

# SEXUAL DEVIANCE OVER THE LIFESPAN

## *Reductions in Deviant Sexual Behavior in the Aging Sex Offender*

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This chapter reviews evidence for reductions in sexually deviant behavior in the aging sex offender. Predominant sentiments in the field and current theoretical accounts hold that sexual deviance is a lifelong individual trait, and that the expression of sexually deviant behavior continues throughout the lifespan. The current chapter considers an alternative view. We argue that although some individual traits and predispositions underlying sexual deviance, such as sexual preferences or antisocial traits, may persist to the end of life, the expression or performance of sexually deviant behavior decreases with age. The chapter is founded on the proposition that the performance of sexual behavior in the human male is determined in large part by blood levels of the male sex hormone testosterone, and that these blood levels decrease with age. In support of this proposition, the present chapter reviews (1) what is known about the male sex hormone testosterone and its relation to sexual drive and behavior, (2) the effects of aging on blood levels of testosterone, (3) the effects of aging on male sexual behavior, (4) the effects of aging on the strength of sexual arousal in sex offenders, and (5) the effects of aging on sexual recidivism in sex offenders released from custody. The chapter also discusses the challenge to the effects of age on recidivism by the proponents of actuarial assessment. Finally, the chapter discusses the implications of age-related decreases in sexually deviant behavior for policy, legislation, and professional practices relating to the assessment, treatment, and management of the aging sex offender.

### **STRONG BELIEF IN LIFELONG SEXUAL DEVIANCE**

There is a strong belief in the field that sexual deviance persists unabated into old age. Three sources of empirical evidence seem to have been most influential in supporting this

view: a highly influential meta-analytic study of recidivism in sex offenders, studies of long-term recidivism in sex offenders, and the development of actuarial instruments that have been demonstrably predictive of recidivism in sex offenders.

In their meta-analytic review of 61 data sets, representing over 23,000 sex offenders, Hanson and Bussière (1998) found that indicators of antisocial behavior and deviant sexual interests were strong predictors of sexual recidivism. Age was identified as only a moderate predictor ( $r = -.13$ ), with younger offenders reoffending at a higher rate. Although the correlation is supportive of the notion that aging leads to reductions in deviant sexual behavior, aging was not identified by the authors as a strong determinant of recidivism in sex offenders.

Two notable studies of recidivism in sex offenders examined recidivism rates 25 years or more after release from custody (Hanson, Steffy, & Gauthier, 1993; Prentky, Lee, Knight, & Cerce, 1997). Although long-term recidivism rates were not found to be high, the mere fact that offenders were still offending so long after release seems to support the notion that sexual deviance persists into the later years. If one considers that the average sex offender is released sometime after age 35, recidivism 25 years later would indicate persistence of sexual deviance to age 60, at least.

Finally, perhaps the most significant advance in the assessment of the sex offender during the past 20 years has been the development and promulgation of actuarial instruments that are demonstrably predictive of recidivism among adult male sexual offenders (Doren, 2002; Hanson, 1997; Quinsey, Harris, Rice, & Cormier, 1998). Hanson and Morton-Bourgon (2004) identified the five most commonly used actuarial instruments as the Violence Risk Appraisal Guide (VRAG; Quinsey et al., 1998), the Sex Offender Risk Appraisal Guide (SORAG; Quinsey et al., 1998), the Rapid Risk Assessment of Sexual Offense Recidivism (RRASOR; Hanson, 1997), the Static-99 (Hanson & Thornton, 1999), and the Minnesota Sex Offender Screening Tool—Revised (MnSOST-R; Epperson et al., 1998). These actuarial instruments consist primarily of items that code “static” or unchanging features of the offender being assessed, reinforcing the idea that offenders cannot or do not change in recidivism risk over time. Three of these instruments code age at assessment as a specific risk factor. For example, on the RRASOR and Static-99, the age of the offender is coded as being younger or older than 25 years of age, with the younger age signifying higher risk. A similar item in the MnSOST-R codes high risk as younger than age 30. These item codings imply that risk for sexual recidivism does not change after the ages of 25–30. The VRAG and SORAG code age at index offense. This is a static age item, since this item response will not change with maturity. In the manuals describing the proper use of these actuarial instruments, there is no advice given concerning adjustments in risk for an older offender. Therefore, from the actuarial perspective, recidivism risk in sex offenders does not decrease due to the aging of the sex offender.

The strong belief in the persistence of sexual deviance into old age is supported by theoretical accounts that posit long-term sexual deviance. Harris and Rice (2003) have articulated this position clearly by stating that “the preponderance of scientific evidence supports the idea that the majority of variance in violent criminal conduct (including sexual aggression) can be attributed to genetically and physiologically based enduring traits that, once initiated, exhibit lifelong persistence under conditions so far observed” (p. 208).

Despite this evidence and these arguments, there are good reasons to question the notion that sexually motivated behaviors of any type—paraphilic or conventional—will

continue unabated throughout a man's middle years and into old age. Such an expectation is at variance with the known facts of human endocrinology and male sexuality.

### THE ROLE OF THE MALE SEX HORMONE TESTOSTERONE

Mammalian gonads and adrenals secrete several male sex hormones called "androgens." All are steroid hormones produced primarily in the Leydig cells in the male testes, although some small amounts of these hormones are produced in the adrenals in both males and females. Testosterone is the most potent and abundant androgen (Seidman, 2005). Close to 98% of testosterone molecules are protein-bound, with approximately one-third of these weakly bound to albumin and the remainder strongly bound to sex-hormone-binding globulin (SHBG). Because the testosterone molecules that are bound with SHBG cannot bind with receptor cells, this component of testosterone has no behavioral effect. Only the non-SHBG-bound testosterone is biologically active ("bioavailable"), including free testosterone and testosterone that is loosely bound to albumin (Seidman, 2005). Free testosterone diffuses into target cells, where it is converted to dihydrotestosterone and estradiol. Testosterone and dihydrotestosterone bind to androgen receptor cells mediating the effects on sexual behavior (Seidman, 2005).

The male sex hormone testosterone plays a critical role in the production of male sexual behavior—a role first identified by the pioneering work of behavioral endocrinologist Frank Beach (1948). When males are castrated, blood levels of testosterone decline rapidly (Coyotupa, Parlow, & Kovacic, 1973). Sexual behavior also declines, but more gradually—sometimes over weeks and months (Beach, 1970). These effects of castration are seen in birds (Hutchinson, 1974), subprimate mammals including rats and guinea pigs (Beach, 1971), nonhuman primates (Phoenix, 1978), and humans (Heim & Hursch, 1979). In the human male (Heim & Hursch, 1979), the majority of castrates show an immediate decline in sexual behavior, while most of the remainder show a gradual decline over weeks or months. A small percentage of human castrates, approximately 10%, show no decline in sexual behavior (Heim & Hursch, 1979).

When men exhibit low levels of total testosterone in their blood (below 300 ng/dl) due to the malfunctioning of their hypothalamic–pituitary–gonadal axis, they are referred to as "hypogonadal." Hypogonadism is characterized by a loss of libido and a loss of both sleep-associated and spontaneous erections (Anderson, Bancroft, & Wu, 1992). Davidson, Kwan, and Greenleaf (1982) utilized a within-subject design to study the effects of injected doses of exogenous testosterone on sexual behavior in six hypogonadal men. These patients received (1) 100 mg of testosterone, (2) 400 mg of testosterone, or (3) placebo. Each patient received each treatment with a gap of 6 weeks between treatments, and the order of treatment was varied among patients to control for treatment order effects. Results indicated that injections of testosterone increased plasma testosterone levels. The effect was temporary (with peak effects 7 days after injection) and dose-dependent (with larger doses producing larger increases in blood levels). The hypogonadal men kept daily diaries of their sexual behavior and penile erections. The largest behavioral effects of testosterone injections were reported 1 week after injection, corresponding to the time of peak effect on plasma testosterone level. Dose-dependent effects of testosterone injections were observed for total erections, nocturnal erections, coital attempts, masturbation, and orgasm, with larger doses producing larger increases in sexual behavior (Davidson et al., 1982).

