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Growth as a mirror: Is endocrine disruption challenging Tanner's concept?

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Abstract

Background—James Tanner coined the expression 'Growth as a Mirror' and summarized in four words the results of more than a century of research on growth. Nineteenth century social reformers saw poor child growth as a reflection of terrible environmental conditions of the working class. Later investigators in anthropology and other fields clarified the connections between poor nutrition, disease, psychosocial stress and poor growth.

Aim—To evaluate the growth as a mirror concept in light of recent studies of endocrine disruption.

Papers and Implications—Pollution is recognized as a prominent component of the modern environment. From studies of many pollutants it is clear that some pollutants depress growth while others speed sexual maturation and increase growth, primarily in weight and fatness. While such unwelcome environmental features do not always suppress growth, growth still mirrors the environment in all its complexity and this relationship is key to understanding growth patterns today. For example, Akwesasne Mohawk adolescents are characterized by high rates of obesity and overweight. Their growth reflects the multiple intersecting influences of psychosocial stress, several pollutant exposures and limited dietary choices.

Conclusion—Although Tanner did not anticipate the myriad influences of pollutants, the growth as a mirror concept continues to have great validity and utility.

Keywords

Growth; endocrine disruption; sexual maturation; persistent organic pollutants

INTRODUCTION

James Tanner coined the expression 'Growth as a Mirror'. The concept embodies Tanner's view of growth as an accurate reflection of the environment. For Tanner and other human biologists, the environment was viewed broadly. It included psychological, social and

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physical elements and ranged from the level of the family to the entire society. The reflection of the environment in growth prompted Tanner to develop auxological epidemiology as a means of interpreting growth at the individual and population levels.

As a student of the history of science and the most knowledgeable commentator on the history of growth studies, he acknowledged that the relationship of child growth to child health that underlies his auxological epidemiology (not auxological epidemiology itself) was recognized by the English social reformers of the 19th century in their efforts to improve the lives of the working class. The concept of ecosensitivity of growth was developed much further by anthropologists, most prominently Franz Boas (Johnston and Little 2012). Correctly identified as the founder of American anthropology, Boas was the first to describe normal age-related biological variation in growth, the concept of developmental age and that acceleration and retardation of growth may have social and ethnic differences. His research concerning the role of the environment on phenotypic plasticity, conducted in the early 20th Century for the US Immigration Commission, was crucial to the demise of racial classifications and established the close connection between environmental conditions and patterns of human physical growth (Stocking 1968; Tanner 1981). This early examination into the nature of growth and body form provided a foundation for other scholars, drawn from anthropology, psychology and clinical pediatrics to continue to investigate the environment-growth interaction and contributed substantially to the creation of Tanner's belief that growth reflected societal and environmental conditions.

This essay analyses the growth as a mirror concept and evaluates it in light of new findings in studies of environment–growth interaction. It particularly emphasizes studies of the effect of human-made environmental features, some of which seem to cause developmental acceleration.

GROWTH AS A MIRROR: IT'S HISTORY AND COMPONENT CONCEPTS

Growth as a mirror was Tanner's most succinct statement on the meaning of human growth. The term first appeared in ArtoDemirjian's edited volume from the IVth International Congress on Auxology in Montreal in 1982. The essay was an evidentiary tour de force convincingly demonstrating that growth patterns differed over time and that, within one time frame, they differed according to social class and other measures of economic well-being. This statement was not the last of his published assertions that growth reflects human conditions, and it was not the first either. Prior to the 1982 Montreal congress he had published his very useful introduction to growth, *Foetus into Man* (Tanner 1978: 219). This ended with a linguistically economical but intellectually complex coda that contained several ideas that are components of his growth as a mirror concept. He wrote,

Provided always that parental size is known, growth emerges as the prime measure of a child's physical and mental health (p. 219).

