

A Bayesian Approach to Investigating
Age-at-Death of Subadults in a Forensic Context

by

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Estimating age at death is among the first steps in the identification of an unknown individual. For subadults, dental formation stage remains the most accurate aging indicator due to minimal environmental impact. Even the most accurate method, however, is affected by “mimicry bias,” where the age profile of the target population “mimics” the age profile of the reference population used to develop the age estimation method. Bayesian statistics and transition analysis can control for this bias in archaeological and forensic samples through calculating the average age of transition from one development phase to another, followed by estimating the probability that someone of a certain age has a given phase of development based on a sample of individuals of known age. Here, Bayesian-derived age ranges related to the dental formation phases of Moorrees *et al.* (1963) were generated using a sample of 201 children of known age (Orthodontics Case File System, Maxwell Museum of Anthropology). In this study, I present age ranges at an exact 50% probability for each tooth at various stages of development. These ranges can be used in forensic cases wishing to control for “mimicry bias” in assessments that rely on age estimation via dental formation.

A Bayesian Approach to Investigating Age-at-Death of Subadults in a Forensic Context

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Introduction

Forensic anthropologists and bioarchaeologists have developed many techniques for estimating the age of skeletal remains. Age-at-death provides insight into mortality patterns in addition to assisting in positive identification of medicolegal cases. Age estimation methods have been developed from categorizing age-related variation in skeletal collections of known age and sex. Methods of age estimation development however suffer from one intrinsic bias: the collection may include more individuals of certain ages than others. As a result, more information on skeletal variation related to age can be observed for certain age categories, which eventually influences which age category an individual of unknown age would be included. This has been termed the “mimicry bias,” for it results in a target population of unknown age mimicking the age distribution of the reference sample (Bocquet-Appel and Masset 1982).

The goal of this thesis is to develop unbiased age ranges for dental formation stages that can be used by forensic anthropologists on subadult individuals. These age ranges are created through the development of ages-at-transition from one dental formation stage to another based on a reference sample of 201 children between the ages of five to twelve from the Maxwell Museum of Anthropology at the University of New Mexico. These transition ages are then recalculated using Bayes’ theorem, which takes into consideration a prior probability of death by age.

This thesis will provide a chapter-by-chapter analysis beginning with an outline of how anthropologists have sought to confront the age mimicry bias, and how Bayesian statistics has become a recognizable tool to control for this bias. Next, I will describe the sample utilized in this study, along with the aging techniques, and statistical methods applied to this sample. I discuss transition analysis and Bayes’ theorem and how they work together to create the

unbiased age ranges in this study. In the results chapter, I review the figures and tables that are the products of this research, and explain the importance and relevance of the age ranges that Bayes' theorem generates. Finally, I discuss the limitations and recommendations for the next step in future research.

Background

One key determination in skeletal biology is age estimation of a skeletonized individual. In medicolegal circumstances, age-at-death is vital to creation of a biological profile and victim identification. In archaeological contexts, age-at-death provides a reflection of population mortality, which can be useful for understanding the demographic dynamics of past populations.

The determination of age-at-death of subadults in particular provides one perspective on ancient population health and fertility, mortality, and migration rates (Chamberlain 2009, Hoppa and Vaupel 2002). Age-at-death distributions for children in past populations rely on skeletal indicators of growth and development. Childhood deaths can be thought of as a reflection of overall population health (Saunders and Barrans 1999). The study of these subadult deaths in the archaeological record provides information on population health, because these individuals experience the greatest risk of death (Reidpath and Allotey 2003). Demographic profiles also can lend information regarding household and family composition, economic organization, social problems, as well as political structure (Kolb 1985, Ubelaker 1992, Waldron 1994, Gowland and Chamberlain 2005, Chamberlain 2006). Developing a reliable estimation of age for the individuals in the population is one of the first steps in filling in the blanks that lead to knowledge of past life-ways.

Skeletal biologists have developed a variety of methods for estimating the age of subadults. In general, the skeletal age of prepubescent children most accurately reflects their chronological age (i.e., actual time since birth), with age estimations becoming progressively more inaccurate through adult age (Stewart and Trotter 1954, Anderson, *et al.* 2009).

Degeneration of an adult occurs at a much less regular rate than the growth and development of a subadult. For this reason, the age ranges that have been calculated through various aging

methods of adults have wider ranges, and are less accurate and reliable. According to Crews (1993) and Zwaan (1999), the growth and development of a juvenile is predetermined more strictly by evolution and genetics than the degeneration processes of an adult. Therefore, skeletal growth characteristics of subadults, such as long-bone lengths (Hoffman 1979), epiphyseal fusion (Scheuer and Black 2000) and dental calcification and eruption (Moorrees *et al.* 1963a, 1963b, Ubelaker 1979, and Schour and Massler 1941) provide more precise and accurate estimations of age than many adult aging methods based on skeletal degeneration (Garvin *et al.* 2012). Tooth formation has been seen to be the most preferred dental aging process because it is less affected by environmental factors, and strongly controlled by genetic factors (Glasstone 1938, Paynter and Grainger 1961, Ubelaker 1999, Smith 1991, Scheuer and Black 2000; Moorrees *et al.* 1963, Fanning 1962, Sapoka and Demirjian 1971), and it covers a much longer growth period than either long bone length or epiphyseal union. Tooth formation provides a continuous series of developmental changes from before birth to about 20 years of age (Demirjian 1986), Moorrees *et al.* (1963a,b) because it includes both permanent and deciduous teeth (Saunders *et al.* 1993). One issue, however, is that specific diseases, such as hypopituitarism and syphilis, can modify the rate of dental development (Bauer 1994, Ubelaker 1999). Thus, this needs to be taken into account if these are prominent diseases in the population being studied.

Schour and Massler first developed a schedule of tooth eruption in 1941 by creating a visual chart of “snapshots” of dental development within particular age ranges (Figure 1). However, this method was hampered by a small sample of diseased children (Al Qahtan, et al. 2010). Also, this method, which requires the researcher to match their sample tooth to a chart

with a defined age, is subject to inter and intra observer error since it does not provide examples of the gradation between stages (Fanning 1961).

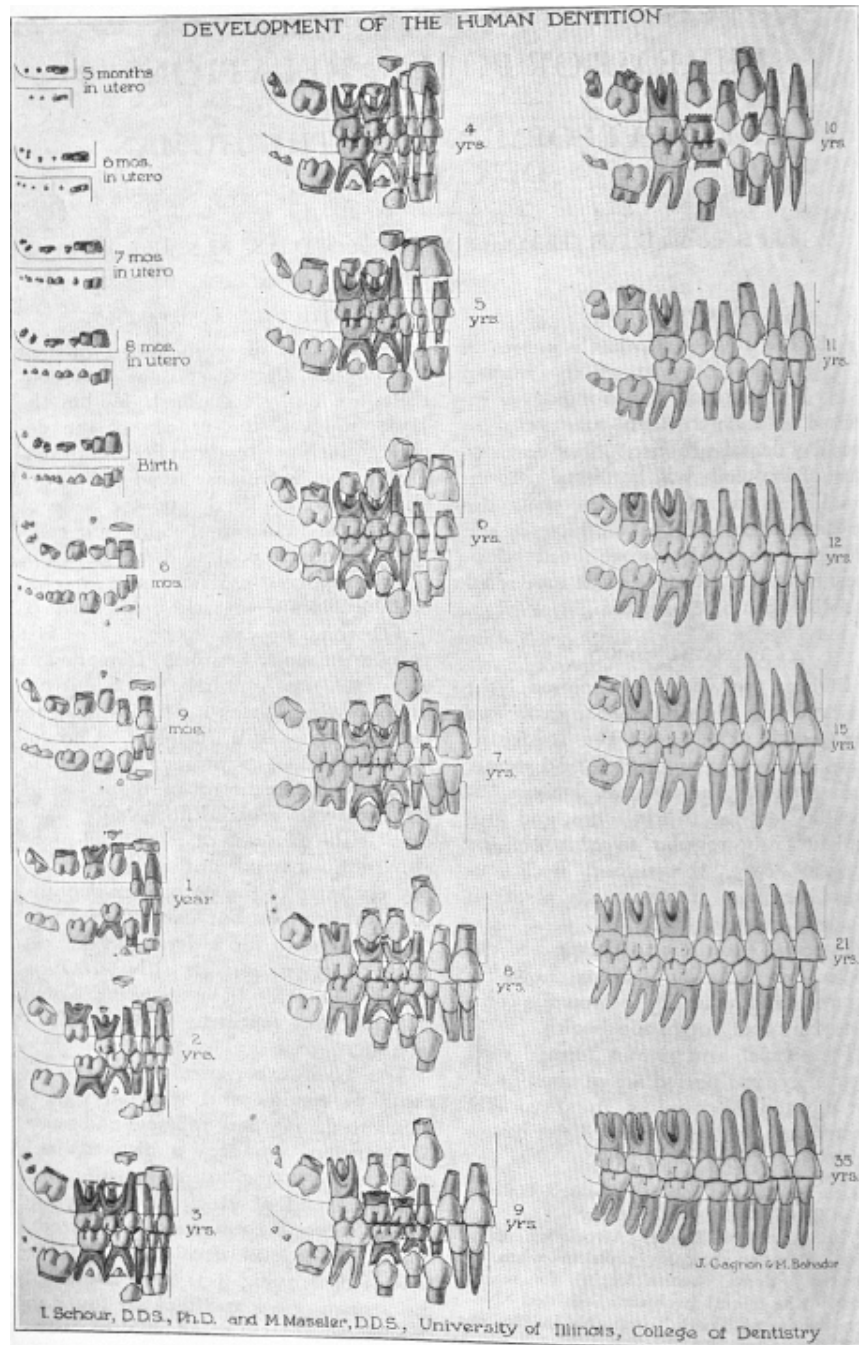
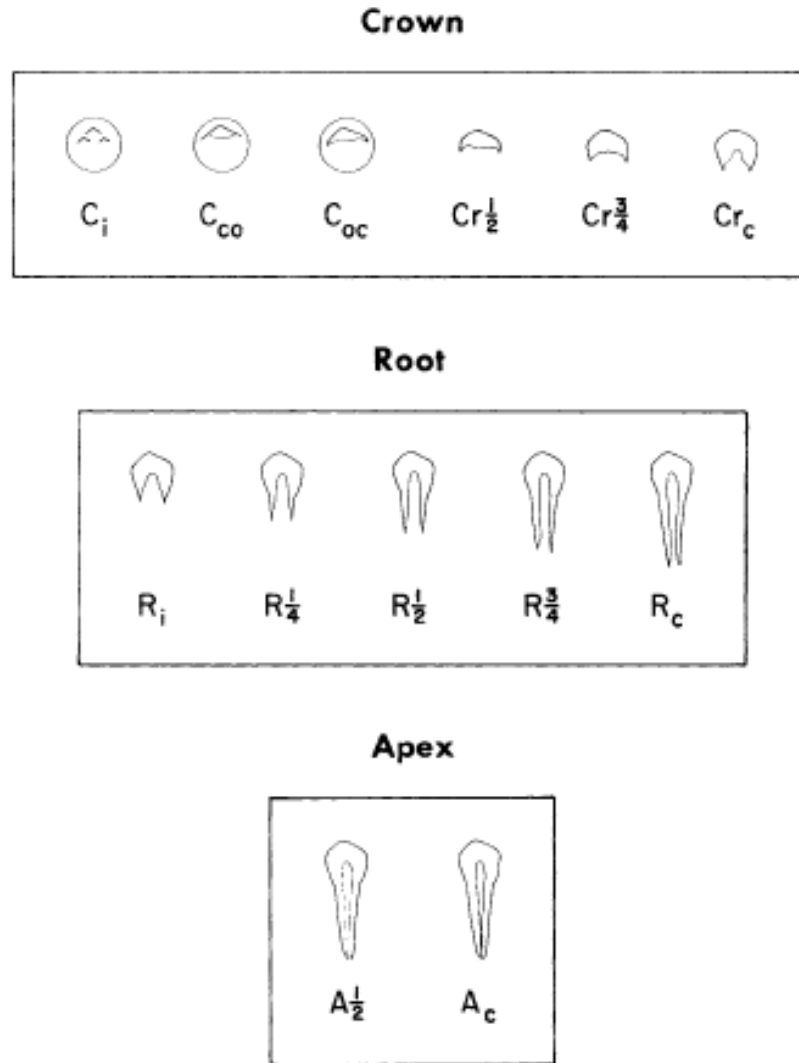


Figure 1: Dental development chart developed by Schour and Massler (1941).

As a result, Moorrees *et al.* (1963a,b) developed an aging technique that breaks down development of a tooth into fourteen different stages to better reflect the continuous process of dental formation. Moorrees and colleagues observed the development of the permanent maxillary and mandibular central and lateral incisors in intraoral radiographs of 48 males and 51 females. This sample consists of children from Boston, which was assembled from the Forsyth Dental Infirmary under the direction of Dr. Harold C. Stuart. In addition, samples of permanent mandibular posterior teeth were derived from the Fels collection, which consists of radiographs of 136 males and 110 females from Ohio. The Fels data were used to obtain the chronology of formation and resorption of the deciduous mandibular molars and canines. Moorrees, *et al.* (1963a) determined dental development by inspecting these radiographs and assigning ratings according to consecutive stages. The distinctions between stages are based on crown and root development, which is displayed in Figures 2 and 3.



TOOTH-FORMATION STAGES AND
THEIR CODED SYMBOLS

Stage	Coded Symbol
Initial cusp formation	C_i
Coalescence of cusps	C_{co}
Cusp outline complete	C_{oc}
Crown $\frac{1}{2}$ complete	$Cr_{\frac{1}{2}}$
Crown $\frac{3}{4}$ complete	$Cr_{\frac{3}{4}}$
Crown complete	Cr_c
Initial root formation	R_i
Initial cleft formation	Cl_i
Root length $\frac{1}{4}$	$R_{\frac{1}{4}}$
Root length $\frac{1}{2}$	$R_{\frac{1}{2}}$
Root length $\frac{3}{4}$	$R_{\frac{3}{4}}$
Root length complete	R_c
Apex $\frac{1}{2}$ closed	$A_{\frac{1}{2}}$
Apical closure complete	A_c

Figure 2: Dental formation chart for single rooted permanent teeth provided by Moorrees, et al., (1963b) with corresponding stages.

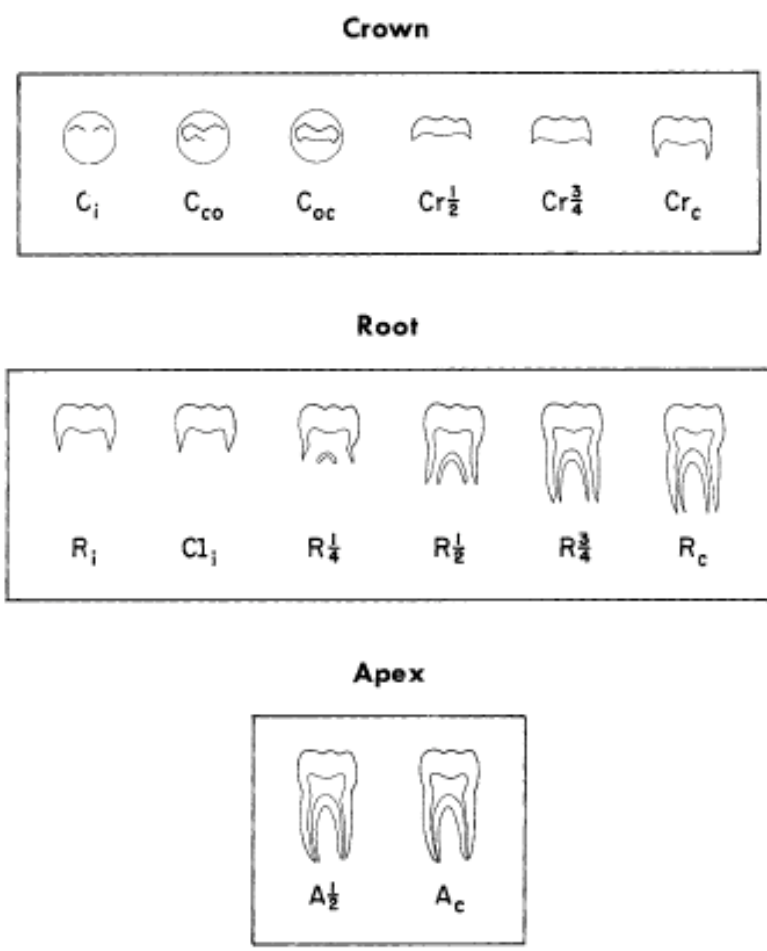


Figure 3: Dental formation chart of permanent mandibular molars developed by Moorrees, *et al.*, 1963b).

Employing these charts for determining dental maturation of a child presents a variety of problems. First of all, Moorrees, *et al.*, (1963b) explains that this method may not be applicable to individuals from populations other than North America, due to possible variation in rates of development. Also, the experience level of the researcher in determining the stage of formation can cause problems (Moorrees, *et al.* 1963b). Another major issue with this method is the difficulty in determining the span of time between transitioning from one stage of development to the next (Moorrees, *et al.* 1963b). Finally, researchers have argued about the amount of dental development that can be observed in radiographs (Fanning 1961).

Demirjian, *et al.*, (1973) attempted to resolve some of the issues of the Moorrees *et al.* method. They analyzed panoramic radiographs of 1446 boys and 1482 girls to develop a dental aging method based on criteria beyond crown and root formation. Rather than focus on changes in root length, their stage descriptions are based on changes such as enamel formation, form of the pulp chamber, and development of root canal. In this system, all mandibular dentition, with the exception of the third molar, are classified into eight stages from the first appearance of calcified points to the closure of the root apex. Although this method has proven to be reliable, it also contains several issues. First of all, the method is only applicable to individuals from 3 to 17 years of age. Also, since this method relies on the stage of development of several teeth at any time, missing teeth can cause difficulty when using this method. Finally, as with many dental development studies, radiographs will be necessary in order to observe and tooth formation stages, as dental development occurs within the jaw.

Subsequent testing of both Demirjian and Moorrees' methods have uncovered issues with accuracy, particularly when applying them to other reference samples of known age from varied populations. Maber *et al.* (2006) and Olze *et al.* (2010) identify Demirjian *et al.*'s method as the most accurate and easiest method for development assessment. Through their assessment, Maber *et al.* (2006) tested accuracy using a Student's *t-test* and found the Demirjian method to overestimate age with a mean accuracy (in years) of 0.25 for males, and 0.23 for females, a standard error of 0.04 for each, with a standard deviation of 0.84 for both in a sample of 491 males and 455 females. However, Demirjian *et al.*'s (1973) technique has been found to overestimate age of individuals in other reference samples (Hägg and Mattson 1986, Poyry *et al.* 1986, Staaf *et al.* 1991, Mörnstad *et al.* 1995, Caro and Contreras 2001, Nykänen *et al.* 1998).

According to Foti, *et al.* (2003), Demirjian *et al.*'s (1973) method was found to overestimate age by almost one year.