Testosterone is necessary or at least important in maintaining libido. Increasing plasma androgens at puberty is correlated with the onset of nocturnal emissions, masturbation, dating, and infatuation (Kemper, 1990). The level of bioavailable testosterone is correlated with sexual thoughts (Meston & Frohlich, 2000). Males with an early onset of androgen secretion develop an early interest in sexuality and erotic fantasies (Feder, 1984). A significant relationship between serum testosterone levels and libido has been found in the following populations: normal men (Anderson et al., 1992; Bagatell, Heiman, Rivier, & Bremner, 1994), normal adolescent boys (Udry, Billy, Morris, Groff, & Raj, 1985), men in or past middle age (Davidson et al., 1983; Tsitouras, Martin, & Harman, 1982), men complaining of loss of sexual interest (O'Carroll & Bancroft, 1984), men with erectile dysfunction (Schiavi, White, Mandeli, & Levine, 1997), and hypogonadal men (Davidson, Camargo, & Smith, 1979; Kwan, Greenleaf, Mann, Crapo, & Davidson, 1983; Luisi & Franchi, 1980; O'Carroll, Shapiro, & Bancroft, 1985). This relationship between blood levels of testosterone and libido will come as no surprise to professionals working in the area of sexual deviance. Medical and pharmacotherapeutic interventions designed to manage sexually deviant behavior have largely employed antiandrogenic medications designed to reduce blood levels of testosterone, libido, and ultimately (of course) sexually deviant behavior (e.g., Bradford & Fedoroff, 2006).

### THE EFFECTS OF AGING ON TESTOSTERONE AND SEXUAL BEHAVIOR

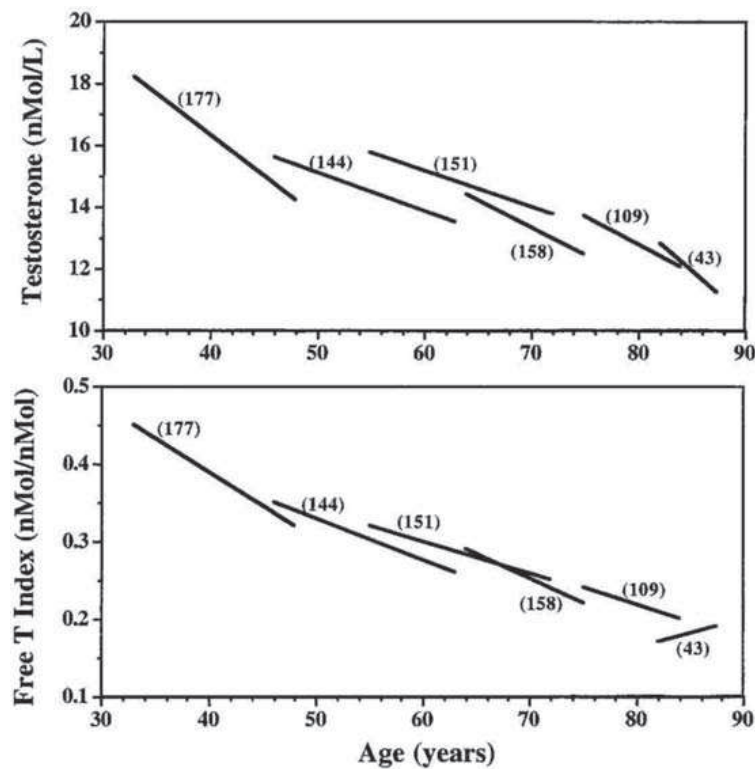
Older men do not experience a sudden cessation of gonadal function and hormone secretion, as occurs in women during menopause. Instead, there is a progressive reduction in male hypothalamic–pituitary–gonadal axis function with age. Testosterone levels decline through both central (pituitary) and peripheral (testicular) mechanisms, and there is an age-related loss of circadian rhythm (Seidman, 2005; Swerdloff & Wang, 1993). Numerous studies have established that levels of both total and bioavailable testosterone peak in early adulthood and thereafter decrease with age through the remainder of the lifespan (e.g., Baker et al., 1976; Denti et al., 2000; Harman, Metter, Tobin, Pearson, & Blackman, 2001; Jankowska, Rogucka, Medras, & Welon, 2000; Ooi et al., 1998; Vermeulen, Goemaere, & Kaufman, 1999).

Gray, Berlin, McKinlay, and Longcope (1991) conducted a meta-analysis of 88 published studies examining the effects of aging on testosterone, and the studies reported conflicting results. In cross-study comparisons, certain research design characteristics (e.g., time of day of blood sampling) and various sample characteristics (e.g., volunteers vs. patients as subjects) were related to both mean testosterone level and the slope of the age–testosterone function. Blood levels of testosterone exhibited a circadian rhythm, with higher levels being recorded in the morning. Studies that did not record blood levels specifically in the morning produced lower blood levels. For subgroups of subjects that did not exclude men with significant illness, the mean testosterone levels were low and did not decline with age. In contrast, studies that recorded blood levels in the morning using subgroups that included only healthy subjects had higher overall testosterone levels and showed a decline of testosterone with age. Therefore, when study methodology has employed appropriate controls for extraneous factors, the relationship between aging and testosterone blood levels indicate a significant effect of age.

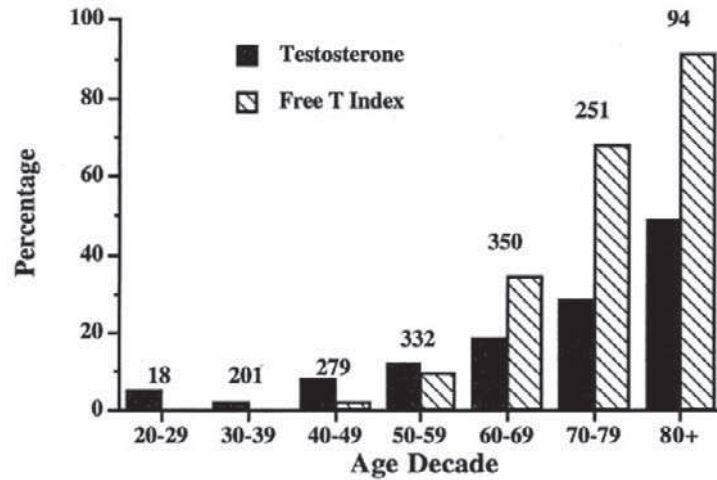
In the Baltimore Longitudinal Study of Aging (Harman et al., 2001)—a study employing both a longitudinal design (the same subjects were measured at different ages)

and a cross-sectional design (different cohorts were tested at different ages)—levels of both total and bioavailable testosterone were seen to decrease in a linear function from age 30 to age 90. Figure 3.1 presents the longitudinal effects of aging on total testosterone and free testosterone (free T index). As can be seen, blood levels of testosterone decrease in a linear fashion from young adulthood to old age.

In the same study, significant numbers of older men could be diagnosed as hypogonadal, in the sense that their blood levels of testosterone had declined below the diagnostic criteria (Harman et al., 2001). Figure 3.2 presents the proportion of different-age samples who met diagnostic criteria for hypogonadism, based on both total and bioavailable testosterone (free T index). As can be seen, the proportion increased significantly with age. Testosterone deficiency in elderly men could be considered a normal aging phenomenon. However, somewhat arbitrarily, age-adjusted norms were not used, and modern medicine regards this state as pathological. This condition in older men has been referred to as “andropause,” and it is considered to be the male equivalent of menopause in women.



