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Race/ethnicity and criminal behavior: Neurohormonal influences

Lee Ellis[a,b,*,1]

[a] Department of Anthropology and Sociology, University of Malaya, Kuala Lumpur, Malaysia
[b] Sociology Department at Minot State University, Minot, ND, Unites States

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ABSTRACT

Evolutionary neuroandrogenic (ENA) theory asserts that brain exposed to androgens plus the brain's ability to learn accounts for most of the sex and age variations in criminal behavior. Here, the theory is extended to explain race/ethnic variations in offending. The article documents that among seven different racial/ethnic groups, blacks have the highest and East Asians have the lowest criminal involvement. Strictly social environmental explanations for race/ethnic differences in criminality appear to be inadequate for explaining these differences. Two main elements of ENA theory are offered in the present context: (a) criminal behavior is promoted by exposing the brain to testosterone and other androgens. (b) rapid postpubertal declines in offending depend heavily on learning ability. Ten lines of evidence concerning average racial/ethnic variations in androgen exposure are reviewed, and four lines of evidence of racial/ethnic differences in learning ability are reviewed. With some exceptions and qualifications, currently-available evidence seems to support the idea that racial/ethnic variations in offending could be at least partially explained by ENA theory. Closing comments are offered to suggest that biosocial approaches to the study of racial/ethnic variations in criminal behavior can help to supplement strictly social environmental theories in criminology.

For over a century, scholars have struggled to explain racial and ethnic differences in criminal behavior (reviewed by Gabbidon, 2015; McNulty & Bellair, 2003). In just the past five years, eight scholarly books have been published dedicated to this topic (Barak, Leighton, & Cotton, 2014; Delgado & Stefancic, 2012; Gabbidon, 2015; Glynn, 2013; Kalunta-Crumpton, 2012; Rowe, 2012; Unnever & Gabbidon, 2011; Walker, Spohn, & DeLone, 2011). And, in a fairly recent presidential address to the American Society of Criminology, Peterson (2012, p. 303) called for "placing race and ethnicity at the center of the study of crime and justice".

As will be shown, nearly all past and contemporary explanations for racial/ethnic variations in criminal behavior consider only social environmental variables as relevant. The present article proposes that the inclusion of neurohormonal variables along with learning ability and sociocultural variables can provide a more complete explanation for the race/ethnic differences in offending that have been documented.

There are five parts to this article. Part I capsulates the nature of race differences in offending. Part II briefly summarizes social environmental explanations for these race/ethnic differences. In Part III, a biosocial theory is presented that could assist in understanding these differences. Part IV reviews a wide array of empirical evidence bearing on possible race/ethnic differences in exposure to testosterone and other androgens as well as race/ethnic differences in learning ability. Part IV is further condensed in Part V in order to match theoretical predictions with the available evidence.

1. Part I

1.1. Summarizing the evidence regarding racial differences in criminality

Part I will include a description of six tables, each one summarizing conclusions derived from studies of crime rates for six different racial/ethnic groups relative to the crime rates for whites in the same country. The reason for making comparisons to whites is that most studies published on race differences in crime have been conducted in white-majority countries, principally the United States, but also Britain, Canada, Australia, and New Zealand.

Each table provides a "numeric snapshot" of the relevant studies according to the types of crimes sampled and whether the data were based on official crime statistics or self-reports. To conserve space, the citations themselves are not presented; instead, see the *Handbook of crime correlates* (Ellis, Beaver, & Wright, 2009, pp. 20–29).

* Corresponding author at: Department of Anthropology and Sociology, University of Malaya, Kuala Lumpur, Malaysia
E-mail address: lee.ellis@hotmail.com.
[1] Semi-retired.

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Table 1
Findings from studies comparing criminality by blacks and by whites. (The numbers inside each cell represent the number of studies located.)

Crime involvement relative to whites	Type and seriousness of offenses							
	Official statistics (e.g., arrest, prosecution, conviction, imprisonment data)						Self-reported data	
	Violent offenses	Property offenses	Drug offenses	Delinquency	General & unspecified offenses	Recidivism	Victimizing & overall offending	Illegal drug & use
Blacks higher/whites lower	96	2	4	75	12	17	12	4
No significant difference	0	0	0	0	0	6	20	8
Blacks lower/whites higher	0	0	0	0	0	0	4	33

1.1.1. Black-white comparisons

The first table pertains to differences in crime rates between blacks and whites. Most of the studies were conducted in the United States. As summarized in Table 1, official data are very consistent in indicating that blacks engage in crime at higher rates than do whites, especially regarding crimes of a violent nature (murder, assault, rape, and robbery). To illustrate, in a recent book on black-white differences in crime, Unnever and Gabbidon (2011, p. xv) stated the following: "African American men – about 6 percent of the population of the United States – account for nearly 60 percent of the robbery arrests in the U.S." In the case of U.S. murders, black rates are about 5.5 times higher than those for whites (Ulmer, Harris, & Steffensmeier, 2012). Many years earlier, Wolfgang and Ferracuti (1967)/2013, p. 152) observed that in Philadelphia "homicide rates for young black males was 27 times higher than for young white males, and the female rate was 23 time greater than for white females."

In addition to the official data, Table 1 also presents a summary of the self-reported data on black-white differences. Regarding the official data, there is no disagreement that black rates are higher. However, one can see that most self-report studies have either found no significant black-white differences or that whites actually have greater involvement than blacks, particularly regarding illegal drug offenses. These inconsistencies need attention before moving on to comparisons involving other racial groups.

Some have interpreted the black-white discrepancies between official statistics and self-reported data as evidence of police biases in law enforcement activities (Turk, 1969; Quinney, 1970; Schur, 1973). However, there are at least three reasons for believing that the official data are closer to reality than the self-report data, at least regarding the most serious offenses.

First, most self-reported offenses pertain to relatively trivial crimes (misdemeanors) such as smoking pot or minor vandalism, whereas most law enforcement statistics have to do with serious crimes (felonies) such as committing assaults, robberies, and major thefts (Eaton & Polk, 1961; Pope, 1979; Schuster, 1981). A few self-report studies have attempted to eliminate the trivial offenses before making black-white comparisons. They have all concluded that blacks actually do self-report higher offending rates than whites, although the differences are still not nearly as great as the differences reflected in official data (Elliott, Huizinga, & Morse, 1986; Hill & Crawford, 1990; Peeples & Loeber, 1994).

Second, one study was able to cross-check offenses that had been self-reported as resulting in an arrest against police records of an arrest for the same individuals. It concluded that white males reported 90% of the offenses on their police records, while black males reported only 67% (Hindelang, Hirschi, & Weis, 1981, p. 177). A few additional studies have also indicated that blacks are more likely than whites to provide "normatively acceptable responses" to questions about involvement in crime, thereby under-reporting to a greater degree (Huizinga & Elliott, 1984; Mensch & Kandel, 1988; Aquilino & Sciuto, 1990).

Third, several U.S. studies have used data from crime victimization surveys as a way of cross-checking the accuracy of official data for violent crimes such as assaults, robberies, and forcible rapes. These studies have revealed that crime victims report their attackers to have been black at much higher rates than they report them being white. This was true for both white and black victims (Blumstein & Cohen, 1987; Hindelang, 1981; Pope, 1979; Wilbanks, 1985; Wolfner & Gelles, 1993).

Finally, it is also relevant to note that nearly all self-report studies of offending (especially those involving illegal drug use) are derived from samples of either high school seniors or of college students. Both of these sampling sources substantially under-estimate black-white differences in crime rates due to the fact that blacks have much higher high school dropout rates than whites (Carpenter & Ramirez, 2007; Rumberger, Ghatak, Poulos, Ritter, & Dornbusch, 1990). Offending rates among all dropouts are considerably higher than the rates for those who complete high school, and especially those who go on to college (Swaim, Beauvais, Chavez, & Oetting, 1997; Townsend, Flisher, & King, 2007). Therefore, when sampling high school and especially college students, one excludes blacks who tend to offend at the highest rates, thereby under-estimating black involvement in self-reported crime relative white involvement.

Overall, the studies that are numerically summarized in Table 1 in conjunction with the qualifications just provided suggest that offending rates are considerably higher for blacks than for whites. This is especially so for serious violent crimes.

1.1.1.1. Hispanic-Anglo Comparisons. Hispanics (Latinos/Latinas) are people whose ancestors are from Spanish/Portuguese-speaking South and Central American countries. Most Hispanics are considered white, but they are distinguished from European whites – sometimes called *Anglos* – by the fact that their skin and hair are usually darker and by their typically having Spanish family names. Also, most Hispanics can trace their ancestry back to Mexico, the Caribbean Islands, or South or Central America rather than to Europe.

Nearly all studies that have compared crime rates of Hispanics with non-Hispanic whites have been conducted in the United States. Table 2 summarizes the research findings from these studies. It shows that official crime statistics agree that Hispanic crime rates are higher than those of non-Hispanic whites. These differences are especially well documented for homicide, with Hispanic rates being roughly three times higher than those for non-Hispanic whites although they are only about half the homicide rates for U.S. blacks (Centerwall, 1984; Martinez, 1996; Pokorny, 1965; Polednak, 1989; Ulmer et al., 2012).

Turning to self-reports, Table 2 shows that most studies have indicated that no significant differences exist between offending rates of Hispanics and non-Hispanic whites, and in a few cases, Hispanic respondents have reported significantly lower rates, especially regarding marijuana use (Flannery, Vazsonyi, & Rowe, 1996) and under-age drinking (Robins, Tipp, & Przybeck, 1991). However, as noted above regarding blacks, Hispanics have considerably higher high school

Table 2
Findings from studies comparing criminality by Hispanics and by non-Hispanic whites. (The numbers inside each cell represent the number of studies located.)

Crime involvement relative to whites	Type and seriousness of offenses							
	Official statistics (e.g., arrest, prosecution, conviction, imprisonment data)						Self-reported data	
	Violent offenses	Property offenses	Drug offenses	Delinquency	General & unspecified offenses	Recidivism	Victimizing & overall offending	Illegal drug & use
Hispanics higher/whites lower	18	0	2	8	4	1	4	9
No significant difference	0	0	0	0	0	0	3	9
Hispanics lower/whites higher	0	0	0	0	0	0	1	5

dropout rates than whites (Carpenter & Ramirez, 2007; Neumann, 1996) and delinquency and drug use among Hispanic dropouts is substantially higher than for their cohorts who complete high school (Henry, Knight, & Thornberry, 2012). As a result, studies limited to high school seniors and/or to college students will substantially under-estimate criminality by Hispanics compared to non-Hispanic whites.

Also, in the case of "harder drugs," such as heroin and cocaine, Hispanics self-report using these particular drugs considerably more than do non-Hispanic whites (Johnston, O'Malley, & Bachman, 1993; Mieczkowski, 1996). This self-reported use of "hard drugs" has been supported by data collected in hospital emergency rooms where Hispanic visitation rates for overdosing on heroin and cocaine far exceeded non-Hispanic white visitation rates (Mieczkowski, 1996).

Overall, studies that have compared rates of offending by Hispanics and non-Hispanic whites (Anglos) using official statistics have consistently concluded that Hispanic rates are more involved even though self-reported data have not found consistent differences. As with the research comparing blacks and whites, much of the seeming inconsistencies between the official statistics and the self-reported data appears due to the failure of nearly all self-report studies to sample persons who dropped out of high school and to the fact that self-reports focus on relatively trivial offenses while official data deal mainly with serious and especially with violent offenses.

1.1.1.2. Native American-white comparisons. Crime rates among Native (Aboriginal) Americans have been studied extensively in the United States and Canada. As summarized in Table 3, nearly all of these studies have found official crime rates for Native Americans to be higher than those for whites. The differences have been especially pronounced for offenses committed by persons under the influence of alcohol (Graves, 1967; Jensen, Stauss, & Harris, 1977; Nettler, 1984; Pope & McNeely, 1981; Roberts & Doob, 1997). In general, violent crime rates involving Native Americans are similar to those of Hispanics (Walker et al., 2011). The only category of offending that may be lower among Native Americans than among whites is property crime (McCone, 1966). Regarding studies based on self-reports, Table 3 shows that even these studies suggest that Native Americans are more involved in illegal drug use than are whites (G. F. Jensen et al., 1977; Wallace & Bachman, 1991).

1.1.1.3. Pacific Islander-white comparisons. Pacific Islanders are persons native to Australia, New Zealand, and Hawaii and other Pacific Islands. For statistical comparisons between whites and Pacific Islanders, most studies have been conducted in Australia and New Zealand. As one can see from viewing Table 4, both official statistics and self-report data are in agreement that Pacific Islanders engage in crime at higher rates than whites.

1.1.1.4. South Asian-white comparisons. South Asians are primarily descendants of countries such as India, Pakistan, Bangladesh, and Malaysia (C. M. Hall & Page, 2012). Because all of these countries cover land that was former colonialized by Britain, quite a number of their citizens immigrated to Britain before their countries gained independence. Today, South Asians comprise about 3% of England's population (D. J. Smith, 1997). Thus, most of the studies of crime rate differences between South Asians and whites have been conducted in England. Table 5 shows a mixed picture regarding any differences in official crime rates between South Asians and whites living in the British Isles. However, self-report studies have consistently indicated that South Asians have higher offending rates than do whites.

1.1.1.5. East Asian-white comparisons. The final category for comparisons involves East Asians. These are persons whose ancestors were from three countries: China, Japan, and Korea. The majority of studies that have compared crime rates by East Asians with whites were conducted in the United States. Table 6 shows that all of the studies based on official data have concluded that whites engage in more crime than do East Asians. Most of the self-report studies have reached the same conclusion, i.e., whites are more involved in crime, especially drug offenses, than East Asians (Jang, 2002). The few exceptions simply concluded that there were no significant differences between the two groups.

Table 3
Findings from studies comparing criminality by Native Americans and by whites. (The numbers inside each cell represent the number of studies located.)