This is a statement about the great value of growth data when dealing with the individual child. Growth measurements are probably the most common measurement made in clinic visits to gauge the well-being of the paediatric patient. It has been used in more exacting clinical situations also. In describing the clearance of HIV infection in a perinatally infected infant one research team judges that the intervention is proven beneficial and complete by virtue of the child's normal growth and development. They write 'The child is 5 years of age at this writing, is HIV-seronegative, and remains well, with normal growth and development and no laboratory or clinical evidence of HIV-1 infection' (Bryson et al. 1995: 833).

In toxicology, weight loss in adults and slowed growth in sub-adults are classic signs of toxicity. Typically, toxicological studies of different substances are calibrated to measure

effects at levels too low to affect growth since any dose that can affect growth is already demonstrably toxic.

Tanner went further with the 'growth as a mirror' concept, beyond the clinical assessment of the individual child. Differences in growth of groups reflected the distribution of what he called 'the substrates for growth'

It [growth] is equally powerful for studying the effect of political organization upon the relative welfare of the various social, cultural and ethnic groups which make up a modern state.

When large differences exist in growth patterns among social groups, the differences reflect differential distribution of the substrates for good growth and the society's valuation of equal health for all social strata. A grossly unequal distribution reflected on the moral character of a society. These ideas put him squarely in the lineage of the 19th century social reformers. He comfortably credited the idea of auxological epidemiology to them. He wrote,

My thesis—that the growth of children amongst the various groups which make up a contemporary society reflects rather accurately the material and moral condition of that society—is no less important for being wholly unoriginal (Tanner 1986: 3).

Tanner's earliest statement on growth patterns of a population reflecting environmental features appears in the seminal text *The Biology of Human Adaptability* (Baker and Weiner 1966: 46).

If we compare the growth of members of a single population under differing social, nutritional, and medical conditions we can see to what extent their present environment falls short of supplying those stimuli and substrates which are necessary if all members of the population are to fulfil the potentialities of their gene complex (emphasis added).

Here and everywhere else Tanner tended to think of the environment as permissive. It permits growth up to the limit of the individual's genetic potential by providing the 'necessary substrates', in the form of a nutritionally adequate diet and freedom from diseases. If conditions were right, the intrinsic growth trajectory of the individual would be expressed fully, but shortfalls in the necessary substrates would hinder growth and produce a downward trajectory.

Growth analysis, or auxological epidemiology to use Tanner's term, was a valuable tool for discovering adverse conditions expressed as poor child growth and to reveal the social and moral values of societies. It developed throughout the 19th and 20th centuries when the primary influences on growth were nutritional deficit and infectious disease, and the related aspects of low SES such as poor preventive healthcare and medical care generally.

If we continue to study different social groups in the population as social and economic development or regression occurs we can see clearly whether or not we have really advanced and whether we have favoured one social group at the expense of others (Baker and Weiner 1966: 46).

The CODA from his 1978, *Foetus Into Man*, also expresses concern about technology. This is not a concern with a mismatch of biology to the environment, but a concern with the over-use of new technology, the intentional, medical modification of the developing person to enhance specific characteristics such as athletic performance or intelligence or to create a citizenry of identical persons perhaps for the purpose of social control.

At the same time it gives us valuable lessons on the way in which our biological heritage and our technological culture interact. It warns us that all too soon we shall

be technically capable of creating monstrously specialized or monstrously similar children. It points to our need to be reconciled with our origins, to see ourselves once again as a part of the natural order; not foetus into angel, nor foetus into monster, but foetus into Man (Tanner 1978: 219).

In short, Tanner's concept of growth as a mirror has four distinct components: (1) growth of the individual child reflects the child's health; (2) the pattern of child growth typical of a population reflects the conditions affecting that population; (3) the degree of differences in growth within a population and the distribution of difference reflects the degree of social differentiation and values regarding social difference; and (4) growth may be altered by purposeful technological alteration. What he did not foresee was *accidental* technological modification, the modification of growth and development through exposure to pollutants that are endocrinologically active.

