



Sex differences across different racial ability levels: Theories of origin and societal consequences



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ABSTRACT

Jensen (1971) found that black girls score 3 IQ points higher than black boys, and white boys 1.5 IQ points higher than white girls. He, nevertheless, concluded that this did not support his Race \times Sex \times Ability interaction theory. Jensen (1998) further analyzed data, some from the National Longitudinal Survey of Youth (NLSY79), and suggested that there is no sex difference in general intelligence, *g*. Other studies have questioned Jensen's null sex difference theory.

The present study tested both theories with data from the ensuing NLSY97 survey, which represents the 15+ million 12–17 year old adolescents living in the US in 1997.

Total sample analyses confirmed the existence of significant inverse white–black IQ sex differences, and disconfirmed the null sex difference theory.

Separate race–age analyses demonstrated, however, that robust IQ sex differences materialize only after age 16, with no white–black interaction. At age 17, female IQ trails male by 3.6–7.03 points in three races, respectively. Classical IQ probability curves foretell that more males than females will enter the highest echelons of society, irrespective of race, and white Male/Female ratios at IQ 145 successfully predicted real-life sex differences in educational and occupational achievement. White males with IQ 55 can be expected to run a very high risk of encountering severe achievement problems, a risk shared to some extent with Hispanic male, but black females with this low IQ can be expected to perform worse than black males.

The paper finally proposed models to account for the origin of sex and race differences in IQ and related educational and occupational differences, involving gene copy numbers, brain size, and steroid hormones. It was suggested that the evolutionary background and physiological nature of sex and race differences explain why social engineering fails to eradicate them.

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1. Introduction

1.1. Problems

Jensen (1998) looked for sex differences in general intelligence, *g*, in the NLSY79 wave of the National Longitudinal Survey of Youth, which used the Armed Services Vocational Aptitude Battery (ASVAB). He concluded that "... the sex difference in *g* is either totally nonexistent or is of uncertain direction and of inconsequential magnitude." (Jensen, 1998, p. 540–41). However, other studies have questioned this conclusion (e.g. Flores-Mendoza et al., 2013; Irwing, 2012; Jackson & Rushton, 2006; Lynn, 1999; Nyborg, 2005; Rushton & Jensen, 2005).

This raises the question of whether there will also be null sex differences after we have examined IQ data from the ensuing NLSY97 CAT-ASVAB computer aided IQ survey, which is the nationally representative study that NLSY79 is not. The NLSY97 analysis could further inform

whether sex differences in *g* and dispersion scores generalize across races.

All this is of interest because Jensen (1971) observed back in the 1960s that the level of black female scholastic achievement was generally higher than that of black males. He therefore decided to test a Race \times Sex \times Ability Interaction Theory (IT) that there is a black female advantage in IQ and no sex difference among whites. Testing the IT interested Jensen, because a black sex difference in IQ could assist in the identification of specific environmental factors in the white–black IQ difference itself. He therefore examined the IT in "... seven highly *g*-loaded tests and a total of more than 20,000 subjects, all of school age and most below age thirteen". After combining the data, Jensen found that black females lead black males by 3 IQ points and white males had a 1.5 IQ point advantage over white females. Even if statistical rejection of the null sex IQ difference theory was not possible, Jensen nevertheless concluded that the IQ differences were too small to explain the sex differences in favor of black female achievement. Contributing to this conclusion was further that the effect was inconsistent for different tests and in different samples, that data was not gathered systematically so as to be representative of the whole black and white populations in

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the United States, and that girls tend to mature earlier than boys. Moreover, several subsequent large-scale studies (Broman, Nichols, & Kennedy, 1975; Jensen & Johnson, 1994; Roberts, 1971; Strauch, 1977), carried little support or even findings running counter to the IT.

A re-examination of Jensen's Race \times Sex \times Ability Interaction Theory (IT) with data from the NLSY97 wave entails several advantages. Contra the NLSY79 data, the NLSY97 data are representative for all adolescents living in the US in 1997. Data are gathered systematically for three major races (and includes a small "Other" race category). All subjects were examined by the same test. Not least, sample size allows data to be meaningfully broken down separately by sex and race for each year between 12 and 17.

A more general problem is that the origin and mechanisms of sex and race differences are still largely unknown despite massive research efforts. One factor here is the extent to which environmental factors contribute to these differences. Another problem is their predictive validity for education and occupation.

1.2. Specific aims and hypotheses

The present study approaches these problems with data from the NLSY97 study in the form of a series of hypotheses:

1. Since most studies find a sex difference, it is hypothesized that males score higher IQ than females.
2. Following Jensen (1971) we hypothesize, that there will be no white-black Race \times Sex \times Ability interaction.
3. Since most studies find race differences in IQ, it is hypothesized that races will also in this study rank themselves whites > Hispanics > blacks.
4. It is hypothesized that a moderate average male advantage in IQ combines, in accordance of classical distribution theory, with larger male distribution scores and predict a male overrepresentation at the very low and very high end of the intelligence distribution.
5. As Ree and Earles (1990) documented that the predictive power of IQ increases with increasingly higher task training demands, it is hypothesized that larger Male/Female probabilities and ratios provide increasingly more precise predictions of actual educational and occupational sex differences in achievement.
6. Visscher, Hill, and Wray (2008) observed that estimates of heritability for IQ from twin studies were "remarkably consistent in the range of 0.5–0.8, across many age groups". It is accordingly hypothesized that sex differences in education and occupations, based on IQ, both reflect their ultimate evolutionary origin, and are grounded in identifiable proximate physiological mechanisms.

1.3. Suggested solutions

The paper endeavors to combine evolutionary with ontogenetic theories in the attempt to associate a causal understanding of current observations of sex and race difference in IQ with their evolutionary origin. It further strives to identify part of the physiological mechanisms behind race and sex differences in IQ (g, Spearman, 1927) and relate this to achievement differentials in order to explain why IQ-related differences are so resilient to change, even after extended goal-directed social engineering.

1.4. The approach

The paper is divided into five parts. Section 2, after the introduction, documents the empirical approach to testing hypotheses 1–4, Section 3 tests hypothesis 5, and Section 4 discusses the empirical findings. Section 5 tests hypothesis 6 by venturing into evolutionary theory in the attempt to fathom the origin and physiological nature of sex and race difference in IQ and related achievement, in an eternal quest for a

unified account of *Molecular Man in a Molecular World* (Nyborg, 1994, 1997).

2. Empirical examination of race and sex differences in NLSY97

2.1. Subjects and analysis

A total of 7127 NLSY97 respondents completed the CAT-ASVAB97 subtests. Of those 5452 came from the cross-sectional sample, 1675 from a supplemental sample, as also young adults from two separate samples. The total sample was reduced for the present analysis to: 1) Only those with complete data, 2) Only whites, Hispanics, and blacks, because there were too few subjects in the "mixed" race group for meaningful breakdowns, and 3) Only 12–17 year olds, because there were too few 18 year olds. This reduced the total number of subjects to 6.912 respondents.

Tables 1a–1c provide age, actual and representative Ns, average PC1 g, and its IQ equivalent, all broken down by sex, race, and age.

The NLSY97 study offers individual case weights to provide an estimate of how many individuals in the United States are represented by each respondent when used in tabulation. According to the manual, the assignment of individual respondent weights involves three types of adjustment: 1) The reciprocal of the probability of selection (associated with housing unit as well as sub-sampling applied to individuals), 2) Differential cooperation rates (based on geographic location, group membership, and within-group sub-classification), and 3) Correction for certain types of random variation associated with sampling as well as sample "under-coverage" (used to conform the sample to Census Bureau estimates of population totals).

The use of weights in the present analyses is associated with the following restrictions and precautions. The weighted mean may not represent the entire population as the analyses are confined to sub-samples only with valid answers on all CAT-ASVAB97 items. Deletion of the difficult-to-interpret "other" races category of subject as well as of the diminutive 18 year old group involved just 180 subjects. Item non-response due to refusals, don't knows, or invalid skips was quite small, so the degree to which the weights are incorrect is probably quite small, so the population estimates presented here will most likely be accurate.

The NLSY97 calculates two types of sampling weight. The first "total sample" weight involves all NLSY97 respondents; these weights (when divided by 100) add up to an estimate of the number of U.S. residents in the sample age range 12–17 years in 1997. The second set named "cross-sectional sample weight" contains weights only for respondents in the cross-sectional sample and all over-sampled cases have a zero weight. These weights are also designed to produce an estimate of the number of U.S. residents in the sample age range, but since there are fewer respondents if the over-sample is omitted, each black or Hispanic (or Latino) respondent in the cross-sectional sample has a larger value. The recommended use of the two sets of weights are as follows. Total sampling weights can be used in race-related tabulations, in order to increase the precision for black and Hispanic (or Latino) youths. Cross-sectional weights can be used in tabulations focusing only on non-black, non-Hispanic youths, or that do not include any analysis by race/ethnicity. Details of sampling are provided by Moore, Pedlow, Krishnamurty, and Wolter (2000).

As the concept of statistical significance becomes notoriously difficult to interpret in analyses involving huge numbers of representative subjects, I preferred to remain on the conservative side in the analyses. The ensuing factorial multivariate tests for statistical significance are therefore (with the few exceptions noted later) based on the much lower actual number of Ns when testing hypotheses. However, representative Ns and IQs are tabulated in Tables 1a to 1c to allow the reader to directly estimate the (minor) IQ differences to results based on actual Ns. Graphically, the differences between IQ age-curves based on

Table 1a
Male general intelligence (first principal component *g* and its IQ equivalents) broken down by race and age (NLSY97 data).

Males											
Race	Age	Actual N	PC1 <i>g</i> ^a	SD	IQ equivalent ^a	SD	Repr. N	PC1 <i>g</i> ^b	SD	IQ equivalent ^b	SD
White	12	279	-0.13	0.88	97.99	13.22	830.254	-0.13	0.88	97.98	13.21
	13	400	0.18	0.88	102.73	13.22	1.102.215	0.18	0.88	102.69	13.19
	14	420	0.34	0.92	105.06	13.78	1.177.627	0.34	0.92	105.05	13.82
	15	393	0.70	0.89	110.51	13.37	1.109.395	0.70	0.89	110.46	13.34
	16	348	0.88	0.90	113.16	13.56	1.054.433	0.88	0.90	113.17	13.57
	17	106	0.93	1.00	114.01	15.04	333.518	0.93	1.00	114.01	14.95
Subtotal		1.946	0.44	0.97	106.60	14.52	5.607.442	0.44	0.97	106.67	14.56
Hispanic	12	92	-0.85	0.81	87.29	12.19	122.176	-0.80	0.90	88.01	13.43
	13	149	-0.53	0.85	92.07	12.81	181.249	-0.39	0.86	94.12	12.89
	14	118	-0.44	0.87	93.36	13.11	171.320	-0.28	0.89	95.83	13.38
	15	144	-0.04	0.91	99.45	13.63	205.883	0.01	0.90	100.14	13.46
	16	134	0.06	0.85	100.93	12.71	186.977	0.08	0.83	101.21	12.50
	17	43	0.22	0.96	103.34	14.39	53.163	0.37	0.97	105.51	14.49
Subtotal		680	-0.29	0.93	95.67	13.94	920.768	-0.20	0.94	97.07	14.03
Black	12	112	-1.11	0.73	83.33	10.90	155.150	-1.10	0.69	83.43	10.36
	13	166	-0.87	0.77	86.91	11.58	227.364	-0.81	0.82	87.79	12.35
	14	197	-0.67	0.91	89.91	13.60	243.350	-0.60	0.91	91.01	13.61
	15	193	-0.47	0.91	92.98	13.72	244.491	-0.37	0.91	94.46	13.62
	16	151	-0.50	0.96	92.47	14.46	211.949	-0.41	0.97	93.82	14.55
	17	44	-0.07	0.91	98.92	13.66	65.835	0.01	0.93	100.09	13.98
Subtotal		863	-0.67	0.90	89.90	13.44	1.148.139	-0.59	0.92	91.12	13.87
Total		3.489	0.03	1.06	100.38	15.92	7.676.349	0.21	1.04	103.19	15.57

^a Based on unweighted data.

^b Based on total sample weighted data / 100.

unweighted data and total sample-weighted data are barely visible, and numerical tabulations differ little.

2.2. Analysis

The CAT-ASVAB97 is basically a vocational ability test, involving subtests like Shop and Auto Information, as seen in Table 2.

Specific vocationally relevant subtests can be sex-biased, and this could affect the reliability of testing theories about general race–sex differences in general intelligence. In order to reduce this risk, I scrutinized several factor-analytic solutions. This indicated that the first unrotated Orthogonal Principal Component PC1 *g*, derived from only those 7 CAT-ASVAB subtests with *g*-loadings >0.80, accounted for

most of the total variability (76%). This factorial approach excluded 4 subtests, including Shop and Auto information, and was chosen as the least sex-biased estimate of general intelligence.

In order to ease interpretation and comparisons to other studies, PC1 *g* is in the following converted to its IQ equivalent scores in accordance with the formula: $15 * stPC1g + 100$.

2.3. Results

2.3.1. Total sample testing

Given that PC1 best reflects general intelligence, *g*, in the CAT-ASVAB test, we may assume that age differences in PC1 *g* largely mirrors subscale score age differences in the 7 most highly *g*-loaded standardized

Table 1b
Female general intelligence (first principal component *g* and its IQ equivalents) broken down by race and age (NLSY97 data).

