The Galton Institute

NEWSLETTER

Galtonia candicans Issue Number 69 December 2008

Cannabis and Psychosis: The Environment Trigger
by
Benjamin Capps and David Nutt

(Centre for Ethics in Medicine and Henry Wellcome L.I.N.E., University of Bristol)

Introduction

This paper addresses the ethical issues that arise from evidence that suggests that some of the negative health effects of cannabis use, in particular psychosis, may be predicted by genetic polymorphisms. We attempt to balance a 'genetic determinist' view of disease (e.g. a 'gene for psychosis') with the concurrent evidence that the phenotypic expression of a gene, such as adult-onset psychosis, may require an environmental trigger. Our primary aim is to offer tentative suggestions as to how the 'post-genomic era' – a period in which society is inundated with genetic data and novel applications of gene modification, screening, and selection – may influence drug policies in the UK, specifically with regard to cannabis use. In light of our emergence from the Genomic Revolution, it is appropriate that society considers the ethico-social implications of the 'nature-nurture' debate in this context.[1]

Cannabis and Psychosis

Genotype has long been associated with various mental health disorders, and with various mental health disorders, and there has been much speculation that there exist 'genes for...' many types of mental illness.[2] However, as the evidence mounts which suggests gene-environment interactions in human development – particularly in the effects of exposure to an environmental pathogen on a person's health is conditional upon her genotype[3] – emphasis has switched from discovering the Medelian 'units' that *cause phenotypes*, to the understanding of the complex relationship between genes and environment.[4]

There is growing evidence to suggest that there is a gene-environment interaction between drug-taking and mental health, that includes known environmental pathogens and risk factors to specific substance-use disorders, and a heterogeneity in responses to these specific causes.[5] One such drug is cannabis, whose use is associated with psychosis: a disorder that is categorised as conditions in which a person's ability to test reality is impaired.[6] A relationship between psychosis, in particular schizophrenia, and cannabis has been suggested for some time see[6] but only recently have Caspi *et al* claimed evidence for a gene-environment interaction in the effects of adolescent-onset cannabis use on adult psychosis.[7] They present data that suggest that cannabis-induced psychosis has a genetic component (a functional polymorphism of the catechol-0-methyltransferase (COMT) gene), thus strengthening the view that gene-hunters can locate gene-to-disorder connections, even for multifactoral disorders, which minimally represent a risk factor for asymptomatic carriers. Others have reported a similar

Contents

Milo Keynes

I am sad to have to announce that Milo Keynes (our current Newsletter Editor) suffered a stroke on August 19th, 2008. After a difficult period he is now back in active rehabilitation and we all wish him a good and speedy recovery from here.

David Galton

link between COMT genotype and psychotic symptoms, but as of yet the exact nature of the gene-environment and gene-gene relationship between psychosis and cannabis use remains unresolved, despite a growing number of other human and animal studies.[8]

Importantly, Caspi *et al* argue that such a connection may only be apparent among individuals in a sample that has been exposed to specific non-genetic risks: specifically adolescent-onset cannabis use. They do not rule out other possible environmental risks and pathogens; and they conclude: 'Our findings suggest that a rôle of some susceptibility genes may be to influence response to these pathogenic environments'.[9] This supports a (not uncontroversial)[10] view that 'Cannabis use... is a component cause, [but also] part of a complex constellation of factors leading to psychosis'.[11] Thus, it is proposed that the risk of developing psychosis is higher in those who are 'genetically vulnerable' to the disease and are exposed at some time to cannabis, but the aetiology and medical link is still difficult to specify. This may include (or an absence of) a family history of, or genetic predisposition to psychosis, and a personal history of unusual 'triggering' (environmental) experiences.

The Regulation of Neuropharmaceuticals and Cannabis

Cannabis is a psychoactive substance (PS), and is in the class of neuropharmaceuticals (pharmacological agents whose primary mode of action is through altering the metabolism of neurotransmitters) many of which are widely used in medicine and for recreational purposes. It is primarily classified as a hallucinogen; its active ingredient is delta-9-tetrahydrocannabinol; and it is normally smoked or ingested. Cannabis is a widely used drug[12] that is currently categorised in the UK as illicit though in a lower class [C] than other drugs such as heroin and cocaine [class A].[13]

Neuropharmaceuticals are often referred to as 'drugs', and tend to be either strictly controlled as *medicines*, or when then fall outside this 'legitimate' use, classified as illicit, and therefore out of bounds for *recreational use*. When PSs are used outside the medical setting, they are seen as being 'misused' on the grounds that they can be categorically regarded as 'harmful' to the user or others around him, and intrinsically related to poverty, mental health and illness, and criminality. There is overwhelming evidence that widespread use of many drugs has massive economic and social costs; however, the link to drug misuse is not always straightforward, and furthermore, it is often not helpful to describe different 'drugs' under a unified heading, since they often have very different pharmacological effects, social acceptability, long and short term psychological effects, medical uses, and legal status.[14]

The health-recreation distinction has significant socio-political results. For example, while medicines used as part of therapy are considered as an acceptable medical intervention if properly prescribed (on the not unproblematic grounds of evidence-based medicine),[15] recreational drug use is often disproportionately associated with social problems and crime,[16] global instability,[17] and the 'global burden of disease'.[18] The comorbid effects of many drugs[19] therefore become a justification for paternalistic government intervention.[20] However, this distinction is not as clear-cut as some legal and social norms would indicate. Most drugs, whether used in medicine or for recreational purposes, have side effects. When used in therapy, these unwanted (and unavoidable) effects are tolerated as a cost of curing, treating or elevating the primary condition. This is despite the large numbers of deaths caused by adverse drug reactions, and the lack of effectiveness for many patients of particular prescribed drugs.[21] This cost-analysis of health benefits is not evident in the recreational use of drugs, since the

autonomous decision to experience a certain mental state, despite the potential physical, psychological, legal and social costs, is a 'voluntary' action (as opposed to the burden of disease that compels drug use on patients). However, many drugs used in medicine have a hedonistic value for recreational users (and evidence suggests that cannabis may also have a medicinal value for some patients), and the therapeutic advantage is replaced by the attainment of 'out of it' experiences. Thus, the legal distinction is one of medical indication (regulated by the Medicines and Healthcare Products Regulatory Agency) *and* the socio-historical context of specific drugs. Thus, understanding the circumstances of drug (mis)use is part of a wider study of the social construction *and* biochemical investigation of the effects of particular drugs.[22]

There is evidence that cannabis has medicinal benefits to offer, such as controlling some forms of pain, alleviating nausea and vomiting due to chemotherapy, treating wasting due to AIDS, and combating muscle spasms associated with multiple sclerosis. However, current research has not proved persuasive to allow licensed use of cannabis, for example as an analgesic.[23] A negative political view of cannabis as a recreational drug remains a dominant political force, because of the suggestions that its use increases the use of other illicit drugs or that it is a 'gateway' drug, encouraging uptake of other drug use,[24] though the evidence for this is weak.[13] The opposition to recognise cannabis as a medical treatment seems to be concerns as to potential side effects, such as hallucinations, delusions and clinically significant schizophrenia,[25] and that there are medical alternatives, for example, for pain management.[26]

