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Why are assumptions passed off as established knowledge?

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ABSTRACT

In this paper we attempt to explain the problems that can arise when assumptions made by experts in their respective fields of Medicine become widely accepted as established knowledge. Our hypothesis is that these problems are in large part attributable to a failure of the experts to follow the principles of logical argument.

Empirical data to evaluate our hypothesis derives from an analysis of the reasoning processes employed in the generation of three syndromes drawn from the clinical discipline of Pain Medicine: myofascial pain, shoulder impingement and central sensitisation.

We demonstrate a failure by the proponents of these syndromes to structure their scientific arguments in a logically valid fashion, which lead them to promote assumptions to the status of facts. In each instance those in relevant scientific journals responsible for content review accepted – and thereby promulgated - this fundamental error in reasoning. The wide acceptance of each of these assumptions as established knowledge affirms our hypothesis. Furthermore, we show that such uncritical acceptance has had significant consequences for many patients.

> "What can be asserted without evidence can also be dismissed without evidence." [Christopher Hitchens, 2007].

Background

According to the World Health Organisation [1], in more than half of cases in which diagnostic errors are made, an error of reasoning can be identified. Such errors can result in failure to provide an accurate and timely explanation of the patient's health problems or to communicate that explanation to the patient. In the practice of Medicine these issues loom large, and their consequences can be serious. How do these errors come about?

Hypothesis

We advance the hypothesis that the source of error lies in the application of fallacious reasoning, in large part due to innate and acquired biases of authors of scientific articles [2,3]. Because scientific journals are the main vehicles for advancing knowledge, editors of these journals appear to have relaxed their pivotal responsibility in detecting errors in reasoning made by eminent persons who submit their work for publication. As a result, the readership will have difficulty in deciding whether the opinions expressed are established scientific facts or whether they are based only on assumptions.

We will evaluate our hypothesis by focusing attention on three clinical examples chosen from the discipline of Pain Medicine, in each of which we identify assumptions that became widely accepted as established fact.

Defining the terminologies

To set the scene, we first explore the terminologies used in the various reasoning processes to be mentioned below. They are derived from the discipline of philosophy, which as well as containing an enormous repository of knowledge provides us with a diverse set of conceptual instruments or tools "to analyse, manipulate and evaluate philosophical concepts, arguments and theories" [4]. The main reasoning tools employed in scientific research are deduction, induction and abduction.

Argument

In philosophy, an argument is the most basic unit of reasoning,

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where an inference from one or more starting points (called a "premise" or "premises") proceeds to an end-point (called a "conclusion") [4]. A premise makes a claim that is either true or false. This raises two questions about the validity of an argument: (i) whether or not the conclusion follows from the premises; (ii) what justifies the use of the premise(s) in the first place. In some instances the truth of a premise has been demonstrated elsewhere, while on other occasions it may be true by definition.

Validity

When an argument has a sound deductive structure it is said to have validity. Validity is guaranteed irrespective of whether the premise and conclusion of an argument are true, however ridiculous they may be. An argument is said to be sound when it is both valid *and* the premises are true. However, "begging the question" is the term used when the premises of an argument assume the truth of its conclusion. It can occur when a particular position springs from wishful thinking or is grounded in ignorance [5].

Explanation

Whereas arguments attempt to demonstrate that something *is* true, explanations attempt to show *how* something is true. According to Lipton [6], to make a scientific claim is to assert that the claim is true. As he puts it, " … scientific inferences are often driven by asking how good an explanation a given hypothesis would provide of the phenomena if it *were* true, and then, if it would be good enough, inferring that it *is* true" (emphasis in the original). An explanation is judged to be more compelling than its rivals when it is supported by established theory [7].

Reasoning by the hypothetico-deductive method

As mentioned above, for a philosophically deductive argument to be sound, the move from premise to conclusion is such that if the premise is true, then the conclusion must also be true. Argumentation allows the formulation of explanatory hypotheses, which are proposed explanations for a phenomenon, but with the important proviso that they have the potential to be refuted [8].