Females											
Race	Age	Actual N	PC1 <i>g</i> ^a	SD	IQ equivalent ^a	SD	Repr. N	PC1 <i>g</i> ^b	SD	IQ equivalent ^b	SD
White	12	238	-0.20	0.75	97.02	11.18	736.932	-0.20	0.74	97.00	11.13
	13	353	0.07	0.76	101.06	11.43	1.034.455	0.07	0.76	101.05	11.40
	14	375	0.35	0.82	105.28	12.25	1.102.124	0.35	0.82	105.26	12.26
	15	412	0.55	0.78	108.31	11.70	1.143.915	0.55	0.78	108.22	11.67
	16	343	0.65	0.76	109.82	11.33	1.064.191	0.66	0.75	109.93	11.27
	17	116	0.69	0.82	110.41	12.27	386.589	0.69	0.81	110.41	12.16
Subtotal		1.837	0.35	0.83	105.25	12.47	5.468.206	0.35	0.83	105.24	12.48
Hispanic	12	92	-0.85	0.77	87.27	11.61	117.734	-0.71	0.84	89.36	12.60
	13	126	-0.62	0.76	90.77	11.47	155.810	-0.53	0.73	92.02	10.95
	14	149	-0.38	0.77	94.35	11.60	177.157	-0.25	0.79	96.29	11.82
	15	148	-0.24	0.80	96.34	12.04	190.422	-0.18	0.75	97.36	11.23
	16	115	0.08	0.83	101.26	12.46	144.233	0.15	0.91	102.21	13.67
	17	35	-0.04	0.79	99.38	11.80	46.591	-0.10	0.78	98.48	11.73
Subtotal		665	-0.36	0.84	94.59	12.60	831.947	-0.27	0.84	95.90	12.66
Black	12	119	-1.03	0.66	84.56	9.95	157.316	-0.90	0.64	86.46	9.62
	13	177	-0.73	0.83	89.05	12.43	228.301	-0.67	0.82	89.97	12.32
	14	179	-0.64	0.86	90.46	12.84	229.181	-0.58	0.88	91.28	13.27
	15	169	-0.30	0.83	95.48	12.48	232.414	-0.31	0.86	95.30	12.94
	16	198	-0.20	0.77	96.94	11.59	234.020	-0.23	0.78	96.48	11.67
	17	79	-0.45	0.88	93.20	13.21	91.028	-0.45	0.88	93.26	13.15
Subtotal		921	-0.53	0.85	91.98	12.77	1.172.260	-0.51	0.85	92.37	12.71
Total		3.423	-0.03	0.93	99.61	13.99	7.472.413	0.15	0.90	102.18	13.55

^a Based on unweighted data.

^b Based on total sample weighted data / 100.

Table 1c
Total sample general intelligence (first principal component *g* and its IQ equivalents) broken down by race (NLSY97 data).

Race	Actual N	PC1 <i>g</i> ^a	SD	IQ equivalent ^a	SD	Repr. N	PC1 <i>g</i> ^b	SD	IQ equivalent ^b	SD
White	3.783	0.40	0.91	105.95	13.58	11,075,648	0.40	0.91	105.97	13.59
Hispanic	1.345	-0.32	0.89	95.14	13.30	1,752,715	-0.23	0.89	96.52	13.41
Black	1.784	-0.60	0.88	91.06	13.22	2,320,399	-0.55	0.89	91.75	13.31
Total	6,912	0.00	1.00	100.00	15.00	15,148,762	0.18	0.97	102.70	14.62

^a Based on unweighted data.
^b Based on total sample weighted data / 100.

CAT-ASVAB sub-tests, from which it was derived. Fig. 1 provides the results of a factorial ANOVA of total sample male–female responses, where PC1 *g* and the 7 highly *g*-loaded standardized subscale scores appear as dependent variables, and sex and age are categorical variables.

Use of total sample response weights would have made the curves legitimately representative for 15+ million 12–17 year old adolescents living in the US in 1997 (conf. Table 1c), but then the vertical bars denoting .95 confidence intervals would be too small to be seen with the naked eye. Fig. 1 therefore provides the highly similar age difference curves based on un-weighted responses, which allows visually informative estimates of confidence intervals under less ambitious circumstances. All statistical main effects remain highly significant, however, even for unweighted data: Sex $F(7, 6.894) = 158.908, p < 0.000$; Age $F(35, 29.003) = 35.318, p < 0.000$; Sex * Age $F(35, 29.003) = 2.435, p < 0.000$.

The curves unsurprisingly confirm that age differences along the orthogonal PC1 *g* scale run largely parallel to age differences along the standardized CAT-ASVAB subtest responses, it was derived from. Fig. 1 reveals that all age difference d-curves follow a largely similar course for males and females, with only minor average sex differences before 15. However, female d-curves reach an asymptote at age 16, and then unexpectedly regress, whereas male curves continue to rise over the full age span. The causes(s) and consequence(s) of this discrepancy are discussed later.

2.3.2. Total sample test of Jensen's null sex IQ difference theory

Fig. 2 provides total sample average male and female IQ differences, broken down by race and statistically tested on basis of unweighted scores, i.e. actual Ns.

Black male IQ trails on black female IQ by about 2 points (male $X = 89.90$ SD 13.44 vs. female $X = 91.98$ SD = 12.70; conf. Tables 1a and 1b). The Hispanic sex difference of 1.08 IQ points is in male favor, but does not reach significance (male $X = 95.67$ SD = 13.94 vs. female $X = 94.59$ SD = 12.60). White females trail white males significantly by 1.35 points (male $X = 106.60$ SD = 14.52 vs. female $X = 105.25$ SD = 12.47). Results of the planned intra-racial multivariate tests for sex differences are texted into Fig. 2. The Sex * Race least squares interaction term is significant ($F(2, 6906) = 9.36, p < 0.000$).

Table 2
CAT-ASVAB97 subtests *g*-loadings for the total sample of whites, Hispanics, and blacks (unweighted data; unrotated Principal Component Extraction).

	<i>g</i> -Loadings
General science	0.879
Arithmetic reasoning	0.874
Word knowledge	0.873
Paragraph comprehension	0.866
Mathematics knowledge	0.863
Mechanical comprehension	0.836
Electronics information	0.829
Assembling objects	0.740
Shop information	0.676
Numerical operations	0.675
Coding speed	0.640
Auto information	0.635

The data supports Jensen's (1971) Race × Sex × Ability Interaction Theory (as long as we disregard age differences, see later), and disconfirms his null sex difference theory.

2.3.3. Race–age separated testing of the Race × Sex × Ability × Age interaction theory

A more detailed racial picture of interactions surfaces when the total sample male–female comparisons in Figs. 1 and 2 are broken down further by age for each race separately. Fig. 3 provides results for whites.

Although white male IQ leads at all ages, the differences do not become statistically significant before age 15. Then, white female IQ levels out earlier than male IQ and tend to stabilize at a lower level, whereas male IQ approaches stabilization at a significantly higher level. The wider confidence intervals observed for both sexes at age 17 – as compared to those for the 16 year olds – are due to the relatively lower number of subjects in this age group. The following main effects reached significance: Sex $F(1, 3771) = 17.8, p < 0.000$; Age $F(5, 3771) = 110.9, p < 0.000$; Sex * Age (Yrs.) $F(5, 3771) = 1.9, p = 0.09$.

Fig. 4 provides age curves for Hispanic sex–age differences in IQ.

Again, no consistent sex differences are seen before age 14. The modest male lead at age 15 disappears again at age 16. Female Hispanic IQ then declines slightly, whereas male IQ rises further. Neither Sex ($F(1, 1333) = 2.41, p = 0.121$) nor Sex * Age ($F(5, 1333) = 1.17, p = 0.319$) interaction reached statistical significance, but Age did: $F(5, 1133) = 38.08, p < 0.000$.

Fig. 5 illustrates the black sex–age IQ differences.

Black male average IQ trails female IQ up to age 15, at which time it reaches a plateau. Black female IQ reaches its zenith at age 16 and then drops markedly, coinciding with a black male IQ rise into a significant lead. The Sex main effects did not reach significance $F(1, 1772) = 1.71, p = 0.191$, but Age did $F(5, 1772) = 33.01, p < 0.000$, and so did Sex * Age interaction $F(5, 1772) = 3.05, p = 0.01$.

Overall, Jensen's (1971) original Race × Sex × Ability Interaction Theory receive support as long as we disregard the sex–age differences among whites and blacks. Hispanic scores testify to no interaction, but neither was this race included in Jensen's racial interaction focus.

What happens to the theory if we control for age by restricting the sample to only 17 year olds? Fig. 6 provides the answer for both unweighted and representative samples of blacks, Hispanics, and whites

Clearly, the black–white interaction theory receives no support from representative post-pubertal IQ data.

Table 3 provides representative racial IQ means and dispersion (SD) averages for the group of 17 year olds.

Here white females trail males by 3.60 points, Hispanic females trail males by 7.03 points, and black females trail males IQ by 6.82 points. The Table further indicates that white male dispersion score is 2.79 points larger than female SD, that Hispanic female trail male SD by 2.76 points, and that black female SD trails male by 0.83. The 17 year old overall sex difference, irrespective of race, amounts to 4.60 points in these representative data.

2.3.4. Means versus dispersions

Fig. 7 graphs the total sample Male/Female distributions along the IQ scale, in order to address the question of how sex differences in total means and dispersion scores translate into sex differences in ratios

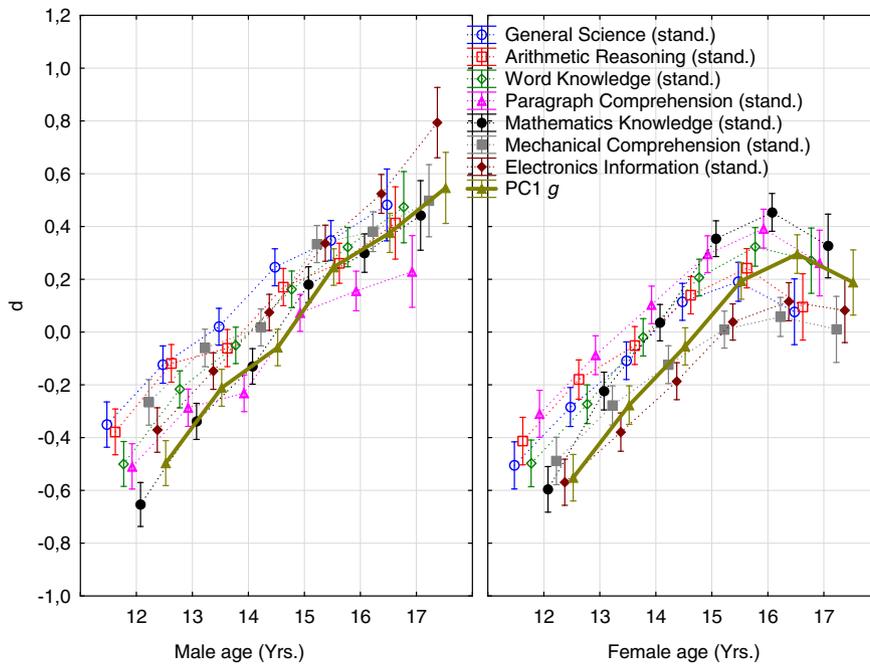


Fig. 1. Total sample unweighted male and female standardized age differences in general intelligence (first principal *g* component, unrotated; fully drawn line), and in 7 highly *g*-loaded CAT-ASVAB subtests (dotted lines). Vertical bars denote 0.95 confidence intervals. Female IQ drops after age 16 whereas male IQ develops further.

and frequencies at extreme ends of the presumed close to normal IQ distribution, *irrespective of race and age*.

The female IQ distribution curve is more narrowly defined around its mean unweighted IQ of 99.61 (SD 13.99), than the male curve, which is visibly flatter around IQ 100.38 (SD 15.92), $F(4, 6910) = 4.63, p = 0.03$. The curves for these particular IQ–SD combinations suggest that males are unmistakably overrepresented at both the lower and higher ends of the IQ distribution, despite the fact that the total sample average IQ sex difference of the total sample amounts to just 0.77 IQ point (compare Tables 1a and 1b). The probability of finding a female with IQ 55 is approximately 6 times lower than finding such a male, and there will be about 3.5 males for each female with IQ 145.

Fig. 8 presents distributions, ratios, and probabilities for the 17 year old white male and female subsamples, calculated from their respective data located in Tables 1a and 1b.

The male IQ average of 114.01 and SD 15.04 combine with female IQ = 110.41 and SD 12.27 to produce a probability for males of about

25 times that of females of identifying a male with IQ as low as 55, and an estimated white Male/Female ratio slightly above 5:1 at IQ 145.

Fig. 9 provides curves for 17 year old Hispanics.

The male IQ of 103.34 and SD 14.39 result in a relatively low probability of finding males in this subsample with IQ 55 (i.e. < 0.10), but there will be an estimated 20 Hispanic males for each female at IQ 145, given the female average IQ of 99.38 and SD 11.80.

Black data presents an interestingly dissimilar picture, as seen in Fig. 10.

The black male 17 year old average IQ of 98.92 and SD 13.66 combine with female IQ 93.20 and SD 13.21 into a highly increased probability of finding dysfunctional black females with IQ 55, relative to black males, and the estimated Male/Female ratio at IQ 145 hovers around 7:1.

