure is due, at least in part in the USA, to the need to have a diagnostic code to receive payment, and it is one reason that genome scans utilizing diagnostic codes have produced so little useful information. Behavioural medicine is one area where veterinary medicine could take a leading role. The field is so new that diagnostic biases are not deeply entrenched, yet the field is developing at a time when neurobehavioural, molecular,

Table 3

Necessary and sufficient conditions for selected behavioural diagnoses discussed in the text (adapted from Overall, 1997a, 1997b, 2004a, 2004b)

*Behavioural diagnosis:* impulse control aggression

*Necessary:* abnormal, inappropriate, out-of-context aggression (threat, challenge, or attack) consistently exhibited by dogs towards people under any circumstance involving passive or active control of the dog’s behaviour or the dog’s access to the behaviour

*Sufficient:* intensification of any offensive aggressive response from the dog upon any passive or active correction or interruption or control of the dog’s behaviour or the dog’s access to the behaviour

*Behavioural diagnosis:* Noise phobia

*Necessary and sufficient conditions:* Sudden and profound, non-graded, extreme response to noise, manifest as intense, active avoidance, escape, or anxiety behaviours associated with the activities of the sympathetic branch of the autonomic nervous system; behaviours can include catatonia or mania concomitant with decreased sensitivity to pain or social stimuli; repeated exposure results in an invariant pattern of response

*Behavioural diagnosis:* obsessive-compulsive disorder

*Necessary condition:* repetitive, stereotypic motor, locomotory, grooming, ingestive, or hallucinogenic behaviours that occur out-of-context to their “normal” occurrence, or in a frequency or duration that is in excess of that required to accomplish the ostensible goal

*Sufficient condition:* as above, in a manner that interferes with the animal’s ability to otherwise function in his or her social environment

*Behavioural diagnosis:* separation anxiety

*Necessary conditions:* physical or behavioural signs of distress exhibited by the animal *only* in the absence of, or lack of access to the client

*Sufficient conditions:* consistent, intensive destruction, elimination, vocalization, or salivation exhibited *only* in the virtual or actual absence of the client; behaviours are most severe close to the separation, and many anxiety-related behaviours (autonomic hyperactivity, increased motor activity, and increased vigilance and scanning) may become apparent as the client exhibits behaviours associated with leaving

and genetic tools have never been more accessible. The conditions that are of interest in veterinary behavioural medicine do not have to be exact analogues of human conditions for this type of attempt at classification to be meritorious (Overall, 2000; Shekhar et al., 2001).

The first publication of necessary and sufficient criteria (Overall, 1994) was intended to provoke a discussion, not to set a standard. The silence was deafening. Subsequent compilations (Overall, 1997a,b,c, 2004a,b, 2005) have produced some discussion, but the focus has unfortunately been on less relevant topics, like philosophical schools of thought (Pageat, 1998; Mills, 2002), rather than on biology. The approach has been taken in more profitable directions in human psychiatry in response to frustration about ‘treatment failures’ in complex conditions (Castellanos and Tannock, 2002). That said, comparisons of data predicated on this classification should engender revisions and refinements of the classification. The classification, itself, is not important: the extent to which it provides a structured, logical, heuristic tool for the development of thought in the field is important.

The terminological debate also includes pitfalls about labels. If what we call something affects the way we think about it, then what we call it is essential. By being careless we have done harm. Consider the issue of ‘dominance’ in dogs. We can no longer leave unaddressed the dangers of employing a terminology that may be unfounded. We must also consider that behaviour is a dynamic process, yet the roles of time, repeated exposure, and learning are all but ignored. By the time most true behavioural problems are recognized by the clients the behaviours and the social relationships between the participants have changed. We can further change these relationships – and in the wrong direction – if we continue to operate within the flawed context that results from the adherence to inapplicable and wrong terminologies.

There are two broad contexts in which the term ‘dominance’ is used with respect to dogs: when describing interactions between dogs and when describing the role the client is recommended to take in interactions with the dog. Neither of these approaches is valid.