Crime involvement relative to whites	Type and seriousness of offenses							
	Official statistics (e.g., arrest, prosecution, conviction, imprisonment data)						Self-reported data	
	Violent offenses	Property offenses	Drug offenses	Delinquency	General & unspecified offenses	Recidivism	Victimizing & overall offending	Illegal drug & use
Native Americans higher/whites lower	18	0	1	3	0	3	1	28
No significant difference	0	0	0	0	0	0	1	0
Native Americans lower/whites higher	0	1	0	0	0	0	0	0

Table 4
Findings from studies comparing various forms of criminality by Pacific Islanders and by whites. (The numbers inside each cell represent the number of studies cited.)

Crime involvement relative to whites	Type and seriousness of offenses							
	Official statistics (e.g., arrest, prosecution, conviction, imprisonment data)						Self-reported data	
	Violent offenses	Property offenses	Drug offenses	Delinquency	General & unspecified offenses	Recidivism	Victimizing & overall offending	Illegal drug & use
Pacific Islanders higher/ whites lower	2	0	0	4	5	2	2	3
No significant difference	0	0	0	0	0	0	0	0
Pacific Islanders lower/ whites higher	0	0	0	0	0	0	0	0

Overall, the evidence summarized in Tables 1 through 6 indicate that criminal behavior is more common among blacks than among whites, among Hispanics than non-Hispanic whites, among Native Americans than whites, and among Pacific Islanders than whites. When crime rates for South Asians and whites have been compared, the official data is very inconsistent regarding any significant differences, although self-reports point toward higher rates among South Asians. Finally, in the case of East Asians, their rates appear to be substantially lower than those for whites, especially regarding official data. Thus, the ordering of crime involvement for these seven racial/ethnic groups is as follows:

$$\text{Blacks} > \text{Hispanics} \approx \text{Native Americans} \approx \text{Pacific Islanders} > \text{Whites}_{(NH)} \approx \text{South Asians} > \text{East Asians}.$$

2. Part II

2.1. Environmental explanation for race differences in offending

Given that criminal behavior is more prevalent in some racial/ethnic groups than in others, this section will briefly review past explanations for these differences (also see McNulty & Bellair, 2003). The proposals are divided into two categories: (a) two having to do with societal or neighborhood variables (such as poverty, ethnic heterogeneity, and racial discrimination) and (b) two focusing on subcultural factors such as traditions and practices that seem to be fairly unique to a given racial/ethnic groups.

2.1.1. Systemic explanations for racial differences in crime

2.1.1.1. Economic racial disparities. Racial differences in income and wealth have been widely documented (Blank, 2001; DesJardins, Ahlburg, & McCall, 2006; Oliver & Shapiro, 2006; Smith, 1995), and several criminologists have proposed that these inequalities are central to racial differences in criminality, especially black-white differences (Pope & McNeely, 1981; Potter, 1991; Rose & McClain, 1990). One article went so far as to suggest that violent crime is the "apparent price of racial and economic inequality in U.S. society" (Blau & Blau, 1982, p. 126).

Studies undertaken to test such a proposal, usually by statistically controlling for income differences between blacks and whites, have been able to account for some of the black-white differences in murder rates with economic factors, but far from all of it (Huff-Corzine, Corzine, & Moore, 1986; Messner & Golden, 1992; Shihadeh & Steffensmeier, 1994; Williams, 1984). When efforts are made to explain why black violent crime rates are at least twice as high as those for other economically disadvantaged groups such as Hispanics and Native Americans, this economically-based hypothesis has garnered little support (Blank, 2001).

Research involving Asians also has failed to support an economic explanation for race differences in criminality. For example, East and South Asians residing in the U.S. and in Canada differ only slightly from the average incomes of whites, depending on the years and geographic regions sampled (Blank, 2001; Sharpe & Abdel-Ghany, 2006; White, Fong, & Cai, 2003; Zeng & Xie, 2004). However, as documented in Part I, the crime rates of South Asians are roughly equal to those for whites, and the rates for East Asians are substantially lower. Overall, economic disparities do not appear to be responsible for most of the racial variations in criminality.

2.1.1.2. Racism and societal discrimination and subordination. Many social scientists have argued that race differences in criminal behavior result from racist attitudes and discriminatory practices by a society's racial majority against its racial minorities (e.g., Dollard, 1937; McNeely & Pope, 1981; Stets & Straus, 1990). This view is prominently featured in the writing of Unnever and Gabbidon (2011). After describing the differences in violent crime between blacks and whites, they state "It is obvious to us that this incredible racial disparity in offending must be related to the past and present racial subordination of African Americans" (p. xv).

According to such reasoning, racial subordination prevents minority races from reaching their educational, social, and financial potential. This in turn creates the social and psychological conditions that promote minority resentment and subsequent involvement in criminal activities. In other words, it is not simply racial inequities in income and wealth that influences racial differences in crime, but emotional

Table 5
Findings from studies comparing various forms of criminality by South Asians and whites. (The numbers inside each cell represent the number of studies cited.)

Crime involvement relative to whites	Type and seriousness of offenses							
	Official statistics (e.g., arrest, prosecution, conviction, imprisonment data)						Self-reported data	
	Violent offenses	Property offenses	Drug offenses	Delinquency	General & unspecified offenses	Recidivism	Victimizing & overall offending	Illegal drug & use
South Asians higher/ whites lower	0	0	0	3	1	0	2	2
No significant difference	0	0	0	1	4	0	0	0
South Asians lower/ whites higher	0	0	0	2	1	0	0	0

Table 6
Findings from studies comparing various forms of criminality by East Asians and by whites. (The numbers inside each cell represent the number of studies cited.)

Crime involvement relative to whites	Type and seriousness of offenses							
	Official statistics (e.g., arrest, prosecution, conviction, imprisonment data)						Self-reported data	
	Violent offenses	Property offenses	Drug offenses	Delinquency	General & unspecified offenses	Recidivism	Victimizing & overall offending	Illegal drug & use
East Asians higher/ whites lower	0	0	0	0	0	0	0	0
No significant difference	0	0	0	0	0	0	2	1
East Asians lower/ whites higher	6	0	0	10	11	0	0	6

responses to those social inequities. Proponents of this perspective envision American society as fostering racial subordination especially of blacks and Hispanics, thereby helping to explain the high crime rates in these groups (Armour, 1997; Staples, 1975).

There are at least four reasons for questioning this type of explanation for the high crime rates by blacks and Hispanics. One comes from noting that in the U.S. many governmental efforts have been undertaken since the 1960s to alleviate poverty, provide more equal educational opportunities, and to outlaw racial discrimination in housing and other accommodations (Lee, 2002). Despite these efforts, the proportional involvement of blacks and Hispanics in crime does not appear to have substantially dissipated relative to crime rates among whites over the past half-century (Blank, 2001; Graglia, 1999; Steffensmeier, Feldmeyer, Harris, & Ulmer, 2011).

Second, Jews were subjected to discrimination in many countries throughout history and yet their crime rates have been found to be unusually *low* (reviewed by Ellis et al., 2009, p. 115). Even in Nazi Germany when governmental policies were strongly anti-Semitic, the crime rates for Jews (derived from *official government data*) were actually lower than crime rates for the German population as a whole (Bonger, 1943; Exner, 1939).

Third, even though many native Britons resent foreigners from former British colonies immigrating to Britain (Daniel, 1968; Furnham & Gunter, 2013; Mayhew, Elliott, & Dowds, 1989), it is only among black immigrants that distinctly higher rates of crime have been documented (Home Office, 1989; Hood, 1992). Rates among South Asian immigrants are roughly the same as those for whites in England (Home Office, 1989; Smith, 1997).

Fourth, in South Africa, blacks constitute the majority of the country's population and since the end of apartheid in 1994, blacks have electorally controlled the government. Nevertheless, black crime rates especially for violent offenses have remained much higher than for the white minority (Duflou, Lamont, & Knobel, 1988), even after the end of apartheid rule (Thomson, 2004).

All in all, racial discrimination and subordination by a majority population against minority groups in a society do not appear to explain much if any of the racial and ethnic variations in crime that have been documented. Attention will now turn to subcultural explanations.

2.1.2. Subcultural explanations for race differences in crime

Two distinguishable subcultural explanations have been offered for why crime rates vary by race. One is the "subculture of violence" hypothesis. The other emphasizes that psychological stress may impact some racial groups in ways that elevate their probability of engaging in crime. Each of these two possibilities are considered below.

2.1.2.1. Subculture of violence. According to the subculture of violence thesis, "the unique historical experiences of blacks have led them to adopt a set of values conducive to violence" (Sampson, 1985, p. 47). Such an idea was first put forth in the 1960s by Wolfgang and Ferracuti (1967). They argued that people living in southern states of the U.S., especially blacks, reside in subcultural neighborhoods where violence has come to be an accepted way of dealing with interpersonal conflicts.

Several proponents of the subculture of violence thesis have extended it to assert that the brutality of nearly two centuries of slavery created a black subculture in which hostility and anger are expressed through violence (Messner, Baller, & Zevenbergen, 2005; Patterson, 1998; Wilson, 1987). Supporting this proposal, studies have found feelings of hostility and anger to be more prevalent among blacks than among whites (Durel et al., 1989; Scherwitz & Rugilies, 1992). Also, blacks have been found to justify the use of violence as a way to solve interpersonal disputes more than whites do (McNeely & Pope, 1981). Overall, various researchers have concluded that the subculture of violence thesis along with poverty and segregation have helped to perpetuate unusually high rates of violent crime among U.S. blacks (Curtis, 1975; Huff-Corzine et al., 1986; Oliver, 1989). At least one research team declared that the subculture of violence thesis "remains the definitive argument for society's role in creating violent criminal behavior" (Cao, Adams, & Jensen, 1997, p. 367).

This subculture of violence explanation, especially one based on a history of slavery, has weaknesses. First, it has only been used to explain the high involvement of blacks in violent crime. This leaves high black involvement in property offenses unexplained. Second, blacks do not only exhibit higher rates of violent crime in southern parts of the U.S. where slavery predominated. Their violent crime rates are also found in the remainder of the U.S. (Lane, 1979), including Hawaii (Blanchard & Blanchard, 1983). Unusually high violent crime among blacks is also found in England (Home Office, 1989; Hood, 1992), Canada (Gartner, 1995), and Israel (Landau & Drapkin, 1968).

2.1.2.2. General stress/strain explanation. A general strain theory (GST) was proposed by Agnew (1985, 1992, 2001) to help explain race differences in criminal behavior (also see Bernard, 1990). Agnew argued that all forms of strain (or stressors) increase the chances of crime by causing negative emotions (especially anger) for those who experience the stressful events. In an expanded reformulation of GST, Agnew (2006, p. 116) offered a list of 12 different types of stressful experiences that could enhance the probability of criminal behavior. These included parental rejection, family conflict and instability, racial discrimination, homelessness and poverty, being chronically unemployed, receiving poor grades in school, and "experiences with prejudice and discrimination based on characteristics like race/ethnicity and gender".

It is true that all of the above-mentioned stressful experiences appear to be associated with criminal behavior (see review by Ellis et al., 2009). Also, these stressful experiences have been found to be more prevalent among racial and ethnic groups with the highest crime rates (Agnew, 2001; Kaufman, Rebellon, Thaxton, & Agnew, 2008; Toomey & Christie, 1990). However, one finds sex differences in rates of poverty and job discrimination against women, and yet males far surpass females in crime (Ellis et al., 2009, pp. 11–19).

Regarding racial differences in negative feelings such as anger

(which GST asserts is an important mediator of the connection between stress and crime), there is little evidence that blacks are more prone toward such feelings than whites. For example, no significant black-white differences in feelings of anger were reported in one study (Mabry & Kiecolt, 2005). And in a study of widows and widowers, blacks were significantly *less* likely to report feeling anger following the death of a spouse than were whites (Carr, 2004). Thus, even though by *objective* standards, blacks suffer greater stress than do whites, fragmentary evidence casts doubt on Agnew's (2006) contention that blacks *feel* greater "anger" than do whites.

As another version of the general stress theory, a few researchers have suggested that after years of racist treatment, many adolescent blacks (especially males) develop a high degree of self-hatred which they express in the form of violence toward others, especially other blacks (Hammond & Yung, 1993, p. 145; Poussaint, 1972). However, this explanation is inconsistent with numerous studies showing that black adolescents usually report having significantly more favorable self-images than do white adolescents (Siegel, Yancey, Aneshensel, & Schuler, 1999). Self-esteem ratings are also higher for blacks than for whites (reviewed by Twenge & Crocker, 2002). Furthermore, East Asians, the racial group with the lowest crime rates, have been shown to have lower self-esteem ratings than whites and certainly than blacks (reviewed by Gray-Little & Hafdahl, 2000; Tashakkori, 1993; Twenge & Crocker, 2002).

2.1.3. Overall assessment of sociocultural explanations

A more in-depth exploration of the various sociocultural explanations for racial variations in criminality has been provided by McNulty and Bellair (2003). Nevertheless, the point being made here is that these proposals all seem to leave much of the variance in racial variations in offending unexplained. In agreement with Wright (2009), I am inclined to believe that sociocultural explanations for most of the racial and ethnic variations in criminal behavior are very inadequate. In particular, why blacks are at least twice as likely to commit murder and serious assaults as are Hispanics, Native Americans, and Pacific Islanders, who are in turn about twice as likely to do so as whites, and why whites and South Asians are roughly equal in their involvement, while all being substantially more involved in violent crime than East Asians remains unresolved by simple social environmental explanations. While social environmental factors are almost certainly relevant, none of them appear to be adequate for explaining most of the race and ethnic differences in offending.

3. Part III

3.1. A biosocial theory of criminal behavior

Two variables – sex and age – are even more associated with criminal behavior than race/ethnicity. Specifically, the vast majority of crimes are committed by males in their second and third decades of life. Nearly 15 years ago, I proposed a theory to account for sex and age variations in such behavior. The theory – called the *evolutionary neuroandrogenic* (*ENA*) *theory* – asserts that differential brain exposure to testosterone is a major determinant of offending behavior (Ellis, 2003, 2005). While the theory is rather simple, it has complex elements in several details (L. Ellis & A.W. Hoskin, 2015). As the theory's name implies, it has two fundamental components, an evolutionary component and a neurohormonal component. Both are briefly described below.