**FIGURE 3.1.** Longitudinal effects of aging on total testosterone and free testosterone. Linear segment plots are shown for men on at least two visits. Each linear segment has a slope equal to the mean of the individual longitudinal slopes and is centered over the medial age for each cohort of men. Numbers in parentheses represent the number of men in each cohort. Segments show significant downward progression at every age, with no significant change in slopes over the entire age range. From Harman, Metter, Tobin, Pearson, and Blackman (2001, p. 727). Copyright 2001 by the Endocrine Society. Reprinted by permission.



**FIGURE 3.2.** Hypogonadism in aging men. Bar height indicates the percentage of men in each 10-year interval with at least one testosterone value in the hypogonadal range: total T < 300 ng/dl (shaded bars) or T/SHGB (free T index) < 5.74 (striped bars). The numbers above each pair of bars indicate the number of men studied in the corresponding decade. The percentage of men with hypogonadism increases progressively from the 20s to the 80s by either criterion. More men have hypogonadism by free T index than by total T after age 50, and there is a progressively greater difference with increasing age between the two criteria. From Harman, Metter, Tobin, Pearson, and Blackman (2001, p. 727). Copyright 2001 by the Endocrine Society. Reprinted by permission.

As menopause does, andropause comprises a constellation of sexual and nonsexual health effects. Age-related reduction in the production of androgens is associated with numerous nonsexual physiological changes, including osteoporosis, decreases in muscle mass, decreases in muscle strength, increases in body fat, and psychological effects including depression and irritability (Seidman, 2005). It might be argued that these nonsexual health effects are simply the effects of aging and are unrelated to deficiencies in the functioning of the hypothalamic–pituitary–gonadal axis. However, these same physiological abnormalities are seen in younger men with hypogonadism, and they can be ameliorated somewhat with hormone (exogenous testosterone) replacement therapy in men of all ages (Harman, 2003). Therefore, these age-related symptoms of ill health are seen to be part of the andropause syndrome.

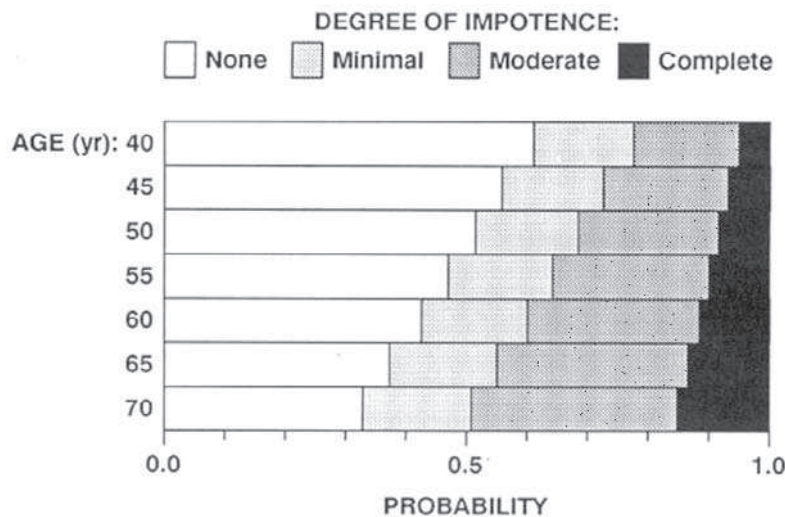
Based on the well-established relationship described above between testosterone and libido, one would therefore expect that the normal decline in testosterone levels with age would be accompanied by a concomitant decrease in libido. The mechanism whereby the decline in testosterone with age influences sexual behavior is not well understood. Although age-related reductions in testosterone availability have been well established, less is known about the effects of age on testosterone utilization. A few authors have suggested that testosterone receptor sites may become less sensitive with age, so that the threshold concentration of testosterone necessary to maintain libido may increase with age (e.g., Baker & Hudson, 1983; Schiavi, 1999, pp. 52–53; Tsitouras et al., 1982). Nevertheless, either or both processes would imply a trend toward decreasing sexual interest or arousability in older men.

Not surprisingly, studies of human sexuality and aging indicate a general decline in the frequency of male sexual behavior and an increase in the number of problems associated with sexual relations with age, including a reduction in the number and quality of

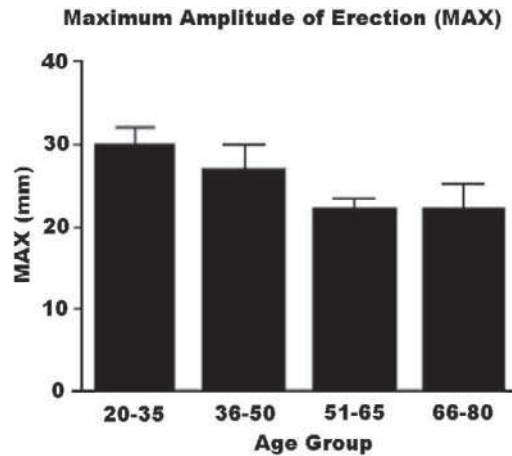
erections (Kaiser et al., 1988; Morley et al., 2000). For example, Feldman, Goldstein, Hatzichristou, Krane, and McKinlay (1994) studied erectile function over different age cohorts and reported that while the prevalence of minimal erectile difficulties remained constant (< 20%) from age 40 to age 80, the prevalence of moderate and complete erectile dysfunction increased so that by age 60, the majority of research participants reported at least minimal erectile dysfunction. These results are presented in Figure 3.3.

The Global Study of Sexual Attitudes and Behaviors (Laumann et al., 2005) was an international survey of various aspects of sex and relationships among adults ages 40–80 years. This survey contacted 13,882 women and 13,618 men worldwide and achieved a response rate of almost 20%. Data were collected through a combination of telephone interview and self-report questionnaire. The authors note that the estimated prevalence of sexual problems reported in this survey was comparable to previously published rates. Several factors were found to be consistently related to the likelihood of sexual problems. In men, lack of interest in sex, erectile difficulties, and inability to achieve orgasm were more prevalent in older men; the older the respondents, the more prevalent these problems became. The survey concluded that sexual problems tend to be more closely associated with physical health and aging among men than among women.

Rowland, Greenleaf, Dorfman, and Davidson (1993) examined the sexual arousal and behavior of 39 healthy sexually functional men ranging in age from 21 to 82. These authors recorded erectile responses to visual erotic stimulation. Figures 3.4 and 3.5 present mean magnitude of erectile responses (Figure 3.4) and mean latency to maximum response (Figure 3.5) for four age cohorts. As can be seen, the magnitude of erectile responses decreased and their latency increased with advanced age. These authors also collected data on self-reported sexual activity and functioning in these same subjects. Results



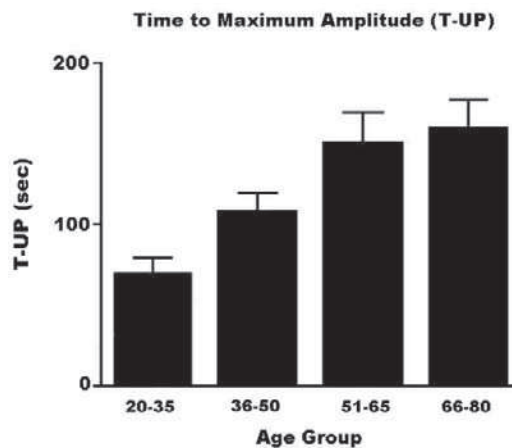
**FIGURE 3.3.** Prevalence in percentage of men of various ages from 40 to 79 years reporting minimal, moderate, or complete erectile dysfunction. Prevalence of minimal erectile dysfunction remains constant across the age spectrum, whereas prevalence of moderate and complete erectile dysfunction increases progressively with age. From Feldman, Goldstein, Hatzichristou, Krane, and McKinlay (1994, p. 56). Copyright 1994 by Lippincott Williams & Wilkins. Reprinted by permission.