Phillips and Van Wyk Kotze (2009) explored the accuracy of the Moorrees, *et al.* (1963) method in comparison to Demirjian, *et al.* (1973) aging method. The study analyzed 914 pantomographic radiographs of children between the ages of three and sixteen. This sample consisted of 442 females and 472 males that received routine dental treatment at the Dental Faculty at Tygerberg in South Africa. They also studied the pantomographic radiographs of 91 Black (Zulu) children with an age range of seven to fifteen. This sample consisted of 44 females and 47 males. Finally, the third sample of children contained 82 females and 71 males for a total of 153 Indian children from Durban, ranging in age from six to sixteen. The age of each child was estimated using the method described by Moorrees, *et al.*, (1963), followed by the Demirjian, *et al.*, (1973) method, and subsequently compared to the known chronological age of the child. As a result, this study showed that in the Tynderberg sample, the Moorrees dental stages underestimated ages in 89.2% of the sample on average by 0.91 years, while the Demirjian method overestimated the ages on average by 0.89 years in 85.7% of the sample. The Moorrees method also underestimated the ages of the Indian children, as 93.7% of the sample lies below the chronological age, and underestimates the Zulu population in 96.7% of the sample. On the other hand, the Demirjian method over estimates age in both the Zulu and Indian populations, in 90% and 79.2% of the sample respectively.

The primary issue facing paleodemographers, however, is that even the most accurate technique will still be impacted by intrinsic sampling bias. As noted above, skeletal biologists develop aging techniques from osteological samples with known, or documented, age-at-death. These samples often are not reflective of the living population, and rarely have equal

distributions of individuals by age. This can result from how these samples are created (e.g., individuals of a certain age may be less likely to donate their body to science or have indigent status that, in the past, would increase their chance of ending up in a study sample) and from the fact that age is a profound predictor of likelihood of death. For example, Demirjian and colleagues' sample of individuals used for their dental development study has a distinctive unequal distribution by age (Figure 4). When assessing human skeletal variation in a sample with an unequal age distribution, researchers can better observe the range of variation in better-represented age categories of, for example, dental development. An example of this is displayed Figure 5, provided by Liversidge, *et al.* (2010). A wide range of bias is visible in Figure 5, where bias in years plotted against reported age for their sample, displayed as N25b. The dotted line in this graph represents a zero bias, when estimated age coincides with actual age.

As a result of this unequal age distribution, skeletal biologists observing a particular dental development stage in an individual of unknown age will more likely associate that stage with one of the adjacent higher-populated age categories in the reference sample, because it is more likely to display that stage due to sampling issues. In reality, individuals in the less-populated age categories could theoretically have that stage of development as well (but because of the smaller sample size, not all development variants that can appear at that stage are displayed in that sample). In the end, the estimated chronological age of the unknown sample becomes biased towards the distribution of the reference population, causing the target population to “mimic” the reference population in terms of age distribution (Bocquet-Appel and Masset 1982, 1985, 1996, Sattenspiel and Harpending 1983, Van Gerven and Armelagos 1983, Buikstra and Konigsberg 1985, Masset and Parzys 1985, Bocquet-Appel 1986, Greene *et al.* 1986, Witter-Backofen 1987, Horowitz *et al.* 1988, Konigsberg and Frankenberg 1992, 2001).

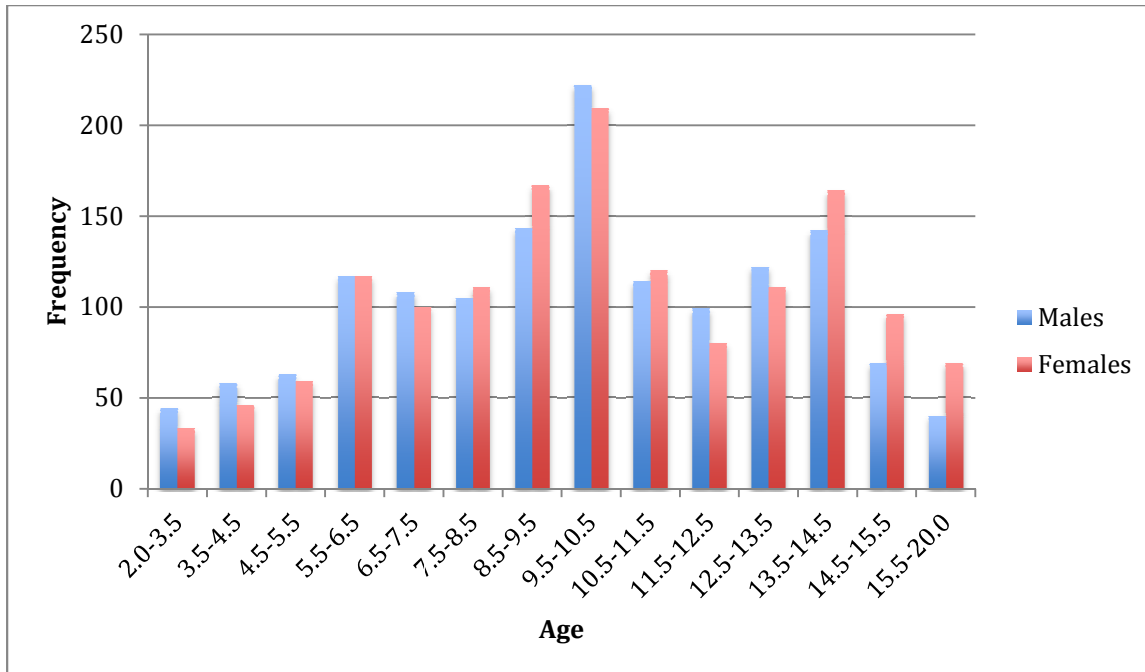


Figure 4: Displaying unequal age distribution from the study conducted by Demirjian, *et al.*, (1973).

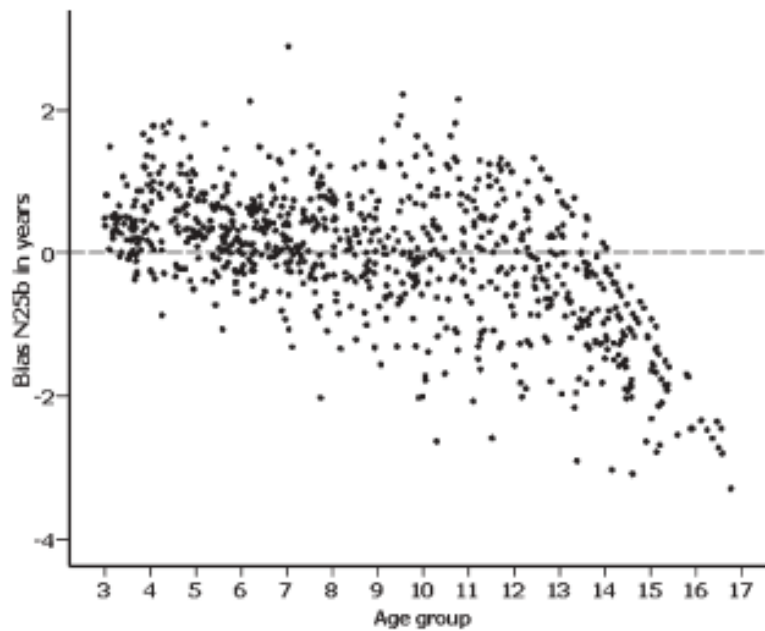


Figure 5: Graph provided by Liversidge, *et al.*, (2010) shown here as an example of a range of variation in an unequal age distribution.

Since this bias was first identified in 1982 by Bocquet-Appel and Masset, paleodemographers have sought statistical and interpretive means for its alleviation. Most efforts have focused on the use of biostatistics to estimate the mortality distribution of samples on the basis of the distribution of age indicator stages. Transition analysis has been used to control for this so-called “age mimicry” (Baldsen 1997, Milner *et al.* 1997, Baldsen *et al.* 1998), by allowing us to make inferences about the timing of transitions from one stage to the next (Baldsen *et al.* 2002). One argument behind transition analysis is that the technique improves age estimation of older individuals, and no longer requires investigators to use an open-ended interval such as 50+ years (Baldsen *et al.* 2002). Also, Baldsen and colleagues suggest that this method addresses the four "analytical difficulties" regarding adult age-at-death estimation (Baldsen *et al.* 2002). These difficulties include: 1) avoiding the large uncertainty associated with age estimation, 2) age mimicry that results from the age-at-death distribution of the reference sample, 3) the most effective way to correlate multiple skeletal indicators of age, and 4) developing methods that code morphological changes as they relate to age. Baldsen, *et al.*, (2002) explain that, "just as no osteologist believes that an exact age can be assigned to any particular skeleton, no one would claim that all skeletons that appear to be roughly the same age can be assigned with equal confidence to a single age interval." Therefore, it becomes clear that every individual with the same skeletal indicator may not come from the same age group (Bethard 2005). The goal of transition analysis is to provide the average age range that an individual transitions from one skeletal stage of development to the next, in order to have a better understanding of the age ranges correlating to a specific stage of development.

Other researchers have attempted to use maximum likelihood estimates (MLEs) of age to estimate the parameters of their statistical models. To construct a maximum likelihood estimate, the first step is to seek the posterior density for an estimate by maximizing the likelihood of observing a fixed data point (such as Suchey's score for pubic symphysis) conditional on the unobservable value being estimated (such as age) (Konigsberg *et al.* 1998). For example, Konigsberg and Herrmann (2002) took a sample of 737 known-age males, whose age was estimated by Suchey's pubic symphysis six-phase scoring system. This sample was divided to create a reference sample of 588 individuals, and a target sample of 149 individuals. Rather than estimating the age of the entire sample, they assumed a normal distribution of age for each score. Knowing the age of some of the population, in this case, 588 people, they take the mean and variance as parameters for their model. By incorporating these parameters, they created a connection between the selected model and the observed data, allowing the model to estimate the age distribution of the remaining 149 individuals based on the Suchey phase of the pubic symphysis.

While transition analysis and MLE analysis can control for some issues related to age estimation, it does not account for the fact that individuals of different ages have varied risks of death, and this can vary from population to population. Also, it is unlikely that all stages of development would have a normal distribution of age. Hazard models have been utilized by many researchers to model survivorship (Wood *et al.* 1992). These models incorporate life tables and data of reported ages from known samples, in order to create an accurate probability of surviving at each particular age. These are known as informed prior probabilities, and can be applied to Bayes' theorem to control for the mimicry bias in age estimation. The output of these models represents the number of individuals in each age group. The survivorship model attempts

to create a line of “best fit” to predict the probability of death at a particular age. The tighter the line fits to the bar graph, the more accurate the prior probability will be. There are several different types of hazard models, including the Gompertz-Makeham, and Siler models.

Konigsberg and Frankenberg (2013) note that one of the issues with the method used by Konigsberg and Herrmann (2002) was the lack of their incorporation of priors, making it a non-Bayesian approach. They state that employing Bayesian methods can more accurately and appropriately approach many of the questions researched by biological anthropologists. In the past, Bayesian statistics tended to be avoided due to the computational burdens. Fortunately, these burdens have recently been removed from the user because of the development of computer simulation methods and related software (Geyer 1992, Gilks *et al.* 1996, Gamerman 1997, Lunn *et al.* 2000, 2009, Brooks *et al.* 2011, Konigsberg and Frankenberg 2013).

Applying Bayes’ Theorem to Age Estimation

Aging individuals utilizes skeletal indicators that provide a morphological age that is linked to a chronological age based on previous studies of reference populations. As noted above, the unequal age distribution of these reference populations results in disparate knowledge of variation of a particular age-related trait across age categories. This then results in an intrinsic bias in age estimation (“age mimicry”) that goes beyond methodological issues. In order to control for this mimicry bias, we need to calculate the probability of what age a skeleton should be based on what we know about risk of death at certain ages and the phase of the particular age indicator observed. This is stated mathematically as $\Pr(c | a)$, where c represents the morphological age indicator stage, and a represents chronological age-at-death (Hoppa and Vaupel 2002). This can be solved using Bayes’ theorem:

$$\Pr(a|c_j) = \frac{\Pr(c_j|a)f(a)}{\int_0^{\infty} \Pr(c_j|x)f(x)dx}$$

Where the variable $f(a)$ is a probability density function for age, $\Pr(a|c_j)$ is the probability that a skeleton died at age a given it has characteristics c_j (in other words, the probability of age given phase); $\Pr(c_j|a)$ is the probability that a skeleton with characteristics c_j died at age a (in other words, the probability of phase given age) (Langley-Shirley *et al.* 2010). $\Pr(a|c_j)$ can be calculated using transition analysis, while $f(a)$ can be calculated by fitting a hazard model to an informative prior distribution (Langley-Shirley *et al.* 2010).

Bayes' theorem has been applied in a number of paleodemographic studies to develop posterior probabilities that express the likelihood that a skeleton showing a particular age-related stage is in fact a particular age. A posterior probability is a conditional probability that is calculated after the prior probability (prior knowledge) is assigned. Bayes' theorem has also been applied to aging methods involving the clavicle, auricular surface, and long bone length (Langley-Shirley and Jantz 2010, Gowland and Chamberlain 2002, 2005; Konigsberg *et al.* 2002). Heuzé and Braga have previously applied Bayesian statistics to dentition in a study in 2008. This study was aimed at further validating the use of Bayesian statistics as a tool to adjust age-at-death distributions to combat the mimicking issue. Unfortunately, however, the small sample size used in this study was not able to validate the use of Bayes' theorem.

Paleodemographers have applied the posterior probabilities developed from Bayesian statistics to best assess age-at-death profiles of ancient populations. For example, Gowland and Chamberlain (2002) used Bayesian statistics to age perinatal skeletal material in archaeological sites in order to re-evaluate the evidence for infanticide in Roman Britain. Through the

incorporation of prior probabilities and Bayes' theorem, this bias was removed and provided them with a more reliable representation of age distribution of the archaeological sample they examined. Looking at nineteen different archaeological sites, these researchers examined a sample of 396 infant long bone diaphyseal lengths. They compared the age distribution of the site obtained through three different methods. These methods include a uniform prior probability, a model prior probability, and the Scheuer, Musgrave and Evans (1980) regression method. These results were compared using the Kolmogorov-Smirnoff statistical test, and showed that there is a statistically significant difference between the age distributions. While the model prior incorporated a higher probability of death at full term, the Scheuer, *et al.* (1980) method displayed a strong central tendency at full term. As a final result of their study, they have shown that this bias can be removed through the incorporation of a realistic prior probability.

Table 1, taken from Gowland and Chamberlain (2002), illustrates the posterior probabilities of long bone length, assuming model prior probabilities they obtained from their study. Generally age estimation of long bone length is generated through a regression equation to calculate age for each individual based on their length (e.g., those of Scheuer et al. 1980), which combined with the other individuals in the sample generates an overall age-at-death mortality profile for this sample. Prior probabilities however can be supplied to a target sample to adjust the population-level mortality profile. For example, if there are 100 individuals in the entire population with a femoral length of 85 mm, then rather than all of those individuals be included in a 44 week-old gestational age category that would result from the regression analysis, 62 individuals would be listed as 44 gestational weeks, 32 as 46 gestational weeks, and 7 as 48 gestational weeks. We could never know which individuals with 85 mm long femora are which age, but that information is not necessary for overall population mortality profiles. Now, age-at-

death of the sample will be less likely to “mimic” the reference sample used to generate the femur regression formula for age.

Table 1: Illustrates the long bone lengths and corresponding ages to be applied in future archaeological cases.

Gest. age (wks)	Long bone length (mm)															
	15–	20–	25–	30–	35–	40–	45–	50–	55–	60–	65–	70–	75–	80–	85–	90–
16	1	0.69														
18		0.31														
20			0.84	0.01												
22			0.16	0.1	0.01											
24				0.89	0.48											
26					0.41	0.27	0.01									
28					0.11	0.3	0.05	0.02								
30						0.42	0.43									
32							0.5	0.53	0.05							
34								0.45	0.57				0.11			
36									0.38	0.41	0.14					
38										0.49	0.34	0.19				
40										0.1	0.28	0.47	0.15	0.3		
42											0.1	0.25	0.64	0.31		
44											0.04	0.08	0.19	0.31		
46												0.01	0.03	0.07	0.62	
48														0.01	0.32	0.55
Total	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

In 2005, Gowland and Chamberlain used Bayesian statistics to examine the demographic structure of a sample of human skeletal remains. The aim of this study was to determine if these plague victims exhibited a catastrophic age structure. Their samples included victims of the 1348 plague from the Royal Mint site and Blackgate site using the auricular surface (Lovejoy et al. 1985) and pubic symphysis (Brooks and Suchey 1990). As comparative reference samples with known age, the authors utilized the Coimbra Identified Skeletal Collection in Portugal and the Spitalfields skeletal collection in London to create a probability model of age. Gowland and Chamberlain used two different model priors in this study, one representing natural attritional mortality, and the other catastrophic mortality. This study differs from many Bayesian employed studies in that their priors were based on the Coale and Demeny model life tables. These tables provide good models for the pre-industrial populations, and were more applicable to this project (Chamberlain 2000, Gowland and Chamberlain 2005). This information provided the source for likelihoods, which became the prior probabilities used in Bayes’ theorem. Using these two

different priors provided them with interesting results. Applying the catastrophic prior showed a markedly different age distribution obtained from the Royal Mint cemetery from the Blackgate cemetery. Their results showed that the Royal Mint cemetery exhibits a catastrophic mortality pattern as a result of the Black Death plague, and all age groups were equally affected. By contrast, the Blackgate site produced an attritional mortality profile, with non-catastrophic conditions (Gowland and Chamberlain 2005).

Bayesian statistics has also been applied to increase the confidence of skeletally-derived age estimations of individuals, more suitable to forensic anthropology. Langley-Shirley and Jantz (2010) incorporate a Bayesian approach to a sample of 1289 clavicles in an attempt to derive robust age ranges for modern individuals to provide updated standards of the American population. Their skeletal sample included 594 individuals from the William F. McCormick Clavicle Collection, and 354 individuals from the Hamann-Todd Osteological Collection. Additionally, they included the raw data from the study by McKern and Stewart (1957). Determination of age was conducted using the five-phase system developed by McKern and Stewart, and a three-phase scoring system. The authors conducted transition analysis to determine the average age at which the transition from one phase to the next occurs. Also, they applied a Gompertz hazard model to an informative prior, based on data from the Forensic Data Bank. Bayesian analysis combined the results of the transition analysis and the hazard parameters produced from the Gompertz model. Through this analysis, they produced age ranges with four different probability percentages (50%, 75%, 90%, and 95%) for each phase for both scoring systems. These age ranges are less sensitive to age mimicry, and can be applied to future forensic studies. The results of this study show that epiphyseal union commences about 4 years earlier on average in modern Americans than from Americans in the early 20th century. This

supports the need for modern standards to assess age in modern individuals (Langley-Shirley and Jantz 2010).