POLLUTANTS AND POOR GROWTH

A pollutant is a material or energy unwanted to some degree because of suspected or proven detriments to the health of humans, animals or other valued features of the environment. Materials may be simple elements such as lead or complex compounds such as pesticides and other hydrocarbon compounds. Energy pollutants include radiation, noise and light. The terms toxicants and pollutants are used interchangeably, but neither is synonymous with 'endocrine disruptor', which is a narrower category of pollutants. Endocrine disruptors are industrial pollutants and pharmaceuticals with the ability to disrupt endocrine system functioning. Until recently, all evidence from studies of pollutants and growth suggested that pollution, at some point in the lifespan, caused poorer growth (for reviews see Schell and Hills 2004; Schell et al. 2010).

Exposure to certain energy sources hinders growth. Radiation exposure from atomic bomb fallout inhibits growth (Sutow et al. 1965; Nakashima et al. 1995), Noise is associated with decreased size at birth (Schell 1982; Kawada 2004) and with reduced height and weight growth in children (Takahashi and Kyo 1968; Schell and Ando 1991). Noise has the ability to induce the stress response, thus it is likely that it inhibits growth by disturbing hormonal regulation of growth during gestation and in the child.

Among the material pollutants, air pollution has been linked to poorer growth. Air pollution is a complex and variable mixture of gases (SO_x, NO_x, CO, O₃) and particulate materials of different sizes; it is associated with reductions in birth weight in many studies (Williams et al. 1977; Djemek et al. 1999; Ritz and Yu 1999; Bobak 2000; Xu et al. 2000; Lee et al. 2003; Liu et al. 2003). Post-natal growth has not been studied so intensively. Those few studies that have been conducted have found decrements in growth associated with components of air pollution (Schmidt and Dolgner 1977; Schlipkoter et al. 1986; Jedrychowski et al. 1999). Cigarette smoke is a particular kind of air pollution and its relation to decreased pre-natal growth is very well established (Hellerstedt et al. 1997).

Lead is a common component of air pollution, though it is absorbed less by inhalation than through the gastrointestinal tract. Lead falls from the atmosphere, either in or out of the home, and is brought to the mouth. Typically blood lead levels rise in early childhood. Hand-to-mouth activity by toddlers living in lead-burdened areas is thought to be the most common means of childhood exposure. Lead is a neurotoxicant causing permanent brain damage at high doses and learning deficits at lower ones (Centers for Disease Control 2000). In many neuro-toxicological investigations child growth is studied as a corollary. Studies of children with moderate lead exposure show growth deficits in height (or length), weight and/or head circumference (Ballew et al. 1999; Hernandez-Avila et al. 2002; Schell et al. 2009a). Even at low levels there is evidence that growth is slowed in proportion to lead

exposure and that the levels able to damage growth and development are surprisingly low (Schell et al. 2010; Afeiche et al. 2011; Burnitz et al. 2012).

Among the organic compounds, several can have a growth inhibiting effect. Recent meta-analysis of 12 European birth cohorts ($n = 7990$ women) found that a $1 \mu\text{g/dL}$ increase in polychlorinated biphenyls (PCBs) (specifically IUPAC #153) in maternal or cord blood was associated with a decrease in birth weight of 150g (95% CI = $-250, -50\text{g}$) (Govarts et al. 2012). Some PCB congeners are similar to dioxin which has been linked to reduced birth weights as well (Tsukimori et al. 2012). This strongly supports much earlier reports of growth reduction associated with acute PCB exposure in newborns and children (Yoshimura 1971); and experimentally in macaques (Allen et al. 1974).

Studies of growth in relation to noise, radiation, air pollution and lead fit the growth as a mirror concept well because growth is reduced in proportion to the exposure to the untoward influences. The effects of PCBs are more complex as they include inhibitory effects, but some apparent stimulatory ones as well, as is described later. In further agreement with Tanner's growth as a mirror, most toxicant exposures tend to concentrate on less powerful members of society and, thus, poor growth still locates poverty and social inequality.