3.1.1. The evolutionary component

ENA theory assumes that, unlike males, females have evolved tendencies to bias their mate choices toward mates who are competent at resource provisioning. In other words, such a female tendency has been naturally selected. Females who primarily use any other criteria in mate selection will be likely to leave fewer offspring in subsequent generations.

The evidence for the existence of such a female bias in all known human societies is substantial. Studies throughout the world have shown that criteria such as "being able to make a living" and "being a good provider" are more important for females when choosing a mate than for males when choosing a mate (Bereczkei, Voros, Gal, & Bernath, 1997; Buss, Shackelford, Kirkpatrick, & Larsen, 2001; Greenlees & McGrew, 1994; Jennions & Petrie, 1997; Moore, Cassidy, Smith, & Perrett, 2006; Waynforth & Dunbar, 1995).

According to ENA theory, this female bias has emanated from the fact that females must make a much heavier investment of time and energy in order to produce offspring than males must make. Therefore, females have been naturally selected to "even the investment ledger" (so to speak) at least a little by seeking mates who are competent (and loyal) resource provisioners. Stated another way, females who bias their mate choices in any other way will leave fewer offspring in subsequent generations than females who prefer mates who appear to be competent and loyal resource provisioners (Cotton, Small, & Pomiankowski, 2006; Edward & Chapman, 2011; Ellis, 2001; Geary, Vigil, & Byrd-Crave, 2004).

Such a female bias has effects upon which males are most likely to leave their genes in subsequent generations at the highest rates. In particular, the bias exerts evolutionary pressure on males to focus time and energy on obtaining resources. A frequent consequence of male efforts to obtain resources is that they end up competing with others, particularly other males, who are doing essentially the same thing.

At least historically, male-male competition over resources is likely to have favored increases in male physical strength and in their abilities to navigate terrain and return home after extensive hunting. In today's context, more complex learning skills may be involved in acquiring resources that are needed to attract and keep mates.

As competition in societies has become increasingly complex and diversified, the ability to learn effective resource acquiring skills has become more challenging, so much so that many males have been favored for flaunting and even exaggerating whatever skills they happen to have. This in turn provides an explanation for why psychopathy is predominantly a male trait (Cale & Lilienfeld, 2002; Yildirim & Derksen, 2012). Once male psychopathic traits evolved, females who were able to discriminate between psychopathic males and males who are genuinely loyal and good at resource acquisitioning would have been favored by natural selection (Ellis, 2005). The end result is that a complex "arms-race" of learned social skills have evolved in both sexes that often entail deception and deception-detection strategies.

Especially in modern societies, males must often learn how to compete without overly irritating those with whom they are competing. If they fail to learn "acceptable" competitive skills, their "opponents" perceive themselves as having been victimized and often seek to retaliate (or they invoke the criminal justice system to retaliate on their behalf). For this reason, young males will often run afoul of the law as they begin their competitive/victimizing activities (L. Ellis & A.W. Hoskin, 2015). Young males with relatively keen abilities to learn and who receive opportunities to practice "acceptable" (i.e., non-criminal) ways of competing for resources and mating opportunities will transition fairly quickly from so-called "crude" competitive/victimizing behavior to "sophisticated" forms of the behavior. Without this learning ability, the transition may take decades to acquire (Boutwell, Barnes, Deaton, & Beaver, 2013).

3.1.2. The neurohormonal component

The evolutionary component of ENA theory is focused on explaining *why* males commit most crimes and *why* most males (especially those with keen learning abilities) quickly transition from "crude" to "sophisticated" forms of competition for resources. As will now be described, the theory's second component seeks to explain *how* competitive/victimizing behavior is promoted and why it gradually moderates with age. In both cases, the evolved human brain plays a central role.

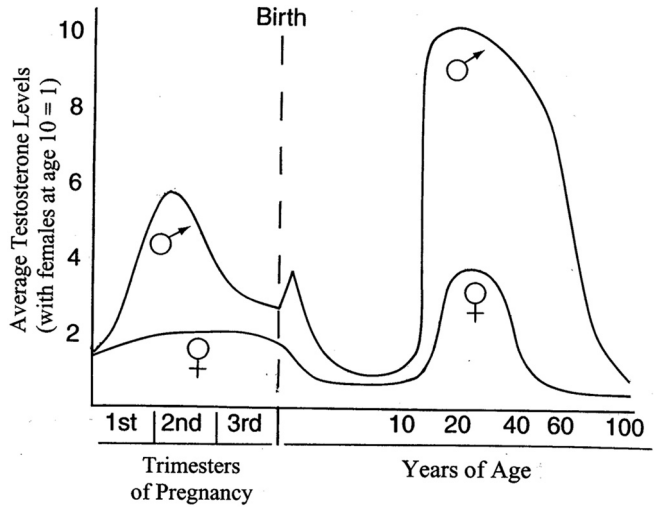

Fig. 1. Testosterone levels of human males and females from conception through old age.

3.1.2.1. Testosterone. To make males more prone toward competitive/victimizing behavior than females, ENA theory stipulates that the primary driver is brain exposure to androgens (so-called "male sex hormones"). *Testosterone* is an especially important androgen in this regard. It is produced primarily by the male testes, although female ovaries and the adrenals of both sexes also produce small amounts.

Recognizing that androgens are produced primarily during two phases of human development is important for understanding how they affect behavior. The first of these two phases is known as the *prenatal* (or *organizational*) *phase*. It occurs before or very shortly following birth. The second phase is called the *postpubertal* (or *activational*) *phase*, and it begins at puberty and lasts to some extent to the end of life.

Fig. 1 illustrates how the levels of testosterone vary on average over the entire human lifetime depending on whether one is a male or a female. Notice that not only are the levels of this hormone higher in males but that the gender differences are greatest prior to and shortly after birth (i.e., prenatally) and then again following the onset of puberty (postpubertally).

High (male-typical) levels of testosterone masculinize bone structures (i.e., lengthening and thickening the bones) (Kenny, Prestwood, Gruman, Marcello, & Raisz, 2001; Leonard et al., 2010) and enlarge and strengthen muscle fibers, especially in the upper torso (Auyeung et al., 2011; Joubert, Tobin, & Lebart, 1994; Morley et al., 1997; Urban, Bodenburg, & Gilkison, 1995). Most importantly, high levels of testosterone masculinize/defeminize an otherwise "default" female brain, thereby producing innumerable changes in cognition and behavior (Marin & Baker, 1998; Vainio, Heikkilä, Kispert, Chin, & McMahon, 1999). In other words, male mammals, including humans, are fundamentally an enlarged and strengthened variant on the female sex (Brennan & Capel, 2004).

In order to produce male mammals, one X chromosome is replaced with a smaller Y chromosome. This latter chromosome contains genes that cause the gonads to be diverted away from their "normal" feminine form (i.e., ovaries) and to take on a masculine form (i.e., testes) instead (Harley, Clarkson, & Argentaro, 2003; Page, Fisher, McGillivray, & Brown, 1990). Once the testes have formed in males, they begin to produce high levels of testosterone and other androgens which cause the muscle, bone, and brain tissue to masculinize/defeminize (Arnold et al., 2004; Rinn & Snyder, 2005).

The above paragraphs come with at least two important qualifications. One is that testosterone exposure needs to be relatively high (i.e., in the male-typical range) prenatally *and* postpubertally in order to fully androgenize bone, muscle, and neuronal tissue (Arnold, 2009; Morris, Jordan, & Breedlove, 2004). However, especially regarding androgenizing the brain, prenatal testosterone levels appear to be much more important than postpubertal testosterone (Berenbaum, 1999; Hines, Brook, & Conway, 2004). In essence, prenatal testosterone determines the extent to which each human is born with a masculine, a feminine brain, or something in between. While some additional androgenizing effects may occur during childhood and adolescence (Sisk & Foster, 2004) and some non-hormonally mediated genetic effects on sexual differentiation also occur (Arnold, 2004; Davies & Wilkinson, 2006), most of the testosterone our brains are exposed to following birth seems to merely activate the brain to fulfill its prenatally programmed masculine/defeminine "destiny".

The second qualification is that androgens appear to interact with testosterone. Particularly important in this regard is the stress hormone, cortisol (Mehta & Prasad, 2015; Montoya, Terburg, Bos, & Van Honk, 2012). Basically, when individuals are challenged or under stress, those with high exposure to testosterone and diminished levels of cortisol have an elevated risk of responding in risky and violent ways. More information along these lines will be presented later.

3.1.2.2. Learning ability. ENA theory asserts that the brain's capacity to learn is largely responsible for the speed with which individuals desist from most forms of criminal behavior following the onset of puberty. As an individual's learning ability goes up, the basic distinction between illegal ("crude") and legal ("sophisticated") forms of competition for resources (and mating opportunities) are more easily appreciated. Thereafter, as an individual's learning ability, augmented with opportunities to acquire and practice competitive/victimizing techniques, increases, his/her behavior will hover within legal boundaries.

Keen learning ability also helps males acquire the knowledge and skills needed to successfully compete for resources within the limits of legality. Of course, having appropriate opportunities to practice those skills may also help to speed the transition from "crude" to "sophisticated" forms of competitive/victimizing behavior. However, males whose learning abilities are impaired may take a decade or even longer to consistently behave in a lawful manner. The fact that androgen levels slowly subside beyond early adulthood (Archer, 2004) should also serve to reduce law-violating behavior.

3.1.2.3. Capsulizing the theory. ENA theory offers an explanation for why competitiveness, risk-taking, dominance-striving, and physical aggression are all correlated with high testosterone exposure. In essence, the theory envisions all of these behavioral tendencies as facilitating male acquisition of resources with which males attract mates and thereby pass their genes on to future generations.

It is important to emphasize that ENA theory does *not* argue that exposing the brain to male-typical levels of androgens automatically causes criminal behavior following the onset of puberty. Instead, it argues that *as brain exposure to testosterone surges at puberty, the prenatally-programmed motivation to strive for resources, status, and mating opportunities will begin to fully activate.* The effects of androgens on aggression are therefore essentially indirect. If an individual's motivations for resources, status, and mating opportunities are curtailed, the probability of physically aggressive counter-measures is a likely outcome.

Furthermore, ENA theory does not contend that combining prenatal and postpubertal androgens determine how long individuals will persist in criminal activities. Instead, learning abilities, especially regarding language, is very important, not as a motivator, but as a facilitator of how quickly the androgen-induced desires for resources, status, and mating opportunities are channeled away from "crude" (largely criminal) to "sophisticated" (largely non-criminal) forms of competition.

4. Part IV

4.1. Applying ENA theory to the study of race differences in offending

In this fourth part to the article, two questions are addressed: first, is it reasonable to believe that average racial/ethnic differences in androgen exposure contributes to racial/ethnic variations in criminality? Second, are there average racial/ethnic differences in learning ability that could contribute to racial/ethnic variations in persistent criminal offending? This latter question follows from ENA theory's assertion that learning ability affects how quickly individuals transition from "crude" (i.e., largely illegal) forms of competitive/victimizing behavior to "sophisticated" (i.e., largely legal) forms of the behavior.

Answering the first of these two questions is difficult partly because only piece-meal research is available. Another hindrance comes from noting that it is *brain exposure* to androgens that is most crucial to variations in criminality. Unfortunately, there is no way to assess brain exposure precisely, especially in living organisms. Thus, all ten indicators of androgen exposure to be discussed are indirect measures derived from sources outside the brain itself. Also, as will be made clear below, androgen production is far from stable over time. In fact, levels produced can vary considerably even from 1 h to the next (Dabbs, 1990; Harris, Rushton, Hampson, & Jackson, 1996).

4.1.1. Criminality and race/ethnic variations in androgen exposure

Do the races vary in terms of average brain exposure to androgens in ways that can account for race variations in offending? While the evidence is complex, most of it points to an affirmative answer. Nonetheless, because androgen levels in the brain itself are all but impossible to directly determine, nearly all of the evidence to be reviewed is derived from other parts of the body.

Ten indicators of androgen exposure will be individually considered regarding how they vary (a) with respect to criminality and (b) according to race and ethnicity. The measures can be subsumed under one of three categories: fluid measures, physiological and health measures, and one gene-based measure.

As a prelude to this endeavor, it is worth bearing in mind that twin studies indicate that circulating androgen levels are roughly 50% heritable, especially for males (Harris et al., 1996; Hoekstra, Bartels, & Boomsma, 2006). This is true even within races although most of the specific controlling genes or their alleles have yet to be identified (Hong et al., 2001).

4.1.1.1. Fluid androgen measures. The results from three fluid measures of androgen exposure (all peripheral to the nervous system) will be presented. One involves sampling amniotic fluid while the other two involve sampling circulating androgens later in life, usually following the onset of puberty.

4.1.1.2. Testosterone in the amniotic fluid. An amniocentesis involves using a syringe to withdraw a small sample of the fluid from the amniotic sack surrounding the fetus while it resides in the mother's womb. It is considered a good sampling source for hormones being produced by the fetus since nearly all of the amniotic fluid is derived from fetal excretions (Blackburn, 2007; Judd, Robinson, Young, & Jones, 1976; van de Beek, Thijssen, Cohen-Kettenis, van Goozen, & Buitelaar, 2004).

4.1.1.2.1. Criminality links. To my knowledge, no study has yet been conducted to determine if androgen levels in the amniotic fluid correlate with later involvement in criminal behavior. ENA theory clearly predicts that such a measure of prenatal androgens should be positively correlated with later involvement in crime, especially for males with low intelligence. Regarding males with high intelligence, the theory would predict that after a short adolescent period of delinquency, there should be a long term involvement in more successful forms of competition for resources such as engaging in successful business and occupational pursuits.

4.1.1.2.2. Race/ethnic links. Only one study was located in which testosterone was measured in the amniotic fluid according to race/ethnicity (Martel & Roberts, 2014). This study had a limited sample size (N = 109), 59% of which were non-Hispanic whites. Therefore, the researchers simply combined the remaining 41% of their sample (consisting of blacks, Hispanics, and Native Americans) into a single comparison group. The results revealed no significant differences in prenatal testosterone levels. To the degree this study provides a valid measure of race/ethnic differences in prenatal androgens, it provides no support for ENA theory.

