

Obesity: the Welfare Regime Hypothesis  
Conference in Oxford  
November 17-18, 2009

## **The history of the obesity epidemic**

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# NICOLAUS STENONIVS



Danish scientist born 1638

Until his 36th year (1674) he described:

- the structure of the brain,
- the existence of exocrine glands,
- the heart as a muscle pump,
- the skeletal muscle structure-function relation explaining contraction,
- and many other things in geology, paleontology and crystallography

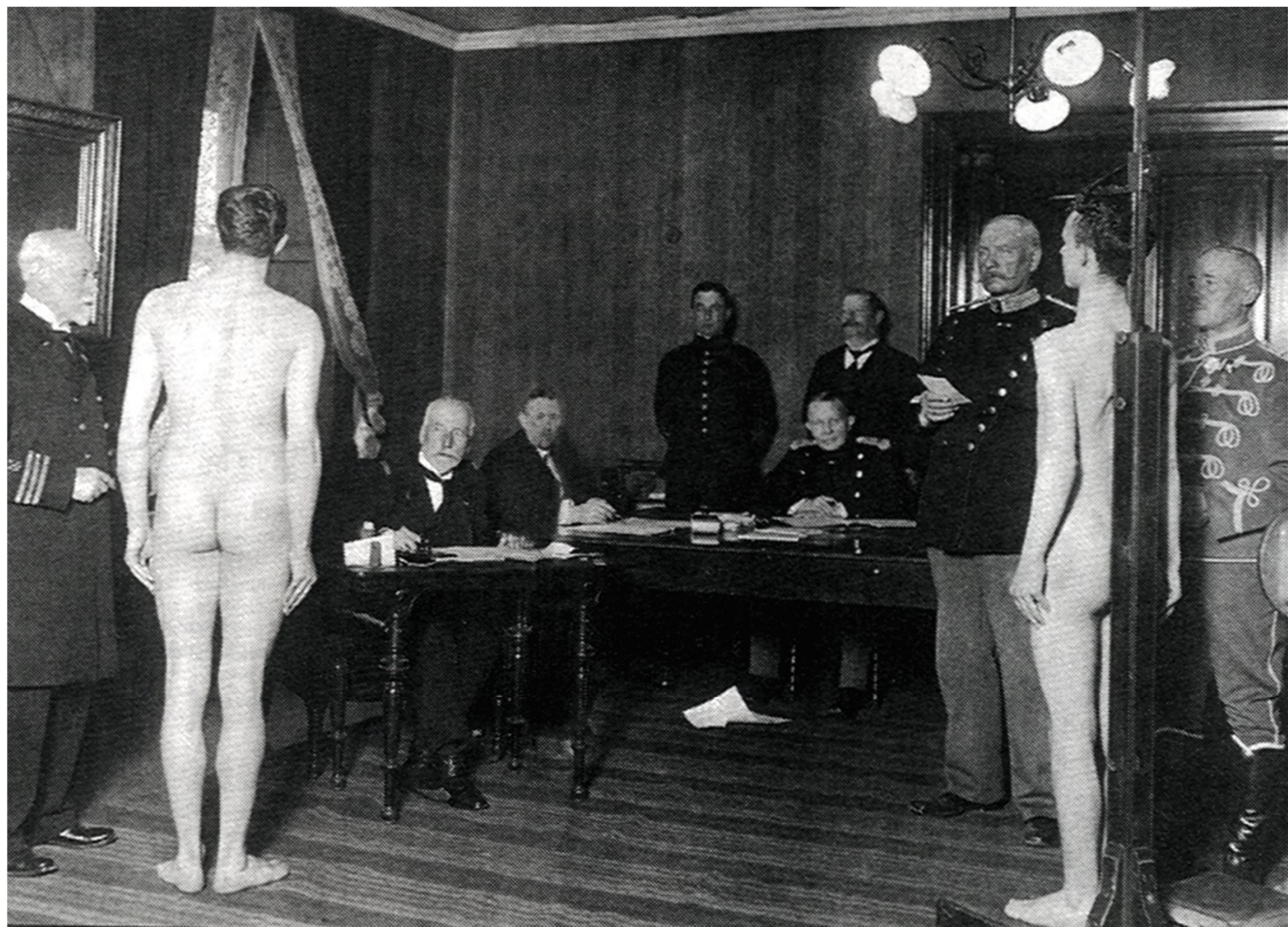
## **Lessons from the prevalence trend studies**

What can we learn from analysis of the rising prevalence of obesity during the last decades?

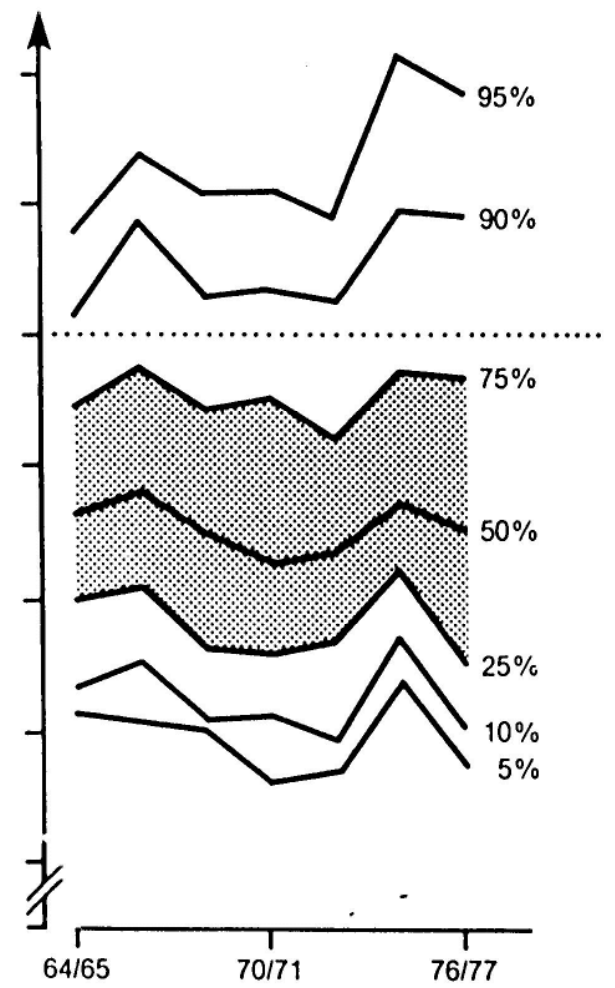
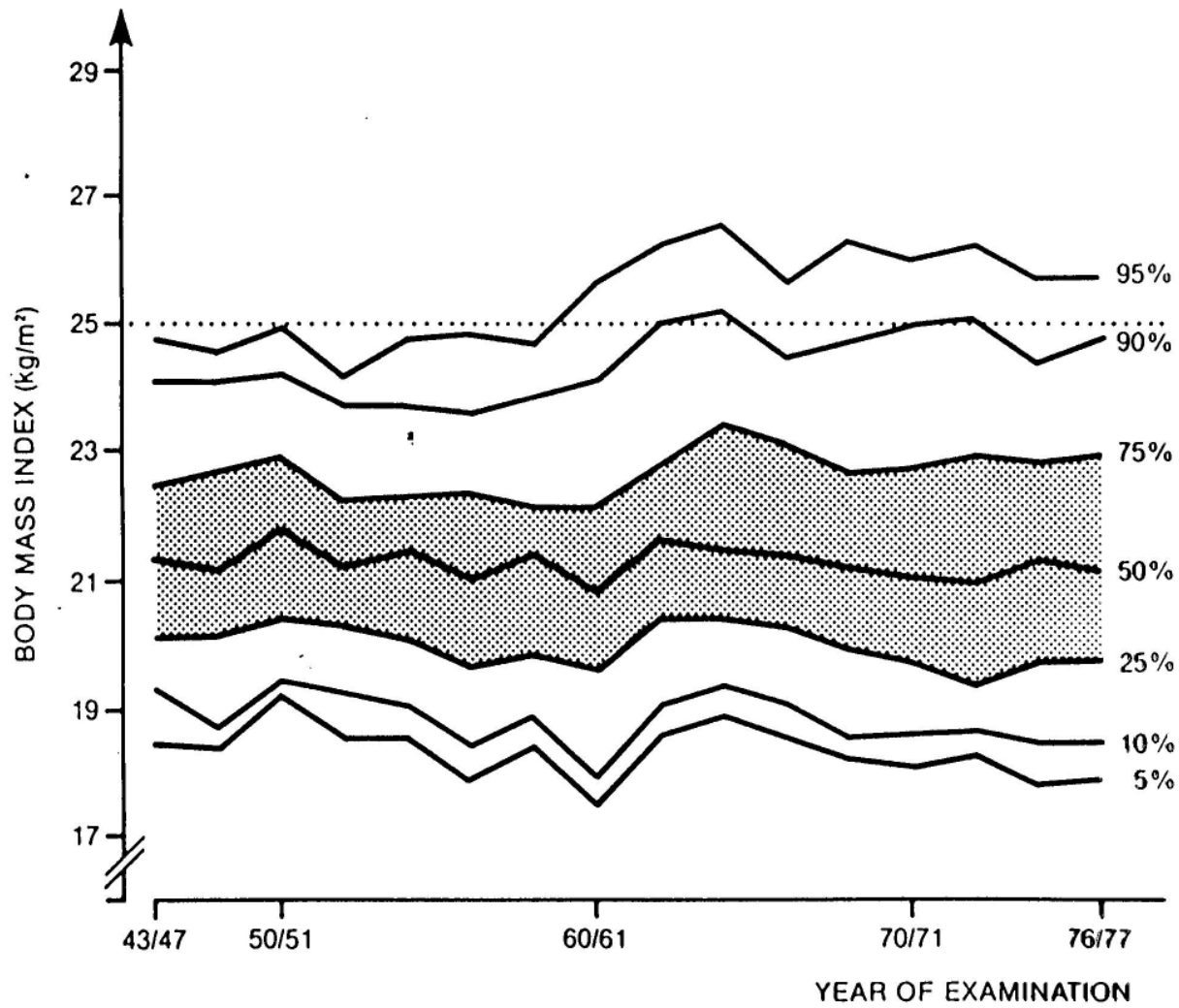
(We assume that the rising prevalence is due to rising incidence rather than rising duration of the condition.)

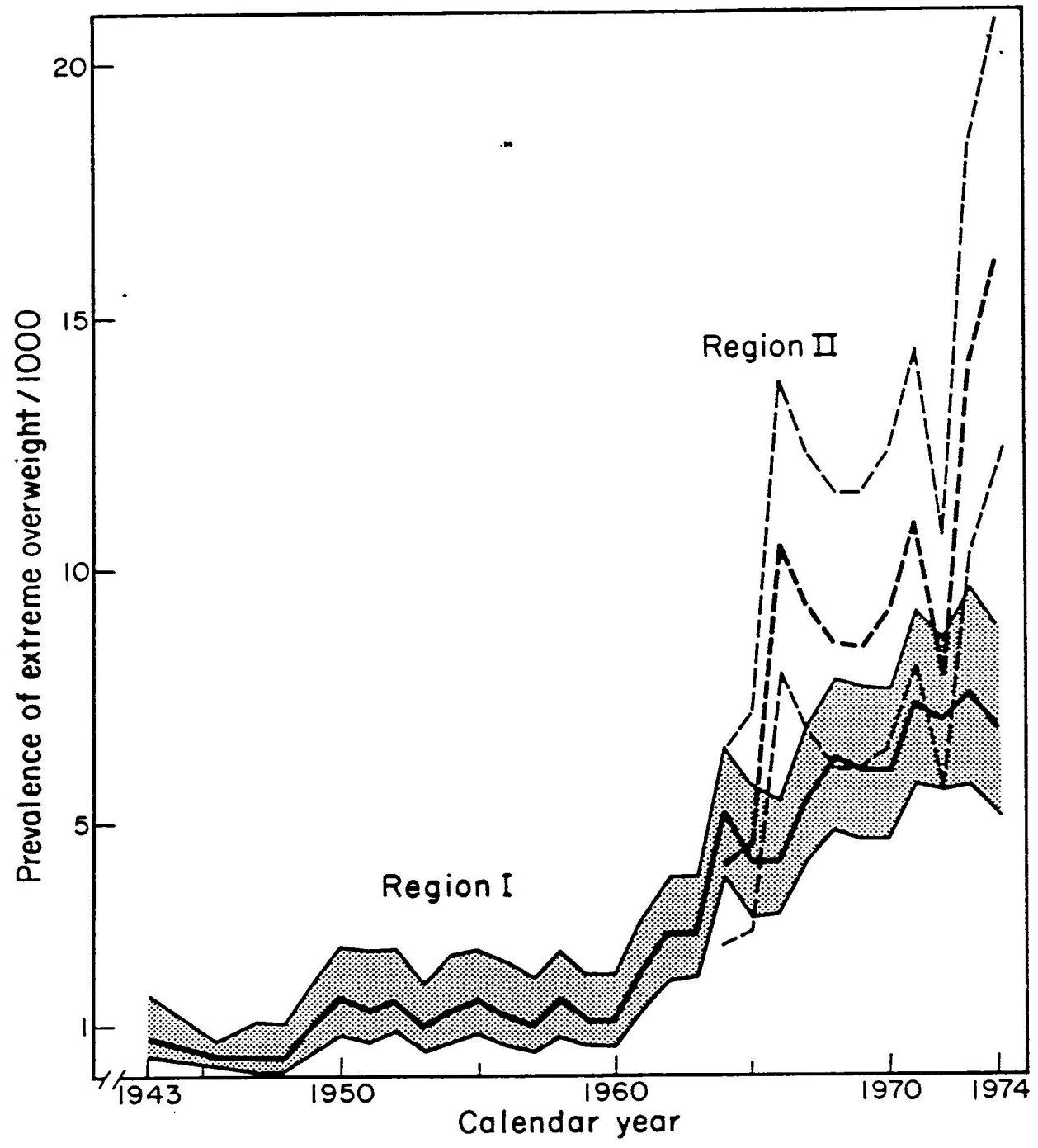
There is at least one – so far unknown – environmental determinant of obesity that has risen before the observed rising prevalence.