ENDOCRINE DISRUPTION

Several pollutants have very different effects from those of noise and lead. Some are capable of interfering with the synthesis, secretion, transport, binding, action or elimination of natural hormones and can therefore affect growth as well as reproduction and behaviour (Crain et al. 2008; Newman et al. 2009; Crinnion 2011). These endocrine disruptors can be antagonistic or agonistic. The different effects depend on the compound itself and its relationship to natural hormones, binding proteins and their receptors. Thus, a compound may act like a natural hormone and bind to the receptor stimulating the cascade of cellular events normally following binding or it may occupy the receptor and simply block natural hormones from stimulating the receptor. This partially explains some of the variation in results from studies of endocrine disruptors.

There is some evidence that exposure to any of the large variety of compounds classed as endocrine disruptors increases length or stature (Leijds et al. 2008; Den Hond et al. 2011). There is more evidence that some toxicants can advance sexual maturation. However, there is considerable variation among the findings, some of which can be attributed to factors that are summarized later, while some variation is inexplicable and the subject of intense research.

The classic example of endocrine disruption from a persistent organic pollutant (POP) is DDT (dichlorodiphenyltrichloroethane). This pesticide was identified in *Silent Spring* (Carson and Darling 1962) as suppressing avian reproduction. Recent studies of human samples have found early onset of breast development (Gladen et al. 2000; Krstevska-Konstantinova et al. 2001) in relation to levels of p,p' -DDE (p,p' -DDE is the chief metabolite of DDT, can be measured in blood and is the commonly used indicator of past DDT exposure), while others do not (Wolff et al. 2008). p,p' -DDE also is associated with an earlier age at menarche in some studies (Vasiliu et al. 2004; Ouyang et al. 2005) but not in others (Gladen et al. 2000; Wolff et al. 2008; Den Hond et al. 2011). As further evidence of p,p' -DDE's disruptive potential, Perry et al. (2006) found that levels of p,p' -DDE were related to disturbed patterns of oestrogen and progesterone levels during the menstrual cycle that could cause difficulties in becoming pregnant and maintaining pregnancy (Perry et al. 2006).

Two other persistent organic pollutants studied frequently are polybromated biphenyls (PBB) (Blanck et al. 2000) and PCBs (Den Hond et al. 2002; Denham et al. 2005; Croes et al. 2009). The study of polyhalogenated biphenyls reveals the complexity of the problem involved in determining effects of POPs because each is not one compound but a family of congeners. There are many possible configurations of carbon substitution with bromine or chlorine that comprise the PBB and PCB groups, respectively. The different structures are expected to substantially influence the type of effects. Evidence of accelerated sexual maturation in the form of early menarche has been reported in two studies of PBBs (Blanck et al. 2000; Ouyang et al. 2005). PCBs are associated with earlier age at menarche (Denham et al. 2005).

There is a considerable amount of research showing that body burdens of some toxicants are associated with greater levels of weight and or fatness and disturbances of glucose metabolism and insulin regulation. Significant and positive relationships between toxicant levels and weight and BMI have been observed across multiple age groupings including infants (Verhulst et al. 2009; Mendez et al. 2011), children and adolescents (Gladen et al. 2000; Smink et al. 2008) and adults (Schildkraut et al. 1999; Pelletier et al. 2002; Karmaus et al. 2009; Elobeid et al. 2010; Dirinck et al. 2011).

Not all individual toxicants affect growth outcomes in the same way. For example, p,p' -DDE consistently shows the strongest relationships to increased BMI or weight, especially in adult subjects (Hovinga et al. 1993; Pelletier et al. 2002; Hue et al. 2007; Karmaus et al. 2009; Elobeid et al. 2010; Dirinck et al. 2011). p,p' -DDE is anti-androgenic, and thus affects the normal hormonal control of fat deposition (Longnecker et al. 2007). However, studies of sub-adults show mixed results, with some evidence demonstrating a negative relationship between p,p' -DDE and BMI (Burns et al. 2012) and others of a positive relationship (Gladen et al. 2000; Den Hond et al. 2011). HCB (hexachlorobenzene) has also shown positive relationships to weight and BMI, in both sub-adults and adults (Pelletier et al. 2002; Smink et al. 2008; Den Hond et al. 2011). Like p,p' -DDE, HCB is an organochlorine molecule that can affect normal metabolic processes. Evidence for PCBs increasing BMI or weight is limited, with some studies showing increased weight growth (Gladen et al. 2000; Pelletier et al. 2002; Verhulst et al. 2009), but these results are not universal (Den Hond et al. 2009; Dirinck et al. 2011).