4.1.1.3. Testosterone in umbilical cord serum. A few studies have assessed prenatal exposure to testosterone by extracting serum from the umbilical cord at the time of birth (Keelan et al., 2012; Robinson et al., 2013; Rohrmann et al., 2009). This sampling source partially reflects testosterone emanating from the mother, but the fact that there are substantial sex differences in umbilical cord levels of testosterone means that it contains testosterone of fetal origins as well (Whitehouse et al., 2015). Thus, there should be a positive correlation between umbilical cord testosterone levels and later involvement in crime.

4.1.1.3.1. Criminality links. No research pertaining to an association between umbilical cord testosterone levels and criminality was located. However, one study found testosterone levels in the umbilical cord was positively correlated with symptoms of externalizing behavior in children (e.g., acting out, chronic disobedience) (Robinson et al., 2013), and multiple studies have shown childhood externalizing behavior to be a good predictor of later delinquency (Ellis et al., 2009, pp. 171–172).

4.1.1.3.2. Race/ethnic links. Are there race/ethnic differences in umbilical cord testosterone? The answer is complicated. Two studies have reported testosterone levels in the umbilical cord blood of blacks and whites, both of which concluded that the levels for blacks were significantly higher (Henderson, Bernstein, Ross, Depue, & Judd, 1988; Troisi et al., 2003). However, two other studies questioned this conclusion. One study of newborn females found no significant black-white differences in the umbilical cord (Agurs-Collins et al., 2012). The other study of males found no significant black-white differences in testosterone, but did report that the ratio of testosterone to SHBG (sex hormone-binding globulin) was higher for blacks (Rohrmann et al., 2009). SHBG is a biochemical that binds to testosterone, thereby preventing testosterone from leaving the blood system, thus rendering testosterone largely unable to affect organ development (including the brain). If this latter finding is accurate, black fetuses could actually be exposed to *less* testosterone than white fetuses, which would be contrary to predictions by ENA theory.

Regarding other racial comparisons, the Rohrmann et al. (2009) study referred to above found that umbilical cord testosterone levels among Hispanics was significantly higher than both blacks and whites. Finally, two comparisons of testosterone levels in the umbilical cord blood of Asians (specifically Chinese) relative to whites (of both sexes) revealed higher levels among the Asians (Lagiou et al., 2011; Troisi et al., 2008).

Overall, more research on links between umbilical cord testosterone, criminality, and race/ethnicity is needed. What is currently available provides little support for and some apparent contradictions to ENA theory.

4.1.1.4. Circulating testosterone. Circulating hormones are those present in the blood system as individuals go about their daily lives. A few studies have measured testosterone levels during childhood (e.g., Caramaschi, Booij, Petitclerc, Boivin, & Tremblay, 2012), but most have focused on postpubertal levels, when male levels skyrocket (see Fig. 1). Nonetheless, the averages associated with sex and age represented in Fig. 1 give no impression of the extent to which circulating levels of testosterone varies within individuals from day to day and even from hour to hour (Liening, Stanton, Saini, & Schultheiss,

Table 7
Findings from studies of black-white differences in circulating testosterone levels.

Study	Age range	Types of testosterone	Ns	Differences (* < 0.05; ** < 0.01)
Abdelrahaman et al., 2005	5–9	Total	47 W plus B combined	Not significant
Asbell et al., 2000	60 +	Total	26 W & 12 B	Not significant
Ellis & Nyborg, 1992	Adults	Total	3654 W_{NH} & 525 B	B > W_{NH}*
Ettinger et al., 1997	25–36	Free/total	157 W & 197 B	B > W*
Gapstur et al., 2002	20–34	Total	695 W & 483 B	B > W*
Heald et al., 2003	Adults	Free/total	55 W & 75 B	Not significant
Hill, Garbaczewski, & Walker, 1984	11–18		251 W & 258 B	Not significant
Hoffman et al., 2005	18–88	Total	21 W & 20 B	Not significant
Hu, Odedina, Reams, Lissaker, & Xu, 2015	12–60 +	Free/total/bioact.	631 W & 355 B	B > W*
Kubricht et al., 1999	Adults	Total	264 W & 189 B	B > W* (under 40) not signif. (over 40)
Kupelian et al., 2008	30–79	Free/total	197 W & 198 B	Not significant
Lee et al., 2010	6–11	Total	228 W_{NH} & 266 $B_{(NH)}$	B > W but not signif.
Litman et al., 2006	30–79	Total/bioactive	710 W & 538 B	Not significant
Lopez et al., 2013	12–19	Total	33 W & 38 B	W_{NH} > B*
Marks et al., 2006	Adults	Total	36 W & 25 B	Not significant
Mazur, 2009	12–90	Total	1637 in total sample	B > W*
Morrison, Sprecher, Biro, Apperson-Hansen, & DiPaola, 2002	10–15	Free	238 W & 251 B	B > W, but not signif.
Orwoll et al., 2006	12–90	Free/total	228 W_{NH} & 266 B_{NH}	B > W* (age & BMI controlled)
Platz et al., 2000	40–75	Total	55 W & 43 B	Not significant
Richards, Svec, Bao, Srinivasan, & Berenson, 1992	6–18	Total	519 W & 519 B	Not significant
Ross et al., 1986	18–22	Total	50 W & 50 B	B > W*
Srinivasan, Freedman, Sundaram, Webber, & Berenson, 1986	11–17	Total	251 W & 258 B	Not significant
Ukkola et al., 2001	30s	Total	202 W & 93 B	Not significant
Winters et al., 2001	18–24	Free/total	23 W & 23 B	B > W** for total T, but not sign. For free T
Wu et al., 1995	60 +	Free/total/bioact.	402 W & 306 B	B > W but not signif.

2010). For this reason, when blood or saliva assays are drawn, the normal procedure is to obtain each person's assay within an hour after awakening, when levels are usually the highest they will be on any given day (Harris et al., 1996; Raff & Sluss, 2008).

4.1.1.4.1. Criminality links. Dozens of studies have been undertaken to determine if circulating testosterone and criminality are correlated. Two reviews of these studies have been published in recent years, both concluding that most studies have reported modest positive associations (Ellis et al., 2009, pp. 208–212; Yildirim & Derksen, 2012, pp. 990–995). Similarly, two different meta-analyses of studies correlating testosterone with various measures of human aggression both concluded that the relationship was modestly positive (Archer, 2001).

4.1.1.4.2. Race/ethnic links. Considerable research has investigated links between race and circulating testosterone levels, most of which have compared black and white males. Table 7 provides a summary of the 25 studies that were located in this regard. As one can see, only nine of these studies reported significantly higher testosterone levels among blacks than among whites. The majority of the studies found no significant differences and one reported that white males actually had higher testosterone levels than black males.

Among the reasons for the inconsistencies are likely to involve sample sizes and age. Specifically, four of the studies were based on fewer than one hundred research participants (e.g., Asbell et al., 2000; Hoffman, Wang, Gallagher, & Heymsfield, 2005; Lopez et al., 2013; Marks, Hess, Dorey, & Macairan, 2006). Regarding age, black-white differences appear to be primarily limited to adolescents and young adults (Ellis & Nyborg, 1992; Gapstur et al., 2002; Kubricht, Williams, Whatley, Pinckard, & Eastham, 1999). As one can see in Table 7, several of the studies reporting no significant differences were based either on preadolescents (Abdelrahaman, Raghavan, Baker, Weinrich, & Winters, 2005; Lee et al., 2010) or older adults (Asbell et al., 2000; Platz, Rimm, Willett, Kantoff, & Giovannucci, 2000). Overall, while the evidence is conflicting, most of the findings based on reasonably large samples drawn from adolescents and young adults do support the conclusion that among males, blacks have somewhat higher levels of testosterone than do whites.

Also worth mentioning is that the study that reported significantly higher testosterone among whites than among blacks statistically controlled for percent body fat and amount of physical activity before making the comparison (Lopez et al., 2013). Testosterone levels have been shown to be inversely correlated with percent body fat (Pasquali et al., 1991; Vermeulen, Goemaere, & Kaufman, 1998) and positively correlated with physical activity (Rudman et al., 1994). Therefore, statistically controlling for these variables prior to making race comparisons could explain why the results reported by Lopez et al. were contrary to all other findings (i.e., higher testosterone for whites compared to blacks).

A recently published meta-analysis sheds additional light on the controversy over black-white differences in circulating testosterone. It concluded that there are modest but significant differences with the levels for black males being higher (Richard et al., 2014). Overall, black males appear to have higher testosterone levels than do white males.

Eight studies were located that sampled Hispanic males and white non-Hispanic males. They concluded that Hispanic males had significantly higher testosterone levels (Lopez et al., 2013; Orwoll et al., 2006; Rohrmann et al., 2007). The remaining five studies all reported that the differences were not statistically significant, albeit the number of Hispanics in several of these studies was small (Ellis & Nyborg, 1992; Kupelian, Hayes, Link, Rosen, & McKinlay, 2008; Litman, Bhasin, Link, Araujo, & McKinlay, 2006; Mazur, 2009; Nyante et al., 2012).

Regarding Native Americans, one Canadian study of male sex offenders compared their circulating testosterone levels with those of white male offenders. It reveals significantly higher levels of testosterone among the Native American sample (Studer, Reddon, & Siminoski, 1997).

In terms of comparing circulating testosterone in which Asians were sampled, a few studies were located. One involved elderly men (mean age = 69) living in Canada and the U.S. It concluded that East Asian-Americans had the highest testosterone levels, following by African-Americans, and whites with the lowest levels (Wu et al., 1995). Similarly, a study comparing Chinese males with white males concluded that the former had significantly higher circulating testosterone levels (Jin, Turner, Zhou, Zhou, & Handelsman, 1999). Another study involving East Asians and whites concluded that Asians had higher

testosterone levels (A.S. Whittemore, L.N. Kolonel et al., 1995; A.S. Whittemore, A.H. Wu, et al., 1995). On the other hand, a study comparing Japanese and Dutch men concluded that the latter group had higher testosterone levels (de Jong et al., 1991). Likewise, a comparison between a sample of Korean men and Swedish men concluded that circulating testosterone levels were substantially higher in the latter (Jakobsson et al., 2006).

Only one study bearing specifically on South Asians was located. It compared Pakistani men living in Britain to native whites and concluded that the whites had significantly higher testosterone levels (Heald et al., 2003).

Overall, the available evidence regarding race differences in circulating testosterone appears to be unsettled except for black-white differences. In this case, black males have higher circulating levels than do white males especially during adolescence and young adulthood. Conclusions concerning other racial/ethnic differences must await more research, although Hispanics and non-Hispanic whites appear to be very similar. East Asians, especially Chinese males, on the other hand, may have higher testosterone levels than whites, a point that will be revisited later.

4.1.1.5. Postpubertal circulating dihydrotestosterone. Dihydrotestosterone (DHT) is a metabolite of testosterone produced by combining a testosterone molecule with an enzyme known as 5-*alpha-reductase* (George, Russell, & Wilson, 1991). In terms of its masculinizing effects on many bodily organs, DHT is considered a more potent androgen than testosterone (Manning, 2009, p. 19; Pettaway, 1999; Schlinger & Callard, 1990).

4.1.1.5.1. Criminality links. No research was located concerning a possible DHT-criminality relationship. However, a significant positive correlation between circulating DHT levels and externalizing behavior among a sample of juveniles was found for males, but not for females (Maras et al., 2003). Also, among young adults in Germany, a positive correlation was found between DHT levels in saliva samples and self-reported aggression (Christiansen & Knussmann, 1987). Also, two Finish studies reported a significant positive correlation between DHT levels and self-reported feeling of anger; one was among a sample of females (von der Pahlen, Lindman et al., 2002) and the other among a sample of males (von der Pahlen, Sarkola et al., 2002).

4.1.1.5.2. Race/ethnic links. Concerning the possibility of race/ethnic differences in circulating DHT levels, one study of adult males found blacks had significantly higher levels of circulating DHT than did both whites and Asians, with no significant difference between the latter two groups (Orwoll et al., 2010). Two other studies involving both sexes of various ages also reported higher DHT levels in blacks than in same-sex whites (Hong et al., 2001; Ukkola et al., 2001). However, one additional study of young to middle aged adults concluded that there were no significant black-white differences in DHT levels (Marks et al., 2006). Overall, more research is need, but most of what has been reported so far points toward higher intra-sex levels among blacks than among whites.

4.1.1.6. Physical indicators of androgen exposure. Physical indictors of androgen exposure are traits that are promoted by exposure to testosterone or some other androgen. In this regard, five indicators will be examined: muscular strength, bone density and length, penis size (for males), the 2D:4D finger length ratio, and incidence of prostate cancer.

4.1.1.7. Muscularity and strength. Testosterone's role in promoting and sustaining muscular strength is beyond question (Auyeung et al., 2011; Joubert et al., 1994; Morley et al., 1997; Sassoon, Gray, & Kelley, 1987; Schroeder et al., 2005). As a powerful anabolic steroid, testosterone ensures that even in infancy, but especially after puberty, males are substantially more muscular and physically stronger than females (reviewed by Ellis, Das, & Buker, 2008, pp. 30–31).

4.1.1.7.1. Criminality links. It is noteworthy that muscularity and physical strength are positively correlated with criminal behavior. Numerous studies have shown that individuals who are involved in offending tend to be more muscular (Blackson & Tarter, 1994; Glueck & Glueck, 1956; Hartl, Monnelli, & Eldeken, 1982; Sampson - & Laub, 2014) and rate themselves as physically stronger (Ellis et al., 2008) than those who appear to offend the least. Even antisocial behavior and aggression prior to puberty have been found positively associated with physical strength (Isen, McGue, & Iacono, 2015).

4.1.1.7.2. Race/ethnic links. Regarding within-sex race differences, research has indicated that relative to whites, blacks have denser striated muscles (Ettinger et al., 1997; Katsiaras et al., 1985) and tend to be physically stronger (Goodpaster et al., 2006). Studies of muscularity and strength among other racial groups were not found.

4.1.1.8. Bone density and stature. Testosterone has been shown to promote the elongation and the density of bones (Kenny et al., 2001; Leonard et al., 2010; Urban et al., 1995). For this reason, males tend to be taller and have denser bones than females in all known human populations (reviewed by Ellis et al., 2008, pp. 14–16, 35). Even among males, circulating testosterone has been shown to be positively correlated with height (Tremblay et al., 1998).