When – at what age in which years – has the exposure taken place? We need to know to find out what it is.

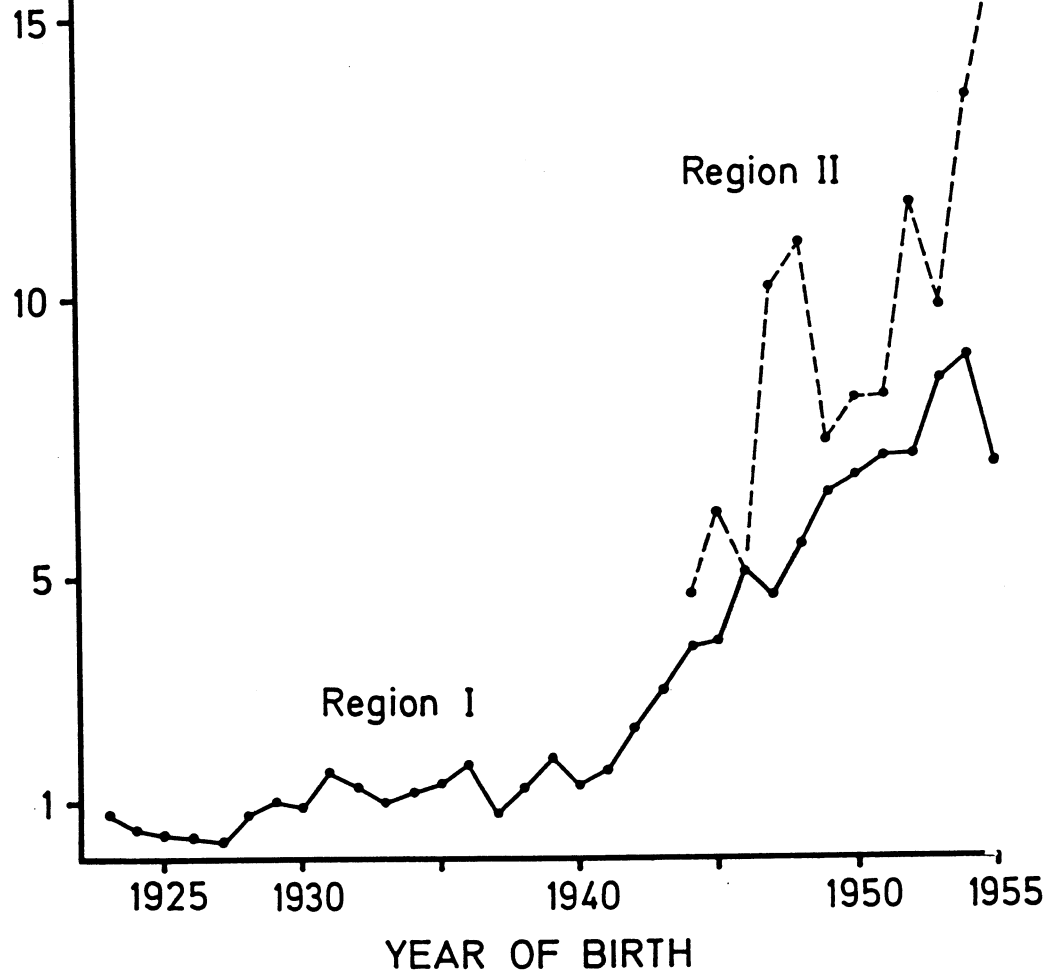


Birth cohorts	Draftees (18-23y)		Boys (6-8y)		Birth weights
	Total	Obese	Total	Obese	Total
1930-33	35159	47	12776	18	0
1934-38	45092	54	20988	20	8772
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<b>Total</b>	<b>708342</b>	<b>6367</b>	<b>163835</b>	<b>528</b>	<b>126391</b>



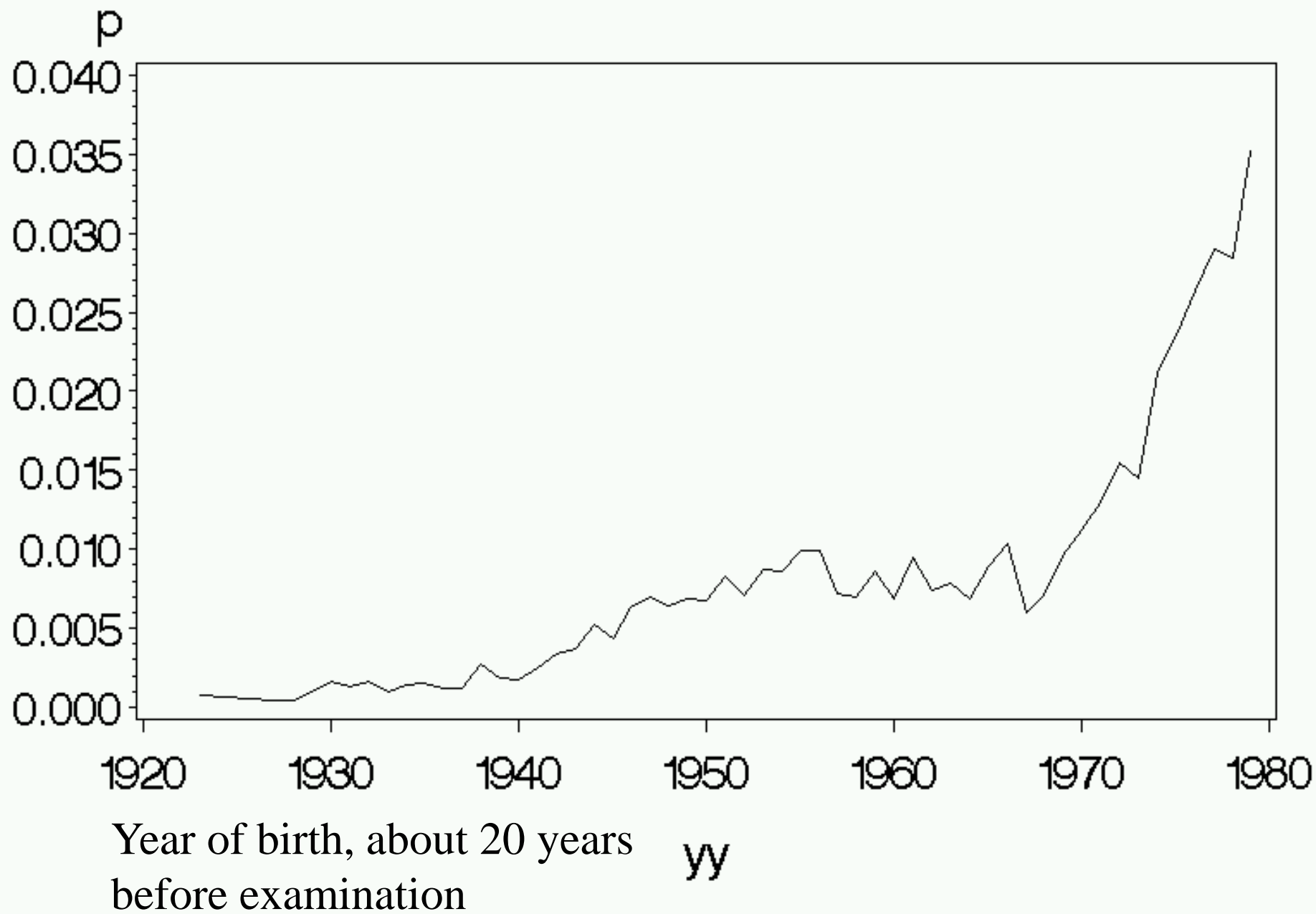


PREVALENCE  
(Per 1000)





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# **At what age did the obesity in the young men develop?**

- School health records may give part of the answer:
- From 1930 - 1983, all school children in Copenhagen attended mandatory annual health exams
  - In public and private schools
  - Weight and height were recorded on health cards
  - Parents reported birth weight at the 1<sup>st</sup> examination

# The health cards

28  
Københavns kommunale Skolevæsen  
Skolelægeinstitutionen.

Helbredskort for PIGER

(Efternavn først) [redacted] *Thomsen*

født den [redacted] 19 *34*

Forældrenes Stilling: [redacted]

Skoleaar	Klasse	Skolens Navn	Skolelægens Navn	Skoleu
19 <i>41/42</i>	<i>2 A</i>	<i>SUNDET</i>	<i>DR. F. M. LARSEN</i>	
19 <i>42/43</i>	<i>2 A</i>	"	"	
19 <i>43/44</i>	<i>3 A</i>	"	"	
19 <i>44/45</i>	<i>4 A</i>	"	"	
19 <i>45/46</i>	<i>5 A</i>	"	"	
19 <i>46/47</i>	<i>1 SEM II</i>	"	"	
19 <i>47/48</i>	<i>2 SEM II</i>	"	"	
19 <i>48/49</i>	<i>3 SEM II</i>	"	"	
19 <i>49/50</i>	<i>4 SEM II</i>	"	"	
19 <i>50/51</i>	<i>R</i>	"	"	

*Differenzvaccination: 2 gangen Besked  
23 - 19 da Hansen.*

*16 - 50 Cerebromucosum: smitsomt ikke.*

*16.50. Vaccination: ✓*

1<sup>o</sup> Undersøgelse Dato: / 19 *41*

Tidligere mere alvorlige Sygdomme:

Er nærmere Beskrivelse af det forefaldne:

Hvorledes er Børnets Udsende? *sundt med tydelig*

Hvorledes er Børnets Bygning? *hækket vedtæt spinkel*

Hvorledes er Børnets Hørd? *ikke hørdet spærnom*

Er Hudlæsion uforekommende? *✓*

Er der Hørbøgelfejl? *✓*

Er der Tegn til Hjertefejl? *✓*

Er der Tegn til Bræk? *✓*

Er der Talefej? *✓*

Er der Tegn til Sygdomme i Nase og Svælg? *Angst - trærd. see ya*

Er der Ørefejl? *✓*

Er der Tegn til Øjensygdomme? *✓*

Er der andre sygelige Tilstande? (Bærende Pladid) *hækket for tid*

Er der Tuberkulose i Hjemmet? *✓*

Var en af Forældrene til Sæde ved Undersøgelsen? *✓*

Hæmoglobinstemmelse:

Undersøgelse:

Skole- aaret

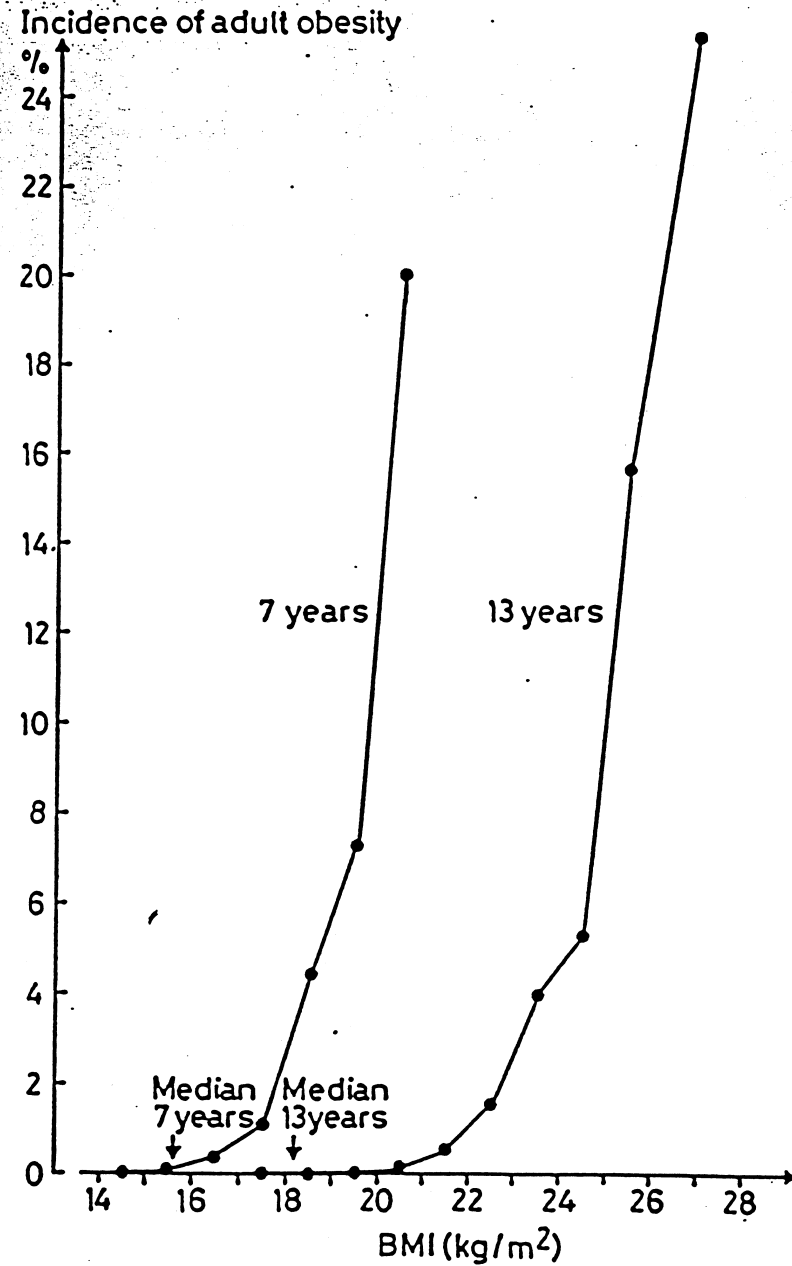
Date	Højde og Vægt		Temperatur
	Højde	Vægt	
16.5.41	117	19	+8.0
19/42	118	19	+10.0
28.9.41	118	20	+5.0
8.11.41	118.5	22.7	+5.0
18.5.43	123.4	22.6	+4.0
19.11.43	125.1	24	+5.0
19/44			
19/45			
19/46			
19/47			
19/48			
10.1.45	133.5	29.2	0
19/49			
19/50			
19/51			
19/52			
23.8.46	141	35	+1.0
19/53			
19/54			
7.12.46	145	37.1	+1.0
19/55			
19/56			
14.1.48	162	41.5	+1.0
19/57			
4.8.48	156	49.2	+1.0
19/58			
19/59			
19/60			
21.1.51	160	49	+1.0
19/61			
19/62			