To sum up, the results so far indicate that, 1) Post-pubertal male SDs are consistently larger than female SDs, 2) The male probability for

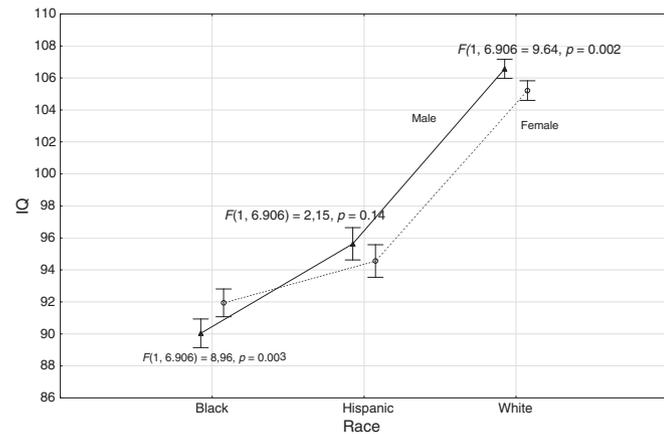


Fig. 2. Racial average male–female IQ differences, N, and results of univariate planned comparisons (NLSY97 data). Vertical bars denote 0.95 confidence intervals. The significant black female and white male IQ leads confirm Jensen’s (1971) theory of Sex × Race × Ability interaction, and disconfirms his (1998) null sex difference theory.

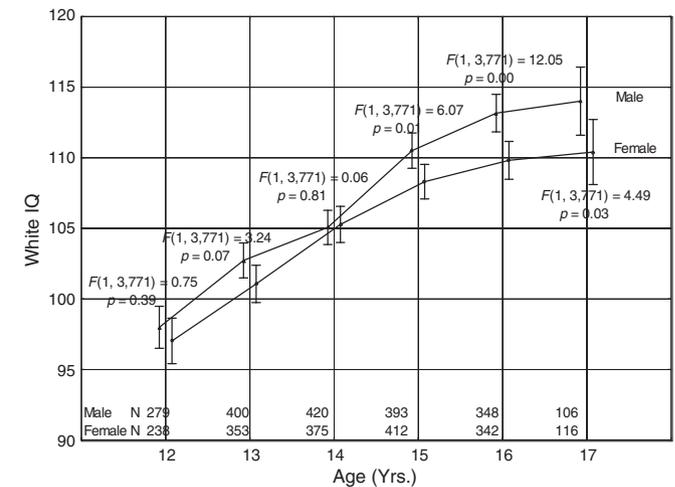


Fig. 3. White average sex differences in IQ across age, N, and results of univariate planned comparisons (NLSY97 data). Vertical bars denote 0.95 confidence intervals. The sex–age difference pattern suggests that white male IQ development stabilizes later and at a higher level than female.

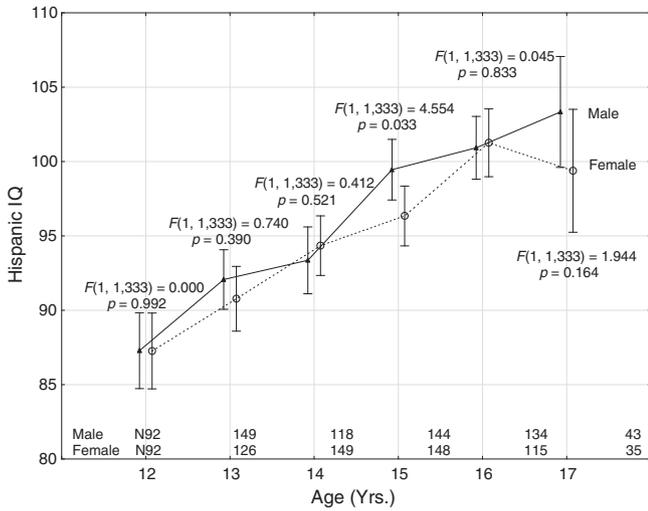


Fig. 4. Hispanic average sex differences in IQ across age, N, and results of univariate planned comparisons (NLSY97 data). Vertical bars denote 0.95 confidence intervals. Sex differences appear sporadic before age 16, followed by a non-significant male IQ lead at age 17, but note low Ns.

having a very low IQ is consistently larger than the female probability, except for blacks, and 3) The Male/Female ratio at the high IQ extreme is always in male favor, even in samples with a miniscule sex difference in average IQ.

2.3.5. The relative meaning of means and variances

Post-pubertal Male/Female ratios, based on means and SDs, will be used in the next section to predict societal consequences in terms of sex differences in real life achievements. We therefore better first clarify the relative importance of sex differences in variance and consider the fact that low-end IQ variability can arise for a number of reasons completely different from those causing high-end IQ variance.

For example, males typically live more dangerous lives than females, so part of the male overrepresentation at the low end of the IQ scale is due to a male bias in IQ reduction through traffic and other accidents, whereas overrepresentation at the high IQ end may be due to favorable gene constellation and hormones modifying brain efficiency.

Deary, Irwing, Der, and Bates (2007) took data from the NLSY79 survey to analyze general intelligence in 1292 opposite-sex siblings with the ASVAB test and its shorter derivative, the Armed Forces Qualification Test (AFQT). They found that females trailed males by about 7% of

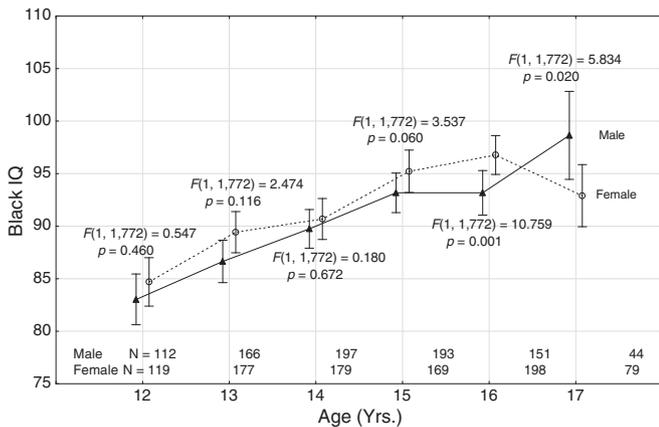


Fig. 5. Black average sex differences in IQ across age, N, and results of univariate planned comparisons. Vertical bars denote 0.95 confidence intervals. A slight early black female IQ lead is followed by a drop after age 16, which, when combined with a male rise, establishes a significant adolescent male IQ lead.

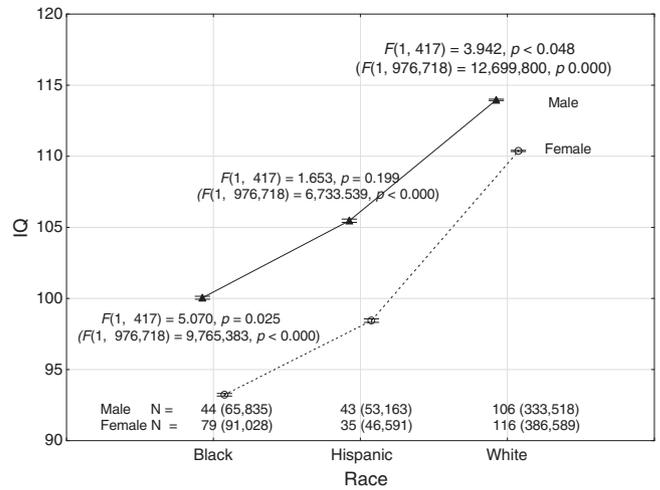


Fig. 6. Sex differences in IQ at age 17 by race, representative Ns, and results of univariate planned comparisons (NLSY97 data). Vertical bars denote 0.95 confidence intervals. Ns and statistical test results are in parentheses. Vertical bars are based on total sample weighted scores / 100. At this age, there is no inverse black–white sex–ability interaction. The significant race–sex differences range from 3.6 IQ points for whites to 6.83 for blacks. The unweighted Hispanic sex difference of 7.03 is not significant, due to low N and considerable dispersion.

a standard deviation in average g score on the ASVAB and AFQT tests, and demonstrated “substantially” less variability. This comes close to the 0.97 IQ point total sample sex difference in the present study after disregarding age and race differences, and is further consistent with the 2.07 SD points lesser female variance, noted in Tables 1a and 1b. In the top 2% AFQT group, Deary et al. (2007) noted that males were overrepresented by almost 2:1, which “... could provide a partial basis for sex differences in intellectual eminence” (my emphasis).

Then again, noting the historical accept of greater male variability in general intelligence, Johnson, Carothers, and Deary (2008) regretted the rare occurrence of clear analyses of the tangible intelligence distributions in representative samples. Drawing on two population-wide surveys of general intelligence in 11 year olds in Scotland, they observed a larger male variability even above the modal IQ level of 105. They, nevertheless, reasoned that despite being present at the high end of the intelligence distribution “sex differences in variability did not appear to account for sex differences in high-level achievement” (my emphasis).

Arden and Plomin (2006) found that males show greater variance in general intelligence from age 2 and concluded that education is not the cause. Lynn, Chen, and Chen (2011) found no sex difference in variance in Raven’s Progressive Matrices before age 7, but from age 10 onwards the variance was significantly greater in their Taiwanese boys.

Sample restriction by age when testing for sex differences in variability is thus important. Is IQ sample restriction also important? One way to address this question is to compare differences in overall Male/Female ratios before and after removing lower-end IQ variance effect. This is possible by first determining the median IQ for the group of 17 year olds, use it as the cut-off point, and then compare the Male/Female ratio for all 17 year olds to that of the subgroup of 17 year olds with IQ scores above the median. Table 4 gives the result of this analysis.

Table 3
Representative sex–race IQ mean and SD differences in 17 years old males and females (NLSY97 data; weighted by total sample weight / 100).

Race	Repr. N	Male Mean	SD	Female Mean	SD	Mean Diff.	SD Diff.
White	720,107	114.01	14.95	110.41	12.16	3.60	2.79
Hispanic	99,754	105.51	14.49	98.48	11.73	7.03	2.76
Black	156,863	100.09	13.98	93.27	13.15	6.82	0.83
Overall	976,724	110.98	15.66	106.38	14.10	4.60	1.56

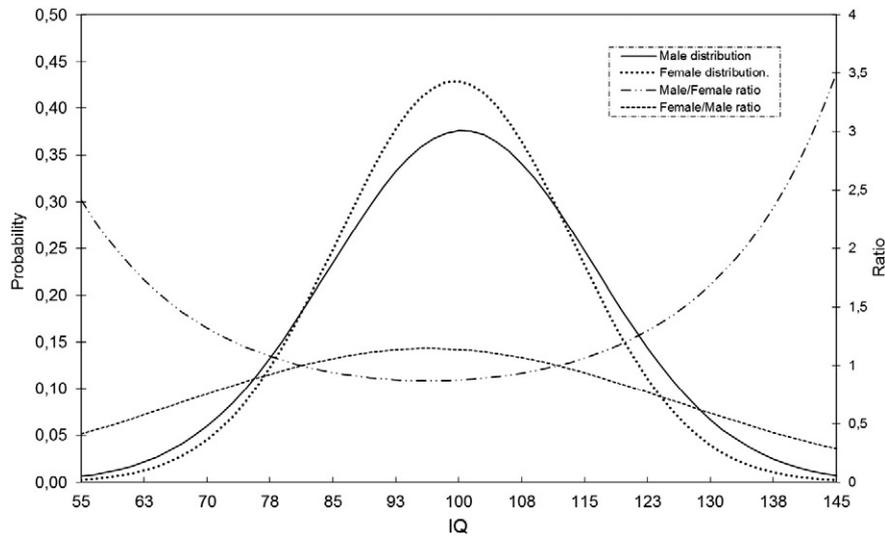


Fig. 7. Total sample sex distributions, probabilities, and ratios as a function of unweighted total means and SDs provided in Tables 1a and 1b (NLSY97 data). The visually obvious sex difference in dispersion scores combines with the small average IQ sex difference in total sample IQ sex difference (i.e. 0.77 points; conf. Tables 1a and 2) to a highly significant male overrepresentation at the low and high end of the IQ distribution.

In unweighted data, the total sample post-pubertal males are 1.17 SD (15.96–14.79) more variable than females, which increases to 1.68 SD (8.79–7.11) in the reduced subsample with IQs above the median. However, the Levene test for sex differences in homogeneity of variance is insignificant for the total sample, but significant for the high IQ subgroup. Using representative total sample weighted data / 100, the sex differences in variability surge to 1.56 (15.66–14.10) and to 2.04 SDs (8.35–6.31), respectively, and the Levene test becomes highly significant in both groups. The Male/Female ratios at IQ 145 are similar all 17 year olds irrespective of weighting data or not, but rise expectedly in the high IQ subgroup to 58:1 for unweighted data, and to 71:1 in weighted data.

Together our results confirm that it is important to master age and sample bias in IQ, also when Male/Female IQ ratios are used for prediction. Another point is that, even if low- and high-end variances may have entirely or partly different reasons along the IQ scale and among males and females, it seems best to include both, when Male/Female IQ ratios are used to predict sex differences in real-life achievement in the general population.