Asking clients and practitioners to identify and then exhibit behaviours that encourage or discourage the ‘dominant’ dog can cause morbidity and mortality for dogs and humans. For example, in a review of dozens of cases involving inter-dog aggression between household dogs,<sup>5</sup> most clients had been advised to support or reinforce the ‘dominant’ dog and that when they did so, the aggression worsened. One could accordingly argue that the clients are not correctly identifying the ‘dominant’ dog, but if a label is causing such difficulties, it may be time to stop using it. The issues of ‘dominance’ and social rank in group interactions comprise one of

the oldest, most confusing, and hotly debated areas in the behavioural literature. It’s important that we understand why this concept has caused problems in the practice of veterinary behavioural medicine.

Cause and effect are confounded. The existence of a hierarchy has been postulated to be a stress-reducing device (Collias, 1953); however, situations where hierarchies are most rigidly maintained are also ones where measures of stress are high (Rowell, 1966). In the traditional scheme the dog who ‘submits’ (generally undefined) or gives way to another as a result of prior interactions is considered the ‘subordinate’ while the individual inducing such behaviour is usually considered the ‘dominant’ animal in the pairing. ‘Dominance’ has been traditionally defined as individual’s ability, generally under controlled situations, to maintain or regulate access to some resource (Landau, 1951; Rowell, 1974). Given that the definition of ‘dominance’ can be further refined as a description of winning or losing staged contests over resources (Archer, 1988; Rooney and Bradshaw, 2004), and that a winning outcome need not confer priority of access to those resources, we must accept that variable distributions of resources (e.g., access to attention, beds, resting sites, toys, food dishes, etc.) will lead to variable hierarchical classifications.

Two concerns deserve redress: (1) the extent to which the labelling of an event, interaction, or pattern of interactions may interfere with our ability to truly understand behaviours and signals, in the relevant context, and (2) the extent to which, if we subscribe to a hierarchical system, we are then tempted or constrained to force all interpretations of behaviours into that system. Such practices have encouraged humans to treat dogs inhumanely under the guise of being ‘dominant’ to them, and have likely resulted in the injury or death of many dogs. In the case of inter-dog aggression such logic leads to ‘reinforcing’ a truly pathological animal as ‘dominant’. These concerns are not new: the potential to mislead was Rowell’s primary concern when she published her ground-breaking study on the intricacies of baboon social interactions (Rowell, 1967). In fact, when free-ranging baboon interactions were classified by behavioural types (e.g., friendly, approach-retreat), and then analyzed according to specific behaviours of the participants, no ‘dominance’ system was noted. In fact, a much more complex, elegant system of interactions that reflected relatedness, age, sex, social history, etc. became apparent.

Most social behaviours, when fully examined, are not characterized by agonistic encounters, but by fluid, context-specific, deferential behaviours (Overall, 1997a). Deference is not analogous to submission or subordination. Deference is about relative status that is freely given, not imposed, and it may vary with context. The animal to which most others defer is the animal that behaves most appropriately given the context, not the

<sup>5</sup> Overall and Dunham, unpublished data, 2004.



animal which must always be at the door first, or must eat first. In fact, a need to control regardless of context is neither adaptive, nor normal. The central and organizing role for deferential behaviours is supported by authors who have looked extensively at social interactions (e.g., Crowell-Davis et al., 2004) when they discuss the variability in the behaviour of high ranking animals. These findings are supported by others that emphasize the importance of understanding when the behaviours are about normal, learning about relative and fluid roles in changing social environments, and when they are about truly pathological behaviour. Because learning works by altering neurochemistry (see discussion in Overall, 2001), we need to understand that both early intervention designed to avert anxiety associated with underlying aggression and pharmacological intervention can help, but neither approach will be used appropriately until the clients can understand the signaling and interactions from the dogs' viewpoints (Rooney et al., 2001). In this world view, diagnosis and treatment are about both understanding the neurochemical changes that occur with learning and repeated exposure, and about becoming humane. This requires that we understand normal ethology and behavioural ontogeny of that species (see, for example, Schoening, 2001; Wirant and McGuire, 2004; Yin and McCowan, 2004).

One key factor that we often neglect is the role of ontogeny and learning in any behavioural problem. The blurring of the lines between normal (the perceived aggressor is truly at risk and aggressive behaviour is adaptive) and abnormal aggression (there is no risk to the aggressor) are real: they are a function of our lack of knowledge about how behavioural conditions develop. In fact, the extent to which an animal deviates from 'normal' in aggression or any other suite of behaviours may depend on ontogeny, multiple gene effects, and pleiotropic environmental effects (Nijhout, 2003) (see Fig. 3). If anxiety-based aggression has a causal pattern similar to other anxiety based conditions like obsessive-compulsive disorder, both a familial or genetic 'predisposition' and a social stressor play roles in the development of the aggression (Overall and Dunham, 2002).

