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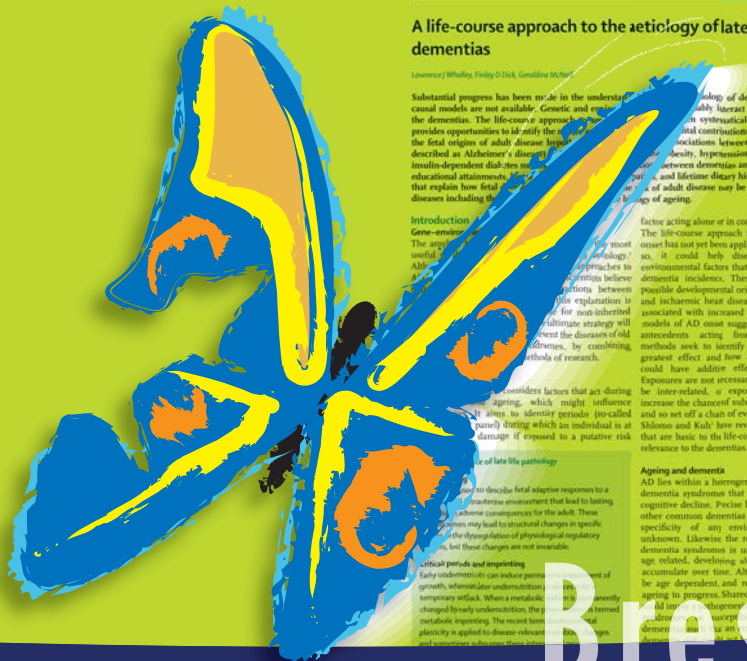
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A life-course approach to the aetiology of late-onset dementias

Lawrence J Whalley, Finlay D Dick, Geraldine Murray

Substantial progress has been made in the understanding of the aetiology of dementias, but comprehensive causal models are not available. Genetic and environmental factors are likely to contribute to the aetiology of dementias. The life-course approach to the aetiology of dementias, provides opportunities to identify the developmental origins of adult disease by examining associations between the dementias (most often described as Alzheimer's disease) and risk factors such as obesity, hypertension, hyperlipidaemia, and non-insulin-dependent diabetes mellitus, low educational attainments, and lifetime dietary history. Biological mechanisms that explain how fetal origins of adult disease may be relevant to many age-related diseases including the dementias are discussed.

Introduction

The aetiology of late-onset dementias is complex. Although genetic and environmental factors are likely to contribute to the aetiology of dementias, the ultimate strategy will be to identify periods (so-called critical periods) during which an individual is at damage if exposed to a putative risk factor acting alone or in combination with other factors. The life-course approach to common diseases of late onset has not yet been applied to AD.¹ However, if done so, it could help disentangle the genetic and environmental factors that contribute to differences in dementia incidence. There are few studies on the possible developmental origins of stroke, hypertension, and ischaemic heart disease (IHD), which are factors associated with increased dementia risk, even though models of AD onset suggest that there are important antecedents acting from conception.² Life-course methods seek to identify when exposures have their greatest effect and how accumulation of exposures could have additive effects over the life course. Exposures are not necessarily independent: insults can be inter-related, or exposure to one factor might increase the chances of subsequent exposure to another and so set off a chain of events leading to disease. Ben-Shlomo and Kuh³ have reviewed key conceptual issues that are basic to the life-course approach and of much relevance to the dementias.

Developmental origins of late life pathology

AD lies within a heterogeneous domain of age-related dementia syndromes that merge with lesser forms of cognitive decline. Precise boundaries between AD and other common dementias are poorly defined, and the specificity of any environmental risk factor is unknown. Likewise the relation between ageing and dementia syndromes is unclear. Dementias could be age related, developing alongside brain changes that accumulate over time. Alternatively, dementias could be age dependent, and rely intimately on biological ageing to progress. Shared environmental risk factors could influence the progression from pre-dementia states to dementia, but the extent of environmental contribution to dementia remains unclear.

Critical periods and imprinting

Early undernutrition can induce permanent growth, whereas later undernutrition is associated with a temporary setback. When a metabolic challenge is followed by early undernutrition, the permanent metabolic imprinting. The recent term 'developmental plasticity' is applied to disease-relevant changes and responses in humans that have been

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Biologia dell'invecchiamento

Comprendere per curare



Brescia

31 maggio 2007

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IL CORSO SARÀ RIVOLTO A
SPECIALISTI NEUROLOGI, GERIATRI E PSICHIATRI

Biologia dell'invecchiamento

Comprendere per curare

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Programma

09.30-10.00 Accoglienza e iscrizione

10.00-11.00 Malattia di Alzheimer fra genetica e ambiente

- influenze genetiche
- influenze socioeconomiche e stili di vita
- manifestazioni cliniche

Angelo Bianchetti, Brescia

11.00-11.30 Discussione

11.30-12.00 Coffee break

12.00-13.00 Dai neurotrasmettitori ai farmaci

- meccanismi biochimici del processo di deterioramento funzionale

- approcci terapeutici

Ferdinando Nicoletti, Roma

13.00-13.30 Discussione

13.30-14.30 Lunch

14.30-15.30 Gli antagonisti del recettore NMDA:
una nuova classe di farmaci

Alessandro Margiotta, Brescia

15.30-16.00 Discussione

16.00-16.30 Chiusura dei lavori

16.30-17.00 Test finale e svolgimento pratiche ECM