A large-scale study of intelligence in Swedish conscripts (Shakeshaft et al., 2015) concluded, that “high intelligence is familial, heritable, and caused by the same genetic and environmental factors responsible for the normal distribution of intelligence”. They also found that “very bright” groups have reduced variance. This is confirmed by Table 4,

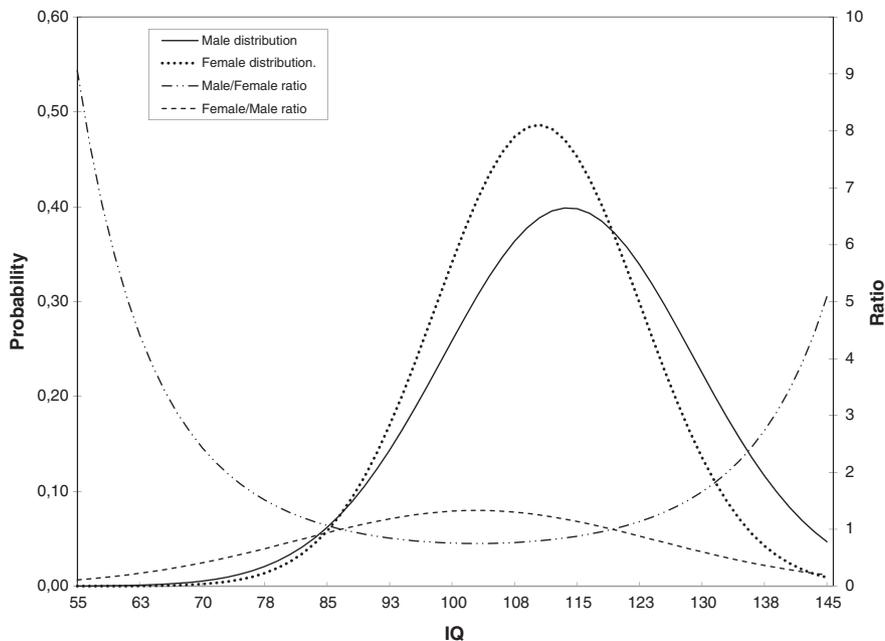


Fig. 8. White 17 year old subsample sex distributions, probabilities, and ratios as a function of their unweighted age means and SDs provided in Tables 1a and 1b (NLSY97 data). For major achievements requiring IQ 145, there will be 5 males for each female, and the probability of acting on a devastatingly low IQ of 55 is many times higher for males.

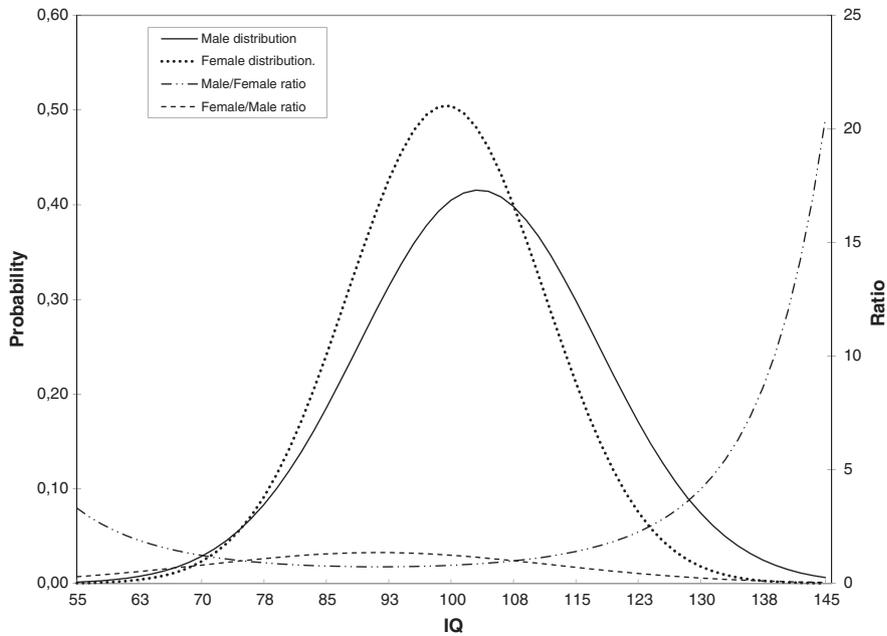


Fig. 9. Hispanic 17 year old subsample sex distributions, probabilities, and ratios as a function of their unweighted age means and SDs provided in Tables 1a and 1b (NLSY97 data). The male probability of low IQs is about 4 times higher than female probability, but there will likely be 20 males for each female with IQ 145.

but the sex difference in SD increased in the high IQ groups by 0.51 (1.68 versus 1.17), and 0.48 (2.04 versus 1.56), respectively.

3. g-Based educational and occupational predictions

3.1. Predicting achievement from g-based IQ Male/Female ratios

Which of the two following statements is correct: 1) High male variance partially explains “sex differences in intellectual eminence” (Deary et al., 2007), or, 2) “sex differences in variability did not appear

to account for sex differences in high-level achievement” (Johnson et al., 2008)?

Before answering this question, it is worth keeping in mind the methodological difference between studies. Deary et al. (2007) based their calculation on 11 year olds at IQ 140, and found 58 boys for each 42 girls there, even despite a miniscule average sex difference in IQ. The present study bases predictions of achievement on the Male/Female ratio of 5:1 at IQ 145 in white 17 year olds (conf. Fig. 8), that is, after the advent of significant sex differences.

We accordingly predict there will be about 80 males for each 20 females in elite educations and occupations.

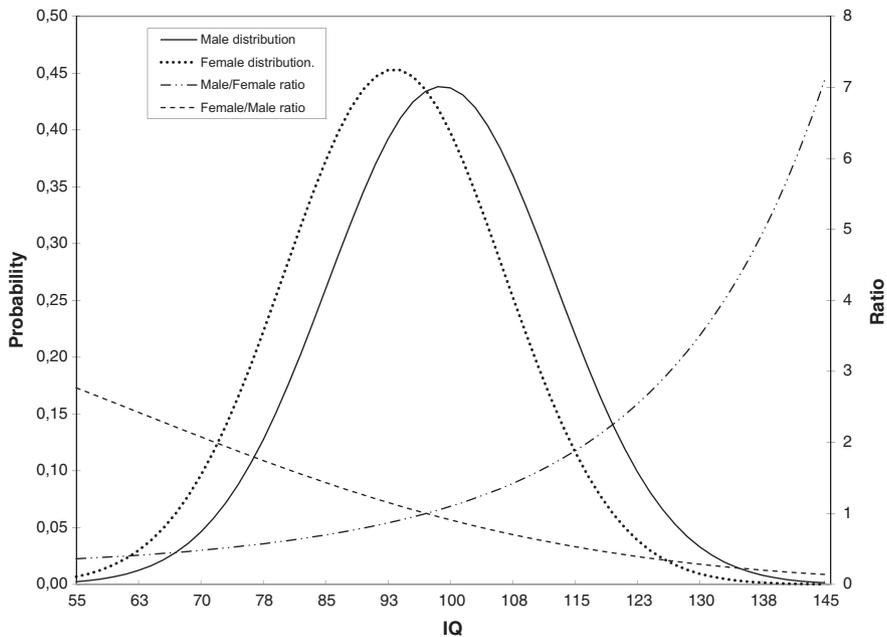


Fig. 10. Black 17 year old subsample sex distributions, probabilities, and ratios as a function of their unweighted age means and SDs provided in Tables 1a and 1b (NLSY97 data). Inconsistent with the higher white and Hispanic male probability of finding dysfunctionally low IQs, it seems that black females have increased probability of suffering from very low IQs, and males dominate at the high end of the IQ scale.

Table 4
Comparing male–female IQ averages, homogeneity of IQ variances, and Male/Female ratios for all 17 year olds, versus subgroups of 17 year olds with IQs above total group medians.

Group	All 17 year olds				Median IQ	Above median subsample 17 year olds			
	N	Average IQ (SD)	Homogeneity ^a	Male/Fe–male ratio at IQ = 145		N	Average IQ (SD)	Homogeneity ^a	Male/Fe–male ratio at IQ = 145
Male ^b	193	108.19 (15.96)	$F = 1.44$			111	119.44 (8.79)	$F = 9.69$	
Female ^b	230	102.82 (14.79)	<i>n.s.</i>	3.5:1	106.43	101	116.00 (7.11)	$p = 0.002$	58:1
Total	423	105.27 (15.54)				212	117.80 (8.20)		
Male ^c	452,516	110.98 (15.66)	$F = 6,858.73$			270,079	121.44 (8.35)	$F = 11,864.78$	
Female ^c	524,208	106.38 (14.10)	$p < 0.000$	3.5:1	109.31	217,330	119.08 (6.31)	$p < 0.000$	71:1
Total	976,724	108.51 (15.02)				487,409	120.38 (7.60)		

^a Levene test for homogeneity (significance means the null-hypothesis about equal male–female IQ variances must be rejected).

^b Based on unweighted data.

^c Based on total sample weighted data / 100.

3.2. License to predict IQ–achievement relations cross-nationally

In order to test the predictive validity of sex IQ ratios in the present study (hypothesis 5), we must first obtain license to generalize from US intellectual conditions to Danish ditto. We thus note with Lynn and Vanhanen (2012) that 1) the average IQ for the USA is 97.5 points and 97.2 for Denmark, that 2) there is considerable cross-national factorial IQ invariance, that 3) there are fairly linear IQ–Occupational status and Income relations (e.g. Nyborg & Jensen, 2001), and that 4) we can apply general classical normal distribution principles across nations. All this provides communality in predictive power – with due caution and perhaps only for some purposes – when we use US white 17 year old male and female IQ data (Tables 1a–1b) to forecast sex differences in white achievements in Denmark, or if we similarly had forecasted sex differences in American achievement from Danish IQ ratios. Obviously, when evaluating the predictions, it is worth remembering, that even if correlation is needed for explanation, it does not prove any particular one.

3.3. Low level educational achievement

Given the inconsistent pre-pubertal sex differences in IQ, illustrated in Figs. 3–5, we predict small if any sex difference in grading in primary and secondary schools. This obviously is wrong. Young US and Danish girls both earn higher grades in school than boys (Danmarks Statistik, 2014a; Jensen, 1998), irrespective of race, perhaps because teachers generally consider girls to be better pupils, doing their homework, and following instructions, with little noise. Calvin, Fernandes, Smith, Visscher, and Deary (2010) found that g correlates ($r = 0.83$) with an educational factor score, but it did not explain sex differences in academic achievement at age 11. The slight IQ advantage of pre-pubertal black females may in fact give them a minor, if only transient, developmental IQ advantage in education before puberty (conf. Fig. 5).

3.4. Higher education

It appears that a similar decoupling of IQ from higher education takes place in the US. A report from the Educational Testing Service (Kirsch, Braun, Sum, & Yamamoto, 2007) thus predicts a “perfect storm” coming soon to the US, as current projections suggest a serious decline in the educational and occupational capabilities, which will make the US lose its competitive edge in world economics. Several international educational comparisons (e.g. PISA) suggest that Denmark and other Nordic countries also suffer increasingly under educational degradation, which currently is explained predominantly in terms of negative social, economic, or school factors.

There is another way to frame this problem. Demographically, there are currently more males than females in all age categories of the Danish population up to age 59 (Danmarks Statistik, 2014b). It is further true that no less than 60% of those earning a university candidate degree in

2010 were female (Styrelsen for Forskning og Innovation, 2013). This indicates that 1) the male numerical population overrepresentation, 2) the appearance of significant white male post-pubertal IQ advantages (conf. Fig. 3), and 3) the increasingly higher Male/Female IQ ratios at the high IQ end (conf. Fig. 8), all become decoupled from the overall linear relationship between IQ and academic achievement at the third level of education. One likely reason for this is the various goal-directed governmentally sponsored equality campaigns, which have dominated Danish educational policy since the 1980s, and have succeeded in setting many more females on the course of higher education. Governmentally sponsored economic reward to faculties hiring more female staff members at universities may have contributed, too (but see below for a recent change). These campaigns seem to trump ability by lowering academic standards. An efficient bulwark against the effects of higher Male/Female IQ ratios seems to be to let in more unqualified female students and staff, and having them succeed in academia by lowering criteria. A further negative factor is that Danish universities are basically financed by the number of grades they pass on, not by the number of students enrolled. A likely unfortunate implication of this is that each failing student means less money for faculty and, indirectly, for research. This may tempt the academic system to not only let in more problematic students, but also to let them graduate.

Interestingly, as the effects of damage done to academia gradually accumulated, some of the sex-specific educational recommendations were recently abandoned. It is also encouraging to note that the degradation of the quality of faculty at Danish universities apparently has been limited chiefly to the lower levels. This can be deduce from the fact that the ratio of males to females increases as we step up the university hierarchy, from Bachelor degrees (BA), to Master degrees (MA), to Ph.Ds, to the sex ratio among full professors, in fair accordance with increasing Male/Female IQ ratios. Thus, despite the current 60% female candidate “overproduction”, only 16% of them get a full professorate. The figures for other countries within Europe are quite similar (Styrelsen for Forskning og Innovation, 2013).

In other words, the selection for higher IQ in intellectually demanding positions in academia is increasingly more consistent with predictions based on the post-pubertal Male/Female ratios provided in Fig. 8.

3.5. Occupational elite achievement

To count as a general principle the predictive power of Male/Female IQ ratios must keep up when also predicting sex differences in intellectually challenging positions in the occupational world. Let us therefore first test whether Male/Female IQ ratios lead to increasingly more reliable predictions of sex differences, the higher we go in the occupational chain of commands. Let us, for example, assume that positioning of the most successful industrial business leaders requires IQ 145 or higher. Let us further base predictions on the 17 year old white Male/Female IQ ratio at IQ 145 in Fig. 8. This circumvents likely complications with predictions involving developmental and racial considerations. The Male/Female sex ratio of 5:1 at IQ 145 let us expect that 20% of all

white Danish females will enter the most important, intellectually demanding, responsible, and powerful positions in society.