An example of the utility of this approach is found in the evolving story of impulse control aggression. Aggression is best defined as an appropriate or inappropriate; inter- or intra-specific challenge, threat, or contest resulting in deference or in combat and resolution (Overall, 1997a). Most abnormal aggressions are the result of underlying anxiety (Overall, 1997a, 2000). Canine and feline anxieties, particularly those involving more extreme responses, appear to have a genetic component (Overall and Dunham, 2002; Reisner et al., 1994). Breeds are the result of artificial selection for specific types of work. The first dogs identified with extreme freezing and social withdrawal, for example, were from

a familial line selected for exquisitely developed pointing behaviours (Murphree, 1973; Murphree et al., 1977). One would expect that the extent to which anxiety was present and deleterious could depend on breed and the task, and that more 'reactive breeds' which are also expected to engage in more complex behaviours (e.g., explosives detection and patrol/human detainment) would be more at risk than are dogs selected for low reactivity, or those trained for and used in more singular tasks like explosives detection.

Dogs share both foraging mode and a social system with humans, and have co-evolved for co-operative work with humans for approximately 135,000 years, with intense selection for specific suites of behavioural traits (e.g., the development of breeds) occurring in the last 12,000–15,000 years (Leonard et al., 2002; Vilà et al., 1997, 1999). Dogs mirror humans in hallmarks of social development (Overall, 1997a). Recent data indicate that dogs are significantly more comparable to humans than are chimpanzees and wolves with regard to the complex social cognition involved in understanding long-distance signals that indicate where food is hidden (Hare and Tomasello, 1999; Hare et al., 1998, 2002; Miklosi et al., 2004; Pongráz et al., 2003; Topál et al., 1997). Dogs are further able to communicate this information to other dogs. Also, like humans, dogs suffer from what we recognize as maladaptive anxiety – that which interferes with normal functioning – which was selected against during the co-evolution of dogs and humans.

There is a growing body of evidence that anxiety can affect the rate at which learning progresses, and various performance capabilities. Additionally, there is evidence that treatment with monoamine re-uptake inhibitors speeds learning of specific tasks in dogs (Mills and Ledger, 2001).

The mechanisms postulated for failed performance associated with anxiety and aggression involve the finding that chronic glucocorticoid excess interferes with long-term potentiation (LTP) and other putative processes associated with learning (Diamond et al., 1992). Chronic exposure has also been proposed to affect hippocampal neuronal structure (Sapolsky, 1996). Viewed in this light chronic cortisol elevation may act as a translational gene regulator in regions of the hippocampus. In the large, but overwhelmingly non-experimental literature on working dogs, the single best predictor of failure in any working dog is fear, and the factor that prohibits most dogs from completing training programmes is their aggressive/fearful/anxious/uncertain response to novel or complex environments (King et al., 2003; Slabbert and Odendaal, 1999; Weiss and Greenberg, 1997). Any tests that can help identify early aspects of fear and anxiety and their effects on aggression will lead to future research on effects of intervention on learning.

One controversial diagnosis, impulse control aggression (formerly called ‘dominance aggression’ in dogs), is about control or access to control in direct social situations involving humans. This is a discrete definition of impulse control aggression and has the advantage of not coupling the challenge to food (food related aggression), toys (possessive aggression), or space (territorial aggression). These aggressions can all be correlates of impulse control aggression and when associated with it may be indicative of a more severe situation. This diagnosis cannot be made on the basis of a one-time event. This definition is radically different from the common descriptions of this aggression that specify that the dog will often react to being pushed on, to being corrected with a leash, or to being pushed from a sofa or a person (Podberscek and Serpell, 1996, 1997; Reisner et al., 1994). This is the primary category of canine aggression in which no warning prior to biting is given (Borchelt, 1983). The classically afflicted dog growls, lunges, snaps or bites if they are stared at, physically manipulated – often when reaching over their head to put on a leash, physically disrupted or moved from a resting site – no matter how gently this is done, and when they are physically or verbally ‘corrected’.

