Available online at www.sciencedirect.com
SCIENCE DIRECT
Neuroscience and Biobehavioral Reviews 29 (2005) 159–168
NEUROSCIENCE AND BIOBEHAVIORAL REVIEWS
www.elsevier.com/locate/neubiorev

Review

The concept of alternative strategies and its relevance to psychiatry and clinical psychology

Alfonso Troisi*

Department of Neurosciences, University of Tor Vergata, via Guattani 14, 00161 Rome, lItaly

Abstract

The intent of this article is to introduce the evolutionary concept of alternative strategies into the fields of psychiatry and clinical psychology. In behavioral ecology, the term alternative strategies refers to the presence of two or more discrete behavioral variants among adults of one sex and one population when those variants serve the same functional end. Often discrete behavioral variants are associated with specific morphological, physiological, and life-history characters. The concept of alternative strategies has been applied to human behavior to explain the origin of some behavioral syndromes that are currently classified as mental disorders or emotional dysfunctions. Antisocial personality could represent a high-risk strategy of social defection associated with resource acquisition and reproduction. Insecure attachment could represent an evolved psychological mechanism that used the quality of parental care received during childhood as a cue for optimizing adult reproductive strategies. Since a major contribution of evolutionary theory is the insight that individual differences are core biological features of any animal species, including *Homo sapiens*, the application of the concept of alternative strategies to psychiatry and clinical psychology can be a powerful antidote to the growing tendency to medicalize human diversity.

Keywords: Alternative strategies; Individual differences; Darwinian psychiatry; Antisocial personality; Insecure attachment; Medicalization

Contents

* Fax: +39 6 4423 2998.
E-mail address: alfonso.troisi@uniroma2.it.

0149-7634/$ - see front matter © 2004 Elsevier Ltd. All rights reserved.
doi:10.1016/j.neubiorev.2004.06.012

That all men are equal is a proposition which, in ordinary times, no sane individual has ever given his assent

Aldous Huxley

1. Introduction

The history of medicine shows that decisions about whether a condition or behavior is best conceived of as a disease or as indicative of health are not made in a social vacuum. In the past, the importation of biases and prejudices into medical classifications of disease has had disastrous consequences for various social groups. For this reason, the study of individual differences requires vigilant and critical conceptual attention. The present and the future of medical research are not immune to the risk of erroneous medicalization. We are rapidly approaching a postgenomic era in which we will know the entire human genome sequence. However, as noted by Plomin [1]: 'There is no single human genome sequence—we each have a unique genome.' (p. 910). Human genome sequencing will reveal thousands of genetic variations among individuals and this will have a tremendous impact on those clinical sciences that are concerned with the study of individual differences. Given the high societal expectations of human molecular genetics, any trait, condition, or behavior associated with a genetic variation is in danger of being construed as a manifestation of disease: 'As more and more genetic variations among individuals are discovered, there will be a rush to label many of these variations as disease-associated.' [2, p. 807].

Evolutionary theory, with its appreciation for biological diversity, can contribute to strengthen the theoretical framework for the study of individual differences in clinical sciences. The intent of this article is to introduce the evolutionary concept of alternative strategies into the fields of psychiatry and clinical psychology. The central body of the article will discuss how alternative strategies are described and classified in behavioral ecology, will give some examples of alternative strategies in non-human primates, and will present data suggesting that two behavioral syndromes that are currently classified as mental disorders or emotional dysfunctions (i.e. antisocial personality and insecure attachment) could be alternative strategies. The paper begins with a brief discussion of how medicine and evolutionary biology differ in their conceptualizations of individual variability and concludes with some reflections about the diagnostic and therapeutic implications of the re-classification of clinical syndromes as alternative strategies.

2. Individual differences in medicine and evolutionary biology

In ordinary medical usage 'normal' has two meanings: 'that which is common' and 'that which is compatible with health'. The fact that the two meanings are often confused reflects the tendency to equate statistical normality with biological normality. The origin of this way of thinking dates back to the Platonic and Aristotelian notion of the 'ideal' to which actual organisms are imperfect approximations [3]. According to typological thinking, homogeneity in a population is the natural state and variation is the result of some sort of interference. A type is postulated for all organisms of a given kind, and deviation from that type requires special explanation. Sir Henry Cohen's [4] definition of disease as 'a quantitative deviation from the normal' (in which by normal he meant the statistical norm) exemplifies the statistical approach to individual differences. Most biological traits are assumed to fall into a normal distribution, with most of the cases in the middle and a few at the extremes. These extremes, which constitute only a small percentage of the total population, are arbitrarily lopped off and labeled 'abnormal' or 'pathological' and the far larger percentage clustering around the middle is arbitrarily called 'normal'. For example, what most clinicians do when they receive a laboratory report is to look up the normal range for the tests in question, where the normal range is traditionally calculated in such a way that it includes 95% of the results found in a random and unbiased sample selected from the general population.

Of course, in medicine, statistical abnormality is not the only criterion of morbidity. Other independent criteria, such impaired function and presence of organic lesion, are commonly applied before deciding that a statistically deviant feature is a manifestation of disease. If an epidemiologist found that 60% of the persons in a society are afflicted with diabetes, no one would be likely to assert that these persons are healthy just because most of them have it. In addition, medicine acknowledges the existence of deviations from the norm which are neutral, like great height, or positively beneficial, like superior intelligence. However, the relevant point here is that medicine subscribes to the pre-Darwinian way of thought that attributes no adaptive significance to individual differences and that

ignores the possibility that within-species variation is actively maintained by natural selection.