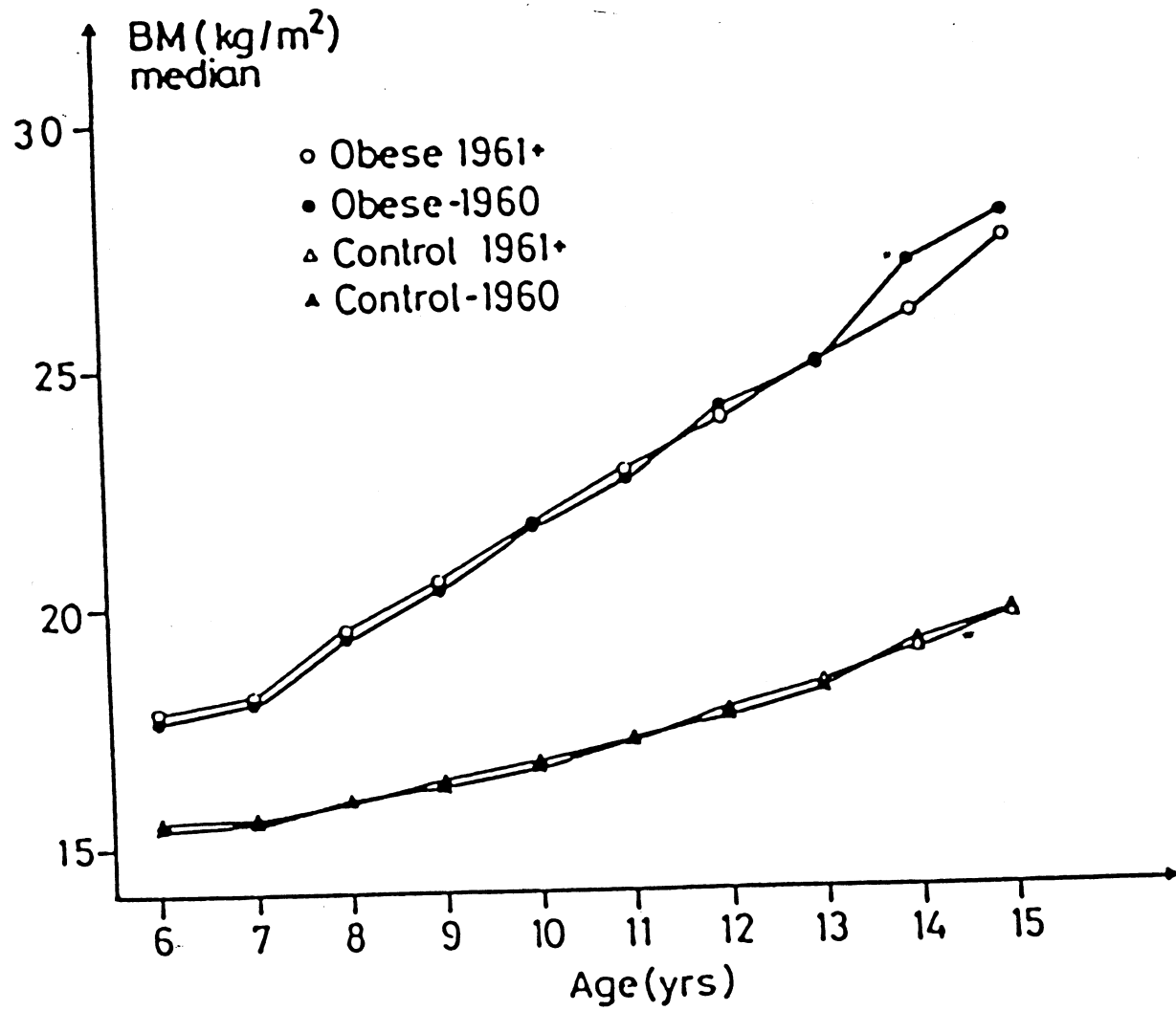
Tuberkulinprøve	Høreprøve	Synsprøve	Bemærkninger angående Børnets Helbred og hygiejniske Forhold samt fornemmelser paa Grund af Sygdom.
-	H. 6	H. 6/6	<i>Se Indtæll. Ligeledes anmærket god. Børnemærket Lot &amp; Drøning. Følge af drøning. Der er en lille god. Hækket i øjet.</i>
-	V. 6	V. 6/6	<i>19/42. Sygdomme 4/4. 9/6. 3/8. 3/9. 3/10. 3/11. 7/12. 2/1. 1/6. 4/2. Følge.</i>
-	H. 6	H. 6/6	
-	V. 6	V. 6/6	
-	H. 6	H. 6	
-	V. 6	V. 6	
-	H. 6	H. 6/6	
-	V. 6	V. 6/6	
-	H. 6	H. 6	
-	V. 6	V. 6	
-	H. 6	H. 6/6	
-	V. 6	V. 6/6	
-	H. 6	H. 6	
-	V. 6	V. 6/6	
-	H. 6	H. 6/6	
-	V. 6	V. 6/6	
-	H. 6	H. 6/6	
-	V. 6	V. 6/6	
-	H. 6	H. 6	
-	V. 6	V. 6	
-	H. 6	H. 6/6	
-	V. 6	V. 6/6	
-	H. 6	H. 6	
-	V. 6	V. 6/6	
-	H. 6	H. 6	
-	V. 6	V. 6	

# The records



Case-control nested in cohort study (designed as a case-cohort study) of childhood BMI in relation to be obese at draft board examination





## **At what age did the obesity in the young men develop?**

- The obesity developed both before, during and after school age.
- The relationship between school age BMI and later risk of obesity was the same before and after the onset of the epidemic.
- Had the onset of the epidemic among the young men been initiated after they left school, then the curves for the obese examined after 1960 would have been lower.

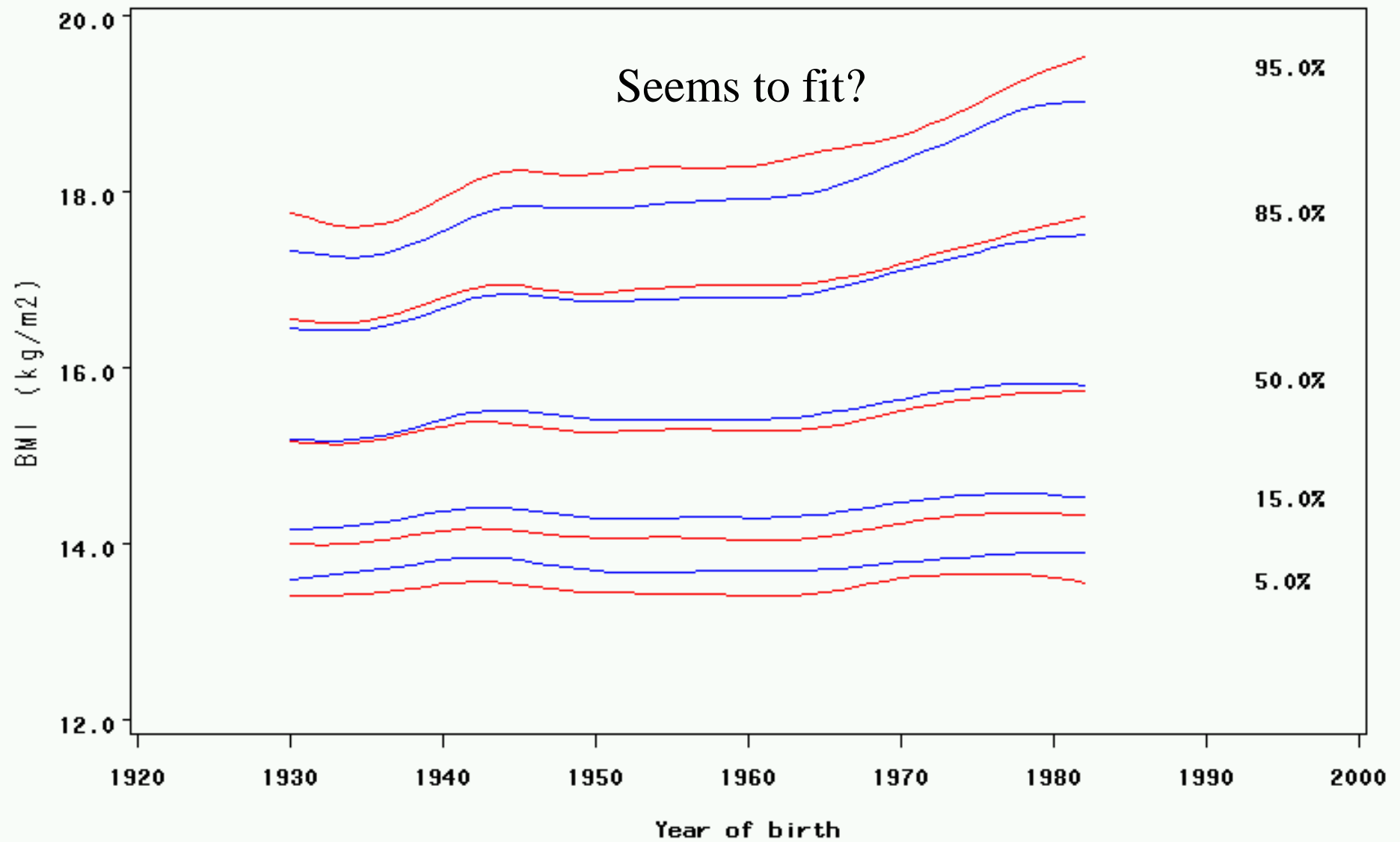


## **At what age did the rise in prevalence first occur?**

- The school health records may give part of the answer
- Could not be answered by the case-cohort sample, because the cohort subsample was too small to address this question.
- Computerization of the weight and height measurements of all children's school records was undertaken

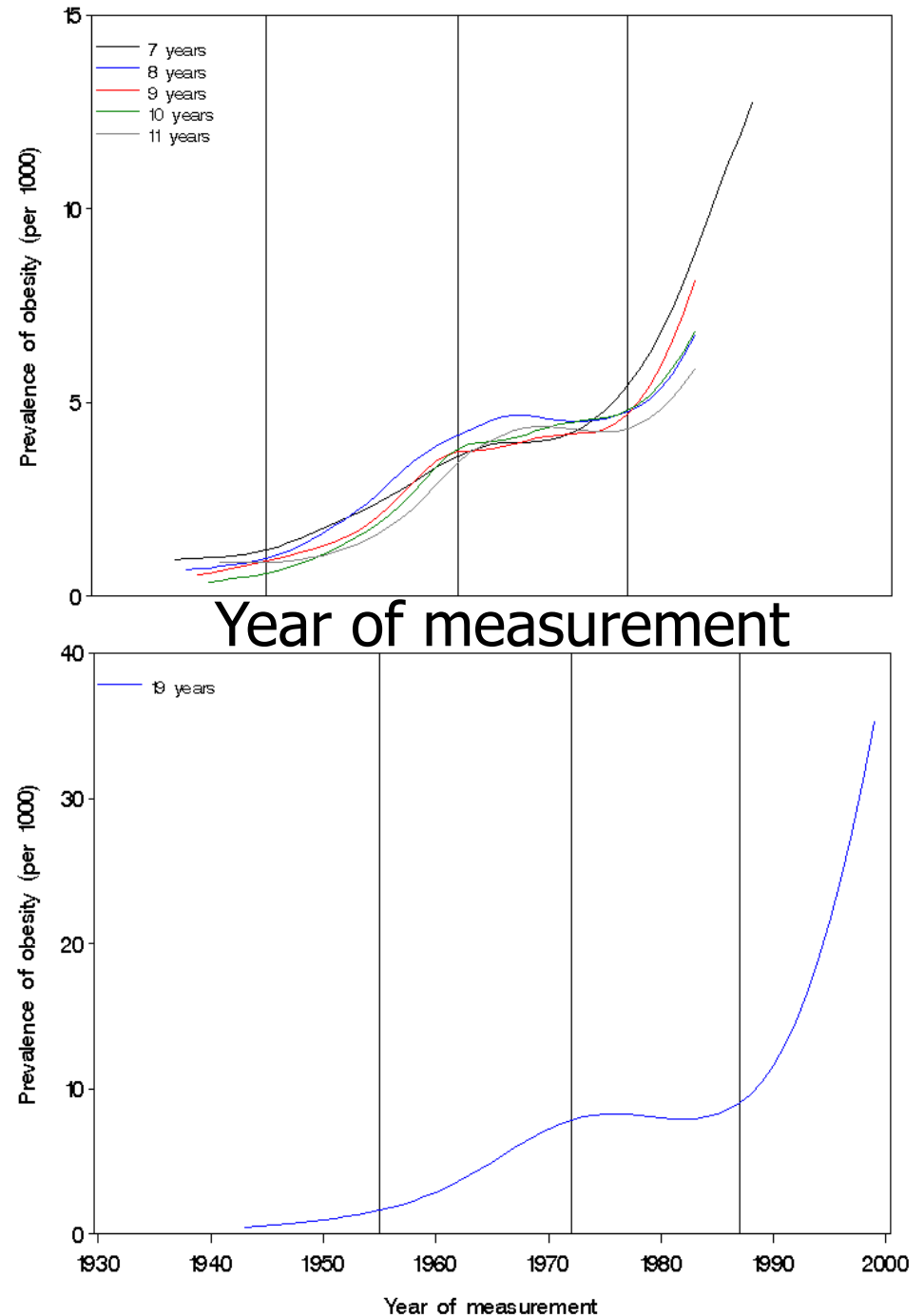
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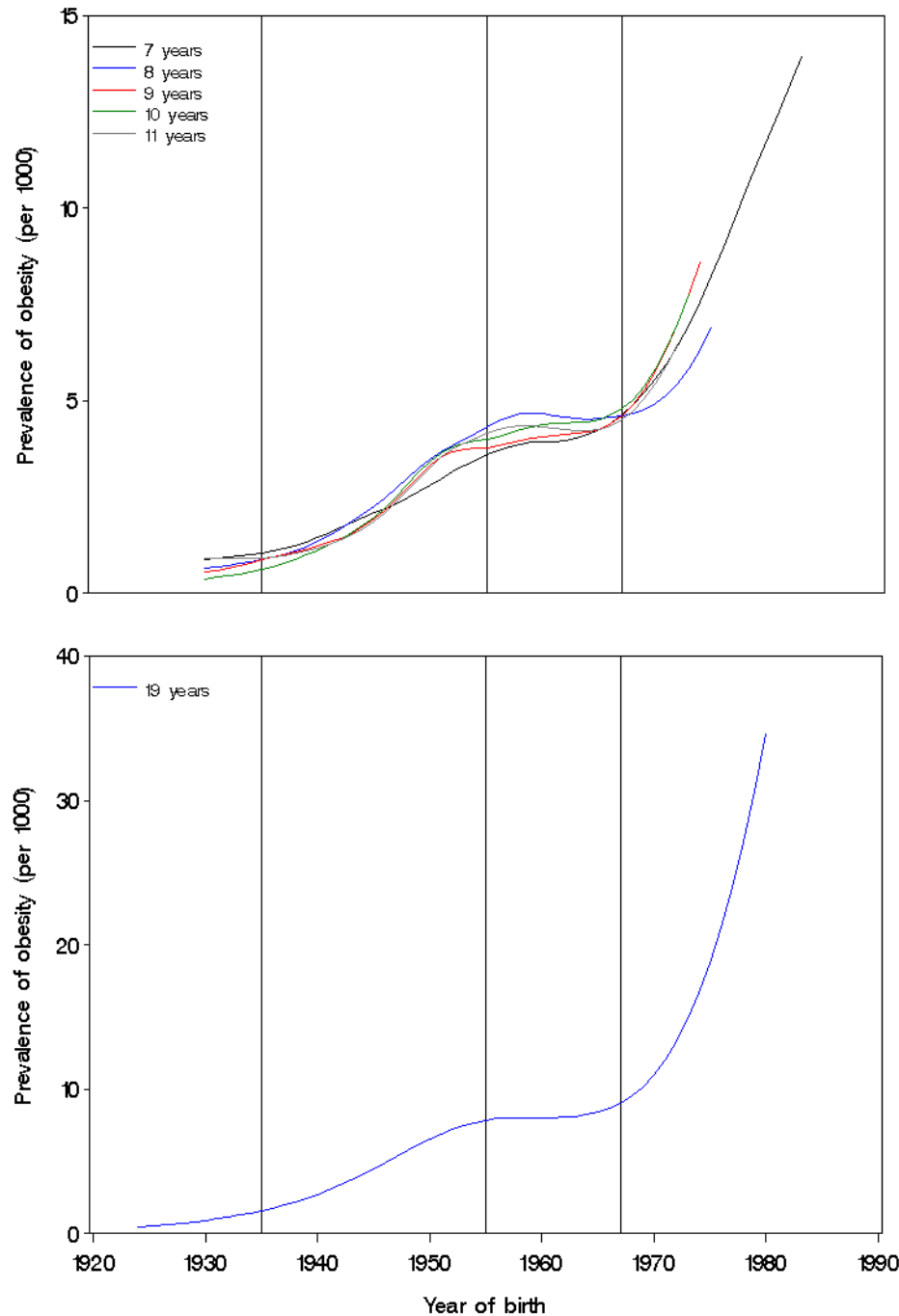
# BMI Percentiles for 7 years old