The Danish organization *Corporate Governance* (2009) presents running statistics for the proportion of females at various corporate boards, which let us test this prediction. Thus, in September 2009, 19% out of *all corporate board positions* were taken up by females, as compared to 19% when females were elected at assembly meetings, and 21% when elected by colleagues (the sex of the relatively few foreign board members was not registered). The corresponding figures for *really big business companies quoted on the stock exchange* were lower: 10% and 5%, with the exception of 30%, respectively. The corresponding 2013 figures are: All corporate board positions 19%, 19%, and 23%, and for *stock exchange noted big business company boards* 23%, 8%, and 31%, respectively (*Corporate Governance*, 2013). Except for the number of females selected by their own colleagues, most figures for 2009 and 2013 come close to the 20% predicted from the Male/Female IQ ratios in Fig. 8, and were lower in the intellectually most challenging big business companies. In other words, the contemporary high-level Danish corporate world is ruled by about 80% males. This resonates well with the estimated 5:1 sex ratio at IQ 145. Moreover, the heavy male bias has not changed much since 2009.

Apparently, for academic faculty and corporate rank order alike, ability increasingly trumps the effects of sexual equality campaigns in situations, where relatively low IQ actors (of both sexes, obviously) become too costly in terms of poor research and teaching, and loss of profit and workplaces. It suggests that underneath all public declarations of equality in academia and industry, there is a silent understanding that vital international university and corporate competitive edges will be blunted by lowering educational and student standards, as well as by academic and managerial incompetence.

The Danish parliament tasked in 1997 Togeby, Andersen, Christiansen, Jørgensen, and Vallgård (2003) to analyze the parliamentary power conditions at the transition to the 21st Century. The committee found, among other things, that males occupy about 80% of the most powerful positions in society. This is entirely consistent with the ratio-based predictions.

It is noteworthy that the reliability of predictions from Male/Female IQ ratios to the highest educational and corporate achievements survives decades of goal-directed governmental campaigns to attain sexual equality in all segments of society. Possible exceptions are found in those lower segments of the educational, occupational, political, and corporate hierarchies, where misplacement with respect to ability is relatively cost-neutral. As soon as the consequences of misplacing low-ability individuals in high-ranking positions become grimmer, up to where just one wrong complex corporate decision may cost thousands of work places or even company or national life, the selection for high-but-scarce intellectual ability becomes paramount, irrespective of sex, race, and creed.

The scientific validity of rationally IQ based selection is defied by sex norming, as when females get preferred when applying for higher positions, in accordance with a certain preset percentage quota. Poulsen (2014; both data and references) has compiled a useful illustrative list of national aberrations from predictions based on sex IQ ratios in terms of the percentage of *Women on Boards* in different countries. The percentages range from .8% in Portugal, 1.4% in Japan, and 2.1% in Italy, over 5% in China and India, 9.7% in the EU, and 15% in the US, up to 18.1% in Denmark, 25.7% in Finland, 26.9% in Sweden, and 44.2% in Norway. Reinforcing the threat to the validity of predictions from ability, is the fact that increasingly more countries, now even cross-national organizations, are considering legislating fixed sex quota. For the EU, the Commission for example suggests a quota of 30% females, but the Parliament prefers 40%.

The large cross-national differences in preferred female percentages reflects the absence of applying rational principles, and this threaten the validity of scientifically based predictions from Male/Female IQ ratio. Moreover, Ree and Earles (1990) illustrated with ASVAB data that *g* explains 28.4% of training success for unskilled military categories, some

60% for various professional categories, and full 77.3% for nuclear weapons and other complex specialist training. Welsh, Watson, and Ree (1990) finally confirmed the unique predictive power of *g* for trainability in comparison to all other factors extracted from that test and from all other sources combined.

One major lesson here is that the higher the *g*-level required for success, the more accurate become predictions based either on *g* alone, or on Male/Female ratios which include effects of the higher male variability. Another, that ratio-based predictions may even be more precise than *g*-alone predictions in samples with a small average sex difference in IQ or *g*, as variability differences count more for ratios than IQ average group differences (conf. Fig. 7).

In conclusion, predictions based on Male/Female ratios suggest that female potential for occupational and educational achievement is under-rated in countries like Portugal, Japan, Italy, China, and India, and over-rated in the EU and the Nordic countries. The ongoing legislation on increasing sex quotas for the EU seems unrelated to imperturbable rational scientific principles, and may be costly. This said, an under-rating of available female intellectual potentials may be equally damaging to a nation's international competitive edge, because average IQ correlates highly with GDP and other economic measures at the national level (Lynn & Vanhanen, 2012). It would profit some nations to cultivate this unseen female high IQ resource.

4. Discussion of data and results

4.1. Data quality

The NLSY97 data wave provides a more accurate basis for testing the null – sex – difference and interaction theories than did the samples used originally by Jensen (1971, 1998). The NLSY97 wave is thus designed explicitly to represent the total black, white and Hispanic populations of adolescent boys and girls living in the United States in 1997. Moreover, it was a serious restriction that the subjects in the Jensen (1971) study were mostly below age thirteen, as we have just documented that sex differences in general intelligence do not show up reliably before 15, even if some sex differences of lower order – like in visuo-spatial abilities – appear reliably earlier. The age range and *Ns* in the present study – from 12 to 17 – further allows for meaningful post-pubertal age-difference breakdowns. Together, this means that the current tests for sex differences and Race \times Sex \times Ability (\times Age) interactions unfolded in a methodologically speaking more appropriate framework than did previous studies.

The results provided seven different insights into the existence of sex, race, and age differences in mean IQ, variability, changes over age, the meaning of the larger male variability for Male/Female ratios and probabilities at extreme IQ values, and the predictive power of Male/Female ratio differences for educational and occupational achievement.

4.2. The female post-pubertal IQ decline

First, *total sample* age-difference standard unit *d*-values and *g* rise fairly evenly for both males and females from age 12 to 15 (Fig. 1). We see this when we inspect the curve for general intelligence, *g*, and each of the seven separate *g*-loaded CAT-ASVAB subscale scores. Even without weighting the data, and thus depending on statistics based on the much lower actual number of subjects, both Sex, Age, and Sex \times Age effects came out statistically significant.

Interestingly, female performance curves drop visibly past age 16, whereas male curves continue to rise to reach their adult asymptote at a later age. This raises hard questions about the most likely endogenous or exogenous cause(s) for the female post-pubertal IQ leveling-off, or even subsequent slight ability decline, as it combines with a steady male rise to materialize in significant post-pubertal sex differences in all three races. A discussion of the evolutionary “why” and the ontogenetic proximate “how” questions is deferred to Section 5, in a quest

for the origin and possible mediating mechanisms behind this developmental phenomenon

4.3. The Race \times Sex \times Ability Interaction Theory

Second, Fig. 2 provides a preliminary total sample age-collapsed basis for testing both Jensen's, 1998 theory of null sex differences, and his Race \times Sex \times Ability Interaction Theory (IT). Planned test comparison were performed in order to favor the two hypotheses under scrutiny, but the statistical analyses were based on the small number of actual Ns, mapped in Tables 1a–1c rather than on the much larger representative number based on weighted data, in order to produce conservative test results.

Under these circumstances, the null sex difference theory must be rejected, as black male IQ trails black female's significantly by 2.08 IQ points (which, by the way, is less than the 3 IQ point significant sex difference Jensen originally found). Moreover, white female IQ trails white males' significantly by 1.35 IQ points (where Jensen observed a 1.5 points difference).

The inverse black–white sex difference provides support for Jensen's IT, and the interaction term is highly significant ($p < 0.000$). Despite this, it seems reasonable to accept Jensen's original conclusion that an inverse black–white IQ sex difference of that size (and inconsistency, see Fig. 5) is of little practical value in explaining a racial difference in educational achievement in environmental terms, as he originally had hoped. Any black female educational advantage apparently has to find another explanation than in their slight (and withering) IQ advantage. The Hispanic total sample sex differences of 1.08 IQ points did not reach statistical significance in unweighted data.

Third, Fig. 3 indicates that the white male IQ lead first becomes significant at age 15. The Hispanic data in Fig. 4 are less regular, perhaps due to the smaller number of Ns, but even they suggest that the male IQ lead appears at, or soon after, age 16, to eventually reach significance at age 17, assisted by the female IQ decline. It is worth noting, that the slight Hispanic female decline after 16 provides part of the explanation for the total sample female regression seen in Fig. 1, because the white female average IQ did not suffer such decline. Age was the only significant main effect in Hispanic data. The remaining explanation for the total sample female IQ decline after age 16 seen in Fig. 1 emanates from the black IQ data in Fig. 5. The slight tendency for young black females to earn higher IQs than black males, even if it is significant at age 16, then turns into an IQ decline at the time when black male IQ seems to recover from its plateau between ages 15 and 16. These two tendencies morph into a significant black young adult male IQ lead. Overall Age and Sex \times Age interaction both turned out as significant main effects, but Sex did not.

Fourth, Fig. 6 presents the final proof that Jensen's Race \times Sex–Ability interaction thesis crumbles post-pubertally. Total sample data are here restricted to only those for the 17 year olds, and thus harvested at a time when an adult sex difference had established itself in all three racial groups. This tabulation is based on using total sample weights / 100, which makes the results representative for the entire population, and explains the sky-high levels of statistical significances added to the Figure (as compared to the modest levels based on unweighted data, also added). At age 17 white males begin to lead white females by 3.60 points (conf. weighted data in Tables 1a and 1b), and black males lead black females by 6.83 IQ points. This means that the black–white Race \times Sex \times Ability interaction theory with its expected meaningful educational implications has completely vanished. The Hispanic sex difference amounts to 7.03 IQ points in male favor.

In general, Jensen (1971) was right in dismissing his own interaction theory, as the small and inconsistent pre-pubertal IQ differences seem of little educational relevance for understanding early race differences in educational achievements, and the race interaction term disappeared entirely in post-pubertal data. However, Sections 3.4 and 3.5 suggested that the mean post-pubertal race and sex differences in IQ, and the

notably larger male variability, have good potentials for correctly predicting later high-level educational and occupational achievements (see also later).

4.4. Lynn's developmental theory versus Jensen's null sex difference theory

Fifth, the age-difference analyses of nationally representative black, Hispanic, and white populations essentially confirm Lynn's (1994, 1999) dual developmental theory: 1) Sex differences in general intelligence appear only sporadic before age 15 and, 2) after puberty, there will be significant IQ differences in male favor. The first part of Lynn's theory is partly confirmed, even if there is an inconsistent and slight male advantage for whites and Hispanics, and a slight female advantage in blacks. The second part of the theory is fully confirmed, as the 17 year male IQ advantage amounts to between 3.60 and 7.03 points across the three races studied, using weighted data (Tables 1a and 1b).

Lynn and Kanazawa (2011) analyzed the multitudes of twentieth century IQ studies, and concluded that some supported and some spoke against Jensen's (1998) null sex difference theory. They noted that several studies disregarded the vital sample age differentiation, and that supportive studies found an adult male IQ advantage ranging between 2.8 and 11.5 IQ points. Meisenberg (2004) analyzed data from the early NLSY79 ASVAB test and found no black or white sex differences in *g* at age 15. However, from age 16 onwards a white male advantage of 4 IQ points grew to 6.5 points at age 22.3, and the 16 old black male advantage of just 1 IQ point grew to 2.15 points among 22.3 year olds. In comparison, the corresponding sex differences for 17 year old whites and blacks in the present analysis of weighted CAT-ASVAB data amounted to 3.60 and 6.83 IQ points, respectively, and to 7.03 for Hispanics (conf. Fig. 6). Lynn and Kanazawa (2011) further inspected large-scale data from the National Child Development Study (NCDS) in a longitudinal analysis, and found that those same girls, who at ages 7 and 11 slightly outscored boys of similar ages, obtained lower average IQ than boys at age 16. According to Lynn's (1994, 1999) developmental theory, this IQ lead reversal is a function of girls earlier maturing, and then boys catch up.

It tells something about the current Zeitgeist that – despite the unambiguous empirical support for Lynn's developmental theory – Lynn and Kanazawa (2011) were able to list up a large number of skeptic contemporary studies, all arguing unambiguously in favor of Jensen's (1998) null sex difference theory. Methodologically, this indicates that not all researchers doing meta-analyses of sex differences in general intelligence control properly for age and/or range restriction in their study. Moreover, not all analytic methods are equals in power to identifying the small adult sex difference (Nyborg, 2005). Saggino et al. (2014) thus found a genuine 7 IQ points (Full Scale WAIS-R) sex advantage in elderly males, but an ensuing Multi-Group Confirmatory Factor Analysis of these same data entirely missed this large difference. Apparently, some of the sophisticated multivariate statistical approaches lack the statistical power to detect small differences, such as sex as compared to race differences, and reports using them add to confusion.

4.5. Extreme post-pubertal Male/Female probabilities and ratios

Sixth, substantial sex and race differences in Male/Female ratios and frequencies at the extremes of the IQ distribution appear when classical distribution theory is applied to group mean IQs and SDs (found in Tables 1a–1c) for post-pubertal 17 year old subjects. The total sample results were illustrative, but the ensuring separate analyses of 17 year olds revealed informative racial differentials in timing and variability.

Fig. 7 thus indicated that there will be about 3.5 males for each female at IQ 145 (+ 3 SD) in the total sample, as well as an overrepresentation of severely retarded males with very low IQ 55. This low IQ lies 15 points under – 2 SDs, which commonly is acknowledged as a sign of mental retardation. The corresponding probability of finding females with this low IQ is about 0.05.