Darwinian theory revolutionized the approach to individual differences. Rather than regarding the variation among members of the same species as an annoying distraction, evolutionary biologists are aware that (1) variation in natural populations is widespread at every level, from gross morphology to DNA sequences, and (2) variation is the fuel for evolution. Natural selection occurs only when differences in some phenotypic characteristic result in consistent differences in rates of survival and reproduction. Selection can operate in different ways on the new variation that continuously originates from recombination and mutation [5]. If intermediate phenotypes are most fit, selection tends to eliminate those phenotypes that deviate in either direction from an optimal average value. This type of selection is called stabilizing selection. The resulting variation is continuous and the extremes constitute only a small percentage of the total population with the far larger percentage clustering around the middle. Thus, if a trait is subject to stabilizing selection, it is not surprising that health (to the extent that it depends on the optimal functioning of that specific trait) coincides with the statistical norm. For this reason, in many instances, statistical and clinical observations are positively and strongly correlated. For example, in laboratory medicine, the statistical mode is often identical with optimal function, as in the count of blood cells or in various chemical titers of plasma substances. However, selection can also produce variation instead of reducing it. If two or more phenotypes have high fitness, but intermediates between them have low fitness, selection is diversifying; that is it acts in favor of two or more modal phenotypes and against those intermediate between them. Diversifying (or disruptive) selection produces discontinuous variation which divides the individuals of a population into two or more sharply distinct forms. Not only may this occur at the anatomical and physiological levels, but also at the behavioral level. Therefore, a variant and statistically deviant behavioral profile may be the product of natural selection and thus it may be an adaptation. This lead us to discuss the concept of alternative strategies.

3. The concept of alternative strategies

In behavioral ecology, the term alternative strategies refers to the presence of two or more discrete behavioral variants among adults of one sex and one population when those variants serve the same functional end, such as more than one way of foraging, or attracting mates, or nesting [6]. Often discrete behavioral variants are associated with specific morphological, physiological, and life-history characters. Some authors prefer to use the word ‘strategy’ to give a complete specification of what an individual will do when competing for a resource, and the word ‘tactic’ for the behavioral components of a strategy. In this article, following Krebs and Davies [7], I prefer to use the word strategy somewhat loosely to describe any alternative behavior pattern used by an individual to compete for a resource.

Traditionally, the behavioral literature has separated alternative phenotypes into those due to genetic differences and those due to environmental or individual cues [8]. Alternative strategies due to genetic differences reflect genetic polymorphisms and are irreversible (i.e. fixed over individual’s lifetime). In contrast, alternative strategies not due to genetic differences imply decision-making processes within the individual and may be reversible. There are two types of decision-making processes. When the frequencies of the two strategies are set by a random decision rule, this is a ‘mixed strategy’ [9]. If the decision rule about which pattern to follow is facultative and made on the basis of environmental or individual conditional cues, then this is referred to as a ‘conditional strategy’.

The evolution of irreversible alternative strategies is likely when the environmental conditions that favor each of the two phenotypes are unpredictable and change frequently. An example of an irreversible alternative strategy reflecting a genetic polymorphism is the reproductive behavior of male coho salmons (*Oncorhynchus kisutch*). In this species, some males have exaggerated snouts and enlarged teeth and they fight for access to the spawning females. Other males lack secondary sexual characters and are relatively cryptic on the breeding grounds. Instead of fighting, they attempt to gain access to females by sneaking. Gross [10] has demonstrated that the two strategies are maintained by frequency dependent selection such that the average reproductive success of each is the same. When two alternative strategies are affected by frequency dependent selection, there exists an equilibrium frequency of the two patterns. As long as the population remains at equilibrium, the two patterns will be, on average, equally successful. Above or below the equilibrium, one pattern does better, thus returning the population to the equilibrium frequency.

Also mixed strategies are the product of frequency dependent selection and have equal success. However, mixed strategies reflect decision-making processes within the individual rather than genetic polymorphisms. These strategies are reversible and individuals can change flexibly from one pattern to another and back again. In digger wasps (*Sphex ichneumoneus*), females have two ways of obtaining a burrow to lay their eggs: they either dig one for themselves or they enter an already dug burrow [11]. Digging a burrow takes much time and energy but employing the alternative strategy implies the risk of entering an already occupied burrow. The fitness of each behavioral phenotype increases as its frequency within the population decreases, that is, which nesting strategy is optimal depends on what the other females in the population are doing. If, for example, all the females were employing the entering strategy, most would

meet another female using the same burrow, there would be lots of fights, and it would certainly pay for a female to go off and dig her own burrow.

Alternative strategies can be conditional on environmental circumstances or individual characteristics such as age, body size, strength or behavioral ability. Alternative conditional strategies are not equally successful and, therefore, have also been called 'making-the-best-of-a-bad-job' strategies [7]. In North American bullfrogs (*Rana catesbiana*), the largest males win the best territories and attract females by croaking. Small, young males are not strong enough to defend a territory and so they behave as satellites, sitting silently near a calling male and attempting to intercept and mate with any females the calling male attracts. The satellite males are not very successful but they make the best of a bad job by parasitizing the largest calling males because they do not have a better chance of getting a female when they are small. This conditional behavioral phenotype is reversible and young males change their strategies from sneaking to fighting as they get older and grow larger and stronger.