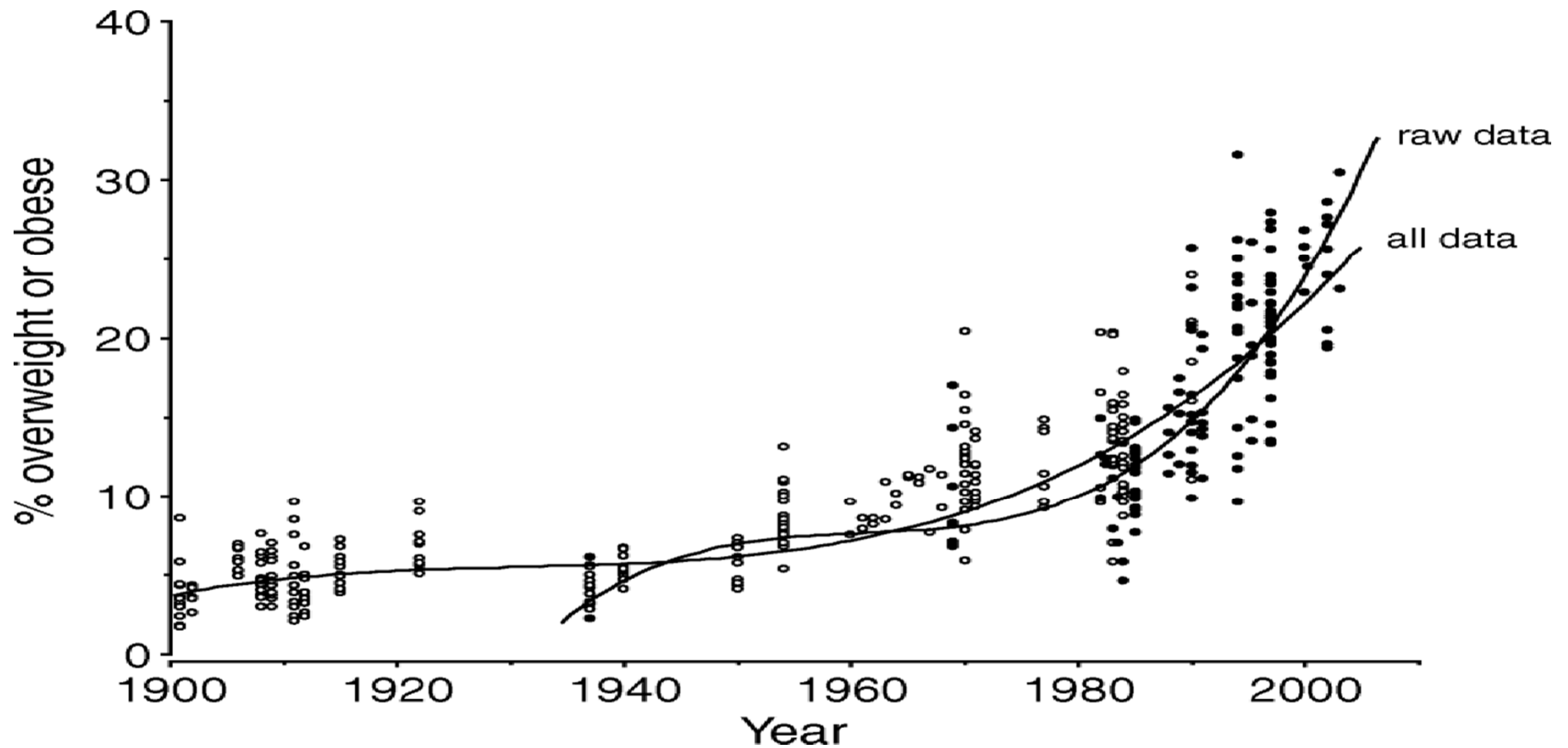
Male=blue, Female=red

- The obesity epidemic is affecting the school-boys much earlier than the draftees and obviously before the economic growth began!
- Also, the environmental changes driving the epidemic did not affect individuals at any of these ages equally.
- Note that the changes occurred about 10 years later in the about 10-year-older young men.

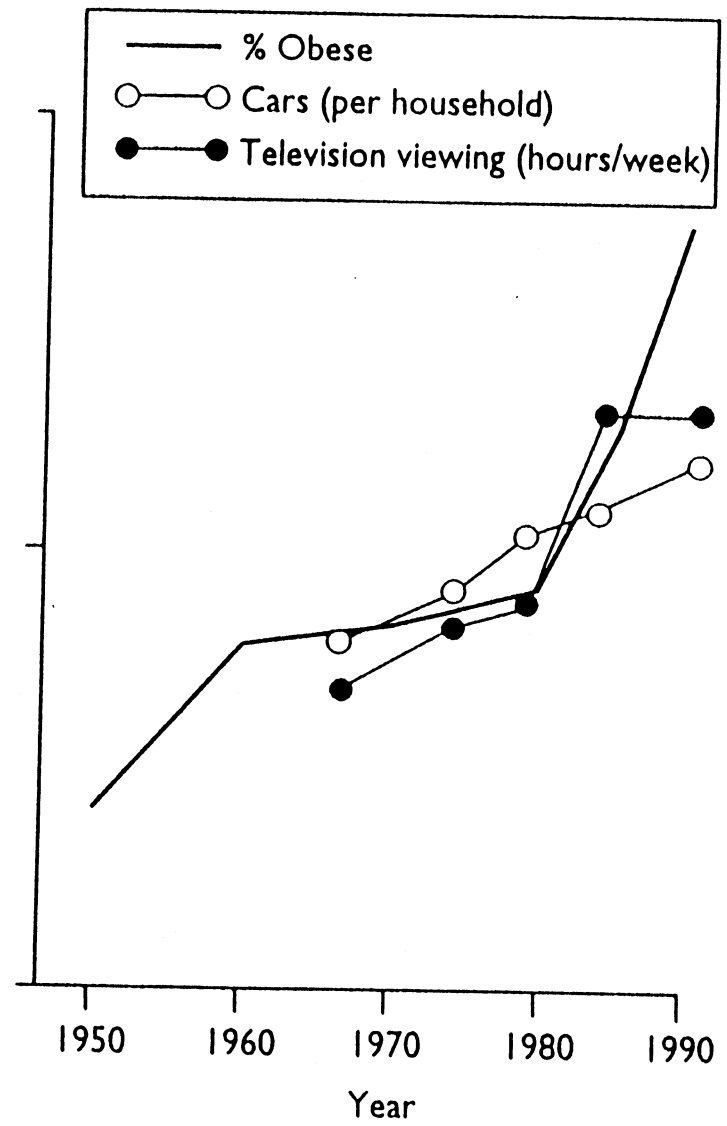
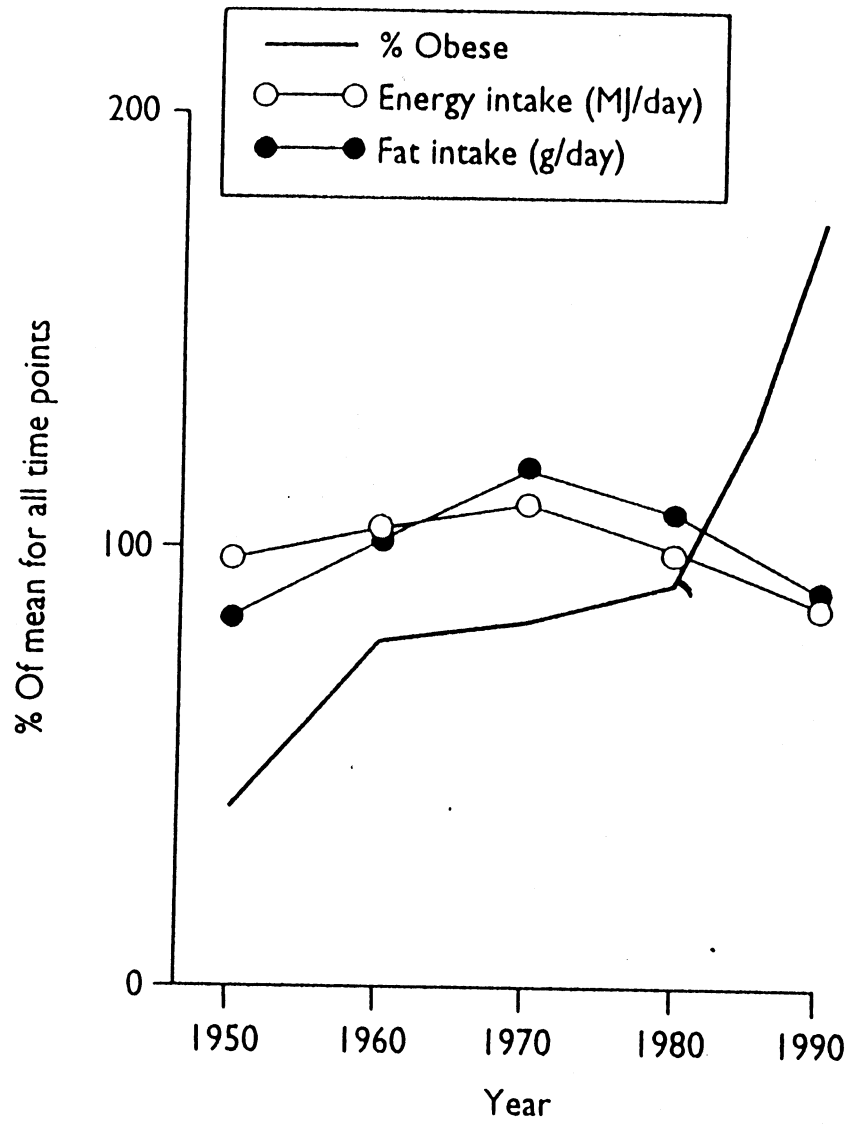




- The trends coincides by year of birth
- Implies that the environmental changes driving the epidemic operate around the time of birth – the individuals may become susceptible within the first years of life.
- This susceptibility may contribute along with the genes to the well known tracking of obesity over the ages.