Fig. 8 further suggests that the high probability of finding males with very low IQs in the total sample (see Fig. 7) is due mainly to the prominence of very low IQ white males. The similar probability curve for male Hispanics with IQ 55 comes close to 0.05 (Fig. 9), and is even close to zero in Fig. 10 for black males. Moreover, this pattern reverses in blacks, as the black Female/Male probability for very low IQ (Fig. 10) hovers around 0.175. These tendencies are camouflaged in multi-race analyses, such as the racial aggregation in Fig. 7, where the Female/Male probability comes close to zero.

Jensen (1998, pp. 367–369) reported that dealing with low IQ blacks gave neither mental retardation teachers nor himself the impression that they had clinical problems, whereas interviewing similarly low-IQ whites pointed to clinically certifiable developmental and clinical problems. This suggested to Jensen that most whites with very low IQs suffer from neural deficits whereas most black low-IQ people display “normal” neural functioning even in the low IQ range.

Developmental and clinical psychologists generally acknowledge that far more boys than girls encounter educational problems, but Fig. 10 suggests that post-pubertal black females are exposed too, which apparently contrast with Jensen's (1971, 1998) observation that pre-pubertal black girls do better in school than white girls. The data suggests, in other words, that the black female pubertal switch from a slight pre-pubertal IQ advantage to a large post-pubertal low-IQ overrepresentation is related to specific events taking place at puberty, which might even be related to similar causes in explaining the post-pubertal Hispanic female IQ decline, illustrated in Fig. 9. Such causes might be genetic or environmental, as very low IQ scores can arise from chromosome anomalies or single-gene disorders, or be due to environmental trauma like head injuries or birth complications (Arden & Plomin, 2006). However, the narrow timeframe for the black and Hispanic female decline points to the existence of more dynamic causal factors acting around puberty. This possibility is modeled in Section 5.

It should be noted that in a more specialized cognitive area, Wai, Cacchio, Putallaz, and Makel (2010) calculated Male/Female ratios for mathematical ability for 1.6 + million 7th grade students in the top 5% in ability across the period 1981–2010. They noted that the ratios were substantially lower than 30 years ago, but had been stable over the past 20 years, and still favored males. In 2012, Wai, Putallaz and Makel (2012) added that, despite the decrease in the Male/Female math ability ratio, the difference is “still likely one factor among many explaining female underrepresentation in some professions”.

4.6. Race and occupational achievement

Seventh, individuals typically rank-order themselves over their lifetime in accordance with their IQ (Gottfredson, 2003), because daily life is an accumulative IQ test (Gordon, 1997) – even if not well standardized. In general, at one extreme, individuals with IQs below 90 tend to end up unemployed or in unskilled jobs, and individuals with IQs above 125 have 50–80% innate potential to become scientists, senior manager, or high-ranking executives. The typical average white > Hispanic > black IQ race rank order reflected in Table 1c leads, when treated in accordance with distribution theory, to the expectation that there will be very few blacks, relatively more Hispanics, and most whites at the very high end of the IQ scale.

This would feed the expectation of a corresponding racial hierarchy of applicants for the highest societal and occupational ladders – everything else equal. But everything is not equal here. Specific environmental factors, such as affirmative action in the US, and obligatory sex quotas in some countries, may cause predictions to fail in forecasting IQ–achievement relations. Despite this noise, we see that the exponentially increasing Male/Female ratios based on combined IQ and variability, enable us to forecast sex differences in achievement with fair accuracy.

It should be noted that, in a related area, Rindermann and Thompson (2013) analyzed US National Assessment of Educational Progress

(NAEP) data from 1971 to 2008, and transformed the educational means and percentiles in reading and mathematics to conventional IQs and SDs. Among other things, they noted that the white-Hispanic gap reduced over the period from 11.59 to 8.46 IQ, and the white–black gap from 16.33 to 9.94 IQ. The authors remark, however, that “Due to its link to school curricula and knowledge (especially in mathematics), the NAEP test is more likely to tap crystallized than fluid intelligence”. This makes it questionable whether these encouraging trends generalize without further ado to the present study based on factor g IQ measurement, which arguable show more race differential stability over time (Rushton & Jensen, 2005).

The possibility also exists, that black male–female-differences may be biased by the high incarceration for black males, but this is unlikely to be a consistent major factor over the full age range of 12–17 years in the present study.

4.7. Empirical conclusions

The findings confirm the existence of race differences in IQ (Rushton & Jensen, 2005). They demonstrate that sex differences in general intelligence first appear robustly around puberty across (three) race(s). They show that Male/Females IQ ratios, calculated at puberty, predict substantial sex differences in educational and occupational achievements with fair accuracy, even if they obviously do not identify the cause(s).

5. The origin and persistence of race and sex differences in IQ and achievement

5.1. The hard problem

Which is the most promising approach to explain the origin, extent, and persistence of these sex and race differences in ability and achievement? Essentially, there are three different approaches (Nyborg, 1997), all illustrated in Fig. 11.

The all surface analyses strive to explain phenotypic individual and group differences in terms of environmental factors at a high level of abstraction. Typically, developmental or behavioral characteristics are explained by characteristics in the socio-cultural-economic environment. Bottom up analyses connect different levels by observing physiological events or genetic differences, and relate them to phenotypic individual or group differences. Top-Down theories connect different levels by taking point of departure in phenotypic phenomena and relate them to underlying physiology or genetics. The all bottom analysis looks for molecular interaction patterns among genotypes (evolved via universal molecular Darwinian selection) and same-level mass-molecular manifestations like body and brain structures, development, behavior, and the construction of culture and society. It is called *Psychology*, in which high-level terms like psyche, mind, culture, and society are substituted by low-level references to the physics and chemistry of brain functions and behavior (Nyborg, 1994; see later).

Ceci, Ginther, Kahn, and Williams (2014) exemplify an all surface approach. Even if these researchers acknowledge that today's female underrepresentation in academia is perhaps not due to yesterday's gender discrimination, they suggest that today's “pre-college factors [barriers] and the subsequent likelihood of majoring in these fields ...” are. Moreover, Ceci and Williams (2011) note that “differential gendered outcomes in the real world result from differences in resources attributable to choices ...” so that we should rather focus on “education and policy changes that will make institutions responsive to differing biological realities of the sexes”, apparently meaning biology is just a factor to be circumvented by reallocating resources. Previously, in 2010, Ceci and Williams (2010) summarized observations from several sources, and concluded that “among a combination of interrelated factors, preferences and choices – both freely made and constrained – are the most significant cause of women's underrepresentation.” In 2009 Ceci, Williams and Barnett (2009) examined possible causal

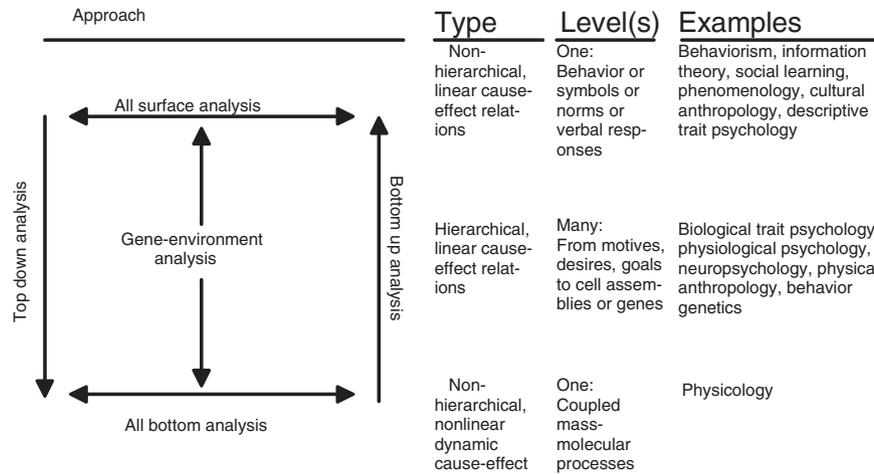


Fig. 11. Types of analytic approaches to abilities and personality (from Nyborg, 1997, modified). See text for explanation.

pathways, and found that the “Biological evidence is contradictory and inconclusive”, so they resolved that “women’s preferences, potentially representing both free and constrained choices, constitute the most powerful explanatory factor; a secondary factor is performance on gate-keeper tests, most likely resulting from sociocultural rather than biological causes.” Taken together, this position reflect a predominantly all surface analysis, according to which biological factors are at best secondary, and can be overridden by environmental means.

Lynn (2008) criticized the consensus position reflected in the 2007 book *Why aren't there more women in science?* edited by Ceci and Williams (2007), for neglecting the male advantages in 1) general intelligence, 2) the larger male dispersion of IQ scores, 3) the male advantage in rotation ability, 4) the larger male interest in science, and 5) the higher male motivation for achievement and career success. It is further an obstinate problem for all surface analyses that, despite centuries of devoted research, we do not know how to use the hitherto identified social causes to override the current persistent race and sex differences in IQ and achievement in empirical causal terms, still less how to safely identify the conditions triggering their origin in the first place. What we know for sure is, that intensive long-term state-funded goal-directed social engineering project, based on principles of (sex) equality, have failed to elicit long-time changes in the heavily male biased societal power balance, at least the one currently on display in modern Denmark. Decades of affirmative actions in the US, based on ideals of racial fairness, have also largely failed, and so have massive attempts (e.g. Project Head Start) to eradicate racial, educational, and economic differences (Herrnstein & Murray, 1994). When posed with the question *How much can we boost IQ and scholastic achievement*, Arthur Jensen’s (1969) (in)famous reply was after 100+ pages careful analysis: *Not much*. According to Fig. 11, the main problem is, that social, cultural, and environmental studies often rely on presumed high-level all surface interactions among environmental factors, typically reflected in non-directional correlations or based on plausibility arguments, but remain unrelated to differential psychological studies of individual and group differences.

Bottom up molecular analyses may hold greater promises. However, to the best of my knowledge, we do not yet have an empirically testable DNA model for the evolutionary origin and persistence of sex and race difference in intelligence and achievement. The studies of phenotypic effects of discrete genes or gene complexes has for a long time not been able to account for more than a few percent of individual IQ variation, and it proves difficult to reproduce even these results. Moreover, they neither identify the pubertal dynamics we have documented, nor the genetic origin of race differences in IQ, even if the possibility of genetic components in current race differences is admitted (Rushton & Jensen, 2005).

A chromosome study of IQ variability by Johnson, Carothers, and Deary (2009) provides an interesting explanation for the larger male variability in terms of a mix of two normal distributions, caused by the dual presence of genes on the X chromosome, one for mental retardation and one for normal population variation in IQ, as a function of sexual selection. Turkheimer and Halpern (2009) commented that the greater male propensity for mental retardation could be accounted for by genes on the X chromosome, and added that no known “intelligence genes” could explain the higher male variability at the right end of the IQ distribution (conf. Table 4).

A top down approach by Lynn and Kanazawa (2011) submitted that a mild tendency for polygyny during human evolutionary history gave early maturing girls a reproductive advantage in their competition with other females for early offspring at a time with optimal egg quality. They thus connected the ultimate evolutionary *why* question with later reproductive differentials via presumed genes, and further related a female maturational advantage to accelerated IQ development. Such a theory may begin to explain individual and racial IQ differentiation at puberty, but neither does it identify the physiological mechanisms involved nor explain the systematic pubertal dynamics, we see in Fig. 1 and discuss later.

All told, the hard problem remains after a century of research using all surface, top down, or bottom up analyses. We are left with the impression that an invisible hand prevents eradication of long-time hardened, consequential, and poorly understood contemporary sex and race differences in IQ and related achievement, which we easily observe in all societies. Still worse, the evidence we have suggests that even costly long-term goal-directed social engineering projects provide no remedy, as the sporadic improvements waters out after a few years. Worst of all, unnecessary scientific controversy, raised by the politically correct, threatens to stifle scientific discussions by turning the use of words like sex, race, and intelligence into taboos.

5.2. The physiological approach to the hard problem

Perhaps, the solution to the hard problem may be to introduce an all bottom comprehensive IQ–Sex–Race–Age model. This model would preferably account for: 1) the evolutionary origin of sex and race differences in IQ, 2) their persistence over centuries, 3) the ontogenetic sex–race–age IQ differentiation seen before puberty (Figs. 1, and 3–5), and 4) the abrupt IQ changes seen after puberty (Figs. 7–10 and Table 4).

Some 20 years ago, I suggested that we begin by identifying some of the physiological mechanisms, presumed germane and active during the ultimate selection process towards the evolution of sex and race differences, and then couple their physico-chemical agents and actions to contemporary ontogenetic proximate physiological mechanisms.

Throughout this operation, the model should include only ultimate and proximate physiological variables with equal causal standing, i.e. there must be at least some bio-chemical similarity between ultimate and proximate agents, their biological mechanisms, and the effects they mediate through receptor molecules in specialized target tissues (Nyborg, 1994, pp. 48–52, 2007). Throughout the development of such an ultimate–proximate connective matrix model, we must strive to prevent committing category errors by jumping between entirely different levels of explanation, e.g. chemical versus conceptual or cultural (Nyborg, 1997), as illustrated in Fig. 12.

A recent study by Davis et al. (2015) may fit the idea of an all bottom approach. These researchers found a numerical relationship between brain size, measures of general and mathematical intelligence, and the copy repeat number on the DUFF 1220 gene site. DUFF 1220 is about 65 amino acids long and located primarily on chromosome 1. DUFF copy number has a long evolutionary past, as copy number increases with still larger brains across species, and with a given species evolutionary proximity to humans (O’Bleness et al., 2012). Assuming that high general and mathematical intelligence reflect optimum physiological conditions of the brain (like Spearman, 1927), we would classify this as an early example of an all bottom approach.