Although the classification of alternative strategies reported above is fine in theory, we cannot ignore two problems deriving from its rigid application. First, as noted by Brockmann [6], it is often difficult to distinguish alternative strategies between those that are genetic and those that are environmental. Environmental and individual conditions affect any type of alternative strategies, and genetic and mixed strategies have often turned out to be a complex set of condition-dependent strategies [8]. Second, over the past 20 years, research has demonstrated a possible role for frequency dependence in all alternative strategies, including those that are condition dependent or based on environmental cues. Thus, although a tripartite classification of alternative strategies can be useful at the descriptive level, we should be aware that (1) all types of alternative strategies (genetic, mixed, and conditional) are influenced by genes, environment, and gene-environment interactions; and that (2) frequency dependence is an intrinsic part of all alternative phenotypes.

3.1. Alternative strategies in non-human primates

One could question the relevance of alternative strategies to human behavior if all the examples from the behavioral literature were limited to insects, fishes or amphibians. Fortunately, in the last few years, the primate literature has documented the existence of alternative strategies not only in Old Word monkey species but also among the great apes.

In mandrills, there are two morphological and behavioral variants of adult males that differ in terms of secondary sexual adornments and reproductive strategies [12]. 'Fatted' males have highly developed sex skin coloration, large testes, high plasma testosterone levels, and fat rumps, whereas 'non-fatted' males have paler sex skin, smaller testes, lower plasma testosterone, and slimmer rumps. While 'fatted' males mate-guard fertile females, less developed males remain in the periphery of the group and mate sneakily with females. Similar intermale differences have been observed in orangutans [13]. In the presence of many dominant males, adolescent male orangutans undergo a developmental arrest: they become fertile but do not develop fully adult secondary sexual features, such as cheek flanges, laryngeal sac, beard and mustaches, large body size, and a musky odor. Developmental arrest is associated with a distinct hormonal profile [14]. Arrested males lack levels of luteinizing hormone (LH), testosterone, and dihydrotestosterone (DHT) necessary for development of secondary sexual traits. However, they have sufficient testicular steroids, LH, and follicle stimulating hormone (FSH) to fully develop primary sexual function and fertility. Like in mandrills, the two morphological variants of male orangutans use different mating strategies [15]. Developed males are frequently involved in male–male aggression, are attractive to females and typically consort with them, and may sire many offspring over a relatively short period of time. In contrast, being inconspicuous and less attractive to females, arrested males adopt a low-cost, low-benefit reproductive strategy based on sneaky matings and forced copulations.

In the short period, the reproductive success of 'non-fatted' male mandrills and arrested male orangutans is lower than that of fully developed males. However, there are advantages associated with the use of the 'sneak and rape' mating strategy. While the 'combat and consort' strategy imposes costs on dominant males in terms of metabolic energy and exposure to intermale aggression, the suppression of secondary sexual traits allows subordinate males to minimize aggression and injury from dominant, fully mature males, while still being able to sire. Both the strategies are maintained by natural selection because the disadvantages associated with each strategy are balanced by advantages in a different context. In both mandrills and orangutans, subordinate males can rapidly switch over to the 'combat and consort' strategy if the density of dominant males decreases. Arrested male orangutans develop into flanged males if a more favorable reproductive situation occurs, and subordinate male mandrills develop secondary sexual traits when they become dominant. Such a flexibility indicates that the 'sneak and rape' strategy is an adaptive alternative strategy based on a continuous assessment of reproductive opportunity and risk of intermale aggression.

3.2. Alternative strategies and abnormal physiology

How would an endocrinologist or a clinical psychiatrist evaluate the health status of non-fatted male mandrills and arrested male orangutans? In all probability, they would agree that these individuals are sick. In reaching such a conclusion, they would apply the criterion that the medical model considers as the most unequivocal for deciding if

a condition qualifies as a genuine disease: the presence of organic lesion. According to the medical model, what is needed to distinguish normal variation from pathology is some kind of organic marker, such as tissue alterations or biochemical abnormality. Non-fatted male mandrills and arrested male orangutans meet this criterion because they not only display deviant behavioral patterns but also have abnormal hormonal profiles.

Because of its apparent objectivity and strict relationship with causal explanations of the patient's symptoms, the view that a disease entity is an altered part of the body came to dominate medical thinking since the pioneering work of Giovanni Battista Morgagni, an eighteenth-century Italian physician who introduced the method of clinical–pathological correlation which consists in tracking down in the organs and tissues the clinical manifestations of disease. During the last 50 years the development of new diagnostic techniques of investigation has expanded the concept of lesion to include physiological, biochemical, and molecular abnormalities, without relinquishing the basic assumption that disease necessarily involves a demonstrable physical abnormality of some sort.

In fact, the objectivity of the criterion of lesion is only apparent. Defining lesion is as difficult as defining disease, and the risk of circular reasoning is always present. For example, the 15th edition of the Encyclopedia Britannica defines lesion as 'a structural or biochemical change in an organ or tissue produced by disease processes'. In logical terms, this means that the definition of lesion is dependent on the definition of disease, not vice versa. Unlike the medical model, evolutionary reasoning suggests that the decision whether a condition is a disease cannot be based on the demonstration of organic pathology because there is no definitive criterion for sorting out morbid physiological alterations from adaptive interindividual variability, unless we judge a condition in terms of functional consequences [16]. The fact that non-fatted male mandrills and arrested male orangutans have abnormal (in the statistical sense) hormonal profiles becomes understandable considering that alternative strategies are often associated with specific morphological and physiological changes. The suppression of secondary sexual traits to minimize aggression and injury from dominant males requires corresponding alterations of the hormonal profile. Thus, the low levels of LH, testosterone, and DHT in these males are an evolved adaptive mechanism, not a lesion.

