No Health without mental health

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The next 40 minutes…

- Childhood psychotic experiences
- Nature-nurture interplay
- Early Mental Health
- LCP versus AL – antisocial behaviour
- Booze, cannabis and other drugs
- Social determinants…
- Suicidality – a case study in what not to do

Retention in the Dunedin Study

Age	Year	Number	Percent*
Birth	1972-73		
3	1975-76	1037	100%
5	1977-78	991	96%
7	1979-80	954	92%
9	1981-82	955	92%
11	1983-84	925	90%
13	1985-86	850	82%
15	1987-88	976	95%
18	1990-91	993	97%
21	1993-94	992	97%
26	1998-99	980	96%
32	2004-05	972	96%
38	2010-12	961	95%
45	2017-18	???	???

** Percentage seen of those who were eligible (i.e. alive) at each age*


Canada
4
USA
11
UK
34
Europe
10
Asia
7
Mid-East
3
Africa
2
Australia
172
Dunedin, n=334
Elsewhere in NZ, n=384

Current research activities include studies of:

- SES inequalities - selection v causation
- Pathways to employment
- Personality continuities across the life-course
- Antisocial behaviour and criminality
- Long-term consequences of childhood adversity
- Maori health/cultural identity
- Cognition and neuropsychology
- Family health history study
- Mental health (including substance abuse)
- Intimate relationships and domestic violence
- Oral health
- Sexual & reproductive health
- Cardiovascular risk factors
- Retinal imaging and endothelial function
- Respiratory health
- Next generation studies (age 3 and age 15 years)

Current research activities (contd)

- Blood based studies
  - Chlamydia trachomatis
  - Herpes immunity
  - Cardiovascular disease risk factors
  - Inflammatory biomarkers
- Genetic studies
  - Mental health phenotypes
  - Asthma/allergy
  - Cardiovascular risk factors
  - Periodontal disease
- Methodological studies
  - Comparison of Dunedin sample with national data
  - Attrition analyses

Psychological Medicine (2011), **41**, 1–6. © Cambridge University Press 2010
doi:10.1017/S0033291710001005

EDITORIAL

Psychotic-like experiences in the general population: characterizing a high-risk group for psychosis

I. Kelleher[1]* and M. Cannon[1,2]

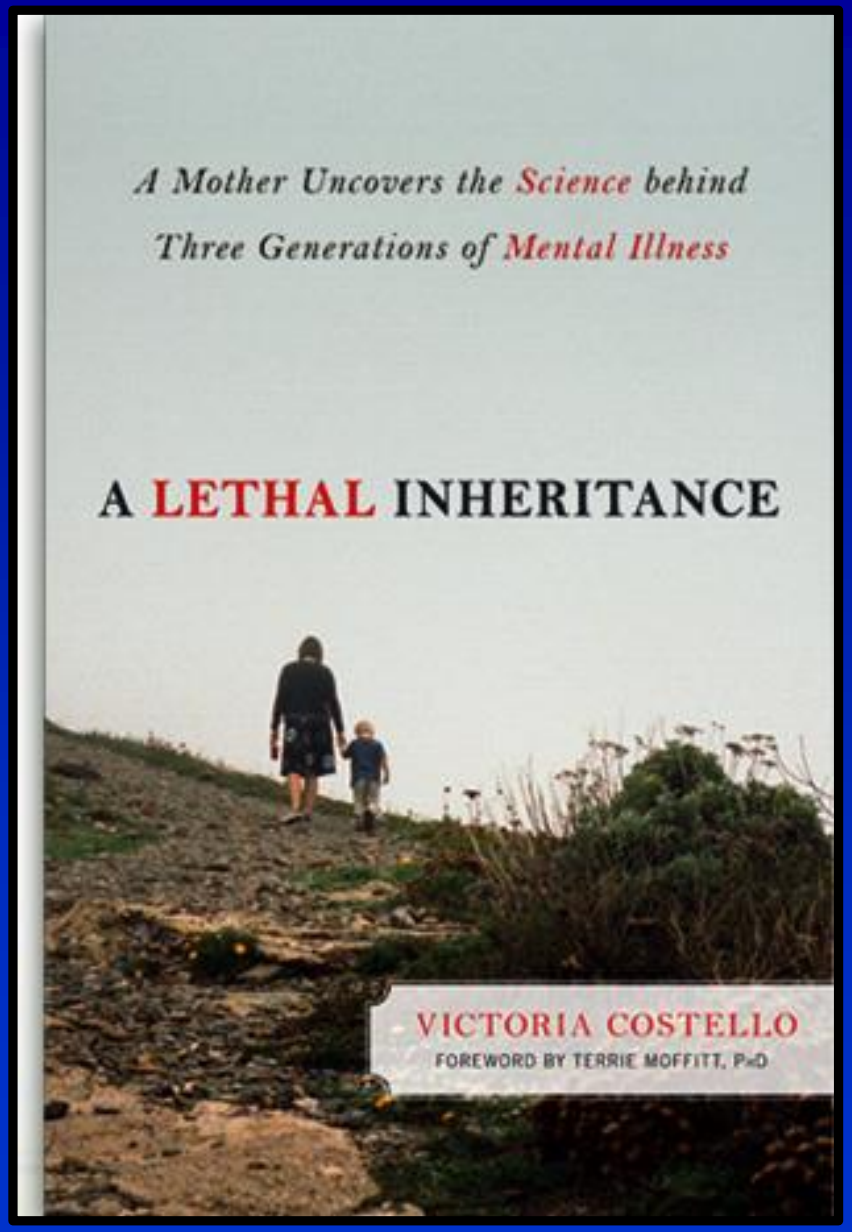
[1] *Department of Psychiatry, Royal College of Surgeons in Ireland, Dublin, Ireland*
[2] *Department of Psychiatry, Beaumont Hospital, Dublin, Ireland*