Cannabis Use in the Post-Genomic Era

We now live in what some call the 'Post-genome era'. Data from the human genome project, and potentially large amounts of raw epidemiological

data stemming from projects such as UK Biobank, provide society with a unprecedented understanding of human inheritance, difference and diseases; and this potentially gives rise to novel genetic 'tests' and escalating lists of diagnosable diseases and risk predispositions. In regard to medicinal drug use, pharmacogenetics (the study of specific genes in the context of the hereditary basis of person-to-person variations in drug response) and pharmacogenomics (the response of the entire genome to drugs; although such terms are often used interchangeably)[27] raise the prospect of tailoring prescriptions to our individual genetic differences. In this way, therapies may be offered which can maximise beneficial effects while minimising side effects and adverse reactions. But this also means that drug users may be able to experience the hedonistic effects of recreational drugs, impervious to many of the risks; so for example, those without a genetic 'predisposition' to addiction may be able switch on and switch off drug use without succumbing to any dependency.

How does the evidence that some people are genetically vulnerable to some of the major deleterious effects of cannabis affect these perspectives? Specifically, how will the possibility that it may be possible to screen individuals before they use cannabis – for medicinal or recreational purposes – and provide them with information of the *risks* for specific health aspects as a direct result of use and for future health problems affect the use and regulation of cannabis? Will we see increased calls for cannabis to be prescribed in therapy? And will the pro-recreational use of cannabis be more forcefully argued?

In light of this growing evidence of genetic susceptibility, understanding the genetic and biochemical aspects of drug use in the post genomic era will have to include an assessment of the social and historical context of use. In this way, we may be able to predict and evaluate the potential legal and social changes that may become integral to perspectives of drug (mis)use and the prediction, prevention and treatment of non-commutable diseases. This paper focuses on cannabis-induced psychosis, which, like many other diseases, has a complex multi-factorial aetiology, involving genetic factors ('genes for…'), non-genetic risk factors (unproven causal predictors) and environmental pathogens (proven causes).[28]

Cannabis and Predictive Genomic Medicine

'Predictive genomic medicine' refers to a branch of 'genomic medicine' which proposes screening healthy individuals to identify those who carry alleles that increase their susceptibility to certain diseases.[29] The goals are to provide early warnings to individuals that certain behaviours may trigger 'disease' genes, and to offer preventative 'treatments' (e.g. gene therapy) or 'avoidance behaviours' that may reduce the likelihood of developing a certain disease in the future. The relationships between genes, behaviour and risk are complex. So when a gene-to-disorder connection is suggested, a careful appraisal of the scientific evidence and the role of the environment and behavioural triggers should be carefully considered.

In the Caspi *et al* report, the authors emphasised the limitations of the study in determining the precise nature of a possible 'psychosis gene' in the context of exposure to environmental pathogens. Thus, a psychosis gene may only be activated if (a) cannabis (or some other psychosis 'causing' drug) is taken; and (b) it is taken at a particular time (e.g. adolescence) or under certain environmental conditions (e.g. the 'threatening' environment associated with illegal drug-market cultures). Psychosis may therefore never be actuated in a 'carrier', despite the presence of a certain gene, if either (a) or (b) are not concurrently present. In such circumstances, it therefore seems incoherent to refer to the presence of a particular genetic variant as a 'gene for…'.

So, should carriers of this variant be told of their risk of developing a psychotic condition if they are exposed to cannabis? The first case may be if cannabis is offered as a therapeutic treatment. Here, patients with a specific genotype that predisposes one to psychosis may be considered as 'at risk' to the deleterious effects of cannabis, and therefore alternatives used instead. The evidence presented in the Caspi *et al* report therefore might suggest policies to rule out the prescription of cannabis, for example, to adolescents.

The problem is, however, that Caspi *et al* present evidence of only one possible path to psychosis. Psychosis is undoubtedly a risk for those without a particular (identified) phenotype and for those who never come into contact with cannabis. Environmental causes may only be contributory to an illness, because exposure to them may not always generate a disorder. In the Caspi study the majority of adolescents exposed to cannabis, even in the val/val group did not develop schizophrenia. Furthermore, there may be (unknown) polygenetic interactions between a number of genes or a number of environmental triggers for psychosis. The heterogeneity associated with a certain illness may suggest a genetic component, but this is also associated with individual differences in temperament, personality, cognition and autonomic physiology. These all may (or may not) also be influenced by genetic and environmental factors.

This raises the specific problem of understanding the nature of 'genetic risk' and 'genetic predispositions', and the feasibility of screening for certain genes. A 'risk' is just that: in this specific case, Caspi et al. suspected that the presence of a specific COMT gene and the adolescent-onset of cannabis use establish a risk to developing psychosis in adulthood. But they realised that this risk was likely to be dependent on a

complex genetic-environment relationship. At present, our knowledge about genes and their roles, and their interplay with environment triggers, is far from sufficient to make reliable predictions about an individual's risk of developing a disease. But does this mean that a policy of screening for suspected alleles for specific diseases is unwarranted? The concern is that a policy of screening would over-emphasise the (cannabis)-gene-psychosis connection, thus leading to neglect for other possible causes of psychosis, specifically the socio-environmental triggers of poor mental health, such as poverty and social deprivation.

There are also questions over the usefulness of such screening programmes. As we have already stated, screening for a 'gene for…' will be futile if the individual is never exposed to the necessary drug or environment trigger. Furthermore, there may be many more environmental pathogens which may affect the user in unforeseen ways, and represent further health risk factors associated to the deleterious effects of cannabis use. For example, smoking cannabis raises risks of tobacco related risks such as lung cancer (and other smoking related illnesses) and addiction, both of which themselves may have a genetic component.[30] Furthermore, other social cost factors should also be considered in assessing the risks of cannabis use, such as those that are related to intoxicated behaviour, for example, an impaired driving ability.[31] Thus, would screening for a specific gene lead to real benefits if it encouraged those with the "safe" genotype to consume more of the drug so putting themselves in the way of more harms? 'Treatments' for genetic phenotypes are probably some way off, such as gene therapy, and therefore abstinence and avoidance of triggers is the only course of action.

With limited resources already a concern for the health service, predictive screening of this kind would likely divert further funds away from proven or more effectual interventions, such as drug education and awareness programmes. However, the usefulness of genomic screening becomes correlatively important as the number of suspected triggers and the prevalence of a particular illness increases; communities in which there is a prevalence of triggered psychosis may benefit from such screening, as may the triaging of genetic screening of those considered to be vulnerable, for example, on the basis of family histories.[32] Thus, uncertainty in predictive screening should not rule out the potential benefits of conveying the relevant genetic risk of developing, and triggers for, a given disease to an individual or group, so that they may avoid specific behaviours and thus minimise the risk of the onset of a disease by this specific trigger.