The above discussion is important because the recent explosion of neuroscience research has revitalized the old idea that the discovery of alterations in brain physiology or anatomy will ultimately solve the problem of distinguishing between psychiatric disorders and the variety of unpleasant or undesirable feelings and behaviors that characterize the human condition. The lesson from the study of alternative mating tactics in non-human primates warns us against the risks of such an approach.

4. Clinical syndromes as alternative strategies

How do these findings relate to human behavior? There is of course nothing new either inside or outside of psychiatry about the fact that people differ. However, what may be new is that the evolutionary origin of these differences is far more important than is usually appreciated, and particularly so in considering the possibility that some clinical syndromes may in fact reflect alternative strategies. Antisocial personality and insecure attachment are two possible candidates for such an interpretation.

4.1. Antisocial personality

As defined in the DSM-IV [17], antisocial personality disorder is a pervasive pattern of disregards for the rights of others associated with distinctive emotional and behavioral features. Individuals with this personality disorder (also known as psychopaths or sociopaths) are frequently deceitful and manipulative in order to gain personal profit. Even though they may display a glib, superficial charm, these subjects lack empathy and tend to be callous, cynical, and contemptuous of the feelings, rights, and suffering of others. Finally, they display a reckless disregard for their personal safety and are free of symptoms of anxiety. Although antisocial personality is classified as a mental disorder in current nosographic systems, some investigators have conceptualized its variant known as 'primary psychopathy' [18] as an alternative strategy because a number of psychological and behavioral traits typical of primary psychopaths could well have contributed to reproductive and survival success in the ancestral environment [19–21]. Reasonable assumptions are that approximately 50% of the persons who could meet the criteria for this disorder go through life undiagnosed and undetected, and they are successful by evolutionary criteria [21]. For example, in a sample of male offenders, Lalumiere et al. [22] found a positive correlation between the Psychopathic Checklist-Revised (PCL-R) score, physical attractiveness and the number of children. In addition, psychopathic offenders scored lower than non-psychopathic offenders on fluctuating asymmetry, a reliable measure of past developmental perturbations.

During our recent evolutionary history, it seems likely that there has been strong selection for cooperation based on reciprocity. To deal with the adaptive challenges posed by the ancestral environment, our ancestors developed a strong propensity to share resources (e.g. food, tools, and knowledge) and to help the sick, the wounded, and the very young. The evolution of cooperation and reciprocal altruism has been possible because of the co-occurrence of a variety of species-typical characteristics including long lifespan, low dispersal rate, life in small, mutually dependent and stable social groups, and a long period of parental care leading to extensive contacts with close relatives over many years [23].

Selection for cooperation and altruism is expected to generate cheating. In a society made up primarily of

cooperators, genes for cheaters can enter the population and remain, provided that, under specific circumstances, the benefits of cheating outweigh the costs. As long as selective pressures for cooperative strategies coexist with counter-pressures for cheating, a mixture of phenotypes will result, such that some sort of statistical equilibrium will be approached. Antisocial personality should thus be expected to be maintained as a low-level, frequency-dependent alternative strategy [20]. Data concerning both the heritability of antisocial personality [24] and its prevalence in the general population [25] are consistent with the hypothesis of frequency-dependent selection. In an evolutionary context, primary psychopathy might thus represent a high-risk strategy of social defection associated with resource acquisition and reproduction.

Interestingly, many different aspects of the psychology and physiology of individuals with antisocial personality that are currently viewed as defective or abnormal traits can be re-interpreted as complex adaptations that have evolved to allow the successful use of a cheating strategy. Sociopaths are characterized by a lack of the social emotions (such as love, shame, guilt, empathy, and remorse) in the absence of any other cognitive or mental deficit. Persons with this disorder are capable of accurately assessing the costs and benefits of short-term social interactions, accurately reading others' behavior rules, utilizing self-monitoring information to alter their strategies, and successfully disguising their intentions. Therefore, their limited capacity to experience social emotions is puzzling considering that such a capacity is present not only in individuals without psychiatric disorders but also in patients suffering from a variety of severe psychopathologies. The lack of social emotions in individuals with antisocial personality becomes understandable considering the evolutionary function of these emotions. Social emotions play a central role as 'commitment devices' [26]. Feeling certain emotions commits an individual to act in certain ways. We know from the work of social psychologists that there is a strict relationship between positive emotions like sympathy and love and altruistic behavior: we are more altruistic toward those we like. On the other hand, negative social emotions experienced before (anxiety) or after (guilt and remorse) defection discourage the use of exploitative and manipulative strategies in social interactions. Not feeling these emotions, individuals with antisocial personality may act where others are constrained from doing so: 'Without love to commit them to cooperation, anxiety to prevent defection, or guilt to inspire repentance, they will remain free to continually play for the short-term benefit.' [20, p. 536].

Also the physiological and neurobiological profiles of individuals with antisocial personality appear to be abnormal in the statistical sense. Compared to control subjects, they are more likely to show orienting responses to novel stimuli, to show less physiological arousal in response to threats of pain or punishment and more tolerance of actual pain or punishment, and to have a lower resting heart rate [27–29]. These distinctive physiological responses reflect reduced noradrenergic functioning and a fearless, stimulation-seeking temperament. A recent study employing magnetic resonance imaging (MRI) has suggested that the low autonomic stress reactivity and the emotional deficits of psychopathic antisocial individuals may be caused by abnormalities of the corpus callosum [30]. Based on the medical model of disease with its emphasis on the concept of lesion, one is tempted to conclude that the organic correlates of antisocial personality definitely demonstrate its morbid nature. However, these statistically deviant biological features could simply represent a complex set of adaptations evolved to favor the use of a high-risk strategy of social defection. If the lack of social emotions prevents experiencing feelings which naturally inhibit the acting out of exploitative and aggressive impulses, so the lack of fear in response to aversive events and the incapacity to form conditioned associations between antisocial behavior and the consequent punishment allow primary sociopaths to persist in using a cheating strategy oriented toward short-term benefits.