Additional studies have found accelerations in growth in other anthropometric dimensions. Waist circumference represents a clearer measure of central adiposity and fat deposition when compared to BMI and can be used to better assess toxicants' effects on adipose tissue. Studies have found positive relationships between waist circumference and organochlorines and phthalates (Lee et al. 2007; Elobeid et al. 2010), although relationships appear to be stronger in females than in males (Hatch et al. 2008). The findings of BMI, weight and waist circumference demonstrate that multiple toxicants, especially organochlorines, are able to disrupt metabolic processes in the body and induce increases in growth in these dimensions.

Some evidence suggests that organochlorine exposure shows positive associations with length and height growth. Studies of adolescents have found positive relationships to height with p,p' -DDE and PCBs (Gladen et al. 2000; Leijds et al. 2008; Den Hond et al. 2011), although findings in adulthood and infancy appear to be non-existent (Karmaus et al. 2009; Verhulst et al. 2009). Adolescence is accompanied by high levels of sex steroids, which can be altered by toxicant exposure. As height during this period is strongly controlled by said steroids, the agonistic properties of certain toxicants may induce more rapid height growth during puberty.

The endocrine disrupting properties of pollutants, whether inhibiting or promoting, highlight the complex relationship between growth and environmental exposures. Tanner and other early auxologists believed that increases in growth outcomes were the result of the removal of deleterious factors, but did not yet understand the properties of different pollutants. These diverse relationships between pollutants and growth outcomes demonstrate the complex nature of growth and its sensitivity to the environment.

COMPLEXITY OF HUMAN EXPOSURES AND THE COMPLEXITY OF GROWTH AT AKWESASNE

James Tanner was quite aware of the complexity of influences upon growth. Indeed, Tanner's work promoted this perspective so well that now human biology characteristically takes a bio-social or bio-cultural approach to human biological variation. The human condition is fundamentally a product of this interaction.

No other analytic approach can produce the level of insight into the pattern of growth and development of youth of the Mohawk Nation at Akwesasne. This community is deeply affected by several intersecting influences, among them exposure to endocrine disrupting pollutants. Growth as a mirror reflects these multiple intersecting forces. Pollution, stress from persecution/acclulturation and nutritional transition all are in force and may influence child growth and other aspects of human biology.

Background

The Akwesasne Mohawk Nation (AMN) is one of several communities comprising the Mohawk Nation, which in turn is one of the tribes comprising the Haudenosaunee or the League of Six Nations (the Iroquois according to French explorers of the 16th century). The Akwesasne community began to be exposed to pollutants after the development of industry on the St Lawrence River in the 1960s and 1970s. The community is located across the St Lawrence River itself, bordering the Canadian provinces of Ontario and Quebec and the state of New York, USA (Figure 1).

Exposure to PCBs was detected in the mid-1980s and an environmental crisis ensued involving concerned citizens of the community and local, state/provincial and national health authorities. The location of the Akwesasne community, bordering multiple municipalities, adds to the complexity of regulations, recourse and stress. While the AMN is a sovereign nation holding treaties with the US and Canada, those nations enforce its environmental laws within the territorial borders of the AMN.

There was good reason for concern. Levels of PCBs among community members who participated in surveys were quite high (Figure 2). Among 10–16.99 year olds participating in one study, the mean level was between the 90th and 95th centiles of US CDC reference values for the US population (Centers for Disease Control 2009; Schell et al. 2012). Levels of *p,p'*-DDE and of lead were well within the range commonly found in the US population.