Recent research shows that psychotic symptoms, or psychotic-like experiences (PLEs), are reported not only by psychosis patients but also by healthy members of the general population. Healthy individuals who report these symptoms are considered to represent a non-clinical psychosis phenotype, and have been demonstrated to be at increased risk of schizophrenia-spectrum disorder. Converging research now shows that this non-clinical psychosis phenotype is familial, heritable and covaries with familial schizophrenia-spectrum disorder. A review of the research also shows that the non-clinical phenotype is associated extensively with schizophrenia-related risk factors, including social, environmental, substance use, obstetric, developmental, anatomical, motor, cognitive, linguistic, intellectual and psychopathological risk factors. The criterion and construct validity of the non-clinical psychosis phenotype with schizophrenia demonstrates that it is a valid population in which to study the aetiology of psychosis. Furthermore, it suggests shared genetic variation between the clinical and non-clinical phenotypes. Much remains to be learned about psychosis by broadening the scope of research to include the non-clinical psychosis phenotype.

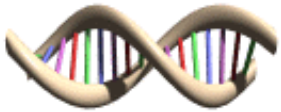
“PLEs share criterion validity with clinical psychosis

A clinical continuum between PLEs and psychotic disorder was demonstrated in an influential paper from the Dunedin birth cohort study (Poulton *et al.* 2000). Children aged 11 years who reported psychotic symptoms were shown to be at a 5- to 16-fold increased risk of schizophrenia-spectrum disorder in adulthood. ..”

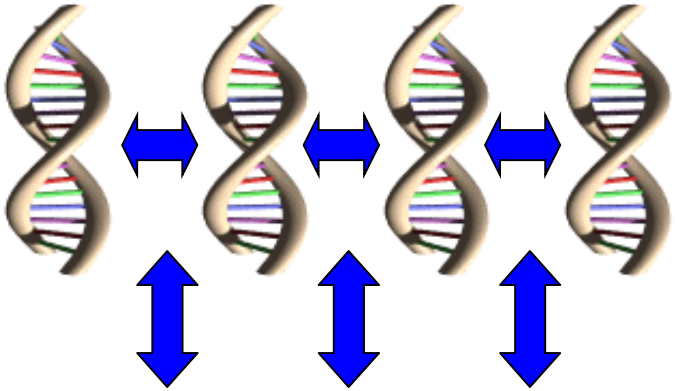
- **Dr Jessie Anderson**
- **DSM-III (1980)**
- **96% follow-up at age 26**



Amherst, NY: Prometheus Books, 2012. http://alethalinheritance.com



Behaviour



Family

Friends

School

Community

Perhaps ironically…

The most cutting-edge genetic research

is the best advertisement I know of for importance of the environment!

The question then becomes…

- When do we intervene; and
- with what to make the biggest difference?

What is the evidence base?

The World Health Organisation's "burden of disease framework" tells us that the three conditions associated with the greatest loss of life and productive years (i.e. living with a disability) are, in order:

1. cardiovascular disease;
2. cancer; and
3. mental health disorders.

