

The Need for Complex Ideas in Anorexia Nervosa: Why Biology, Environment, and Psyche All Matter, Why Therapists Make Mistakes, and Why Clinical Benchmarks Are Needed for Managing Weight Correction

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ABSTRACT

Anorexia nervosa remains an enigma and its clinical challenge is intimidating. But the potential for new insights has been advancing, largely as a result of elegant research in the neurosciences that has modeled behavioral processes resembling key features of the illness. Unfortunately, many in the eating disorder field seem to know little of this work or the implication it holds for treatment philosophy. Instead, the knowledge void has been taken up recently by a

host of misguided notions about etiology, blatantly dismissive attitudes toward psychological concepts, and ill-conceived beliefs about therapy priorities. This article is a clinical perspective on these issues. © 2012 by Wiley Periodicals, Inc.

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Introductory Comments

This article has a long history. It dates back to a July morning in 1974, when the lead author walked through the doors of the UCLA Neuropsychiatric Hospital and was welcomed almost immediately by a 13-year old patient whose ghoulish appearance was so horrifying I could do little more than stare incredulously. My greeter was unfazed by the reaction—as though she had seen it so many times it no longer touched her own sensibilities; if it ever did. After the introductions, she said—a bit over-proudly I thought—that she had anorexia nervosa (AN) and weighed 52 pounds. I distinctly remember thinking I had seen something like this before; then I remem-

bered. It was 1960. I was 11 years old and had come to my grandmother's hospital bed to say goodbye for the very last time; she died the following day from colon cancer. When I asked my mother what made her so thin, she said it was the cancer; on the day my grandmother died she weighed only 69 pounds. I remember thinking this had to be a mistake—a grown person couldn't be that thin.

As for my new acquaintance, she was only a fraction as old as my grandmother, but “thin” didn't even come close to describing the skeleton that stood before me. Her skin had all but melted into the bones beneath and her head—it appeared so shrunken that her eyes dangled perilously from their orbits, seemingly held in place only by a single remaining sliver of muscle; I was certain they were going to drop at any moment. Like the memory of my grandmother's appearance on her final day, my first encounter with AN remains equally well preserved; you never forget images like these. I also recall the brief exchange we had. She asked if I knew what AN was. I said I didn't, so she volunteered to teach me. She described the illness as a way of controlling things. When I asked how, she said, “by losing weight,” as if the answer was obvious. But when I asked, “What exactly is it you're trying to control and why did you lose so much weight?” she just stared blankly. After a brief pause she went on to say that everyone—hospital staff, friends, teachers, and family—is worrying about her—her father especially (who I soon learned suffered from manic depression)—but they're exaggerating the danger, she added; then she abruptly walked away.

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This, I thought, was extraordinary—and intimidating. I had no idea what to make of it, or how to go about helping someone like this.

I first became interested in the nature and causes of mental illness in high school. Now, standing right in front of me was something tangible; tangible, but too complex for an inexperienced clinician to comprehend. Ironically, 6 months before setting out to write this article I received an email from my greeter; I had neither seen nor heard from her since she discharged from UCLA more than 36 years ago. She said she didn't know why, but that she wanted everyone to know (several members of the original treatment team remain) that at 49 years of age she was long recovered, had two healthy children, and enjoyed working as a nurse in a community hospital near her home; she included a picture. Then shortly thereafter, as if I needed a reminder about AN's less merciful side, another message came; this one from the husband of kindly 49-year-old patient whose recent treatment at UCLA was her first. Unfortunately, traveling to California seeking treatment 34 years after her illness began, it was too late. He thanked us for trying, but said his wife recently told him and their two sons that she had grown weary from the struggle; a week ago she went to sleep for the last time, the three of them by her side.

The purpose of science is to advance knowledge about phenomena not well understood. In the process, certain ideas gain prominence, skeptical attitudes emerge, and resulting conflicts of opinion are debated. That theoretical ideas diverge is to be expected, especially in the clinical sciences; in psychiatry, it almost seems a given. Even when knowledge of illness and treatment appear to approach the problem adequately, arguments in psychiatry can linger without any single resolution enjoying general acceptance. For some it's a frustrating state of affairs; others see it as incentive for extending more rigorous analyses to novel ideas. Then again, in a far different way, the conflict of ideas sometimes justifies in the minds of still others that all beliefs are created equal. But their argument of equivalence arises only because the clinical phenomena we study and treat are not easily graspable.

This article focuses on controversies that have recently emerged in regard to AN. As the subtitle titles make clear, it is a long one, and the questions we take up are touchy. On the face of it, it reflects a renewed interest in AN that many in the eating disorder community take as reason for optimism that progress is at hand. Progress is indeed being made, but serious concerns overshadow what should be

an opportune time for new learning. They are broadly related to the different sources of information that clinician and academician access to increase their fund of knowledge, and how each generalizes empirical and experiential knowledge to the clinical setting.

It is an exciting time for eating disorders research because the opportunities for advancing knowledge are now rich. But ironically, there is also a knowledge chasm that is wide. Simply stated, in spite of a wealth of empirical and experiential knowledge that brings attention to the complexities embedded in AN, a counter-intuitive view now prevails in the minds of many, one that portrays the illness and its treatment in ways that do not suffice as truth. The two main sources of controversy center on (1) the explanatory role of causal genes and abnormal biology, and (2) whether family-based treatment for weight correction should have primacy in managing younger patients. That these are important topics for empirical investigation with implications we need to take seriously is not in question. Our concern is how quickly this research came to legitimize, through some unknown parity of reasoning, faulty propositions about causality and mental function in AN, and attitudes that many insist must command its treatment. This article offers an analysis of these beliefs—not only in relationship to other areas of scientific study, but also in the context of our clinical work—and show why they lack plausibility and thus fail to satisfy any acceptable criterion of a priori knowledge. We will not be arguing that a case can be made at this time for a specific theory about AN, only that certain increasingly popular ideas present an unbalanced picture of the illness, lack believability, and risk having undesirable consequences should therapists predicate interventions on their acceptance as irrefutable truths. The key point we will present is that whatever the origins of AN, the processes involved are quite intricate. We will stress this premise repeatedly, because we think the repetitions are necessary. Many of the points we will be making apply equally well to bulimia nervosa, but since we knew the article would be lengthy we elected to focus on AN alone.

In essence, the article asks which notions about AN—concerning vulnerability and psychopathology on the one hand, and subjectivity and therapy on the other—are rational to believe. In framing our answer, we will make reference to new research findings—because knowledge that accrues from science should inform clinical practice—and the every day challenges we confront taking care of patients—because inferences we draw about these

encounters can refine theoretical knowledge of how the human psyche forms and adapts to its surround.

That conflicts of opinion exist about causal factors in AN and what therapy attitude is the most justified is an interesting point, but unsurprising given how clinicians form beliefs about psychopathology and treatment; which leads to touchy questions: What brings well-intentioned researchers or therapists to disparage certain concepts without first considering whether it's rational to do so? We've witnessed it in the past 2 years—an insistence that they be retracted, as if the only conceivable reason for invoking them in the first place is self-interest or bias. And why do well-intentioned therapists wind up doing bad things? The reason our subtitles are apt is because the conflicts of opinion that have risen to the center of debate in our field actually lost validity some time ago—a fact many in our field seem unaware of.

Well conceived scientific investigation creates knowledge and theory that have plausibility. One would hope, then, that unity of opinion should follow as new understanding is disseminated to practitioners, patients, and the public; unfortunately, it's not so easy. Making the case for credible knowledge in psychiatry when different views of abnormality vie for primacy has long been challenging and it will remain so if rival theories are defended too narrowly, if research focuses on one element of knowledge to the neglect of others, and if new findings quickly generalize without waiting for corroboration from other sources. This is precisely why patience and skepticism are indispensable virtues when it comes to making sense of human actions that spin a story of puzzling complexity; when there is no single, sufficiently adequate testimony to their nature, when evidence is often overridden by counter-evidence, and when presumed truths are, in fact, assumptions devoid of evidential support. Applied to AN, these are hardly subtle distinctions considering that the narrowest of conceptions about its nature and treatment have taken hold in the opinion of many.

How much about AN can be spelled out by the effects of genes and biology and whether or not there is sufficient justification at this time for family-based management of weight restoration to replace other treatment models in the care of patients (in particular children and teens, as well as young adults still residing at home), are intriguing questions. As we already stated, each notion has been supporting ground for real progress. Clearly, a significant body of research shows that inherited vulnerability is an individually necessary compo-

nent of AN, justifying the idea that eating disorders should be seen in the same light as other biologically based mental illnesses (BBMI; see Klump et al.¹ for background). Likewise, the well-executed study by Lock et al.,² showing a greater benefit of family-based therapy (FBT) compared with individual therapy in promoting short-term remission in youth with AN, represents a meaningful contribution to clinical care. Unfortunately, the implications of these paradigms were quickly overstated and they are now being invoked by many as single, defensible solutions to the thicket of clinical challenges that AN presents. So questions arise: Do the BBMI and FBT paradigms, at least in the way they are being justified, offer coherent and substantive frameworks for approaching the illness? And can the divisive controversies that have emerged over their meaning and treatment implications be sensibly resolved? We believe they can.

But it will require adopting an overarching notion about human motivation that is inarguably factual. Simply stated, that to understand why in the long arc of human development some people thrive while others remain beholden to odd motives and self-defeating acts requires the subtlest appreciation of how complex processes shape behavior and self-concept; the complexity of human life, including its deviancies, is fact. So does it not stand to reason that broadly conceived approaches to behavior change should also occupy a prominent place in our attitudes about treatment when it comes to an illness that is emblematic of how challenging psychiatric treatment can be?

The need for openness to new knowledge has another important subtext: that some of the more notable successes in psychiatric therapeutics over the last half-century arose from a serendipity that turned conventional notions about biology upside down. Even empirically validated psychological therapies have been “hijacked” unexpectedly by principles of their own, corresponding little to original ideas about mechanisms and mediators of change (best illustrated by early studies of cognitive behavioral therapy for depression). This doesn't mean theory should be set aside when discussing treatment models for AN, as some have argued; to the contrary. As knowledge of human development strengthens, shining a brighter light on how normal and abnormal behavior evolves through environmental regulations of gene expression and a myriad of other complex processes, we have more to take account of. This is why it isn't closure around simple models that's needed, but rather efforts that seek connections between once separate lines of inquiry. So the good thing is that eating disorders

have finally entered the research mainstream; the not so good thing is that many in the field have become entrenched in single-sided notions, overlooking the value of combining insights from different theoretical and clinical traditions.