Individual Choices and Drug Misuse

One of the central considerations in the ethical analysis of drug use is that of autonomy. Discussions on 'autonomy' can lead to confusion because of heterogeneity of understanding in the term. In this regard, autonomy does not stand in isolation (as an absolute right to do as one wills), but a reasonable claim that one should have minimal access to the goods that allow chosen purposes to be reached. This cannot be achieved in isolation, and requires an other-regarding commitment in the interests of socio-political stability.[33] From this starting point, we can clarify certain aspects of the genetic-environment debate.

First of all, it should be noted that drugs are a standing challenge to the conceptual and empirical basis of autonomy.[34] Psychoactive drug use has a direct effect on our ability to be autonomous. Ashcroft argues, that tobacco, for example, may be used by some smokers to enhance their sense of autonomy through assisting in focussing their attention, whereas addiction to tobacco smoking is a very visible mark of the limits of the autonomous will's ability to act on itself. Cannabis, on the other hand, is often used precisely to disinhibit the will and to get 'out of it', as a mark of our dissatisfaction with the nagging demands of autonomy. Ashcroft concludes that 'at a deeper level, this curiosity about the relation between drugs and autonomy can lead to a whole series of worries about the extent to which our human capacities of rationality and choice are malleable to (if not determined by) physical interventions in our physical being, the ways in which third parties may for various reasons want to weaken or alter our physical or social bodies, and the ways our present choices may bind our future selves'.[35]

Drug misuse is therefore dependent in part on *motivation* and the factors which drive an individual to embark upon a particular project. Specifically, we are concerned with the motivation that leads one to misuse a certain drug, and whether an explanation regarding genetic risks will cause a positive change in lifestyle or an avoidance of certain triggers. The reasons for drug misuse will be a complex relationship between personal situation (including financial status and pre-existing mental health illness) and environmental circumstance (e.g. peer pressure or the availability of specific drugs). Thus, drug misuse may not simply be a matter of a choice made at a particular time, and will be affected by the personal assessment of the complex relationships between previous (and prospective) actions, decisions, and experiences. Furthermore, experimenting with drugs (i.e. limited misuse) and habitual use (caused by addiction and craving) will likewise be strongly effectual on later choices. These temporal-specific 'choices' will therefore often dictate the health effects of specific drugs.[36] For example, a one-off use of cannabis in adulthood may be insufficient to trigger psychosis, unlike adolescent use, or prolonged or chronic use.

However, knowledge of genetic

predispositions, whether to an illness possibly triggered by cannabis use (e.g. psychosis), an illness directly related to drug use (e.g. addiction), or an illness indirectly associated to drug use (e.g. lung cancer), may influence or change the user's choices: in this case, to avoid or abstain from cannabis use. Thus genetic information may be used to prevent cannabis use, and thereby reduce the prevalence of drug-induced psychosis. For example, in a Dutch cohort, it was estimated that lack of exposure to cannabis would have reduced the incidence of psychosis requiring treatment by as much as 50%,[37] and is similarly reflected in a Swedish cohort, showing that the use of cannabis increased the risk of schizophrenia by 30%.[38] However, evidence also suggests that an increase in current cannabis use has only had a marginal effect on increasing the reported cases of drug-induced psychosis.[39]

However, there is already evidence that inappropriate communication of genetic predispositions, triggers and risks may lead to negative behaviour with regard to health. For example, being told that one has a 'gene for addiction' may lead to demoralisation with regards to stopping smoking, or that the absence of the same 'gene' may lead to a false belief of being impervious to the negative effects of tobacco.[40] (Again, what is this 'gene for…' if the carrier is never exposed to an addiction-triggering drug?). Of particular concern for society will be if the use of such information contributes to an increase in cannabis use, and a correlative increase in, for example, road traffic accidents, and encourages social problems associated with drug misuse. Any information with regard to the presence of a 'disease' gene should be carefully communicated, since its presence may lead to raised anxiety and reduced self-confidence despite there only being a risk of susceptibility. Past or current users may become anxious if they have the gene; for some this may be the motivation necessary to make them stop, for others it may lead to resignation to their 'genetic future'. The degree of anxiety will be partially dependent on a distinction between the presence of low risk disease predispositions which represent phenotypes dependent on many related factors, and 'mendelian' diseases which represent genetic profiles which are strongly predictive, such as the gene associated with Huntington's disease; in the case of the latter, counselling is important to avoid fatalistic tendencies and suicidal acts.[41]

Concentrating on the genetic causes of disease avoids dealing with social problems that may trigger certain conditions (which are amenable to genetic screening). Caspi *et al* recognise the association that may or may not be present between psychosis and environmental triggers: they specifically mention the possible psychosis-exaggerating effect of illicit lifestyles. Such lifestyles often (but not always) go hand in hand with poverty and low social status. Genomic medicine therefore risks targeting at-risk individuals or communities, rather than addressing the criminal and social environments that may trigger (or lower the risk-threshold of) psychosis.

Conclusions: Changing Policies?

How will the genomic era affect cannabis policies in the UK? In the physician-patient relationship there will normally be a acceptable degree of paternalism-led choice: that the patient has presented themselves for treatment, and therefore may be prescribed drugs under the advice of her doctor or presiding medical professional. In this case, the use of genomic medicine becomes an issue of physician-mediated 'best interests'; that if cannabis would be appropriate as a therapy (based on evidence which may or may not be available in the current literature), then it may be offered. But, if a predisposition were detected, then alternatives would be more suitable. Thus, 'genes for…' become targets for avoiding certain medical interventions (minimising the 'risk'); and specifically, thus straightforwardly avoiding the environment pathogen that may cause the onset of psychosis.

This simple case however, belies the complexity of the social implications for genetic-environment research. As mentioned above, genetic information raises the possibility of both increasing one's autonomy, by enabling individuals to make better-informed choices, while also leading to further restrictions on individual liberty. Should tests become available for 'genes for psychosis' we will have to contend with the issues of whether such tests should be routinely offered. The current debate about cannabis-triggered psychosis illustrates many of the issues society will have to deal with as gene-illness associations become more and more common. We have already seen that should cannabis be offered in the medical setting, then offering (or even mandating) 'risk of psychosis' screening may be beneficial for patients and in *their* interests. However, the mandated testing for drug misuse and concurrent screens for 'disease genes' may be used to dictate medical provision in light of scarce resources: for example clinical decisions may become prejudiced by positive results, especially if an undue weight is placed on taking responsibility for past choices.