Endocrine disruption of weight-related hormonal control

Among Akwesasne adolescents the level of PCBs was found to be associated with changes in thyroid economy. Thyroid stimulating hormone was significantly elevated in relation to PCBs, particularly the ones with long half-lives, and the level of free thyroxin was depressed (Schell et al. 2008). This is the classic pattern for depressed thyroid activity. Given the role of thyroid hormones in energy regulation and maintaining healthy body weight, the relationship suggested that PCBs could contribute to disorders of the thyroid economy and to overweight. The fact that the relationship was strongest with PCB congeners with long

half-lives in combination with the fact that it was not associated with congeners indicating current exposure, suggested that an earlier exposure was responsible for the effects. This in turn suggests some type of thyroid programming earlier in the lives of these adolescents.

Although a mechanism for thyroid programming by any toxicant is not known, diminished thyroid activity may be due to autoimmune pathology. PCB exposure may incite autoimmune activity that involves the thyroid. When the Akwesasne adolescents were studied again ~ 4 years later, those with elevated anti-thyroid peroxidase antibody (TPOAb) had significantly higher levels of PCBs and *p,p'*-DDE (Schell et al. 2009b). Elevated TPOAb indicates autoimmune activity. It is a very common sign of autoimmune disease acting against the thyroid (e.g. Hashimoto's thyroiditis) and accompanies many other autoimmune disorders. Significantly, the relationships with PCBs were with the persistent forms and not the ones reflecting current exposure, again suggesting that much earlier exposures were responsible.

Endocrine disruption: A source for overweight?

The concern that diminished thyroid activity may stimulate weight gain and a higher frequency of overweight and obesity is plausible given results from anthropometric studies of the Akwesasne 10–16.9 year olds. Average weights of boys tracked along the age-specific 90th centile of CDC reference values and weights of girls approximated the 85th centile in every year, while both boys' and girls' heights approximated the 50th centiles (Gallo et al. 2005). Consequently, BMIs are high; average BMIs of boys are at or above the 90th centile and those of girls at or above the 85th centile. Fully, 59% of the youth were either overweight or obese. Further, the extra weight was demonstrably located in thicker skin-folds and located more centrally than at the appendicular sites (Gallo et al. 2007).

The greater weight for height may not seem surprising given the observation that many North American Native American children and youth measured in the last third of the 20th century were heavier than US reference values while of normal height (Schell et al. 2006). What is surprising is the rapidity with which the obesity epidemic has overtaken this particular community and the coincidence in timing with the discovery of pollutant exposure.

Over the last quarter of the 20th century the Akwesasne Mohawk youth were measured by trained anthropometrists twice, once in 1981 (Pfeiffer and Dibblee 1982; Helmuth 1983) and again by our team over the period 1996–2000 (Figure 3). Comparison of weights between those two surveys shows that the average weight of each age cohort of males increased by 11–22 kg and averaged 15.4kg, while no change in average heights occurred (Schell and Gallo 2012). Weight changes in females occurred also but were not as substantial. The gender difference has been typical of the obesity epidemic (Wang and Beydoun 2007; Brewis 2011).

Changes in thyroid economy are probable contributors to the increased frequency of overweight and obesity but are not the entire picture: growth mirrors more than exposure to toxicants. Weight is inextricably related to diets and food choice has become an issue at Akwesasne. Residents face the difficult dilemma of following tradition and consuming locally raised or caught food (fish, game, plants) that might be contaminated or consuming more store-bought items characteristically high in fat, calories and sodium to avoid toxicant exposure. Fear of local foods was once based on suspicion, but now has an empirical basis. Young Akwesasne adults who claimed in interview to have eaten locally produced foods in the past year had significantly higher levels of PCBs, particularly ones reflecting past exposure rather than recent exposure (Schell et al. 2012). Meanwhile, the frequency of overweight and obesity is known to the community and to its health services with

consequent concern for diabetes and other diseases related to overweight. Both alternatives of the dietary dilemma have negative consequences.