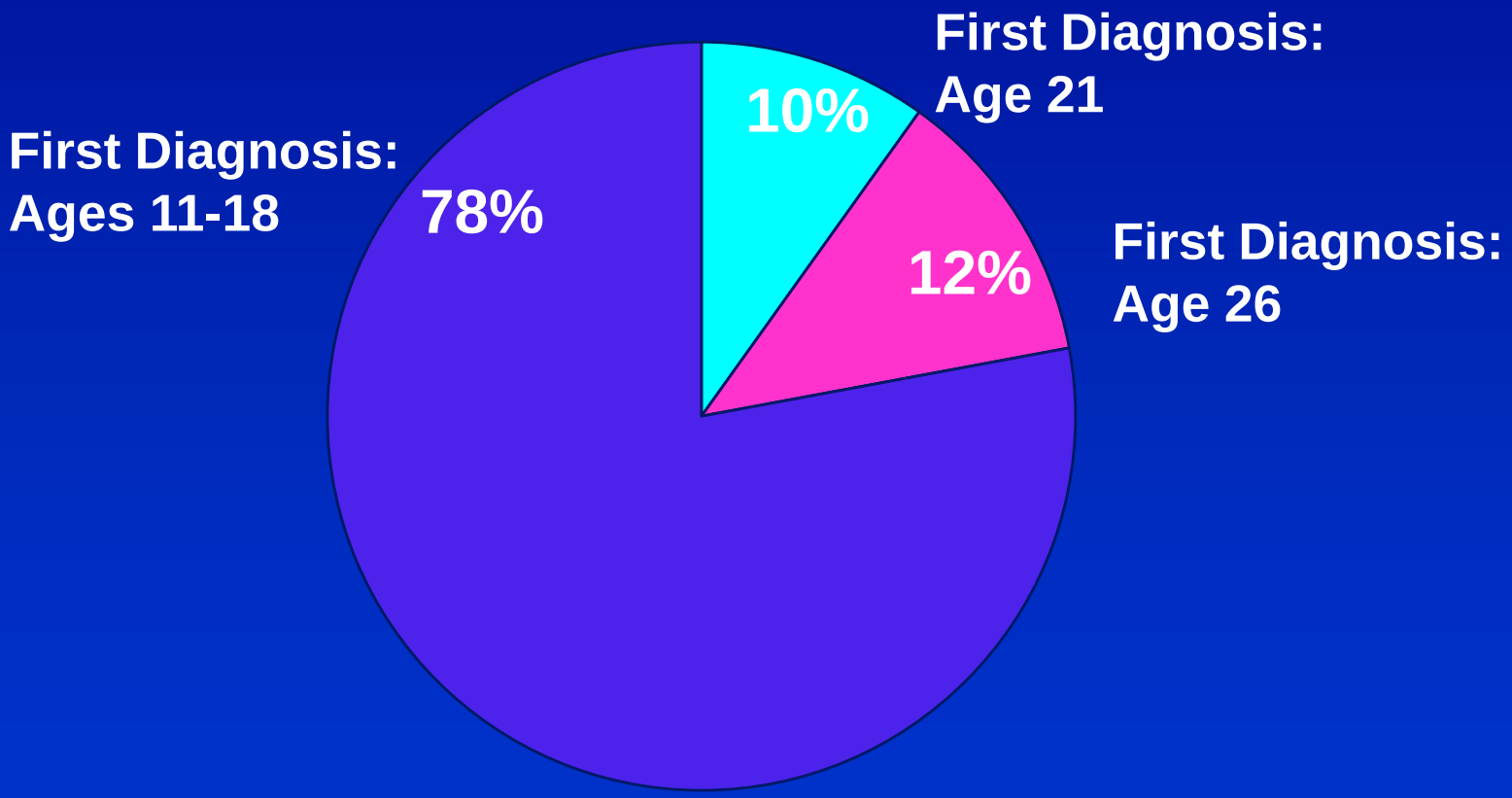
Mental health

In 2003, we showed that among adults with a psychiatric disorder, 50% had had a psychiatric diagnosis by the age of 15, and three-quarters by age 18 (i.e. by the end of adolescence).

Some have interpreted these findings to suggest that approximately half of all mental health disorders could be averted were we to intervene effectively during adolescence or earlier.

Kim-Cohen et al, *Archives of General Psychiatry*, 2003, 60: 709-719

¾ of adult patients of mental-health professionals have a juvenile disorder history



The take home message

Dealing with nascent or emerging mental health problems effectively, early on, could reduce:

- **an enormous amount of suffering borne by both the individual and the community; and**
- **a significant amount of lost productivity when viewed across the totality of a persons working life.**
- **The distinction between child and adolescent v adult psychiatry/psychology is a nonsense.**

Antisocial behaviour

The Dunedin study has arguably one of the most detailed [illegible] tisocial behaviour

- [illegible] aviour [illegible] course

- [illegible] orising [illegible] begin [illegible] ur in

The implications for intervention

- Early onset life-course persistent group: you need to intervene with both child and their family as early as possible

- Adolescent-onset group: the worst thing you can do is use a group intervention approach, given that their behaviour is partly driven by peer influence – individual interventions are required

NB: Prison is a group intervention which tends to expand rather than diminish the antisocial repertoire.

Bad behaviour = bad health

Antisocial behaviour *that emerges early in life and persists over time* is not only associated with

- **poor mental health;**
- **bad relationships; and**
- **criminal behaviour in adulthood**

but also increased risk for a range of physical health problems:

- **Heart disease and stroke (x 3)**
- **Symptoms of chronic bronchitis (x 3)**
- **Gum disease (x 4)**
- **Herpes (x 2)**
- **Smoking (x 10)**
- **Injuries (x 4)**
- **High rates of hospitalisation (x3)**

Odgers et al, *Archives of General Psychiatry*, 2007, 64: 476-484

No. 03-633

In The
Supreme Court of the United States

DONALD P. ROPER, SUPERINTENDENT,
POTOSI CORRECTIONAL CENTER,

Petitioner

v.

CHRISTOPHER SIMMONS

On Writ Of Certiorari To The Supreme Court Of Missouri

BRIEF FOR THE AMERICAN PSYCHOLOGICAL ASSOCIATION, AND THE MISSOURI PSYCHOLOGICAL ASSOCIATION AS *AMICI CURIAE* SUPPORTING RESPONDENT