4.1.1.8.1. Criminality links. Only two studies were located specifically pertaining to a possible relationship between height and criminality. Both of them had to do with officially detected delinquency among males and concluded that height was positively correlated with involvement (Tremblay et al., 1998; Felson, 1996). Another study reported that overall body size at twelve years of age was positively associated with having been convicted of a violent crime later in life (Ikäheimo et al., 2007). Also, one study concluded that childhood aggression among boys was positively correlated with height at three years of age (Raine, Reynolds, Venables, Mednick, & Farrington, 1998). Nonetheless, no research on a possible association between criminality and bone density was located.

4.1.1.8.2. Race/ethnic links. Regarding height and race, findings regarding black-white differences have been somewhat mixed. Some studies have found blacks being taller than whites (Berkey, Dockery, Wang, Wypij, & Ferris, 1993; Garn & Clark, 1976; Hamill, Johnston, & Lemeshow, 1973; Seeman, 2000) while other reported no significant differences (Ogden, 2004; Wang et al., 1996). In the case of Asians-white differences, one multinational study of adult twins (Hur et al., 2008) and one U.S. study of adults in general concluded that whites are substantially taller on average than Asians (Wang et al., 1996). One study of Hispanics in the U.S. concluded that they were significantly shorter than non-Hispanic whites within both sexes (Ogden, 2004).

Turning to bone density, from childhood through adulthood, blacks have repeatedly been shown to have greater bone density than whites among both sexes (Aloia, 2008; Ettinger et al., 1997; Hui et al., 2003; Kleerekoper et al., 1994; Li, Specker, Ho, & Tsang, 1989; Pollock et al., 2011; Seeman, 2000; Taaffe et al., 2001; Wang et al., 1996). Two studies were located that obtained bone density measurements from Hispanics and Asians (unspecific as to East or South Asians) in conjunction with data from blacks and whites. Both studies concluded that whites, Hispanics, and Asians were all similar in bone density, although for a few of the skeletal sites sampled, Hispanics and Asians had the lowest bone density, with whites being intermediate, and again blacks having the greatest bone density (Bachrach, Hastie, Wang, Narasimhan, & Marcus, 1999; Wang et al., 1996). One additional study limited to female non-Hispanic whites, Hispanics, and blacks concluded that the only truly distinctive group were blacks with significantly higher bone density (Berenson, Breitkopf, Newman, & Rahman, 2009).

4.1.1.9. Penis size. Experiments with laboratory animals have shown that prenatal testosterone plays an important role in promoting post-pubertal male penis size (Herman, Jones, Mann, & Wallen, 2000; Yucel,

Cavalcanti, Desouza, Wang, & Baskin, 2003). Furthermore, a recent study reported that penis length among a sample of Egyptian baby boys was positively correlated with both free and total testosterone levels in the umbilical cord (Mohamed, Abdou, Hamza, & Hussein, 2015).

4.1.1.9.1. Criminality links. Regarding the potential relevance of penis size to criminology, two studies (both based on the same data set) have been published. They reported significant positive correlations between self-assessed penis size and self-reported involvement in delinquency, especially regarding violent delinquency (Ellis & Das, 2013; Ellis et al., 2008).

4.1.1.9.2. Race/ethnic links. While it is a sensitive topic to deal with and stereotypes abound, a few studies have attempted to determine if racial/ethnic differences in penis lengths and diameters exist. Most of these studies are based on self-measurements in flaccid, stretched, and/or erect conditions. Three studies reported that the average penis length of black males were significantly longer and greater in diameter than those of whites (Nobile, 1982; Gebhard, Johnson, & Kinsey, 1979; Rushton & Bogaert, 1987). However, a recently-reported fourth study found longer penises for blacks than for whites, although the difference was not statistical significant (Herbenick, Reece, Schick, & Sanders, 2014). Data for four other racial/ethnic groupings were also included in this latter study – i.e., Asians (without distinguishing South Asians and East Asians), Pacific Islanders, American Indians, and Hispanics – none of which differed significantly from blacks or whites.

Two studies provided estimates of racial variations in penis size by comparing data from individuals of different countries rather than individuals of specific racial groupings (Lynn, 2013; Templer, 2002). Both concluded that significantly larger penises were found for males in predominantly black countries (mainly those in sub-Sahara Africa) relative to males in mainly Asian countries (without being specific regarding South Asian or East Asian). Males in European countries had penises whose lengths were between those from the sub-Saharan and the Asian countries.

Overall, most of the evidence supports the view that black males have larger penises than do white males, and that the penises of Asian males are smaller than those of white males. This, of course, supports the view that among males, blacks are exposed to higher levels of androgens than whites and especially Asians. Nevertheless, intra-race differences appear to be far greater than average differences between racial/ethnic groups.

4.1.1.10. 2D:4D finger length ratio. Over the past couple of decades, researchers have conducted many studies in which a rather simple noninvasive method for assessing prenatal testosterone exposure known as the *2D:4D ratio* has been utilized. This procedure involves measuring the relative length of the 2nd and 4th fingers, usually of the right hand (Manning & Fink, 2008). Although it is considered valid (Hampson, Ellis, & Tenk, 2008; Lutchmaya, Baron-Cohen, Raggatt, Knickmeyer,- & Manning, 2004), the 2D:4D ratio is only modest in terms of reliability (Hell & Päßler, 2011; Lippa, 2003; Putz, Gaulin, Sporter, & McBurney, 2004). A specific study in this regard revealed that there was a significant, albeit weak, correlation between 2D:4D and an amniocentesis measure of prenatal testosterone (McIntyre, 2006). Thus, large sample sizes are needed in order to confidently expect to detect correlations between 2D:4D and some dependent variable such as criminality.

The details on how the 2D:4D ratio is altered by testosterone exposure is not fully understood, but it certainly reflects the fact noted earlier that testosterone promotes bone growth (including the bones in the fingers). Also, the digits of the fetus are apparently not exposed to exactly the same amounts of testosterone as they slowly elongate. The end result is that the longer the 4th finger is relative to the 2nd finger, the more testosterone one was exposed to during prenatal development. Of course, male exposure is substantially greater than female exposure; thus males have been repeatedly shown to have lower 2D:4D ratios on average than females (Gillam, McDonald, Ebling, & Mayhew, 2008; Hönekopp & Watson, 2010; Kraemer et al., 2006; Manning, Scutt, Wilson, & Lewis-Jones, 1998; Manning, Churchill, & Peters, 2007).

4.1.1.10.1. Criminality links. Most studies undertaken to determine if 2D:4D ratios correlate with criminality have provided affirmative answers. Specifically, studies of traffic violations by males (Schwerdtfeger, Heims, & Heer, 2010) and of males with one or more criminal convictions versus no convictions (Hanoch, Gummerum,- & Rolison, 2012) both concluded that 2D:4D ratios were inversely correlated with probabilities of offending. More recently, a colleague and I conducted two studies of self-reported offending and found that within both sexes, 2D:4D ratios on the right hand were inversely associated with offending (L. Ellis & A. Hoskin, 2015; Hoskin & Ellis, 2015). However, one study that compared male convicts with males without criminal convictions found no significant differences (Anderson, 2012).

It is also worth mentioning that contrary to expectations, a study of 2D:4D and self-reported psychopathy among females found a significant *positive* correlation (Blanchard & Lyons, 2010). And a two-country study of children with externalizing behavior concluded that 2D:4D was significantly correlated inversely with such behavior (as expected) in an Austrian sample but not in a British sample (Fink, Manning, Williams, & Podmore-Nappin, 2007).

Studies have also sought to determine if 2D:4D is associated with various forms of physical aggression. In accordance with predictions, significant inverse correlations have been reported especially for males (Bailey & Hurd, 2005; Kuepper & Hennig, 2007; McIntyre et al., 2007). The same is true for aggressive dominance (van der Meij, Almela, Buunk, Dubbs, & Salvador, 2012). Bearing in mind that 2D:4D appears to be largely determined prenatally (Hönekopp, Bartholdt, Beier, & Liebert, 2007), it seems noteworthy that one of these studies (Bailey & Hurd, 2005) reported that the correlation between 2D:4D and physical aggression was actually stronger than the average correlation between *circulating* testosterone and physical aggression derived from a meta-analysis (Archer, Birring, & Wu, 1998). Overall, the low reliability of 2D:4D as a measure of prenatal androgen exposure continues to pose an obstacle to assessing any association between prenatal androgens and traits such as criminality, psychopathy, and physical aggression. In this regard, a meta-analysis of the relevant studies concluded that no significant association was identified (Pratt, Turanovic, & Cullen, 2016).

4.1.1.10.2. Race/ethnic links. Regarding race differences in 2D:4D ratios, several studies have compared blacks and whites within one or both sexes. Among males, findings have consistently revealed that blacks have lower ratios than do whites, thus indicating that blacks were exposed to higher prenatal testosterone levels (Ellis, Lykins, & Ratnasingan, 2013; Knickmeyer, Woolson, Hamer, Konneker, & Gilmore, 2011; Manning, 2008; Manning & Fink, 2008; Manning, Henzi, Venkatramana, Martin, & Singh, 2003; Manning, Stewart, Bundred, & Trivers, 2004; Waters et al., 2013).

Regarding studies of other racial/ethnic groups, the evidence is mixed, possibly due to most studies being based on small sample sizes. In this regard, one Canadian study compared 104 whites and 55 Asians and concluded there were no significant differences (Hurd, Vaillancourt, & Dinsdale, 2011). A U.S. study consisting of 105 whites and 24 or fewer of other racial groups also reported no significant differences (Hone & McCullough, 2012). Another U.S. study of infants found insignificant differences between whites and a small group of Asians (Knickmeyer et al., 2011).

Two studies were based on an impressively large survey sponsored by the British Broadcasting Corporation with more than a quarter million respondents from all parts of the world. Using somewhat different criteria for assessing race/ethnicity, both studies reported significant race/ethnic differences in average 2D:4D ratios. One study grouped respondents into three categories: whites, Hispanics, and Asians. It concluded that especially for males, Asians had the lowest

2D:4D ratios, followed by Hispanics, and then by white non-Hispanics (Lippa, 2003). This suggests that Asian males were exposed to the highest prenatal testosterone levels of these three racial/ethnic groups. The second study compiled respondents into five groups: whites, blacks, Chinese, non-Chinese Asians, and Middle Easterners. This study concluded that the ordering of 2D:4D for these groups were as follows: whites > non-Chinese Asians > Middle Easterners > blacks > Chinese (Manning et al., 2007). Overall, both studies (albeit using the same large data set) concluded that Asian males, especially Chinese males, have 2D:4D ratios that are lower or equivalent to those for black males. This suggests that East Asians are being exposed to extremely high prenatal testosterone. I will return to this point again later, since it seems to have a genetic explanation involving race differences in so-called *androgen receptors*.

4.1.1.11. PSA levels and prostate cancer. Although the biochemistry is complex, studies have found that high long term androgen exposure is associated with elevated prostate-specific antigen (PSA) levels and with diagnosed prostate cancer (Curran & Bihrle, 1999; Young et al., 1991). A positive association between androgen exposure and the probability of developing prostate cancer has also been well established (Barrett-Connor, Garland, McPhillips, Khaw, & Wingard, 1990; Gaston, Kim, Singh, Ford, & Mohler, 2003; Morales, 2002). ENA theory leads one to hypothesize that both PSA levels and the incidence of prostate cancer should be associated with offending probabilities.

4.1.1.11.1. Criminality links. No research was located pertaining to the possibility that either PSA levels or prostate cancer risks are associated with criminal behavior. Therefore, the possibility of a correlation remains to be investigated.

4.1.1.11.2. Race/ethnic links. Studies have shown that PSA levels are substantially higher among black males than among white males (Abdalla, Ray, Ray, Vaida, & Vijayakumar, 1998; Morgan et al., 1996; A.S. Whittemore, L.N. Kolonel et al., 1995; A.S. Whittemore, A.H. Wu, et al., 1995). Regarding prostate cancer, black males have more than twice the incidence rate of white males (Altekruse et al., 2010; Curado, Edwards, Shin, & H. R., 2007; Hoffman et al., 2001; Mathew, Elting, Cooksley, Owen, & Lin, 2005; Nelson & Witte, 2002; Peters & Armstrong, 2005; Platz & Giovannucci, 2006; Powell, Bock, Ruterbusch, & Sakr, 2010; Sidney et al., 1991; Yamoah, Stone, & Stock, 2011). This is true not only in the U.S. where most studies have been conducted, but also in the Brazil (Bouchardy et al., 1991) and the Caribbean Islands (Glover et al., 1998).

Dietary and other within-country environmental factors (including differential access to medical treatment) have been investigated as contributors to race differences in prostate cancer, but their relevance appears to be minimal (Hayes et al., 1999; A.S. Whittemore, L.N. Kolonel et al., 1995; A.S. Whittemore, A.H. Wu, et al., 1995). Instead, genetic factors have been strongly implicated (Yang, Addai, Ittmann, Wheeler, & Thompson, 2000), including genes regulating androgen production and sensitivity (Morley et al., 1997). Some research has indicated that high rates of conversion of testosterone into dihydrotestosterone due to a gene promoting 5α-reductase enzyme synthesis (as discussed earlier) seems to increase the risk of prostate cancer, at least among blacks (Makridakis et al., 1999; Pettaway, 1999).

A number of studies of PSA levels and prostate cancer risks among races other than blacks and whites have been reported. These studies indicate that East Asian males have PSA levels and prostate cancer risks that are well below those for any other known racial or ethnic group (de Jong et al., 1991; Oesterling et al., 1995; Shimizu et al., 1991; Yamoah et al., 2011). This conclusion is consistent with evidence of unusually low prostate cancer incidence and mortality rates in both eastern and southern Asian countries (Center et al., 2012).

Four studies of PSA levels and/or prostate cancer involving Hispanic males with non-Hispanic white males were found. Two reported PSA levels to be significantly higher for Hispanics than for non-Hispanic whites (Abdalla et al., 1998; Yamoah et al., 2011). The third study reported Hispanic PSA levels being nearly equal to levels in non-Hispanic whites, while their prostate cancer risks were, if anything, slightly lower than those of non-Hispanic whites (Hoffman et al., 2001). The fourth study conducted in California reported that Hispanics had lower rates of prostate cancer than did non-Hispanic whites (Shimizu et al., 1991).