The well-known scientific method is based upon a number of observations that lead to an explanatory theory, to the formulation of hypotheses derived from the theory, and to their testing.

The basic principle of the hypothetico-deductive method as enunciated by Karl Popper can be summarised as follows [8]:

> "Start with a hypothesis and a set of given conditions[1], deduce what facts follow from them and then conduct experiments to see if those facts hold and hence whether the hypothesis is false".

Popper argued that only attempts at refutation can lead to advances in knowledge [8]:

> "If observation shows that the predicted effect is definitely absent, then the theory is simply refuted." (p.36)

Reasoning by induction

When an unfamiliar phenomenon is encountered, a clinician might attempt to explain it by first appealing to a "general law" of different categories based upon past experience, and then by arguing that the particular phenomenon is analogous to those encountered and successfully managed in the past [4]. This process is called inductive reasoning (or generalisation), which has also been described as the "logic of experience" [9].

In contrast to hypothetico-deductive reasoning, which delivers a conclusion with a degree of confidence, induction can only arrive at conclusions with a degree of "probability". As most bodily processes (i.e. data sets) are hidden from the observer's view and are not necessarily uniform or predictable, reasoning by induction can be problematic in the context of medical decision-making and practice [10]. Thus what was observed to have happened in the past cannot be used as a guide to what has yet to occur.

Reasoning by abduction

In view of the limitations of the processes of deduction and induction when used in the clinical situation, other forms of reasoning are necessary. Abduction utilises previous instances of hypothesis formation to generate similar hypotheses. Collections of facts or observations (i.e. premises) are studied and a set of possible hypotheses is devised in an attempt to explain them [7]. Only those hypotheses that have biological plausibility are worthy of development, necessitating further evaluative work [7].

The role of assumptions

An assumption is anything said or believed that is accepted without proof as true or is certain to happen. In the theory of formal logic, assumptions are premises [11]. But because the burden of proof has to stop somewhere, assumptions fulfil an important role in day-to-day life by allowing us to fill in large gaps in our knowledge.

Some 1600 years ago, Augustine of Hippo [354–430 CE] made the same point in his Confessions [12]:

> "I began to realise that I believed countless things which I had never seen or which had taken place when I was not there to see – so many events in the history of the world, so many facts about places and towns which I had never seen, and so much that I believed on the word of friends or doctors or various other people. Unless we took these things on trust, we should accomplish absolutely nothing in this life". (Book 6, p.5)

Wittgenstein [13] was also of this opinion, "we just can't investigate everything, and for that reason we are forced to rest content with assumption." (§343)

Yet, as we will demonstrate, assumptions should lead to testable hypotheses. A challenging situation arises in Medicine when those who make assumptions neglect to do this. They come to believe they are statements of truth and pass them off to others as established knowledge [14].

Evaluation of the hypothesis

Our hypothesis will be evaluated using empirical data obtained from a close analysis of the three examples we have chosen.

The rise and fall of myofascial pain syndrome

In 1952 Travell and Rinzler [15] proposed that pain felt in voluntary muscles could be "*myofascial*" in origin. They postulated a feedback cycle whereby "trigger areas in myofascial structures can maintain pain cycles indefinitely". Maintenance of the cycle was attributed to bombardment of the central nervous system by impulses from the trigger areas themselves. That is, their "pain cycle" is self-perpetuating [16].

Travell and Rinzler [15] reported finding patterns of referred pain for 32 voluntary muscles and, based on their collective observations, compiled anatomical charts of these areas, which varied in location from person to person.