My study will allow those using dental eruption for age estimation to use the posterior probabilities that I will calculate to create a less biased age-at-death distribution of individuals. The posterior probabilities of being a certain age while displaying a specific stage of dental development can be utilized by forensic anthropologists wishing to have an unbiased age range based on dental development. There are gaps that exist with the research concerning the age mimicry bias and Bayesian statistics, including the specific circumstances in which these methods can be applied. Since dental development is not as affected by environmental factors, and teeth have great preservation, I will be applying Bayes' theorem to this aging method in order for it to be applicable in most cases. Starting with a precise aging technique will allow for more accurate results, and valid age ranges that are applicable to unknown samples of a forensic context. As Boldsen, *et al.*, (2002) suggest, I am creating a method that is applicable on the individual level. I will incorporate two different informative priors in an attempt to provide resultant age estimates that will be free of the age mimicry bias

Materials and Methods

For this project, I randomly sampled 201 individuals from the Orthodontics Case File System of the Maxwell Museum of Anthropology at the University of New Mexico. This collection is comprised of approximately 5,000 orthodontics records donated to the Maxwell Museum in 2005 for research purposes by a retiring orthodontist (Dr. James Economides) in Albuquerque, NM. This anonymous collection contains treatment histories, oral images, x-rays, and dental casts of patients with known age, sex, and ancestry. Of these individuals, 3,053 are eighteen years of age or younger and have at least one x-ray image available. Unfortunately, the radiographs that were supplied for my sample were not derived from a pediatric orthodontist. Therefore, the youngest individual in my sample is five years of age, and the oldest is twelve since formation of dentition other than the variable third molar ceases around this age.

In order to collect data from medical records of possibly still-living individuals, I was required to submit a report of my research to the Institutional Review Board (IRB) at East Carolina University. My study was certified exempt due to the non-invasive, and anonymous nature of this project, and assigned the IRB identification number of UMCIRB 13-001213. A copy of the letter of approval can be viewed in Appendix 6.

Radiographs of the dentition of the individuals from this sample were used to score dental formation using the method developed by Moorrees *et al.* (1963). This method categorizes tooth formation by the fraction of crown, root, and apical completion observed (Figures 2 and 3). The left upper and lower incisors, canines, premolars, and first and second molars were used in this analysis. Due to the variability of the third molar, it was excluded from this research. I created contingency tables documenting the stage of growth of permanent teeth in the sample and the recorded age of each individual by sex. Then, I used these contingency tables

to conduct transition analysis in the statistical package R (www.r-project.org; see Appendix 1 for R code). This utilized a cumulative probit model on the natural log of age to determine the age range that an individual transitions from one of Moorrees' dental development phases to the next. This cumulative probit model assumes that the age indicator states are ordinal categorical, or that there are separate phases that occur in a set order. Because of the older age in this sample, the dental phases observed did not start at the first possible phase of growth. Therefore, the tooth development stage based on Moorrees and colleagues had to be restaged in order to create consecutive stages starting at the value 1 (i.e. if the Moorrees phases were recorded as "7, 9, 10" the restaging was recorded "1, 2, 3"). This process assigns the same standard deviation to each transition, and the natural log scale assures that the transition distribution is log-normal (Langley-Shirley and Jantz, 2010). The output of the transition analysis provided the standard deviation and mean ages of transition for each stage and tooth of each sex.

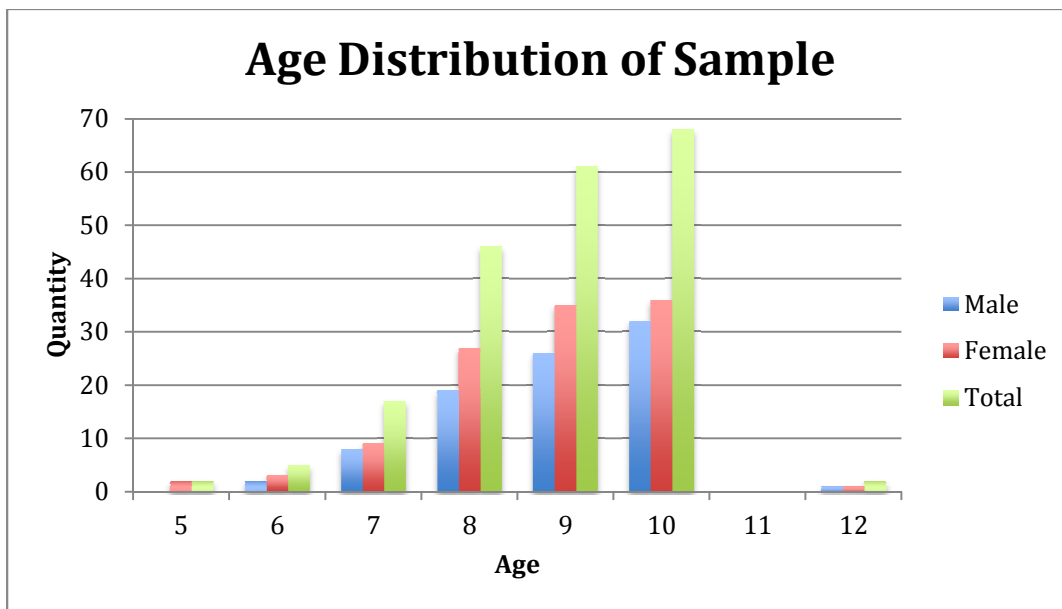


Figure 6: Age distribution of the Maxwell Museum sample, illustrating the unequal number of individuals at each age.

For development of most age estimation techniques, the analysis would stop there.

However, as seen in Figure 6, the Maxwell Museum sample used to derive our transition stages for dental age estimation contains unequal numbers of individuals at each age. Therefore, any age estimation using only the transition stages developed will suffer from “mimic bias,” where more individuals would be placed into the ages with the most number of individuals (e.g., 8, 9, and 10) due to the methodological issues described earlier. A Bayesian approach thus was used to calculate “adjusted” age ranges based not only on the maximum likelihood of age-at-transition, but also the likelihood of death at a particular age. Bayes’ Theorem calculates the posterior probability that an individual should be a certain age (a) taking into consideration not only the age indicator observed (c), but also the probability of death at a certain age [$f(a)$, or the informative prior probability, where ω is the upper limit of the life span].

$$\Pr(a|c) = \frac{\Pr(c|a) f(a)}{\int_0^{\omega} \Pr(c|a) f(a) da}$$

In this study, I computed a hazard model of mortality profiles, one for normal childhood mortality for the year 2008 and one related to childhood violent deaths from the years 2005-2010. The 2008 normal mortality prior probability is based on the age at death distribution of U.S. children under the age of 15 dying in that year, data provided by the National Center for Health Statistics (Arias 2010). The violent deaths of subadults during 2005-2010 has a slightly different mortality profile, and is used to illuminate how different prior probabilities of death can impact the results of the Bayesian analysis. The data for this second prior include the number of violent deaths classified as unintentional firearm, homicide, legal intervention, and undetermined intent of children from birth to 15 years by sex and age in years based on the data from 16 U.S. states. The 2008 general mortality profile can provide prior probabilities of death applicable for

age estimation of modern forensic cases, and the 2005-2010 in particular for cases involving violent deaths of children.

A Gompertz-Makeham survivorship model was applied to the samples of U.S. children in 2008 and those dying of violent means during 2005-2010 to general prior probabilities for the Bayesian model. This survivorship curve represents number of individuals (survivors) in each age group. As the groups increase in age, the quantity of individuals decreases. The survivorship model attempts to create a line of “best fit” to predict the probability of death at a particular age. The tighter the line fits to the bar graph, the more accurate the probability will be. Survivorship curves of males and females for normal and violent mortality, as described above, were calculated in R (see Appendix 2 and 3 for code) with the following equations and are shown in Figures 12 and 13.

$$S(a) = \exp\left[-\alpha_1 a + \frac{\alpha_2}{\beta} (1 - e^{\beta a})\right]$$

$$h(a) = \alpha_1 + \alpha_2 e^{\beta a}$$

Although this is the way survivorship and hazard equations are traditionally written, I had to reparameterize them to be consistent with the R package as follows:

$$S(t) = \exp\left[-a_2(t - bot) + \frac{a_3}{b_3} [1 - e^{b_3(t-bot)}]\right]$$

For Bayesian analyses, it may be necessary to center time at a specific point. The variable *bot* serves as this centering parameter. In this case *bot*=0 since we were interested in dental development occurring from birth. However, if this equation was to be used for an adult aging method in which the degenerative processes begin at, say age 20, then *bot* would be equal to 20.

Finally, the results of the transition analysis and survivorship curve representing probability of death at a particular age were brought together through Bayes' Theorem in order to create highest posterior densities (HPDs), based on the survivorship curves, that an individual dying at stage a displays skeletal stage c . This analysis was completed for each tooth by sex in R (Appendix 4). HPDs were calculated at the 50% level. This is read similarly to a confidence interval, where 50% of children with that stage of formation should fall into the range, with 25% above the range, and 25% falling below the range. However an HPD graph differs from a confidence interval in that it provides exact probabilities, in this case a 50% probability of an age range at a particular stage for a specific tooth. Using the statistical models highlighted in this chapter, I present data and interpretations on the correlation of dental development with skeletal age.

Results

The dental age of 201 individuals was determined from radiographs using the method defined by Moorrees, *et al.* (1963). First, the age at transition from one stage to the next was determined through transition analysis, which provided age at transition distributions. These are displayed in Figures 7-10. Once these ages were calculated for each tooth, they were incorporated into the calculation of the highest posterior density, providing an exact 50% probability age range for each tooth at a specific stage.

Transition Analysis Results

The stage transition age ranges were calculated for each observed dental stage for each tooth (with the exception of the third molar) by sex. Figures 7 through 10 represent the age-at-transition distributions for this sample. Figure 7 displays upper male teeth, while Figure 8 contains lower male teeth. These are followed by Figures 9 and 10, which display upper female teeth and lower female teeth respectively. Each graph illustrates the labeled transitions (from L-R) between their respective stages of 10 and 11, 11 and 12, 12 and 13, and so on. The shift in age based on the dental growth process clearly emerges, as does the increased variability when children transition in the later stages.

The transition analysis provides an age interval at which the subadult transitions from one phase to the next. This calculation also provides the standard deviation of those ages. These results are displayed in Tables 2 and 3. These age intervals translate into variables used to determine the highest posterior density and 50% probability age ranges for each tooth at each stage.

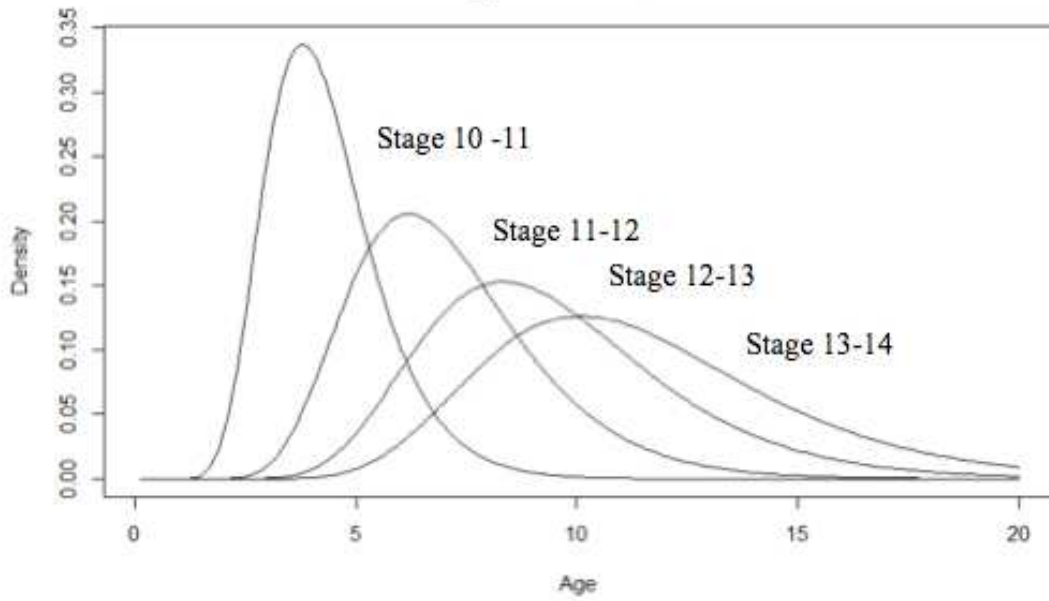
Table 2: Transition ages and standard deviations for each phase calculated for females through transition analysis.

	UI1	UI2	UC	UPM1	UPM2	UM1	UM2	LI1	LI2	LC	LPM1	LPM2	LM1	LM2
Phase 3	-	-	-	-	-	-	-	-	-	-	-	-	-	5.00987017
Phase 4	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Phase 5	-	-	-	4.92870364	-	-	5.01652268	-	-	-	-	5.65410596	-	5.98206604
Phase 6	-	-	-	6.09822046	6.15989049	-	5.87571057	-	-	-	4.95474152	6.25878452	-	7.2773079
Phase 7	-	-	4.06692831	7.54324907	8.15152479	-	7.98267845	-	-	-	7.17149399	8.16596576	-	8.25060062
Phase 8	-	-	-	-	-	-	9.88869317	-	-	-	-	-	-	9.01343282
Phase 9	-	5.21273672	6.5759891	9.18495666	9.89717141	2.48768684	11.1369677	4.8290415	-	7.36656118	8.88455166	9.58101944	-	9.93810144
Phase 10	5.22076008	6.89290071	8.33193732	9.9483529	11.3222747	4.65109697	13.1155559	-	6.23177996	8.46469513	9.88367099	10.3471902	5.15949135	11.4162982
Phase 11	6.89099854	8.11165568	10.1744635	10.8317106	11.6790425	6.59023956	14.2709785	6.45398649	7.10618833	9.71124022	11.2782151	11.8668524	6.12635527	12.0660413
Phase 12	8.20700452	9.99619979	11.7745803	11.6953462	12.4848262	9.78543935	15.2212884	7.50705316	8.30739432	10.9216057	12.5097332	12.8488588	7.72232126	12.626225
Phase 13	10.5331666	12.1867951	14.7132434	12.665351	-	14.9956491	-	8.91460551	10.4482343	11.9067564	13.2019164	13.9382241	9.91911794	-
Phase 14	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Standard Deviation	2.24234447	2.70964772	3.79287326	2.72569806	2.4041755	4.88034628	3.81056376	1.72358215	1.8268054	1.82549991	2.95122175	3.01021794	2.07996474	2.68769853

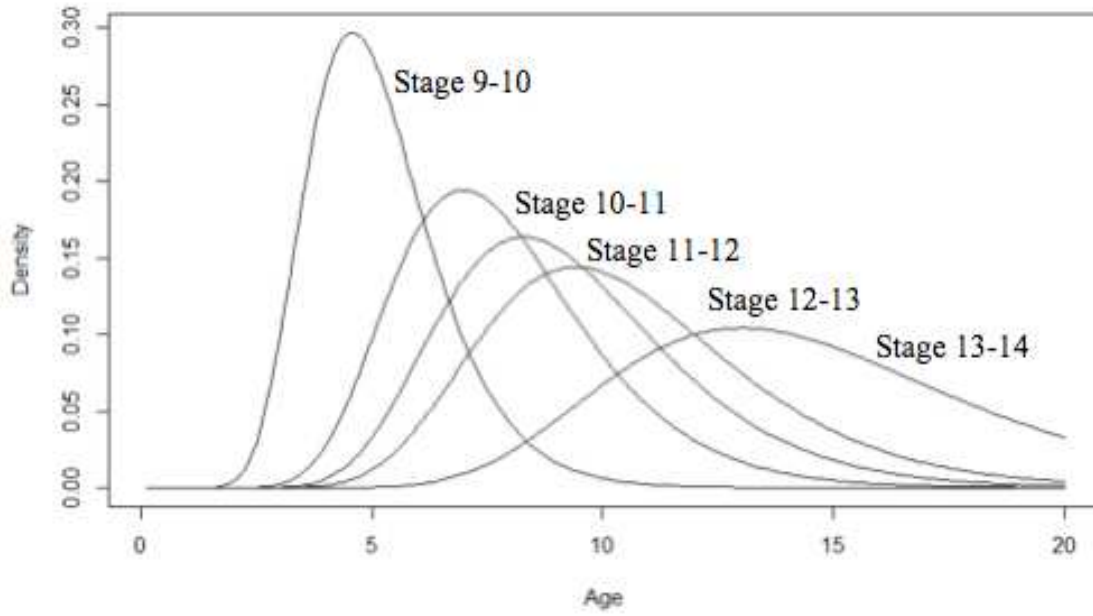
Table 3: Transition ages and standard deviations for each phase calculated for males through transition analysis.