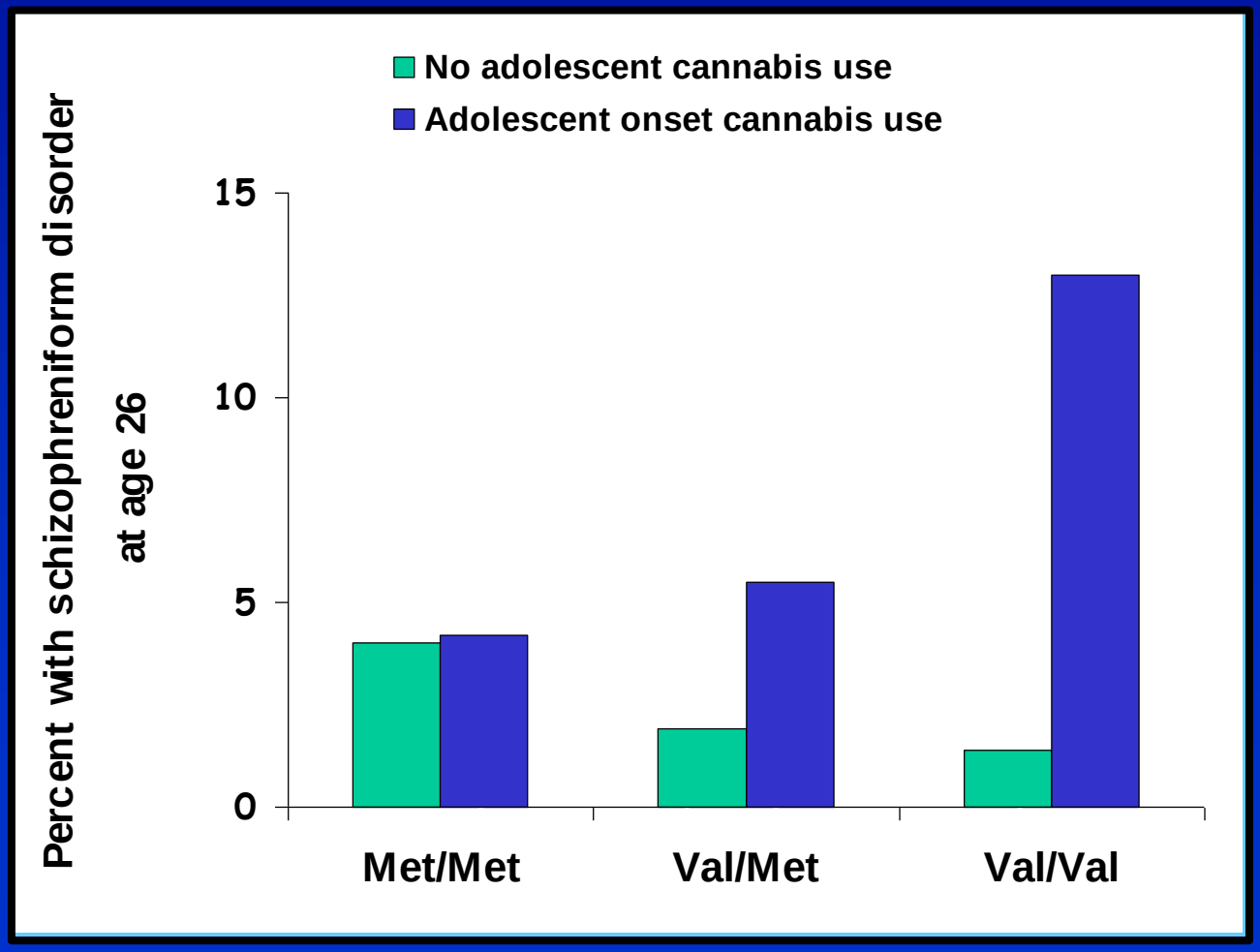
Cannabis and Psychosis

Arseneault, L., Cannon, M., Poulton, R., Murray, R. M., Caspi, A., and Moffitt, T. E. Cannabis use in adolescence and risk for adult psychosis: longitudinal prospective study. *BMJ, 2002, 325(7374): 1212-1213.*

Caspi, A., Moffitt, T. E., Cannon, M., McClay, J., Murray, R. M., Harrington, H. L., Taylor, A., Arseneault, L., Williams, B. S., Braithwaite, A., Poulton, R., and Craig, I. Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the COMT gene: Longitudinal evidence of a gene x environment interaction. *Biological Psychiatry, 2005 , 57: 1117-1127.*

Fergusson, D. M., Poulton, R., Smith, P. F., and Boden, J. M. Cannabis and psychosis: a summary and synthesis of the evidence. *BMJ, 2006, 332(Jan): 172-175.*

The influence of early-onset cannabis use on adult psychosis IS moderated by COMT genotype



Booze & Other Drugs

Odgers, et al (2008). Is it important to prevent early exposure to drugs and alcohol among adolescents? *Psychological Science,* Vol. 19(10), 1037-1044.

Effects of Early Substance Exposure on Adolescents' Adult Outcomes, Before and After Propensity-Score Matching

Adult outcome	Before propensity-score matching: Mean risk: No early exposure (n = 813)	Before propensity-score matching: Mean risk: Early exposure (n = 114)	Before propensity-score matching: Effect size	After propensity-score matching: Mean risk: No early exposure (n = 342)	After propensity-score matching: Mean risk: Early exposure (n = 114)	After propensity-score matching: Effect size
Substance dependence at age 32 (%)	11.1	28.8	3.25** (2.04–5.18)	14.7	28.8	2.25** (1.35–3.73)
Herpes infection at age 32 (%)	17.0	28.0	1.90** (1.18–3.07)	16.1	28.0	2.02** (1.18–3.44)
Early pregnancy (prior to age 21)[a] (%)	18.1	44.7	3.65** (1.93–6.86)	22.5	44.7	2.78** (1.38–5.57)
No educational qualifications by age 32 (%)	14.7	32.1	2.74** (1.76–4.27)	19.3	32.1	1.97** (1.22–3.19)
Number of criminal convictions between ages 17 and 32[b]	1.31	7.12	5.35** (2.85–10.05)	1.74	7.12	3.95** (2.23–6.97)

Note. Study members were categorized as early-exposed (i.e., exposed to alcohol or drugs on multiple occasions before age 15) versus non-early-exposed (i.e., not exposed to alcohol or drugs on multiple occasions before age 15). The reported effect sizes are odds ratios for all outcomes except number of criminal convictions, for which incidence-rate ratios are reported. The numbers in parentheses are 95% confidence intervals. All findings remained statistically significant after adjustment of standard errors via bootstrapping in PSMATCH2. [a]Early pregnancy was estimated for females only and was defined as having at least one pregnancy prior to age 21; sex-specific propensity scores were used for this female-only analysis. [b]Negative binomial regressions were applied to model incidence-rate ratios for the number of criminal convictions.

