Human handedness and the concept of developmental stability

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Abstract

A model is proposed to explain the etiology of pathological handedness. Developmental instability, caused by elevated genotypic homozygosity, environmental disturbances, or their interaction, overrides programmed laterality and handedness in the same way that it perturbs the bilaterally symmetrical expression of morphological and metric traits. The model predicts that pathological handedness should be elevated among individuals with higher than average homozygosity and individuals who have developed under unfavorable uterine environments. Suggestions are offered for specific populations in which the predictions may be tested.

Introduction

Left hand preference, or sinistrality, is found in about ten percent of the population. Recent findings show that this direction and frequency of hand preference can be detected in utero by the end of the first trimester (Hepper et al., 1990). Most researchers agree that hand preference has a hereditary component, but not all agree as to the genetic mechanisms involved (reviewed in Levy, 1977). Annett (1985) favors the role of a single gene with two alleles, the one for dextrality being dominant. Other researchers feel the hand preference results from a complex interaction of multiple genetic and non-genetic factors (Porac & Coren, 1981). While handedness is often dichotomized, it is more appropriately expressed as a continuum, because individuals who are right or left handed differ in the degree to which the nonpreferred hand is actually used in specific tasks (Annett, 1985). Thus, the literature contains terms like nonright-handed or mixed-handedness.

To complicate matters, an elevated incidence of nonright-handers has been repeatedly found in a number of different subpopulations. Significant departures from anticipated dextrality have been reported among individuals with a number of clinically defined phenotypes including autism (Soper et al., 1986), schizophrenia (Green et al., 1989), dyslexia (Annett, 1985) mental retardation (Soper et al., 1987), low birth weight and prematurity (Searleman et al., 1989), and immune disease (Geschwind & Behan, 1982). Among nonclinically defined or normal subpopulations, handedness distributions have been reported to vary with factors like maternal age (Coren, 1990), life expectancy (Coren & Halpern, 1991) intellectual abilities (Kelshaw & Annett, 1983; Benbow, 1986), and even college major (Fry, 1990).

While the above references are relatively recent, an excess of nondextrality, especially among clinically defined sub-populations, has been noted and of interest for a long time, leading Satz (1973) to apply the term 'pathological left-handedness' to those individuals exhibiting left hand preference despite being genetically right-handed. The concept can be extended to right-handers as well, when dextrality is not expected based on family history. Use of the term 'pathological' in describing unanticipated shifts from right or left hand preference has some unfortunate connotations, but is now firmly entrenched in the literature. A model for the origin of pathological left-handedness was first described by Satz (1972) and developed more completely in subsequent contributions by Soper and Satz (1984), Porac and Coren (1981), Coren and Searleman (1990), and Coren and Halpern (1991).

In general, their model starts with 90% of the
population programmed to be right-handed and 10% left-handed. Superimposed upon this, 10% of the entire population is caused by some unspecified pathological intervention to change from their programmed hand preference. Numerically, this means that 9% of the population shifts from right- to left-handedness while only 1% shifts in the other direction, resulting in an excess of nonright-handers. The exact nature of any pathological disturbance has not been unequivocally identified. Brain injuries before age six have been observed to shift hand preference (Satz, 1972), but this sort of trauma can only explain a small fraction of cases of handedness shifts.

A good deal of evidence has been gathered to support a strong association of birth related stressors with the shift in handedness (reviewed in Searleman et al., 1989). However, complications at the time of delivery may, in many cases, reflect perturbations of earlier developmental events. These early interferences are compatible with the idea of a 'Rare Trait Marker' (Coren & Searleman, 1990) and could be either of genetic or of nongenetic origin. Furthermore, not all individuals undergoing birth stress exhibit departures from anticipated handedness. Therefore, any model to explain pathological handedness must be able to account for its occurrence as well as its absence.

My purpose is not to discriminate among models proposed for the inheritance of handedness. However, there is a biological principle linking both population and developmental genetics that I think has value in explaining the etiology of what has become known as pathological left- (and right-) handedness. Below I describe the concept of developmental stability and offer a model in which genetic and/or environmental perturbations of developmental homeostasis can result in deviations from normal laterality patterns observed in certain sub-populations.

The concept of developmental stability

Developmental stability, or homeostasis, is the ability of an organism to develop according to its ontogenetic program despite adverse environmental conditions (Waddington, 1957). A variety of measures are typically employed in assessing developmental stability in animals. Major and minor physical anomalies (Waldrop et al., 1971), sometimes known as 'phenodeviants' (Lerner, 1954), provide evidence of disruptive influences during development. The most widely used measure is called fluctuating asymmetry (FA), seen in paired bilateral traits (Palmer & Strobeck, 1986).

Fluctuating asymmetry, or FA, is only one kind of asymmetry seen in organisms with bilateral symmetry. In order to understand the biological significance of FA, it is helpful to first describe the other kinds of asymmetries observed in animals (Fig. 1). One of these is directional asymmetry (DA). In DA, all members of a species show consistent structural or functional bias for a particular side of the body.

Fig. 1. Three different kinds of asymmetry found in bilaterally symmetrical organisms. DA or directional asymmetry is seen when the mean for the trait always exhibits the same right OR left bias for the species. In antisymmetry, or AS, there is always a bias for one side, but half of the population shows a bias for each side. Fluctuating asymmetry, FA, occurs when the distribution of right minus left values follows a normal curve.