As for most other behavioural conditions, this aggression commonly develops during social maturity when neurochemistry undergoes changes; however dogs exhibiting this behavioural abnormality at social maturity tend to be male, whereas when females are affected they exhibit the behavioural pathology in puppyhood (Overall, 1995; Overall and Beebe, 1997). The average age of onset for affected males is ~12 months, but is ~8 months for females, a statistically significant difference.<sup>6</sup> The range of ages of onset also varies significantly for the sexes. Intact females less than one year of age are over-represented when compared with other age and sex groups for which this diagnosis pertains. This divergent phenotypic pattern of the problem behaviours suggests mechanisms involving multiple causality.

Little work has been done either post-mortem on neuroanatomy or cytoarchitectural facets of these conditions, or ante-mortem using imaging studies of impulse control aggression or impulsivity per se. Limbic system responses have been related to impulsive risk-taking, behavioural timing, and time judgements (Nedergaard et al., 2002; Nelson and Chiavegatto, 2001). The serotonin system has been implicated in both canine impulse control aggression and in human impulsivity. Affected dogs in one study (Reisner et al., 1996), but not in a better controlled, replicated study (Mertens, 2000), had lower CSF levels of 5-hydroxyindol acetic acid (5-HIAA) and homovanillic acid (HVA), metabolites of serotonin and dopamine, respectively, post-mortem

than do control dogs. Although there is evidence that CSF HVA level may be a function of breed, CSF 5-HIAA levels appear to be decreased irrespective of breed. Afflicted dogs differ from all other aggressive dogs based on data from urinary metabolic screens: these dogs consistently manifest excretion of glutamine, the metabolite of the excitatory amino acid glutamate, in their urine (Overall, 1997c). Given that the cytotoxic effects of glutamate are now well established, this pattern warrants further study (Faden, 1989; Trotti et al., 1998). Finally, these dogs respond to treatment with TCAs and SSRIs when combined with behaviour modification (Dodman et al., 1996; Overall, 1995).

In the early stages of the condition, the dogs improve quickly and dramatically if they are given a kind, reliable rule structure for interaction (e.g., they must sit and be calm before they get any kind of attention). This is a clue that the provocative behaviour exhibited by the dog may be more about soliciting information from and about the social environment than it is about pushy, manipulative behaviour. In fact, within the population of dogs developing the behaviour at social maturity, at least two phenotypic groups have been identified: (1) those dogs that are not able to use the social cues in the human environment to modulate their reactivity and who become explosive when they reach their stimulus threshold, and (2) those dogs that are uncertain of the human social environment and provoke it to gain information about what expected social responses and consequences could be (Fatjo et al., 2005; Overall, 1997a). Both of these pathological representations are forms of rule structures that have gone wrong. Keys to treatment include replacement with rule structures with clear and humane expectations.

Affected dogs come from family lines were, on average, one half the dogs in each litter show signs of the condition by social maturity. (18–24 months). Once identified within a breed or familial line, the condition appears each generation. Breeds that have been commonly represented in specific populations include English springer spaniel (Reisner et al., 1994), American Cocker spaniels, Dalmatian, golden retriever, German shepherd, Labrador retriever, and Rottweiler (Overall, 1997a) in the USA, English Cocker spaniels in the UK (Podberscek and Serpell, 1996, 1997), and golden retrievers in Europe. A collaborative effort is currently underway to use genome scans and mapping for this condition and others involving aggression and anxiety.<sup>7</sup>

Breeds, by definition, are the result of canalised genetic variation, and when a trait appears in a breed line it is likely that there is accompanying line breeding

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<sup>6</sup> Overall KL, Dunham AE, Frank D, 2004, ms.

which can be identified by multi-generational pedigrees. It is not unusual to have three or four generations of dogs available for examination within any affected pedigree. Use of multi-generational dog families also allows us to examine individual differences instead of averaging across groups. Furthermore, families and breeds of dogs are also ideal for haplotype analysis in a way that is almost impossible to accomplish in humans. Such analyses may better link structure and function than do analyses of groups (Plomin and Kisslyn, 2001).

Finally, although much of our information about canine aggression comes from specific breeds of dogs, none of this information implies that the entire membership of any breed is aggressive, nor does it support legislation that bans breeds. A review of the data on dog bites indicates that popular and numerically dominant breeds are most frequently involved in bites, bites to children are relatively common, most bites to children are to male children, and most fatal dog bites involve male children. To outlaw some breeds, especially in the absence of the needed demographic and risk data, would not make us safer, and the illusion that it would is a dangerous one (Kahn et al., 2003, 2004; Overall and Love, 2001; Schoening, 2001; De Keuster et al., 2005).

