

Declining Worldwide Sperm Counts: Disproving a Myth

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Much has been made in the medical and lay literature of an alleged decline in human sperm counts worldwide. More than 100 articles have appeared in the peer-reviewed literature in the past 50 years on this topic. As discussed in more detail later, these articles vary widely in the quality of their methodology and their fundamental results. Most studies have found no decline, an increase, or mixed results in assessing changes in sperm parameters. A few studies have shown an unambiguous decline. It is from the latter studies, however, that the lay media and various advocacy groups have drawn the “findings” supporting theories that purport to explain the “decline” in sperm counts. The primary theory supported by these groups is that minute environmental levels of chemicals acting as “hormone mimics” or “endocrine disruptors” are responsible for this alleged deterioration. The term “endocrine disruptor” refers to chemical substances that exhibit some degree of estrogen-like activity. Although there is no question that estrogenic compounds can be potent modulators of biochemical and physiologic function in high doses, the implication that in utero or adult exposure to low levels of environmental “endocrine disruptors” produces clinically detectable effects in humans is highly uncertain.

Data from the handful of peer-reviewed articles showing a decline in sperm counts and other semen parameters have been quoted often enough in the media and even among researchers that this “fact” has achieved the quality of a paradigm. A dispassionate review of the research to date,

however, firmly repudiates not only the alleged connections between “endocrine disruptors” and declines in semen quality but also the declines themselves. Far from being a worldwide and well-proved phenomenon, declines in semen quality are, at best, a highly local phenomenon with an unknown cause and, at worst, a collective artifact arising from the observation of a highly variable physical attribute (sperm counts) with a relatively low-resolution tool (retrospective analysis of non-randomized study populations).

This article explores in detail the issue of the alleged decline in semen quality. The impetus for a comprehensive re-evaluation at this time is threefold: (1) the potential impact of a real decline in semen quality and subsequent human fertility is a priori critical to human welfare; (2) governments have begun to enact “anti-endocrine disruptor” legislation that is based, in part, on selected portions of the published data about semen quality; and (3) confusion and misinformation about semen quality remain widespread in lay and professional circles.

Sources of error

At first blush, it might seem almost trivial to obtain for semen quality the same type of widely accepted physiologic norms that have been determined for other bodily fluids and functions, such as blood or blood pressure. A host of difficulties conspire to make semen the least well understood bodily fluid, however, in terms of the distribution of its normal parameters in the general population. Obtaining human semen for scientific analysis is logistically difficult. As many authors have pointed out, the fact that semen is almost universally obtained by masturbation has

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placed profound limits on the ability of researchers to adequately study this issue. If collection of semen samples were as straightforward as obtaining blood samples, the nature of semen quality changes over time (if any) would have been determined decisively decades ago. A prospective, longitudinal study of semen parameters in a large, multicenter, randomized study of community-dwelling men, although time-consuming and expensive, would provide highly reliable data. Of nearly equal quality would be an analysis of a suitably sized population of randomly selected community-dwelling men analyzed by birth cohort. Unfortunately, neither of these high-quality observational tools has been used to investigate the phenomenon of semen quality because of the logistical and emotional obstacles posed by the means of obtaining semen in a timely and well-controlled manner.

Researchers in the past 50 years have studied populations of men who have provided semen samples for reasons such as donation to sperm banks, evaluation for male factor infertility, prevasectomy evaluation, infertility evaluation for a couple, and donation for use with assisted reproductive techniques, such as *in vitro* fertilization or intracytoplasmic sperm injection. None of these populations represents a random sample of the population at large, and each presents a selection bias, although some of these study populations are more likely to be biased than others. For example, men who provide semen samples as part of a couple's infertility evaluation in which the female partner is later determined to be the source of the infertility could plausibly be considered nearly representative of the general male population because their inclusion for testing is unrelated to the semen donor's potential fertility. Other types of male study populations are more likely to be biased, however. Semen donors, for example, may have been screened for problems known to affect fertility or may have been selected precisely because a prior semen analysis showed a robust fertility. Male donors to *in vitro* fertilization or intracytoplasmic sperm injection programs are more likely than normal to have low fertility, regardless of the fertility status of their partner.

The lack of truly randomized, community-dwelling study populations has posed fundamental limits on our ability to say what is "normal" in terms of semen parameters and renders illegitimate any attempts to generalize from a particular study of semen or semen change over time to the male population at large.