Travell, who later became the personal physician to the late

[1] The term "given conditions" refers to those *a priori* theories provisionally accepted as being true.

president John F. Kennedy [17], presented her work at the first World Congress on Pain held in Florence in 1976, when the trigger areas became known as "*trigger points*" [18]. She then joined with Simons, already an American national hero as the first astronaut to ascend in a balloon to the height of 19 miles [19], to publish what became a highly influential book that formalised the construct of myofascial pain arising from trigger points [20]. Their prestigious societal status facilitated the uncritical acceptance of their premise, which resulted in a consensus as to both the existence and nature of myofascial trigger points.

Refutation of the myofascial pain syndrome construct

Even at the time of proposition of Myofascial Pain Syndrome (MPS), the then famous neurologist Karel Lewit revealed that the diagrams of referred "myofascial" pain from "trigger points" had "sometimes been chosen arbitrarily, there being no accepted standard" [21].

A thorough investigation found the original argument presented for the existence of trigger points and their responsibility for MPS to be invalid because the premises are not true and the theory is not testable [22,23]. Despite extensive research in experimental animals and humans conducted over three decades, no agreement has been reached on the definition of a "trigger point" and its true nature remains obscure [24].

The construct of MPS therefore collapses as do any modalities of treatment that rely upon the reality of "trigger points" as concrete entities [25]. Furthermore, the concept of a self-sustaining "pain cycle" rests upon the flawed argument that an experience (i.e. pain), can also be conceptualised as a concrete entity responsible for neurophysiological events. This is the fallacy of reification [26]. The epistemic failure was to assume the veracity of the untested proposition of "trigger points" and to reject any evidence that might contradict that assumption [16].

How "shoulder impingement syndrome" became a popular orthopaedic condition

In 1972, orthopaedic surgeon Charles Neer [27,28] proposed the concept of shoulder *sub-acromial impingement syndrome*. His anecdotal surgical experience led him to believe that 95% of shoulder pain is due to impingement of the supraspinatus tendon beneath the acromion process. Neer was a world famous and respected surgeon, and there can be no doubt that his reputation contributed to the acceptance of his article [29]. Medical professionals and the public alike came to accept "impingement syndrome" as a genuine clinical entity.

Refutation of the construct

Neer's reasoning process was that of abduction, whereby his many repeated observations led him to adopt what he believed was the most likely hypothesis to explain his successes. He believed they were entirely attributable to the particular operative procedure he had performed. This logical fallacy is known as *post hoc ergo propter hoc*, where the effect is attributed to the preceding event.

Although he meticulously recorded his procedures and findings, Neer did not follow the exactitude that characterises scientific studies (i.e. controlling for variables and various confounders, including the placebo effect that is now known to play a significant role in determining the outcomes of many surgical procedures) [30]. Furthermore, he used the favourable outcomes as validation for his argument but neglected to consider alternative explanations.

How a postulated mechanism became a disease

The North American rheumatologist Yunus [31–33] argued that fibromyalgia syndrome and other similarly vague conditions, e.g., myofascial pain syndrome, irritable bowel syndrome, chronic fatigue syndrome, headache, and restless legs syndrome share several clinical characteristics, including pain, disturbed sleep, fatigue, hyperalgesia, and the absence of identifiable underlying tissue pathology.

Yunus proposed the concept of "*central sensitivity syndromes*" (CSS) as his favoured unifying explanation for these observed clinical phenomena. His explanation drew upon the neurophysiological concept of central sensitisation [34] characterised by "*hyperexcitement of the central neurons*" arising from neurohormonal dysfunctions, which resulted in amplified, widespread and persistent pain.

