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EDITORIAL

Why psychogeriatrics starts right after adolescence

Mara Parellada

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The borders between pediatric and adult medical specialties are always somewhat artificial, and based on spurious considerations, such as usually attaining the age of majority, i.e., 18 years; however, the borders between so-called child and adolescent (C&A) psychiatry and adult psychiatry are particularly questionable. Compared with other mammals, Homo sapiens take a long time to reach maturity: adult sexual function is not reached until around 13–15 years of age, and the brain is not fully developed until at least age 25.

The symptoms of severe mental disorders do typically start in young people (their vulnerability can be traced to infancy, or even in utero) [1]. The vast majority of mental disorders can be divided into two broad categories: those with childhood onset, such as ASD, ADHD, conduct disorders, and most anxiety disorders; and those that incubate over neurodevelopment and erupt in adolescence and young adulthood (e.g., schizophrenia, bipolar disorders).

Five of Erikson's eight stages of life occur before age 18. A substrate of biological changes runs in parallel to these psychological stages; indeed, most changes in brain development occur before the third decade of life. Absolute cortical volume peaks at 9.3 years in males, and at 8 years in females; the convex hull area rises robustly from age 3 in both sexes, reaching its peak in males at 15.2 years and in females at 11.8 years [2]. The visual, auditory, and limbic cortices, which myelinate early, show a more linear pattern of aging than the frontal and parietal neocortices, which continue myelination into adulthood. Except for the posterior temporal regions, which have a more protracted course of cortical maturation and continue to gain gray matter density up to age 30, the other regions (dorsal frontal and parietal association cortices, on both the lateral and interhemispheric surfaces) see a decline in their gray matter density between the ages of 7 and 60 [3]. Brains do not develop normally in the absence of critical genetic signaling, or in the absence of essential environmental input. The key to understanding the origins and emergence of both the brain and behavior lies in understanding how inherited and environmental factors are engaged in the dynamic and interactive processes that define and guide development of the neurobehavioral system [4]—and this knowledge is key for the work of child psychologists and psychiatrists.

It is well known that chronic stress alters brain conformation. For example, animal studies show that the hippocampus, through stress-induced dendritic retraction, becomes vulnerable to neurotoxicity, even after the stress has stopped; the retraction protects the neuron from glutamate damage during the stressful situation, but this may come at the price of interfering with normal development and learning [5]. Smaller hippocampi have been associated with childhood maltreatment, as well as major depression and post-traumatic stress disorder [6, 7]. In addition to the effect on brain development of cumulative exposure to stressful situations that may exceed the brain's buffering capacity to restore the stress system to its baseline status, exposure to stressful situations during sensitive periods of development can become embedded into regulatory physiological processes. Epigenetic changes, such as DNA

M. Parellada (✉)
Child and Adolescent Psychiatry Department,
Instituto de Investigación Sanitaria Gregorio Marañón, IiSGM,
Hospital General Universitario Gregorio Marañón, CIBERSAM,
Ibiza 43, 28009 Madrid, Spain
e-mail: parellada@hggm.es

M. Parellada
Hospital General Universitario Gregorio Marañón,
CIBERSAM, Ibiza 43, 28009 Madrid, Spain

methylation changes in the stress system, have been shown to occur in response to environmental cues, such as maternal care in rodents [8], and are also present in autopsies of human adults with a history of childhood maltreatment [8, 9]. In the same vein, if a given circuit underlying any brain function is left unstimulated during its developmentally sensitive period, the brain function served by that circuit could be permanently compromised [10].

Plasticity of the brain seems to be lifelong, but it is greater in early years. Cerebral plasticity is the dynamic potential of the brain to reorganize itself during ontogeny, learning, or following damage. Mechanisms such as modulations of synaptic efficacy, unmasking of latent connections (including dendritic remodeling), phenotypic modifications and neurogenesis have been identified [11]. Recoveries from trauma, as well as changes in brain regions sustaining common functions due to trauma or developmental problems, typically occur in young people. A better understanding of these mechanisms of brain reshaping is needed to advance in the therapeutics of children exposed to early traumatic experiences and those affected by neurodevelopmental disorders. Sensitive developmental windows are posited in the early years; the period immediately after a function is gained could be particularly sensitive, as shown in studies of auditory processing, where both sensitivity and plasticity are highest during the days following hearing onset [12]. The same was shown for the development of the visual cortex a few decades ago, and this is probably true for most neural circuits, such as those that support bonding and language learning, and many others.

Investment in deeper research on the impact of intervention to enhance resilience and protective factors, and diminish the effect of risk factors, will pay off, and is therefore necessary. Regarding secondary prevention, we need to investigate whether we could parallel cancer research and treatment; as Nobel Laureate Lee Hartwell said, "If you detect it at stage 1 or 2 most patients survive; if you detect it at stage 3 or 4 most patients die." Clinical staging of disorders is becoming popular in psychiatry [13], and treating patients at the early stages of disorders are proving effective. Psychological intervention in common mental disorders with psychotic symptoms seems to diminish the expression of full-blown psychotic disorders [14, 15]. Moreover, providing clinical care for patients with high-risk states may diminish the associated distress, improve functioning and reduce the duration of untreated psychosis, should it emerge (which correlates greatly with a positive future outcome). Early contact with services after the onset of any disorder is likely to diminish suffering and burden [16].

Perhaps the time has come to change the specialties' names, and talk about neurodevelopmental psychiatry, adult psychiatry, and neurodeterioration or aging psychiatry. With this perspective, adult psychiatry would shrink, and neurodevelopmental psychiatry would expand.

But no matter what we call them, these underlying realities are not reflected in the provision of services, the expertise of professionals, or investment in research. Data from the WHO's 2004 Global Burden of Disease study shows that worldwide, the main cause of YLDs in 10–24-year-old is neuropsychiatric disorders (45 %). The main risk factors for incident DALYs in 10–24-year-old were alcohol (7 % of DALYs), unsafe sex (4 %), iron deficiency (3 %), lack of contraception (2 %), and illicit drug use (2 %) [17]. The number one cause of disability in the age range 10–14 is unipolar depression, which accounts for 5.7 % of total DALYs, while unipolar depression and schizophrenia are the two leading causes in the age range 15–19 (accounting for 15.2 % of total DALYs) [17]. With these data in hand, C&A psychiatry should be receiving the majority of mental health resources. However, psychiatry currently seems to deal mainly with tertiary prevention.

Early detection means finding disorders before they worsen—before they devastate a person's life. Scientific evidence increasingly supports going beyond that approach, to embrace the promotion of healthy neurodevelopment and the reduction of toxic environmental triggers to prevent lifelong mental disorders before they occur.

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