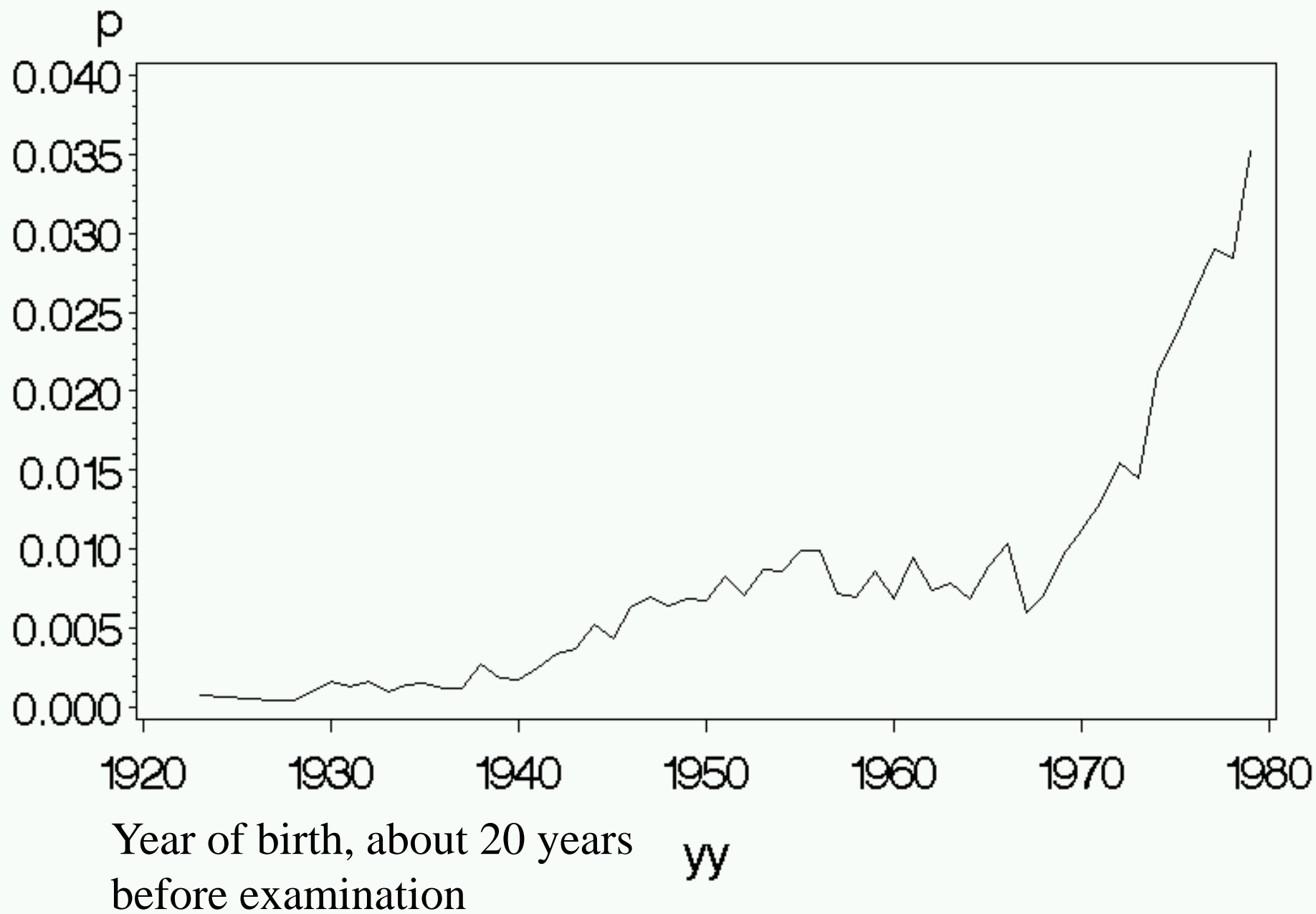
**Prevalence of overweight and obesity for 5–15-year-old Australian children. Lines of best fit (3<sup>rd</sup> order polynomial) are shown for all data combined ( $r^2=0.83$ ; RMSR = 2.2%) and for raw data only ( $r^2=0.75$ , RMSR = 3.3%). Open circles are age and gender specific prevalence rates based on descriptive data. Filled circles are derived from raw data. Each circle represents an age and gender slice for a single study.**



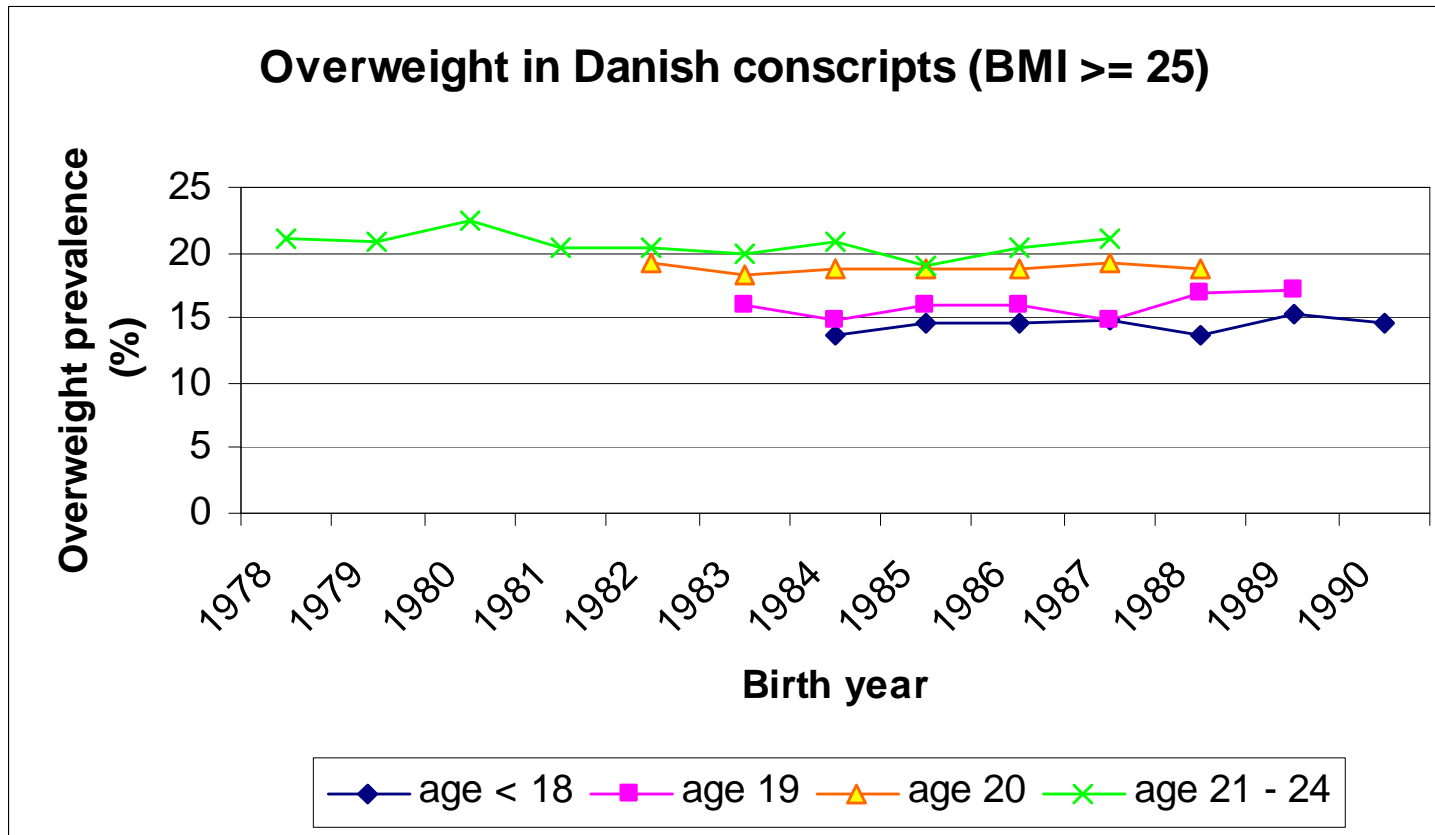
# Expectations about the recent trends in the obesity epidemic

- The epidemic of obesity has been developing rapidly over the past decades in most parts of the world.
- Prediction has been made that the trend would continue in the forthcoming decades and that a majority of the people would be obese in 10-20 years time in several countries.
- Assuming unchanged relations between obesity and associated health problems, co-morbidities, and mortality, this prediction translates to huge public health problems that would challenge all sectors of the societies, inside and outside the health care sector.
- Recent evidence suggests that the epidemic is taking a break