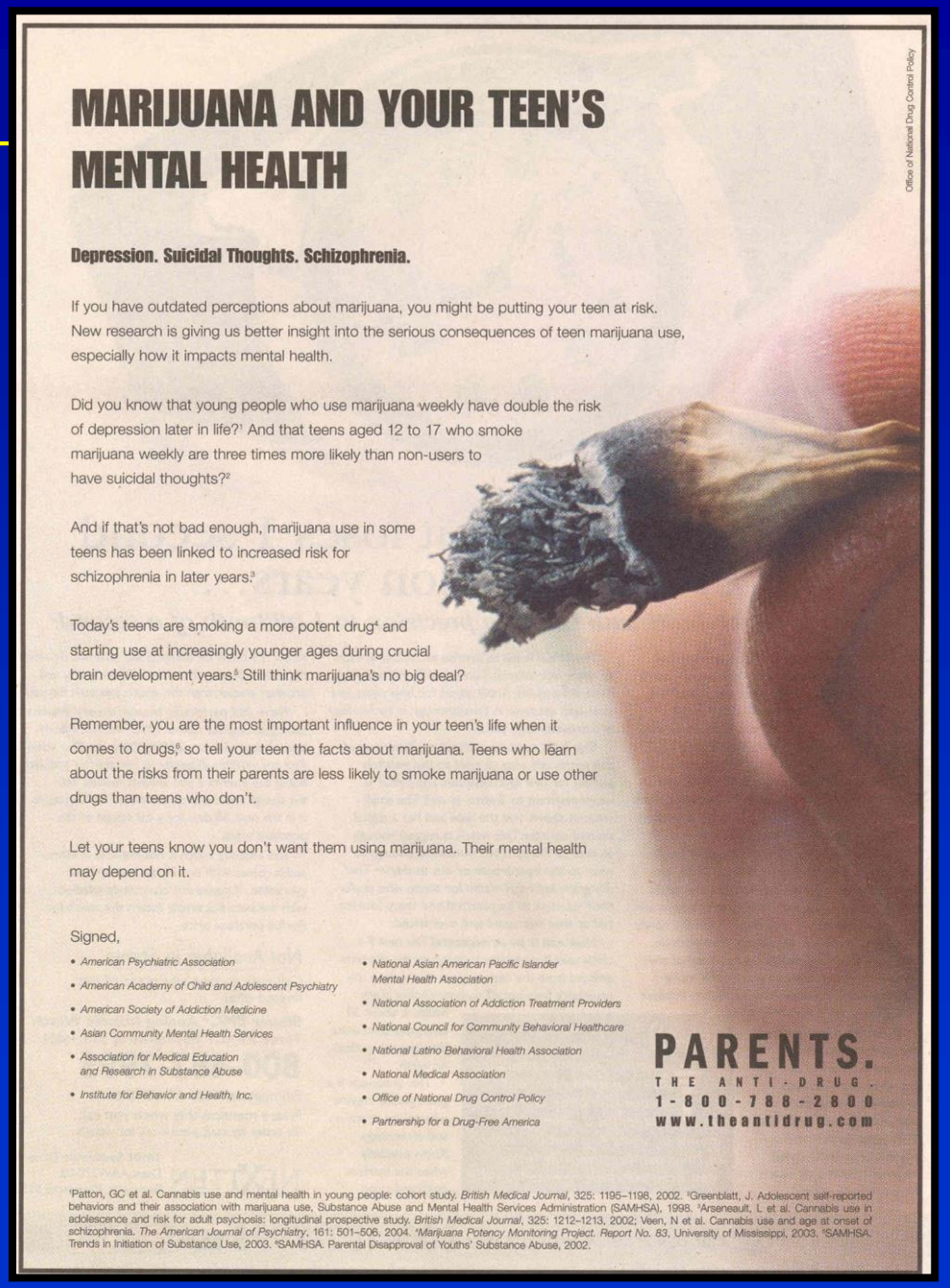
**$p < .05$.

Psychological SCIENCE

Public Health Implications

Office of National Drug Control Policy – Executive Office of the President, **USA**, 2005.

Advisory Council on the Misuse of Drugs, London, **United Kingdom**. September 2005.



MARIJUANA AND YOUR TEEN'S MENTAL HEALTH

Office of National Drug Control Policy

Depression. Suicidal Thoughts. Schizophrenia.

If you have outdated perceptions about marijuana, you might be putting your teen at risk. New research is giving us better insight into the serious consequences of teen marijuana use, especially how it impacts mental health.

Did you know that young people who use marijuana weekly have double the risk of depression later in life?[1] And that teens aged 12 to 17 who smoke marijuana weekly are three times more likely than non-users to have suicidal thoughts?[2]

And if that's not bad enough, marijuana use in some teens has been linked to increased risk for schizophrenia in later years.[3]

Today's teens are smoking a more potent drug[4] and starting use at increasingly younger ages during crucial brain development years.[5] Still think marijuana's no big deal?

Remember, you are the most important influence in your teen's life when it comes to drugs,[6] so tell your teen the facts about marijuana. Teens who learn about the risks from their parents are less likely to smoke marijuana or use other drugs than teens who don't.

Let your teens know you don't want them using marijuana. Their mental health may depend on it.

Signed,

- *American Psychiatric Association*
- *American Academy of Child and Adolescent Psychiatry*
- *American Society of Addiction Medicine*
- *Asian Community Mental Health Services*
- *Association for Medical Education and Research in Substance Abuse*
- *Institute for Behavior and Health, Inc.*
- *National Asian American Pacific Islander Mental Health Association*
- *National Association of Addiction Treatment Providers*
- *National Council for Community Behavioral Healthcare*
- *National Latino Behavioral Health Association*
- *National Medical Association*
- *Office of National Drug Control Policy*
- *Partnership for a Drug-Free America*

PARENTS.
THE ANTI-DRUG.
1-800-788-2800
www.theantidrug.com

[1]Patton, GC et al. Cannabis use and mental health in young people: cohort study. *British Medical Journal*, 325: 1195–1198, 2002. [2]Greenblatt, J. Adolescent self-reported behaviors and their association with marijuana use, Substance Abuse and Mental Health Services Administration (SAMHSA), 1998. [3]Arseneault, L et al. Cannabis use in adolescence and risk for adult psychosis: longitudinal prospective study. *British Medical Journal*, 325: 1212–1213, 2002; Veen, N et al. Cannabis use and age at onset of schizophrenia. *The American Journal of Psychiatry*, 161: 501–506, 2004. [4]*Marijuana Potency Monitoring Project. Report No. 83*, University of Mississippi, 2003. [5]SAMHSA. Trends in Initiation of Substance Use, 2003. [6]SAMHSA. Parental Disapproval of Youths' Substance Abuse, 2002.