	UI1	UI2	UC	UPM1	UPM2	UM1	UM2	LI1	LI2	LC	LPM1	LPM2	LM1	LM2
Phase 5	-	-	-	-	-	-	4.76693719	-	-	-	-	4.52075041	-	5.22757762
Phase 6	-	-	-	5.43203047	6.15989049	-	5.07394185	-	-	-	5.62758839	5.40002185	-	6.93360583
Phase 7	-	-	5.2465987	7.72417484	8.15152479	-	5.65592688	-	-	5.16154008	7.75886453	8.63106583	-	8.57085933
Phase 8	-	-	-	-	-	-	8.26408486	-	-	-	-	-	-	9.43566822
Phase 9	-	4.9432549	7.49463177	9.47094793	9.89717141	4.22314453	10.664527	-	-	7.52717646	-	9.93892633	-	10.7487665
Phase 10	4.13091528	7.55053937	9.43200789	10.7100976	11.3222747	5.46168855	12.4793964	-	5.92307654	9.35744235	10.4129997	11.893678	6.04623038	12.0773645
Phase 11	6.77684057	8.97334934	11.0084482	11.4963547	11.6790425	7.42158763	14.6484423	5.98358568	7.554444	10.7925815	11.3247093	13.0599723	7.30771033	14.0510623
Phase 12	9.10689665	10.1967598	12.8650586	12.1604028	12.4848262	10.1355191	16.3342805	7.35688787	8.33171236	12.6748916	11.8524903	13.8105943	8.15659661	-
Phase 13	11.0427268	14.1151826	-	-	-	12.6121294	-	8.73060789	10.4947801	16.0231251	13.705593	-	9.67971056	-
Phase 14	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Standard Deviation	2.98480644	3.392446	2.97044579	2.53890844	2.4041755	3.42308864	4.49281464	1.37351111	1.89938464	3.83682406	2.93774262	3.6389865	1.52517826	3.0160512

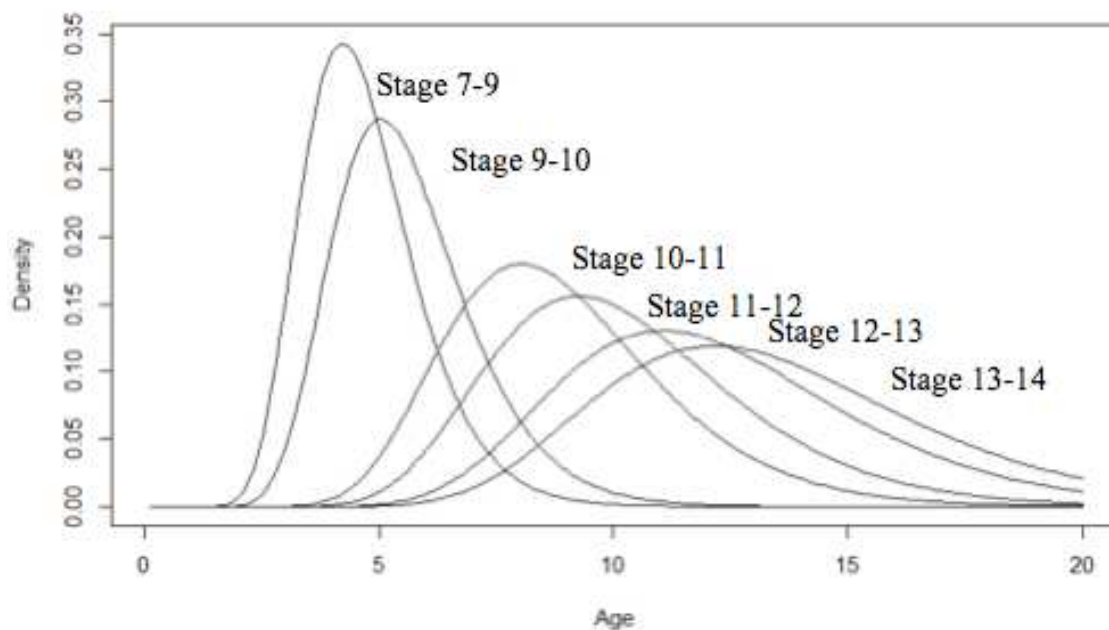
Upper Incisor 1, Male



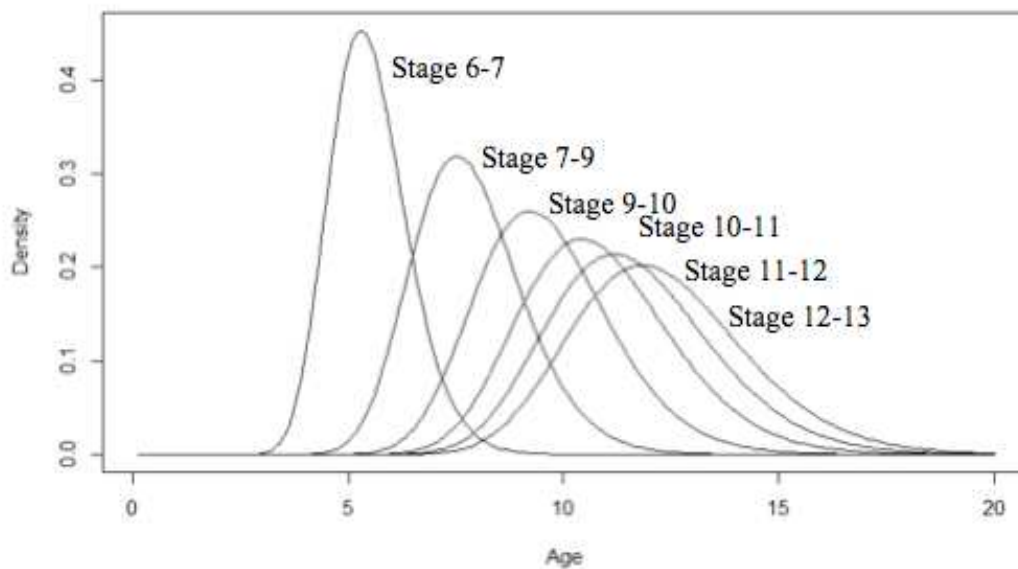
Upper Incisor 2, Male



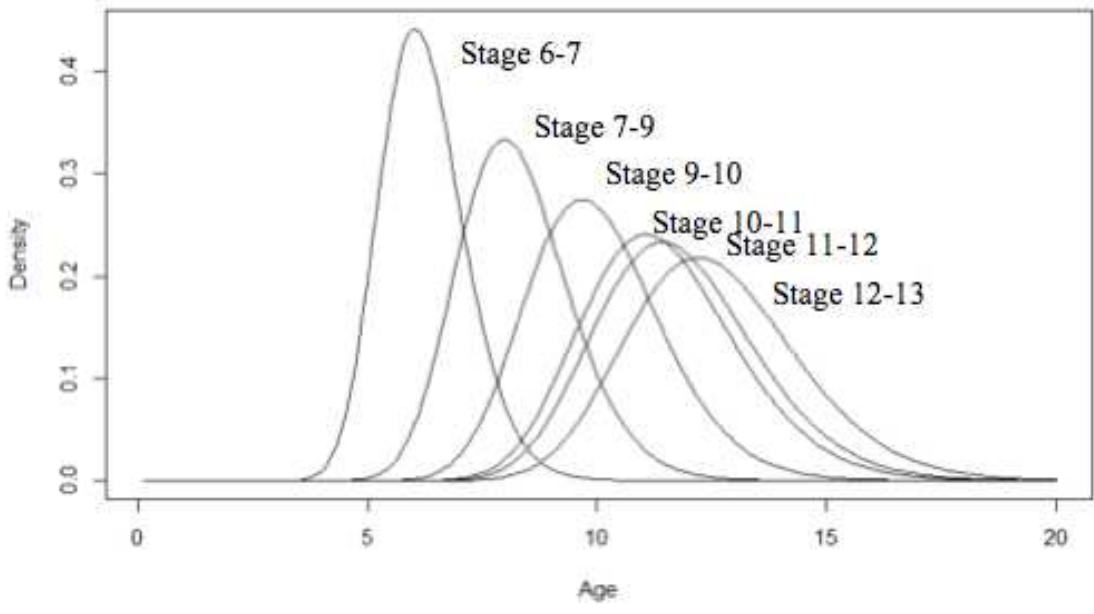
Upper Canine, Male



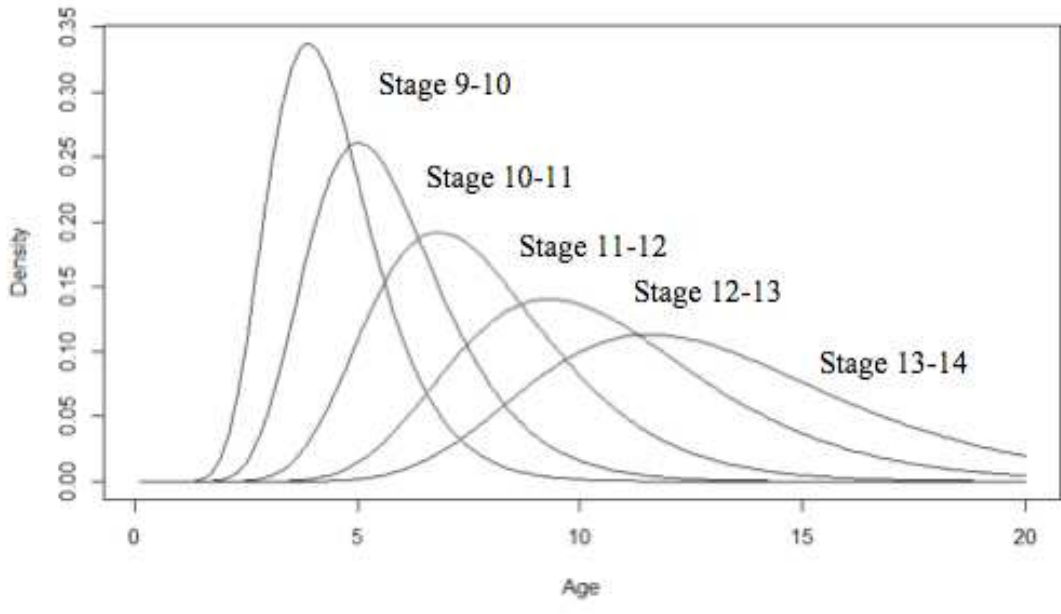
Upper Premolar 1, Male



Upper Premolar 2, Male



Upper Molar 1, Male



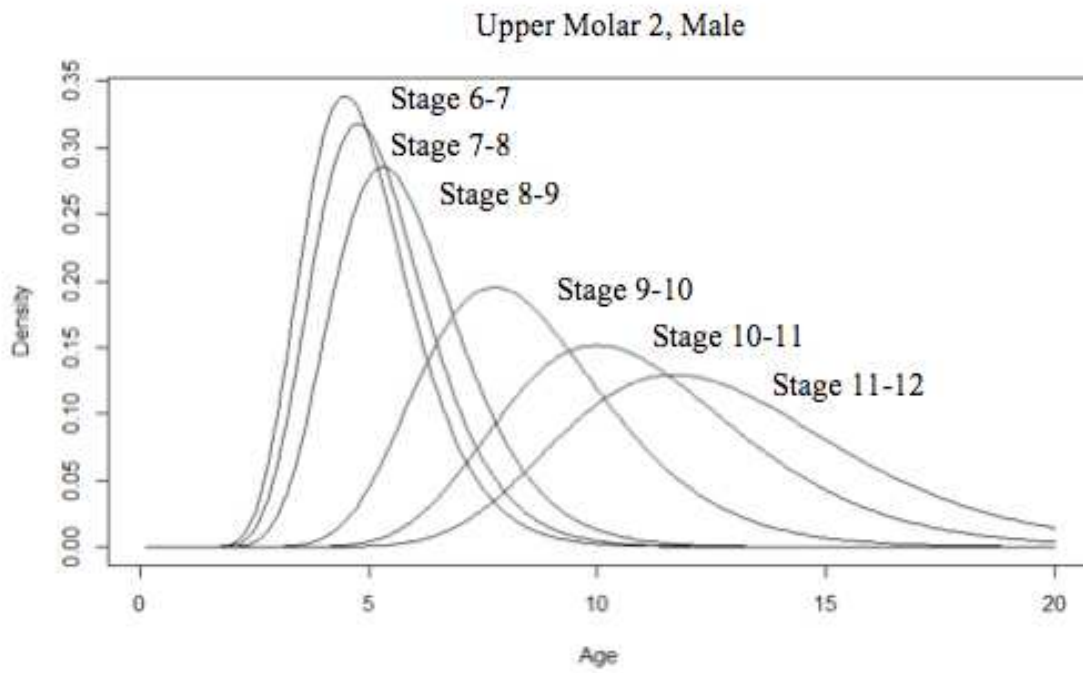
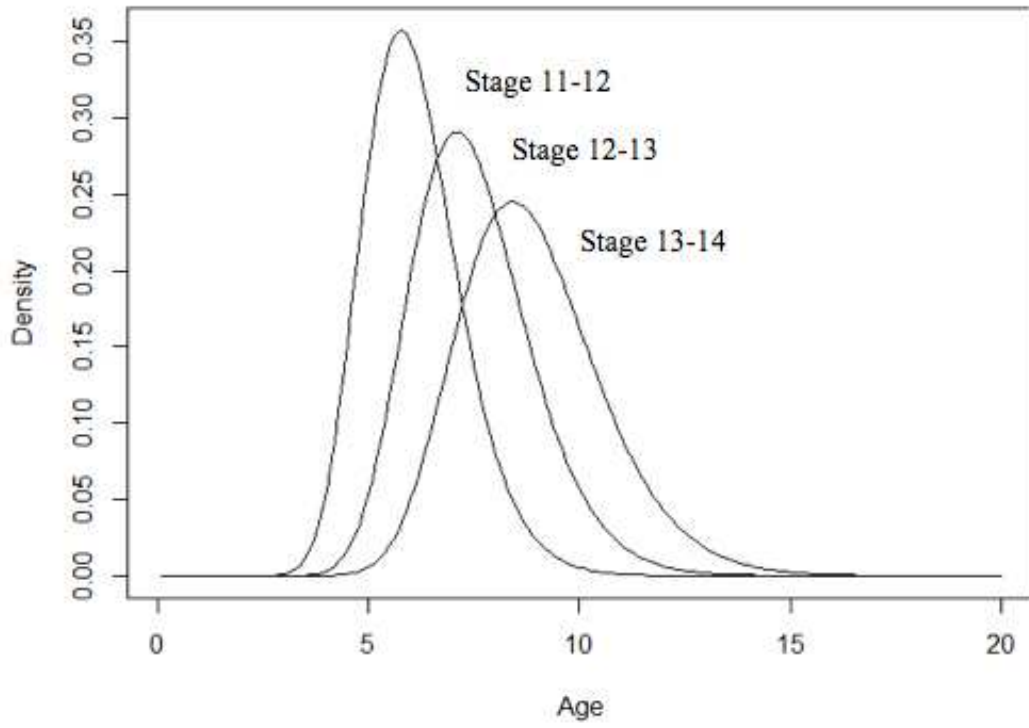
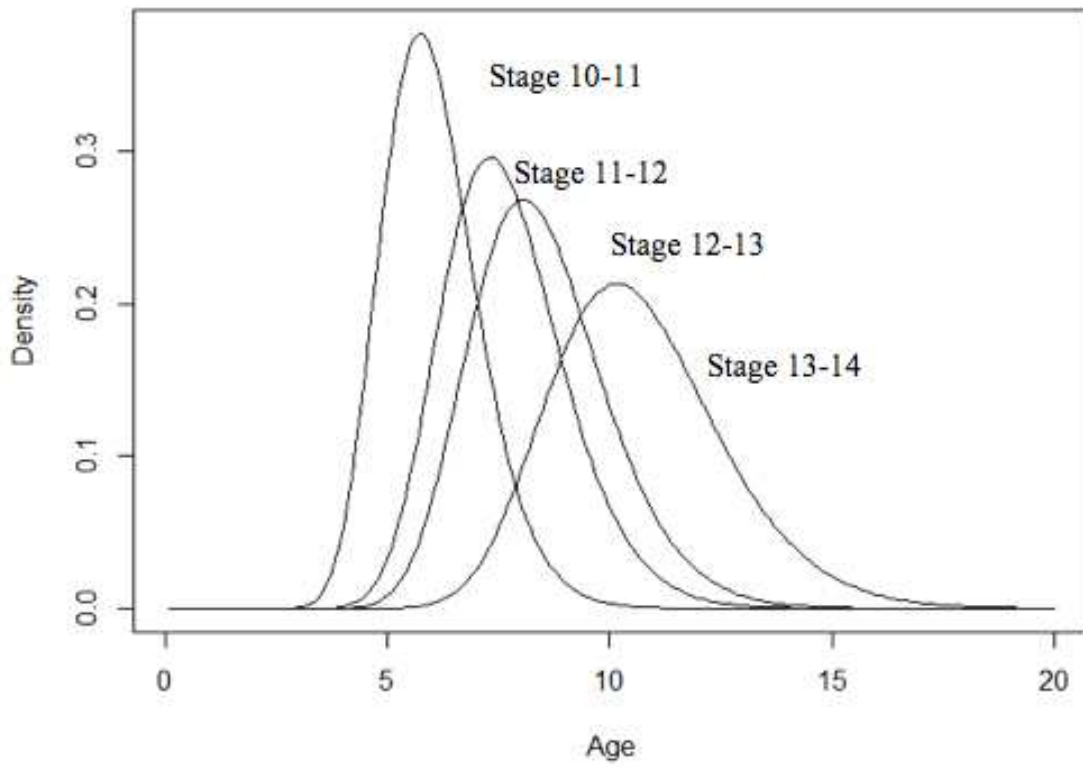


Figure 7: Displays the age-at-transition distribution between dental development stages for all upper male teeth.