4.2. Insecure attachment

The attachment theory is one of the most influential model proposed to explain the relationship between early experience and adult personality and social behavior. According to attachment theory, infants develop expectations about their caregivers' availability and responsiveness based on the quality of parental care they receive. These expectations then serve as the basis for the development of mental representations of the self and of the other ('internal working models' in the terminology of attachment theory) that influence later psychosocial functioning. Infants with emotionally available caregivers develop a model of the self as loved and valued, and a model of the other as loving. When infants instead have experiences that lead them to expect caregivers to be rejecting or undependable, they develop a model of the self as unloved or rejected, and a model of the other as unloving or rejecting. As adults, these persons do not expect that significant others will be available when needed, and they develop insecure strategies for coping with their distress.

Based on observations of infant behavior during reunion with the mother after a brief experimental separation, Ainsworth et al. [31] documented three basic patterns of attachment: secure, ambivalent, and avoidant. Upon reunion, securely attached children use their mothers to regulate and attenuate their distress, often resuming other activities (e.g. play and exploration) quickly after calming down. In contrast, avoidant children retract from their mothers upon reunion, choosing to control and dissipate their negative affect in a self-reliant manner. Ambivalently attached children make inconsistent and conflicted attempts to derive comfort and support from their mothers, often

intermixing clinginess with overt anger. A number of studies employing self-report measures have shown that the major patterns of attachment organization described in children (secure, ambivalent, and avoidant) have corresponding analogs among adults.

The traditional perspective of clinical psychology views secure attachment as the healthy pattern and the insecure patterns as reflecting some kind of emotional dysfunction if not pathology. In contrast with such a normative approach, Belsky and co-workers [32,33] have advanced the hypothesis that, in the ancestral environment, all three patterns of attachment were equally adaptive in terms of promoting reproductive fitness in the ecological niches that gave rise to them. According to this hypothesis, attachment patterns represent evolved psychological mechanisms that used the quality of parental care received during childhood as a cue for optimizing adult reproductive strategies.

The Belsky et al. model builds on the life history theory. In many long-lived species, including humans, when to reproduce and how many resources to invest in each offspring are two basic 'decisions' that have a major impact on reproductive fitness. The 'choice' between a parenting effort strategy (characterized by delayed maturation, discriminative sexual behavior, low fertility, and high parental investment) and a mating effort strategy (early maturation, promiscuous sexual behavior, high fertility, and low parental investment) depends on prevailing ecological conditions [34]. Under adverse environmental conditions where the flow of resources is chronically low or unpredictable, it can be adaptive to 'choose' a quantity-oriented strategy, by channeling more effort toward rapid physical development, earlier mating, and multiple, short-term relationships. Delayed maturation and reproduction under conditions of environmental risk and uncertainty may cost individuals dearly, since they are more likely to die before reproducing. In contrast, environments in which resources are plentiful and relationship ties are more reciprocal and enduring should foster a quality-oriented strategy consisting in slower physical development, later mating, and long-term pair bonds structured around greater parental investment. In such environments, reproductive fitness should be enhanced by deferring reproduction until prospective parents have acquired the skills and resources needed to maximize the quality of each offspring.

According to the life history model, in the ancestral environment, the principal evolutionary function of attachment patterns was to translate information about the availability and predictability of resources (including parental care and attention) into behavioral strategies for promoting reproductive fitness. Whether reproductive fitness is actually enhanced in the modern environment is less central to the validity of the evolutionary hypothesis than the predictions that each attachment pattern should be associated with different sexual and parental behaviors and that such differences should remain operative today.

Consistent with these predictions, a number of studies have found that secure individuals are likely to engage in enduring romantic relationships and have both the desire and capacity to invest heavily in parental care. In contrast, avoidant individuals frequently report involvement in one-night stands and sex outside established relationships [35], and they tend to be cold, remote, and unsupportive parents [36]. Even more convincing is the evidence linking early experience, patterns of attachment, somatic growth and reproductive development. If less responsive parental care received during childhood should foster the development of a mating effort strategy, then individuals with an insecure pattern of attachment should show accelerated somatic growth and early pubertal maturation. In line with these predictions, studies of pubertal timing in girls have found that stressful family environments are associated with early menarche whereas an affectionate and responsive parental style is associated with delayed puberty [37]. Raine et al. [27] have found that large body size at age 3 years but not at 11 years was related to fearlessness and aggression at age 11 years, indicating a critical period for the development of an association between rapid physical growth and antisocial behavior (a behavior pattern often associated with avoidant attachment [38]).

Although the above reported data are provocative and potentially important, the evolutionary model of attachment patterns as alternative life history strategies has still limited empirical validity. For instance, the evolutionary significance of ambivalent attachment is not clear. Belsky [33] has proposed that ambivalence may reflect an evolved 'helper-at-the-nest' reproductive strategy, in which parenting effort is directed toward kin. According this hypothesis, because of their extreme emotional dependence, ambivalent individuals (especially female firstborns) had the tendency to remain in the native group and to assume the role of sterile helpers to their parents and siblings (increasing the direct reproductive success of kin, and thereby their inclusive fitness). As yet, there is no empirical evidence supporting this clearly speculative interpretation of the adaptive significance of ambivalent attachment in the ancestral environment.