Overall, using PSA and especially the prevalence of prostate cancer as biomarkers for male exposure to testosterone and other androgens supports the conclusion that blacks have higher exposure than do whites. Hispanics and non-Hispanic whites seem to be roughly equivalent, while Asians appear to have relatively low PSA levels and low prostate cancer risks, suggesting that their exposure to androgens is relatively low.

4.1.1.12. Androgen receptor CAG repeat genes. The last line of evidence to be reviewed concerning possible links between criminality, race/ethnicity, and androgens involves the prevalence of proteins known as *androgen receptors* and the genes that control these proteins, called *CAG repeat genes* (or simply *CAG repeats*). Androgen receptor proteins are found in most cells throughout the body and play a critical role in mediating testosterone's masculinizing/defeminizing effects on muscular, skeletal, or neurological organs (Rubinow & Schmidt, 1996).

It is important to note that the greater the number of CAG repeats one has, the *less* effective androgens tend to be in terms of masculinizing/defeminizing the body (La Spada, Wilson, Lubahn, Harding, & Fischbeck, 1991; Sato et al., 2004). In other words, individuals can have very high amounts of testosterone and DHT throughout their bodies, but if their cells have numerous AR CAG repeats, the ability of these androgens to masculinize/defeminize the body (including the brain) will be curtailed (Chamberlain, Driver, & Miesfeld, 1994).

The number of androgen receptors one has is genetically coded by a single gene on the X chromosome (Brown et al., 1989; Chamberlain et al., 1994; Zitzmann & Nieschlag, 2003). Many versions (or alleles) of this gene have evolved regarding the number of repeats it produces. These repeats vary from as few as 11 to as many as 37 (Edwards, Hammond, Jin, Caskey, & Chakraborty, 1992; Platz et al., 2000; Tut, Ghadessy, Trifiro, Pinsky, & Yong, 1997). And again, the more CAG repeats there are on the X chromosome, the *fewer* the number of androgen receptors an individual will have (Allen, Zoghbi, Moseley, Rosenblatt, & Belmont, 1992; Giovannucci et al., 1997; Igarashi et al., 1992; La Spada et al., 1991; Manning et al., 2003). Of course, the fewer the number of androgen receptors, the less impact testosterone and other androgens will be allowed to have on muscle, bone, and brain tissue.

4.1.1.12.1. Criminality links. Two recent studies reported evidence that AR CAG repeats are inversely associated with involvement in criminal behavior. One was a study conducted in India involving over five hundred men, roughly half of whom were convicted of rape. The rapists had significantly fewer CAG repeats than did control males (Rajender et al., 2008). A study conducted in China also found the number of the AR CAG repeats was significantly fewer among violent male criminals than among males with no criminal convictions (Cheng, Hong, Liao, & Tsai, 2006). Findings from these two studies suggest that male criminality, particularly of a violent nature, is more common among males with relatively few CAG repeat alleles in their AR controlling gene.

Furthermore, one study revealed an inverse correlation between the number of CAG repeats in AR genes and symptoms of childhood conduct disorders among boys (Comings, Chen, Wu, & Muhleman, 1999). Three other studies found the number of AR CAG repeats negatively correlated with impulsivity among males (Aluja, García, Blanch, & Fibla, 2011; Vermeersch, T'Sjoen, Kaufman, Vincke, & Van Houtte, 2010; Westberg, Henningsson, Landén, et al., 2009). The relevance of these latter studies is that both childhood conduct disorders and impulsivity are statistically associated with criminality (reviewed

by Ellis et al., 2009, pp. 7–8, 122–126). One recent study also reported an inverse correlation between AR CAG repeats and self-reported aggression among male Tanzanians (Butovskaya et al., 2015).

4.1.1.12.2. Race/ethnic links. Research has provided considerable evidence of race differences in the number of AR CAG repeats. In particular, research has consistently shown that blacks have the fewest average numbers. Specifically, the mean number of repeats for blacks is in the range of 19 ± 3.5sd. Whites (Europeans) usually have intermediate numbers (Mean = 21 ± 3.0sd), and East Asian populations have the greatest number (Mean = 22.5 ± 3.5sd) (Ackerman et al., 2012; Bennett, Price, Kim, et al., 2002; Edwards et al., 1992; Hsing et al., 2000; Hurd et al., 2011; Irvine, Mimi, Ross, & Coetzee, 1995; Manning, 2007; Nelson & Witte, 2002; Platz et al., 2000; Ross et al., 1986; Sartor, Zheng, & Eastham, 1999; Sasagawa et al., 2001; Suzuki et al., 2002; Tut et al., 1997).

For racial/ethnic differences other than blacks, white, and East Asians, the average AR CAG repeat numbers is less well established. Two studies comparing Hispanics and non-Hispanic whites were located. One reported that the average number of repeats among Hispanics was slightly but significantly fewer than those of non-Hispanic whites (Balic et al., 2002) while the other study reported the opposite pattern (Davis-Dao et al., 2011).

In the case of South Asians, the only estimates that could be found were based on two studies conducted in India. One study obtained a mean number of AR CAG repeats of 21.7 ± 0.18 (Thangaraj et al., 2002) and the other reported a mean of 22.2 ± 1.5 (Dhillon & Husain, 2003). If one averages these two means, the result is 21.95, which is slightly higher than the 21.0 estimate for whites (Europeans) and slightly lower the 22.5 estimate for East Asians. No normative data on AR CAG repeats for Native Americans or Pacific Islanders were found.

The black < white < Asian ordering of AR CAG repeats may be helpful in solving a puzzle regarding race differences in androgen exposure. As discussed earlier, studies of both circulating testosterone levels and of the 2D:4D ratios indicated that East Asian males are exposed to higher prenatal testosterone than any other racial groups (Manning, 2008, p. 29; Orwoll et al., 2006). However, physiological indicators of androgen exposure (such as musculature and height) suggest that Asian males are *less* androgenized than males of any other race (Manning, 2008, p. 29). It seems possible to explain these seeming inconsistencies by assuming that the high number of AR CAG repeats among Asians, especially East Asians, has the effect of inhibiting much of the physiological effects of androgens relative to other racial groups. This hypothesis needs to be tested.

Overall, the evidence for average race/ethnic differences in AR CAG repeats appears to be consistent. If Native Americans and Pacific Islanders are set aside for lack of evidence, and the racial/ethnic groups are ordered in terms of their average AR CAG repeats, the order would be as follows: Blacks < White Non-Hispanics ≈ Hispanics < South Asians < East Asians. Such an ordering is quite similar to the ordering of race/ethnic differences in offending rates noted in Part I.

4.1.2. Criminality and race/ethnic variations in learning ability

As noted earlier, ENA theory asserts that due to evolutionary forces, two main variables impact involvement in criminal behavior. The first variable is brain exposure to androgens, with high exposure being associated with greater involvement. Learning ability is the second variable. The lower one's learning ability, the more time individuals take to transition from “crude” (often criminal) forms of competitive/victimizing behavior to “sophisticated” (usually non-criminal) forms of the same behavior. Therefore, indicators of learning ability should inversely correlate with the length of time an individual remains involved in criminal behavior following the onset of puberty. Four measures of learning ability will be given consideration below: intelligence, academic performance, learning disabilities, and brain size.

4.1.2.1. Intelligence. Even before specific tests of intelligence were developed, few doubted that humans exhibit a great deal of between-individual variation in their intellectual abilities. Around the start of the 20th Century, educational psychologists developed a series of intellectual challenges for children that were found useful in forecasting how well these children would perform in school (Jensen, 1998; Mackintosh, 2011). They called the resulting scores *intelligence*. Because intelligence was found to vary simply according to chronological age, at least until early adulthood, the scores were often adjusted to reflect changes associated with age, resulting in what is known as *IQ* (for *intelligence quotient*) (Embretson, 1992; Jensen, 1998).

4.1.2.1.1. Criminality Links. For decades, studies have been undertaken to determine if intelligence and involvement in criminal behavior are correlated. The findings have overwhelming supported the conclusion that these two variables are inversely related (reviewed by Burhan, Kurniawan, Sidek, & Mohamad, 2014; Ellis & Walsh, 2003; Hirschi & Hindelang, 1977). When attention is given to repeat offenders, especially for violent crimes, the IQ-offending associations are especially strong. On average, repeat offenders score at least ten points lower than do non-offenders (Kratzer & Hodgins, 1999; Moffitt, Gabrielli, Mednick, & Schulsinger, 1981; Wolfgang, Figlio, & Sellin, 1972). Furthermore, the most pronounced differences between serious offenders and non-offenders usually involves the linguistic portions of intelligence tests rather than portions having to do with math and spatial reasoning (Walsh & Beyer, 1986; Yeudall, Fromm-Auch, & Davies, 1982).

4.1.2.1.2. Race/ethnic links. Turning attention to the controversial issue of race differences in intelligence, studies have repeatedly documented race/ethnic differences in average intelligence test scores. These studies have consistently shown that whites score higher than blacks by roughly 12–15 points (Nisbett, 1998; Nyborg, 2015; Rushton & Jensen, 2006) and that East Asians score about 5 points higher than whites (Herrnstein & Murray, 1994; Jensen, 1998; Jensen & Inouye, 1980). The IQ scores of Hispanics appear to be about 8 points lower than those for whites (Hartmann, Kruuse, & Nyborg, 2007; Nyborg, 2015). Scores by Native Americans are similar to those of Hispanics (Naglieri & Ronning, 2000). The scores for South Asians and Pacific Islanders are both typically 5–6 points lower than those for whites (Lynn, 2006; Lynn & Vanhanen, 2002).

4.1.2.2. Academic performance. Academic performance is typically measured with an individual's grade point average (GPA). While this measure is certainly not a perfect gage of learning ability, it is still useful when combined with other measures. In this regard, studies have shown that GPA (especially for core courses that are required of all students) typically correlates strongly with scores on intelligence tests, i.e., r = 0.40 to 0.50 (Chamorro-Premuzic & Furnham, 2008; Cucina, Peyton, Su, & Byle, 2016; O'Connor & Paunonen, 2007).

4.1.2.2.1. Criminality links. Several dozen studies have been undertaken to determine if academic performance and criminality are related. Consistent with ENA theory, the evidence has overwhelmingly concluded that these two variables are inversely correlated (reviewed by Ellis et al., 2009, pp. 150–153).

4.1.2.2.2. Race/ethnic links. Studies of racial/ethnic differences in academic performance have unanimously concluded that Asians outperform whites (Hsin & Xie, 2014; Kao, 1995). However, differences between East Asians and many South Asians in this regard appear to be minimal (Kao, 1995). Whites in turn have higher GPAs than Hispanics (J. Lee, 2002), who in turn usually average higher than blacks (see review Kao & Thompson, 2003; Miller, 2004). A few studies have also indicated that whites obtain substantially higher average grades than do Pacific Islanders (Kao, 1995) and Native Americans (Cucina et al., 2016; Kerbo, 1981).

4.1.2.3. Learning disabilities. Another indicator of learning ability, especially regarding language acquisition, involves the concept of

learning disabilities (Mallett, 2014; Rucklidge, McLean, & Bateup, 2013). Unlike intelligence, which is measured on a continuous scale, learning disabilities are usually treated as a dichotomous variable, i.e., individuals either have or do not have one or more learning disabilities. This makes the measurement of learning disabilities relatively crude, since it is more realistic to believe that learning disabilities are actually present (in nearly all of us) to varying degrees not in an either-or fashion.

4.1.2.3.1. Criminality links. Despite the imprecision in measuring the presence or absence of learning disabilities, the variable has been part of numerous studies of criminal behavior. Research has consistently shown that diagnosed learning disabilities are more common among delinquents and criminals than among persons in general (Grigorenko, 2006; Mannuzza, Klein, & Moulton, 2008; Prentice & Kelly, 1973; Stattin & Klackenberg-Larsson, 1993; West & Farrington, 1973).

4.1.2.3.2. Race/ethnic links. Regarding race differences in learning disabilities, patterns appear to be similar to those for IQ scores, but in reverse order. Specifically, black rates of learning disabilities are roughly twice as high as those for whites (Ong-Dean, 2006). In two studies, both based on extensive comparisons among U.S. children (Coutinho, Oswald, & Best, 2002; Shifrer, Muller, & Callahan, 2011), the conclusion was reached that relative to whites, the prevalence of learning disabilities were all greater for blacks, Hispanics, and Native American. The lowest prevalence was among non-Hispanic whites and Asians (without distinguishing East and South Asians). Another U.S. study confirmed the high rate among blacks, but reported that they were followed closely by Native Americans, then by whites, and finally by Asians/Pacific Islanders (Zhang & Katsiyannis, 2002). This study did not distinguish Hispanics from non-Hispanic whites, nor did it report the proportion of Asians and Pacific Islanders in its sample.

One study in England compared whites and South Asians. It reported that learning disabilities were of equal prevalence for these two groups (McGrother, Bhaumik, Thorp, Watson, & Taub, 2002).

4.1.2.4. Brain size. Beginning in the 1920s, studies were undertaken to determine if learning ability, inferred from intelligence test scores, might be correlated with brain size. Most of these studies reported low positive correlations in this regard, i.e., in the range of 0.10 and 0.25 (reviewed by Ellis, 1994; Christensen, Anstey, Leach, & Mackinnon, 2008). However, as the 20th Century drew to a close, more precise measurement of brain size became possible using magnetic resonance imaging. This method of estimating brain size has shown that the correlation between brain size and IQ scores are actually in the range of 0.40 to 0.45 (Andreasen et al., 1993; Roth & Dicke, 2005; Rushton & Ankney, 2009; Wickett, Vernon, & Lee, 1994). It is therefore reasonable to consider brain size itself as a rough proxy measure of learning ability.