Section 5.3 takes the idea of an all bottom approach to the molecular origin of sexual differentiation further, while Section 5.4 ventures into the origin of race differences.

5.3. The evolutionary origin and mechanisms of early sex differences

In briefest possible outlines, about a billion years ago a sequence of amino acids or RNA fractured under environmental stress during multicellular evolution and formed a small segment, which allowed a transition to take form from cellular asexual and hermaphroditic modes to the sexual reproductive modes we see in most contemporary animals.

Later, but still long time ago, some of the few functional genes on what later became known as the Y chromosome promoted, in the humanoid fetus, the evolution of testicular tissues capable of synthesizing steroid chemicals, such as androgens, which together with evolving adrenal gland production, morphed originally neutral fetal tissues into masculinized bodily, brain, and behavioral development. This immensely complex male molecular machinery displays several seemingly paradoxical effects. One is that androgens, in particular testosterone (T), also masculinizes after being reduced by enzymes to dihydrotestosterone, but feminize after being aromatized by other enzymes into so-called female estrogens. Steroids like estrogens, in particular estradiol (E₂), is needed for stimulating female fetal and pubertal developments of body, brain, and behavioral traits in the once sex neutral fetus.

This description pays little justice to the functional genomic complexity of primates, as more general biological effects of steroid agents and their physiological machinery depend further on familial gene constitution, dose, timing, receptor distribution and satiation. All these

physiological events are embedded in vastly complex genomic interactions, in which the effects of multiple positive and negative genetic feed-back and feed-forward systems mix — sometimes curvilinearly, sometimes nonlinearly — with specific and more general short- and/or long-term environmental impacts.

Luckily, most of this breathtaking complexity follows decipherable rules, because Nature is a tinkerer (Jacob, 1977). She routinely mixes well-known agents from thoroughly tested combinations during evolution, rather than being a relentless de novo inventor. We accordingly do not have to follow each molecule but can focus on systematically coupled mass-molecular events in known target tissues, as in psycho-neuro-endocrinology, and treat data by sufficiently powerful computers.

Early nascent individual sex hormonal differentiation thus gave various classical Darwinian pressures a certain range of possibilities to select among, which in turn promoted the evolution of still more contrasting sexual body, brain, and behavioral types. The early African Androtype (Nyborg, 1994) responded favorably to further selection for male fighting, aggressive defense of valued territory, and mating, in warm eco-niches. His hunting behavior mediated further specialized evolution of those abilities and technical skills, which favored male survival in primeval Africa. The early female Estrotype (Nyborg, 1994) responded physiologically favorable to further selection for improved gestation, lactation, caring, and gathering behavior. Improved social and verbal fluency skills optimized female and progeny survival in the deep wood or at the Savanna camp site. In the end, these evolving male and female covariant trait combinations exerted synergistic effects, which favored collective survival.

Physiologically, “androgens and estrogens are for two good reasons well suited for coordinating the timetables for male and female body, brain and behavioral development and for making the necessary reproductive adjustment to the opposite sex possible...” ... with the possibility of manifesting ... “important compromise solutions in one and the same individual” (Nyborg, 1994, p. 51). “Steroid processes are the modus vivendi of sexually reproducing species, and with even minor disturbances of the steroid system, extinction is guaranteed.” (Nyborg, 1994, p. 52).

In fact, Darwin’s principle of *Sexual Selection in Relation to Sex* (1871) works only on basis of proper steroid balances, as sex hormones provide the physico-chemical differentiation upon which male selection of preferred females unfolds. In polygamous humans, Androtypic males fight for access to Estrotypic females, and dominant males have a higher probability of getting access, even if reproductive success in part also depends on female strategic preferences.

The following sections provide an ontogenetic model for Androtypic and Estrotypic modulation of co-variant trait development. However, in order to be able to fit race into the full model, we first have to attend to the question of the advent of the race differences in IQ, mapped in Table 1c.

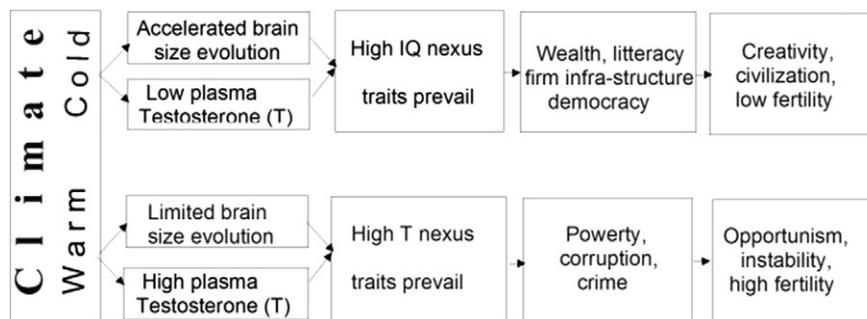


Fig. 12. The IQ/T-Geo-Climatic (GCO) model. Warm climate results in slow brain and IQ evolution, and favors aggression through selection for high plasma testosterone (T), which results in opportunistic, unstable societal organization, with high fertility rates. Colder climates increasingly accelerate brain and IQ evolution, and favor low T and increased altruism, which results in complex societal organization, with low fertility rates. Rudiments of this prolonged ancestral temperature eco-niche selection process along a North–South gradient are, what we today recognize as geo-climatic (previously categorized as race) differences in intelligence, personality, and societal organization.

5.4. The evolutionary origin and mechanisms of early race differences in IQ

When and why did human IQ race differences appear during evolution, and why are they also so impervious to changes? The inverse IQ/T-Geo-Climatic Origin (GCO) model in Fig. 12 provides a preliminary answer.

The model is inspired by Richard Lynn's (1991, 1997, 2006) *Cold Winter* theory. Briefly, selective Darwinian forces in the tropical eco-niches gradually favored DUFF copy number evolution, and its associated brain growth and efficiency in sub-humans as well as in antediluvian African residents. Once prehistoric northbound migrants undertook their northbound Exodus into still colder eco-niches, they became subjected to still harsher selection for Duff numbers, accelerated brain size, and related IQ development. Only those capable of judiciously planning winter supplies, inventing warm shelters, and developing new hunting tactics, could survive in the cold. A potential steroid aspect of migration is that male hunters and later farmers were possibly further selected for low plasma T levels. A high T levels may be favorable for survival in pre-historic Africa, where bellicose territorial defense of place and resources was a matter of life or death, but cold eco-niches allow small bands to survive only if less aggressive low-T male hunters altruistically share warm shelters and the sparse and widely dispersed protein resources with family and friends. Ample aggression and selfish greed would reduce the odds of collective survival in the cold.

In this way the inverse IQ/T-Geo-Climatic Origin, GCO, model in Fig. 12 explains how at least 195,000 years of progressively colder selection inversely favored the evolution of larger brains, higher IQ, and lower T and physical aggression levels. Geo-climatic migration enlarged the initially small physiologically based differences in brain size, IQ and altruistic behavior along the North–South gradient, we can still trace today in rudimentary form as racial, geographical, or national differences in IQ and personality (Nyborg, 2012).

The copy repeat number counts on the DUFF 1220 gene site by the Davis and O'Bleness groups, and by others, provide fascinating information about the molecular side of the rate of brain size and physiological general intelligence evolution. The number count story thus goes far back on the evolutionary scale, from sub-primates with no copies, over Old World monkeys with 35 copies and larger brains, up to Modern World chimpanzees with 125 copies and, finally, up to contemporary humans with 270 + copies, and the relatively speaking largest brains and highest IQs. Within the human end of the scale we see a range of about 8 copy number variations, with higher IQ individuals having more copies, larger brains, and more optimal brain performance than those individuals finding math difficult. Independent confirmation of the workings of the DUFF 1220 – brain size molecular machinery would justify the GCO model prediction that copy count numbers increase linearly with northbound distance migrated away from the Equator, skin color, brain size, IQ and level of societal organization (Nyborg, 2013a).

Ordinary race terminology commonly elicits controversy. It should be noted that the GCO model redefines “race” in geo-climatic terms. Race refers here to a classification of the remote offspring of differently colored ancient interbreeding groups, subsisting for extended periods within eco-niches with fuzzy climatic borders. The geo-climatic evolutionary selective forces can conveniently be defined in terms of the evolutionary–migratory distance covered along the geo-climatic south–north continuum since the ancestral African exodus, even if they have to be adjusted for transient back-and-forth migrations due to major local climatic changes like glaciation periods and large-scale natural disasters (Nyborg, in preparation).

Empirically speaking, skin color correlates .90 + with IQ (Meisenberg, 2004; Templer & Arikawa, 2006), but color obviously does not cause IQ. The two just co-vary. An important indicator is latitudinal distance from the Equator, which correlates .80 with IQ, and $-.89$ with skin color (Nyborg, in preparation). Then again, distance is just another indicator variable, and not the causal agent. The real causal agent is the parameter

variation in the selective challenges offered by the different eco-niches traveled through. This parameter varies in accordance with the average annual temperature. The idea is that physical average world temperatures dictate eco-niche differences, which exert inverse migratory selective effects on DUFF 1220 copy number, brain size, IQ, and T-levels, among other things.

The starting point for the migratory south–north gradient is, in other words, the traditional racial “black” classifications, which refers to the modified physiological constitution of the remote offspring of those early ancestral evolutionary forebears, who successfully survived, slowly evolved, but remained within Africa at the warmest end of the geo-climatic scale. Some selection chanced even within African latitudes, causing higher DUFF copy number counts, larger brains, higher IQs, lower T, and budding ancient cultures in the colder northern areas of Africa, in distinction to Equatorial regions. Afro-American IQ may tell another story, as slaves were brought up north very fast, and then their genotype was gradually mixed with “white genes” (Jensen, 1998). This process will slowly decrease the white–black IQ gap, now amounting to about one standard deviation (e.g. Nyborg & Jensen, 2001). The northernmost end on the geo-climatic south–north gradient refers to the earlier yellow/Asian/Oriental classifications. As the “yellow race” went farthest north, they will, according to GCO model, show the highest average DUFF 1220 copy counts, the largest brains, the highest IQs, and the lowest T – lower than whites and blacks (Rushton, in Nyborg, 2013b). In between these two endpoints on the migratory scale we find more or less brownish or pale physiological constitutions, all the remote offspring of those ancestral African forebears having been graded in proportion to migratory distance traveled from the Equator and the harshness of the cold selection they were subjected to, which in turn favored DUFF copy numbers, brain size, and IQ evolution, and reduced T (Nyborg, 2013a).

In other words, the GCO model in Fig. 12 summarizes progressive evolution as follows: The further north the primordial black African migrants went, the lesser sun, the colder temperature, the lighter skin, the more demanding eco-niche, the higher DUFF 1220 count, the larger brains and higher IQs, the lower T, reduced physical dominance and increased formal dominance, the more altruism, the more “civilized” societal organization – and the lower the fertility. In this way the GCO model accounts for the prehistoric origin of geo-climatic [race] differences in terms of progressive evolution by cold eco-niche selection targeting named physiological parameters.

So far we have outlined when and how geo-climatic sex and “race” differences in physiology, brain size and IQ, T, and personality, conceivably arose. The comprehensive all bottom analysis now calls for a physiologically linking to proximal modeling of essentially similar ontogenetic mechanisms, which could assist in explaining the dynamics of contemporary sex differences in IQ, brain size, personality, and achievement, previously documented.

5.5. The ontogenetics of sexual differentiation of IQ and personality

A model pretending to cover highly complex dynamic physiological ontogenetic relationships is currently best seen as a heuristics tool – even if it springs from more than a hundred years of laboratory and field research within and across psychology, neurology, and endocrinology. However, certain basic observations are confidently established.

We know for sure that sex hormones modify familial gene expression, particularly, but not exclusively, during fetal life and around puberty (Nyborg, 1994). Moreover, animal manipulation and natural human variation studies indicate that prenatal hormones can entirely overrule genotypic sex and turn “female” XX genotypes into phenotypic males, and “male” XY fetuses into phenotypic females. We also know that hormone variations guide many more subtle aspects along the sexually differentiated male and female developmental paths, and affect early and late interests, personality, and intelligence in foreseeable directions. Early and late hormonal variations exert

permanent organizational body and brain effects, but fetal, pubertal, and old age hormones also promote on-off or graded organismic events, depending on dose, timing, and the presence of receptor molecules. We also know that hormones are sensitive to environmental factors (McEwen, 2001).

Hormones mediate the sexual expression of familial propensities and performances in man and other animals. Particularly pertinent in the present context is the observation that T is a major player in coordinating a whole packet of male characteristics (T-nexus traits), and E₂ in coordinating a packet of female E₂-nexus traits. This includes development of secondary sexual characteristics, behavioral and personality traits, and IQ (Nyborg, 2013a).

A model for the dynamic hormonal modulation of pubertal events is of particular interest in the present context, because puberty is the time when significant coordinated sex differences in body, brain size and organization, and general intelligence take place, as just documented. All this coincide with marked pubertal surges in plasma T and E₂ concentrations.