**FIGURE 3.4.** Mean maximum amplitude (mm) of erectile responses recorded via circumferential penile plethysmography in response to presentations of erotic stimulation. Means are plotted for four age cohorts. Standard deviations are indicated by the hatch marks. From Rowland, Greenleaf, Dorfman, and Davidson (1993, p. 552). Copyright 1993 by Kluwer Academic/Plenum Publishers. Reprinted by permission.

indicated significant age-related decreases in the frequency of sexual activity, including intercourse, orgasm, and masturbation. Perhaps surprisingly, participants in this study reported no reduction in sexual satisfaction with age.

It might be argued that these reductions in sexual arousability with age are due to psychological effects. Perhaps the erotic stimuli are found to be less attractive or stimulating among older participants. However, Karacan, Salis, Thornby, and Williams (1976) re-



**FIGURE 3.5.** Mean latency to maximum amplitude (in seconds) of erectile responses recorded via circumferential penile plethysmography in response to presentations of erotic stimulation. Means are plotted for four age cohorts. Standard deviations are indicated by the hatch marks. From Rowland, Greenleaf, Dorfman, and Davidson (1993, p. 552). Copyright 1993 by Kluwer Academic/Plenum Publishers. Reprinted by permission.

corded nocturnal penile tumescence in the course of a study of sleep. Plotting the frequency, duration, and rigidity of nocturnal erections over the age of the research participants, these authors reported that the participants' nocturnal erections peaked at age 13 and declined slowly with age. These data would suggest that the observed aging effects on sexual arousal are due to biological, not psychological, mechanisms.

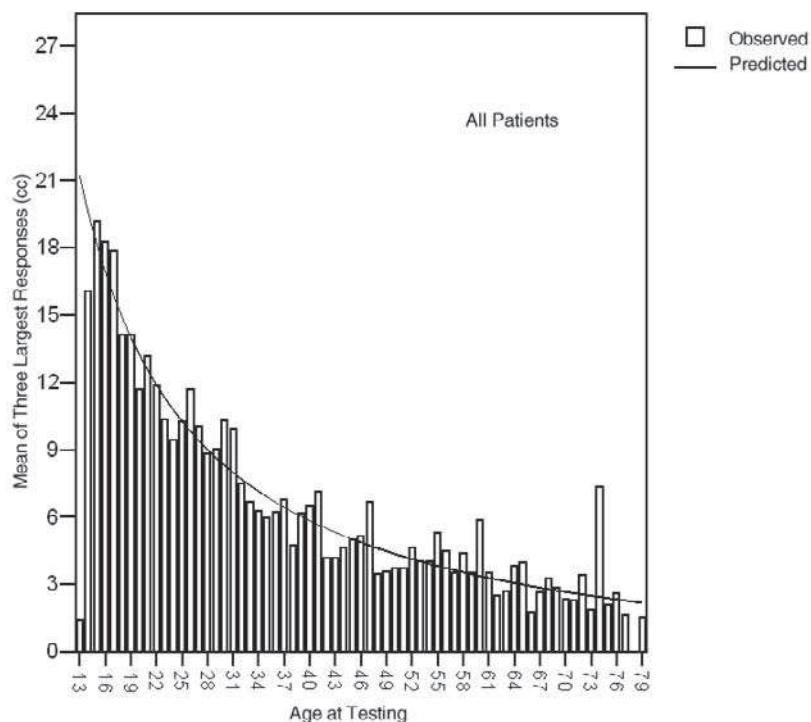
### **THE EFFECTS OF AGING ON SEXUAL AROUSAL IN THE SEX OFFENDER**

The current literature supports the notion that sexual arousal decreases with age in sex offenders. Hall (1992) examined the relationship between age and erectile responses measured by circumferential penile plethysmography in 169 inpatient adult male sex offenders ranging from 20 to 66 years of age. Age accounted for a significant proportion of the variance in arousal, and arousability was inversely related to age. This reduction in sex offender arousability seems to begin at an early age. Kaemingk, Koselka, Becker, and Kaplan (1995) examined the relationship between age and erectile responses measured by circumferential penile plethysmography in 104 adolescent sex offenders ranging in age from 13 to 17 years. Somewhat surprisingly (given the restricted age range), age accounted for a significant proportion of the variance in arousal, with the younger adolescents showing erectile responses to a greater number of stimulus presentations, and demonstrating a greater mean percentage of full erection scores across stimulus presentations.

Using volumetric penile plethysmography, we (Blanchard & Barbaree, 2005) examined the strength of arousal as a function of age among more than 1,400 sex offenders referred for phallometric testing to the Kurt Freund Phallometric Laboratory. The dependent measure of penile response was a standard quantity in the laboratory, the "output index" or OI (Freund, 1967). This is the average of the three greatest responses to any stimulus category except "neutral," where penile response is expressed in cubic centimeters (cc) of blood volume increase from the start of a trial. As measured by our laboratory equipment, full erection for the average patient corresponds to a blood volume increase of 20–25 cc. Figure 3.6 shows the relation between penile response and age. The bars represent the mean observed blood volume increase for patients of every age from 13 to 79. The amplitude of penile response declined steeply from almost full erection in adolescence to lower levels at about age 30; it continued to decline after that, but at a lower rate. It is clear from the observed data that the decrease would be better described by a curved than by a straight line. The line of best fit was plotted as the reciprocal of age.

### **THE EFFECTS OF AGING ON SEXUAL RECIDIVISM IN SEX OFFENDERS**

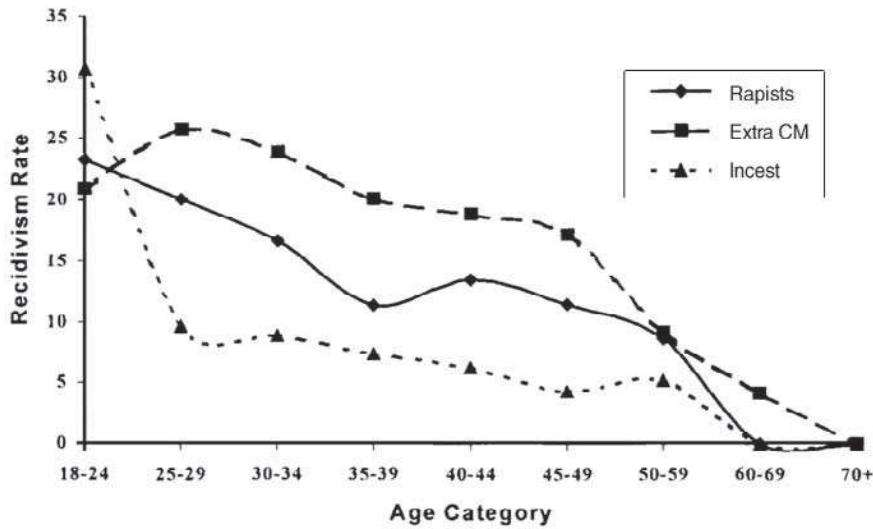
Castration was evaluated as a treatment for sex offenders in studies conducted in Europe in the 1960s and 1970s. These studies were quite consistent in their finding that castration of adult sex offenders causes a substantial reduction in the rate of recidivism (Freund, 1980; Heim & Hirsch, 1979). The mechanism for this decrease in sexual recidivism is thought to be reductions in the male sex hormone testosterone brought about by castration (Freund, 1980). Therefore, given the reductions in blood levels of testosterone that occur with age, we would predict that recidivism should decrease in sex offenders with aging.



**FIGURE 3.6.** Phallometric response of all patients as a function of age. The bars represent the mean observed blood volume increase (i.e., mean OI) for patients of every age. The curved line is the predicted blood volume increase for patients of every age, as computed by linear regression. From Blanchard and Barbaree (2005, p. 448). Copyright 2005 by Kluwer Academic/Plenum Publishers. Reprinted by permission.