4.1.2.4.1. Criminality links. If (a) brain size is an indicator of learning ability, and (b) ENA theory is correct in asserting that learning ability contributes to rapid transitioning away from criminal behavior following the onset of puberty, one should find an inverse correlation between brain size and involvement in crime. The only study bearing on this possibility was one conducted among an age cohort of over 5000 Finish males. It reported that head size (a relatively crude proxy for brain size) was significantly smaller among the males who had been convicted of violent crimes relative to males with no conviction record. However, the head size of males convicted of non-violent crimes did not differ significantly from the non-convicted males (Ikäheimo et al., 2007). Obviously, more research is in order regarding brain size and criminality.

4.1.2.4.2. Race/ethnic links. Regarding the possibility that brain size has a bearing on racial/ethnic variations in offending, considerable research was located. One compared blacks, whites, and East Asians. It reported that the average brain size for blacks was 1270 cm^3, significantly smaller than that for whites at 1340 cm^3. East Asians had the highest average brain size at 1360 cm^3 (Rushton, 2010). Two additional studies obtained estimates of the average brain size of blacks, Hispanics, and non-Hispanic whites. Both reported average brain sizes for blacks were lowest and non-Hispanic whites were highest, with Hispanics being intermediate (DeCarli et al., 2008; Lynn, 1996). Another study that was limited to comparing whites and blacks concluded that the brains of whites are significantly larger (Ho, Roessmann, Straumfjord, & Monroe, 1980).

5. Part V

5.1. A tabular condensation of part IV for succinct interpretation

Part IV reviewed the evidence pertaining to the relevance of ENA theory to race and ethnic differences in criminal behavior. In particular, it sought to determine the extent to which ten indicators of androgen exposure and four indicators of learning ability are associated with (a) criminality and (b) race and ethnicity in ways predicted by the theory. Give that the overall picture provided in Part IV is difficult to decipher due to its having reviewed an array of studies using many operational measures of similar variables and sometimes conflicting results, Part V will provide a summary of the evidence. The greatest condensation of all is offered in Table 8, which Part V will now describe.

5.1.1. Brain exposure to androgens

As noted earlier, it is essentially impossible to measure brain exposure to androgens directly. Also, because timing of the exposure and the specific androgens involved are important, one must rely on a variety of peripheral indicators of such exposure. Ten such indicators were considered.

1. Testosterone in the amniotic fluid. Central to ENA theory is the idea that higher levels of *prenatal* testosterone (along with other androgens) promote criminality. The single best measure of prenatal androgen exposure comes from assays drawn from amniotic fluid (van de Beek et al., 2004). Therefore, there is probably no better way to critically test ENA theory than to correlate androgen levels in the amniotic fluid with criminal involvement following the onset of puberty. Unfortunately, no such research has yet been reported.

Regarding race/ethnic differences in testosterone levels in the amniotic fluid, meager evidence from one study with a fairly small sample size was located. It indicated that when the levels for non-Hispanic whites were compared to the combined levels from three other racial/ethnic groups (blacks, Hispanics, and Native Americans), the differences were non-significant, obviously not a finding that is supportive of ENA theory. Thus, there is virtually no evidence bearing on ENA theory's prediction that prenatal exposure to androgens (a) contributes to criminal behavior or (b) that racial/ethnic groups with the highest rates of crime are exposed to more prenatal androgens than groups with the lowest rates.

2. Testosterone in the umbilical cord. Androgen levels in the umbilical cord largely reflect hormones being transmitted by the mother to the fetus around the time of birth. Therefore, this sampling source is less relevant to ENA theory than levels found in the amniotic fluid (especially during the second trimester when most sexual differentiation of the brain is thought to occur) (van de Beek et al., 2004). Nevertheless, it is reasonable to assume that androgens in the umbilical cord reflect to some extent how much a fetus was being exposed to at least in the weeks leading up to birth.

As shown in Table 8, little evidence was located bearing on the possibility that umbilical cord testosterone levels are associated with offending later in life. This evidence was limited to a study suggesting that high umbilical cord levels were positively correlated with

Table 8
A summary of evidence regarding ten indicators of androgen exposure and four indicators of learning ability relative to their associations with criminality (column A) and race/ethnicity (column B).

Race/ethnic ranking for criminal involvement Blacks (B) > Hispanics (H) ≈ Native Americans (NA) ≈ Pacific Islanders (PI) > Non-Hispanic Whites (W_{NH}) ≈ South Asians (SA) > East Asians (EA)		
Indicators of androgen exposure and learning ability	Associations with criminality (A)	Racial/ethnic order for each androgen or learning ability indicator (Bolded – well established; Standard type – fairly well established; *Italicized* – possible) (B)
1. Testosterone in the amniotic fluid	No evidence	$W_{NH} \approx B + H + NA$ [no evidence for PI, SA, or EA]
2. Testosterone in the umbilical cord	Very little evidence	$B > W_{NH}$ EA $> W_{NH}$ [no evidence for H, NA, PI, or SA]
3. Circulating testosterone	Modest pos. cor. well established	B $> W_{NH}$ H $\approx W_{NH}$ *NA* $> W_{NH}$ *EA* $> W_{NH}$ W_{NH} > *SA* [no evidence for PI]
4. Circulating dihydrotestosterone	Strong evidence of a signif. pos. cor.	B $> W_{NH}$ $W_{NH} \approx$ EA [no evidence for H, NA, PI, or SA]
5. Muscularity and strength	Strong evidence of a signif. pos. cor.	B $> W_{NH}$ > EA W_{NH} > H [no evidence for NA, PI, or SA]
6. Bone density and elongation	Criminals possibly taller than normal	B $> W_{NH}$ > EA W_{NH} > H [no evidence for NA, PI, or SA]
7. Penis size	Minimal evidence of a positive cor.	B > W > EA [no evidence for H, NA, PI, or SA]
8. 2D:4D finger length ratio	Possible inverse correlation	EA < **B** < **W_{NH}** [no evidence for H, NA, PI, or SA]
9. PSA levels & prostate cancer rates	No evidence	**B** > **W_{NH}** ≈ H > **EA** [no evidence for NA, PI, or SA]
10. Number of AR CAG repeats	Fewer repeats cor. with criminality	**B** < H < **W_{NH}** < SA **W_{NH}** < **EA** [no evidence for NA or PI]
1. Intelligence scores	Well established inverse correlation	**B** < H ≈ NA < **W_{NH}** < **EA** W_{NH} = SA [no evidence for PI]
2. Academic performance	Well established inverse correlation	B < H ≈ NA ≈ PI ≈ NA < W_{NH} < *SA* ≈ *EA*
3. Prevalence of learn disabilities	Fairly well estab. positive correl.	B > NA > $W_{NH} \approx$ *EA* > *SA* [no evidence for H or PI]
4. Brain size	Possible inverse correlation	**B** < **W_{NH}** < **EA** H < W_{NH} [no evidence for NA or PI]

disruptive/externalizing behavior in childhood by male offspring, behavior that has been found statistically associated with criminality following puberty (summarized by Ellis et al., 2009, p. 171).

Regarding race/ethnic differences, evidence was mixed regarding the possibility that umbilical cord levels of testosterone differed between blacks and whites. In the case of East Asians, the available evidence suggests that their umbilical cord testosterone levels were actually higher than those for whites, which directly challenges ENA theory's prediction that high prenatal androgens are associated with elevated involvement in crime. Overall, considerably more research is needed before drawing firm conclusions about how testosterone levels in the umbilical cord might help to explain race/ethnic differences in offending.

3. Postpubertal (circulating) testosterone. From a theoretical standpoint, postpubertal androgens play an important secondary role in causing intra-sex variations in criminal behavior. This is because postpubertal androgens tend to fully activate and engage the alterations in brain structures and functions that were laid down by prenatal androgen exposure. While of secondary importance, postpubertal androgens are more easily measured (in both saliva and the blood), and have therefore been studied much more than prenatal exposure

As shown in Table 8, the available evidence suggests that even within each sex, but especially among adolescent and young adult males, high circulating testosterone correlates positively with criminal involvement. Regarding race/ethnicity, within both males and females, blacks appear to have significantly higher circulating testosterone levels than do whites (although the differences are relatively modest). This tentative conclusion was reached by assembling the twenty-sex studies summarized in Table 7 along with a recent meta-analysis of most of the same studies (Richard et al., 2014). Also, Native Americans may have higher levels than whites. Thus, the patterns for blacks, whites, and Native Americans coincide with differences in their rates of offending. However, Hispanics and non-Hispanic whites appear not to differ in circulating testosterone levels, and some of the research suggests that whites had higher levels than South Asians (even though these two groups appear to have more or less equal offending probabilities). Most challenging of all regarding ENA theory and circulating testosterone was the evidence that East Asians, with their lower offending rates, may have higher circulating testosterone levels than whites.

4. Postpubertal (circulating) dihydrotestosterone. DHT is an especially potent androgen in terms of influencing bodily tissue. As with testosterone itself, DHT can be measured in the blood and saliva following the onset of puberty. Table 7 notes that considerable evidence supports the conclusion that DHT levels are positively correlated with criminal behavior.

In the case of race/ethnicity, Table 8 shows that DHT seems to be higher among blacks than among non-Hispanic whites, and among Native Americans than non-Hispanic whites. However, no differences have been found between Hispanics and non-Hispanic whites, and the levels for non-Hispanic whites appear to be higher than for South Asians. Particularly, troublesome to ENA theory once again involved East Asians. While their offending rates are lower than those of whites, the research on race differences in DHT suggests that theirs is higher than for the average same-sex whites.

5. Muscularity and strength. Muscularity is highly influenced by androgens. To have maximum effect, the androgen levels need to be high both prenatally and postpubertally. Table 8 indicates that muscularity and physical strength are positively associated with criminality. In the case of race/ethnic differences, most of the evidence is limited to differences between blacks and whites. These studies agree that among both sexes, blacks are more muscular and physically stronger than whites of the same age.
6. Bone density and elongation. Androgens are known to promote bone growth and thickness, thus largely accounting for why males are on average taller than females. Not much research is available on possible connections between height and criminality, but as reflected in Table 8, there is a little evidence that at least among males, offenders are slightly taller than non-offenders. In the case of bone density, no research has been undertaken to assess its association with criminality.

Regarding race/ethnicity, most studies have indicated that East Asians are shorter than whites and that blacks may be slightly taller than whites. Also, blacks have been shown to have denser bones than other racial groups. If so, these differences would roughly conform to differences in offending probabilities as ENA theory predicts. However, non-Hispanic whites appear to be taller than Hispanics, which does not conform to the theory's predictions.

7. Penis size. While being a sensitive topic to discuss, research has shown that the length and girth of male penises are promoted by exposure to androgens, particularly testosterone and DHT. The androgen exposure is most effective when it occurs prenatally and is then augmented with additional high exposure following the onset of puberty. As noted in Table 8, there are two studies, both based on the same data set, suggesting that self-reported penis size is positively correlated with self-reported offending. Regarding race differences, most relevant studies have indicated that black males have larger penises on average than do East Asian males, with white males being intermediate.
8. 2D:4D finger length ratio. Research has indicated that the relative length of the fourth (ring) finger compared to the second (pointing) finger (especially on the right hand) provides a noninvasive rough indicator of how much testosterone one was exposed to prenatally (Manning, 2008). The lower the 2D:4D ratio, the more the exposure. As predicted by ENA theory, most studies cited in Part IV show that prenatal androgen exposure predicts criminality.

Regarding race/ethnicity, the evidence is reflected in Table 7. Blacks appear to have lower ratios than do whites, suggesting they are exposed to higher levels of prenatal testosterone as predicted by ENA theory. However, East Asians seem to have ratios that are even lower than those for blacks, which is contrary to predictions by ENA theory. However, as will be discussed shortly, there may be way to explain these discordant findings on race and exposure to androgens (based on 2D:4D measurement) in terms of varying “sensitivity” to androgens that is linked to race differences in androgen receptors.

9. PSA levels and prostate cancer rates. The levels of prostate-specific antigen (PSA) and the rate at which males are diagnosed with prostate cancer both appear to be promoted by exposure to androgens. However, the timing of this exposure is not confidently known.

Table 8 shows that no research has yet been undertaken to determine if either of these measures are higher among offenders than non-offenders. ENA theory predicts that positive correlations will be found.

The available research on race/ethnic differences in PSA levels and in prostate cancer prevalence have consistently shown that black males suffer much more than do East Asians, with whites being intermediate. However, non-Hispanic white males and Hispanic males do not appear to differ.

10. AR CAG repeat alleles. The number of CAG repeat alleles for the androgen receptor gene helps regulate the effects of androgens on the body. The fewer the number of repeats one has, the more sensitive one is to androgens (Ackerman et al., 2012). Said another way, the fewer the number of CAG repeats in the genetic allele for androgen receptors one inherits, the more androgens will masculinize ones bodily organs (such as the muscles, the genitals, the skeletal structures, and, of course, the brain)

As reflected in Table 8, considerable evidence has accumulated to suggest that AR CAG repeat numbers are inversely correlated with involvement in crime and related behavioral tendencies. This table also shows that East Asians on average have the greatest number of AR CAG repeats while blacks have the fewest. Both Hispanics and non-Hispanic whites appear to be roughly intermediate in this regard.

These racial/ethnic patterns in AR CAG repeats are fairly consistent with predictions by ENA theory regarding androgenic influences on criminal behavior. Specifically, because blacks have the fewest AR CAG repeats, they tend to exhibit the greatest effects of androgen exposure, and, of course, androgen exposure is hypothesized to promote criminality. At the opposite extreme, because East Asians have the greatest number of AR CAG repeats, they should be least inclined toward criminal behavior. However, the tentative finding that Hispanics and non-Hispanic whites are nearly equal in terms of AR CAG repeats does not match with evidence of higher offending rates among Hispanics.

Obviously, more research is needed on AR CAG repeats in relationship to criminality and to race/ethnicity. Besides clarifying the nature of their relationships, it would be informative to determine if bodily organs such as bone, musculature, genitals, and the nervous system are all equally dependent upon these repeats.