Social determinants

- Sought to understand why children exposed to different adverse psycho-social experiences are at elevated risk for age-related disease.
- Tested whether adverse childhood experiences predict enduring abnormalities and stress sensitive biological systems: the nervous, immune and endocrine/metabolic systems.
- Three childhood predictors:
  - Socio-economic disadvantage
  - Maltreatment
  - Social isolation
- Three adult outcomes:
  - Depression
  - Inflammation
  - Clustering of metabolic risk markers

Prediction of three age-related-disease risks in adults with different levels of exposure to adverse childhood experiences and established developmental risk factors

	Risk Ratio (95% CI)					
	Panel 1: Major Depression		Panel 2: hsCRP >3 mg/L		Panel 3: Clustering of Metabolic Risk Markers	
	Bivariate[a]	Multivariate[b]	Bivariate[a]	Multivariate[b]	Bivariate[a]	Multivariate[b]
Adverse Childhood Experiences						
Childhood SES						
High	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Average	0.78 (0.53-1.15)	0.90 (0.60-1.34)	1.59 (1.00-2.52)	1.55 (0.98-2.46)	1.53 (0.89-2.61)	1.52 (0.89-2.57)
Low	1.22 (0.80-1.87)	1.14 (0.72-1.79)	1.96 (1.19-3.25)	1.63 (0.98-2.70)	2.65 (1.52-4.62)	2.11 (1.20-3.70)
Childhood maltreatment						
No	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Probable	1.18 (0.84-1.66)	1.07 (0.76-1.51)	1.18 (0.87-1.60)	1.16 (0.85-1.58)	1.56 (1.13-2.14)	1.39 (1.01-1.93)
Definite	2.28 (1.58-3.27)	1.69 (1.13-2.55)	1.80 (1.26-2.58)	1.56 (1.08-2.26)	1.28 (0.77-2.11)	1.04 (0.65-1.67)
Childhood social isolation						
Very low	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Low	1.32 (0.80-2.16)	1.35 (0.84-2.17)	1.29 (0.83-2.02)	1.31 (0.84-2.05)	1.12 (0.64-1.95)	1.14 (0.67-1.95)
High	1.22 (0.74-2.00)	1.20 (0.74-1.95)	1.42 (0.92-2.20)	1.39 (0.91-2.15)	1.52 (0.90-2.55)	1.34 (0.81-2.24)
Very high	1.99 (1.25-3.17)	1.76 (1.12-2.77)	1.62 (1.05-2.50)	1.60 (1.04-2.47)	2.34 (1.43-3.83)	1.96 (1.21-3.17)
Established Developmental Risk Factors						
Family history	1.88[c] (1.36-2.61)	1.71[c] (1.23-2.39)	. . .	. . .	1.74[d] (1.28-2.38)	1.49[d] (1.09-2.03)
Birth weight	0.72 (0.54-0.96)	0.78 (0.59-1.04)	0.74 (0.57-0.95)	0.78 (0.61-1.01)	1.16 (0.86-1.56)	0.91 (0.68-1.21)
Childhood BMI	1.07 (0.91-1.27)	1.10 (0.93-1.29)	1.12 (0.97-1.30)	1.13 (0.99-1.30)	1.58 (1.41-1.78)	1.53 (1.35-1.73)

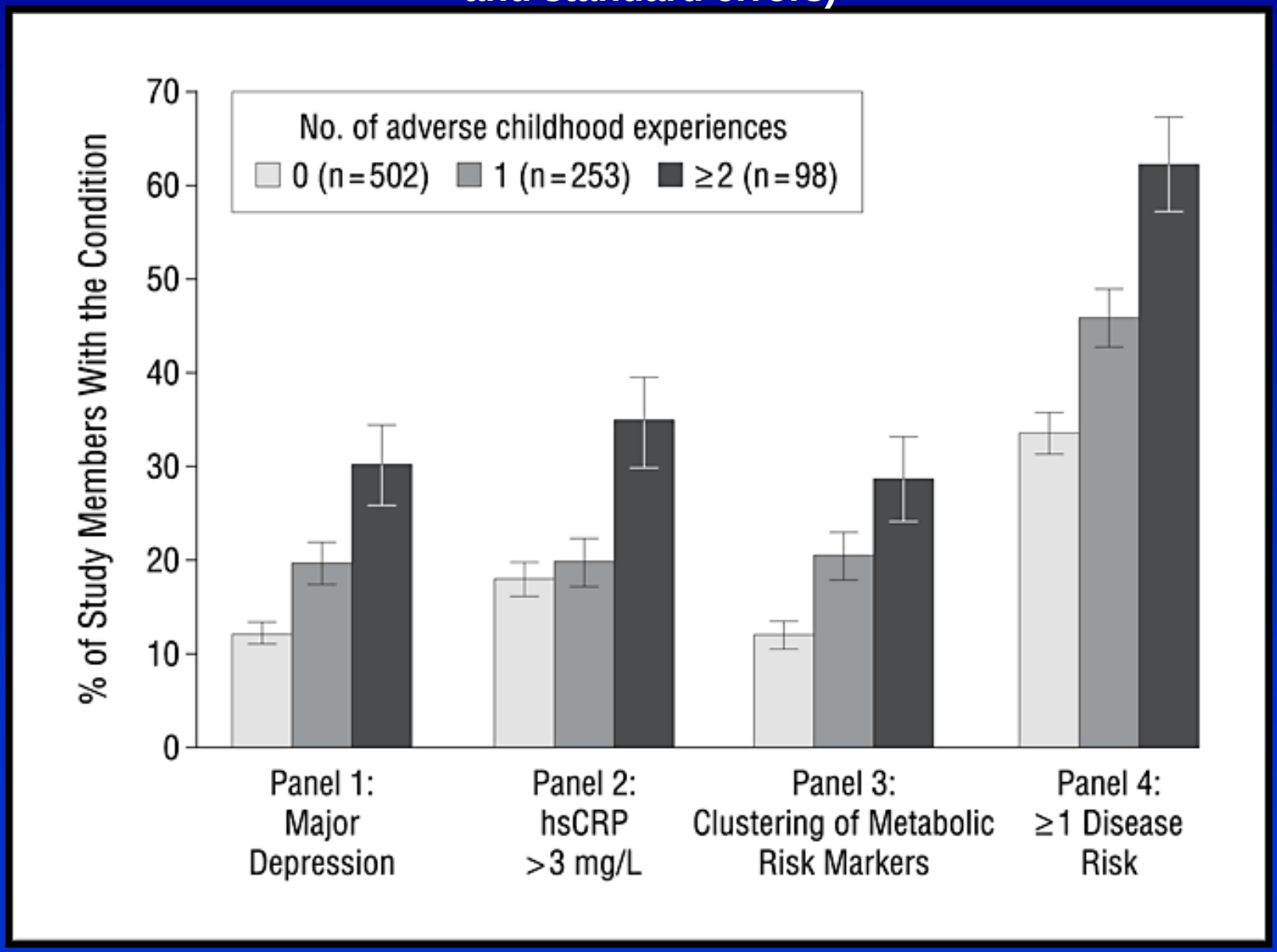
Danese, A. et al. *Arch Pediatr Adolesc Med,* 2009;163:1135-1143.