Hanson (2002) has examined the effects of age at release on rates of sexual recidivism in a large sample of sex offenders. The results indicated that these sex offenders' risk for recidivism decreased with age at release. According to Hanson's description, the patterns of decline differed among rapists, child molesters, and incest offenders. In incest offenders, from an initial peak at ages 18–24, there was a rapid decline to below 10% at ages 25–29, with a continuing gradual decline to age 60, after which there were no incidents of reoffense in these samples. In nonfamilial child molesters, the rate of recidivism peaked when offenders were released at ages 25–29, then gradually declined to release at age 50, at which time the rate of decline increased markedly to release at age 70. Rapists showed a gradual decline in rates of recidivism from 18 years to 60 years. Recidivism rates are plotted for the different age cohorts in Figure 3.7.

When we examine the plotted rates of recidivism over age at release, there are two issues that require mention and clarification—one minor, the other more important. The minor issue is that the age intervals on the abscissa are unequal, and therefore the plots in Figure 3.7 are distorted somewhat. The more important issue concerns the fact that the statistically significant differences between groups in pattern of decline identified by Hanson (2002) are entirely due to performance in the youngest age group (18–24 years) of extrafamilial child molesters. Although only linear trends were evident in the rapists and incest offenders, logistic regression identified a significant curvilinear component to

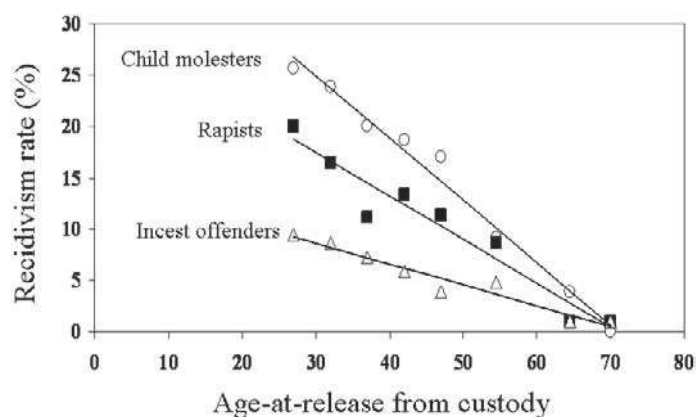


**FIGURE 3.7.** Recidivism rates (%) plotted for nine age cohorts from 18 to 70+ years of age. Separate plots are provided for three separate offender groups: incest offenders, extrafamilial child molesters, and rapists. From Hanson (2002, p. 1054). Copyright 2002 by Sage Publications. Reprinted by permission.

the trend of recidivism over age at release in extrafamilial child molesters (Hanson, 2002). Examination of Figure 3.7 indicates that recidivism rates increased from the youngest (18–24) to the next youngest (24–29) age group in these offenders. Thereafter, from age 25 on, rates of recidivism among the extrafamilial child molesters decreased in an approximately linear fashion. Our focus in the present chapter is on changes in behavior that occur with aging. For these reasons, and for the sake of simplicity, our discussion of the Hanson data will exclude consideration of the youngest (18–24 years) age group.

In a previous paper, we (Barbaree, Blanchard, & Langton, 2003) replotted the Hanson (2002) data for offenders released between the ages of 25 and 70+ (see Figure 3.8). The recidivism rate for each offender subgroup (child molesters, rapists, incest offenders) was plotted over the midpoint of the appropriate age interval. For each group, a regression equation was calculated, and a regression line was plotted. Also, we calculated correlations between observed and predicted rates of recidivism and found that all correlations were above .97, indicating that the regression lines were good fits to the observed data. A number of aspects of these data become very clear from this replotting. In the Hanson data set, (1) recidivism in sex offenders declined from the late 20s; (2) the decline can be best characterized as a linear decline over age at release in all offender subgroups; (3) the decline ended at age 70, and at that age the estimated recidivism rate was zero for all subgroups; and (4) in their youth, sex offender subgroups differed in the rate at which they recidivated, with child molesters showing the highest rate and incest offenders showing the lowest rate.

We (Barbaree et al., 2003) then examined sexual recidivism in a sample of sex offenders ( $N = 477$ ) released from a Canadian federal penitentiary and followed for an average of almost 5 years. It was hypothesized that sexual recidivism would decrease in a linear fashion as a function of age at release. The regression constants derived from Hanson (2002) as described above provided a basis for making a precise prediction of the

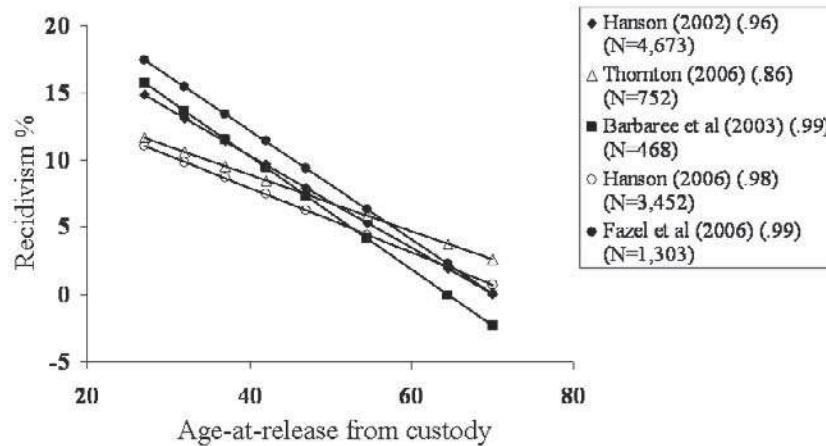


**FIGURE 3.8.** Recidivism rates (%) replotted as a function of age at release from custody in child molesters, rapists, and incest offenders age 25 and older. From Barbaree, Blanchard, and Langton (2003, p. 64). Copyright 2003 by the New York Academy of Sciences. Reprinted by permission.

values of the regression constants in our study. We divided our sample into age cohorts (21–30; 31–40; 41–50; 51+) and calculated a failure (recidivism) rate for each age cohort. Calculating a regression equation describing the reductions in recidivism over age for our data yielded almost exactly the same slope and intercept (regression constants) as we had derived from the data reported by Hanson.

Between the publication of our 2003 paper and the time of our writing this chapter, three additional studies of the relations between recidivism and age in sex offenders have been published. Fazel, Sjöstedt, Långström, and Grann (2006) followed all adult male sex offenders released from prison in Sweden between 1993 and 1997 ( $N = 1,303$ ) and recorded criminal convictions for an average of 8.9 years. They divided their sample into age cohorts (< 25; 25–39; 40–54; 55+). Recidivism rates decreased significantly in older cohorts.<sup>1</sup> Thornton (2006) followed a large nationally representative sample ( $N = 752$ ) of sex offenders for a period of 10 years after their release from prison in the United Kingdom. Dividing his sample by age cohort (< 18; 18–24; 25–39; 40–59; 60+), he found that the rate of sexual recidivism declined generally with age. Finally, Hanson (2006) followed a very large sample ( $N = 3,425$ , compiled over eight separate samples) of sex offenders released from prison in North America and the United Kingdom. Hanson calculated 5-year recidivism rates for different age cohorts (18–24; 25–39; 40–49; 50–59; 60+).

In order to compare the results of these five studies (Barbaree et al., 2003; Fazel et al., 2006;<sup>1</sup> Hanson, 2002, 2006; Thornton, 2006) and to highlight similarities among the studies' findings, we plotted their results in a comparable format on one graph. Since the focus in this chapter is on declines in sexual behavior after the peak in testosterone levels in the mid-20s, we eliminated data for age groups below age 25. Since these studies used age cohorts with different class intervals, we calculated a regression equation separately for each study. To estimate the goodness of fit of the obtained straight line, we calculated a correlation between the expected and observed values obtained in each study. Since each of these studies used a different follow-up period, we adjusted the recidivism rates proportionally to correct to a 5-year follow-up period. We then plotted the adjusted regression lines for these five studies in one figure, presented here as Figure 3.9. These regression equations were all good representations of the data obtained from each study,



**FIGURE 3.9.** Replotted recidivism as a function of age at release from custody corrected to 5 years' time at risk (data from Barbaree et al., 2003; Fazel et al., 2006; Hanson, 2002, 2006; Thornton, 2006) (total  $N = 8,879$ ). Correlations indicate goodness of fit of the regression line to observed recidivism over age in these studies.

with correlations ranging from .86 to .99. These studies showed a very consistent effect of age at release across studies—namely, a linear decline in recidivism from age 25 to age 70.