Another source of potential error in studies of semen quality is the highly variable nature of the subject in question. Attributes such as sperm count, semen volume, and sperm morphology not only vary widely between individuals but also vary widely *within* individuals. Semen quality is sensitive to the following variables:

- Abstinence time (the amount of time since the previous ejaculation). Longer abstinence times lead to higher sperm counts, higher semen volumes, and a higher percentage of sperm displaying abnormal morphology. In turn, abstinence time varies with such things as a man's age, his current level of sexual activity, and his general health.
- Scrotal temperature. The Sertoli cells of the testicles are temperature sensitive and must be several degrees cooler than normal body temperature to function properly. Anything that either temporarily or chronically raises scrotal temperature can depress semen quality [1]. Such phenomena as fever, hot tubs, exposure to high-temperature working conditions, and occupations that require long periods of sitting have been shown to affect sperm quality.
- Season. Some, but not all, studies of semen quality have shown seasonal fluctuations in mean sperm counts, with averages highest in springtime and lowest in summer [2].
- Smoking. Chronic smokers show a 13% to 17% decline in sperm counts, according to a meta-analysis of 20 studies [3]. Variations in the incidence of smoking between regions or over time may alter mean sperm counts.
- Marijuana use. Various animal, *in vitro*, and human studies have demonstrated deleterious effects on sperm parameters—including sperm counts—of tetrahydrocannabinol and chronic use of marijuana [4]. Changing regional or temporal trends in the use of marijuana may be a confounding factor in studies of semen quality.

An additional factor that has yet to be explained adequately contributes to the difficulties of scientifically determining population norms for semen and assessing any changes to those norms over time. Semen parameters have been repeatedly shown to vary significantly with geographic region. Even careful studies using identical laboratory methods on similar populations of men recruited for similar reasons have found this effect. For example, a study of 1283 men

from three regions of the United States found a mean sperm concentration in California of 72.7×10^6 sperm/mL, whereas the mean concentration in Minnesota was 100.8×10^6 sperm/mL, and in New York it was 131.5×10^6 sperm/mL [5]. As demonstrated in the following sections, failure to take such variation into account can completely invalidate studies of semen characteristics.

Studies of semen quality have been hampered by three fundamental sources of potential error: inability to study a truly random population of community-dwelling men, wide inherent inter- and intrasubject variation in semen parameters, and wide and unpredictable geographic variations in semen quality. The authors of the best studies of semen quality in the past 50 years are cognizant of some or all of these potential sources of error. Some studies, for example, have attempted to control for variables such as abstinence time or have chosen subjects only from the subpopulations of men that are least likely to be biased in comparison to the population at large. Many studies have not taken these potential sources of error into account, however. The failure to address such errors in some studies has been compounded by additional methodologic or statistical errors.

A flawed pivotal study

Before 1992, several small-scale or regional studies of men seeking medical help for infertility suggested a decline in sperm counts or other semen parameters in primarily European countries [6–15]. In the same period, however, a large US study found no decline in semen parameters [16]. The divergence in the results of these studies remained a topic of professional discussion and debate during these years but did not reach a wider audience. This thinking changed with the publication in 1992 of a paper by Elisabeth Carlsen and two colleagues from the University of Copenhagen, Denmark [17]. Entitled “Evidence for decreasing quality of semen during past 50 years,” this meta-analysis of 61 previous studies gained worldwide media attention. The attention was caused by the surprising magnitude of the findings (a nearly 50% drop in sperm count from 113×10^6 sperm/mL in 1940 to only 66×10^6 sperm/mL in 1990) and the fact that the authors suggested a cause for the decline: “compounds with estrogen-like activity or other environmental or endogenous factors.” This study’s findings dovetailed with pre-existing concerns in many quarters about

the potential hazards of environmental pollutants, such as herbicides, pesticides, and chemical contamination of all sorts. Although the relevant medical community reacted quickly to the paper with skepticism about its results and criticism of its methodologies, the popular interpretations of the study were unreserved, and subsequent critiques in the medical literature received little, if any, popular notice. As a result, the Carlsen paper has had an impact on the popular imagination and mindset of many environmental advocates that is far out of proportion to its actual scientific value. I argue that the paper has no scientific value. Its primary importance is that it acted as a stimulus for more careful researchers to explore the complex issue of semen quality.