As Yunus [33] asserted:

> "Central sensitisation (CS) is clinically and physiologically characterised by hyperalgesia (excessive sensitivity to a normally painful (sic) stimulus), allodynia, expansion of receptive field (that is likely to explain widespread pain), a prolonged electrophysiological discharge (that may explain the chronic nature of pain), and an after-stimulus unpleasant pain (e.g., burning, throbbing, and paresthesia) that lasts longer than that observed in normal controls following a noxious stimulus." (p.341)

By way of extrapolation, he argued that the "remarkable overall hyperexcitement of the central neurons" might explain the hypersensitivity to many environmental stimuli (e.g., noise, weather, stress). Yunus then suggested, "CS becomes self-sustained without further stimuli, even minor, because of long-term central nervous system (CNS) neuroplasticity, and is probably accentuated with chronicity in human diseases." (p.342)

This proposition contained three scientifically tenuous themes:

- Firstly, central sensitisation *of nociception* was adopted as the model from which an unjustified generalisation to "central sensitisation" was made.
- Secondly it was stated that the biology of CSS is "based on neuroendocrine aberrations (including CS) that interact with psychosocial factors to cause a number of symptoms", without evidence being offered.
- Thirdly, "long-term CNS plasticity" was invoked for the ability of CS to sustain itself, which is a circular argument.

Although Yunus conceded: "central sensitisation may not be the only pathophysiological mechanism for CSS *disease* (emphasis added)", he believed his argument was plausible and therefore viable [33]. Was his argument sound? Did his conclusion follow from his premises?

Refutation

Yunus [33] noted the definition of the word "pathology" went beyond "structural pathology" to include "functional changes that result from disease process." In relation to CSS he argued: "the definition of pathology should include both structural and neuroendocrine-immune (NEI) changes". In so doing, he failed to identify the tautology inherent in a proposition that appears to be true (i.e. his definition of pathology) because it includes all possibilities. Moreover, Yunus begged the question by elevating CS, a postulated mechanism, to become a "disease" of the same name. These failures led him to formulate a hopelessly circular argument: "multiple symptoms in a CSS condition are due to its association with multiple other CSS conditions affecting different systems" [33]. However, a comprehensive review of the scientific evidence from the field of neuroimaging did not support the concept of CSS [35].

The consequences arising from each assumption

Each of these three examples is characterised by an untested but plausible assumption being accepted as truth, without the implications of each theory being rigorously tested in a hypothetico-deductive paradigm that actively seeks refutation. How was this allowed to happen?

Myofascial pain syndrome

The uncritical acceptance in the peer-reviewed musculoskeletal (MSK) literature of the ideas advanced by Travell and Simons, saw MPS as a common pain condition [36] with a burgeoning scientific literature [37] and the establishment of a prolific international industry offering training courses and certifications to accredit those who wished to treat supposed myofascial trigger points. Various health care providers, including physicians, physiotherapists, osteopaths and chiropractors, were taught how to diagnose and treat patients with a condition known as MPS. The fundamental premise of MPS has been shown to be false, a myth, a mirage. Yet an inestimably large number of patients around the world have undergone treatment that was irrational and no more effective than placebo. Regardless of criticisms of their logic, proponents of MPS continue to use favorable outcomes from clinical trials as validation of their construct and justification for their use of various therapeutic modalities (e.g. dry needling and trigger point massage etc.). Their reasoning exemplifies the logical fallacy known as *post hoc ergo propter hoc*. We suggest that eliminating the term MPS from the current medical taxonomy would advance the study of tender but ostensibly normal tissues and allow better and scientifically plausible explanations, such as neuroinflammation [16,38–41], for the clinical phenomena, and discourage the use of forms of treatment that have no scientific foundation.

Shoulder impingement

When assessed by today's scientific standards, Neer's original article, which has been cited over 3000 times, can be considered only as an opinion piece or a conjecture [42]. No number of citations of the same phenomenon (apparent confirmation) confirms the validity of the observation [43]. To believe otherwise is known as the "illusory truth effect" [44].

Today, some four decades after the publication of Neer's proposition and despite its comprehensive refutation, belief in "impingement syndrome" as a real clinical entity remains intact [42,45,46]. The acceptance of Neer's assumptions by orthopedic surgeons as established knowledge has led to "sub-acromial decompression, which became one of the most commonly performed shoulder operations globally (approximately 21,000 decompression procedures per year in the United Kingdom and many times more in the United States), although it has been shown to be no more beneficial than placebo [47].