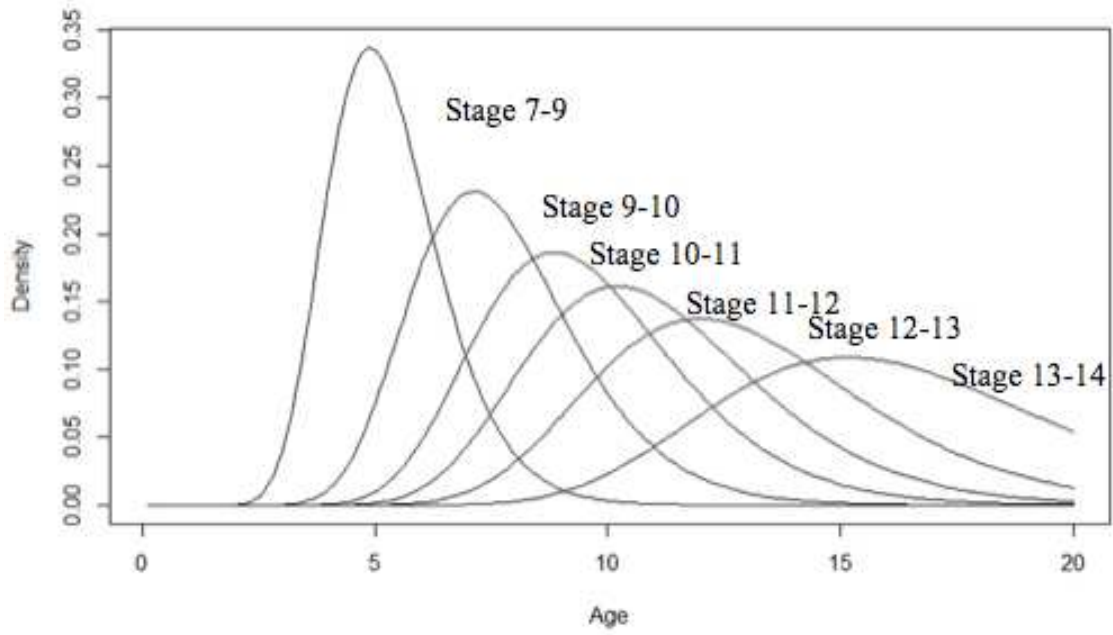
Lower Incisor 1, Male



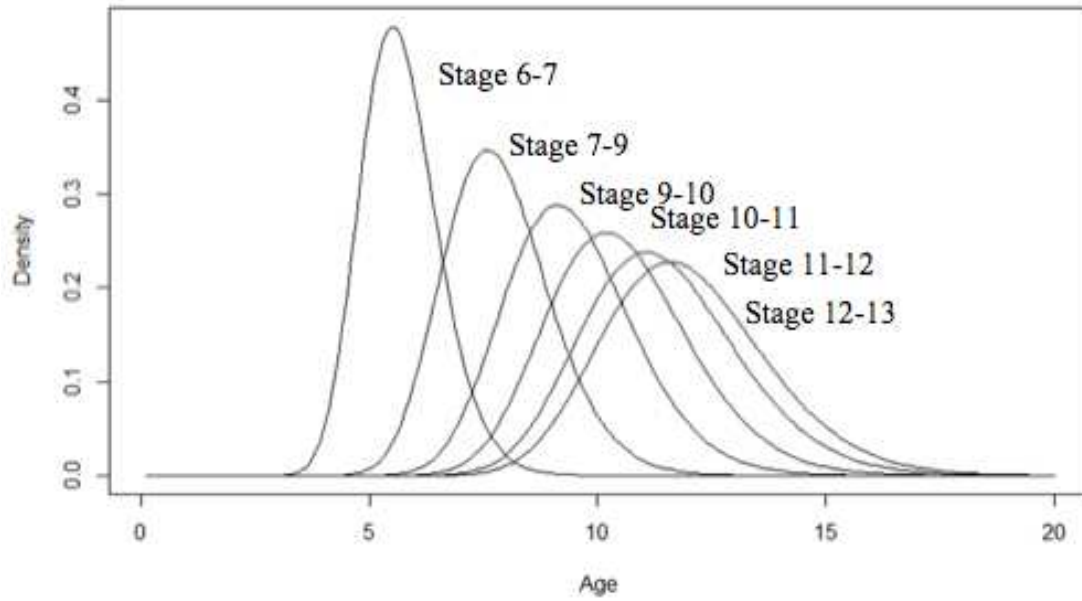
Lower Incisor 2, Male



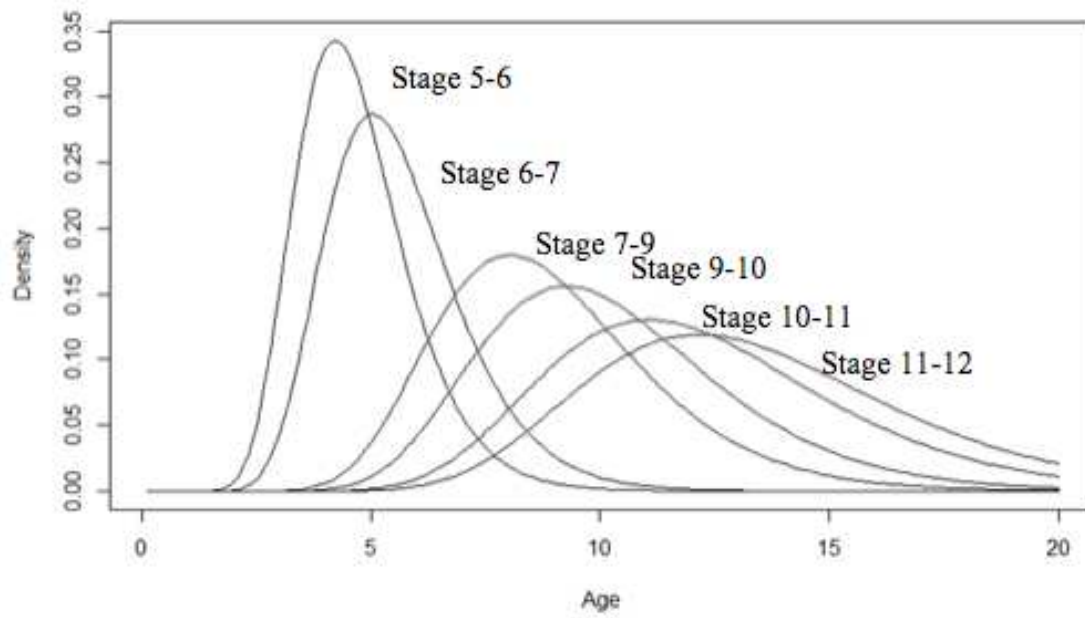
Lower Canine, Male



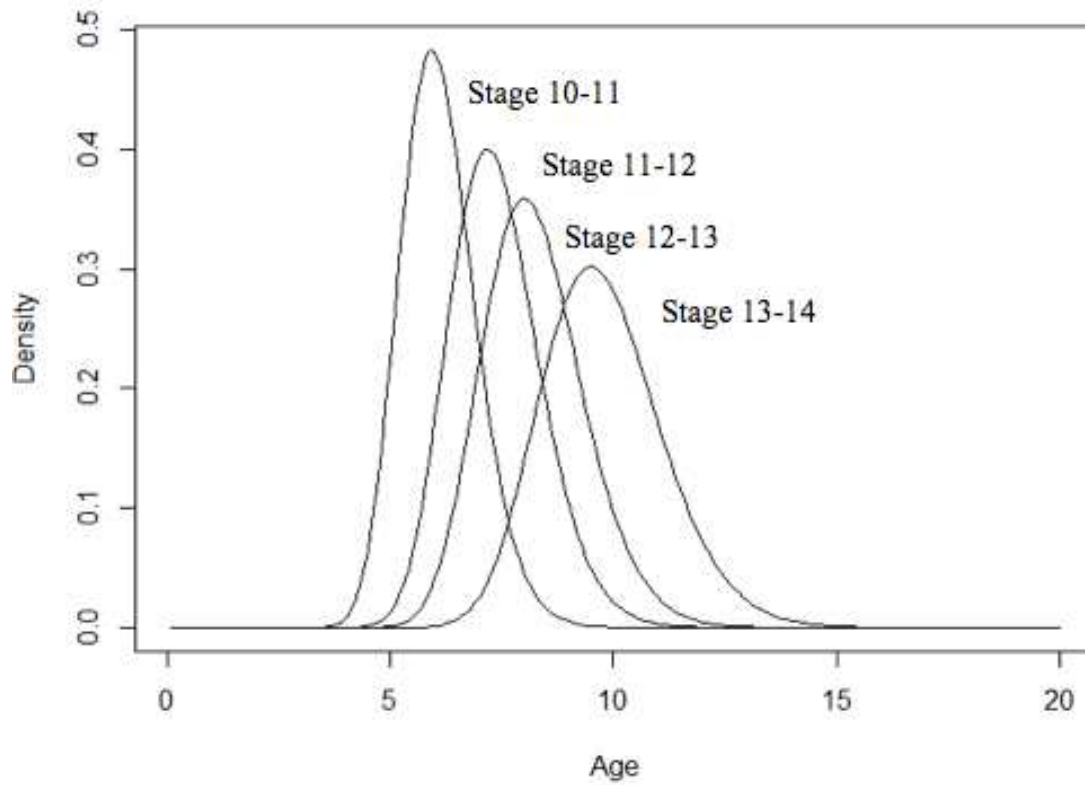
Lower Premolar 1, Male



Lower Premolar 2, Male



Lower Molar 1, Male



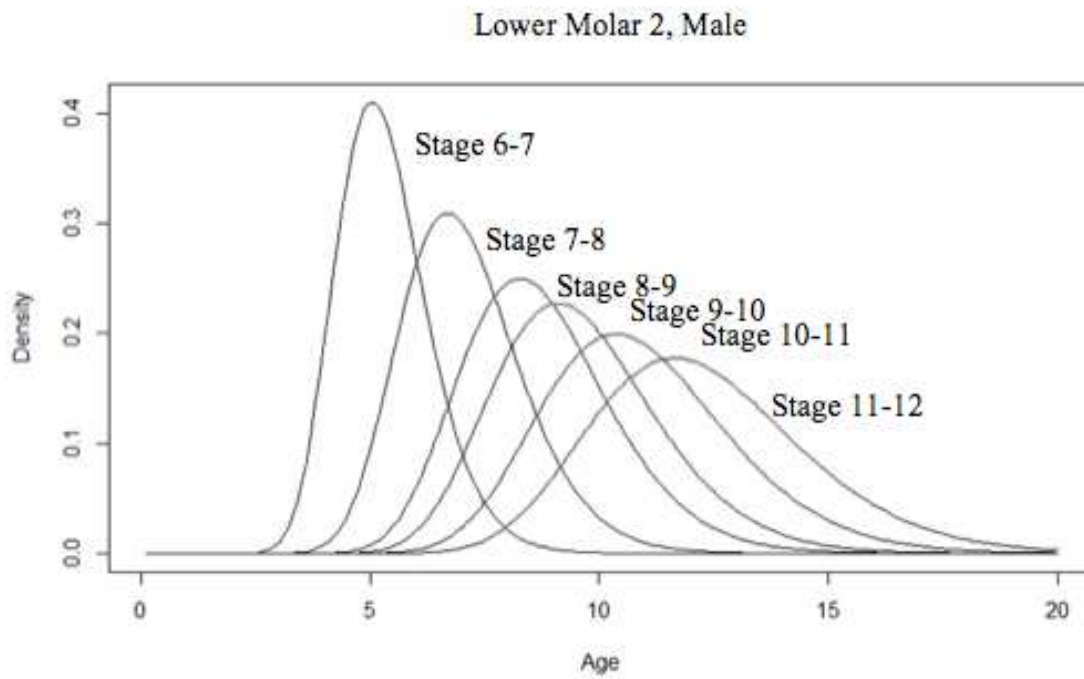
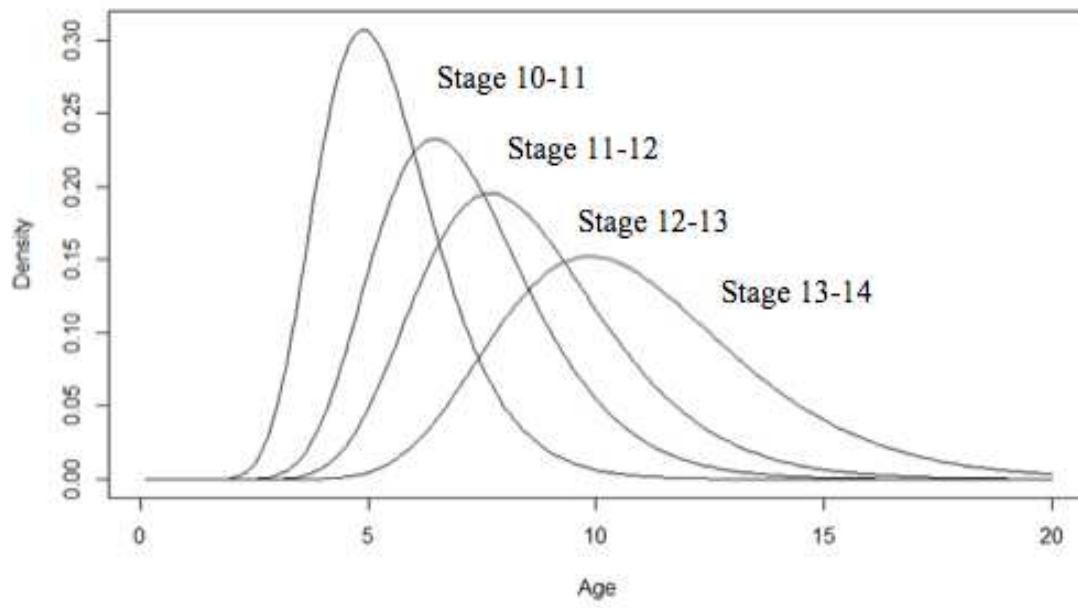
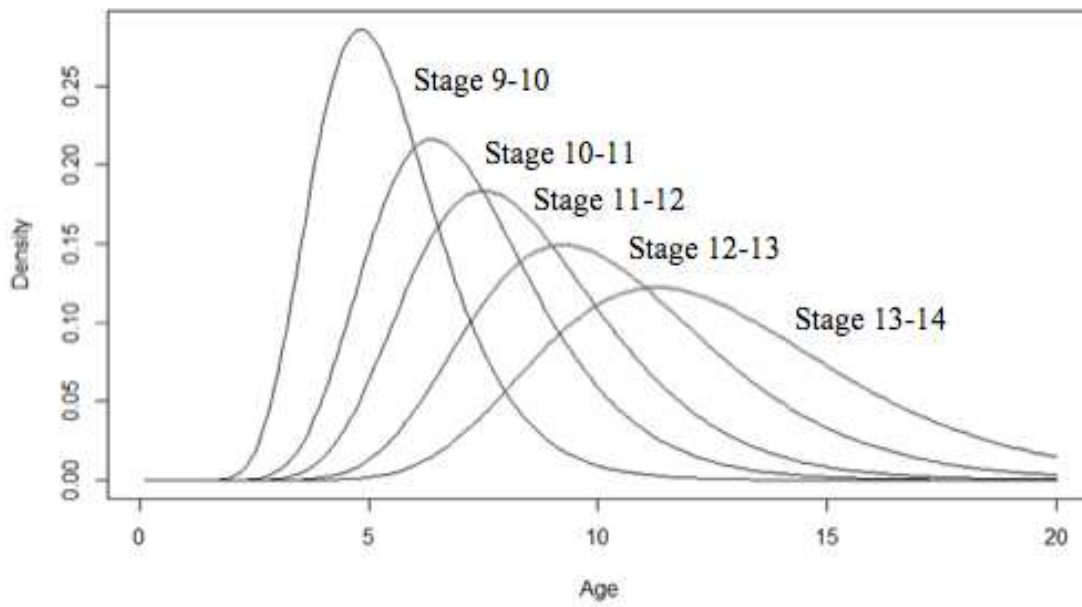


Figure 8: Displays the age-at-transition distribution between dental development stages for all lower male teeth.