Although the Carlsen paper already has been thoroughly “discredited in a number of professional articles” [18–22], its stature as a pivotal study warrants a summary of its major weaknesses:

- Variability across the 61 studies in the methods and protocols used for sperm collection and measurement
- Inability to control for period of abstinence in study subjects
- Inability to control for lifestyle factors, such as cigarette smoking or recreational drug use
- Failure to include studies that were available and conducted within the time period of the meta-analysis that fail to show a decline or report sperm concentrations higher than other studies included in the meta-analysis from the same time period
- Failure to account for geographic variation among studies (Of the studies from before 1970, all were from the United States, and 80% of these were from New York, where sperm counts (then and now) are the highest. After 1970, only three studies were from the United States, and many were from third-world countries, where sperm counts were low. A reanalysis of the Carlsen meta-analysis that accounts for this geographic variation shows no decline in sperm counts [Fig. 1].)
- Use of an inappropriate statistical analysis. A comprehensive statistical reanalysis of the Carlsen study [23] showed that the linear regression model used was inappropriate because the data distribution was highly non-uniform—most data were collected between 1970 and 1990. When quadratic or spline regression models were used (even when the data were uncorrected for geographic

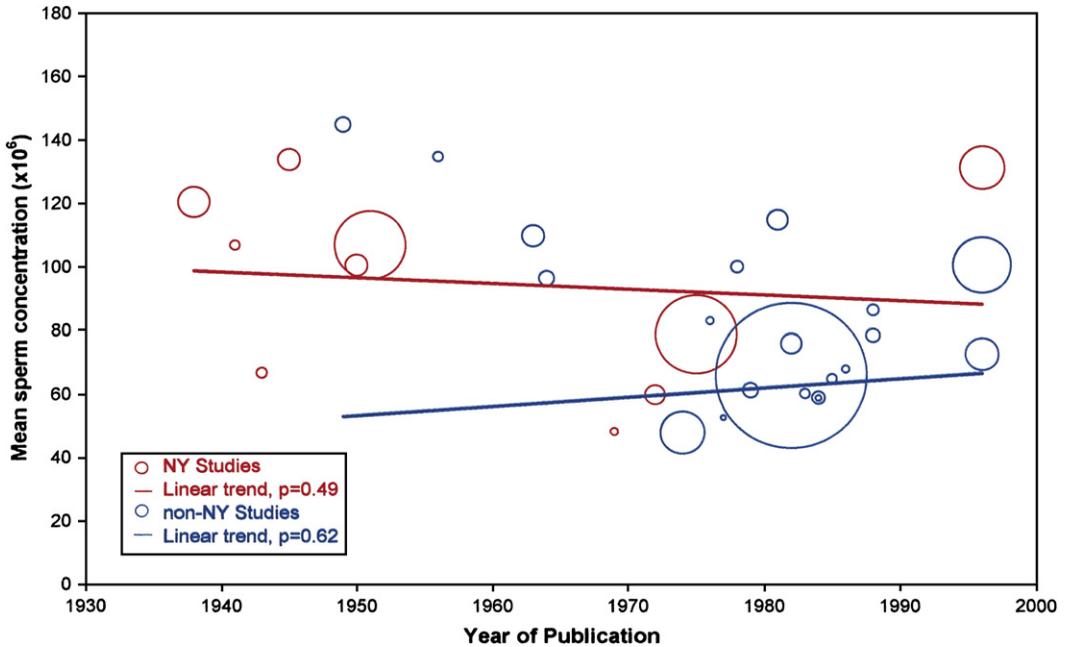


Fig. 1. Re-analysis of data from Carlsen et al shows no decline in sperm counts (*blue regression line*) when data from New York are excluded. Bubble size corresponds to number of men in study. (From Saidi J, Chang D, Goluboff E, et al. Declining sperm counts in the United States? A critical review. *J Urol* 1999;161:460–2; with permission.)

variation) the data show mean sperm counts increasing since 1940.

Any of these weaknesses alone would serve to cast the results of a scientific study in doubt. Taken together, they justify a deep skepticism regarding the Carlsen study and a removal of the study from consideration in any review of evidence supporting a decline in sperm counts or other semen parameters.

The balance of evidence

Although without use in a scientific sense, the Carlsen paper did stimulate more than a decade's worth of scientific research, most of it more methodologically sound than the Carlsen paper and written by authors who made far less sweeping extrapolations to the general population and possible causative agents. The 31 major studies published after the Carlsen paper that reported on time trends of semen parameters have been reviewed. Six of these studies showed clear evidence of a decline in either sperm counts or sperm counts and other semen parameters in a given location and period of time (Table 1). Sixteen studies unambiguously showed no decline (either

no change or an increase) in semen parameters (Table 2), and 5 studies showed ambiguous results (Table 3). This latter group includes studies such as the 1995 report by Comhaire and colleagues [24], in which motility and normal morphology showed significant decreases but total sperm count was unchanged across the 17-year study period. The other studies in this group reported similarly conflicting results.