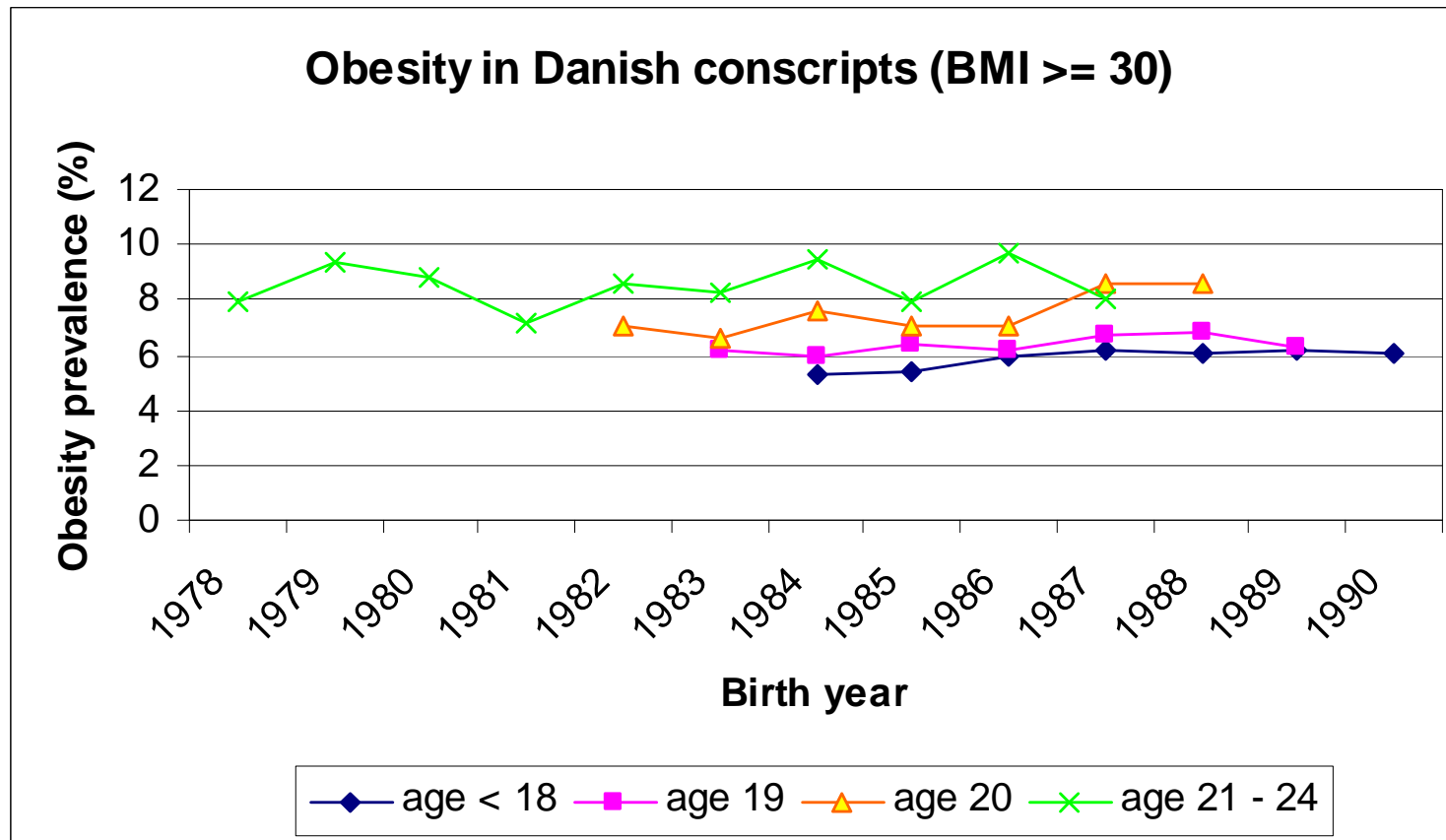


# Overweight in Danish young men



<http://www.forsvaretsuddannelser.dk/ForsvaretsDagOgVaernepligt/Pages/bmi.aspx>

# Obesity in Danish young men



<http://www.forsvaretsuddannelser.dk/ForsvaretsDagOgVaernepligt/Pages/bmi.aspx>

Figure 1

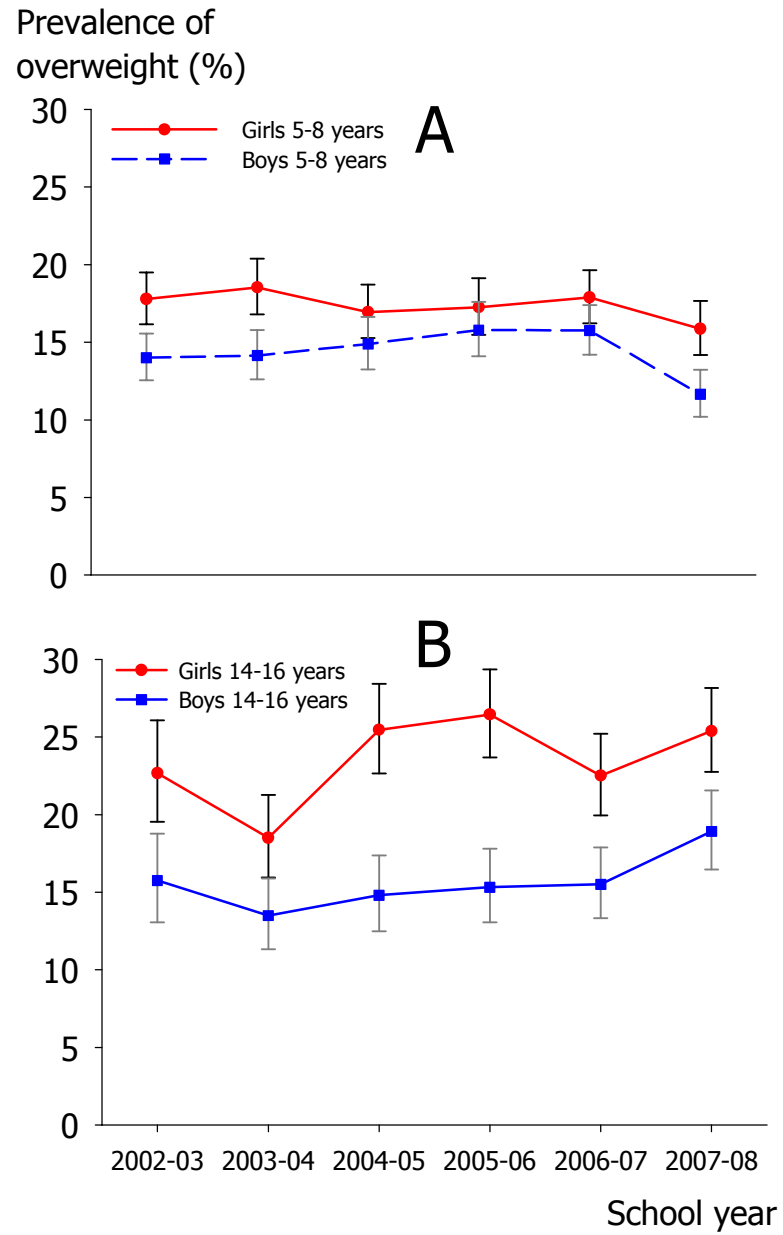
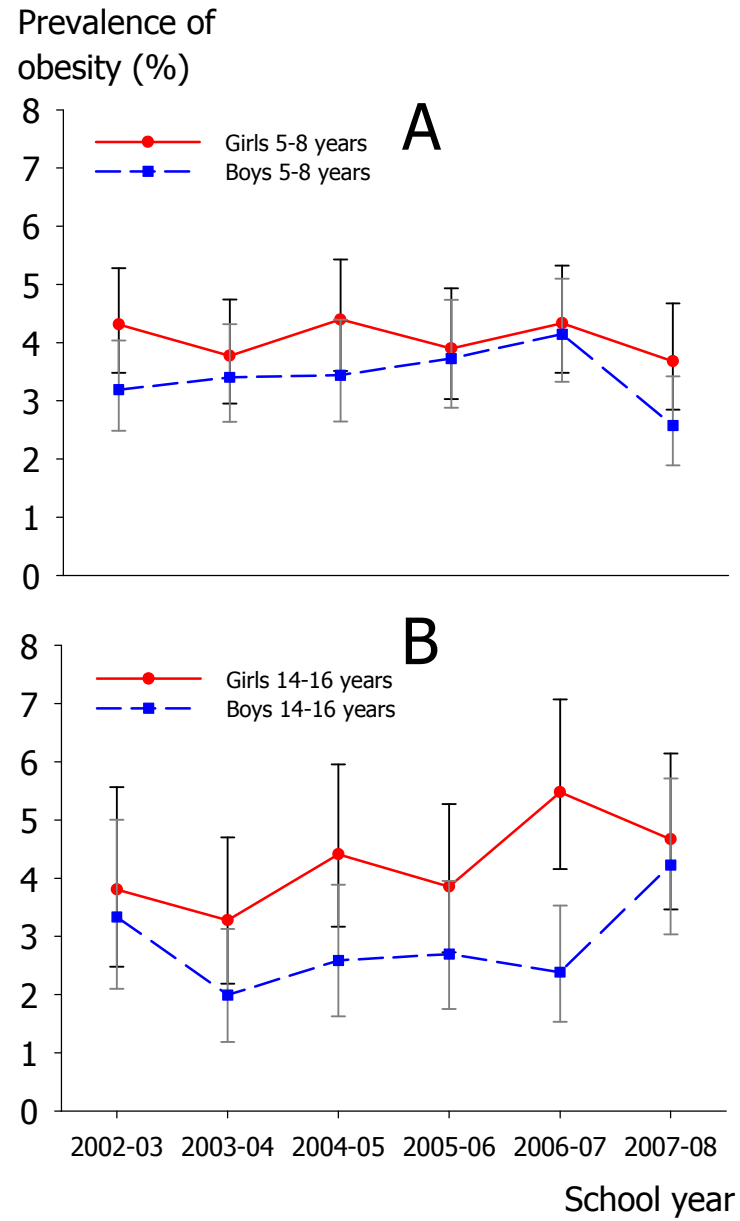


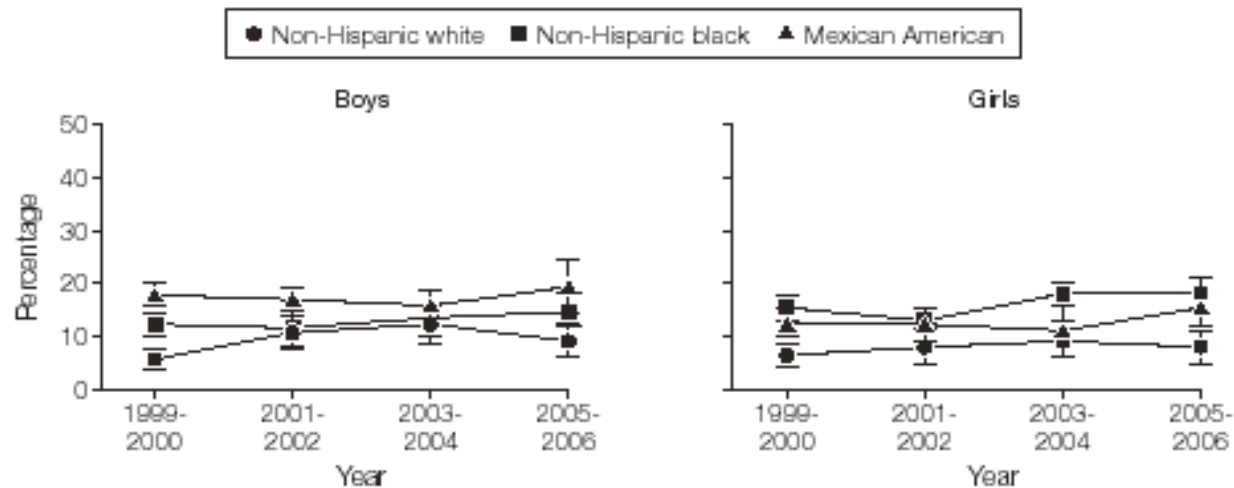
Figure 2



# U.S.A. children and adolescents

(National survey – 97<sup>th</sup> percentile)

**Figure 1.** Body Mass Index for Age at or Above the 97th Percentile by Race/Ethnicity in 1999-2006



Error bars indicate 95% confidence intervals. Participants were aged 2 through 19 years.

Ogden et al. High body mass index for age among US children and adolescents, 2003-2006; JAMA, 2008.

## Summary

- The prevalence of obesity and overweight has increased dramatically during the past 60 years.
- The epidemic has developed in two waves, about 30 years apart, between three stable periods
- There seems to be conditioning causes of the epidemic around time of birth.
- These causes may constitute a permanent susceptibility that contribute to the tracking of obesity in later life.

## **Find the target for prevention?**

If the perinatal origin of the epidemic is true, it will pave the way for a preventable target in a limited time window at an age, where preventive actions are commonly accepted, such as vaccinations.

The obesity epidemic is by nature a historical phenomenon, and I may require researchers in history to identify the driver of the epidemic.

The understanding of the history of the epidemic may provide the tools to avoid it in the future!





The obesity epidemic taking a break, but it may just  
be silence before the storm!

I hope my dream come through before:  
...that we one day may be able to prevent obesity by  
a specific, short-term modifications in the early  
postnatal life where a pre-conditioning for  
development of obesity seems to be set.


Thank you to all my collaborators and to you for your attention  
Have a safe trip into the future!

# NICOLAUS STENONIVS



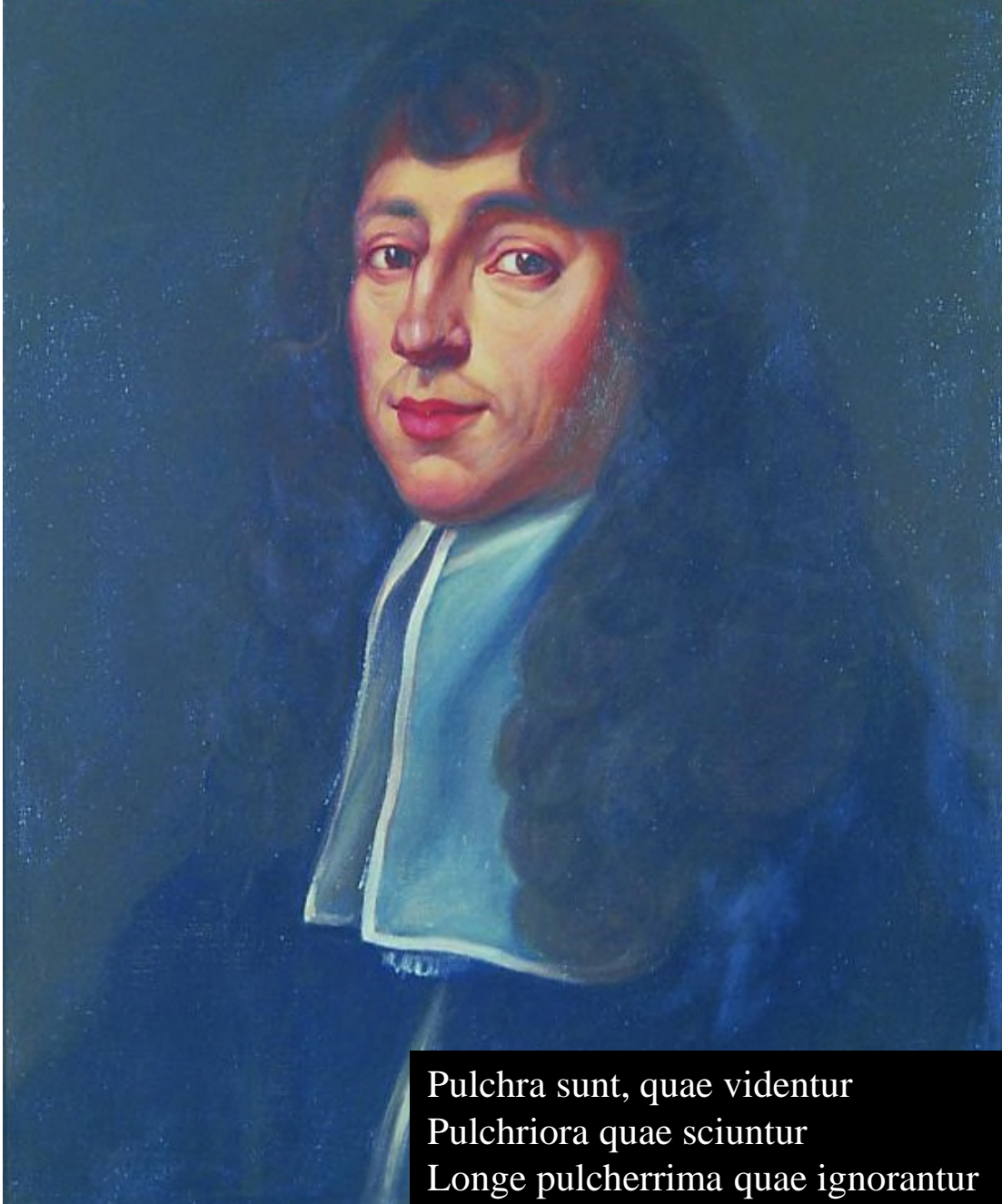
When 36 years old  
Realising that he could not get  
Deeper in understanding by science  
He converted to Roman Catholicism  
Became priest  
Bishop in Northern Germany  
Died very poor 48 years old in 1686  
Became Saint 1988

# NICOLAUS STENONIVS

A portrait of Nicolaus Stenonius, a 17th-century Danish naturalist and theologian. He is depicted from the chest up, wearing a dark, heavy coat with a large fur collar and a white cravat. The background is a dark, textured blue. The name 'NICOLAUS STENONIVS' is written in gold, serif capital letters at the top of the image.

Pulchra sunt, quae videntur  
Pulchriora quae sciuntur  
Longe pulcherrima quae ignorantur

# NICOLAUS STENONIVS



Pulchra sunt, quae videntur  
Pulchriora quae sciuntur  
Longe pulcherrima quae ignorantur

Beautiful is what we see

More beautiful is what we know

Most beautiful is what we do not know



# Key problems in assessment

- Even though the studies are conducted in geographically defined populations, migrations and developments may induce changes over time in the composition of the population with regard to characteristics associated with obesity prevalence, such as gender, age, ethnic groups and social classes.
- The same methodology (sampling design, measurement techniques, analytical definitions and estimation methods) should be used across the time period.
- Declining response and participation rates by increasing degree of obesity and over time should be taken into account when using data based on voluntary provision of information from the individuals.

# Key problems in interpretation 1

- Recent trends over short time periods should be interpreted on the background of the long-term preceding history of the epidemic.
- Longer time periods may be needed to ascertain the trends.
- Is stabilization of the prevalence by time reflecting a mixture of opposite trends, some still increasing, some decreasing?
- With the observed non-linear development of the epidemic, predictions into the future become entirely dependent on assumptions made about the future non-linear pattern.

## Key problems in interpretation 2

- Are the changes reflecting corresponding changes in the concurrent pressure from the obesogenic environment or is it due to preceding changes in perinatal predisposing determinants, possibly transmitted across generations (changes by year of birth vs current calendar years)?
- Are the changes in prevalence due to changes in incidence or persistence or both (prevalence = incidence x duration)
- Are there differential changes in the various types of obesity defined by body fat distribution?