As cannabis is categorised as an illicit drug this complicates the current debate considerably. We do not here want to engage in whether current drug – and specifically cannabis – policies are effective[42] or coherent.[43] Our concern is what role genomic information may have in the ethical debate. While cannabis possession remains an 'arrestable offence', there seems little of benefit in offering routine 'psychosis' screening; users of cannabis could simply open themselves to criminal sanctions and the stigmatisation of 'drug misuse'. The message is therefore if one makes choices that transgress into illegal action, then one must also accept the risks, including possible health costs. Of course, this

means that society picks up the cost when users present themselves for treatment (as well as the cost of policing policies). Such logic equally applies to screening specific populations. The paper by Caspi *et al* specifically addresses the issue of adolescent-onset of cannabis use and the risk of psychosis in later life. But again, logic suggests that adolescents should be encouraged to avoid cannabis use because of the criminal risks, rather than provide potentially burdensome (or encouraging) genetic information.

If cannabis use policies continue to be liberalised (again, we do not here question issues of ideology, and believe genetic information has no role in this debate) – and personal use became acceptable – then genetic information regarding predispositions and risk would enable users (and potential users) to make informed choices. A possible increase in the legal use of cannabis may lead to an increase in incidents of psychosis, or drug related economic and social costs. We may therefore also see a proportional increase in programmes of screening and testing, and more innovative and advanced detection measures, resulting in stricter control measures to dissuade inappropriate and risky use. For some, this represents an unacceptable intrusion into personal choices and individual interests.[44] With regard to drug-related illness, enabling individuals to avoid such risks may prove to be beneficial for some carriers of the 'risk' gene. The use of this information by them therefore becomes a matter of personal responsibility, and questions whether individuals should knowingly expose themselves to risks when they have such information? The choice that an individual makes in this regard is interwoven with debates surrounding freewill and agency; which themselves are bound by the debates over the possible genetic basis of behaviour, and its relationship between explanation and justification of behaviour. In this regard, the contrast can be clearly seen between the debates concerning the causes and explanations of drug addiction as either a 'disease of the will' or a 'pathology of choice'.[45]

The 'choice', therefore, is not necessarily an easy one to make, since avoiding the 'wrong choice' could be impossible for some depending on a genetic vulnerability and exposure to certain environment triggers or pathogens found in areas of social deprivation, poverty and socially excluded communities. Avoiding risks may therefore be an issue for society to solve, by (the difficult task of) removing the social environments that encourage drug misuse. 'Reintegration' programmes are more likely to succeed through providing housing, education, employment opportunities, and family or social support, rather then genetic screening.

The evidence suggests, therefore, that we are no closer to a settled position with regard to acceptable cannabis use, not least because the distinction between legitimate therapeutic drug use and illicit recreational misuse, in the context of the relationship between genetics, environmental triggers, individual motivations, and social situation, remains unclear. There still remains the enduring question of the role of nature and nurture on our personal and social development. We now have at our disposal unprecedented information regarding the genetic basis of agency (i.e. the genes that dispose us to particular paths, such as the onset of diseases, character traits, risks and predispositions) and the myriad of environmental factors that contribute, limit and shape our lives. We still do not know many aspects of the genetic basis of disease and how environment contributes to a particular pathology, let alone how each influences the other; specifically, we are unable to determine the extent to which some genetic phenotypes are dependent and conditional on exposure to an environmental risk or pathogen. The paper by Caspi *et al* is a contribution to this debate, but it fails to resolve the matter in either direction. Eventually determining the exact nature of the genetic-environmental interaction in drug use and its consequences will require a multidisciplinary approach, including fundamental input from neuroscience, biochemistry, social study and genetic epidemiology.

Endnotes:
[1]Merikangas, K. and Risch, N. 2003. Will the Genomics Revolution Revolutionize Psychiatry? *American Journal of Psychiatry* 160:625-635.
[2]Kendler, K. 2005. 'A Gene For...': The Nature of Gene Action in Psychiatric Disorders. *American Journal of Psychiatry* 162: 1243-1252.
[3]Rutter, M. and Silberg, J. 2002. Gene-Environment Interplay in Relation to Emotional and Behavioral Disturbance. *Annual Review of Psychology* 53: 463-490.
[4]Insel, T. and Collins, F. 2003. Psychiatry in the Genomics Era. *American Journal of Psychiatry* 160: 616-620; Hamer, D. 2002. Rethinking Behavior Genetics. *Science* 298: 71-72.
[5]Moffitt, T., Caspi, A. and Rutter, M. 2005. Strategy for Investigating Interactions Between Measured Genes and Measured Environments. *Archives of Genetics and Psychiatry* 62: 473-481.
[6]Fergusson, D., Horwood, L. and Ridder, E. 2005. Tests of Causal Linkages Between Cannabis Use and Psychotic Symptoms. *Addiction* 100: 354-366; Hall, W, and Degenhardt, L. 2000. Cannabis and Psychosis: A Review of Clinical and Epidemiological Evidence. *Australian and New Zealand Journal of*

Psychiatry 34: 26-34; McKay, D. and Tennant C. 2000. Is The Grass Greener? The Link Between Cannabis and Psychosis. *Australian Medical Journal* 172: 284-286; Thirthalli, J. and Benegal, V. 2006. Psychosis Among Substance Users. *Current Opinion in Psychiatry* 19: 239-245.

[7]Caspi, A, Moffitt, T., Cannon, M., McClay, J., Murray, R., Harrington, H., Taylor, A., Arseneault, L., Williams, B., Braithwaite, A., Poulton, R., and Craig, I. 2005. Moderation of the Effect of Adolescent-Onset Cannabis Use on Adult Psychosis by a Functional Polymorphism in the Catechol-O-methyltransferase Gene: Longitudinal Evidence of a Gene X Environment Interaction. *Biological Psychiatry* 57: 1117-27.

[8]Hall, W. 1008. Cannabis and Psychosis. *Drug and Alcohol Review* 17: 433–444; Henquet, C., Rosa, A., Krabbendam, L., Papiol, S., Fananas, L., Drukker, M., Ramaekers, J. and van Os, J. 2006. An Experimental Study of Catechol-O-Methyltransferase Val(158)Met Moderation of Delta-9-Tetrahydrocannabinol-Induced Effects on Psychosis and Cognition. *Neuropsychopharmacology* Aug 23; Advance Online Publication doi: 10.1038/sj.npp.1301197.

[9]Caspi et al. *op. cit.* note 7, p. 1126.