Prediction of number of age-related-disease risks in adults with different levels of exposure to adverse childhood experiences and established risk factors

	Incidence Rate Ratio (95% CI)[a]			
	No. of Age-Related-Disease Risks at Age 32 y[b]			
	Panel 1: Bivariate Analysis	Panel 2: Adverse Childhood Experiences Model	Panel 3: Developmental Risks Model	Panel 4: Life-Course Model
Adverse Childhood Experiences				
Childhood SES				
High	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Average	1.33 (0.99-1.80)	1.36 (1.00-1.85)	1.38 (1.02-1.88)	1.36 (1.00-1.86)
Low	1.89 (1.36-2.62)	1.66 (1.19-2.33)	1.60 (1.14-2.26)	1.55 (1.09-2.21)
Childhood maltreatment				
No	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Probable	1.37 (1.11-1.69)	1.28 (1.03-1.59)	1.27 (1.02-1.57)	1.26 (1.01-1.56)
Definite	1.81 (1.38-2.38)	1.59 (1.19-2.11)	1.50 (1.12-2.01)	1.55 (1.15-2.08)
Childhood social isolation				
Very low	1 [Reference]	1 [Reference]	1 [Reference]	1 [Reference]
Low	1.25 (0.91-1.71)	1.24 (0.91-1.71)	1.26 (0.92-1.72)	1.26 (0.92-1.73)
High	1.35 (0.99-1.84)	1.31 (0.96-1.79)	1.27 (0.93-1.74)	1.29 (0.94-1.76)
Very high	1.87 (1.38-2.51)	1.73 (1.27-2.34)	1.66 (1.22-2.25)	1.63 (1.20-2.21)
Established Developmental Risk Factors				
Family history of depression	1.33 (1.09-1.62)	...	1.25 (1.02-1.53)	1.23 (1.00-1.50)
Family history of CV disease	1.30 (1.05-1.60)	...	1.24 (1.00-1.54)	1.24 (0.99-1.54)
Birth weight	0.81 (0.68-0.97)	...	0.78 (0.65-0.94)	0.79 (0.66-0.95)
Childhood BMI	1.22 (1.10-1.35)	...	1.22 (1.10-1.35)	1.22 (1.10-1.35)
Established Concurrent Risk Factors				
Adult SES	0.79 (0.69-0.91)	...	...	0.88 (0.75-1.02)
Adult smoking	1.05 (0.96-1.16)	...	...	0.96 (0.87-1.07)
Adult physical activity	0.94 (0.86-1.02)	...	...	0.94 (0.86-1.03)
Adult diet	0.99 (0.90-1.08)	...	...	1.00 (0.91-1.11)

Danese, A. et al. *Arch Pediatr Adolesc Med,* 2009;163:1135-1143.

Distribution of mean (SD) age-related-disease risks at age 32 years with different levels of exposure to adverse childhood experiences (percentages and standard errors)



Danese, A. et al. *Arch Pediatr Adolesc Med* , 2009;163:1135-1143.

Take home messages

- The enduring consequences of adverse childhood experiences were not explained by established developmental or concurrent risk factors.

- Four important findings are apparent:
  - First, our results indicate that groups of children exposed to different adverse experiences do not necessarily overlap.

    This suggests that different interventions are needed to tackle each adverse childhood experience.

- **Second, our results indicate that children exposed to a greater number of adverse experiences have a greater number of age-related disease risks in adult life.**

  The cumulative effect of adverse childhood experiences points to new opportunities for disease prevention.

- **Third, our results indicate that children exposed to adverse psychosocial experiences have enduring abnormalities in multiple biological systems.**

  Although some specificity was observed (e.g. SES does not predict depression), the overall picture was that adverse childhood experiences may simultaneously affect nervous, immune and endocrine/metabolic functioning in adulthood.