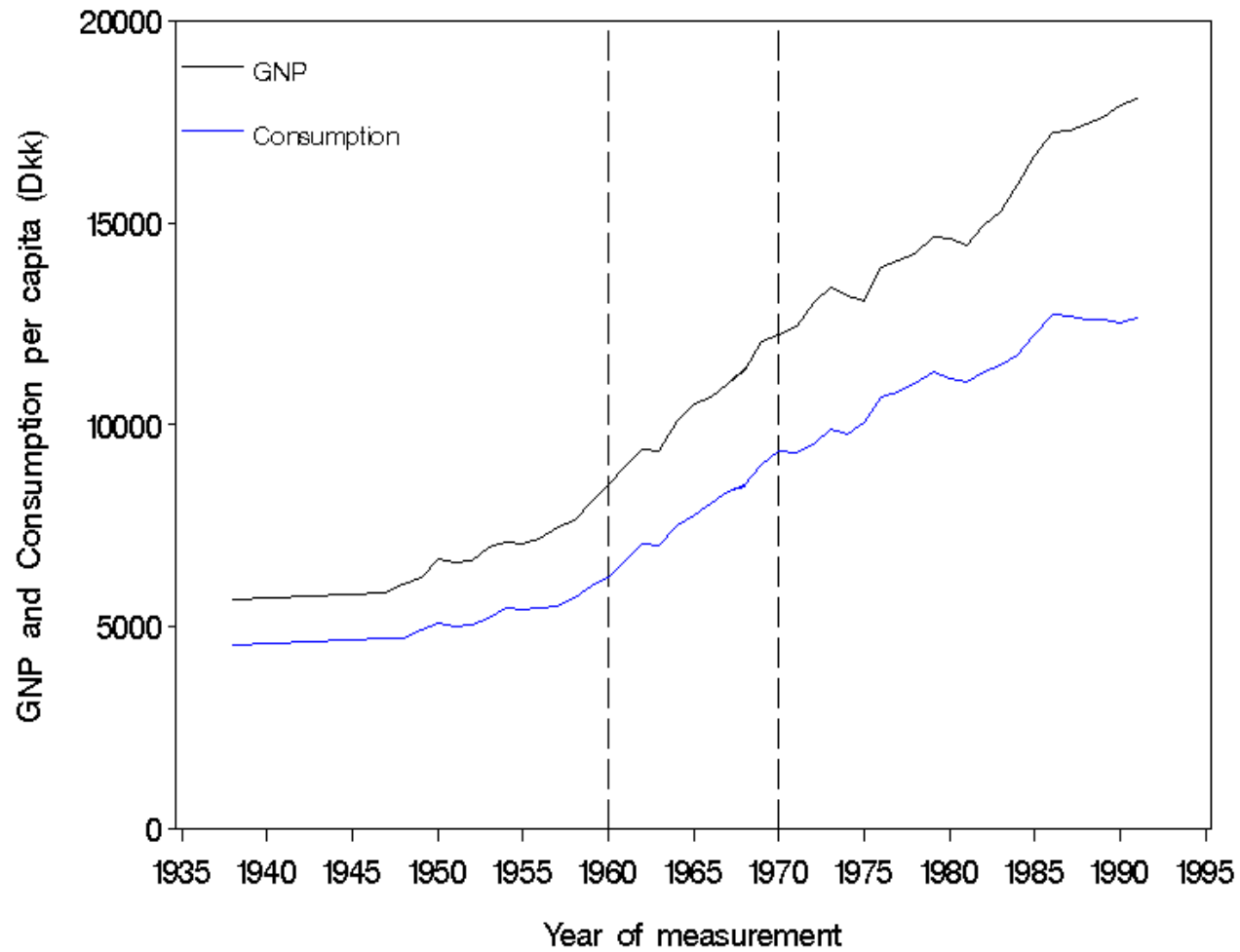


## **Economic measures**

Realising the obesogenic environment may be a multitude of elements in the society, could global economic measures be used as a measure of this environment?

The expectation is of course that the changes in the environment would facilitate increased food intake and less physical activity which hence would lead to the epidemic developing in parallel with the economic growth in a society like the Danish one.

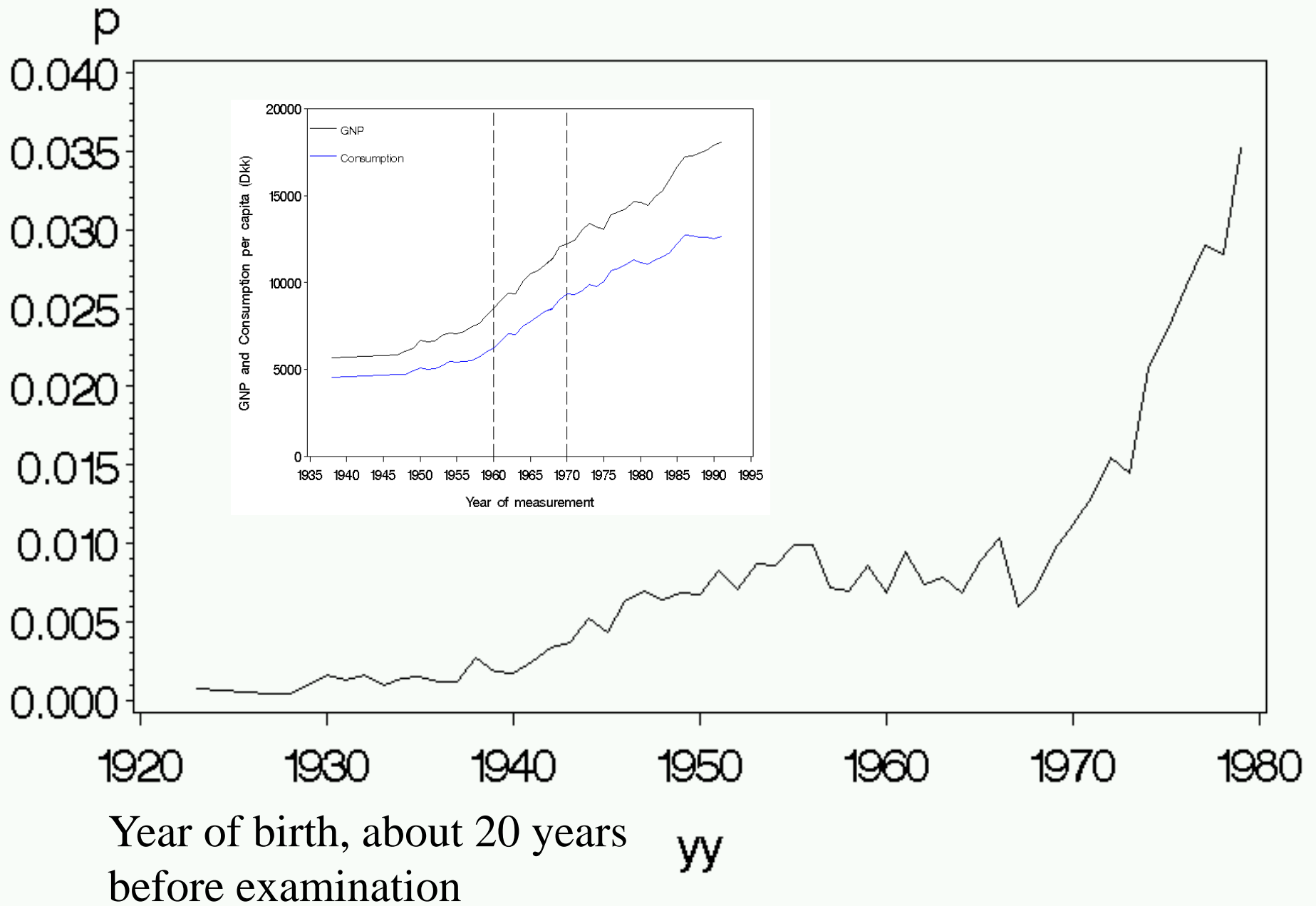
## Development of the 'obesogenic environment' assessed by global economic measures in Denmark



## **Expected time course**

The question now is if the obesity epidemic did develop in the same way with a sufficient lag time between changes in the indicators of the obesogenic environment and the changes in occurrence of obesity?

If this is not the case, we can discard these measures as adequate indicators of the obesogenic environment creating the changes in the development.



## **Seems to fit?**

The increase in obesity prevalence among the young men began a few years after the take off in economic growth and a presumably susceptible subset reacted to this change, but if the epidemic developed earlier, ie between 1942 and 1950, it would not fit...

What happened after 1974, when the economic growth continued?

At what age did the obesity in the young men develop and at what age did rise in prevalence begin?

Answers to these questions would provide two critical tests of the hypothesis.

## **However, it did not fit...**

The increase in obesity prevalence after 1974 (birth year around 1954) did not fit with the continued economic growth.

While the economy continued to grow, there was a leveling off in the prevalence and stable period of about 20 years, followed by a new sharp increase.

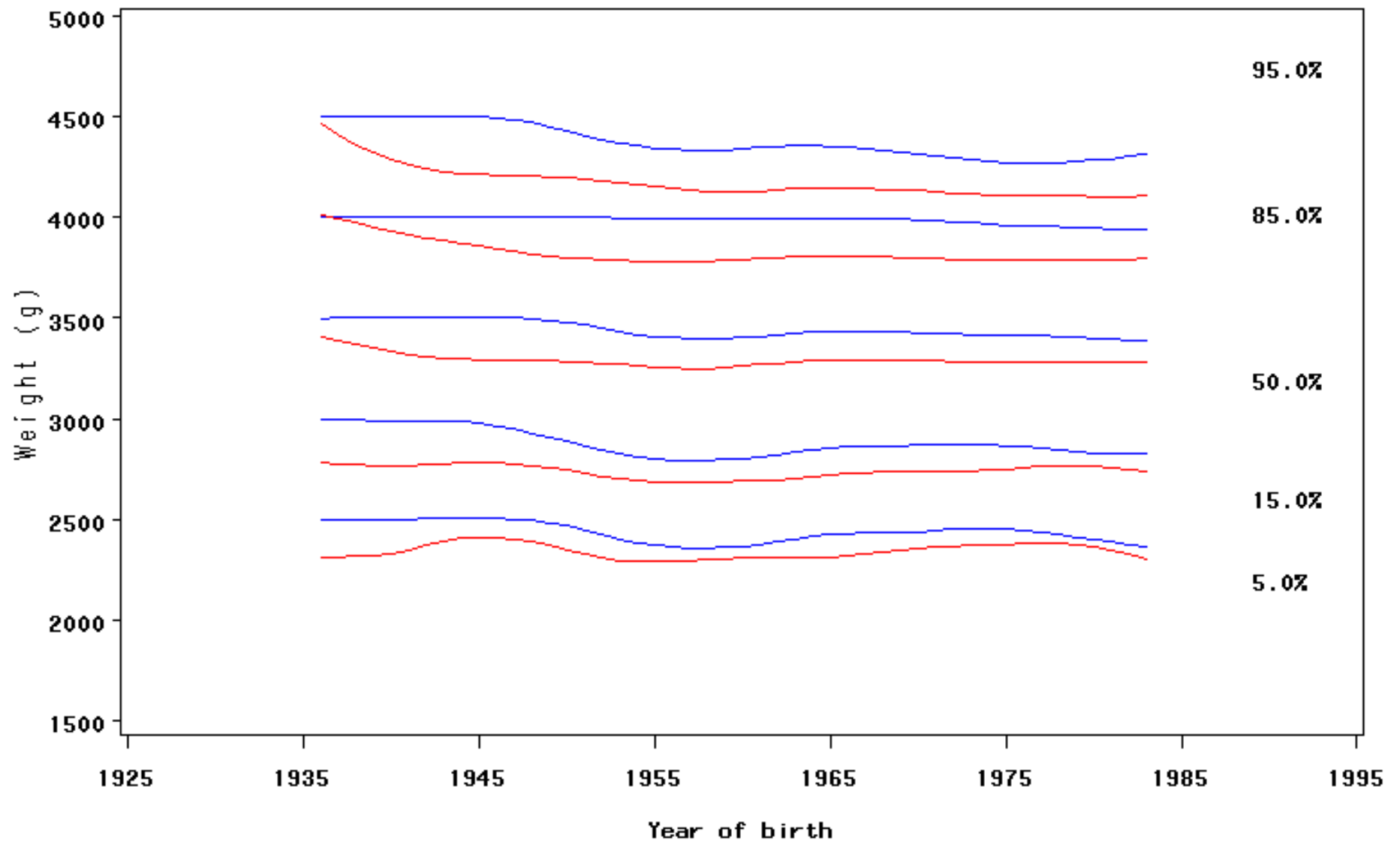
## **New questions – some answers**

- At what age before age 7 years did the epidemic take off?  
Don't know yet.
- Is it taking off already before birth?  
Let's see.
- Is birth weight related to later risk of obesity?  
Let's see
- Is the association between birth weight and later obesity changing as the epidemic evolves?  
Let's see

Birth cohorts	Draftees		Boys		Birth weights
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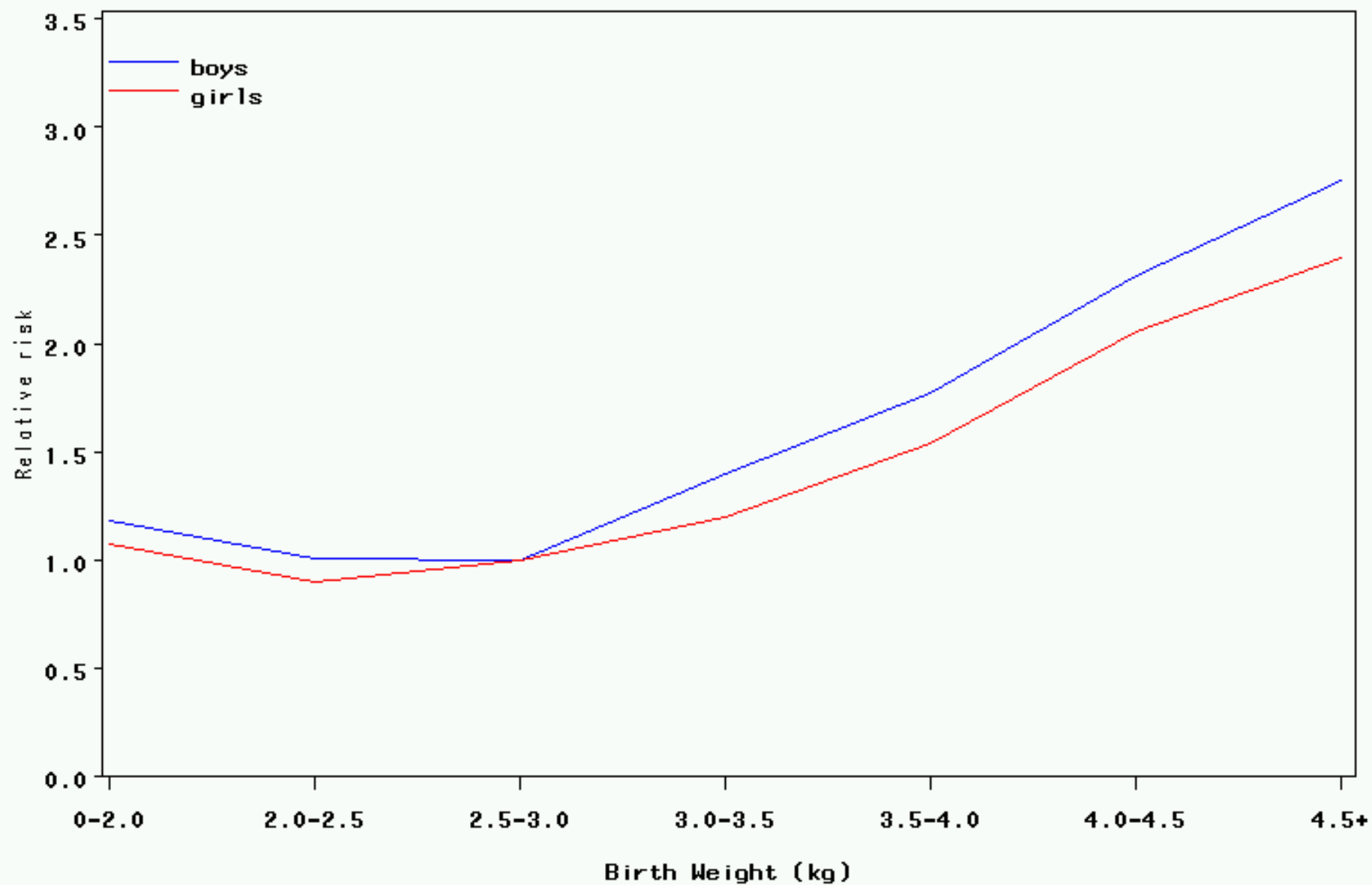
# Birth weight