The Illusion of Consensus

It isn't that consensus among us is completely lacking; it's that it's illusory. Witness any discussion of AN and points of agreement are many; or so it seems. Regarding psychopathology, we agree that its symptoms quickly assume a will of their own and that in its more severe form the consequences are grave. We also appreciate that the illness has a stubbornness so persuasive it brings patients to do things different from what we plan for them. Parents describe it best: a transformation of their child's manner seemingly out of nowhere, as mystifying as it is frightening, often leaving them exhausted, in despair, and resentful. AN can not be explained simply, nor is the remedy predictable, because so many of its features—the suddenness of its onset, the rapidly peaking intensity, its ego-syntonic character—are inaccessible to single-focused ideas.

Also uncontroversial is that heritable factors operate at an important level in etiology. It's when discussion turns to equally strong evidence from research outside of eating disorders that environmental factors also play a role in pathological behavior, sometimes enhancing, sometimes mitigating, the vulnerabilities conferred by genes that consensus frays. Why acrimony ensues as soon as this notion is introduced is an intriguing question, especially when evidence of gene-by-environment interaction in psychological development is incontrovertible, and given that the evidence is strongest for phenotypes having a plausible clinical parallel to AN: stress sensitivity, cognitive bias toward threat, neuroticism, anxiety—even activity-based anorexia.^{3,4} To frame the irony differently, here we are bickering about whether or not rearing influences should be included in causal and treatment paradigms, at the same time that neuroscience-based models of psychiatric disorder elegantly show why inheritance shouldn't receive singular attention any more than early attachment deficits or family discord should. Simply put, the more we learn about molecular codes that play a role in vulnerability, the better is our appreciation that the origins of abnormal behavior travel a far distance from inherited variations of DNA.

Clinically, we easily agree that AN is an intimidating challenge and that therapists who take it on should have the highest level of skill development to help patients battle its emotional sway. But this is hardly the prevailing ideology in our professional dialogue. Instead, many in the field are attempting to reduce complex challenges to rudimentary ideas which then quickly take on such broad significance that a treatment model is born impromptu—some even insisting it should be adopted as our treatment of choice. Why? If complexity is bluntly etched not only in the psychopathology of AN but also in the challenges it creates, shouldn't this very same notion be a constant thread in the ideas we introduce during treatment? And if narrow ideas sufficed, why does the treatment of AN often veer off course and prove disappointing? As an example, consider the notion that eating and restoring weight to normal weight while preventing compensatory behaviors models extinction learning; in the most rudimentary sense this is true, but to think this rises to the level of an explanatory paradigm is short-sighted.

So we are not saying that opinions about AN shouldn't vary. It's when the intensity with which they are defended rests on assumptions too narrow to represent a powerful conceptual principle that knowledge suffers, along with patient care. If clear thinking is what we want in our professional and public discourse—it's certainly what our patients and families deserve—then entrenched viewpoints that ignore the many levels of analysis needed for explaining an illness layered in complexity must be set aside. And there is another point. Since errors in managing AN are sometimes set in motion before treatment actually begins (an assertion less incongruous than it sounds), modifying attitudes founded on sweeping generalizations may help to avoid these blunders from occurring in the first place. This article is, in effect, a dialectic that underscores why our field is wracked by confusion, mistrust, and divisiveness when it need not be.

The Impetus for This Clinical Perspective

It arose from a heated conversation that took place over dinner at a recent national meeting of eating disorder professionals. Present were the authors, several practitioners, and the founding member of a parent advocacy group with ties to the Academy for Eating Disorders (AED). The conversation eventually fixed on a question that was of particular concern to this parent, ironically enough harkening

back to the lament of 19th century pioneers—William Gull and Ernest-Charles Lasegue—that both practitioner and family often went too far in indulging the patient’s wish to manage eating on their own. Listening to this parent’s account of her daughter’s treatment by a “specialist” she initially assumed trustworthy, it was clear how the treatment went awry and why her anger was deserved. ‘Why do therapists wait too long before realizing their patient can not gain weight?’ was the question she put to us.

Unfortunately, therapist complacency in addressing malnutrition remains, giving credence to this parent’s belief that clear thinking about how to approach AN’s intransigence is still lacking. So as the conversation came to a close our dinner companion suggested, with further encouragement from others at the table, that the authors—friends and colleagues for nearly four decades—write a commentary on this issue and that it be accompanied by a proposal: that outpatient management follow definable benchmarks for determining when a therapist should stop doing what has not been helpful and replace it with something that might be; specifically, a level of care offering greater support for weight gain and reduction of psychopathology. We acknowledged this was a worthy suggestion because as far as we know, little has been written about specific, treatment-focused benchmarks in AN.

But as we began to write, we felt that a discussion focused on benchmarks alone would not suffice because while they can be plausibly described, implementing them at a point in treatment when symptoms are worsening or progress has stalled is a unique challenge; and as our key point argues, not many challenges in AN can be managed without convincing insights and strong clinical skills. We wanted our younger colleagues to understand this; it became an important part of the reason we are writing this article.

Our Backgrounds

Obviously, we wouldn’t have written it if we didn’t consider the question posed to us at dinner to be meaningful and answerable. Still, a perspective is personal, so we thought a brief summary of our backgrounds would be important.

Our careers have been long, nearing 40 years now. We were fortunate that our doctoral training in clinical psychology crossed several theoretical domains—social learning theory, cognitive and de-

velopmental psychology and psychopathology, principles of behavioral and psychodynamic theory, and brain–behavior relationships—and that after graduating we devoted much time to connecting research with patient-based experience; also, bio-phobic we’re not. The lead author’s masters and dissertation research investigated sleep electrophysiology in newborns at risk for severe mental disorder, and both of us have collaborated with authors of the AED Position Paper on Eating Disorders as Biologically Based Mental Illness¹ in the Price Foundation and National Institute of Mental Health funded studies of genetic factors in eating disorders.⁵ In addition, MS was a founding Fellow of the American Academy of Clinical Psychopharmacology and is a longstanding member of the Society of Biological Psychiatry, and CJ is a Principal Investigator in an NIMH-funded multicenter study comparing family behavioral treatment to systemic family therapy in adolescents with AN. Similarly, our academic and postdoctoral experiences were further strengthened by supervised experience in interdisciplinary clinical settings from multiple therapeutic viewpoints. Finally, we have spent our entire career working in combined service and research environments that provide care to the most seriously ill children and adults (and families), as well as to those less impaired. Between us, the number of patients and families we have treated privately, treatments that we have supervised directly in the centers we direct, and consultations we have conducted with juvenile and adult patients (and families), is on the order of 12,000.

The summary will strike some as overly self-regarding, but seeing how ideas on etiology and treatment are now as easily accepted as they are dismissed, there is no stand-in for theoretical diversity and depth of clinical experience when taking on matters about which so many in the Academy disagree. Thus, we feel well prepared to address the disciplinary blinders and doctrinaire attitudes now hindering the rapprochement of ideas our field urgently needs.

Our Framework

Modeling treatment on advances in our understanding of pathological processes in AN has long proven difficult; this is why controversy can be a good thing. But the sort of debate we need is not possible if arguments depend on fixed beliefs that are at odds with the very research cited in their defense, and if new treatment ideas are greeted either with gushing enthusiasm or disdain, rather

than caution and humility. To be clear, we are focusing on the BBMI and FBT paradigms not because we disagree with them—there is good empirical support for the original concepts—but because a larger body of research shows that the interpretation of each needs softening, and because many have come to see the BBMI paradigm as justifying a therapy philosophy we believe is not only unwarranted, but contrary to good clinical care. What we mean here is that an increasingly genocentric view of AN is being overinterpreted to mean that weight correction is an absolute biological prerequisite for any treatment that is more “psychological” in character. At first blush, this seems a reasonable notion; but it needs to be nuanced. Obviously, if low body weight continues so will psychopathology and no therapy-derived insight is compelling enough to stand on its own in reversing the disease; behavior change is, of course, crucial to anyone’s definition of recovery; without weight correction the prospect for sustained recovery is nil. Just the same, there is no evidence—empirical or clinical—showing that normal weight is a necessary prerequisite for initiating meaningful psychological dialogue, or that psychotherapeutic dialogue can not be facilitative of weight change. It’s a bit like mixing apples and turnips. A patient 50 pounds below a BMI of 19—confused, disorganized, unable to retain short-term information, and emotionally erratic—is unquestionably ill-suited for psychotherapy of any sort. But this patient is considerably different from one who is 30 pounds underweight, ingesting food, and though compelled by similar fears is nevertheless committed to an examination of the conflict that has taken hold of his or her mind. Simply stated, normalization of weight is not the absolutely essential starting point for using thought, reason, and insight as foundations for change.

There is another paradox of sorts that needs mention here. A fundamental principle of FBT holds that parents must separate the symptoms of AN from the person who bears them. It’s an eminently sensible concept because the separation, if successful, can buffer against additional personal and family strain; especially for the patient, whose already harsh self-disparagement is injury enough. Just the same, there are histories in which parental attitudes, environmental strain, and psychopathology are not so easily segregated and the interventional efforts needed are more involved.

So our concerns are (1), the absence of support for many of the clinical assumptions the BBMI and FBT paradigms have given rise to, and (2), a philosophical attitude about treatment that is becoming

far too circumscribed, moving in a direction that suggests—we now hear it stated often—that only techniques supported by controlled, empirical study deserve consideration. Again, to eliminate any misunderstanding, we are not contrarians assailing empirical research on biology or treatment techniques. Biology is an essential field of inquiry and the study of FBT by Lock et al. is a signal development with important implications. What we worry about is that many therapists have reified the effects of genes and took the results reported by Lock et al. to mean that FBT is the only justified treatment for young people with AN, overlooking the fact that 50% of the participants who received it were unremitted. In our view, the present disunity in our field underscores three worrisome trends: (1) that many therapists apparently see no place for the sort of clinical wisdom that can never be manualized; (2) that the emphasis on empirically validated interventions is drawing attention away from more broad-based training experiences; and (3) that therapists who will one day encounter very ill patients are not being prepared adequately for taking on the many complex predicaments they will face. In short, although it is without question that FBT will suffice as a first-line intervention for some young patients with AN (most likely those whose vulnerability load is less extreme), to insist it is the only treatment modality that deserves consideration doesn’t translate well for clinicians who have seen AN’s many faces over many years.

So the dialect underscores the need for attitudes about the BBMI and FBT paradigms to move in the direction of perspective taking: an appreciation of their value, but with finer distinctions that take into account knowledge gained from long-term clinical experience and translational research showing why a more inclusive vision of its complexity is imperative.