Based on the data reviewed above, it would be reasonable to conclude that when sex offenders are released from custody at different ages, they show age-related decreases in recidivism. The best description of the age function is a gradual linear decrease in recidivism rates from age 25 to age 70, at which point the estimated recidivism rate is near zero. This age function is similar to that described earlier in this chapter for blood levels of testosterone, for sexual arousal in normal men, and for sexual arousal in sex offenders. Additionally, these reductions in sexual recidivism are very similar to reductions in nonsexual recidivism (both violent and nonviolent) among nonsexual criminals (Hirschi & Gottfredson, 1983; Sampson & Laub, 2003, 2005).

With the support of these empirical data, the present chapter proposes the addition of a new category of risk factor for sexual recidivism—namely, maturation. In the current discussion of risk factors, static risk factors are those characteristics of offenders related to recidivism that do not change over time. Dynamic risk factors are those that may change over time. In contrast to both of these factors, maturation is a risk factor that exhibits changes over time; however, unlike dynamic risk, the change is predictable and inexorable, and once maturational changes occur, they are not reversed under normal circumstances.

### THE ACTUARIAL CHALLENGE TO THE EFFECTS OF AGE ON RECIDIVISM

Despite the wealth of data presented above indicating changes in recidivism risk with age, there is no general or widespread acceptance of maturation as an important risk factor among the proponents or developers of actuarial risk instruments. Specifically, there is no acceptance of the necessity for adjustments to estimates of risk in the older sex offender.

Hanson (2006) has suggested that adjustments in actuarial risk estimates might be appropriate after age 60, but discourages any adjustments before that age. Other proponents and developers of actuarial instruments argue against any adjustments being made for older sex offenders. Their argument is based on their challenge to the validity of age-related decreases in recidivism (Harris & Rice, 2007).

The proponents of actuarial assessment argue that the apparent effects of age on sexual recidivism are actually due to actuarial risk. Their argument has a number of interrelated components.<sup>2</sup> First, they argue that the evidence for the age effect is based on cross-sectional rather than longitudinal data. They point out that age at release is confounded with actuarial risk. Offenders who are released at an older age are also lower in actuarial risk. Second, they argue that age at first offense (a static age variable) is a more powerfully predictive age variable than age at release (a maturational age variable). They postulate that high-risk offenders will, on a probabilistic basis, offend earlier in life, and that these same high-risk offenders will persist in offending into old age. Finally, the proponents of actuarial assessment argue that once the effects of actuarial risk (scores on the actuarial instrument and age at first offense) have been taken into account, age at release adds nothing to the prediction equation. They argue that because age at release does not enter the prediction equation, there is no significant aging effect to be recognized, and therefore no adjustments to risk estimates for the older sex offender. We deal with each of these arguments below.

### THE CONFOUND BETWEEN AGE AT RELEASE AND ACTUARIAL RISK

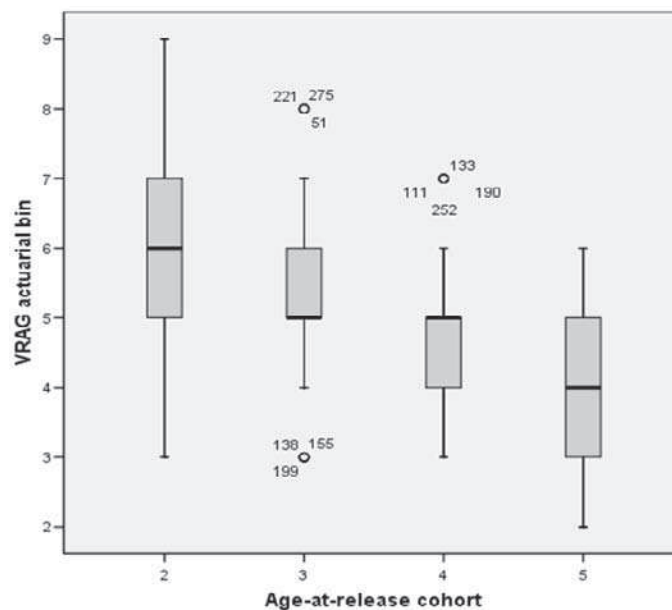
In our data set (see Barbaree, Langton, & Peacock, 2006), age at release was correlated with total actuarial scores on the VRAG, SORAG, Static-99, and MnSOST-R,  $r$ 's (309) =  $-.498$ ,  $-.380$ ,  $-.233$ , and  $-.159$ , respectively, all  $p$ 's < .01. Therefore, the proponents of actuarial assessment are correct in saying that age at release and actuarial risk are confounded. Offenders who are older at release have lower actuarial scores. However, the problem with confounds is that they can be legitimately argued either way. If lower actuarial scores among older offenders can be used to explain lower recidivism among older offenders, then older age at release can be used to explain lower rates of recidivism among offenders with lower actuarial scores.

In an effort to illustrate how age at release might be used to explain actuarial risk, we (Barbaree, Langton, & Blanchard, 2007) compared mean age at release between and among subgroups of sex offenders who were assigned different scores on items contained in the VRAG and SORAG. We found 9 items out of the 12 and 14 items on the VRAG and SORAG, respectively, for which mean age at release was significantly different between or among groups with different item responses, and where the older age group(s) were assigned the lower risk score. These items were as follows: lived with both biological parents to age 16, elementary school maladjustment, alcohol problems, marital status, criminal history score nonviolent, sex offenses only against girls < 14 years of age, failure on prior conditional release, age at index offense, and Psychopathy Checklist—Revised score.

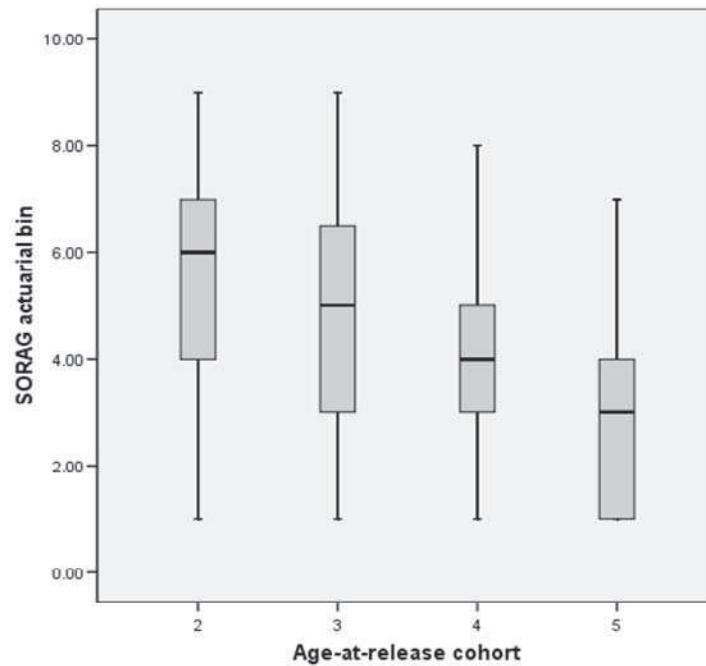
It is not clear why these offender subgroups defined by their item responses would have different mean ages at release. A possible explanation is based on the actuarial method itself—the statistical method used in the development of actuarial instruments. In pure actuarial risk assessment, the items have no meaning. They are selected and

weighted based on their empirical relationship with outcome. If the studies we reviewed above are correct in concluding that recidivism decreases with age, we should not be surprised that the VRAG/SORAG item responses showed a statistical relationship with age at release. In other words, age at release, actuarial item responses, and recidivism are intercorrelated. Age at release is imbedded in the actuarial instruments—not just in items whose nominal meaning has some bearing on some measure of age, but also in items whose meaning has no obvious conceptual relationship to age.