The dietary dilemma is felt in the context of a centuries long history of oppression of American Indians that the Akwesasne community has experienced severely (Goodman-Draper 1994). More recent attempts to destroy American Indian cultures through forced residence of American Indian children at government schools and by other means have imbued many American Indian communities with the desire to resist any force that further undermines their culture. Avoiding contaminated local foods that have been part of the traditional diet represents a loss of Mohawk culture (Akwesasne Task Force On The Environment 1997). Avoiding a few foods may seem trivial to people accustomed to depending on grocery markets and who fish and hunt for recreation, but it is not to native people. The cultural damage is deeper than forced food choices (Arquette et al. 2002; Schell et al. 2012). The consumption of traditional local foods required traditional activities of hunting, fishing and farming as well as traditional means of preparing foods. The methods for success in these activities were passed on from one generation to the next. These traditions and the ties between generations are really what are at stake rather than just cuisine.

Just as Tanner and the auxological epidemiologists of the 19th century saw the connection between larger politico-economic forces and the poor growth of manual-class children, it is the larger political forces operating at Akwesasne that make avoiding toxicants in food a destructive influence on local Mohawk culture. In order to allow Akwesasne residents to continue catching, preparing and eating locally-caught foods that are free of contaminants, the local sources of pollution would have to be stopped and past environmental damage redressed. However, expeditiously cleaning up all contamination that has entered the St Lawrence River and the food chain is a process mired in legalities. Recall that there are two Canadian provinces and one US state involved and, therefore, two federal governments. There are court-adjudicated interpretations of risk and danger that adhere to western culture and legal definitions, not indigenous ones. Instead of dealing with the larger forces that hinder clean-up and full restoration of the environment to permit consumption of locally caught foods, the public health establishment response is a message to avoid the food sources of contamination. This approach removes immediate responsibility from the corporations that polluted, along with the government agencies charged with environmental protection, and places the responsibility for a safe diet on the individual resident who must avoid the hazard. This means a daily decision about food choice with consequent concern and stress. Stress is a feeling much talked about among Akwesasne residents, Our investigations were not designed to test its affects but based on other studies of stress, overweight and health (Tamashiro and Moran 2010), it contributes to the burden of risks that have accumulated in the community and is seen as pertaining to the high rates of diabetes, cardiovascular disease and the generally low life expectancy that characterized North American Native American communities (Indian Health Service 2002, 2009; Barnes et al 2010).

The considerable weights of Akwesasne adolescents and young adults is a major health problem, as high weights are associated with numerous poor health outcomes. The high weights represent the intersection of numerous forces acting throughout the sub-adult pattern of growth. It is a reflection of these environmental forces and, as such, growth acts as a mirror of these complex forces.

Pollutants and sexual maturation

The pattern of sexual maturation among the Akwesasne adolescent females has been investigated through studies of menarcheal age and age at Tanner stages and in boys through an investigation of testosterone levels.

Akwesasne adolescent girls, 10–16.9 years of age, were asked if they had experienced menarche at the time of interview. Probit analysis was used with this *status quo* data to calculate the median age at menarche, which was 13.2 years. This is comparable to US girls. Binary logistic regression analysis was used to test the effects of toxicants while controlling for common influences on the age at menarche (Denham et al. 2005). Age at menarche was earlier in relation to the level of PCBs in the girls at the time of blood draw. Doubling the mean PCB level was associated with a predicted age at menarche that was 6 months earlier than the average age. On the other hand, higher lead levels were associated with later age at menarche (Figure 4).

A complimentary analysis of girls' age at entry to Tanner Stage IV was conducted. Girls were asked to self-assess their Tanner Stage, which they did in private with the aid of line drawings and written cues to identify marker components of each stage. Tanner Stage IV is highly correlated with age at menarche and the results were similar: age at entry into Tanner Stage IV was negatively associated with PCB level, greater PCB level was associated with younger ages (Gallo and Schell 2008). Among boys the effect was quite different. Testosterone levels of boys were significantly lower in relation to PCB levels and higher in relation to p,p' -DDE levels. Clearly compounds that bear some resemblance to steroid hormones involved in sexual maturation act differently on males and females. Also, two compounds, p,p' -DDE and PCBs, although both endocrine disruptors and considered pollutants can produce opposite effects, most likely due to differences in their structure.