The summaries that follow do not include studies that simply reanalyzed existing data from Carlsen or others (although all of them contradict the “declining sperm” hypothesis), nor do they include studies that examined or critiqued in general ways some of the methodologic issues involved in the debate over alleged changes in semen parameters.

As can be seen from the tables, six studies with a combined *N* of 9215 reported an unambiguous decline in one or more semen parameters (including sperm count). The magnitudes of the reported declines in sperm counts (either in terms of total count or as sperm concentration/density) varied from a 16% decline to a 31.5% decline in the study period. In general, the authors of these studies attempted as best they could to control or account for some of the variables discussed previously.

Table 1
Studies showing unambiguous decline in sperm count

Date	Author(s)	Sample size (<i>N</i>)	Study period	Location	Major findings
1995	Auger et al [28]	1351	1973–1992	France	<ol style="list-style-type: none"> 1. Mean volume: no change 2. Mean sperm concentration declined 2.1% per year from 89 million/mL to 60 million/mL 3. Percent motile sperm decreased 0.6% per year 4. Percent normal sperm decreased by 0.5% per year
1996	Irvine et al [26]	577	1984–1995	Scotland	<ol style="list-style-type: none"> 1. Sperm concentration fell from 98 million/mL before 1959 to 78 million/mL among donors born after 1970 2. Total motile sperm fell from 169 million to 129 million 3. Concentration declined 2.1%/y 4. Motility increased 0.18%/y
1996	Adamopoulos	2385	1977–1993	Greece	<ol style="list-style-type: none"> 1. Total sperm count declined from 154.3 million to 130.1 million 2. No significant drop in semen volume
1998	Bonde [31]	1196	1986–1995	Denmark	<ol style="list-style-type: none"> 1. Median sperm concentration dropped from 63 million/mL in men born 1937–1949 to 52 million/mL in men born 1970 and later 2. Median total sperm count dropped from 206 million and 117 million, respectively
1999	Bilotta [35]	1068	1981–1995	Italy	<ol style="list-style-type: none"> 1. 31% decline in sperm concentration over the study period 2. 8% decline in motility 3. 9% decline in sperm with “typical morphology”
2003	Almagor [36]	2638	1990–2000	Israel	<ol style="list-style-type: none"> 1. Sperm count declined by 5.2 million each year 2. Motility declined by 0.5% per year
Total		9215			

In comparison, 16 studies with a combined *N* nearly ten times that of the “pro-decline” studies (103,313) found no decline or a slight increase in the sperm counts of their respective populations. These authors took pains to control for variables such as abstinence time and variation in laboratory techniques and analyzed their data using appropriate statistical techniques. As noted earlier, however, all of these studies, like the “pro-decline” studies, relied on populations of men who do not necessarily represent the general male population and thus share the weakness of all studies to date. With that qualification understood, many of these studies are careful and rigorous and more than sufficiently powered to provide reliable data. If there were an actual decline in sperm counts in

these study populations, one can assume that these analyses would have identified it.

An objective observer can draw two firm conclusions from this summary:

1. There is no “worldwide” decline in sperm counts or other semen parameters.
2. No correlations can be drawn from these studies about a causative role for “endocrine disruptors.”

The question of geographic variation in sperm counts

Both of these conclusions deserve elaboration. Although the evidence firmly refutes the reality of