In one sense, that we have come to this critical juncture isn’t unexpected. In a field where soft ideas have long taken precedence, paradigms supported by empirically verified observations will naturally demand strong attention, as they should. But as we said earlier, there is a caveat: that belief about complex clinical issues is useful only if it portrays knowledge accurately and comprehensively. On the surface, the proposition is straightforward; in reality, it’s anything but; because debate over what we should or should not believe inevitably takes place in an interpersonal context of one person (or group) trying to convince another that the idea they have invested time and effort in is a fallacy. This is where the intellectual analysis of ideas becomes tricky, because human nature being what

it is it doesn't take much to violate the operative notion that this form of discourse must be emotion neutral. Realistically, the only viable solution to the problem of close mindedness is to carefully consider if the ideas we justify as knowledge are defensible regardless of personal ideology, and to resist turning a blind eye to ideas we may be inclined to reject spontaneously and uncritically. As a concrete example, it would mean that if we held strongly to psychoanalytic principals, we would not reject out of hand evidence of a cognitive mechanism operating in symptom formation. Cutting straight to the point, however strong our commitment might be to an ideology it should never cordon us off from considering the possible relevance of other concepts about human behavior.

So we unapologetically acknowledge that a purpose of this article is to challenge ideas tied to the BBMI and FBT paradigms that many now assume to be fact; to offer an understanding of why they hold only a piece of truth about AN—an important piece to be sure—but not a whole truth, and to see that when a less demanding theoretical and clinical calculus is applied to causal biology and psychopathology that is complex it inevitably faces disappointment. Our objective thus brings us to several touchy questions: Why do our treatments help some but not others? What brings well-intentioned care givers to do unwise things? And why has there been precious little discussion at recent Academy meetings about the importance of approaching AN with diverse treatment skills?

The Challenge and Its Complexities

That the questions are timely should be clear to anyone who has attempted to fully understand the illness. AN expresses a more or less lawful history and clinical presentation, but once it takes hold its clinical borders shift quickly in directions not easily anticipated. We do our best to warn patients that they are feeling the pull of misguided ideas, but they are tone deaf to the standard logic applied to health. To them, only one wisdom matters: whatever actions succeed in losing more weight is a sensible alliance to strike. Of course the search for inherited genes should continue; because the effort is as important to AN as to any other psychiatric illness. AN is certainly a multilocus condition whose eventual expression depends on multiple predisposing traits, chief among which are anxious worry and hypervigilance, reward deficits, compulsiveness of habits, and an obsessional thought structure. But to assume that everything about it is

encoded in the genome, that a single biological calculus will crack open the mystery because environmental influences are trivial, that our culture's embrace of a thin body ideal is the primary nongenetic trigger that warrants focus in prevention efforts are notions that seriously misjudge all of what the illness embodies.

Another facet of AN particularly unnerving, especially for the less seasoned therapist, is that the clinical acumen therapists need builds slowly but the fear the illness incites is instantaneous. And it's a different sort of fear, one that cares little to nothing about our background—student, beginner, scholar, or therapy veteran—what we look like, the gentleness of our manner, even the dedication we bring to the work is immaterial. AN is maddening because the interpersonal context it creates is impersonal and insolent, which is why it doesn't take long for patients to turn uncooperative and for things to feel out of our hands. And when it does, the risk for treatment to lurch back and forth in ways perceived by patient and family as incoherent or needlessly inflexible—or, maybe too flexible—will be great. This much is certain: when the fear strikes, challenges will mount quickly and before long skeptical loved ones will start questioning our every move; this is when we learn never to presume that treatment approach in AN is straightforward; not because it never is, but because in so many cases it isn't. The common lesson learned in clinical work is that the skill set needed to keep the management of AN on track transcends what is portrayed in the most rigorously articulated treatment manual.

Historical Footnotes

In 1982, Paul Garfinkel and David Garner published a highly regarded discussion of the multidimensional character of AN.⁶ Johnson and Connor followed in 1987⁷ with a similar book exploring bulimia nervosa from a bio-psycho-social perspective. Thus, well over two decades ago there was awareness that eating disorders would require analyses that kept multiple levels of discourse in play, each having unique value. But history fades quickly and this bit of seminal discourse now sits on the side lines, paid little more than lip service—acknowledged, but more as an afterthought. So at the same time that our annual workshops and plenary titles are stressing integration and synthesis, resistance to setting aside favored ideas remains strong. Again, we are not decrying differences of opinion—it is a sacred part of scientific and clinical debate. But there is no opportunity for sober dialogue about ideas relevant to issues of causality or

our treatments when arguments are laced with vitriol and the points argued have been shown by other science to be false or overstated. If the uncivil tone of arguments that scrolled across the AED List Serve last year is indication of our field's readiness for synthesis and integration, we are in trouble.

On the Importance of Integrative Models of Etiology and Treatment

Returning to our keynote, complexity is the foremost principle of human development, as crucial to understanding how healthy behavior evolves as it is to decoding how behavior turns pathological. Because of this truism, paradigms that ignore complexity inevitably sacrifice real world applicability, not because their broad outline is incorrect, but because they do not go far enough in their explanations. In our view, the reason we are in the throes of rancorous argument about BBMI and FBT is because the implications of this complexity are being ignored.

It is not an inherent contradiction to argue that paradigms can be informative at the same time they are incomplete. BBMI warrants attention because in addition to advancing knowledge of possible causal mechanisms it has had far ranging implications for health care policy in the United States—the inclusion of eating disorders among the list of insurance parity diagnoses being a case in point. Similarly, though only a single controlled study, the report on FBT by Lock et al. was well executed and its main result is straightforward; but the study's safety parameters required participants to be medically stable for outpatient treatment and to be 75% or greater of ideal body weight. These are not criticisms, merely clarifications meant to draw a contrast between what is, and what is not, indicated by a single study and why good sense must guide its interpretation; clearly, good sense in its interpretation has, thus far, been missing.

This is how scientific advances often come with unfortunate trade-offs, and why a measured interpretation of their meaning is needed to lower the risk of pronouncements the research never intended. We know the authors of the AED Position Paper on BBMI and are confident they do not hold to some, or most, of the ideas the paradigm immediately gave rise to. As for FBT, the clinical insights gained by the research were almost immediately muddled by inexcusably sharp-tongued criticism and undeserved pronouncements about treatment philosophy. So let's take a breath, step back, and remember that both praise and criticism must be cautiously and smartly rendered. Genes and biol-

ogy matter greatly in vulnerability to AN, but environmental influences can matter too. As for FBT, it is neither the Holy Grail of AN treatment nor something villainous.

Revisiting Predisposing Biology

By their very nature, all psychiatric disorders are extremes of human nature. However, the phenomenology of AN stands apart from other mental disorders, not simply its strangeness, but more the ease with which patients shrug off its seriousness. Morton smartly drew attention to this more than three centuries ago⁸ in his treatise on tuberculosis, when he attributed an odd indifference to malnutrition among certain of his wasted patients not to a pathogen, but rather to “passions of the mind.” Modern epidemiological research confirms, with cold facts, the implications: should AN progress in severity the risk of premature death from starvation or suicide rises dramatically. To be in the presence of someone so withered yet so driven, who insists their wasted appearance—if they acknowledge it at all—is more illusion than omen of approaching death, triggers a suffocating fear. Then, to watch helplessly as this unforgiving madness stretches starvation to a degree of physical endurance that seems inconceivable, even taking precedence over the frantic pleas of loved ones to try one more bite—it's a grimly visceral tragedy not justly described; to think biology is not involved in some way is hard to fathom.

But the more common menace of AN is not death; it is the risk of a long, persisting illness, usually with symptoms of lesser intensity but that still impact quality of life. Why the illness is fatal to some but not others, why some recover fully while others struggle a lifetime, are critically important questions for which there are no immediate answers. But one generalization stands up to reason: that when vulnerability is at the tail of the bell-shaped curve the risks of symptom persistence, and possibly death, are greater. Even before evidence of genetic influence in eating disorders came to light there were other commonsense reasons to presume biology was at play; it is hard to imagine otherwise with a madness that comes on quickly without any truly rational explanation. But now, with decades of evidence showing that inherited influences are present in most, if not all, major psychiatric illness, the evidence pertaining to AN is incontrovertible: (a) eating disorders are transmitted in families; (b) concordance is greater in identical compared to nonidentical twin pairs; (c) characteristics associated with AN (regimented behavior and perfectionistic attitudes, aversion to

risk and low reward seeking, anxious worry and elevated vigilance) appear to be tags of vulnerability as they are present before signs of weight loss and are also reflected in its core diagnostic symptoms; (d) anxiety states are not limited to patients only, but also occur more frequently in relatives of persons with AN compared to relatives of non-AN controls; (e) as common as body image concerns and weight dissatisfaction are in the general female population, AN is, by comparison, rare; (f) alterations in brain physiology mediating reward seeking and anxiety have been reported, some persisting after normal weight has been restored. This concisely summarizes the strength of the BBMI paradigm (supporting references can be found elsewhere.⁹⁻¹¹

It's when it comes to the question of what specific elements of AN and its outcome are encoded in the genome and where other sources intervene that we are at a loss. Indeed, if there is a single, overarching seminal idea emerging from recent neuroscience research it is that psychiatric illness reveals processes more elusive than the effects of vulnerability genes alone. So we turn next to the striking contrast between this body of new knowledge and what many in the Academy deem to be justified beliefs. Some of the statements listed below were made at the 2010 and 2011 Academy meetings, others we picked up from the AED List Serve. They are paraphrased—some are embellished—but only to make the point that it has become easy in our field for misunderstanding, misattribution, and plain lack of knowledge to stand in for clinical wisdom.

1. It is now proven that AN is a brain disease; this explains why patients behave strangely and say illogical things—their actions, perceptions, and utterances are irrational because their brain is.
2. AN is a genetic disorder; this is why you have it forever and why psychosocial factors are less relevant in causation or in determining outcome; features once thought to be part of its psychological realm are really effects of its genetic underpinnings, having no unique significance of their own.
3. The BBMI model doesn't deserve the strong attention it is receiving because the methods now used to study brain biology are prone to over-interpretation.
4. Because AN is a brain-based illness, family turmoil should be viewed only as a byproduct of the frustration the illness sows; weight correction needs to take priority for these tensions to resolve; things said by patients

about their relationships, family ones included, should not to be taken too seriously.

5. Psychotherapy cannot, and should not, take place until the brain is mended by restoring weight to normal.
6. Family-based behavioral therapy is the only acceptable method for treating young patients.
7. The FBT approach to weight restoration is crude and atheoretical, unjustly diverting attention away from critical psychological needs of the patient; it disparages psychotherapy.

What is instructive about this list of dueling assertions, and worrying, is that it didn't take long for biology to be turned on its head in ways the Klump et al.¹ Position Paper never intended, and for treatment research focused on FBT to be wildly generalized. It is little wonder that so many patients and families tell us they feel at a loss, not knowing who to turn to for sound, factual advice.