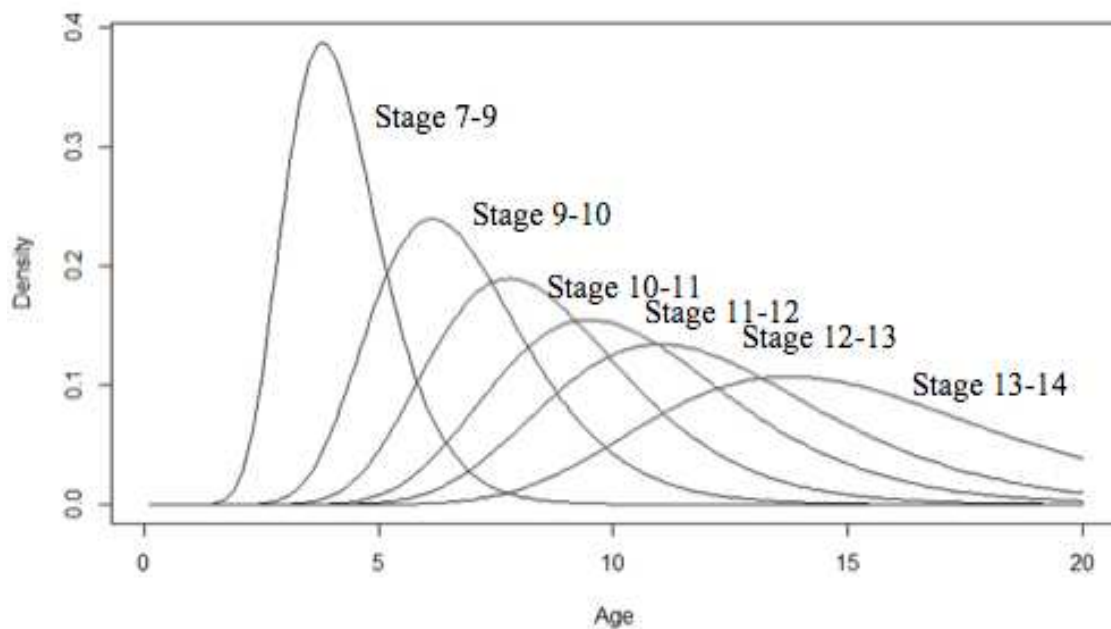
Upper Incisor 1, Female



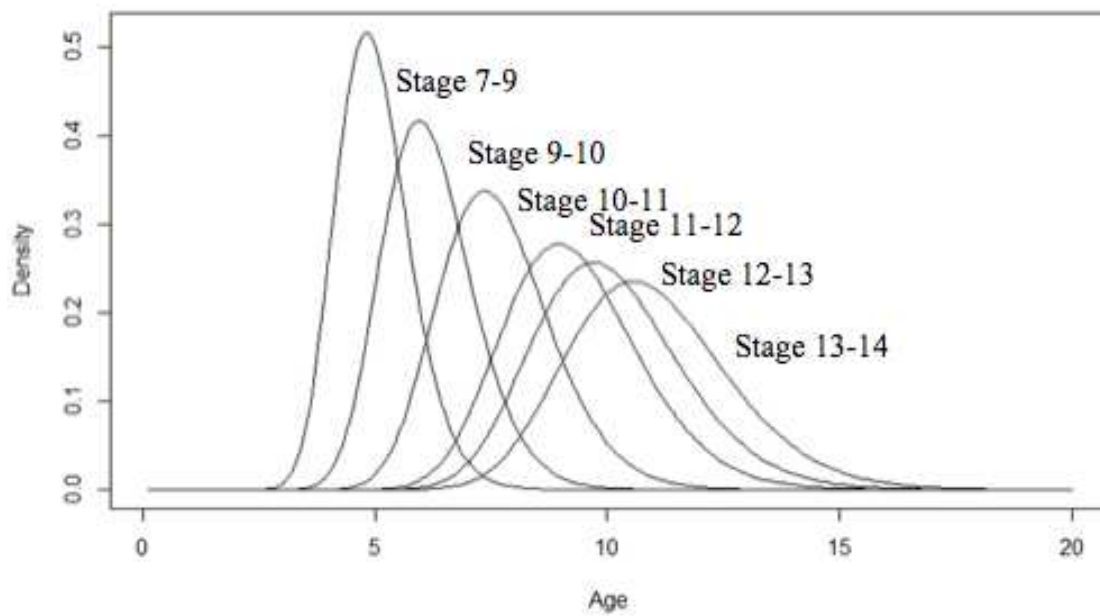
Upper Incisor 2, Female



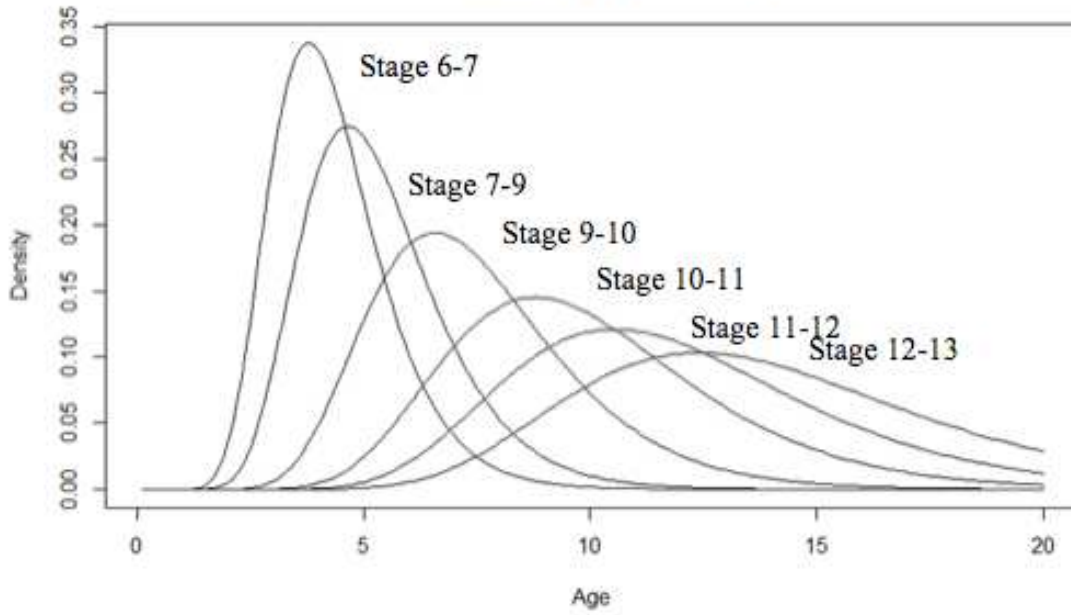
Upper Canine, Female



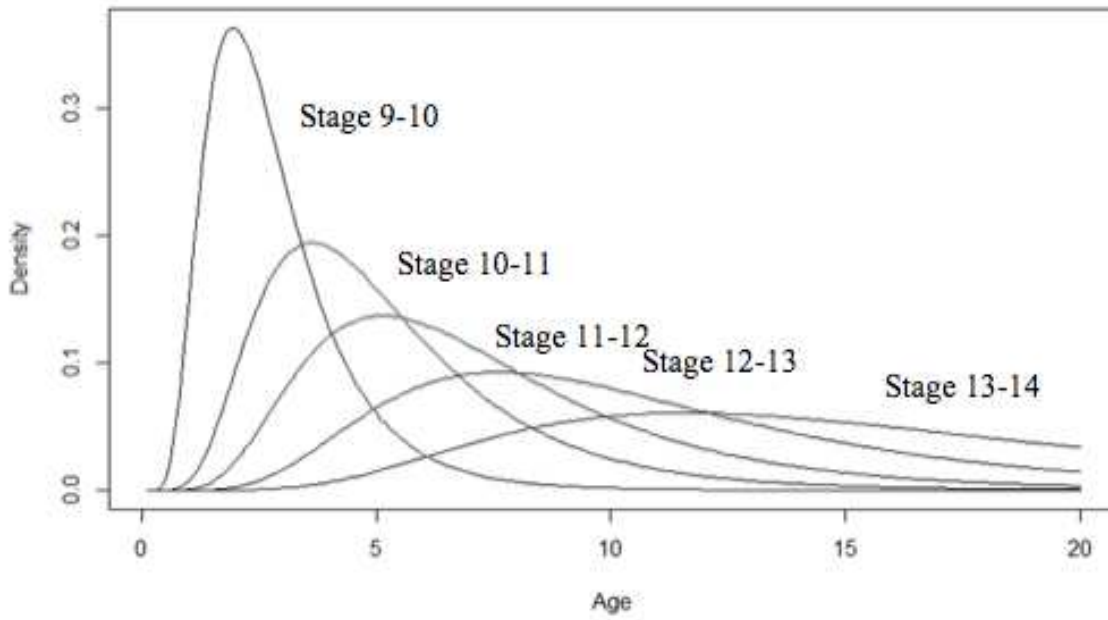
Upper Premolar 1, Female



Upper Premolar 2, Female



Upper Molar 1, Female



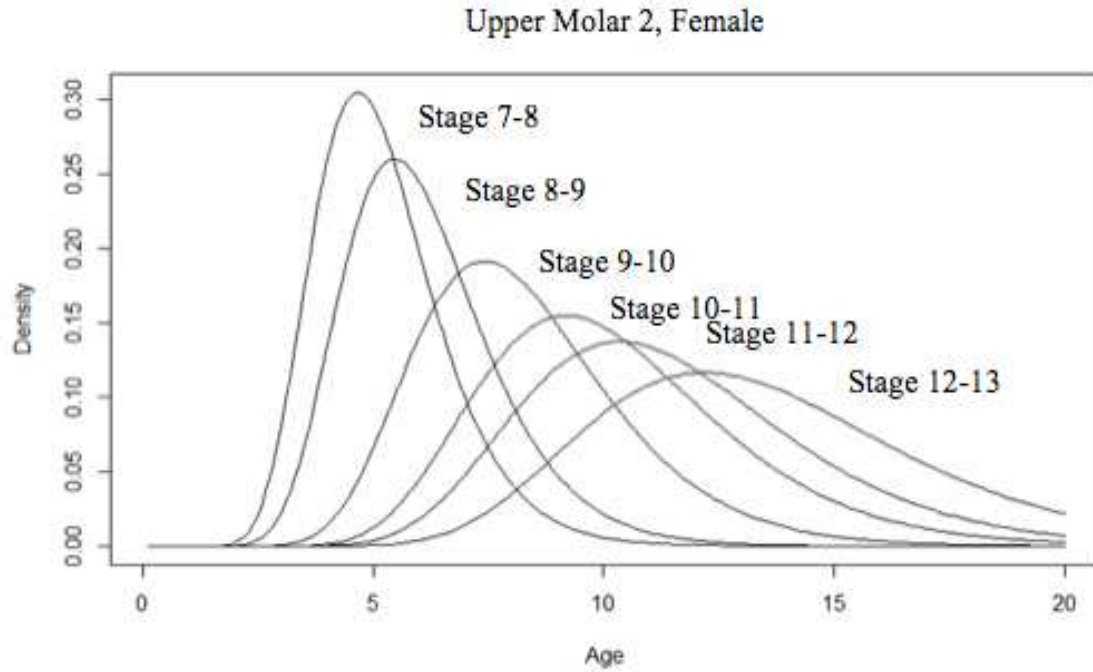
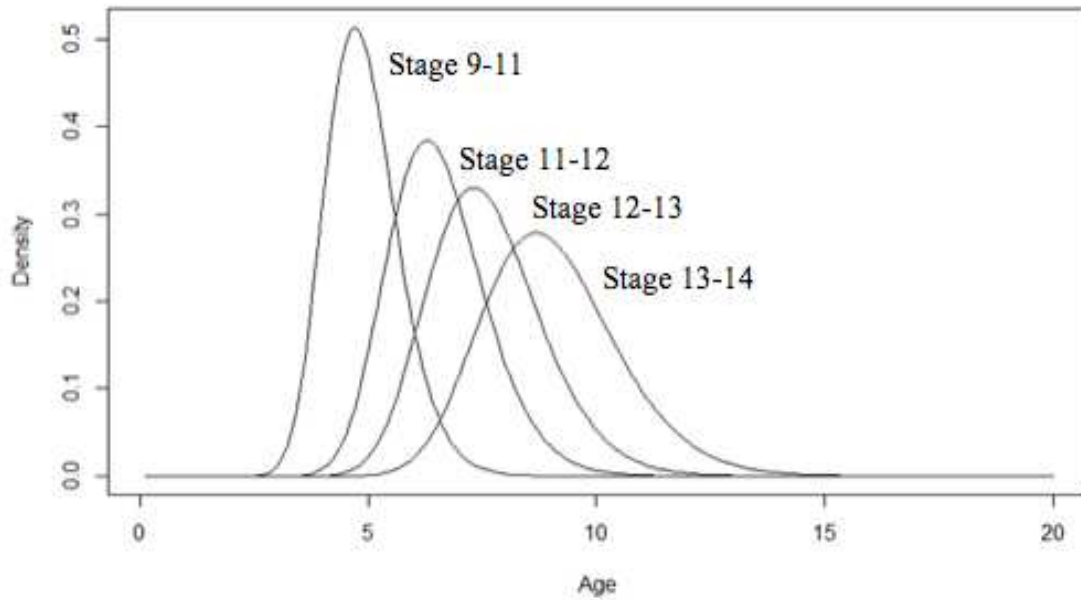
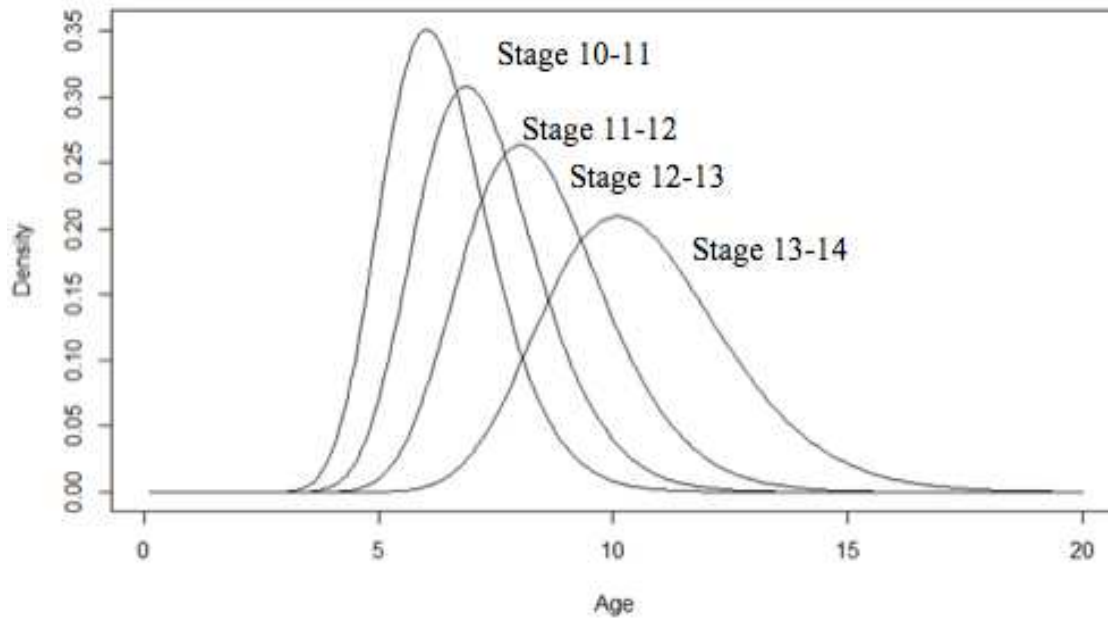


Figure 9: Displays the age-at-transition distributions between dental development stages for all upper female teeth.

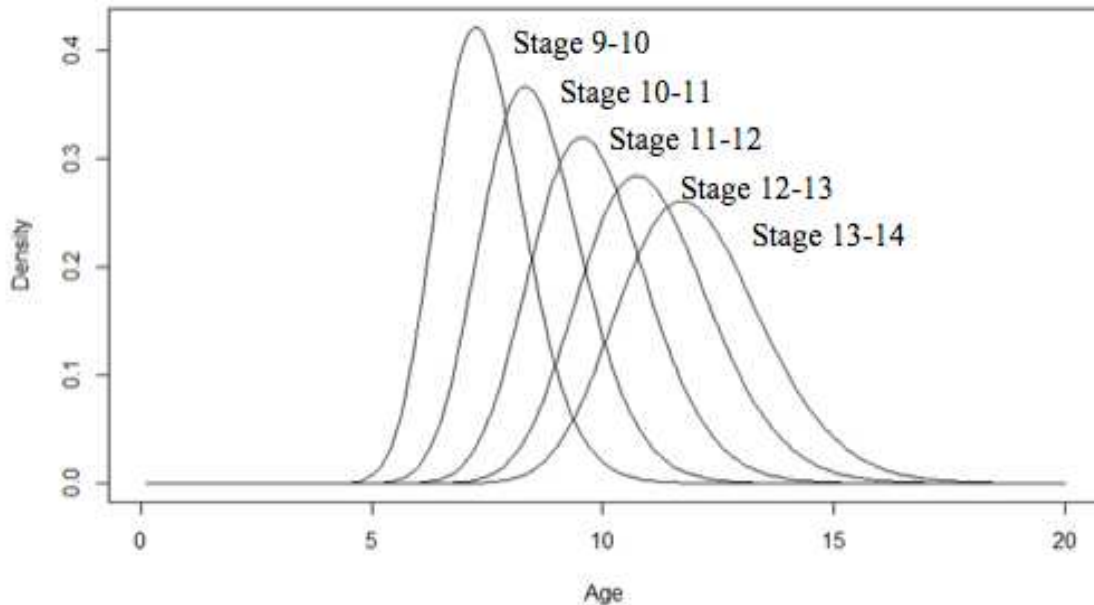
Lower Incisor 1, Female



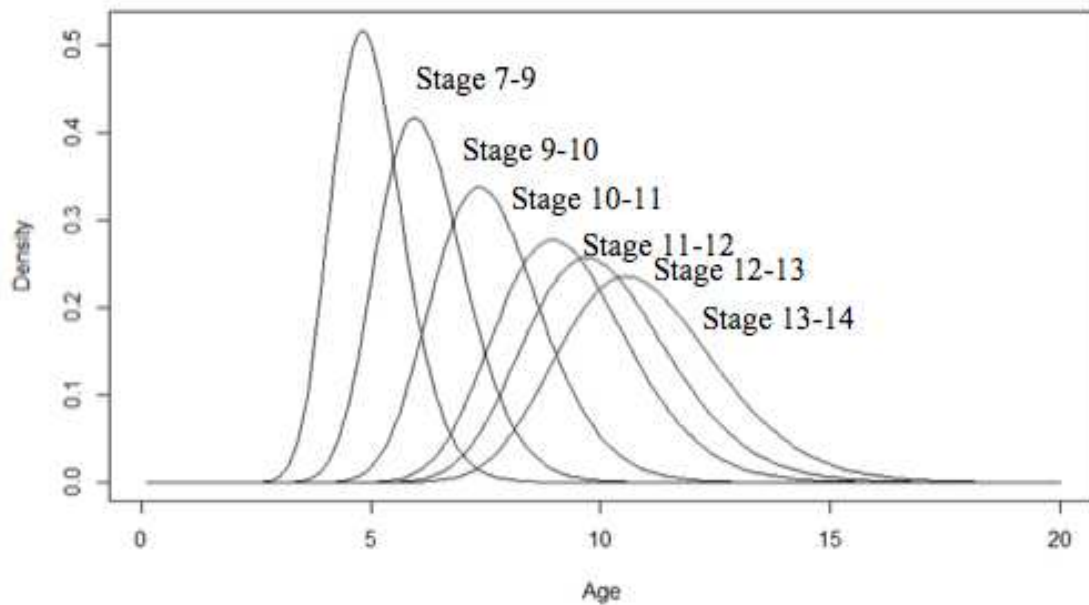
Lower Incisor 2, Female



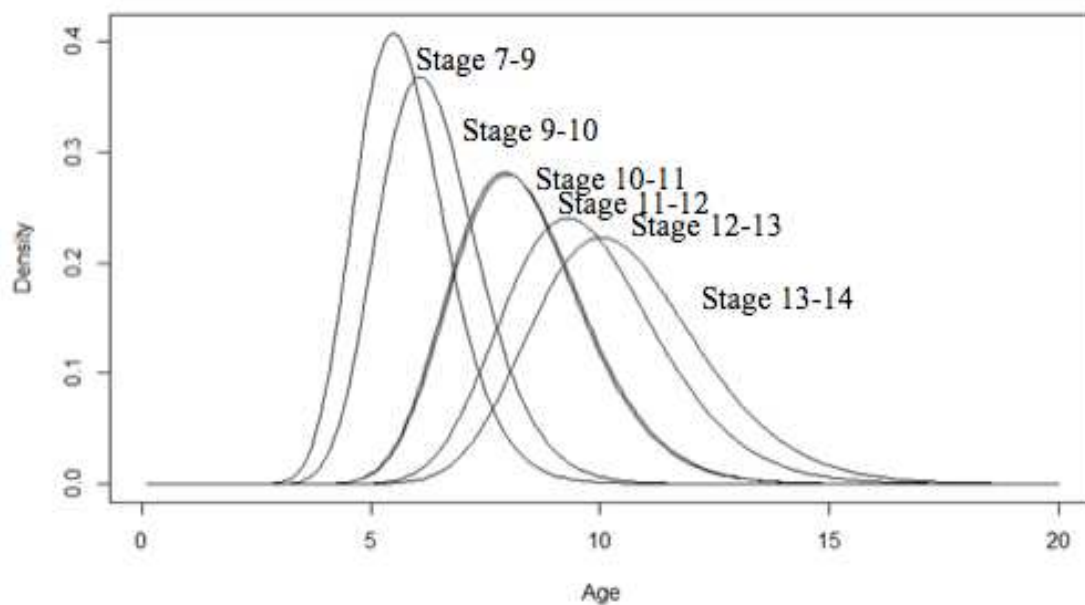
Lower Canine, Female



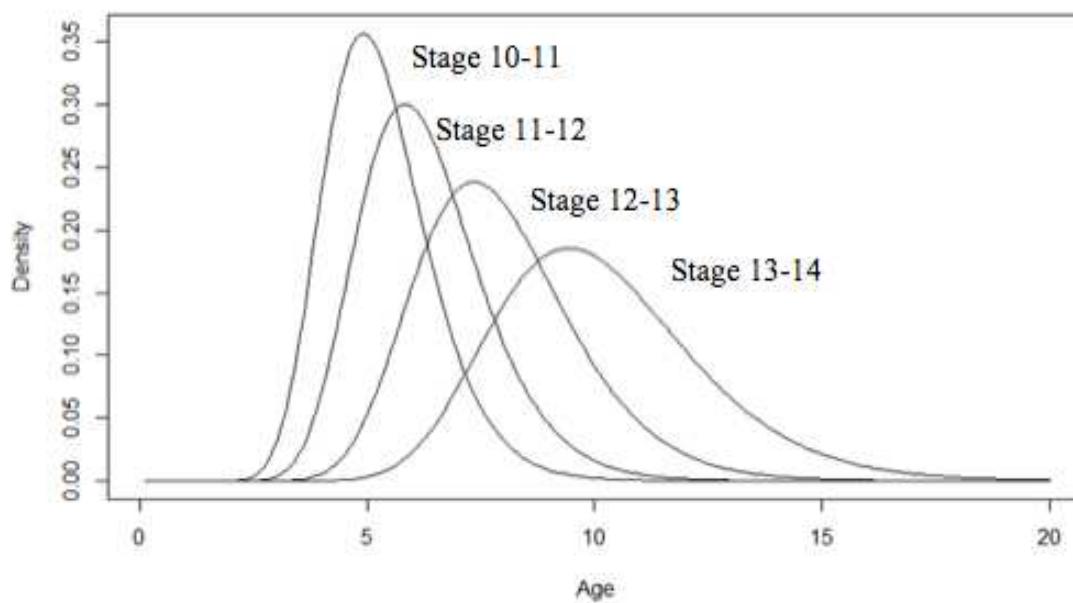
Lower Premolar 1, Female



Lower Premolar 2, Female



Lower Molar 1, Female



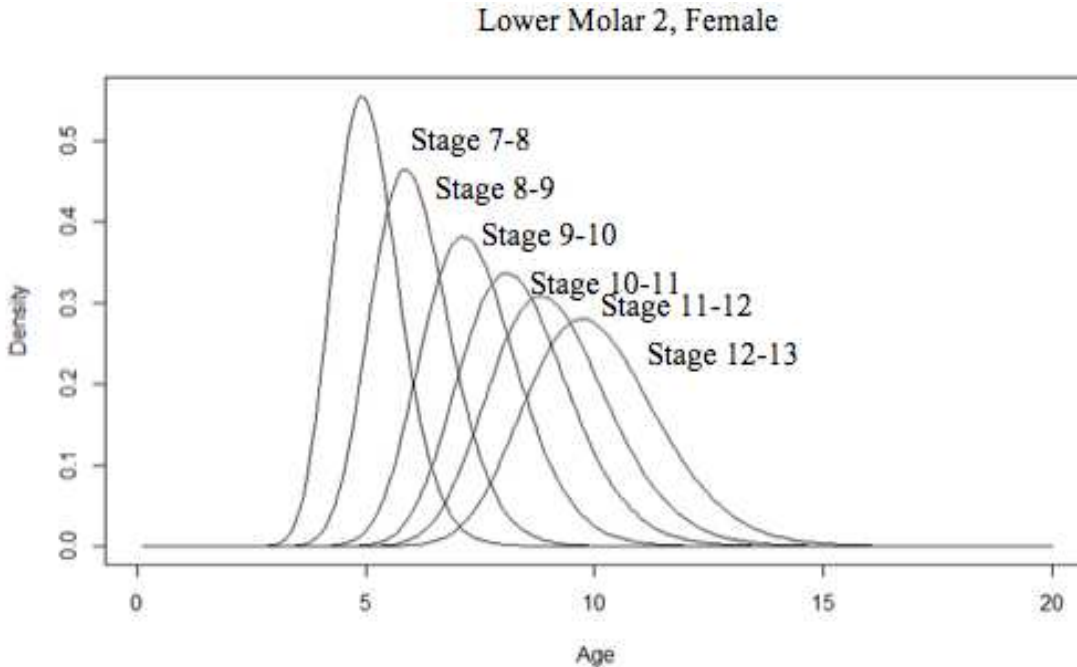


Figure 10: Age-at-transition distributions between dental development stages for all lower teeth of females.