Male = blue, Female = red

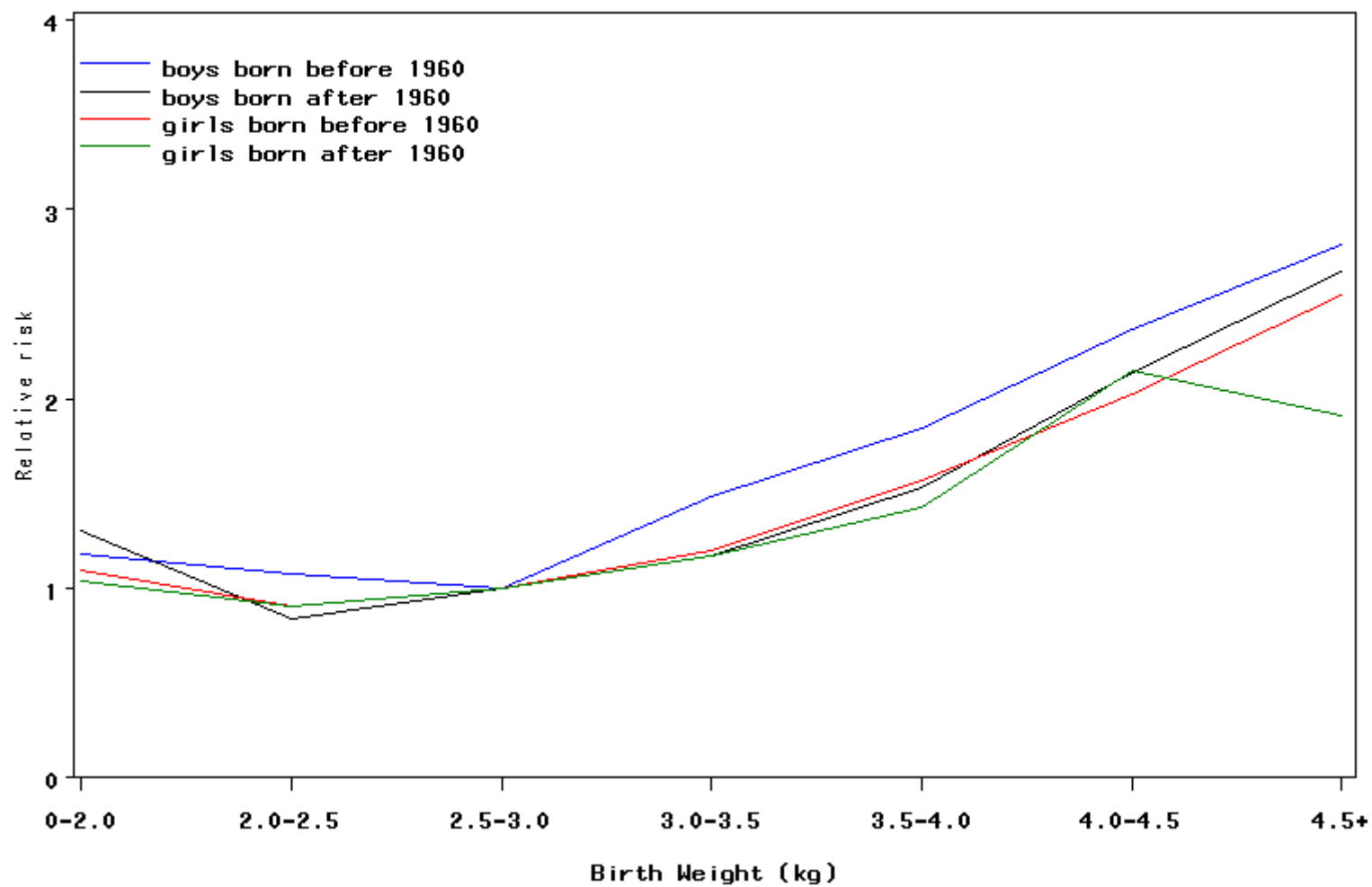
# Overweight vs Birth Weight

Age (year)=8



# Overweight vs Birth Weight

Age (year)=8

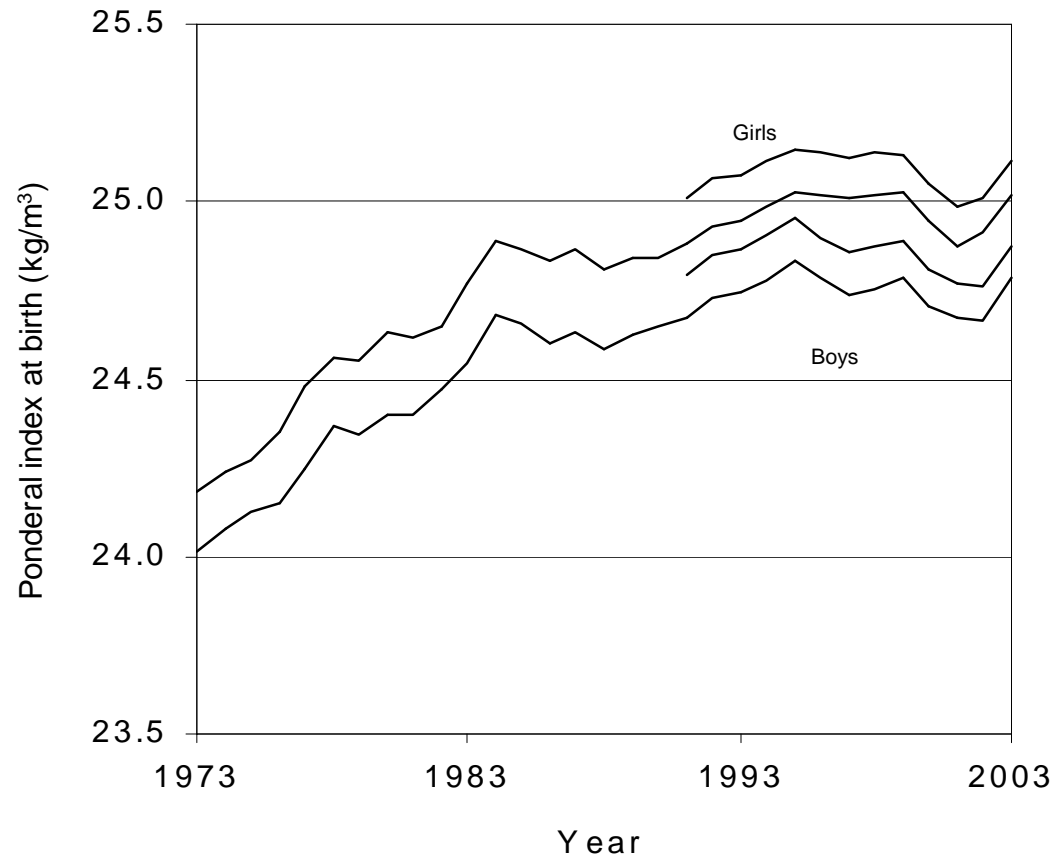


## **Pre- or postnatal exposures?**

Is the causes operating already before birth, and if so, do they lead to increased birth weight?

## More questions

- Has the birth weight increased during recent years?  
Let's see.
- If so, what are the consequences for the obesity epidemic?
- Is maternal prepregnancy obesity or pregnancy weight gain driving the epidemic?



## **Weightless adiposity at birth?**

Even though birth weight is related to later risk of developing obesity, there is no indication that the epidemic is manifest already at that time in life.

It cannot of course be excluded that the the exposure to the crucial causes and their action has begun before birth without leading to increased birth weight (the 'weightless adiposity' induced by dietary exposure to adipogenic polyunsaturated fatty acids according to Gerald Ailhaud).

## **More questions – some answers**

- Has the birth weight increased during recent years? - Yes
- If so, what are the consequences for the obesity epidemic?  
- Don't know yet.
- Is maternal prepregnancy obesity and/or pregnancy weight gain contributing to the epidemic? - Perhaps.



## **Is it the caring for the children that has changed?**

One hypothetical possibility of development of individual biological susceptibility could be some form of chronic stress, particularly derived from sustained insecurity in life, that via psycho-neuro-endocrine mechanisms act on the adipose tissue fat balance.

We have evidence that adverse psychosocial conditions are associated with increased risk of obesity later in life, and could speculate that similar mechanisms may be driving the epidemic.

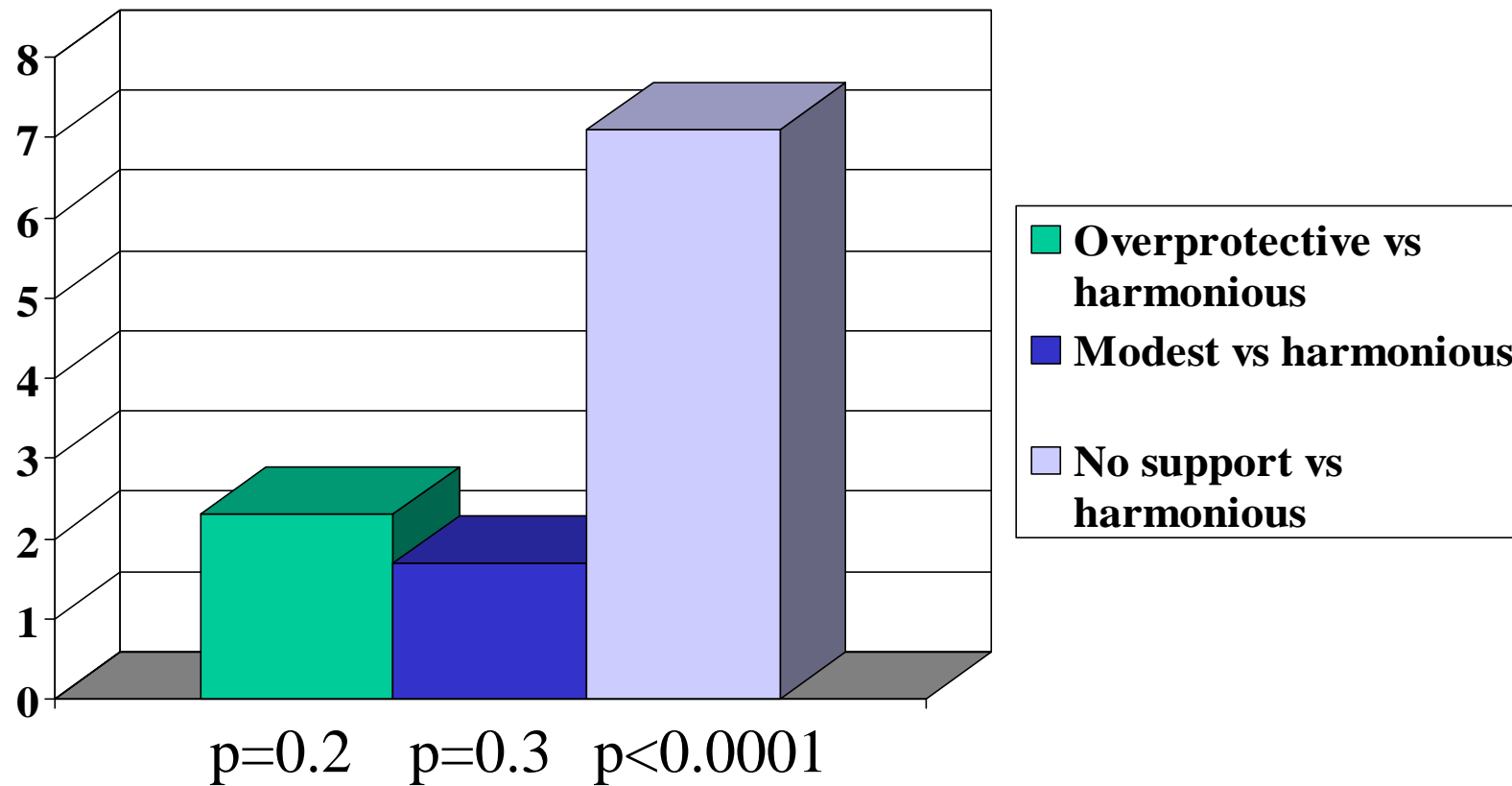
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## Parental neglect during childhood and increased risk of obesity in young adulthood

Inge Lissau, Thorkild I A Sørensen

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Odds ratio for obesity by parental support perceived by the teacher



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# Parental neglect during childhood and increased risk of obesity in young adulthood

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Odds ratio for obesity by appearance at school health exa