Expanding the Vistas of Biology, Development, and Environment

We shouldn't forget that opinions about causality and treatment of mental illness from the early 20th century forward were as wide ranging and contentiously debated as were attitudes about AN. What's different today is that ideologies take hold rapidly because science technology and the conceptual principles on which it rests are more sophisticated, and new research findings now appear almost daily. The wrinkle is that while the roles of inheritance and biology in AN are now well accepted, the translational implications of research findings in allied fields have been slow to enter our dialogue. As we said earlier, the good thing is that research on eating disorders has entered the biomedical and psychological mainstream; the not so good thing is that many Academy members are woefully unfamiliar with this new science. This, we believe, is why the BBMI and FBT paradigms have needlessly become crucibles for dissention and acrimony and why it is important that our field become better acquainted with the bullet points that follow (interested readers are referred elsewhere for a summary of primary sources¹¹):

1. Not only are genes and environment correlated, the effects of each coevolve and interact, which is to say that neither influence is fully deterministic. Regarding the temperament that underlies AN, these correlations

- explain why anxious, worry prone, and stubbornly perfectionistic children not only seek out certain types of social environments, they also “invite” them; because genes and the biology they express make it so. By the same token, because genes and environments also meet randomly, they can clash. For this reason, everyday experiences never guarantee the predictability that children vulnerable to AN prefer and need, which they mistakenly believe they can achieve by imposing strict order on their daily routines. The ultimate “failure” of their innately formed discipline to achieve the unfailingly ordered world they seek is difficult for people with AN to concede and it may be one reason they are prone to the harsh self-judgments of inadequacy, passivity, and mediocrity they bear.
2. Brain circuitry is not fixed, but rather adapts functionally to the environment and then depends on it for its continuing expression. Moreover, neural circuits have plasticity and thus support different, and at times contrasting, motivational drives depending on the environment’s emotional tone; this is why circuit activity is a less than reliable biomarker for abnormal behavior. The search for general and specific indicators of AN risk should continue, but none presently exist nor have we yet identified neurobehavioral correlates of discrete treatment responsive or unresponsive subgroups.
 3. Neurochemical changes brought on by stress (including exposure to adverse rearing environments) not only strengthen the encoding of memories for negative emotional events, they also reshape brain morphology in regions linked to anxiety, fear learning, and reward seeking. Particularly intriguing in this regard is recent evidence (see¹² for background) that even less extreme variations in human care giving can impact the trajectory of developing brain systems that mediate affective and motivational characteristics. For example, whereas the left and right frontal hemispheres are differentially specialized for processing appetitive versus aversive stimuli, respectively (the left frontal region mediating approach and appetitive behavior; the right frontal region promoting avoidance, fear, and behavioral inhibition), a greater right compared with left frontal activation occurs when children are reared in adverse environments—a shift that is associated with increased risk for internalizing behaviors (some of the very same phenotypes characteristic of AN).
 4. It is now clear that environmental stress mechanistically increases anxiety proneness by over-sensitizing fear-generating structures in the limbic brain (amygdala nuclei) and disrupting prefrontal modulation of this region. But the converse is equally powerful: that rearing in environments characterized by parental warmth can silence genes that otherwise promote anxiety,¹³ a finding that may possibly shed at least some light on why positive family relations have been linked to better short- and long-term outcomes in AN.^{14,15} In short, the social world plays an impressively large role in shaping behavioral outcomes. Referring back to points 2 and 3, this is why it has been recently argued¹⁶ that so-called vulnerability genes might actually support adaptation in a context dependent manner: programming fear/avoidance when rearing has been adverse so the child can respond with increased vigilance and avoidance when later facing environments that are anxiogenic (i.e., stress-inducing, novel, or generating discomfort in any way; also note here that discomfort with novelty and diminished motivation by reward are common in AN), but promoting appetitive motivation when rearing is favorable (note again, the link between positive family relationships and more favorable outcome in AN).
 5. A final point concerns a newly described genetic mechanism—epigenesis—whose effects may underlie at least some of the patterns described above. In contrast to inheritance of a nucleotide sequence, epigenesis refers to a heritable, nonstructural modification of the genome in which gene expression is set-off by environmental events. A classic illustration¹³ is the developmental effect on hippocampal–hypothalamic regulation of stress that results from variation in licking and grooming of newborn rat pups by mothers (a model of maternal care). The effect is robust, with high grooming mothers producing offspring with less stress reactivity (i.e., less nervousness). Interestingly, epigenetic studies have also shown that stress to a parent alters not only their behavior, but that of future generations—even when these offspring receive no direct exposure to environmental stress themselves.¹⁷ Exactly how this cross-generation transmission of susceptibility occurs isn’t known—behavioral modeling

of parenting behavior from one generation to the next, or stress effects on germ cells which are then transmitted across generations are possibilities—but whatever the mechanism the effect supports the provocative idea that environmental programming is operative even at the embryonic level. The take home message is that continuities between stress, development, and parent behavior begin to form early and the dynamic processes involved can suddenly activate feelings of threat in offspring when novel environments are encountered later in life. But the converse is equally true: when a rearing environment signals a low likelihood of stress, fear behavior is less automatic and the potential negative consequences of later stress exposure are reduced because HPA and limbic arousal is better regulated by learning experiences that promote inhibitory brain processes.

The general implication of this body of research is obvious: that for paradigms of AN to be complete requires their studious appraisal of the many strands of vulnerability involved in symptom development and illness progression, including: susceptibility genes; the effects exerted by environmental stress on the expression of inherited traits; and other nongenetic effects in the programming of long-lasting behavioral patterns. What is significant here is not only the heuristic value of this science, it is that when translated appropriately it can further help therapist, patient, and family make better sense of an illness that seems ungraspable. And for this very same reason it justifies what should already be a common sense notion: that our therapies should not overlook the strains of life that potentially maintain or accentuate vulnerability, and that interventions targeting the family, school, or interpersonal environment can have benefit. Indeed, evidence from outside our field (research on youth with mood disorders) is showing that multifocus, family-based therapies for stress-ridden, emotionally reactive families can buffer sensitivities that high risk genes lay down.¹⁸

In sum, the reason long-term outcomes in pathological behavior are not easily predicted is because while genes influence the outline of our temperament, environmental conditions program and moderate inherited tendencies through complex feedback loops that determine how behavioral patterns eventually stabilize; simple and linear our development is not. Inheritance wires biology, biology expresses behavior and is also substrate for the experiences behavior generates, experience then

rewires biology in order that behavioral phenotypes adapt optimally to unique environmental “demands,” and from this dizzying cascade of interweaving events the mind’s consciousness forms.

So after taking account of how genes constrain adjustment, why would we dispute clinical formulations that properly reflect other far reaching influences that shape the mental subtleties inherent to self-concept and motivated acts? Why should this be, when science now shows that whatever genetic predisposition to psychological illness sets in motion, the effects unfold in a social milieu whose imprints of learning, experience, and expectation also enter into the long, cumulative causal chain from which a host of ideas arise—some rational, others imagined and far-fetched? This is why we submit that AN can not be understood comprehensively unless the fundamentally important notion of complexity is given credence; that after biology, the rearing environment—the larger social context too—can imbue symptoms with deeply felt, personal undertones that if not taken seriously can significantly short-change our treatments.

We now offer three vignettes which makes the point. We include them as an integral part of the discussion because through the lens of our perspective each is a perfectly good acid test of how we are doing at reflecting the synthesis that we hold to be crucially important to our daily work.

Clinical Illustrations

S is a 17 year female whose illness has lingered without remission for 3 years; at the commencement of this round of treatment her BMI was 12.8. The youngest of three children, S excelled in school—an independent thinker who stubbornly resisted outside assistance even when confronted with challenges that stymied her; insisted that her day be strictly organized from the time she rose until her bedtime and that certain routines were essential for “correct” behavior; and exhibited nervousness from an early age, especially at the possibility of making mistakes or not knowing the answer to a question a teacher might unexpectedly throw her way. Everyone in the family described S as unusually disciplined in all aspects of her behavior and would unhesitatingly sacrifice personal needs for the wellbeing of others. S had friends, but she secretly disdained any peer who, in her eyes, squandered their free time, preferring to fill her own free time with a myriad of “worthy” goals and objectives she believed would hone skills that would prepare her for future pursuits. S vehemently

insisted her self-esteem was strong. But following the start of her illness she admitted to a feeling described as “hollowness,” causing her to fear that her once steely resolve was weakening. She chastised herself for secretly wishing she could relax more and be spontaneous like her friends. As treatment progressed, she started to acknowledge, begrudgingly at first, that she first longed for a greater sense of freedom—“to just be like everyone else”—around age 11, and that her self-esteem was, in truth, always shaky. It was at roughly this same time in her life that her father, who described himself as painfully shy and nervous, was suddenly laid off due to the economic downturn and though the family was well set financially, he grew withdrawn and sullen. Witnessing this, S felt torn between caring for him and seeking greater personal comfort and support from friends and other family. Struggling to reconcile the conflicting urges, she reigned herself in more, refocused on school, and gave herself unselfishly to family needs; it was not the first time she felt so inclined. When she was 8 an older sibling took seriously ill, a family-wide strain that lingers. Looking back, her father volunteered that the stress of this illness was probably aggravated by his inherent nervousness, which he was sure impacted S more than anyone else. Adding to the story, S’s older sibling described both her parents as worriers, but that mother covered it better by periods of excessive drinking, which S admitted she had witnessed but never felt comfortable talking about. S described her parents’ as being prone to worry over even minor matters and assuming the worst of all possible outcomes when challenged in any way. Two weeks following her hospital admission she spontaneously expressed an opinion that her illness served many purposes: suppressing hunger made her feel her character was strong; eating as little as possible enhanced her discipline, which, she asserted, was crucial to aiding her family to cope with her sibling’s illness; and that losing weight was not only key to excelling in school—irrefutable proof of self-restraint and single-mindedness—it also pulled her attention away from worry and anger over her mother’s drinking and her father’s passivity. In a family session roughly three weeks post-admission, the therapist unexpectedly pushed hard for each family member to discuss, in detail, the nature and content of every one of their strains; to describe not only the specific points of conflict within the family’s shared experience that incited their emotions, but also what issues intensified their worry the most. S appeared instantaneously uncomfortable. This was noticed by the therapist, who asked that S not interrupt the flow of

discussion but rather sit quietly, observe, take notice of anything that stood out, and feel whatever discomfort she was having; but first, S was asked why she blanched. She said she was afraid that her parents would not welcome this line of question, would grow uncomfortable, and that later at home the adverse effects of being challenged in this way would spill out and that she would feel compelled to pick up the pieces by calling her mother throughout the next day to see how she was managing. The therapist’s response was, “Then let this happen.” The following day, S spoke of the session. She described the unease it generated as she listened to her parents’ revelations, and the need she also felt to keep her unease in check. Nevertheless, she and her family appreciated the importance of what they were asked to jointly experience. What unfolded in subsequent sessions was further discussion of the role discipline had long played in her development, her instinctive unease whenever emotion—of any sort—was triggered by social or academic challenge or excitement, the alarm triggered by seeing her family in distress, and the conviction that food avoidance was not only a measure of the discipline needed to effectively reign herself in so should could offer an anchor of support to her parents, but also an effective distraction from an array of personal and family related concerns. S and her family are working well together, engaged in treatment with openness and commitment. S also states she is more accepting of the need for her family to play a supervisory role in her eating and weight gain now that they are speaking more honestly about things that had been buried for years and managing their emotional life more effectively. Her weight gain is still triggering, but the wisdom she is expressing in her individual and group therapy is genuine.