5. Methodological issues

A number of methodological issues complicate the potential use of the concept of alternative strategies in psychiatry and clinical psychology. First, alternative strategies are discrete behavioral phenotypes, and the distinction between discrete and continuous variation is often difficult in the study of both 'normal' human behavior and clinical phenotypes [39,40]. For example, the controversy whether antisocial personality is a dimension or a category is still unresolved. The question is further complicated by data indicating that antisocial personality may be a heterogeneous construct, including a variant which

is best described as a taxon (i.e. primary psychopathy) and a variant which is best described as a dimension (i.e. secondary psychopathy) [18].

Second, in re-interpreting a variant behavioral phenotype as adaptive, it is important to distinguish between the beneficial effects of the behavior pattern that have been selected and its possible, non-selected beneficial effects. For instance, compared to a healthy child of the same age, an autistic child can be much less distressed by the separation from his mother, but this does not mean that the highly disabling syndrome of infantile autism has evolved in the ancestral environment as a means to cope with maternal loss. Third and most important, adaptive stories are easy to create and hard to falsify, and the risk of using inappropriate or insufficient standards of evidence for identifying adaptations and their functions is always present in the evolutionary study of human behavior. Such a risk is even higher in the field of evolutionary psychopathology where it is getting more and more popular the practice of picking a disorder out from current nosologies (which are of dubious validity) and inventing an adaptive explanation for its existence (see [21], for a detailed critique of the abuse of the adaptationist approach in evolutionary psychiatry).

Since the publication of the early critiques to adaptationism [41], theoretical evolutionary biology has made several advances that can help advocates of the adaptationist program to avoid storytelling and outlandish explanations in their attempts to elucidate the selection pressures that forged human behavior and psychological traits [42]. In particular, evolutionary psychologists have developed reasonable standards of evidence that could be used to identify adaptations. To qualify as an adaptation, a behavioral or psychological trait must meet not only the engineering criteria originally proposed by Williams [43] (i.e. high efficiency, high complexity, and high modularity) but also the criteria of low phenotypic variance, low genotypic variance, universality across cultures, universality across pre-history and history, and functional design promoting fitness under ancestral conditions [44]. However, some of these criteria are not very applicable to behavioral and psychological adaptations that evolved as components of alternative strategies. The criteria of low phenotypic variance, low genotypic variance, and zero heritability are applicable to those traits that are the product of stabilizing selection. If for a given trait there is an intermediate optimum at which fitness benefits are maximized relative to fitness costs, stabilizing selection should tend to eliminate genetic variation underlying the trait and lead to low levels of phenotypic variation by making the trait's expression resistant to environmental perturbations. Without any genetic variation in the trait, heritability (defined as the genetic variance divided by the phenotypic variance, $h^2 = V_G/V_P$) should not be higher than zero because everyone has the same genes. Hence, the criteria of low phenotypic variance, low genotypic variance, and zero heritability are not applicable to alternative strategies that are the product of diversifying (not stabilizing) selection as well as the criterion of high efficiency is not applicable to the 'making-the-best-of-a-bad-job' strategies. The development of new and better criteria for identifying behavioral and psychological adaptations, including alternative strategies, should be a major task for evolutionary psychology and psychiatry over the coming years. The development of these criteria should take into account the findings that are emerging from behavioral genomics and that show the importance of polymorphisms, pleiotropic effects (i.e. genes having multiple phenotypic expressions), oligogenic transmission (i.e. a small number of genes of moderate effect), and polygenic transmission (i.e. many genes of small effect) for the evolution of personality traits [45].

6. Clinical implications

Being related to the more general issue of re-defining disease in evolutionary terms [16], the application of the concept of alternative strategies in psychiatry and clinical psychology has diagnostic and therapeutic implications. As these clinical sciences necessarily engage the enormous range of human diversity, they also must move beyond narrowly normative diagnostics and conceptualizations of the healthy ideal to consider the evolutionary origins of individual differences [46]. In psychiatry, the medicalization of the human condition [47] is a long-standing problem. In the past, cultural prejudices and political aims were the major causes of the social construction of mental illness, as for example in the (in)famous case of drapetomania (the 'mental disease' causing black slaves to run away) [48]. In the last few years, the social construction of mental illness has partly been replaced by the corporate construction of psychiatric disorder. Pharmaceutical companies are actively involved in sponsoring the diagnostic definition of new diseases and promoting them to both prescribers and consumers [49]. It seems that some new behavior is medicalized every day. The recent psychiatric literature has witnessed the conversion of sexual desire into an 'addiction' complete with support groups, and excessive fear of social interaction into a mental disorder treatable with medication. One study [50] has coined the term 'female sexual dysfunction' to refer to sexual difficulties such as lack of desire for sex, anxiety about sexual performance, and problems with lubrication that are present in 43% of the American women aged 18–59, even though leading sex researchers have argued that these difficulties may reflect healthy and functional responses in women faced with stress, tiredness, or threatening patterns of behavior from their partners [51].

The necessity of avoiding medicalization of individual differences is not simply an intellectual exercise. The concept of disease acts not only to describe and explain, but also to enjoin to action. For this reason, labeling a psychological or behavioral condition as sick may have

serious individual and social consequences. At the individual level, inappropriate medicalization carries the dangers of self-reproach, social stigma, inappropriate treatment decisions, and iatrogenic illness. At the social level, the resources invested in diagnosing and treating healthy people are likely to be diverted away from preventing and treating individuals with real diseases. Since a major contribution of evolutionary theory is the insight that individual differences are core biological features of any animal species, including *Homo sapiens*, the application of the concept of alternative strategies to psychiatry and clinical psychology can be a powerful antidote to the growing tendency to medicalize human diversity.