Table 2
Studies finding no decline or an increase in sperm count

Date	Author(s)	Sample size (N)	Study period	Location	Major findings
1996	Bujan [30]	302	1977–1992	France	1. Sperm counts remained constant after adjustment for age of donors
1996	Paulsen	510	1972–1993	United States	1. No decrease in sperm count, volume, sperm concentration, or normal morphology
1996	Vierula	238	NA	Finland	1. Mean sperm concentration was high (133.9 million/mL) and unchanged across the study period 2. Total sperm count and sperm density were unchanged 3. No trends up or down when data were analyzed by birth cohort
1996	Fisch [5,22]	1283	1970–1994	United States	1. Statistically significant increase in sperm concentration over study period from mean of 77 million/mL to 89 million/mL (0.65% increase/year) 2. Motility also constant, although mean volume decreased slightly
1997	Berling	718	1985–1995	Sweden	1. Sperm concentration rose from 46 million/mL in 1985 to 64 million/mL in 1995 2. Sperm with normal morphology rose from 58% to 66.4% 3. Volume declined
1997	Benshushan	188	1980–1995	Israel	1. Volume increased 5.1%/y 2. Total motile sperm count rose 7.7%/y 3. Motility increased 0.27%/y
1997	Handelsman	689	1980–1995	Australia	1. Overall mean for period was 69 million/mL 2. No significant change in semen volume, total sperm count, or sperm concentration over study period
1997	Rasmussen [32]	1055	1950–1970	Denmark	1. Variation found in semen parameters year to year but no decline observed over the period studied 2. Comparison of four birth cohorts revealed no association with changes in sperm quality
1998	Emanuel [21]	374	1971–1994	United States	1. No significant differences between mean or median sperm counts between subjects in modern group compared with 1000 subjects in MacLeod and Gold's 1951 study
1998	Younglai	48,968	1984–1996	Canada	1. Linear regression analysis of the means of each of 11 centers studied over study period showed no significant trend
1999	Andolz	20,411	1960–1996	Spain	1. 0.2% decline in volume/year 2. 0.04% increase in sperm count/year 3. 0.4% increase in motility 4. 3.6% decline in normal sperm/year

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Table 2 (continued)

Date	Author(s)	Sample size (N)	Study period	Location	Major findings
1999	Gyllenberg [2]	1927	1977–1995	Denmark	<ol style="list-style-type: none"> 1. Increase in mean sperm concentration from 53 million/mL to 72.7 million/mL 2. Increase in total sperm count from 166 million to 227 million 3. Sperm motility declined from 74.2% “excellent” motility in 1977–1980 to 41.9% “excellent” in 1993–1995.
1999	Zorn	2343	1983–1996	Slovenia	<ol style="list-style-type: none"> 1. Volume, concentration, and total sperm count did not change in study period 2. Sperm concentration analyzed by birth cohort showed a decline from 1950–1960, then an increase after 1960
2000	Acacio	1347	1951–1997	United States	<ol style="list-style-type: none"> 1. No decline in sperm concentration found when compared with MacLeod data from 1951 and 1979
2000	Tae Seo	22,249	1989–1998	Korea	<ol style="list-style-type: none"> 1. Mean sperm concentration was 60.5 million/mL 2. No change in concentration, volume, or motility in study period
2001	Itoh	711	1975–1998	Japan	<ol style="list-style-type: none"> 1. Volume was unchanged 2. Sperm concentration was 70.9 million/mL in early study compared with 79.6 million/mL in later Study
Total		103,313			

a worldwide decline in sperm counts, these studies clearly demonstrate that semen parameters vary dramatically geographically and temporally. In addition to the previously mentioned data from the United States, a study completed in Europe in 2001 found significant differences in mean sperm count and other semen parameters among fertile men in Denmark, France, Scotland, and Finland [25]. For example, sperm concentration in Copenhagen, Denmark was only 74% that of Turku, Finland.

It is important to recognize that even large interregional differences in sperm count, sperm concentration, or semen volume may not have high clinical relevance because these parameters are only weakly associated with male fertility [26]. On the other hand, sperm motility and morphology are more strongly associated with fertility, and many of the studies reviewed herein demonstrate that these parameters also vary widely.

Currently, no data exist to explain the observed geographic variations in semen parameters. The range of possible causative agents is large and includes the following candidates:

- As-yet undetected differences in laboratory techniques, methods of analysis and

interpretation, subject recruitment, or subject health and lifestyle differences between regions

- Differences in sexual behavior that would alter mean abstinence times between regions
- Differences in emotional/psychologic stress between selected populations of men
- Genetic differences among populations
- Variation in lifestyle factors, such as smoking or recreational drug use
- In utero exposure of male subjects to compounds with mutagenic or teratogenic potential
- Exposure of adult men to differences in environmental pollutants, such as lead or industrial chemicals found in herbicides, pesticides, or other materials

The data assembled to date fail to support any particular causative agent. No useful correlations have been found that would point to differences in exposure to industrial pollutants [27]. The lack of association between rural and urban areas or between areas with known high levels of air pollution and those with less pollution suggests that these factors are unlikely to explain the differences observed. The regional differences described to