The example of Akwesasne adolescent development highlights the complex relationship between growth and environmental exposures, as PCBs are associated with accelerated maturation and lead is associated with delayed maturation in girls. In individuals exposed to toxicants with opposing effects, maturation may appear normal if the levels of each balance the others. Thus, it is necessary to account for the effects of specific toxicants if studying environmental exposures. Analysing specific influences on growth is always more precise than analysing combinations whether they be combinations of toxicants or of other factors. This is well known in urban rural comparisons of growth where the samples may show no differences in growth or size despite there being substantial differences in the impact of specific influences on growth because the net effects of the combinations of environmental factors in each are equal. Growth is a mirror but it is also a summary of the environment; only analysis of specific features will reveal what growth is mirroring. In the case of any specific toxicant the situation is additionally complex because the timing and duration of exposure also affects the relationship between maturation and the toxicant.

CONCLUSION

The pattern of growth of the Akwesasne Mohawk adolescents is a product of multiple, intersecting environmental influences. Together they are reflected in the mirror of growth. One of these is diet, another stress, a third is socioeconomic position and a fourth is industrial pollution that has entered the environment and food chain. All occur against a background of five centuries of cultural oppression. Diet, stress and socioeconomic circumstances are familiar factors to auxologists. Pollutants are becoming better known. Their effects on growth are numerous, reflecting their myriad chemical compositions. If one thing is known about pollutants now, it is that they exist in huge variety and produce a near equal variety of effects. Some stimulate growth while others inhibit it. The mirror of growth

is still reflective, but not exactly as Tanner portrayed it when major environmental influences were different.

The growth as a mirror concept was developed in the middle of the 20th century. Then the numerous longitudinal growth studies reflected the desire to learn what normal growth was and to understand variation in growth in relatively well-off circumstances. Those looking at growth in less developed countries were primarily concerned with poor nutrition and malnutrition. Growth reflected the intertwined variables of nutrition, disease and socioeconomic circumstances. Since then other concerns have emerged, such as psychosocial stress, and these have been incorporated into the growth as a mirror concept. Most effects of pollutants also are reflected in patterns of inhibited growth, but not all. The recognition that some unwelcome influences may not inhibit growth but stimulate it raises a possible challenge to the widely respected concept.

This is not the first time there has been a challenge. If growth is a mirror, what pattern of growth illustrates poor circumstances and health and what pattern illustrates the ideal ones? It is simplistic to assume that larger size or faster growth is necessarily better, although there is good evidence that in poor communities larger persons are healthier than smaller ones (Martorell 1989; Johnston and Low 1995; Nandy et al. 2004; Kuzawa et al 2010). The obesity epidemic is one such challenge, since larger is clearly not better. Thus, growth, either too small or too large or too early or too late, reflects poor circumstances surrounding development. This interpretation encompasses results from studies of pollutants as well as the current obesity epidemic. It also emphasizes the importance of developing clear expectations for growth patterns among healthy individuals.

Growth as a mirror may continue to be true, but the equation to good health now needs refinement. More than before it requires the ability to recognize growth patterns that are indicative of greater and lesser health. The understanding of tradeoffs between growth early in life and diseases later places the interpretation of growth inhibition or stimulation in the context of the lifespan. As researchers, physicians and health professionals better understand these relationships, the equation of growth with health will be refined and more accurate.

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Figure 1.
Map of the Akwesasne Mohawk Nation.

Figure 2. Levels of the sum of seven PCBs among Akwesasne youth in comparison to similarly-aged US youth (CDC 2009).

Figure 3. Weight (kg) of Akwesasne (St Regis) males and females measured in 1996–2000 (Gallo et al. 2005) and 1979 (Pfeiffer and Dibblee 1982; Helmuth 1983).

Figure 4. Median age at menarche based on logistic regression model controlling for SES and other toxicants (Denham et al. 2005).