A correct application of evolutionary knowledge in order to distinguish between adaptive behavioral variation and true psychopathologies should not necessarily lead to the conclusion that therapeutic intervention should be limited to mental and behavioral conditions that are biologically maladaptive. In contemporary medicine, many therapeutic interventions address problems that are not diseases but that are associated with subjective suffering or undesirable consequences. Cosmetic surgery and anti-aging therapies are just two examples. It is unrealistic to think that psychiatry and clinical psychology will remain extraneous to this process that is changing cultural expectations toward medical therapies. Because one of the basic aims of medicine is to alleviate human suffering, an understanding of the evolutionary foundations of individual differences should translate into more effective ways for promoting personal and social well-being, not into the search for natural laws determining what is therapeutically right or wrong. Hence, since evolved human behaviors may sometimes be a source of considerable individual distress and social concern, it is not a contradiction that a clinician embracing an evolutionary view of individual differences informs the patient that his/her condition is not a true mental disorder and, at the same time, accepts to help the patient with the therapeutic means that are used to treat true psychopathologies.

Assume for the moment that the evolutionary hypothesis of the origin of avoidant attachment is correct. An understanding of the selective pressures that favored the evolution of a 'low-commitment' alternative strategy in the ancestral environment by no means questions the legitimacy of the efforts to prevent and treat avoidant attachment in the modern environment. In our society, individuals with avoidant attachment are likely to be considered unreliable romantic partners and bad parents. They often seem cool and somewhat remote, and tend to dismiss the importance of relationships as well as feelings. Compared to individuals with secure attachment, they are more likely to respond to high levels of environmental stress with psychosomatic symptoms and dysfunctional anger [52]. In addition, when affected by chronic diseases that require lifetime treatment such as diabetes, they show poor compliance to prescribed therapy [53]. All these aspects may impose much suffering not only to individuals with avoidant attachment but also to their spouses, children, and friends. It is clear that, if a person with avoidant attachment asks for help, the decision to begin a psychotherapy has nothing to do with the consideration that avoidant attachment may represent a psychological mechanisms evolved to optimize adult reproductive strategies.

In conclusion, the message that this paper would convey to clinicians is that the concept of alternative strategies overturns a cherished myth about evolution and human nature, the myth that an evolutionary perspective inevitably promotes a view of human behavior as a mosaic of invariant, species-universal features that reflect optimally designed adaptations. Rather, the analysis of alternative strategies shows that the condition of health (as defined in evolutionary terms) is compatible with the presence of imperfection, constraints, trade-offs between competing demands, and a wide range of individual differences.

References

[1] Plomin R. Individual differences research in a postgenomic era. Pers Individ Diff 2002;33:909–20.
[2] Temple LKF, McLeod RS, Gallinger S, Wright JG. Defining disease in the genomics era. Science 2001;293:807–8.
[3] Mayr E. The growth of biological thought. Cambridge, MA: Harvard University Press; 1982.
[4] Cohen H. The evolution of the concept of disease. In: Caplan AL, Engelhardt Jr HT, McCartney JJ, editors. Concepts of health and disease: interdisciplinary perspectives. Reading, MA: Addison-Wesley; 1981. p. 209–20.
[5] Ridley M. Evolution, 2nd ed. Blackwell: Oxford; 1996.
[6] Brockmann HJ. The evolution of alternative strategies and tactics. Adv Study Behav 2001;30:1–51.
[7] Krebs JR, Davis NB. An introduction to behavioural ecology, 2nd ed. Blackwell: Oxford; 1987.
[8] Gross MR. Alternative reproductive strategies and tactics: diversity within sexes. Trends Ecol Evol 1996;11:92–7.
[9] Maynard Smith J. Evolution and the theory of games. Cambridge, UK: Cambridge University Press; 1982.
[10] Gross MR. Disruptive selection for alternative life histories in salmon. Nature 1985;313:47–8.
[11] Brockmann HJ, Grafen A, Dawkins R. Evolutionary stable nesting strategy in a digger wasp. J Theor Biol 1979;7:473–96.
[12] Setchell JM, Dixson AF. Arrested development of secondary sexual adornments in subordinate adult male mandrills (*Mandrillus sphinx*). Am J Phys Anthropol 2001;115:245–52.
[13] Maggioncalda AN, Czekala NM, Sapolsky RM. Growth hormone and thyroid stimulating hormone concentrations in captive male orangutans: implications for understanding developmental arrest. Am J Primatol 2000;50:67–76.
[14] Maggioncalda AN, Sapolsky RM, Czekala NM. Reproductive hormone profiles in captive male orangutans: implications for understanding developmental arrest. Am J Phys Anthrop 1999;109: 19–32.
[15] Galdikas B. Adult male sociality and reproductive tactics among orangutans at Tanjung Puting. Folia Primatologica 1985;45:9–24.
[16] Troisi A, McGuire MT. Darwinian psychiatry and the concept of mental disorder. Neuroendocrinol Lett 2002;23(suppl. 4):31–8.