L is a 14-year-old girl with an 8-month history of illness. Very petite, people think she is 11 years old; she finds this comforting and is equally unfazed by her BMI of 11.2, hoping she can lose 10 additional pounds. L is unusually frank when discussing this, attributing her wish to be small to constant self-doubt, anxiety, and resentment at having to grow older when, in her view, her living skills are still poor. She has been compliant with meals, but she frets daily about not being prepared for what others naturally expect of a teenager; she would much prefer living with her parents forever. Her parents state these characteristics have shadowed L’s development from an early age. They are supportive and loving people and came to treatment eager to explore anything and everything the treatment team thought might help them, and their daughter.

Two weeks following the start of treatment L confided to her therapist that there was something in her history that her parents were not acknowledging: her father's heavy night time drinking, which, for several years, she witnessed with extreme fear. When she was 12, L divulged to her mother what she had seen, and that she was constantly afraid because her father was obviously ignoring the seriousness of this problem; mother assured L everything was okay. L told her therapist that as she was losing weight she caught herself wondering if it might compel her father to take better care of his own health. Shortly after revealing this in her individual therapy she discussed it openly in a family session. Choosing her words carefully, she said she knew her body image was its own problem, tied in some way to her anxieties, but there was hypocrisy in her father worrying so much about her appearance while ignoring his own health. Father was genuinely moved by L's directness, especially as neither he nor her mother had ever seen this before. He admitted he had ignored the impact of his behavior on his family, and L's mother acknowledged, as well, that she had been reluctant to confront her husband. L also asked her father whether her weight gain would cause him to see her as a "stronger" person and, if so, would this encourage him to start drinking again? His assurance that it would not happen—that he would seek treatment and stay with it—was welcomed by L. Although her weight concerns did not sharply lessen, she said her father's response to her emotions helped ease what had been a powerful anxiety. As a result she felt more at ease taking meals with them.

M is 29 years old, haunted by an 18-year history of uninterrupted AN. Her drive to excel surfaced early and it remains unrelenting, usually leaving her exhausted and unfulfilled; M says she knows its source is innate. She also remembers being a "high strung" child, a trait she shares with both parents; anxiety and alcohol abuse are prevalent across three generations of relatives on both sides. When signs of puberty emerged at age 11, she remembered recoiling, apprehensive that adolescence would be a distraction, dulling her drive to achieve excellence in every sphere and reducing her productivity. So when her weight jumped several pounds when she 11, she was convinced her discipline had indeed collapsed. In less than 3 months her weight had dropped to 61 pounds. Speaking of this period in her life M remembered feeling startled by puberty because it was unnervingly sudden; so were the changes emerging in her school friends, and at home: strange new hair styles, rising hem lines, flirting with boys and adopting odd new

curiosities, and her parents' constant squabbling without either one asking for a reprieve. It was, according to M, too much for her rigid character to take in. And the strain didn't stop here. When M wasn't fretting about her homework, her calories, or how to relate to an adolescent world she couldn't fathom, she worried about each of her parent's wellness. Her father was a reclusive, emotionally limited man without friends, her mother a diabetic since childhood who was significantly overweight and who neglected herself; her fear was that each would desert her. M recognized that her relationship with her parents was "complicated." Although they were neither emotionally nor physically expressive and rarely did they make attempts to engage with her, she loved them deeply and considered them unique and special people. At the same time, it disturbed her that it was always a nonrelative adult who set in motion each of her three hospital admissions when her weight dropped to alarming levels. Reflecting on this part of the history, M said she learned early on to care for herself and believed that isolation and rigid over-control of dependency was normative; however, as adolescence crossed over into adulthood she grew restless, bitter, and torn when pondering why her parents said little about her emaciated appearance. At present, M is nearing completion of a PhD in public health administration. She has returned to treatment in despair, questioning if her life has purpose and feeling the deep void that isolation from others has left in its wake. It has been an aching pain she has covered with fits of anger, convinced that any desire for intimate connection is, in the end, farcical; that if her own parents neglected the seriousness of her illness since she was 11, how could any other human being deem her worthy of care now? She is compliant with meals, but she disdains eating, regarding it not as a quintessentially human need but rather a "mortifying" admission of "passivity," no different than wishing desperately for something that will never be—human connection. She says that the conflict of ideals raging in her head has become tyrannical—between self-denial and disdain for nurturing (and nourishment, which she regards as its symbolic equivalent) on the one hand, and wanting to be free of the misery her illness on the other. "The more I think back to how lonely I was growing up and how empty our family life was, the angrier I become; that's when my obsession to lose more weight starts screaming so loud I can't stand it. It's crazy. What I always admired about my father was his discipline. And now he doesn't seem to care that this is what's going to kill me." From the time she was young, M,

like many others with AN, felt in her bones, “If I’m not everything, I’ll be nothing.”

These are complex case histories drawn from countless others. They have been included not only because they give voice to the struggle that AN often is, but also to provide context for the points we have been discussing, and those that will follow. The complexities illustrated are not seen in every case of AN, but when they are we need to think twice before offering pronouncements about the meaninglessness of events in the nonbiological realm and insisting that one-sided notions of determinism and illness pathways are adequate. The indelible mark of genes is obvious here, not only in the temperamental anxiety, rigidity, and low hedonic drive that mark the development of these young women, but also in the psychological environment their parents have shaped. But beyond this, rearing environments and the demands they place on children can also be less than optimal for psychological health and for this reason must be taken into account. It is a factual point, not an accusation, and to mention the obvious counterpoint—family rearing in other cases of AN is completely unremarkable—almost seems superfluous. Clearly, family rearing is not causal in any primary sense, nor is there any evidence to suggest it is required for every case AN to emerge; but neither is it irrelevant. Genes are surely the starting point for these life trajectories, selecting for particular traits that, depending on environmental conditions, will react to the psychological “demands” of the environment within a predetermined range and with a particular phenomenology. Whatever the demand is (to lessen the load brought on by a member’s illness; an imagined, or overtly imposed, “duty” to make up for a misbehaving sibling’s negative impact on family life; a perceived—rightly or wrongly—parental short-coming; adjusting to rearing low in nurturing or one burdened by instability, harshness, or trauma), the strain and hardship it can set off should not be relegated in importance. Good science proves the point: symptoms of psychological illness do not exist in an impersonal vacuum. The unfortunate fact of development for people vulnerable to AN is that it represents a trade-off: inherited genes program for certain forms of adaptation (rigid control, avoidance, and restraint) when novelty, stress, and emotional discord are encountered, but the benefits patients attribute to these patterns are illusory and their future consequences can be grim. In short, to hold to the notion that biology explains most of what is core in AN and FBT is the only treatment that matters will explain too little to really help patient and family

resolve struggles that are achingly complex. This is why a broad vision of treatment is needed and why our concern with therapeutic nihilism is a real one.

The concern is hardly cheap hyperbole (also see Waller’s¹⁹ thoughtful appraisal of how motivation is currently viewed in eating disorder circles). In recent consultations we have listened to patients complain that anger they had been expressing in their therapy had been discounted—“a part of the disorder,” said their therapists, who assured it will cease once weight increased. Yes, some outbursts are just that—irrational anger set off by the reasonable efforts of loved ones or therapists to maintain a steady hand. But every upset emotion a distortion wrought by starvation? The patients we are referring to here had upsets completely unrelated to the need for weight gain. This increasingly popular notion—that the heated emotion of patients and the seemingly volitional nature of their actions is driven by biology alone and will be tempered by refeeding—deserves a heavy dose of skepticism because it is too superficial a notion to permit understanding of the reactive processes that, over time, link biology to its context and shape ideas that are also central to AN: about motivation, identity, conflict, and maturational fears. The three case histories make the point that AN is an adaptation that biology alone can not wrap itself around.

Other Clinical Challenges: Why Experience, Skill, and Complex Ideas Matter

Thus, the question arises: How well are the current emphases on biology and manualized treatments preparing young therapists for the challenges they will soon encounter? And given recent attention on outpatient, family-based therapy and the strong allegiance pledged by many therapists to the model, let’s also consider the question of whether or not AN’s more extreme morbidity is being taken too lightly. Unfortunately, here, too, the concerns are justified.

We have recently crossed swords with providers oddly opposed to hospital-based care, not only for patients at low weight for periods ranging from months to years, but also for those whose weight was on the decline. The argument has been that inpatient treatment is “known” to be ineffective (in fact, there isn’t a stitch of evidence that supports this statement), and that if their patient is exposed to ones hospitalized they will only learn dishonesty and deviousness; yes, this can happen. But the

logic here is so obviously tortured you wonder how it leaves someone's mouth uncensored: "I insist on protecting my patient by maintaining the present treatment course at the risk of even greater weight loss, further bone decay, more psychological malaise, and an even stronger conviction that weight loss is the ultimate salvation." It's this sort of foolishness that leaves anyone experienced in the care of very ill patients scratching their head in bafflement and dismay. Perhaps more irritating is that this absurdity is almost always argued by therapists who have never worked in a hospital setting. Of course hospitalization carries significant risk; we hear the horror stories quit often. But the problems arise mainly in settings where the treatment approach is either incoherent or coercive; because the program's leadership and staff are minimally skilled and the treatment philosophy is superficially narrow and pedestrian. Sadly, the effects of poor treatment, no matter where it takes place, can be far reaching. Beyond the branding effect—all hospitals/day hospitals/therapists should be avoided—the lack of geographically accessible, high-quality inpatient programs (or limited availability of skilled therapists), only compounds the problem. But shrill attacks from practitioners who know little to nothing about the high quality of care that is possible in well-regarded treatment centers and who fail to grasp the urgency of intervening with more intense levels of management when AN advances are difficult to stomach.

The Therapist's Task

Judging the intensity of the dispositional traits that foreshadow AN is a critical task; this is because once irrational attitudes about weight, appearance, and dietary restraint unfold these factors play a role in driving symptom intensities, their resistance to rational argument, and the strength of the reward weight loss brings. To have this knowledge is to be forewarned because when extreme habit rigidity and anxiety converge, AN's remedy is far more difficult and the self-belittling ideas, shame, and convictions of inadequacy that patients harbor are highly resist to challenge. So taking into consideration how difficult it is to predict the future, we come to the question: How, and when, should we react to malnutrition that is not reversing? Intervening early offers a potential advantage, but even then treatment is beset by challenges for which immediate, easy to effect, solutions are sometime lacking. This is why the question is a crucial one. But first we consider some other treatment-related challenges for which solutions are not always immediate.