This temporal co-variation suggests that mechanisms of dynamic pubertal hormone modulation relate to solving the ontogenetic “how” problems. We seem at the brink of identifying the particular surges in specific hormones, which modulate familial genes, and prime their mechanisms of co-variant biological expression of bodily and physiological brain-based IQ functions. We may now begin to associate post-pubertal (if not pre-pubertal) Male/Female IQ sex ratios with dynamic physiological events, which in turn would allow us to associate sex differences in high- and low-level lifetime achievements with their molecular gene-neuro-hormonal basis. Add to this that the number of DUFF 1220 copy repeats, relates linearly to brain size, general intelligence and mathematics achievement, and we may begin to see how it is possible to combine all the above phenomena in terms of an all-Bottom molecular analysis of individual and group differences, even if we often have to look for relatively weak global effects.

5.6. The General Trait Covariance (GTC) model

The General Trait Covariance (GTC) model in Fig. 13 suggests ways in which hormones sculpt body, brain, and behavioral development.

The model “generates testable predictions about harmonized body, brain, intellectual, and personality development based on modulation of parental DNA, testosterone/estradiol (T/E₂) balance, and experiences. Optimum brain, intellectual and personality development depends on medium and balanced hormone concentrations, at the cost of sexual differentiation for both sexes, in accordance with a universal economy principle” (Nyborg, 1994, chapter 13). Conversely, maximum sexual differentiation accompanies high and contrasting T and E₂ concentrations, respectively, at the cost of optimum intellectual and personality development in modern societies — the inverse of Hi-IQ/Lo-T relationships.

The “Optimum Range Principle” (Nyborg, 1994, chapter 9), implies that females, whether animal and human, tend on average to “overshoot” the optimal brain value for full expression of highly g-loaded abilities at puberty. This is due to the considerable female rise in E₂, which is needed at the time to guide the molecular trade-off with other vital female body and brain developmental timetables. Some of the proximal neuro-hormonal mechanisms behind these events are described in Nyborg (1994, chapters 3 and 6). The hormonal Optimum Range Principle explains the female post-pubertal decline in IQ and CAT-ASVAB subtest scores seen in Figs. 1 and 3–5.

5.6.1. Migratory applications of the GTC model

The model was recently used in a study of the previously described migratory cold selection effects as a function of climatic variations (Nyborg, 2013a). It generated a series of testable evolutionary prediction about individual, sexual, and racial IQ, and brain and body development. The results of the study supported the notion that prehistoric geoclimatic sex-related IQ differentiation relates to current proximate ontogenetic neuro-hormonal mechanisms at puberty for good ultimate evolutionary reasons.

5.6.2. Menstrual cycles

Another way to illustrate how dynamic E₂ mechanisms monitor the genetic expression of familial abilities is to take advantage of the fact that female E₂ cycles monthly up and down, with low values around the first flow-day of menstruation and high values around the 14th day. These changes in plasma E₂ levels will, according to the GTC model, result in monthly inverse expressions of various female g-loaded lower level

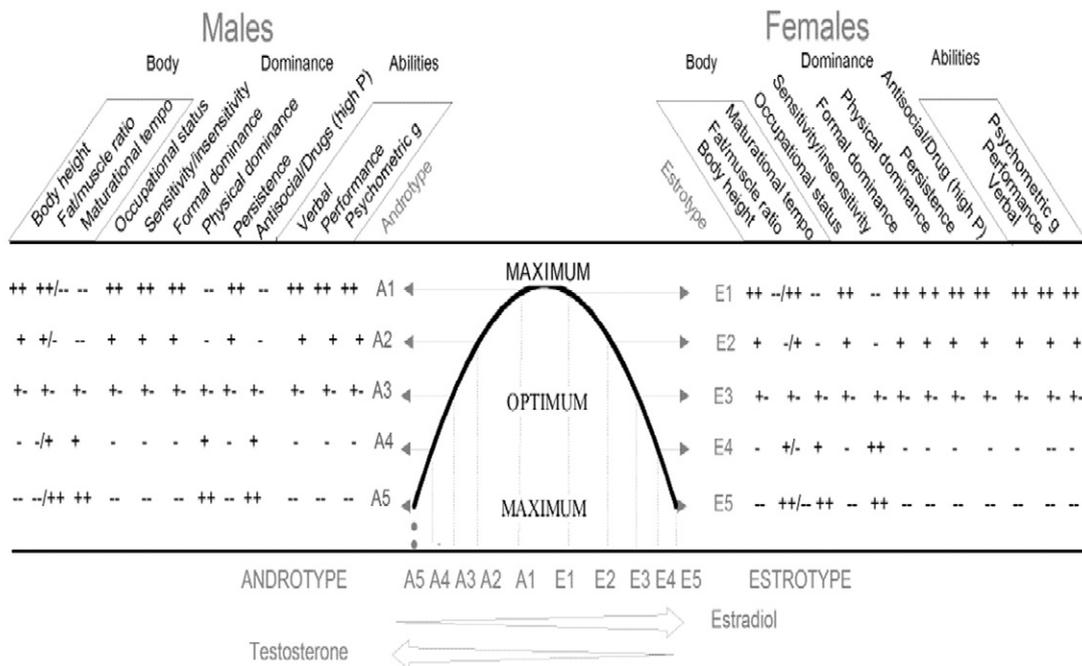


Fig. 13. The General Trait Covariance model (Nyborg, 1994). For explanation, see text.

abilities. Given the Optimum Range Principle, we can hypothesize that most human females “overshoot” the E_2 brain optimum level for “male” abilities mid-cyclically, and less so or perhaps not at all around the first flow-day. To the extent plasma E_2 level relates to g -loaded traits like “male dominated” spatial and “female dominated” verbal abilities, we can expect that the “male” spatial abilities would be temporarily suppressed mid-cyclically in females, but not around their first menstrual flow-day. Conversely, we can expect that “female dominated” verbal and fine-motor skills are enhanced mid-cyclically with high E_2 .

Several studies confirm the overshoot-suppress hypothesis. [Klaiber, Broverman, Vogel, Abraham and Cone \(1971\)](#), [Klaiber, Broverman, Vogel, Abraham and Steen \(1971\)](#) and [Klaiber, Broverman, Vogel and Kobayashi \(1974\)](#) thus monitored spatial ability over three consecutive menstrual cycles and found that it was enhanced in periods when E_2 was low and suppressed when E_2 was high. Similar results are obtained by others ([Anderson, 1972](#); [Dor-Shav, 1976](#); [Hampson, 1986, 1988](#); [Hampson & Kimura, 1987, 1988](#); [Hughes, 1983](#); [Silverman & Phillips, 1991](#)). [Hampson and Kimura \(1988\)](#) controlled for mood changes but this did not alter the observed hormone–ability relationships. [Hampson \(1989\)](#) finally observed that progesterone did not disturb the E_2 effect of subduing the expression of spatial ability, and that individual E_2 values related as expected curvilinearly to spatial ability when measured by space relations subtest, but this generalized neither to Hidden Figures nor to Rod-and-Frame test performances.

5.6.3. Human clinical studies

Clinical substitution therapy studies also illustrate the optimum E_2 brain value principles and the overshoot-suppress principles. Girls and women with Turner’s syndrome lack some X chromosome material and do not produce normal amounts of sex hormones. This calls naturally for supplementary cyclic estrogen/gestagen treatment. It then appears that untreated and long-term treated (i.e. on average 8 years of treatment) score uniformly low on various spatial ability tests and do poorly in mathematics, whereas those receiving about one year of treatment performed on par with their control sisters ([Nielsen, Nyborg, & Dahl, 1971](#); [Nyborg & Nielsen, 1981](#)). The retrospective nature in these analyses and the scant control over treatment schedules prevent firm conclusions based on such studies alone. However, a later pseudo-experimental study of Turner patients examined the effects of six different controlled substitution treatment regimes. Growth hormone exerted little effect on abilities, whether given alone or in combination with other treatments. In contrast, E_2 treatment appeared to accelerate the development of specific abilities so that after one year of treatment they were on par with normal control girls. Two years of treatment with Oxandrolone (OX, and anabolic steroid) even brought Turner girls on par with normal control boys, but the following one year of E_2 treatment seemed to inhibit the passing superior spatial ability in the androgen-treated group (conf. Figure 9.1 in [Nyborg, 1994, p. 98](#)). Again, it is sobering to note that the number of girls in each treatment group was small (between five and eleven), and that the investigators were aware of the particular treatment schedule (but not of the unexpected ability outcome). Instead of referring to data from double-blind prospective studies with normal female controls, these results come from case studies of a relatively rare syndrome.

5.6.4. Animal application

The human hormone–brain–behavior relations prescribed by the model are further supported by rodent data. Pre-pubertal female and male rats display similar levels of g -loaded visuo-spatial abilities, as measured by various maze learning and memory tests. However, at the zenith of considerable hormone increases in plasma hormone levels at puberty male rats stop performing better, and female rats begin to make more errors at the conclusion of the marked pubertal rise in E_2 . This compares to the human pattern seen in Hispanics and black females at puberty ([Figs. 4 and 5](#)). E_2 surges are equally important for the full maturation of female secondary sexual characteristics in man,

and other animals. Moreover, early female castration prevents the female pubertal regression in visuo-spatial abilities, and E_2 treatment makes male rats begin making more errors than they did before treatment (e.g. [Dawson, 1972](#); [Dawson, Cheung, & Lau, 1975](#)). [Williams, Barnett, and Meck \(1990\)](#) later confirmed that controlled early sex hormone exposures selectively affect performance.

5.6.5. Empirical summary

In other words, chemically identical sex hormones exert permanent structural body and brain effects in humans during fetal and pubertal periods as in rodents, in addition to having transient effects, all depending on dosage, developmental timing, and the presence, distribution, and saturation of molecular receptors in specific target tissues. They exert permanent or passing modulation of the phenotypic expression of sex-dimorphic traits, depending on dose and timing, all in accordance with the “Enhance–Suppress” principle ([Nyborg, 1994, chapter 7](#)). This supports the GTC model.

Moreover, when combining animal evidence with the outcome of studies of normal human hormonal cyclicality and the clinical evidence we might – however tentatively – presume, that the natural pubertal female surge in E_2 (or other steroid derivatives) results in a hormonal overshoot phenomenon. As such, it may be partly or totally responsible for the observed decline in ability in female post-pubertal rats, in E_2 -treated male rats, and in Turner patients after prolonged E_2 treatment, as measured by g and the highly g -loaded subtest scores in the CAT-ASVAB test. Young blacks (and perhaps also Hispanics) males have higher plasma hormone levels than whites ([Ellis & Nyborg, 1992](#); [Nyborg, 2013a,b](#); [Ross et al., 1986](#)). By analogy we would expect them to overshoot, on average, more than whites. This might explain the de facto female post-pubertal decline in abilities in these two races, and the early arrest in white female IQ development.

Obviously, without better-controlled dedicated research we can presently only speculate about how to best explain these complex hormonal–brain–IQ–behavior cause–effect relations.

5.7. Concluding remark

Section 5 of this paper marked an aspiration to combine the ultimate IQ/T-Geo-Climatic Origin (GCO) with the proximate General Trait Covariance (GTC), in the attempt to explain the empirical findings reported in the previous parts of the paper in terms of complex gene–hormone–brain–behavior relations, and to relate them to their evolutionary past. This is arguable the most direct way to associate the ancient physiological mechanisms behind prehistoric selective changes in sexual reproductive modes and sexual differentiation proper, with selective changes in evolutionarily speaking much later geo-climatic (previously “racial”) DNA selection in northbound migrants, as a function of their ventures into still colder climates. The association of prehistoric steroid and other selection to current dynamic proximate physiological agents was made with a keen eye on similarity in causal standing of agents and mechanisms, while avoiding committing the category errors illustrated in [Fig. 11](#). The study of contemporary simpler species with more or less complete forms of sexual reproduction can also be illuminating.

By aligning presumed prehistoric with contemporary causal variables in such a comprehensive analysis, we may begin to realize the intricate origin of why and how chemically identical steroid hormones flow everywhere in the body – then and now – but exerted specific biological effects only when receptor molecules – also under selection – were present in target tissues. These steroids modulated the expression of historic DNA material, as they now modulate today’s further selected and mutated DNA in the family line, in both cases to phenotypic expression upon which evolution can work – then and now.

By physiologically relating ancient Darwinian selective effects to the rudimentary form of contemporary differences in body, brain, and behavioral functioning, we may begin to appreciate the amazing historic

continuity of steroid and neuro-hormonal chemistry during Nature's constant tinkering and optimizing. This paves the way for a better causal understanding of today's sex differences and the current geo-climatically caused north-south differences in body, intelligence, personality, achievement, and societal organization – so easy to observe and so difficult to change. The paper thus rounds up suggestions for the rough outlines of a comprehensive theory for the origin and persistence of geo-climatic and sex differences.

The next phase is to thoroughly test the models, fill in the devilish details and cut out the fragile parts. By so doing we may – on the way – come to actualize Spearman's dream: "The ultimate task is to unravel the material basis of physical g, whereby physiology will achieve the greatest of all its triumphs" (Spearman, 1927).

Undoubtedly, an important part of this process consists of carefully modeling interaction effects of genes, hormones, and environmental factors, with brain and body differentiation, and to monitor how all this affect – in multi-way interactions – specific and general life achievement measures in a coherent, consistent, and causally satisfying way, including the possibility of tight experimental control (Nyborg, 1994). The ultimate goal of psychology is to seamlessly integrate all the relevant molecular phylo- and ontogenetic facets with molecular physico-chemical aspects of the environment in a unified all bottom analysis, aspiring to a complete mapping of the purely physico-chemical origin and existence of "Molecular Man in a Molecular World" (Nyborg, 1997, 2007, 2013a). No doubt, many contemporary colleagues will see this program as too farfetched or even downright impossible, but not all do (Eysenck, 1996).

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