[17] American Psychiatric Association. Diagnostic and statistical manual of mental disorders, fourth edition (DSM-IV), 4th ed. Washington, DC: American Psychiatric Association; 1994.
[18] Skeem JL, Poythress N, Edens JF, Lilienfeld SO, Cale EM. Psychopatic personality or personalities? Exploring potential variants of psychopathy and their implications for risk assessment Aggr Viol Behav 2003;8:513–46.
[19] Harpending H, Sobus J. Sociopathy as an adaptation. Ethol Sociobiol 1987;8(suppl. 3):63s–772.
[20] Mealy L. The sociobiology of sociopathy: an integrated evolutionary model. Behav Brain Sci 1995;18:523–99.
[21] McGuire MT, Troisi A. Darwinian psychiatry. New York: Oxford University Press; 1998.
[22] Lalumiere ML, Harris GT, Rice ME. Psychopathy and developmental instability. Evol Hum Behav 2001;22:75–92.
[23] Trivers R. Social evolution. Menlo Park, CA: Benjamin/Cummings Publishing Co; 1985.
[24] Slutske WS. The genetics of antisocial behavior. Curr Psychiatry Rep 2001;3:158–62.
[25] Skilling TA, Quinsey VL, Craig W. Evidence of a taxon underlying serious antisocial behavior in boys. Crim Justice Behav 2001;28: 450–70.
[26] Frank RH. Passions within reason: the strategic role of the emotions. New York: Norton and Company; 1988.
[27] Raine A, Reynolds C, Venables PH, Mednick SA, Farrington DP. Fearlessness, stimulation-seeking, and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. Arch Gen Psychiatry 1998;55:745–51.
[28] Herpertz SC, Werth U, Lukas G, Qunaibi M, Schuerkens A, Kunert HJ, et al. Emotion in criminal offenders with psychopathy and borderline personality disorder. Arch Gen Psychiatry 2001;58: 737–45.
[29] Raine A. Biosocial studies of antisocial and violent behavior in children and adults: a review. J Abnorm Child Psychol 2002;30: 311–26.
[30] Raine A, Lencz T, Taylor K, Hellige JB, Bihrle S, Lacasse L, et al. Corpus callosum abnormalities in psychopathic antisocial individuals. Arch Gen Psychiatry 2003;60:1134–42.
[31] Ainsworth MDS, Blehar MC, Waters E, Wall S. Patterns of attachment: a psychological study of the strange situation. Hillsdale, NJ: Erlbaum; 1978.
[32] Belsky J, Steinberg L, Draper P. Childhood experience, interpersonal development and reproductive strategy: an evolutionary theory of socialization. Child Dev 1991;62:647–70.
[33] Belsky J. Modern evolutionary theory and patterns of attachment. In: Cassidy J, Shaver PR, editors. Handbook of attachment: theory, research, and clinical applications. New York: The Guilford Press; 1999. p. 141–61.
[34] Chisholm J. The evolutionary ecology of attachment organization. Hum Nature 1996;7:1–38.
[35] Simpson JA, Gangstead SW. Individual differences in sociosexuality: evidence for convergent and discriminative validity. J Pers Soc Psychol 1991;60:833–70.
[36] Van IJzendoorn MH. Adult attachment representations, parental responsiveness, and infant attachment: a meta-analysis on the predictive validity of the adult attachment interview. Psychol Bull 1995;117:387–403.
[37] Ellis BJ, Garber J. Psychosocial antecedents of variation in girls' pubertal timing: maternal depression, stepfather presence, and marital and family stress. Child Dev 2000;71:485–501.
[38] Rosenstein D, Horowitz HA. Adolescent attachment and psychopathology. J Consult Clin Psychol 1996;64:244–53.
[39] Lenzenweger MF. Consideration of the challenges, complications, and pitfalls of taxometric analysis. J Abnorm Psychol 2004;113: 10–23.
[40] Meehl PE. What's in a taxon. J Abnorm Psychol 2004;113:39–43.
[41] Gould SJ, Lewontin RC. The spandrels of San Marco and the Panglossian paradigm: a critique of the adaptationist programme. Proc R Soc Lond, Series B 1979;205:581–98.
[42] Andrews PW, Gangestad SW, Matthews D. Adaptationism—how to carry out an exptationist program. Behav Brain Sci 2002;25:489–553.
[43] Williams GC. Adaptation and natural selection. Princeton, NJ: Princeton University Press; 1966.
[44] Miller G. Mental traits as fitness indicators: expanding evolutionary psychology's adaptationism. Ann New York Acad Sci 2000;907: 62–74.
[45] Plomin R, DeFries JC, Craig IW, McGuffin P. Behavioral genomics. In: Plomin R, DeFries JC, Craig IW, McGuffin P, editors. Behavioral genetics in the postgenomics era. Washington, DC: American Psychological Press; 2003. p. 531–40.
[46] Troisi A, McGuire MT. Evolution and mental health. In: Friedman HS, editor. Encyclopedia of mental health, vol. 2. San Diego: Academic Press; 1998. p. 173–81.
[47] Chodoff P. The medicalization of the human condition. Psychiatr Serv 2002;53:627–8.
[48] Cartwright SA. Report on the diseases and physical peculiarities of the negro races. The New Orleans Med Surg J 1851;7:707–9.
[49] Moynihan R, Heath I, Henry D. Selling sickness: the pharmaceutical industry and disease mongering. Br Med J 2002;324:886–90.
[50] Laumann EO, Paik A, Rosen RC. Sexual dysfunction in the United States: prevalence and predictors. J Am Med Ass 1999;281:537–44.
[51] Bancroft J. The medicalization of female sexual dysfunction: the need for caution. Arch Sex Behav 2002;31:451–5.
[52] Mikulincer M, Florian V, Weller A. Attachment styles, coping strategies, and posttraumatic psychological distress: the impact of the Gulf War in Israel. J Pers Soc Psychol 1993;64:817–26.
[53] Ciechanowski PS, Katon WJ, Russo JE, Walker EA. The patient-provider relationship: attachment theory and adherence to treatment in diabetes. Am J Psychiatry 2001;158:29–35.