The postulated mechanism becomes a brain disorder

The CSS concept was not only championed in the MSK peer reviewed literature, but also taken further by some rheumatologists who introduced the concept of "*centralised*" pain [33,48–51].

Arnold et al. [52] asserted that fibromyalgia exemplifies "centralised" pain and that it results from "persistent neuronal dysfunction". They argued: "Aberrant neurochemical processing of sensory signals in the central nervous system (CNS) may lower the threshold of pain, amplify normal sensory signals, and alter gene expression, thereby leading to hypersensitivity and central sensitisation that result in chronic pain".

Two questions arise from this assertion: how might "alter(ed) gene expression" lead to "*hypersensitivity*", and what do "hypersensitivity" and "central sensitisation" refer to? According to Clauw [51], "the pathophysiological hallmark is a sensitised or hyperactive central nervous system that leads to an increased volume control or gain on pain and sensory processing [51]." This statement can be rewritten as follows: "Increased CNS activity leads to increased pain and sensory processing", which is a circular argument.

Sluka and Clauw [53] explained the overlapping conditions comprising CSS as manifestations of an underlying brain disorder with "-similar underlying pathology with alterations in central nervous system function leading to augmented nociceptive processing and the development of CNS-mediated somatic symptoms of fatigue, sleep, memory and mood difficulties".

In summary, central sensitisation syndromes are characterized by the development of CNS-mediated symptoms attributable to altered brain function. Or, to put it another way, a cluster of "CNS-mediated symptoms" allows the inference of a brain disorder called "central sensitisation". This is a circular argument, which resulted in a false consensus whereby clinicians and patients were led to view fibromyalgia as a disease of the brain. The false consensus unnecessarily exposed patients so labelled to the declaration that chronic pain is a disease in its own right, which was then, and still is, a controversial proposition [54].

Refuting the hypothesis

Diagnostic errors in medicine are typically multifocal in origin and faulty clinical reasoning is only one recognised factor [55]. But as Graber et al. [55] acknowledge, examining someone's clinical reasoning can be difficult. Should the clinical reasoning process used by the proponents of each of the conditions be soundly based, our hypothesis is refuted.

Although we have argued that the assumptions made by the initial proponents of each of these examples turned out to be wrong, we have not explained their widespread perpetuation. We suspect that the "snowball effect" to citations was in large part responsible [43,56]. This effect is seen when other authors are more likely to cite a published paper because of its previous citations, rather than for its particular content or for the quality of its reasoning.

Affirming the hypothesis

The problems that can arise from failing to recognise assumptions as such become obvious when people other than the proponent believe that they are true and therefore reliable facts. The three examples drawn from the field of Pain Medicine show the harm that can ensue when assumptions made with the persuasive power of authority turn out to be wrong. As we found in each example, important issues of medical diagnosis and management were dependent upon the veracity of the respective assumptions, each of which turned out to be wrong. This is why we are also ethically obliged to acknowledge the possibility that even our deeply held beliefs may be wrong.

As we have shown from these examples, discerning the difference between valid and invalid arguments requires all clinicians, researchers and journal editors to carefully scrutinise both their content and the reasoning processes used to arrive at their conclusions. A failure to do so will continue to produce avoidable errors in medical diagnosis and management and call into question the credibility of the relevant scientific literature. No matter how good our observations, faulty reasoning will invariably lead us from the truth, with adverse consequences for patients, the profession, and our society [5].

In the spirit of preventing further instances of this phenomenon, we encourage the adoption of three logical principles that ought to be invoked in the processes of review: (i) Assumptions should lead to testable hypotheses; (ii) Repeated observations of the same phenomenon do not add to the validity of the initial hypothesis; (iii) Assumptions may become facts only after the hypotheses that they generate have withstood rigorous testing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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