[10]For example: 'Available evidence does not strongly support an important causal relation between cannabis use and psychosocial harm, but cannot exclude the hypothesis that such a relation exists'; MacLeod, J., Oakes, R., Copello, A., Crome, I., Egger, M., Hickman, M., Oppenkowski, T., Stokes-Lampard, H. and Smith, G. 2004. The Psychosocial Consequences of Use of Cannabis and Other Illicit Drugs: Systematic Review of Longitudinal, General Population Studies. *Lancet* 363**:** 1579–1588; and: 'Antecedent cannabis use appears to act as a risk factor in the onset of schizophrenia, especially in vulnerable people, but also in people without prior history'; Smit, F., Bolier, L. and Cuijpers, P. 2004. Cannabis Use and the Risk of Later Schizophrenia: A Review. *Addiction* 99: 425–430.

[11]Arseneault, L., Cannon, M., Witton, J. and Murray, R. 2004. Cannabis as a Potential Causal Factor in Schizophrenia. In: Castle, D. and Murray, R. eds. *Marijuana and Madness: Psychiatry and Neurobiology*. Cambridge. Cambridge University Press. pp. 101–118.

[12]Miller, P. and Plant, M. 2002. Heavy Cannabis Use Among UK Teenagers: An Exploration. *Drug and Alcohol Dependence* 65: 235-242; World Health Organisation. 1997. *Cannabis: A Health Perspective*. Programme on Substance Abuse, Division of Mental Health and Prevention of Substance Abuse. Geneva. World Health Organisation. Report no. WHO/MSA/PSA/97.4.

[13]Misuse of Drugs Act 1971; cannabis reclassified as a Class C drug under the Drugs Act 2005.

[14]In: Caan, W. and De Belleroche, J. eds. 2002. *Drink, Drugs and Dependence: From Science to Clinical Practice*. London. Routledge.

[15]Ashcroft, R. 2004. Current Epistemological Problems in Evidence Based Medicine. *Journal of Medical Ethics* 30: 131-135.

[16]Stevens, A. Trace, M. and Bewley–Taylor. 2005. *Reducing Drug Related Crime: An Overview of the Global Evidence, Report Five*. The Beckley Foundation Drug Policy Programme. London. The Beckley Foundation.

[17]Roberts, M., Klein, A. and Trace, M. date?. *Towards A Review of Global Policies on Illegal Drugs, Report One*. The Beckley Foundation Drug Policy Programme & Drugscope Report. London. The Beckley Foundation.

[18]World Health Organisation. 2004. *Neuroscience of Psychoactive Substance Use and Dependence*. Geneva. Geneva. World Health Organisation.

[19]Swadi, H. and Bobier, C. 2003 Substance Use Disorder Comorbidity Among Inpatient Youths with Psychiatric Disorder. *Australian and New Zealand Journal of Psychiatry* 37: 294-298. See also: Hunt, N. with contributions from: Ashton, M., Lenton, S., Mitcheson, L., Nelles, B. and Stimson, G. date? *A Review of the Evidence-Base for Harm Reduction Approaches to Drug Use*. London. Forward Thinking on Drugs: A Release Initiative. http://www.forward-thinking-on-drugs.org/review2.html

[20]Kleinig, J. 2004. Ethical Issues in Substance Use Intervention. *Substance Use & Misuse* 39: 369-398.

[21]Morgan, D. 2001. *Issues in Medical Law and Ethics*. London. Cavendish. 15.

[22]Berridge, V. 2001. Illicit Drugs and Internationalism: The Forgotten Dimension. *Medical History* 45: 282–288.

[23]Dyer, D. 2006. Prosecutors Take a Tough Line on Cannabis Supplied to Relieve Pain. *Guardian.* July 17.

[24]Fergusson, D. and Horwood, L. 2000. Does Cannabis Use Encourage Other Forms of Illicit Drug Use? *Addiction* 95: 505-520.

[25]Rey, J. and Tennant, C. 2002. Editorials: Cannabis and Mental Health. *BMJ* 325: 1183-1184.

[26]Kalso, E. 2001. Cannabinoids for Pain and Nausea: Some Evidence but is there Any Need? *BMJ* 323: 13-16.

[27]Evans, W. and Relling, M. 1999. Pharmacogenomics: Translating Functional Genomics into Rational Therapeutics. *Science* 286: 487-491.

[28]Caspi, A. and Moffitt, T. 2006. Gene-Environment Interactions in Psychiatry: Joining Forces with Neuroscience. *Nature Reviews Neuroscience* 7: 583-59

[29]Hall, W., Morley, K. and Lucke, J. 2004. The Prediction of Disease Risk in Genomic Medicine. *EMBO Reports* 5: S22-S26.

[30]Hall, W., Madden, P., and Lynskey, M. 2002. The Genetics of Tobacco Use: Methods, Findings and Policy Implications. *Tobacco Control* 11:119-124.

[31]Bates, M. and Blakely, T. 1999. Role of Cannabis in Motor Vehicle Crashes. *Epidemiological Reviews* 21: 222-232; Blows, S., Ivers, R., Connor, J., Ameratunga, S., Woodward, M. and Norton, R. 2005. Marijuana Use and Car Crash Injury. *Addiction* 100: 605-611.

[32]Morgan, C., Dazzan, P., Morgan, K., Jones, P., Harrison, G., Leff, J., Murray R., and Fearon, P. 2006. First Episode Psychosis and Ethnicity: Initial Findings From the AESOP Study. *World Psychiatry*

5: 40-46; Semple, D., McIntosh, A. and Lawrie, S. 2005. Cannabis as a Risk Factor for Psychosis: Systematic Review. *Journal of Psychopharmacology* 19: 187-19

[33]Capps, B. 2008. Authoritative Regulation and the Stem Cell Debate. *Bioethics* 22: 43-55.

[34]Ashcroft, R. 2004. Drugs symposium: Introduction. *Journal of Medical Ethics* 30: 332.

[35]Ibid.

[36]Ashcroft, R., Campbell, A. and Capps, B. 2005. Ethical Aspects of Developments in Neuroscience and Drug Addiction (Chapter IV). *Foresight: Drug Futures 2025?* Brain Science, Addiction and Drugs Project. London. Department of Science and Technology.

[37]Van Os, J., Bak, M., Hanssen, M., Bijl, R., de Graaf, R. and Verdoux, H. 2002. Cannabis Use and Psychosis: A Longitudinal Population-Based Study. *American Journal of Epidemiology* 156: 319-327.

[38]Zammit, S., Allebeck, P., Andreasson, S., Lundberg, I. and Lewis, G. 2002. Self Reported Cannabis Use as a Risk Factor for Schizophrenia in Swedish Conscripts of 1969: Historical Cohort Study. *BMJ* 325: 1199-1201.

[39]Hall, W. 2006. Is Cannabis Use Psychotogenic? *Lancet* 367: 193-195.

[40]Hall et al. *op. cit.* note 30.

[41]Terrenoire, G. 1992. Huntington's Disease and the Ethics of Genetic Prediction. *Journal of Medical Ethics* 18: 75–78.

[42]Roberts, M., Bewley-Taylor, D. and Trace, M. 2005. *Facing the Future: The Challenge for National and International Drug Policy, Report Six*. The Beckley Foundation Drug Programme. The Beckley Foundation. London.