Table 3
Studies presenting ambiguous results

Date	Author(s)	Sample size (<i>N</i>)	Study period	Location	Major findings
1995	Comhaire [24]	360	NA	Belgium	<ol style="list-style-type: none"> 1. Motility and morphology exhibited “highly significant decreases” 2. Total sperm count did <i>not</i> decrease 3. 40% of donors after 1990 exhibited “subnormal” sperm compared with only 5% of group investigated before 1980
1996	Van Waeleghem	416	NA	Belgium	<ol style="list-style-type: none"> 1. Volume increased slightly 2. Mean concentration declined by 12.4 million/mL in study period 3. Sperm count was unchanged 4. Normal morphology decreased from 39.2% in 1977–1980 to 26.6% in 1990–1995
1996	DeMouzon	7714	1989–1995	France	<ol style="list-style-type: none"> 1. No decline in sperm counts when data were analyzed by year of collection 2. Sperm counts declined “regularly” for men born from 1950 to 1975
1997	Ye Zheng	8608	1968–1992	Denmark	<ol style="list-style-type: none"> 1. Semen quantity and quality did not decline with increasing year of birth during the entire period from 1922 to 1972 2. From 1950 onward there was a gradual decline in sperm count and normal sperm forms but not in semen volume 3. Decline in total sperm count was 1.9 million/mL per year of advancing year of birth
1999	Ulstein	5180	1975–1994	Norway	<ol style="list-style-type: none"> 1. Two subgroups of study subjects showed declines in semen parameters 2. Subgroup of men with previous children did not show decline in semen parameters

date warrant further research, some of which is ongoing [28].

It is similarly impossible to draw any meaningful conclusions from the conflicting studies exploring temporal changes in semen parameters. Although the preponderance of data strongly suggests that there has been no decline in sperm counts or concentration in the past few decades, the handful of well-conducted studies reviewed herein that came to opposite conclusions cannot be dismissed. These data may be real for the specific region and time period examined. If so, then the observed declines are highly localized. For example, a study of men in the vicinity of Paris showed a decline [29], whereas a study of men in the Toulouse region of southern France showed no decline [30]. Likewise, one study of Danish men showed a decline [31], whereas another study that drew men from the same small

country showed no decline over the 20-year study period [32].

Explanations for a decline in the regions studied include all of the potential causative factors mentioned and another that has not received much attention to date. It is well known that weight gain in men, particularly the deposition of adipose tissue around the waist, can depress serum total testosterone levels and increase serum estradiol levels [33]. Given the epidemic of obesity in the developed world in the past few decades, a gain in mean male weight coupled with type 2 diabetes and metabolic syndrome, which are related to weight gain, could explain some of the observed declines of sperm in specific populations of men studied. Weight gain has not been included in the data analysis of any of the studies of changing semen parameters conducted to date. It is worth noting that

contrary to the data on an alleged decline in semen parameters, good data from randomized, adequately sized populations of community-dwelling men show a clear and consistent decline in mean testosterone levels [34]. Such declines also may contribute to a decline in semen parameters in the few locations in which this phenomenon has been observed, although this has not yet been explored scientifically. It is certainly another area ripe for further investigation.

Summary

This article critically examines allegations for a worldwide decline in sperm counts and other semen parameters, as most visibly presented by Carlsen and colleagues in their 1992 paper. Despite the lack of scientific support for this hypothesis and for related claims that a “decline” is related to “endocrine disruptors,” these constructs remain firmly entrenched in the popular literature and are being used, in part, as a justification for legislation banning certain suspected “disruptors,” such as phthalates. A review of the data amassed to date on this issue clearly demonstrates that the bulk of the evidence refutes claims for a widespread decline in semen parameters. The initial study by Carlsen and colleagues has been criticized widely and thoroughly, and the number of methodologic flaws contained in the study warrants its exclusion from any review of data supporting a decline. The total population of subjects in which no decline in semen parameters was found is ten times larger than the population of men in whom a decline was found.

Even granting the reality of the declines reported for a handful of localized regions, no conclusions can be drawn from any of the existing studies about the role of putative causative agents. The range of such agents is wide, and no associations have yet been found between any of the reported declines and exposure of the men involved (either in utero or as adults) to “endocrine disrupting” compounds. The cause (or causes) of the well-documented geographic variations in semen parameters deserves further investigation; however, the evidence accumulated to date showing no decline in sperm counts or sperm concentration in populations throughout the developed world should be accepted. Advocates of “endocrine disruptor” theories are unjustified in using an alleged “decline” in semen parameters to support their cause, and public officials should be advised that this “leg” of supposed evidence for

proposed legislation is weak to the point of breaking.

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