One is the grim reality of finite resources. Motivation for treatment may be strong and family support unwavering, but if funds needed to support an extended period of care for a dangerously underweight person at a respected treatment program are not available, alternative solutions may be few; this is a circumstance that can only be described as heart wrenching.

A second is no less urgent, but is more universal: that of treatment refusal or nonadherence, a challenge that becomes especially worrisome when patients are at the age of consent. Avoidance of care in AN is not so much a battle of wills as a confrontation between opposing values and perspectives—of patient, family, and practitioner. Having been a part of the struggle more than once, it is hard to capture in words the urgency of a family's desperation, our own as well, when a dangerously wasted person enters a courtroom to serve as a platform for legal debate about mental competency and the freedom to choose one's fate. The debate is less contentious when risk of death is imminent and mental deterioration is indisputable, but only because there is no legal barrier to physician intervention within a medical facility when risk is imminent; even the most zealous patient rights advocate shies away from a defense of free choice under these circumstances. It's when the sufferer appears capable of satisfying the broad legal definition of competency in spite of malnutrition that the outcome is less certain. Having sat through the proceedings many times, there isn't prose strong enough to describe the tension—parents and loved ones, therapist, too—all obliging court etiquette by gritting teeth in order to remain silent witness to legal arguments that defy reason.

This is why the question of what can be done before the opinions of attorneys and judges are engaged is so important. And shouldn't these issue come up for detailed discussion in the very first contact with a potential patient (and family)? Isn't this the logical time to prepare them for what is to come, both in general and in the many particulars of the illness, in the hope that a travesty can be averted—to discuss the nature of AN, what drives incomprehensible objections to weight gain, what in the case history might impact on long-term outcome, and the different levels of care that may be needed? This is the role of consultation, a task far different from motivational enhancement (again, see Waller's¹⁹ cautionary points). We would argue, and strongly, that for an illness as challenging and enigmatic as AN consultation is an essential prerequisite to the initiation of treatment; unfortunately, it is frequently ignored, as we will show. Our

point in citing these examples is that given the challenge they entail, it is difficult to sustain a case for teaching about AN that focuses on narrow ideas and narrow clinical training.

We naturally hope for our science to become more generative, but in the meantime it isn't as if we are completely in the dark. Managing AN is hard, but at least some of the challenges can be tempered if met by a clinical wisdom that is appropriately balanced by humility; unless we are too hostile to some ideas and too favorably disposed to others. What we hope we have made clear is that to successfully integrate pragmatic strategies supported by clinical research and experience into an overarching management that better assists patient and family, clinicians must know about the science outlined above, learn how it applies, and come to the work very well prepared. It's when the challenge is met by inexperience and skills narrowly developed that fear takes hold, and the impulse to see the illness in more categorical terms—to insist on the superiority of single, specific interventions—will be strong; we have seen this many times in supervising the casework of less experienced therapists. Maintaining poise as symptoms escalate is difficult under any circumstance, but when training and experience lack diversity and depth the straightforwardness of less abstract concepts becomes appealing. Simply put, experience too limited and clinical training too selective will bring frustration and fear should the initial presentation be severe, should progress stall, and should weight start to decline.

At the Margins: What Patients and Parents Are Not Being Told

Exactly what are parents and patients being told about the “new” paradigms for understanding AN? Well, they certainly hear more about genes and the brain; but unfortunately, without much clarification or context, the result of which has been no small measure of confusion and misunderstanding as they are to wonder in private if they were just told they/their child has “brain damage” or a “genetic defect.” It goes without saying that explanations serve a clinical purpose only if they are authoritative and complete; if not, they do harm since fragments of truth are never a good substitute for no explanation at all.

This is why it is not a good thing for patients and the public to be told that AN is a genetic disorder, but little else; not to be told: obstacles stand in the

way of identifying causative genes and determining how genes, brain physiology, and behavior interrelate; what genetic risk actually means; that stress in the environment can worsen vulnerabilities by programming anxiety proneness and negative reactions to later occurring stressful events; what connection causative genes may have to core features of the illness (anxiety and fear proneness, compulsiveness of habits, and low appetitive motivation); that genetic effects are not necessarily permanent; that predisposing genes can also have positive adaptive effects—i.e., discipline and regimentation are virtues, but in AN these virtues are “hijacked” in an effort to restore a veneer of competence as the strains of maturation become too much to live with; that behavior is shaped by environment too, because genes, biology, and the social context, which includes family life, are interdependent.

On top of these omissions, we now hear therapists insist that giving credence to environmental influences in AN is outdated, a theory refuted long ago, and that doing so amounts to unjustified scolding of parents for being malevolent and causative. This is so patently at odds with science it suggests that the BBMI and FBT paradigms' most ardent defenders know the least about it (again, we are not referring to any of the authors of the BBMI and FBT papers^{1,2}). It's another oddly inverted, contradictory logic—pitting a general premise (genes and biology play a role in risk; FBT can be effective) against others also well supported by elegant research (how natural processes inevitably link genes, brain function, and environmental adversity together; FBT is not universally effective).

Our point is that a growing, integrative science convincingly shows there is no basis whatsoever to claim that reference to environmental conditions is resurrecting outdated theories of psychogenic etiology. Rather, it argues:

1. AN frequently involves more (in psychological terms) than what is transmitted by heredity.
2. Invoking an interplay of biology and environment does not vilify families any more than it argues family disturbance is a causal prerequisite.
3. The notion of stress engendered vulnerability is not at odds with treatment models that see families as critical partners in care; it argues that broad attention must be given to sources of intrafamilial strain and the need for other forms of therapeutic dialogue to reduce it. Ignoring the potential adverse effects of environments that can increase childhood anxiety

is not helpful to families or patients given current scientific data showing these effects can be long lasting, and that changing a rearing environment's emotional tone can be beneficial.

Importantly, our experience has been that nearly every family who has heard this broadly sketched viewpoint has been entirely receptive to the concepts outlined, did not feel vilified, and welcomed knowing about their implications. So from the clinical perspective, the message that modern neuroscience underscores is: (1) that an invigorated focus on therapy skill is warranted, addressing not only the individual's belief structure, but also the social context in which they live; and (2) applying technique, whether in manual form or instructed, can be valuable, but more is needed, especially when it comes to work with difficult cases. In our view, the further needed element is not easily measured, but patients and families feel its presence and they speak of it often. It is not one single thing, but rather a set of skills with different facets: the uniquely refined ability of the therapist to sit long hours sifting patiently and thoughtfully through strains and secrets the human psyche can easily cloak; insight into what this messy tangle of conflicting tensions, puzzling emotions, and disparaging self-beliefs reveals about a patient's (and family's) misery; the ability to translate this understanding into prose eloquent enough, and delivered with the strength of conviction needed, for our patient (and family) to "feel" they best give it deeper thought; and then to steer the treatment in the direction needed and escalate its intensity should progress lag. To appreciate science is one thing, but in the clinical realm there is no substitute for well-honed skills, intuitiveness, and decisiveness when facing AN's challenge.

Other Research Relevant to Treatment Management

We know that our treatments are imperfect, but intervening soon after illness onset probably has a better chance of restoring health in persons with AN than when treatment waits until middle or late adulthood, at least judging from the poor results of adult treatment trials. This being the case, there are three crucially relevant empirical observations that inform the use of benchmarks in treatment management:

1. Because weight gain during the course of treatment is often partial, psychopathological

symptoms can persist for years. For some, the pattern remains stable, for others symptoms wax and wane in intensity through adult life.

2. Prospective research in other psychiatric conditions—e.g., schizophrenia²⁰ and major affective illnesses²¹—shows that even when very mild symptoms persist, the risk of later relapse is greatly increased. For an illustration in AN, the reader is referred to Lowe et al.²² and Strober et al.²³
3. There is literature in the pharmacotherapy and cognitive behavior therapy (CBT) of depression,²⁴ CBT for bulimia nervosa,²⁵ and FBT for AN²⁶ showing that greater symptom reduction in the very early weeks of these (time limited) therapies predicts a more robust end-of-treatment outcome. In other words, if early treatment improvement is nil the likelihood of a favorable short-term outcome is low. The implication for managing the trajectory of weight gain in the early phase of outpatient therapy couldn't be more obvious.

Benchmarks and the Context for Their Application

Having laid out a strong justification for treating AN as a complex set of deterministic processes and for adopting a comprehensive attitude toward its treatment, we come to what may seem a technically simple question: What definable benchmarks can be sensibly used to determine when a course of outpatient treatment in AN has reached its limit of benefit and should give way to a higher level of care? But we also hope the reader now better appreciates why the question is not so straightforward; because it's not easily separated from the perspective taking we have been stressing, or from the connecting links between the research cited above and the keynote of this article. There are several points:

1. Even when clinical improvement is noteworthy, the continuing presence of mild symptoms should not be taken lightly as an inherent destabilizing process remains active.
2. Lack of early weight gain in outpatient therapy may place a limit on what can be achieved over time, at least in the short-term.
3. Complex processes are in play; to assume that single interventions are by themselves satisfactory is assuming too much.

4. Therapist inexperience can be iatrogenic, its effects swinging in directions that seem entirely haphazard: extreme laxity when containment is needed, rigidly enforced control when additional time is warranted to see if the treatment course can evolve without adverse consequence. And let's not forget in this regard that a patient's avowed enthusiasm for eating is often the last refuge for a fear that a more intense treatment is coming. This is why it is important to understand that the appearance of change is not necessarily meaningful change and why failure to recognize the distinction can have serious repercussions.
5. As illness lengthens so does its pernicious effect on physical health and self efficacy; this is why allowing low body to linger is not only inexcusable, it is dangerous. In short, it behooves therapists to be aware of observations both within and outside our field that draw attention to why it is important to set boundaries that may have practical clinical relevance.

Still, it's easy to miss the context of the message: benchmarks will not have the consequence intended unless they are a seamless part of a larger ideology; specifically, the many premises that justify seeing AN as a complex illness requiring variable aspects of management and therapeutic approach. Benchmarks should not be treated as descriptive criteria; rather, they are elements in an overarching concept of care, transmitting knowledge about the illness and its management that patients and families should learn from. Even if they are systematically imposed they will be on shaky grounds if attempted by clinicians whose theoretical and clinical perspectives are narrow.