[43]Hayry, M. 2004. Prescribing Cannabis: Freedom, Autonomy, and Values. *Journal of Medical Ethics* 30: 333-336.

[44]Centre for Cognitive Liberty and Ethics. 2004. *Threats to Cognitative Liberty: Pharmacotherapy and the Future of the Drug War*. Davis, California: CCLE

[44]Ashcroft, A. and Franey, C. 2004. Further Ethical and Social Issues in Using a Cocaine Vaccine: Response to Hall and Carter. *Journal of Medical Ethics* 30: 341-343.

PROFESSOR JOHN THODAY

1916 - 2008

John Marion Thoday, who died recently days short of reaching his 92nd birthday, was born in 1916. He claimed that he could remember, as a child, being taken to see the celebrations at the end of WW1 in November 1918. He came from an academic family, his father at different times occupying chairs of botany in South Africa, Egypt and the University College of North Wales, Bangor.

John Thoday's life was one of academic distinction following a lively youthful period. He read botany at Bangor graduating just prior to the start of WW2 (and maintained a life-long interest in plants). He then started as a research student at Trinity College, Cambridge in the autumn of 1939. Soon, however, he entered the RAF to work on photographic intelligence, a logical deployment of his talents given his deep interest in cytological studies. Among those whom he encountered in this phase of his life was Enoch Powell, at that time the youngest (29) one star general in the British Army, and someone whose ability in politics greatly impressed John even though their political beliefs did not coincide.

Following demobilisation in 1945 he resumed work on his Ph D and in 1946 moved to the Mount Vernon Hospital and Radium Institute. His research here resulted in a discovery of great fundamental significance namely that biological damage resulting from exposure to ionising radiation is greatly increased in the presence of oxygen. This discovery had both academic and practical importance.

In 1947 he was appointed to the University of Sheffield charged with developing the discipline of genetics and by 1954 had succeeded in establishing an independent department of that subject which he headed until 1960 when he succeeded Sir Ronald Fisher as Balfour Professor of Genetics at Cambridge. It was in the Sheffield years that he achieved a second important advance in genetical knowledge. He had made a decision to switch his research interest to the study of quantitative and quasi quantitative characters and with the help of a succession of able research students attracted by his enthusiasm and innovative approaches he was essentially the first to demonstrate that the genes influencing such characters can be mapped and their effects measured with some accuracy. Although he probably would not have emphasised the point, it is worth noting that that this work also indicated that quantitative trait loci (QTL) as we now denote them must vary considerably (at least in some instances) in the magnitude of their effects on a specific character.

During his time in Cambridge his manifold abilities and in particular his insight and ability to develop thinking in unconventional directions were deployed in several ways. In addition to systematic and shrewd effort devoted to strengthening his own discipline and in encouraging and supporting junior colleagues he was much involved in needed reform in teaching programmes across the biological sciences and in offering a broadly based degree course in his own subject. He also played a very significant role in strategic planning and implementation at university level as well as playing a full part in the academic and social life of Emmanuel College.

John's distinction in science was recognised by his election, in 1965, to Fellowship of the Royal Society. He also served as President of the Genetical Society in 1975-77 and was a member of the Galton Institute Council, in one form or another, for virtually thirty years.

He is survived by his wife of many years Doris, a daughter and a son. He was in a number of respects a fortunate man, not least in that he remained reasonably well until shortly before his death - a circumstance all the more remarkable in that his lifetime consumption of cigarettes must have amounted to a large six figure number. In the lottery of the genes he drew a good ticket and all who knew him would be glad of that fact.

John A Beardmore

IUSSP seminar on Trade-offs in Female Life Histories:
Raising New Questions in an Integrative Framework

Bristol, United Kingdom
23-25 July 2008

Organized by the IUSSP Scientific Panel on Evolutionary Perspectives in Demography in collaboration with the **University of Bristol**

With financial support from the British Academy, the British Society for Population Studies and The Galton Institute

Report by
Rebecca Sear and Paul Mathews

The goal of this seminar was to bring together researchers from a variety of disciplines to explore female life history. Evolutionary biologists have developed life history theory to identify the trade-offs individuals must make in allocating resources between growth, reproduction and the maintenance of body condition. Demographers, anthropologists and other human scientists have increasingly focused on these trade-offs, in an attempt to understand the patterning of fertility, mortality, growth and ageing across human populations. This 2nd meeting of the IUSSP Evolutionary Perspectives on Demography Panel brought together 36 participants from the fields of demography, anthropology, physiology and biology to explore this issue.

Four plenary talks and 25 short presentations were given at the seminar, grouped into 7 sessions. These themes represented both key stages in female life history (age at first birth; birth intervals and pregnancy; parental investment; and grandmotherhood) and the applications of such life history analyses (to the demographic transition;

and theoretical models of life history). The 29 papers were very broad ranging, encompassing empirical research across the developed and developing world, and including the use of historical datasets; theoretical work on the mathematical modelling of life history traits; comparative work on primate life histories; conceptual and review papers synthesising previous work on life histories to generate new hypotheses; and the implications of such research for policy. A key theme to emerge from the seminar was the importance of getting the methodology right in explaining life history trade-offs. In particular, the difficulty of understanding variation in life history trade-offs given the problem of heterogeneity between women was much discussed. Here a dialogue between disciplines such as demography and evolutionary biology is important in furthering the field, since both can bring their expertise to bear on this complex issue. The importance of an integrated understanding of female life history for making policy decisions was also highlighted: without an in-depth understanding of *why* female life histories vary, it is extremely difficult to develop effective policy which aims to improve women's lives. These wide ranging papers will be published in a special issue of the *American Journal of Human Biology* next year, in order to provide an integrative framework for future research in this area.

The meeting was held at the University of Bristol, 23-25 July 2008. The organisers were Mhairi Gibson (local host) and Rebecca Sear, with help from the IUSSP Panel on Evolutionary Perspectives on Demography, particularly Monique Borgerhoff-Mulder and Ulrich Mueller. As well as the generous financial and administrative support offered by the IUSSP, this meeting received financial sponsorship from the British Academy, the British Society for Population Studies and the Galton Institute, and administrative support from BIRTHA (Bristol Institute for Research in the Humanities and Arts).

The first plenary of the workshop,

given by Professor Kristen Hawkes from the University of Utah, set the tone for the workshop by exploring in detail the problem of how heterogeneity between women can obscure life history trade-offs. If women differ in their genetic endowment or in their access to resources, then life history trade-offs will be occurring across the surface of numerous different curves. This will make it difficult to observe trade-offs at the population level. This plenary highlighted the importance of using appropriate methodology to try and account for this problem, which in turn highlighted the necessity for interdisciplinary work in this area. Demographers are also concerned with the problem of heterogeneity and have been working to produce solutions in recent years. Anthropologists and evolutionary biologists must engage with and help to further these methodological advances, in order to advance their own research into life history theory.