HPD Results

Figure 11 demonstrates the impact of including an informed prior probability in age estimation for this data set. The black line represents the probability of death at a particular age, and the red line displays how it is shifted when the informed prior probability, which is represented by the blue line, is incorporated. It is important to note that the blue line is only representative of the informed prior probability, and not meant to be compared to the black and red lines. The informed prior probability (blue line) is what takes the probability of death at a particular age (black line) to the shifted and more precise probability of death at a particular age, or posterior probability (red line), which is the goal of this research.

HPD male molar 2, C3/4, US Violent Deaths 2010 prior

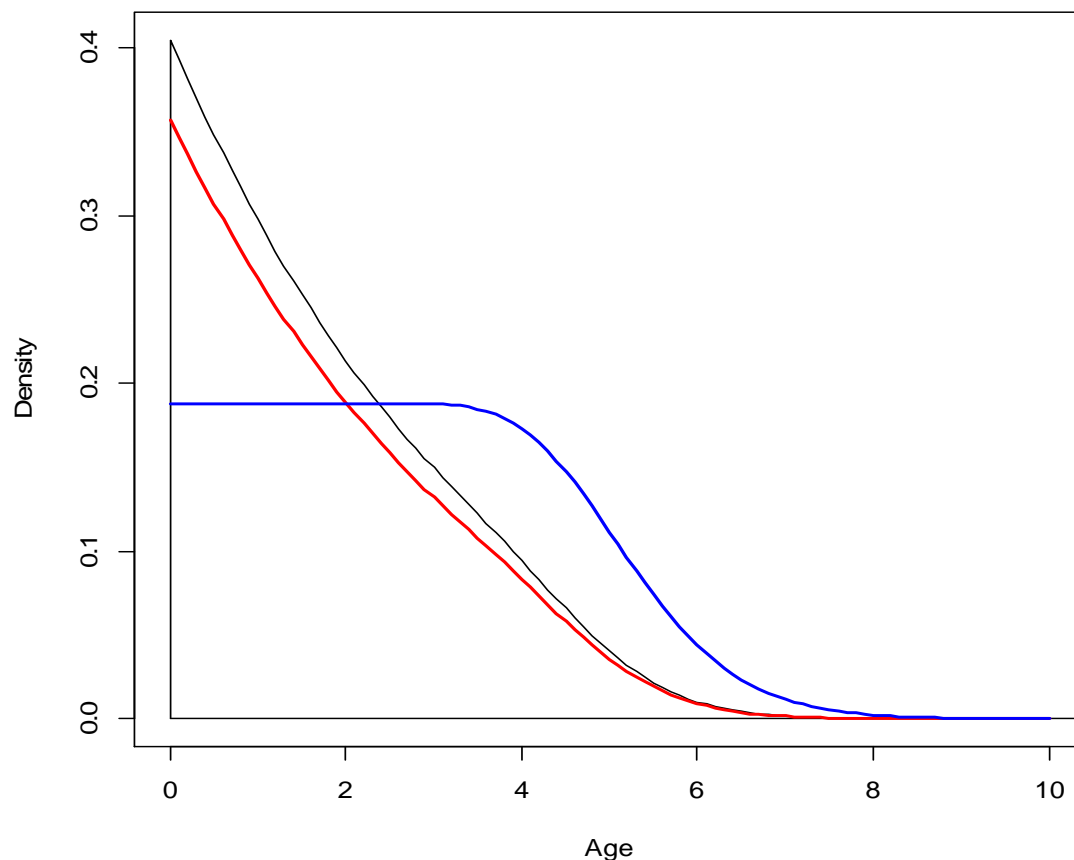


Figure 11: This graph, provided by Lyle Konigsberg, shows the impact of including an informed prior probability in age estimation for this data set. The blue line shows the likelihood that an individual is a particular age based on their dental development stage (in this case, crown $\frac{3}{4}$ complete for males). The red line is the informative prior probability of death at a particular age; in this case US violent deaths in 2010. The black thin line is the posterior density for age-at-death based on informative prior and likelihood.

Tables 4 through 7 present the Highest Posterior Density (HPD) and 50% probability age ranges for each tooth at each stage by sex and informed prior probability. This analysis used the male and female hazard parameters obtained for the Gompertz model (illustrated in Figures 12 and 13), providing the priors for US female mortality, 2008 ($\alpha_3= 0.498$, $\beta_3= -0.093$); US female violent deaths, 2010 ($\alpha_3= 0.415$, $\beta_3= -0.065$); US male mortality, 2008 ($\alpha_3= 0.479$, $\beta_3= -0.093$); and US male violent deaths, 2010 ($\alpha_3= 0.357$, $\beta_3= -0.0599$). These survivorship graphs illustrate an increased risk of death for subadults under the age of fifteen, with a drastic decrease in

survivability after the infant ages. It is possible that the slope would not have as extreme if the graph extended past the age of fifteen. As displayed in the graphs, the confidence curves have a loose fit for younger individuals, but become progressively tighter through the later ages. This demonstrates that the Gompertz model is less stable for lower ages. It is possible that this could be remedied by adjusting the age ranges for these younger individuals, such as changing them to monthly intervals for the first two to four years of life, rather than by year. This could create a confidence curve with a tighter fit, allowing for more accurate priors for individuals under the age of four.

When comparing the 2008 survivorship curve to the 2010 curve, we see that there is a slightly wider confidence interval for the 2008 data with more extreme priors. This implies that the 2008 priors will have a slightly greater impact on the data than the 2010 prior. The comparison of male and female curves shows little difference between the widths of the intervals. However, there is a difference in the posterior probabilities that are calculated. Therefore, each of the priors will provide different age ranges.

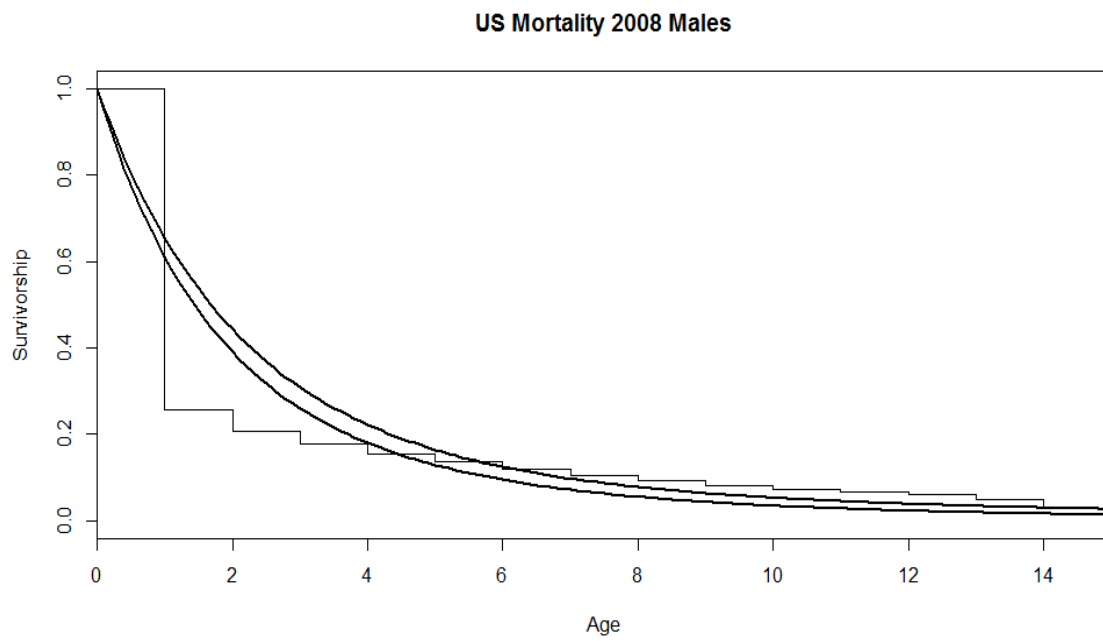
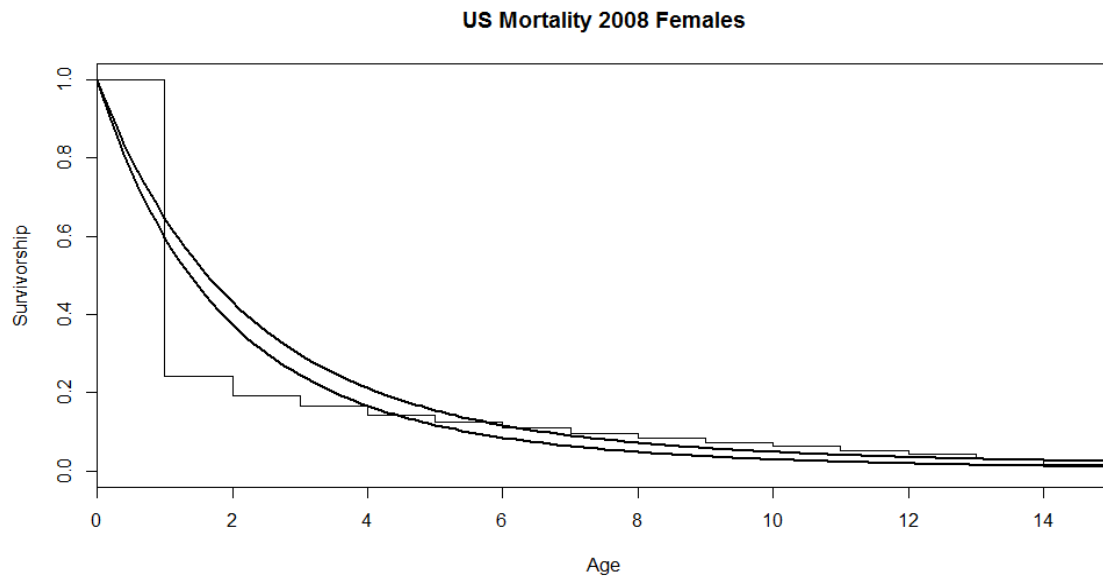


Figure 12: The 95% confidence interval on a Gompertz Makeham mortality model for male and females dying in 2008.

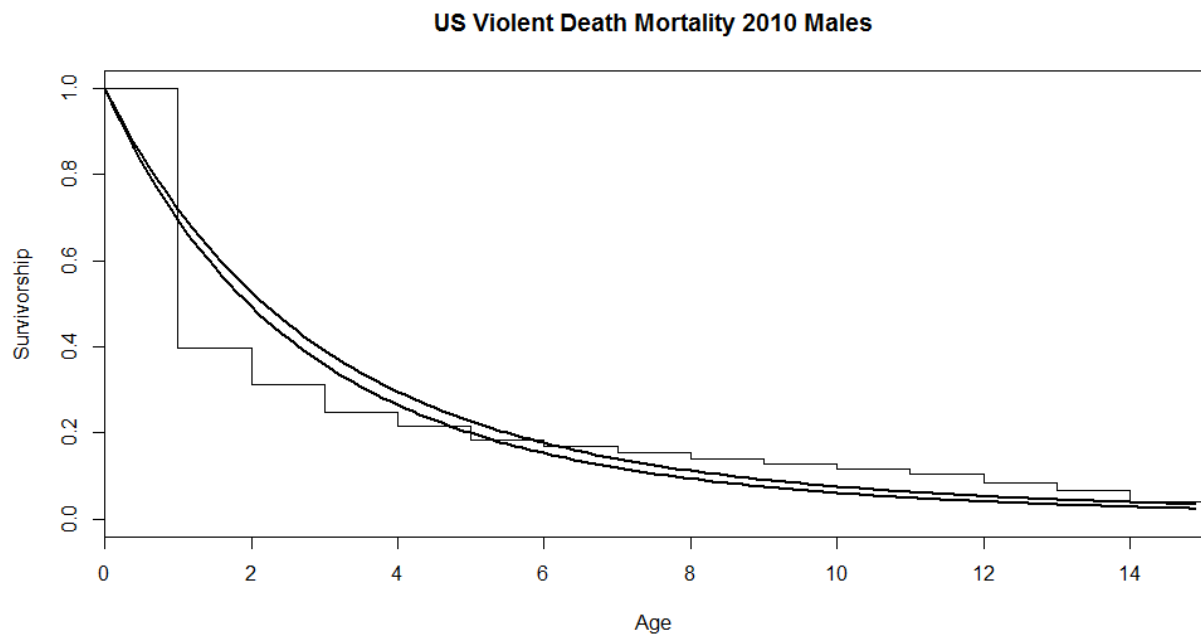
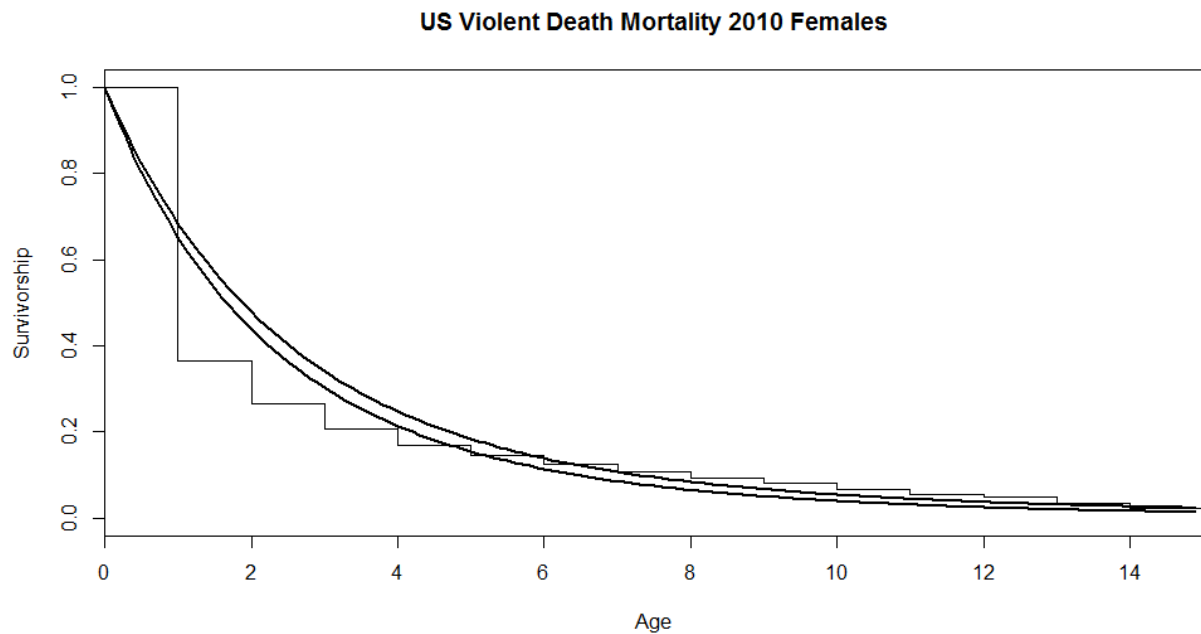
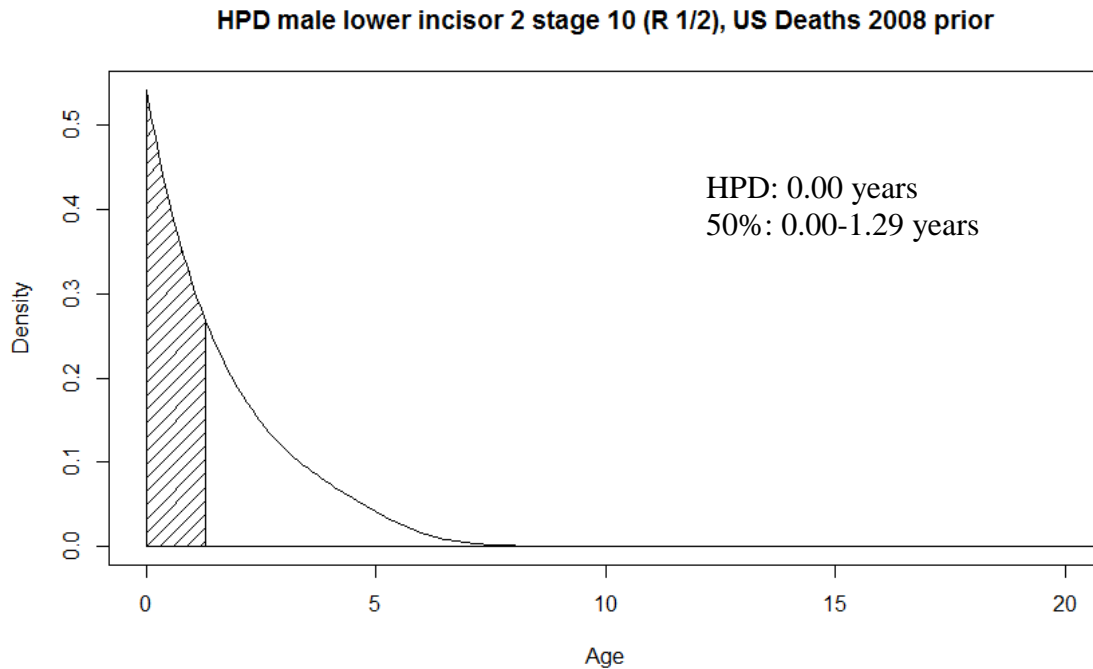


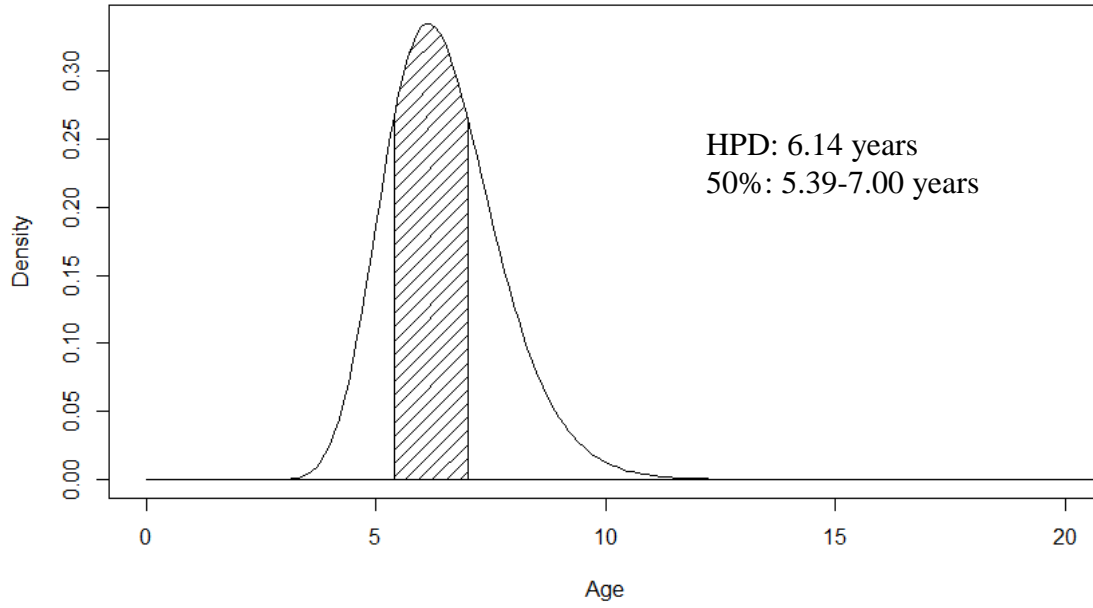
Figure 13: The 95% confidence interval on a Gompertz Makeham mortality model for male and females dying of violent deaths in 2010.