5.1.1.1. Overall assessment regarding androgen exposure. While numerous gaps and seeming inconsistencies remain to be addressed, the information highlighted in Table 8 brings one to the following tentative conclusions:

(a) Androgen exposure is associated with criminality in ways predicted by ENA theory.
(b) Androgen exposure generally varies according to race/ethnicity in ways that also conform to predictions by ENA theory. In particular, the racial/ethnic groups with the highest androgen exposures engage in crime at higher rates than the groups with the lowest exposures.
(c) The most glaring exception to the association between race/ethnicity and testosterone levels involves East Asians. Even though their involvement in crime is lower than any other racial/ethnic group yet studied, their testosterone levels also appear to be among the highest of all groups. This seeming theoretical contradiction could be explainable in terms of East Asians having greater average numbers of AR CAG repeats than other racial/ethnic group.

5.1.2. Learning ability

The other key component of ENA theory involves learning ability. Below, evidence is highlighted concerning how criminal behavior and race/ethnicity seem to vary regarding each of the four indicators of learning ability: intelligence scores, academic performance, prevalence of learning disabilities, and brain size.

1. Intelligence scores. Much has been written on the topic of race/ethnic differences in intelligence (see reviews by Jencks & Phillips, 2011; Neisser et al., 1996). Space does not permit coverage of the numerous issues still needing further exploration in this regard. However, as shown in Table 8, some fairly firm conclusions are possible. Regarding involvement in criminal behavior, the evidence is clear that persons with low intelligence are more likely to be involved, and to remain so for more years following puberty than persons with relatively high intelligence. Table 8 also reveals that blacks score lower than non-Hispanic whites, with Hispanics and Native American being roughly intermediate. South Asians and whites are roughly equal, but the highest average scores are attained by East Asians.
2. Academic performance. As shown in Table 8, academic performance, as measured by grade point averages, tends to be lowest among blacks, followed by Hispanics, Native Americans, and Pacific Islanders, all of whom score lower than non-Hispanic whites. The latter in turn typically have lower GPAs than do nearly all Asian groups, particularly those ancestral to East Asia.
3. Prevalence of learning disabilities. Table 8 summarily indicates that being diagnosed with learning disabilities is associated with criminality. In terms of race/ethnic differences, the highest prevalence of

learning disabilities seems to be among blacks, then Native Americans, with non-Hispanic whites and East Asians both being low, but South Asians possibly being lowest of all.

4. Brain size. The fourth indicator of learning ability was brain size, a variable strongly associated with intelligence scores, i.e., ~0.45 (McDaniel, 2005). For this reason, ENA theory predicts that brain size will be inversely correlated with persistent criminality. Currently only one study linking brain size to involvement in criminal behavior has been reported; it revealed a significant inverse relationship for violent crime but not for other types of offenses.

As shown in Table 8, research on the possibility of racial/ethnic variations in brain size is consistent in indicating that blacks have smaller brains on average than whites and that East Asians have the largest brains. Non-Hispanic whites appear to have somewhat larger brains than Hispanics. These patterns conform to predictions of race/ethnic differences in offending by ENA theory but more detailed comparisons are in order.

6. Discussion

The best scientific theories are those that not only explain what is currently known about a targeted phenomenon, but also suggest additional testable hypotheses (Worrall, 1982). ENA theory was designed to account for the widely-documented conclusion that throughout the world most offenders are males in their teens and twenties (Ellis et al., 2009, pp. 11–20). Because the theory specifies that sex and age differences in offending are largely the result of brain exposure to androgens and the brain's ability to learn, one can use the theory to derive additional hypotheses. For example, individuals with high brain exposure to androgens and/or low ability to learn should be more involved in crime than individuals with low androgen levels and/or high learning ability. Therefore, while ENA theory was not specifically designed to deal with race/ethnic differences in criminal behavior, it clearly leads one to suspect such connections. After documenting the nature of race/ethnic differences in offending, the article drew upon published findings regarding relationships between (a) androgen exposure and (b) learning ability, on the one hand, and (c) racial/ethnic variations in criminality, on the other hand.

As shown in Part IV (and then summarized in Part V), considerable evidence indicates that race/ethnic differences in androgen exposure and learning ability at least roughly parallels race/ethnic differences in criminal behavior. Nevertheless, there are some exceptions and many gaps in the information that was located. As research continues to accumulate on how androgen exposure and learning abilities may (or may not) contribute to race/ethnic differences in criminality, the theory's strengths and weaknesses should become more apparent.

At least two other social scientists have suggested that race differences in androgen exposure may be contributing to race differences in criminality (Mazur, 1995; Walsh, 2003). The main differences between their proposals and the one offered here is that the present proposal (a) is very specifically theory-driven and (b) drew upon more contemporary research evidence.

6.1.1. A personal comment

The issues addressed in this article are bound to irritate many people. For this reason, I would like to make a personal remark. As a criminologist/sociologist, I am obviously very interested in better understanding criminal behavior, including how it varies by race and ethnicity. However, as a world citizen, I know some readers will construe my ideas as racist. Such a charge is easily make but all but impossible to refute. And, once such a label has been applied, many hesitate to give serious attention to the scientific merits of the arguments that are offered. So, for what it's worth, I reject the label, and assure anyone who might propose it that they don't know me very well. My only request is that the evidence and argument be consider in the spirit of being proposals worthy of future scientific exploration.

6.1.2. The biosocial nature of race/ethnicity

In the past, one reason for not considering biological variables as part of the explanation for race differences in human behavior has been the view that *race* is a social, not a biological, concept (Helms, 1990; Smedley & Smedley, 2005). I disagree, believing instead that *race* is actually a *biosocial* concept (also see Lin & Kelsey, 2000). In the words of Telles and Paschel (2014, p. 864) “race is both physical and cultural”. From a biosocial perspective, genetic, hormonal, and neurological factors are all likely to be interacting with sociocultural factors to influence racial and ethnic variations in human behavior.

Biosocial theorists have no problem investigating the possibility that racial and ethnic differences in criminality are biologically influenced. For example, they can readily accept the evidence that the number of AR CAG repeats vary according to race (Hsing et al., 2000; Sartor et al., 1999). Those who consider race to be *simply* a social construct must be befuddled by such evidence and at a loss to explain it.

6.1.3. Testosterone's effects on behavior

Some have characterized the effects of testosterone on behavior as making individuals prone to anger and aggression (Harris et al., 1996; Sluyter et al., 2000). While statistical associations between testosterone and anger certainly exist, my reading of the literature is more in line with ideas offered by Birger et al. (2003), Mazur (2005), and Mehta and Prasad (2015). These writers have all converged on the hypothesis that *testosterone drives dominance and status striving* (probably in interaction with low cortisol) to promote aggression when dominance and status appear to be blocked. Put another way, “high testosterone levels encourage dominance-seeking behaviors, which put the individual into situations in which frustration of dominance can occur” (Birger et al., 2003, p. 656). Overall, when status attainment is being frustrated, especially by others acting in a counter-dominant fashion, anger and physical conflicts are common outcomes (for additional arguments to this effect, see Mazur & Booth, 1998; McAndrew, 2009; Mehta & Prasad, 2015). I hypothesize that a great deal of crime, especially violent crime, can be explained in this way and that some of this variation in offending is related to race and ethnicity.

To illustrate the view that brain exposure to testosterone causes dominance-seeking behavior and violence when frustrated by another dominance-seeking actor, imagine two high-androgen males socially interacting with one another. Each one will be motivated to establish and/or maintain his or her own sense of dominance over the other. The chances of violent confrontations are ever present, especially if their abilities to learn nonviolent dominance-maintaining tactics were never very extensive (Felson & Steadman, 1983). Along the same lines, imagine a high-androgen male with low learning ability interacting with a police officer suspecting him of an offense. When he faces the prospects of being arrested, he will interpret the event as a dominance challenge, thereby triggering a violent response (Stewart, Schreck, & Simons, 2006).

6.1.4. Capsulizing ENA theory

In ultimate (i.e., evolutionary) terms, ENA theory asserts that males are more involved in crime than females due to an evolved female preference for mates who are (or at least appear to be) reliable resource provisioners. To accommodate this female preference, males have evolved genetic tendencies to produce relatively high levels of testosterone and other androgens that enhance their drives to acquire resources and maintain status. These male efforts frequently entail victimizing others in ways that can provoke retaliation. Theoretically, the criminal justice system has evolved to help minimize these interpersonal tit-for-tat escalations in violence.

Adolescent behavior reflecting desires for resources and status are

often crude, manifesting themselves in ways that people often resent and seek to suppress (L. Ellis, A.W. Hoskin, 2015). By full adulthood, most individuals with highly androgenized brains (i.e., predominantly males) will have transitioned from crude to refined forms of competitive/victimizing behavior, the latter typically taking the form of normal occupational and financial activities. The theory asserts that learning ability, as well as opportunities to learn forms of competition that minimally victimize others, largely determine how fast individuals transition from crude to refined forms of competitiveness.

In essence, ENA theory contends that males are genetically programmed to comply with female preferences for mates who are competent resource provisioners. As a result, most males exhibit competitive and status-striving behavior that frequently victimize others until they learn to restrict their competiveness to largely lawful acts. The more androgens their brains are exposed to and the more limited their abilities to learn, the higher the probability of their criminality persisting.

6.1.5. Biosocial vs. social environmental explanations of race/ethnic differences in criminality

There is nothing in ENA theory that contradicts environmental explanations for race/ethnic differences in criminal behavior *except* to assert that these explanations are incomplete. In a well-organized review of environmental explanations followed by an analysis of new data, McNulty and Bellair (2003) revealed that fairly similar environmental factors seem to account for high involvement in violent crime by blacks, Hispanics, and Native Americans. The factors identified by these authors mainly had to do with poorly integrated families, "depleted resources", and "weak social bonds (e.g., to school)" (p. 735). Nothing in the present article refutes the relevance of these variables. Rather, the present article implies that poverty, family discord, and loose bonds to school and other social institutions may themselves be influenced by high brain exposure to androgens and to low learning ability (Ellis & Hoskin, 2015b).

Because race and ethnicity are more than a social concept – they are biosocial concepts (as discussed earlier) – races and even ethnic groups are bound to vary in part due to genetics. Some of the relevant genes are likely to influence both androgen levels (Harris, Vernon, & Boomsma, 1998; Hoekstra et al., 2006) and learning ability (Haworth et al., 2009; Oliver & Plomin, 2007). These genetically-influenced racial variations in turn are likely to influence racial and ethnic variations in poverty, family discord, and poor school performance, thereby helping to explain why there are persistent racial variations in these traits. All of these traits in turn can affect the probability of criminal behavior.

6.1.6. Future directions

Most aspects of ENA theory need to be examined in greater detail. In particular, the role of AR CAG repeats in androgenizing the brain and the possibility that it affects racial/ethnic variations in criminality is currently little more than a hypothesis. Furthermore, the role of other hormones interacting with androgens should be carefully explored. For example, the stress hormone cortisol may interact with androgens in ways that could impact aggression and crime (Mehta & Prasad, 2015; Montoya et al., 2012). There is already preliminary evidence that both androgen levels and cortisol levels vary according to race and ethnicity (Kornienko, Clemans, Out, & Granger, 2014; Mazur, 1995; Tremblay et al., 1997). However, these links are complex due in part to multiple environmental experiences throughout life altering the production and release of both sex hormones and stress hormones (Mays, Cochran, & Barnes, 2007; Mazur, 1995; Tyrka et al., 2010).

Regarding the role of learning in affecting criminality, the focus of this article was on intelligence and academic performance. However, other important aspects of learning ability, such as working memory and executive functioning, need to be examined regarding how they too may vary along racial/ethnic lines and how these variations could affect offending behavior. Some promising research along these lines have already been published (Paschall & Fishbein, 2002; Sarsour et al., 2011).

7. Conclusion

This article builds on what has been described as the "intersection of the neural and social sciences" (Noble et al., 2015, p. 774). At this intersection, neurologically-based social science is already spilling over into the discipline of criminology. This spillover calls on criminologists to familiarize themselves with how the brain works in ways that influence human behavior that sometimes runs afoul of criminal statutes. In particular, criminologists should test theory-driven hypotheses that include neurological and hormonal variables. This article reviews evidence bearing on some of the efforts that have been made so far regarding racial/ethnic variations in criminality. The information was organized into five parts.

Part I showed that racial groups can be roughly ordered in terms of their average involvement in criminal behavior as follows:

$$\text{Blacks} > \text{Hispanics} \approx \text{Native Americans} \approx \text{Pacific Islanders} > \text{Whites}_{(NH)} \approx \text{South Asians} > \text{East Asians}.$$

In Part II, a review was provided of past efforts to explain race differences in offending simply in terms of sociocultural variables. While informative, none of these explanations seem to account for most of the racial/ethnic variations in criminality revealed in Part I.

Part III provided an overview of ENA theory, a theory first formulated over a decade ago primarily to explain sex and age variations in offending. At a neurological level, two main variables drive ENA theory: exposure to androgens and learning ability. In essence, as androgen exposure becomes greater and as learning ability becomes diminished, offending probabilities increase.

Part IV sought to determine if ENA theory could help to explain race/ethnic differences in criminality better than mere sociocultural theories. This was done by reviewing the literature regarding ten different indicators of androgen exposure and four different measures of learning ability.

Finally, Part V condensed the information provided in Part IV into a table, thereby providing a birds-eye view of the extent to which current evidence matches theoretical predictions. This condensation indicates that there is currently modest support for the theory, although some inconsistencies and several important gaps in the available evidence are noteworthy. Central to ENA theory is the hypothesis that prenatal testosterone increases the probability of offending, and the evidence based on the 2D:4D finger length measure is still inconclusive. Unfortunately, no study based on amniotic fluid measures of prenatal testosterone has yet been reported.

From a scientific perspective, the most direct challenge to ENA theory's application to race/ethnic differences in criminal behavior comes from evidence that East Asian males often have levels of testosterone that are *higher* than those for white males and possibly even for black males. This is obviously contrary to theoretical expectations since East Asian males exhibit lower crime rates than either of do white and especially black males. Nevertheless, as noted in Parts IV and V, it *may* be possible to explain this seeming inconsistency in terms of racial/ethnic variations in AR CAG repeats. Specifically, the high number of AR CAG repeats in East Asian populations may prevent much of the testosterone they produce from masculinizing muscular, bone, genital, and neurological tissue relative to whites and especially blacks (both of who have fewer AR CAG repeats).

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