- **Fourth, our results indicate that children exposed to adverse experiences are more likely to have age-related disease risks in adult life, regardless of their family liability for disease, birth weight, childhood weight, and adult SES and health behaviours.**

Modifying established risk factors is unlikely to wholly mitigate the economic health burden associated with adverse childhood experiences.

Promoting healthy psychosocial experiences for children may be necessary to improve the quality of longer lives and reduce health care costs across the lifecourse.

The New Yorker, Mar 21, 2011

A REPORTER AT LARGE

THE POVERTY CLINIC

Can a stressful childhood make you a sick adult?

BY PAUL TOUGH

Nadine Burke at her San Francisco clinic. Photograph by Alessandra Sanguinetti.

The Longitudinal Study: A Bridge to the Future

John F. McDermott, M.D.

Our *Journal* like most scientific journals, has a dual function: to develop a knowledge base for the field and to provide the latest information for clinical practice. For years, there was little connection between the two. Research was dominated by cross-sectional, retrospective studies of highly selected clinical cases, with all the distortions inherent in that method. Then, in the 1980s, came our own "Framingham studies," prospective longitudinal studies of community-based samples. They years—from infancy into preschool, from preadolescence through adolescence—and into adulthood. The evaluations were labor-intensive, emphasizing face-to-face standardized assessments supplemented by parent and teacher reports. (The Dunedin Study group was also evaluated at ages 26 and 32. The next assessment is planned for age 35.)

Although the study was correlational, these factors, and the high retention rate (well over 90%), strengthened the potential for causal inferences. As

Journal of the American Academy of Child & Adolescent Psychiatry, 2011, 50(4): 317-319.

These studies came not just from the United States, but also from around the world. One of the most unique, one that separated it from the others, was the Dunedin Multidisciplinary Health and Development Study from the University of Otago Medical School in New Zealand. The investigators

I used to think that epidemiology would draw us a much-needed map of childhood disorder, but there is much more to it than that.

Projects like the Dunedin Study are our "Global Positioning System" for clinical practice in the future. &

Mental Health Commission of Canada

Commission de la santé mentale du Canada

Making the Case for Investing in Mental Health in Canada

1 in 5 people in Canada lives with a mental illness each year

THE JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY

ACAMH

Journal of Child Psychology and Psychiatry 54:7 (2013), pp 791–799

doi:10.1111/jcpp.12062

Diagnostic transitions from childhood to adolescence to early adulthood

William E. Copeland,[1] Carol E. Adair,[2, 3] Paul Smetanin,[4] David Stiff,[4] Carla Briante,[5] Ian Colman,[6] David Fergusson,[7] John Horwood,[7] Richie Poulton,[8] E. Jane Costello,[1] and Adrian Angold[1]

[1]Psychiatry and Behavioral Sciences, Duke University, Durham, NC, USA; [2]Mental Health Commission of Canada, Calgary, AB, Canada; [3]Departments of Psychiatry and of Community Health Sciences, University of Calgary, Calgary, AB, Canada; [4]Risk Analytica, North York Corporate Centre, Toronto, ON, Canada; [5]Southwest College of Naturopathic Medicine, Tempe, AZ, USA; [6]Epidemiology and Community Medicine, University of Ottawa, Edmonton, AB, Canada; [7]Psychological Medicine, Christchurch School of Medicine and Health Sciences, University of Otago, Christchurch; [8]Preventive and Social Medicine, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand

Conclusions: Having a disorder in childhood or adolescence is a potent risk factor for a range of psychiatric problems later in development. These findings provide further support for prevention and early life intervention efforts and suggest that treatment at younger ages, while justified in its own right, may also have potential to reduce the risk for disorders later in development.

Research

JAMA Psychiatry. 2014;71(2):119-127. doi:10.1001/jamapsychiatry.2013.2803
Published online December 4, 2013.

Original Investigation

Suicide Attempt in Young People
A Signal for Long-term Health Care and Social Needs

Sidra J. Goldman-Mellor, PhD; Avshalom Caspi, PhD; HonaLee Harrington, BA; Sean Hogan, MSW; Shyamala Nada-Raja, PhD; Richie Poulton, PhD; Terrie E. Moffitt, PhD

RESULTS As adults approaching midlife, young suicide attempters were significantly more likely to have persistent mental health problems (eg, depression, substance dependence, and additional suicide attempts) compared with nonattempters. They were also more likely to have physical health problems (eg, metabolic syndrome and elevated inflammation). They engaged in more violence (eg, violent crime and intimate partner abuse) and needed more social support (eg, long-term welfare receipt and unemployment). Furthermore, they reported being lonelier and less satisfied with their lives. These associations remained after adjustment for youth psychiatric diagnoses and social class.

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Effective strategies for suicide prevention in New Zealand: a review of the evidence

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Abstract

A national suicide prevention strategy for New Zealand was developed in 2006. There is relatively little strong evidence for the efficacy of many existing suicide prevention initiatives, and this area has frequently been captured by strong claims about the effectiveness of programmes that have not been adequately evaluated. This paper provides a conceptual framework for classifying suicide prevention initiatives, reviews evidence for their effectiveness, and makes recommendations for initiatives to be undertaken as part of suicide prevention activities in New Zealand.

The available evidence thus far suggests that the most promising interventions likely to be effective in reducing suicidal behaviours are medical practitioner and gatekeeper education, and restriction of access to lethal means of suicide. This evidence also suggests a clear agenda for research, which includes evaluating interventions and prevention programmes, developing model and demonstration projects, identifying meaningful outcome measures, and refining and identifying the critical elements of effective programmes.

OFFICE OF THE PRIME MINISTER'S SCIENCE ADVISORY COMMITTEE

The role of evidence in policy formation and implementation

A report from the Prime Minister's Chief Science Advisor

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