And there is yet another crucial point: that partial improvement in AN is often heralded as an important achievement for patients. This is a tricky issue because depending on the context of the illness and its course, it may be; in fact, it's a more touchy and complicated matter. Patients need to feel there is hope for change and that their therapist appreciates how hard the struggle to gain weight is; even when it seems effortless it rarely is. But empathy with what is an agonizing struggle needs to be cautiously reconciled with the danger of complacency. Many times we hear patients say—their therapists too—that the illness has been stable for some time; that this is a good thing given how bad things used to be. Perhaps, but stasis is not justification for complacency, especially when the patient is a child

or young adult. As the research just highlighted shows, symptoms that linger confer more than the eventual risk of bone disease and relapse; in time it also brings a rise in psychological inertia. We hope for a therapy that will one day prove transforming, but for now AN is frustrating because change typically comes in orders of magnitude too small to discern. It's a clinical fact that underscores one other: as the duration of a stably low weight lengthens, insidious shaping processes are evolving that continue to feed an already ruthless self judgment, ultimately forming the belief that "I can never adapt to adult life unless I am thin." And since many patients remain underweight for long periods of time while continuing in the same outpatient treatment, how can we not wonder if the eating disorder community is aware of this?

Add to this concern two more examples of how patients equivocate when they receive a recommendation that they intensify their care. They come from many hundreds of consultations (some initiated by parents, some requested by the therapist, some from patients themselves) that sought our advice on the status of a current therapy. We are referring specifically to consultations that begin with the assertion: "Things now are so much better than before." Maybe so, but sometimes the words are less a comment on progress and hope for a better future than an unskillfully cloaked fear that a higher level of care is precisely what the consultant will advise. Patients may be clumsy in how they minimize the seriousness of their illness, but this doesn't mean they lack intuition. What better way to soften the fear of being told they need something more than by ending the consultation before it begins with a pretext for why it wasn't needed in the first place.

A second obfuscation (some related statistics to follow) is seen with patients who are not currently under treatment by a mental health professional (some having discontinued this treatment) but instead are receiving supportive care from other care givers (e.g., nutritionist, spiritual counselor, pediatrician, school guidance counselor, or nurse), often for lengthy periods in spite of negligible weight gain; it is a common scenario. Why this begs many questions, but as AN is fundamentally a psychological/psychiatric illness, this form of solo, nonpsychological "counseling" is not, and should never be, a trade-off for mental health treatment, even when the patient has declined it. Many of these care givers have taken strong exception to our criticism of the arrangement, but we stand by the reproach. Apart from the incongruity (not to mention the expense) of sustaining such contact

when the patient's commitment to, or tolerance of, weight gain is nil, the idea that it is vital because it remains the only available support may be a noble one, but a contact that lacks neither reference to the facts of the patient's psychopathology, nor one able of probing the varied, complex reasons why self-examination is shunned, only fosters the illusion that care and support is actually being given; it is not. It isn't that the arrangement lacks benefit whatsoever; it's that, at best, it's a superficial one. We have known far too many patients, stable but with attenuated symptoms for many years, who ultimately paid a heavy price for the relapse that eventually came. Simply stated, it's our view—and a strong one—that nonmental health allied care should never be initiated or continued without a patient having first established a treatment that is psychological in nature. And should that relationship end referral of the patient for consultation should be arranged immediately and the allied care should cease. Patients may refuse, but the idea that this is a *de facto* justification for sustaining an arrangement that offers little further therapeutic challenge is setting a dangerous precedent. The opinion may strike some as harsh, maybe even tantamount to an unethical abandonment of the patient (it is not), but as Vandereycken and Meerman note in their excellent book on AN,²⁷ it is far better to interrupt the illusion of "treatment" when conditions for a genuine therapy are absent than to press forward when resistance to change is too great. How to approach the dilemma is considered below.

As the above examples imply, benchmarks serve the purpose of holding off the adverse complications that lack of weight gain, or weight decline, eventually bring. But they will have the greatest utility when they rest on a comprehensive attitude toward care, in the same way that their public health implications hinge on their wider dissemination. So we hope the reader will see accept their utility and apply them widely in outpatient practice.

Having said this, we are not claiming there is a general theory that lends validity to the benchmarks we will describe, or that implementing them guarantees that the hold AN has on patients will be decisively broken. Benchmarks are merely a frame of reference for approaching a particular problem, not a set of testable hypotheses. Still, they have an association with scientifically supported observations and because an immense amount of clinical experience provides further testimony, we believe

the general rationale for their application is broadly correct and that significant adverse consequences follow when outpatient care lacks a framework of which benchmarks are a part. The time points in the algorithms described were not empirically derived, but neither are they entirely arbitrarily. Again, their objective is to avoid blunders via a pragmatic frame of reference for approaching a particular problem.

Low Body Weight and Other Foundations for Benchmarks

Obviously, the prime reason for linking benchmarks to a time line for deciding when outpatient care should transition to one more intensive is the malignant effects of unremitting malnutrition; but a brief caveat concerning the relationship between low weight and outcome warrants comment. Clearly, people who stay ill naturally have poorer outcomes, but what mediates the association of low weight and long-term outcome is not yet understood. For example, we don't know if the mechanisms involved are at a cellular level; whether low weight is reinforced over time in ways psychological, interpersonal, or biological, or via interactions between all three; whether low weight is a proxy for other factors more discreetly linked to treatment failure and chronicity; or how many years of unremitting illness are required before recovery can be declared unfeasible. In our experience, it is common for patients and families to be told that a very low BMI is predictive of poor outcome; but as Steinhausen¹⁴ shows, this pronouncement is incorrect. It isn't body mass at the beginning of treatment that predicts poorer long-term outcome, it's the persistence of low body weight that does.

Some Personal Observations as Testimony to the Need of Benchmarks

They are drawn from many consultations with both adolescents and adults, of which three stand out.

First, it is striking how many patients remain in outpatient care for extended stretches of time, from months to decades, without anyone in the treatment team ever having advised referral to a higher level of care for weight restoration and a more comprehensive treatment. And we are not referring here to patients for whom such a recommendation was made but refused.

Second, many adolescents (as well as young adults who still reside with parents) enter treatment without any assistance being offered to

parents—no preparation, assistance, guidance, or discussion whatsoever, even when families were asking to participate and fully capable of doing so. The rationale typically given for separating parents from the child's therapy—to preserve the child's confidentiality—is difficult to defend since maintaining a strong individual psychotherapy and giving direction to parents need not be mutually exclusive.

Third, among the 226 consultations conducted by MS between January 2007 and December 2010 where treatment involved a multidisciplinary team of professionals, lack of cohesion in the team's leadership structure along with poorly articulated rationales given for decisions that were rendered over the course of treatment were frequent complaints. Indeed, when patients/families were asked, "Who in this team is in charge, and who do you turn to for guidance?" there were blank stares. Moreover, critically important and complex decisions (e.g., timing of calorie changes; whether it was, or was not, appropriate to participate in exercise or recreational activities; setting of the target weight and prescribed weekly weight gain; when to back off temporarily on calorie increases to allow symptoms to stabilize; whether vacations should be permitted or delayed) were often delegated to certain members of the team without input from others and rendered without any involvement of the therapist, the effect of which was that clinical factors of central importance did not inform decision making in any logically consistent or sensible way. Indeed, the irritation of parents, "We never know who is in charge," was palpable. For an illness so baffling, this state of affairs is not simply counterintuitive, it is just plain wrong. If we accept that AN is an inherently complex illness, why undertake its treatment without first laying out clear, a priori rules governing how decisions that clearly require an account of how psychological, pathological, physical issues interact will be rendered? And given that many interacting factors are at play in AN, it is imperative that all treatment decisions reflect knowledge of these deterministic trends, what they mean, and the function—in psychological terms—they serve. Naturally, certain decisions depend uniquely on the patient's physical condition and here the physician's authority is absolute. But outside of this general point, treatment decisions that arise often in AN can not, in our opinion, be rendered clearly without a single person guiding the process. And in our view, the single best resource for understanding the complexities at play is, of course, the therapist; but ironically, rarely is the

therapist involved in this way. To the contrary, in these consultations many therapists told MS they preferred to defer management decisions to others, which, as the consultation ultimately made clear, proved a tactical and conceptual error; because for patient and family, uncertainty and mistrust arose as a result. Decision making in AN is inevitably a reactive process because it shifts as symptoms wax and wane; this is why collaborative discussion is essential. But at day's end the clinician with the single greatest knowledge of the case and its context must construct the narrative and bring coherence to whatever overarching decisions are needed. Who else can realistically do this but the therapist? The idea that extending the therapist's role in this way can contaminate the therapy is not new, and we are not making light of the potential for conflict. Still, we have worked conjointly with patient and family members for decades and the occasions when therapy shut down because boundaries were muddled were few. But this was because the nature, parameters, rationale, and transference implications of the arrangement were thoroughly discussed before treatment actually began.

Last, and perhaps most disconcerting in the light of what we have discussed, rarely do either patient or family receive a formal and detailed introduction to the nature of AN, including the challenges to come if treatment is to be attempted. Among the 226 families referred to above who were currently receiving outpatient care and who sought consultation with MS about the viability of this treatment, I asked whether they had received, at any time from any treating therapist, an initial consultation that discussed in depth: (a) the defining characteristics of AN; (b) what background factors precede its onset; (c) how and why it is believed to be self-reinforcing; (d) why patients often state they can not accept life without it; (e) what is known about risk factors and how these factors collectively shape the symptoms that unfold; (f) why symptoms become so resolute, and so quickly; (g) the different course patterns and outcomes the illness takes, including the risks of chronicity and premature death; (h) what bench marks would guide decisions about when to stop one treatment in order to consider alternatives; (i) how parents/significant others can assist their child/partner in managing the symptoms and in promoting weight restoration; (j) what influences in the environment are favorable, and those that might hinder progress; (k) short- and long-term medical effects; (l) and what is known at present about the value and purpose of different therapeutic modalities. To our amazement, in only

two cases – less than one percent – was such a comprehensive pre-treatment preparation given. This is simply inexcusable.

Benchmark Algorithms

When to Consider Inpatient Care as a First-Line Intervention

No attempt has yet been made to determine if there is a specific clinical boundary that warrants inpatient versus outpatient care; neither is there a specific weight threshold (below 75% of ideal body weight is frequently cited) nor an infallible course indicator that supports the imperative of hospitalization, beyond, of course, trending signs of cardiac, hematologic, kidney, or liver function abnormality. However, based on experience with patients who enter into outpatient care first, the likelihood of failing this treatment is high when any of the following clinical features are present, and especially when they co-occur.

1. A steeply declining trajectory in body weight, especially when weight is already below 75% of expected weight for age and height.
2. Irrefutable insistence that further weight loss is needed, or justifiable, because of an overweight or “obese” appearance.
3. History of an extreme degree of regimentation or compulsiveness in behavioral routines from early in life; extreme fear of maturational challenge; a history of trauma, extreme hyperactivity (multiple hours of unrestrained activity), or comorbidity with major depression or obsessive compulsive disorder when their symptom intensity results in impairment on their own, or is compromising weight restoration.