After this opening, the workshop continued with a further three plenary talks and seven sessions of proffered papers. Four of the seven workshop sessions concerned key stages in female life history: age at first birth, pregnancy and birth intervals, parental investment and grandmotherhood. One session focussed exclusively on a particularly important life history trade-off, that between investment in reproduction and investment in somatic maintenance. The remaining two sessions concerned the applications of such life history analyses: how such work can shed light on the demographic transition, and how such research can be used to generate overarching models of female life history.

The second plenary of the workshop began the debate over whether fertility is traded off against longevity. Professor Emily Grundy, from the Centre for Population Studies at the London School of Hygiene and Tropical Medicine, presented data from England & Wales, Norway and the US to show that the relationship between investment in reproduction and later life outcomes can

differ even in industrialised countries with similarly low mortality and fertility rates. This plenary was neatly complemented by a paper which presented very similar analyses of historical data from Quebec and Utah. Alain Gagnon, of the University of Western Ontario, began by remarking that the Quebeçois data has been used in three previous studies to show a positive relationship between fertility and longevity, a negative relationship, and no relationship at all. Again, this illustrates the importance of getting methodology right when analysing the tricky issue of life history trade-offs. Grazyna Jasienska, from Jagiellonian University, drew this session to a close by highlighting gaps in the existing literature on this relationship in the hope that focussing attention on such lacunae will stimulate research which closes these gaps.

The third plenary, by Professor Gillian Bentley of the University of Durham, was a first-rate demonstration of integrative research. Gillian brought together demography, evolutionary biology and physiology in her research on the effects of developmental environment on female reproductive physiology. She and her group have used the natural experiment of migration to investigate the influence of developmental environment on female reproductive hormones, by comparing such hormones in Bangladeshi migrants from Sylhet to the UK, with women still resident in Sylhet and UK women. This physiological theme was picked up in the session in birth intervals and pregnancy, with papers by Virgina Vizthum, of the University of Indiana, and Claudia Valeggia, of the University of Pennsylvania, investigating the hormonal correlates of pregnancy loss and the resumption of post-partum fecundity respectively. Both papers involved small-scale studies of non-Western populations (a Bolivian agropastoralist community and an indigenous Argentinean population), in dramatic contrast to the large scale datasets used by Grundy and Gagnon, demonstrating the diversity of evidence brought to bear on female life histories during the workshop.

Perhaps the most diverse session, however, in terms of empirical evidence was the session on parents and parental investment. In this four-paper session, Katherine Hinde, from UCLA, demonstrated sex-biased parental investment in rhesus macaques. David Lawson, from University College London, followed this with his analysis of the trade-off between wealth and family size in the large-scale, longitudinal UK database ALSPAC (Avon Longitudinal Study of Parents and Children). Eshetu Gurmu's paper (from the University of Addis Ababa) switched focus to the developing world, with his study of marital dissolution in Addis Ababa. Finally, Kai Pierre Willfuehr, from Giessen University, ended the session with his analysis of another historical dataset, this time from northern Germany. All studies highlighted the need to take context, particularly, though by no means exclusively, access to resources, into account when analysing parental investment decisions.

The final plenary, by Professor Beverly Strassmann of the University of Michigan, was a wide-ranging lecture which showcased Professor Strassmann's long-term anthropological research project among the Dogon in Mali. A number of different trade-offs were considered during this plenary, which also highlighted that women do not make life history decisions in isolation, but in the context of a partnership. Such partnerships may be characterised by sexual conflict. Conflict between partners may be particularly pronounced in this highly polygynous society, and much of Professor Strassmann's research has focused on the implications of polygyny for women's life history strategies.

Perhaps two key themes dominated the workshop, one methodological, one with both empirical and theoretical implications. Already discussed is the emergence of a consensus that researchers across disciplines must unite to develop rigorous methodology to analyse life history trade-offs. This issue was discussed at several points during the

workshop in the context of moving forward our understanding of female life history trade-offs by appropriately controlling for heterogeneity between women. Ruth Mace's (University College London) paper, on contraceptive uptake in the Gambia, emphasised how interactions between demographers and anthropologists have already improved the methods for investigating life history trade-offs. Anthropologists are now increasingly making use of statistical methods such as event history analysis, which can incorporate time varying covariates. Such techniques are vitally important for interpreting the dynamic processes that occur during the life course.

The other theme that emerged was the importance of kin in female life histories. A number of empirical papers demonstrated the key importance of relatives in female life history, including Donna Leonetti's (University of Washington) analysis of first births in two Indian communities, Monique Borgerhoff Mulder's (UC Davis) paper on contraceptive use in rural Tanzania, Brooke Scelza's (UCLA) paper on grandmaternal investment in Martu Aborigines, Australia and David Coall's (University of Basel) paper on grandparental investment in modern Switzerland. Papers by Meredith Reiches (Harvard University), Jonathan Wells (Institute of Child Health, University College London) and Lesley Newson (University of Exeter) all explored the theoretical implications of the importance of kin. Reiches's paper considered how female life history trade-offs would be better understood in the context of 'pooled energy budgets', that is the energy budgets of not just the woman herself but all her relatives, including her partner, who may be contributing to her reproductive effort. Wells's paper discussed the importance of kin in the context of genomic imprinting, and how this may have influenced the evolution of female reproductive behaviour. Lesley Newson brought the influence of kin to bear on the puzzle of the demographic transition, and suggested that part of the

explanation for low fertility in industrialised societies is a lack of kin support, given the evidence that kin provide both encouragement and practical support for raising children.

Much of the research presented at the workshop has important policy implications, touched upon by the presenters. For example, Alejandra Nũnez-de-la-Mora's paper on the trade-off between immune defence and reproduction highlighted the need to take a holistic approach to development, since a reduction in disease prevalence may inadvertently lead to an increase in fecundity. One paper focused exclusively on the implications of such life history research for policy-makers. Sarah Johns, from the University of Kent at Canterbury, demonstrated how an integrative understanding of female life history trade-offs can be used to inform policy surrounding teenage motherhood, a subject of some concern to the UK government, as well as a number of other industrialised countries. She suggested that teenage motherhood is a rational strategy in the context of social marginalisation and low life expectancies. Therefore policies targeted solely at improving adolescent's knowledge of contraception are unlikely to result in substantial decreases in the instance of teenage pregnancy, without also tackling social inequality and poverty.

Programme of Events

Plenary: Kristen Hawkes
Vital rates in human & chimpanzee populations: how within species variation complicates cross-species comparisons.

Session: Contraception and the Demographic Transition
Chair: Rebecca Sear
Monique Borgerhoff Mulder
Women's Fertility Preferences and Family Planning: Tradeoffs, Biased Cultural Transmission, or Men?