The link between separation anxiety and noise and thunderstorm phobia provides another excellent example of how a modern synthesis can provide a useful approach for understanding anxiety. Anxiety disorders are among the most common health concerns in human medicine (Narrow et al., 2002), as they are for pet dogs. Like humans, dogs with one anxiety-related diagnosis frequently have other anxiety-related diagnoses (Overall et al., 2001; Overall and Dunham, 2002), suggesting the existence of some putative genetic or neurochemical liability (Gractacos et al., 2001; Smoller and Tsuang, 1998; Scherrer et al., 2000).

Although there are few quantitative clinical studies on anxious dogs, those focusing on separation anxiety (Overall et al., 2001) and obsessive-compulsive disorder (Overall and Dunham, 2002) have shown that a high percentage of affected patients experience other, co-morbid anxiety disorders (~90% and 75%, respectively). In the case of separation anxiety, the co-morbid diagnosis is usually noise or thunderstorm phobia. While the data are few owing to the nature of retrospective studies, heightened noise reactivity or fear as a young dog may predispose the individual to the later development of separation anxiety. If so, this strongly suggests that associations between various anxiety and 'mood' conditions (e.g., depression and anxiety; panic and social phobias, etc.) may be the result of increased risk that is either the direct result of a shared underlying cause of the initial disorder, or the indirect result of neurochemical and/or molecular changes that occur because of the initial disorder.

One data set (Overall et al., 2001) showed that the conditional probability that a canine patient has separation anxiety, given that they have noise phobia is high (88%) and approximately the same as if they have thunderstorm phobia (87%). However, the probability that a patient has noise phobia is higher (74%) than the probability that they have thunderstorm phobia (61%), given that either have separation anxiety. The finding that the probability of having a noise phobia, given the presence of a thunderstorm phobia, is not equivalent to the converse (90% versus 76%), supports the hypothesis that neurochemical responses to noise are different from those to thunderstorms. This conclusion, when combined with the data on the relationships between noise and separation anxiety, suggest that, the behavioural phenotypes and endophenotypes are manifestations of repeated exposure and LTP. In other words, phenotypes change with time and learning and these behavioural changes are reflections of the underlying neurochemical pathology.

The property of unpredictability/uncertainty associated with thunderstorms may have a role in shaping the neurochemical and behavioural responses to anxiety provoking situations, suggesting future areas of exploration for understanding anxiety-related responses in dogs. Interestingly, although noise and thunderstorm phobic patients and those with separation anxiety share non-specific signs, members of each diagnostic group experience distinct physiological responses to the provocative intravenous lactate test and non-overlapping behavioural responses (Overall and Dunham, 2003; Overall et al., 2002), suggesting that finding biomarkers for anxiety may be a realistic expectation.

Another region in which biomarkers may be important is in assessment of how well animals, within a diagnostic category, respond to medications. Understanding the genetic polymorphisms that render humans rapid, slow, or moderate metabolisers of psychotropic drugs has improved the prognosis for 'non-responders' (Tribut et al., 2002). Additionally, these polymorphic groups may suggest variation in mechanism of the underlying problem, and make mapping some of these conditions a more efficient endeavour.

### *2.1. Final thoughts – a changing paradigm and new synthesis*

When done correctly, science provides us with paradigms by which we can learn whether something is true. Veterinary behavioural medicine is fundamentally about two things: (1) pushing the frontiers of neurobehavioural genetics and modern ethology to learn about variability in behaviours of companion animals and how to best address that variability in a world that is becoming increasingly complex for pets, and (2) using this new knowledge to become more humane. The results of

anticipated behavioural studies should help re-homing centers in their mission. The time has come to encourage a new synthesis and paradigm shift in the field of veterinary behavioural medicine. Science must proceed divorced from human judgements, jealousies, misperception, and an unfettered reliance on people's opinions (*sensu Hsu and Serpell, 2003*). We must commit ourselves to collaborative, publishable endeavours rooted in hypothesis testing and data collection. It is hoped that this symposium represents a significant step in that direction.

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