Some will ask how these indicators are to be operationalized so they can be applied systematically; but this is where the sort of clinical judgment that experience hones is important. Similarly, it is intuitive that when more than one of the listed features is present concurrently the impairment this results in is too difficult to interrupt in the outpatient setting. For these reasons the rationale for moving sooner rather than later to inpatient care is sound. We are not saying it is absolutely impossible for outpatient care to succeed when very low body weight (or these other clinical features) is present—we know of cases—but it is rare.

When to End Outpatient Care

This is by far the most crucial scenario for benchmark application because it is the more common one. Our recommended algorithms for escalating the level of care are as follows; they are broken down by the presenting circumstance.

Scenario 1: When Outpatient Care Has Been Attempted for an Underweight Child/Adolescent/Young Adult, Regardless of Previous Treatment History

Beginning treatment de novo with an underweight child or teen is the paradigmatic illustration of benchmarking the level of care needed to minimize risk of a deeper and more entrenched psychopathology. In fact, we are increasingly being asked by parents, “When do we know what we’re doing is not working?” It is a complicated question for several reasons: because the treatment of AN requires time, so changing course too soon isn’t always a good thing; there is no uniformity in how “low weight” is defined; and switching to another therapist, one more skilled, can sometimes reverse the course dramatically. But knowing that a spontaneous remission is extremely rare, that bone demineralization accrues quickly, and that as symptoms intensify self-esteem and adaptive competency suffer, we urge that outpatient therapy be stepped up to hospital care when any of the following circumstances apply:

1. If weight declines steadily over the first 3 weeks of treatment (or following consultation if no treatment was initiated). In our experience, this trajectory becomes difficult to interrupt thereafter.
2. Weight is initially stable, but there is a negligible average weight gain (or a waxing and waning pattern of increases and decreases) by the end of month two of treatment (or following an initial consultation). In our experience, a steady, uninterrupted increase in weight back to normal body mass becomes increasingly less likely after this point.
3. There is initial weight gain, but the slope of the increase levels off prior to the patient achieving full weight restoration, and this flattened pattern remains unaltered for at least 6 continuous weeks.

If, per chance, there is a change of therapist, the algorithms recycle immediately.

Scenario 2: Initiating Outpatient Treatment for the First Time in an Older Adult at Low Body Weight

Here, we are presuming the patient has been ill for at least 5 years. It might be argued that because illness duration is longer the urgency of more intensive treatment is proportionately greater; but so is the patient's language of resistance, and since time will be needed before we have a good sense of the precise issues involved in the history of such a patient a more lenient algorithm is prudent. So assuming the patient is judged by a physician's examination to be medically stable, the algorithm needs to allow greater time for the patient's struggle to play out. Accordingly, we recommend the transition to a higher level of care when:

1. Weight is declining steadily in the first three weeks after commencing treatment.
2. Weight is initially stable, but the patient is unable to initiate, and then sustain, a steady increase in weight by the end of month three of treatment; in our experience, uninterrupted weight gain after this point is increasingly unlikely with such a patient.
3. Weight increases initially, but the slope of this increase then levels off and remains so for 3 continuous months.

Scenario 3: Initiating Outpatient Treatment with a Young Adult Who Has Had a Prior Failed Treatment

Here we recommend the same criteria as in Scenario 1.

It is also legitimate to ask whether these should be explicit rules; but it should be remembered that the time points derive from experience. Beyond the questions that naturally arise about algorithms, what these address in a fundamental sense, and we think reasonably given the material reviewed, is the danger of passivity in decision making; because over time, the pull of irrational habits, conflicted motivation, and the mind's attitude in AN grow stronger and as they do the resistance to change becomes more formidable.

What to Do When Patients/Families Reject the Recommendation of a Higher Level of Care?

As Vandereycken and Meerman note,²⁷ therapists have as much "right" to discontinue treatment as patients do; except here, the rationale is stronger as it is informed by an important clinical wisdom. Patients withdraw from treatment due to fear of what they are being asked to confront and the emo-

tional discomfort that results (we are not speaking here of ending a treatment that is poorly executed or one attempted by an unskilled therapist). But for therapists, the decision comes after a lack of meaningful progress over an extended period, or upon a patient's refusal to step up their level of care when it is deemed acutely necessary. There is no question these are difficult, sometimes painful, decisions; we consider them reluctantly because of the original commitment we made to the patient's well-being and our abiding hope that one day our patient will enjoy a future less encumbered by withering self-deprivation. But returning to an earlier caution, continuing a level of care that is unprofitable and not likely to have benefit in the foreseeable future is not treatment, and to carry on as if it carries significant risk. For this reason, discontinuing treatment may well be the only action persuasive enough to convey the urgency of what the therapist feels, and what the patient needs, regardless of what they ultimately decide to do.

But it is not a finite decision, and this is an important point. Instead, we recommend for the time being only a temporary interruption of care; specifically, if an impasse has been reached we recommend reconvening in 1 month's time for further assessment of the patient's circumstances. The rationale of the interruption is explained by incorporating the premises discussed throughout this article; hope is expressed that the patient (and/or family) will soon reconsider the refusal of a higher level of care; the risks of further refusal are discussed straightforwardly; interim follow-up with a physician is strongly recommended; possible underlying reasons for the patient's reluctance to contemplate treatment of greater intensity are outlined, emphasizing the psychopathological issues involved, including as much detail in the explanation as possible; and finally, the possibility that the treatment may end at this time is acknowledged. Should the patient return, laboratory results and symptom intensities are reviewed and signs indicating a worsening of the patient's physical or clinical state are discussed in detail, along with the patient's/family's current worries, or, perhaps, their professed lack of concern (but rarely is a blasé attitude seen). If the original recommendation is rebuffed at the 1-month follow-up, a second follow-up in 3 months is offered and if the patient agrees the therapist again expresses hope that the return visit will take place. Yet a third, but final, 3-month follow-up is scheduled if the refusal persists.

In our experience, the outcomes are entirely unpredictable: some patients reconsider quickly, accepting the higher level of care because their

condition has worsened, or the intensity of their misery has greatly increased; some continue to refuse, but ultimately reconsider at a later time; others seek treatment elsewhere, sometimes showing noteworthy improvement; and still others never return and we never hear from them again. Finally, as discussed elsewhere,²⁸ for some adult patients the only recourse is a supportive management where the interventions and objectives are carefully measured.

Concluding Words and a Postscript on Consultation

We have presented an argument for understanding the mystery and danger of AN in the broadest context possible. In doing so, we have highlighted lessons being taught by numerous credible studies about biology, development, and life experience – lessons germane to psychopathology and to its treatment. We made the point that genes and biology are the fundamentally important starting point for understanding, because compulsiveness of habit, anxiety, low reward seeking, and behavioral regimentation are heritable phenotypes that ‘power’ the illness and sustain its self-rewarding properties. But equally important to this understanding is that these traits can be ‘opportunisticly exploited’ by the psyche of AN to sustain its adaptive, self-rewarding effects. Whether we think the transformative process is unconscious or volitional is beside the point since the dividing line is too faintly drawn to determine where one ends and the other begins. Still, even though vulnerability begins with genes and biology, to assume there is neither intentionality nor volition involved at any level of the illness is a fallacy; biology and willfulness are not mutually exclusive processes. So what isn’t beside the point is that genes and biology should be viewed in functional terms, that the clinical implications of this viewpoint are many, and that molecular neuroscience reminds us that life experiences play a role in illness because even irrational mental states adapt to social pressures in a causal chain that shapes the course of development over many years. This is why throughout the article we stressed that a narrow conceptual vision of AN can never suffice as clinical theory because making pragmatic interventions the centerpiece of therapy is not only too deliberate, it is insufficiently sensitive to the contextual factors that can either promote or arrest symptom progression.

So coming to the end, we return to the touchy question of where things go wrong and why. Also,

why is it that in preparing to treat a psychiatric illness we unhesitatingly agree is more complex and potentially threatening to health than any other, therapists rarely begin with a detailed, comprehensive, authoritative empirical and clinical account that establishes: what, exactly, is being treated; how the illness evolves and what processes underlie its self-perpetuating character; why the treatment is likely to be challenging and in what ways; how long a period of care may be needed, the elements of that care, and what risks result if the treatment should end prematurely; what benchmarks need to be followed to decide when more intense intervention is essential, and why; what behavioral, psychological, and life style changes signal that the end of treatment is at hand; and what personal, parent, family, and social strains may be important to consider, and to address therapeutically? Why does this rarely occur? We believe the reasons are several:

1. insufficient clinical experience; lack of breadth in academic and clinical training; and little exposure to other areas of psychopathology relevant to eating disorders;
2. a commitment to doctrinaire ideas and quickness to write off alternative possibilities for care;
3. lack of training and experience in combined clinical and academic settings that offer comprehensive, multidisciplinary, and higher levels of care, and a misguided disdain for hospital-based treatment.

How to put into words unfamiliar to either patient or family the many reasons why weight gain has become a fear so paralyzing it is resisted at all costs, and how to give far ranging explanations of what gives rise to the illness and the absurdly wild beliefs that become attached to its symptoms, are questions that underscore the essential wisdom of making far ranging clinical skills and diverse theoretical knowledge our foundation. Not only does it allow for answers to the questions patients and families surely have, it also articulates the unspoken resistances to change and the withering emotional highs and lows that may soon erupt for which everyone, therapist too, must prepare. This, in our experience, is the dialogue that has proved the most robustly effective means of helping parents (and other loved) separate illness from the person who bears the affliction; any other preparation is sterile and incomplete.

Who doesn’t wish for a treatment that can alleviate suffering quickly? But to think there is a decisive way of accomplishing it is risky and naïve. Teaching parents skills to assist with weight gain is a good

thing; we should do it. What is not a good thing is failing to recognize it must coincide with a seasoned ability to infer when the conditions for its success may not be present and how other interventions can assist. We know from colleagues, many interactions with therapists, and quite extensive experience with patients and families, that in the torrent of excitement that greeted FBT, it, and it alone, has assumed center stage in the care being delivered by many practitioners and many treatment centers whose knowledge of the illness is marginal. We understand this; when treatment service options are limited, a pragmatic solution is needed, and quickly. Still, it shouldn't overshadow the need for platforms of teaching and skill building that are more comprehensive. And let us not forget another basic truth about people who struggle and the loved ones who understand little of how to help. Ultimately, it is the therapist's knowledge of the mysteries involved and the wisdom they display in rendering their clinical judgments that instills hope and builds faith that what is being done follows a rationale that must be honored. Without this, the bond that tethers our patient to our treatment is a fragile one.

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