Since these nine items constitute the majority of actuarial items on the VRAG and SORAG, it would be expected that the combination of these items in a total score would reflect an important relationship with age at release. Figures 3.10 and 3.11 present box plots (SPSS version 14) for the VRAG and SORAG actuarial bin scores, respectively, with actuarial bin scores plotted over age-at-release cohorts (21–30; 31–40; 41–50; 51+). For both the VRAG and SORAG, mean actuarial bin scores decreased over age in a linear fashion. For the youngest cohort, the median actuarial bin was at or near the 6th bin, whereas for the oldest cohort, the median actuarial bin was at or near the 4th and 3rd bins for the VRAG and SORAG, respectively. Analyses of trend (using an unweighted means correction for unequal  $N$ ) indicated that for both the VRAG,  $F_{\text{linear}}(1, 307) = 92.666, p < .001$ , and the SORAG,  $F_{\text{linear}}(1, 307) = 53.539, p < .001$ , the linear function of actuarial bin over age at release was highly significant. Tests of the deviations from linearity were not significant (both  $F$ 's  $< 1.00$ ). If recidivism decreases with age, as indicated



**FIGURE 3.10.** Box plots of the mean VRAG bin scores plotted as a function of age-at-release cohort. The box depicts scores from the 25th to the 75th percentiles, and the horizontal line through the box represents the median score. The “whiskers” represent the range of values in the sample from lowest to highest. Outliers are excluded from the box plot but indicated on the figure. Age-at-release cohorts 2 through 5 were released from custody at 21–30, 31–40, 41–50, and 51+ years of age, respectively.



**FIGURE 3.11.** Box plots of the mean SORAG bin scores plotted as a function of age-at-release cohort. The box depicts scores from the 25th to the 75th percentiles, and the horizontal line through the box represents the median score. The “whiskers” represent the range of values in the sample from lowest to highest. Outliers are excluded from the box plot but indicated on the figure. Age-at-release cohorts 2 through 5 were released from custody at 21–30, 31–40, 41–50, and 51+ years of age, respectively.

in the review above, lower recidivism among offenders who obtain a low actuarial score may be due to their older age at release, not their actuarial risk.

To examine the role of age at release in the prediction of recidivism by the VRAG and SORAG, we (Barbaree et al., 2007) regressed item scores and actuarial bin scores over age at release and saved the residual scores as “age-corrected” actuarial scores. The residual scores had no correlation with age at release. We then compared the age-corrected actuarial scores with the original actuarial scores in their ability to predict recidivism via receiver operating characteristic analysis. For both actuarial items and actuarial bin scores, the original scores were significantly superior in predictive ability to the comparable age-corrected scores. These analyses indicated that age at release has contributed to the ability of the VRAG and SORAG to predict recidivism in sex offenders, at least in our data set.

In the empirical process of selecting items for the VRAG and SORAG, the developers of these actuarial instruments have unwittingly selected items that reflect distinctive characteristics of younger offenders. By virtue of the fact that younger offenders are more likely to reoffend sexually, for reasons reviewed above (including higher blood levels of testosterone), the items that identified younger members of the sample are related to recidivism. By selecting numerous such items and combining them in an actuarial instrument, the total score is predictive of recidivism. In this way, the predictive accuracy of an actuarial instrument can be said to borrow some of its predictive power from the relationship between aging and recidivism in the sex offender.

For Harris and Rice (2007) and the anonymous reviewers of our manuscripts, the fact that age at release does not add to the predictive equation after actuarial scores and age at first offense are entered into the equation leads them to the conclusion that there is no effect of aging on recidivism in sex offenders. We disagree and feel that their logic is flawed. As we have demonstrated (Barbaree et al., 2007), the effects of aging are imbedded in their actuarial instruments. When actuarial scores are entered into the equation first, the effects of aging already contribute to the predictive accuracy of their instruments. In this way, the subsequent “test” of age at release is preempted. The variance due to age at release is at least partly used up in the prior test of the actuarial score. Testing the ability of actuarial scores to predict recidivism is not an appropriate or fair statistical method of evaluating the effects of aging on recidivism.

Ultimately, the actuarial argument seems unconvincing to us. By arguing that there is no reduction in recidivism with age, the proponents and developers of the actuarial instruments are arguing that sexual recidivism is the only index of sexual behavior in the human male that does not decrease with age. (As an aside, they are also arguing that it is the only criminal behavior in men that does not decrease with age. Due to space limitations in this chapter, we have not reviewed the substantial literature on the effects of age on criminal recidivism.) While we have to agree that there is a confound between age at release and actuarial risk, we feel that the more convincing interpretation of the data in all its aspects is that sexual recidivism does decrease with age, and that actuarial risk cannot account for all of the decline observed in the published studies.

### **AGE AT FIRST OFFENSE VERSUS AGE AT RELEASE**

Harris and Rice (2007) have investigated the contribution of age at release to prediction of recidivism in the samples of violent offenders that were used in the development of the VRAG and SORAG. They have concluded that although age at release predicted violent recidivism, it did not do so as well as age at first offense and other actuarial variables. They have concluded that age at release provides no useful additional information concerning recidivism risk.

This argument seems unconvincing to us. These two indices of age are entirely and profoundly different. Age at first offense is a static factor. Once an offender has committed a criminal offense, his age at first offense is determined for the remainder of his life. This index of age bears no relationship whatever to the aging processes we have reviewed and described earlier in this chapter. In contrast, age at release is a maturational variable, in that it reflects the level of maturity of the individual when he is released from custody. This variable is determined by many factors, including age at index offense, sentence length or length of incarceration, parole decision making, and so on. But importantly, age at release changes over time, and with it the level of risk posed by the offender.

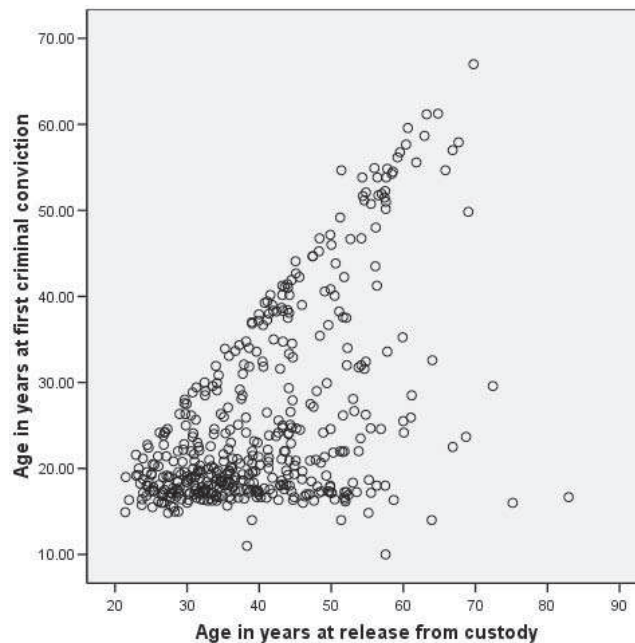
Their argument is unconvincing to us, partly because we do not understand why the power of age at first offense to predict recidivism takes anything away from the significance of age at release as a predictor. In other words, whether or not age at first offense is a significant predictor of recidivism seems to us to be totally independent of the predictive ability of age at release. Aging can have its effect on recidivism whether or not higher-risk youthful offenders are convicted of crimes at a young age.

The only possible argument we can think of that could be made in respect of this issue is that age at first offense and age at release are correlated in these samples, and there-

fore, from a statistical perspective, their collinearity requires us to disentangle their effects. This correlation might be the result of a large proportion of sex offender samples consisting of first-time offenders for whom age at release and age at first conviction are separated by sentence length. The actuarial argument might be that the age at release is not an important predictor and is only a statistical predictor due to its correlation with what Harris and Rice regard as the primary static actuarial age predictor, age at first offense.