Ruth Mace
Social Influences on the Decision to Start Using Contraception: a Study From Rural Gambia

Mary Shenk
Causes and Consequences of the Demographic Transition in Urban South

India: Transitions in Total Fertility and Age of First Reproduction

Lesley Newson
Cultural versus Reproductive Success: Why Does Economic Development Bring New Trade-offs for Women at all Stages of Life?

Session: Age at First Birth
Chair: Grazyna Jasienska

Karen Kramer
Early First Birth among Pumé Foragers. Implications of a Pooled Energy Budget to Life History Tradeoffs

Donna Leonetti
Age at First Reproduction in the Context of Differing Kinship Ecologies

Sarah Johns
Teenage Pregnancy and Motherhood: How Might Evolutionary Theory Inform Policy?

Session: Birth Intervals and Pregnancy
Chair: Barry Bogin

Virginia Vitzthum
Modulation of Reproductive Investment during Early Pregnancy

Claudia Valeggia
Interactions Between Metabolic and Reproductive Functions in the Resumption of Postpartum Fecundity

Daryl Shanley
A Mathematical Modelling Framework for the Study of Optimal Interbirth Intervals

Plenary: Emily Grundy
Biology or Sociology? What Can We Learn From Associations Between Reproductive Histories and Later Life Mortality in Contemporary European Populations

Session: Parents and Parental Investment
Chair: Tom Dickins

Katherine Hinde
Lactational Performance in Primiparous and Multiparous Rhesus Macaques: Milk Energy Density and Milk Yield

David Lawson
Trade-offs in Modern Parenting: a Longitudinal Study of Sibling Competition for Parental Investment

Kai Pierre Willfuehr
Is There a Trade-off Between Early Versus Late Survival? Long-term Consequences of Early Parental Loss in the 18th to 19th Century Krummhörn Population

Eshetu Gurmu
Determinants of Marital Dissolution in Addis Ababa (Ethiopia)

Session: Fertility, Somatic Mainte-

nance and Survival
Chair: Virginia Vitzthum

Alejandra Nunez-de-la-Mora
Trade-offs Between Growth, Maintenance and Reproduction in Human Female Life History: What Do We Know?

Ilona Nenko
Relationship Between Fertility and Body Size and Shape: An Empirical Test of the Covert Maternal Depletion Syndrome Hypothesis

Alain Gagnon
Are There Trade-offs Between Fertility and Survival to Old Ages? Evidence From Three Large Historical Demographic Databases

Grazyna Jasienska
Reproduction and Lifespan: Overall Energy Budgets, Trade-offs, Intergenerational Costs and Costs Neglected by Research

Plenary: Gillian Bentley
Developmental and Environmental Trade-offs in Female Fecundity

Plenary: Beverly Strassmann
Menopause and the Trade-off Between Offspring Number and Offspring Quality

Session:Grandmothering and Allocare
Chair: Monique Borgerhoff Mulder

David Waynforth
Mothers' Decisions to Provide Direct Care to Their Infants Versus Using Daycare and Family-based Childcare: a Life-history Perspective with Data on British Mothers and Children

Brooke Scelza
The Grandmaternal Niche: Critical caretaking among Martu Aborigines

David Coall
Grandparental Investment: The Influence of Early Reproduction and Family Size

Jonathan Wells
Cooperative Breeding and the Evolution of Flexibility in Female Reproductive Behaviour

Session: Models of Life History
Chair: Mhairi Gibson

Duncan Gillespie
When Fecundity Does Not Equal Fitness: Evidence of an Offspring Quantity-Quality Trade-off in Preindustrial Humans

Barry Bogin
Childhood, Adolescence, Fertility and the Variant Nature of Human Life History

Meredith Reiches
Pooled Energy Budget and Human Life History

Letter to the Editor

Dear Sir

Apparent IQ stability across several decades in an extended family

Much has been made of the world-wide increase in intelligence quotient scores amounting to 0.3 points per annum. However, this is a statistical result and apparently backed by very few longitudinal studies of actual families.

Intelligence is considered to be between 50 - 70% inherited according to the various observers, and the rise has been blamed upon better nutrition, test sophistication and even playing computer games that might stimulate visual abilities.

I provide here an eleven decade presentation of IQ results in an extended Pembrokeshire family that is currently undergoing diaspora. The tests used had a Standard Deviation of 20 and the results corrected for age at the time of testing.

Volkmar Weiss (personal communication) suggested that the high mean level of IQ is due to the author only attending the houses with the best cooks.

Whilst there may be some truth in this a couple of pedigrees have been included showing both high and low levels of tested intelligence indicating a possible local lack of dominant mediocre genes. In addition an important local council within the County found that eighteen of its twenty six members had been measured by the author, exposing relationships that had not previously been realised. The family has found local eminence for about four centuries which may indicate abilities above the average.

James R Flynn considers that the reported rise in IQ is an artifact based on the sub-tests developed in a generally pre-scientific age of concrete thinking but still used on subjects maturing in a post-scientific phase when generalities are more important, thus providing a skewed result. Common sense must support this. In the family there is little significant difference in IQ across the time span.

The oldest survivors of their pre-twentieth century cohort were probably the fittest, the brightest and therefore the longest lived. Those surviving the first World War (1899-1918) seem most depressed but as a farming group they always fed well and few died in any war.

Although IQ remains the same the proportion of first degree with as low a score as 118, which one would have thought impossible. Nevertheless it is a recruitment of all available talent, ranging from astronomers to fine arts but with a bias towards biological sciences. The earliest degree was in the pre-1898 birth decade, when in theology, the individual recorded the highest IQ ever, in his college. That was only equalled once later and none quite reached brilliance (IQ 160).

The two related pedigrees show one group that remains successful by choosing suitable breeding mates, and a second, where the breeding is haphazard and suffers from having a lady in a former generation who is reported to have had some undiagnosed mental malady which from biased verbal reports seems to have been bipolar. Many of her descendants fail to thrive.

Dyslexia is common throughout the family and this may possibly account for many with low IQ scores, who, in the real world has married for wealth or land gain. It may be that Pembrokeshire is a little behind the times.

Patrick James

RESULTS				
Birth Decade	Number	Mean IQ	S.D.	Percentage with First Degrees Degrees
Pre 1898	13	128.0	11.3	(1 individual)
1899 - 1908	26	120.6	15.7	0
1909 - 1918	26	118.0	20.1	3.8
1919 - 1928	35	128.4	23.9	28.6
1929 - 1938	46	127.5	16.8	28.3
1939 - 1948	37	123.5	19.1	24.3
1949 - 1958	37	123.9	21.1	35.0
1959 - 1968	68	125.9	18.5	29.4
1969 - 1978	29	120.0	23.2	31.0
1979 - 1988	16	127.6	18.0	-
1989 - 1998	4	133	-	-