To investigate the relationship between age at first offense and age at release, we conducted a further supplemental analysis of our data set (Barbaree et al., 2003, 2007). We constructed a scatter plot showing the statistical relationship between these two age variables. It is presented in Figure 3.12. As will be obvious on inspection of this figure, there is no meaningful relationship between these two indices of age, at least in our data set. Although there is a mathematical correlation between the two age variables,  $r(466) = .55$ ,  $p < .001$ , the correlation is a statistical artifact. Since it is impossible for offenders to be released at an age younger than the age of their first offense, the scatter plot is empty above the diagonal, forcing a mathematical correlation. Taking this artifact into account while carefully inspecting the scatter plot leads the reasonable observer to the conclusion that there is no meaningful relationship between these variables.

Harris and Rice (2007) have reported that age at first offense predicts recidivism. They argue further that the effects of age at release are insignificant or small when age at release is entered into the equation after age at first offense. These findings are used by these authors to argue for the primacy of the static actuarial age variable. Our counter-argument is based on the spurious correlation between the two age variables depicted in



**FIGURE 3.12.** Scatter plot plotting the age at first conviction in months over the age at release in years in a sample ( $N = 468$ ) of sex offenders. The plot is empty above the diagonal because an offender cannot be released from custody at an age younger than the age of his first conviction.

Figure 3.12. When age at first offense is entered first, it preempts a fair test of age at release. At least some of the variance due to age at release has already been used up in the test of the static age variable. It seems to us that in efforts to make a fair assessment of the aging effect, the relative strength of predictive clout between the two age variables is irrelevant. We would argue that if there is any effect of age at release, after reasonable controls for actuarial risk (i.e., free of the confound with age at release), then we must conclude that aging has a significant effect on recidivism in the sex offender.

### **THE NEED TO ADJUST ESTIMATES OF RISK IN THE OLDER OFFENDER**

Although the actuarial test developers have unwittingly capitalized on the relationship between age at release and recidivism, they deny the effects of aging when applying the instrument score to the evaluation of risk in the individual sex offender. Because actuarial items are predominantly static items, a high actuarial score obtained by a young man will follow him unchanged for the rest of his life, despite repeated scoring by various evaluators. So even while the offender's risk is decreasing as part of the maturational process, the actuarial instrument confirms his static risk for the remainder of his life. Ironically, the validity of the actuarial instrument that asserts his ongoing risk is based partly on the effects of age on recidivism (Barbaree et al., 2007).

In arguing against the need for adjustments in the older offender, the proponents of actuarial assessment state that the existing actuarial instruments weight age more or less appropriately, and that no further adjustments for aging are required. We feel that this represents a significant error in logic. Actuarial items code characteristics of offenders at a particular point in time, usually the date of the assessment as release from custody is imminent (or, in research, the date of the file from which the items are coded). The test of the predictive accuracy of the actuarial test is based on an analysis of relations between the data recorded at the time of the assessment and later recidivism. The maturational variable (age at release) contributes to the prediction equation, either directly or indirectly, through contrasts between different offenders released at different ages. These between-offender contrasts contribute to the predictive power of the actuarial instruments, because offenders released at a younger age recidivate at a higher rate. However, age as a maturational risk factor is a within-offender contrast. Because the instruments were developed without any explicit recognition of maturation as a potential risk factor, and because the resulting items sets do not include any items that reflect changes in risk that occur after the age of 30, the actuarial instruments are not sensitive to the within-offender changes that occur with age. In order to accommodate age as a maturational risk factor, an assessment instrument must make adjustments to estimates of risk as the offender ages.

### **CONSTRUCTING A PREDICTION EQUATION THAT IS SENSITIVE TO THE EFFECTS OF AGING**

Our method of testing the effects of age controlled for actuarial risk and for the spurious correlation between age at first offense and age at release. Our method recognized that age at release is embedded in actuarial risk scores. We (Barbaree et al., 2005a) used stepwise Cox regression to test the effects of age at release. In the first step, to control for ac-

tuarial risk, we entered five actuarial factors that had been calculated in an earlier factor analysis of static actuarial items (Barbaree et al., 2006). These factors were Antisocial Behavior, Persistence, Child Sexual Abuse, Detached Predatory Behavior, and Male Victim(s). These factors were found to be orthogonal (uncorrelated) to a sixth factor, Young and Single, and therefore were thought to be relatively independent of age at release. In the second step, we entered age at first offense. Finally, in the last step, we entered age at release. For both sexual and violent recidivism, we found that age at release was a significant predictor of recidivism, even after controlling for actuarial risk and age at first offense. Hazard ratios derived from the Cox regression indicated that recidivism risk decreased by approximately 5% each year.

### SUMMARY AND FUTURE DIRECTIONS

The present chapter has been founded on the premise that the male sex hormone testosterone plays a significant role in the production of sexual behavior in the human male, and that age-related reductions in blood levels of the hormone result in concomitant reductions in numerous indices of male sexual behavior in the aging man. Age-dependent reductions have been observed in libido, coitus, erectile frequency and quality, and frequency of masturbation. Age-dependent increases have been observed in sexual problems in the aging man, including erectile dysfunction and lack of interest in sexual relations. Sex offenders are not immune to these effects of aging. Specific age-related effects have been observed in sex offenders' level or amplitude of sexual arousal and in their recidivism. Both sexual arousal and recidivism decrease in sex offenders with age.

These effects of aging are not widely accepted or recognized in the field. Importantly, current methods of assessment of sex offenders do not account for the effects of aging. In other words, no adjustments or allowances are typically made in estimates of recidivism risk in the current best-practice approaches to assessment. Current assessment methods are based on the development and validation of actuarial instruments. In the studies that have led to percentage estimates of recidivism over 5, 10, and 15 years after release, offenders in the research samples were released from custody in their mid-30s. Currently recommended practice is to apply these percentages to sex offenders at all ages, even elderly offenders. Methods of adjusting these percentages have been recommended. Rich Wollert (2006) has described a method of adjusting estimates of risk for older offenders, based on Hanson's (2002) data. Nevertheless, proponents of actuarial assessment have not recognized or accepted this method of adjustment, and such adjustments are not recommended by them.

Professional standards guiding the use of psychological tests warn against the use of tests if such use may be discriminatory on the basis of age, race, culture, or other factors. If the person being tested is substantially different from the individuals who made up the standardization sample for the test, the test should be applied with extreme caution. The current chapter makes the point that direct application of current actuarial instruments to elderly sex offenders is potentially discriminatory.

The implications of the aging effects reviewed in this chapter are profound. Current policy and legislation in most Western jurisdictions target sex offenders for civil commitment or long-term incapacitation when the offenders are in their middle years. Current practice is to continue the detention of many of these offenders on into old age. Incarceration of these offenders is grossly expensive and seems unjustified if risk is generally

lower in the aged sex offender. One solution is the revamping of the current risk assessment methodologies to accommodate reductions in the performance of sexually deviant behavior in the older sex offender.

## NOTES

1. The Swedish data showed exceptionally low rates of sexual recidivism, much lower than those reported for the U.K. and North American data here or in previous reports. Allowing for possible differences between Sweden and other jurisdictions in the way sexual crimes are charged, prosecuted, and tried, including possible differences in plea bargaining, we chose to use the Swedish data reflecting “any violent” reconviction—a statistic that included all sexual offenses. These percentages were very similar to those reported in the U.K. and North American data for sexual recidivism.
2. The actuarial position is presented in part in Harris and Rice (2007) and has been articulated by numerous anonymous reviewers of our manuscripts on the effects of aging on sexual recidivism submitted for publication (Barbaree, Langton, & Peacock, 2005a, 2005b), communicated to one of us (Howard E. Barbaree) by Karl Hanson (associate editor of *Sexual Abuse: A Journal of Research and Treatment*). We are unable to identify the individual sources of all these arguments, and we therefore simply refer in this chapter to the general actuarial position or argument.

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