The priors derived from the Gompertz survivorship curves were incorporated into the calculation of the highest posterior density (HPD) of age for each tooth at each stage. Due to the large number of graphs produced by this analysis, only the results of the lower second incisors

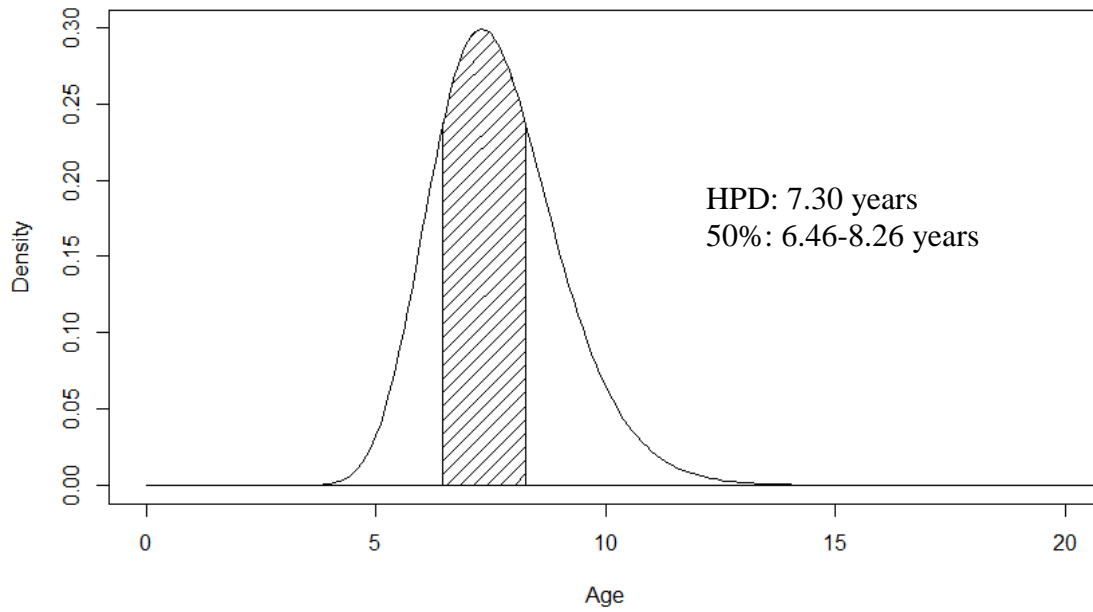
will be displayed in the paper, while the remaining graphs can be viewed in Appendix 5. The results of the 50% HPD of age for each lower second incisor stage for males are presented in Figure 14. There is a slight increase in age with each successive stage of transition, which is expected based on the transition analysis. These graphs provide exact probabilities, and a 50% probability that an individual displaying a certain Moorrees stage will fall in the specified age range. With these graphs, a stage for any tooth can be chosen at random with a corresponding age and the graphs will provide an accurate probability for the chosen scenario.



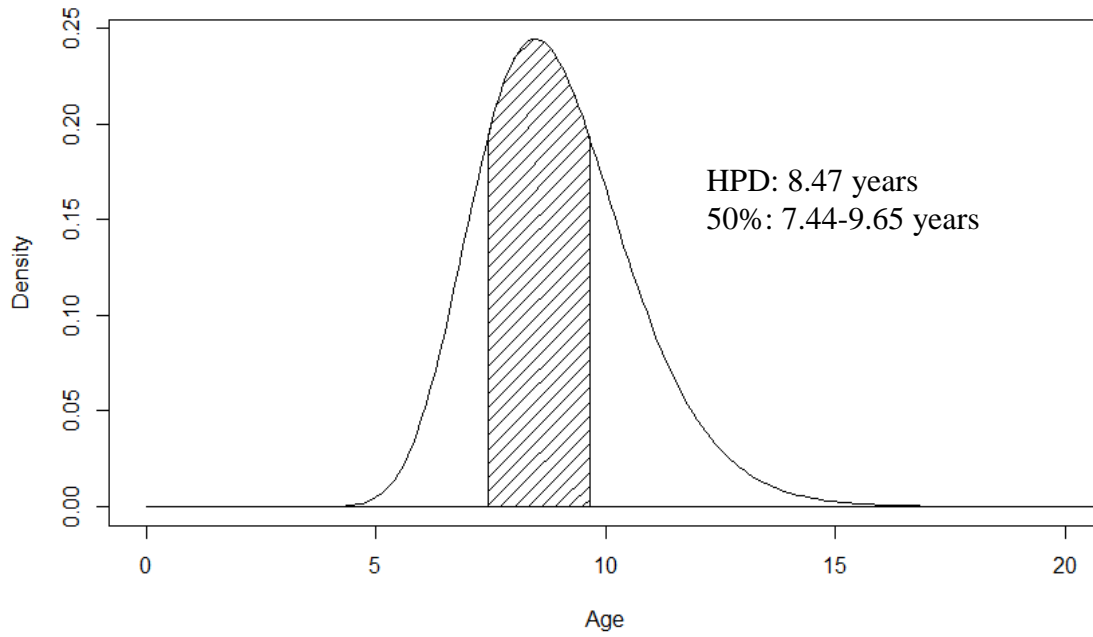
HPD male lower incisor 2 stage 11 (R 3/4), US Deaths 2008 prior



HPD male lower incisor 2 stage 12 (Rc), US Deaths 2008 prior



HPD male lower incisor 2 stage 13 (A 1/2), US Deaths 2008 prior



HPD male lower incisor 2 stage 14 (Ac), US Deaths 2008 prior

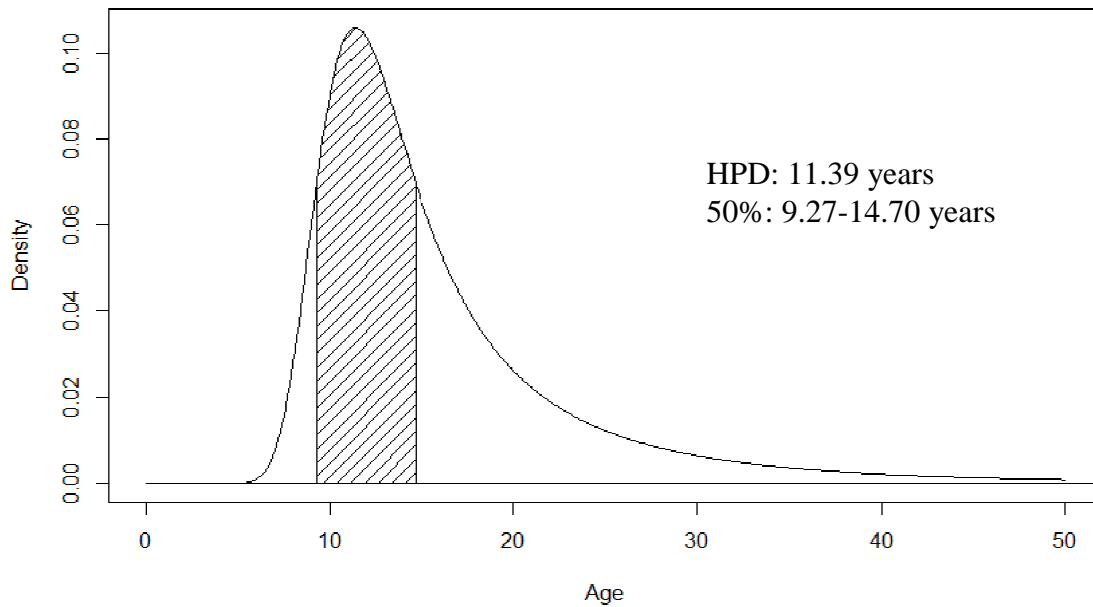


Figure 14: 50% HPD of individuals showing a particular stage of dental development at a particular stage, for stages 10 – 14 of the lower second incisor in males

The HPD tables (Tables 4-7), distinguished by sex and informed prior used, display the highest posterior density, which is age with the highest probability for each tooth at the specified stage, as well as the exact age range that falls within the 50% probability. The hash mark in these tables denote phases that were not observable, due a lack of representation of these age groups in my sample. The difference was provided for each age range to show the increase in width of the ranges as the ages increase. This is due to the small sample size, and the low numbers of individuals aged 11-12, causing Bayes' theorem to compensate by creating wider ranges to ensure the exact 50% probability. Although these widths are greater, they have the same likelihood as the more narrow widths.

Although there is always room for improvement, the HPD graphs presented here predict an exact 50% probability of age for each tooth at a specified Moorrees' tooth development stage. This becomes especially useful and more reliable when multiple teeth are examined. For example, a female upper canine at stage 11 and a female upper first molar at stage 13 provide age ranges of 6.39-9.15 and 4.67-8.96 respectively. If a female child is found with their upper canines and first molars at that stage, the age ranges can be combined to give a smaller and more reliable age range of 6.39-8.96. This only becomes more reliable with the addition of more teeth.

Table 4: Female Highest Posterior Density (HPD), Deaths 2008 Prior

Phase	Tooth	UI1	UI2	UC	UPM1	UPM2	UM1	UM2	LI1	LI2	LC	LPM1	LPM2	LM1	LM2
5	HPD	-	-	-	-	-	-	-	-	-	-	-	-	-	5.21
	50%	-	-	-	-	-	-	-	-	-	-	-	-	-	4.70-5.78
	<i>Difference</i>	-	-	-	-	-	-	-	-	-	-	-	-	-	1.08
6	HPD	-	-	-	5.16	3.91	-	4.69	-	-	-	-	5.51	-	6.23
	50%	-	-	-	4.60-5.79	3.19-4.78	-	3.90-5.63	-	-	-	-	4.86-6.25	-	5.61-6.92
	<i>Difference</i>	-	-	-	1.19	1.59	-	1.73	-	-	-	-	1.39	-	1.31
7	HPD	-	-	-	6.33	4.94	-	5.72	-	-	-	5.41	6.49	-	7.32
	50%	-	-	-	6.65-7.10	4.01-6.09	-	4.73-6.92	-	-	-	4.71-6.23	5.67-7.43	-	6.62-8.10
	<i>Difference</i>	-	-	-	0.45	2.08	-	2.19	-	-	-	1.52	1.76	-	1.48
8	HPD	-	-	-	-	-	-	7.31	-	-	-	-	-	-	8.14
	50%	-	-	-	-	-	-	6.07-8.80	-	-	-	-	-	-	7.37-8.99
	<i>Difference</i>	-	-	-	-	-	-	2.73	-	-	-	-	-	-	1.62
9	HPD	-	-	4.37	7.73	6.6	-	8.57	-	-	-	7.29	8.03	-	8.91
	50%	-	-	3.59-5.33	6.90-8.66	5.37-8.11	-	7.14-10.29	-	-	-	6.43-8.28	7.06-9.13	-	8.07-9.84
	<i>Difference</i>	-	-	1.74	1.76	2.74	-	3.15	-	-	-	1.85	2.07	-	1.77
10	HPD	-	5.05	6.24	8.9	8.23	2.43	9.73	-	-	7.53	8.58	9.04	-	9.98
	50%	-	4.16-6.12	5.21-7.48	7.99-9.92	6.73-10.08	1.70-3.44	8.11-11.70	-	-	6.87-8.25	7.59-9.68	7.97-10.25	-	9.02-11.04
	<i>Difference</i>	-	1.96	2.27	1.93	3.35	1.74	3.59	-	-	1.38	2.09	2.28	-	2.02
11	HPD	5.16	6.22	7.65	9.64	9.63	3.63	10.97	5.19	6.13	8.62	9.61	9.99	5.06	11.01
	50%	4.31-6.17	5.15-7.51	6.39-9.15	8.66-10.75	7.86-11.81	2.60-5.04	9.18-13.12	4.59-5.88	5.40-6.96	7.87-9.45	8.50-10.86	8.79-11.35	4.34-5.89	9.98-12.15
	<i>Difference</i>	1.86	2.36	2.76	2.09	3.95	2.44	3.94	1.29	1.56	1.58	2.36	2.56	1.55	2.17
12	HPD	6.4	7.33	9	10.44	11.26	4.78	11.77	6.5	7.02	9.78	10.78	11.12	6.08	11.57
	50%	5.37-7.62	6.06-8.87	7.54-10.76	9.37-11.63	9.17-13.83	3.44-6.65	9.80-14.15	5.81-7.29	6.18-7.98	8.94-10.71	9.55-12.18	9.81-12.61	5.21-7.10	10.49-12.77
	<i>Difference</i>	2.25	2.81	3.22	2.26	4.66	3.21	4.35	1.48	1.8	1.77	2.63	2.8	1.89	2.28
13	HPD	7.72	8.83	10.62	11.26	15.08	6.46	14.74	7.59	8.39	10.83	11.66	12.02	7.6	13.76
	50%	6.46-9.24	7.31-10.69	8.85-12.74	10.11-12.55	11.56-20.18	4.67-8.96	11.47-19.48	6.77-8.51	7.35-9.58	9.90-11.84	10.34-13.15	10.60-13.63	6.50-8.89	11.56-17.31
	<i>Difference</i>	2.78	3.38	3.89	2.44	8.62	4.29	8.01	1.74	2.23	1.94	2.81	3.03	2.39	5.75
14	HPD	10.69	11.9	14.45	13.73	-	9.63	-	9.8	11.26	13.05	14.14	14.82	10.46	-
	50%	8.30-14.25	9.15-15.95	11.29-19.03	11.42-17.35	-	6.52-14.55	-	8.05-12.64	9.15-14.53	11.07-16.40	11.62-17.96	12.14-18.83	8.29-13.74	-
	<i>Difference</i>	5.95	6.8	7.74	5.93	-	8.03	-	4.59	5.38	5.33	6.34	6.69	5.45	-

Table 5: Female Highest Posterior Density (HPD), Violent Deaths 2010 Prior

Phase	Tooth	UI1	UI2	UC	UPM1	UPM2	UM1	UM2	LI1	LI2	LC	LPM1	LPM2	LM1	LM2
5	HPD	-	-	-	-	-	-	-	-	-	-	-	-	-	5.23
	50%	-	-	-	-	-	-	-	-	-	-	-	-	-	4.72-5.80
	<i>Difference</i>	-	-	-	-	-	-	-	-	-	-	-	-	-	1.08
6	HPD	-	-	-	5.19	3.97	-	4.75	-	-	-	-	5.55	-	6.26
	50%	-	-	-	4.63-5.82	3.24-4.86	-	3.96-5.71	-	-	-	-	4.89-6.29	-	5.64-6.95
	<i>Difference</i>	-	-	-	1.19	1.62	-	1.75	-	-	-	-	1.4	-	1.31
7	HPD	-	-	-	6.37	5.03	-	5.8	-	-	-	5.45	6.53	-	7.35
	50%	-	-	-	5.68-7.14	4.08-6.20	-	4.80-7.01	-	-	-	4.75-6.28	5.71-7.48	-	6.65-8.12
	<i>Difference</i>	-	-	-	1.46	2.12	-	2.21	-	-	-	1.53	1.77	-	1.47
8	HPD	-	-	-	-	-	-	7.39	-	-	-	-	-	-	8.16
	50%	-	-	-	-	-	-	6.15-8.89	-	-	-	-	-	-	7.40-9.01
	<i>Difference</i>	-	-	-	-	-	-	2.74	-	-	-	-	-	-	1.61
9	HPD	-	-	4.44	7.76	6.7	-	8.64	-	-	-	7.33	8.07	-	8.93
	50%	-	-	3.65-5.42	6.93-8.69	5.46-8.22	-	7.21-10.35	-	-	-	6.47-8.32	7.10-9.17	-	8.09-9.86
	<i>Difference</i>	-	-	1.77	1.76	2.76	-	3.14	-	-	-	1.85	2.07	-	1.77
10	HPD	-	5.12	6.32	8.93	8.32	2.52	9.79	-	-	7.55	8.61	9.07	-	9.99
	50%	-	4.23-6.21	5.28-7.56	8.02-9.94	6.81-10.16	1.77-3.58	8.17-11.73	-	-	6.89-8.27	7.63-9.71	8.00-10.28	-	9.03-11.05
	<i>Difference</i>	-	1.98	2.28	1.92	3.35	1.81	3.56	-	-	1.38	2.08	2.28	-	2.02
11	HPD	5.23	6.3	7.72	9.66	9.69	3.78	11	5.23	6.17	8.64	9.63	10.01	5.11	11.02
	50%	4.37-6.25	5.23-7.59	6.46-9.22	8.68-10.76	7.93-11.85	2.71-5.23	9.18-13.19	4.62-5.92	5.43-7.00	7.89-9.46	8.53-10.88	8.82-11.37	4.39-5.94	9.99-12.16
	<i>Difference</i>	1.88	2.36	2.76	2.08	3.92	2.52	4.01	1.3	1.57	1.57	2.35	2.55	1.55	2.17
12	HPD	6.47	7.42	9.06	10.45	11.28	4.98	11.78	6.54	7.06	9.79	10.8	11.13	6.14	11.58
	50%	5.44-7.70	6.14-8.96	7.60-10.81	9.39-11.64	9.22-13.82	3.59-6.88	9.83-14.12	5.84-7.32	6.22-8.02	8.95-10.72	9.57-12.19	9.82-12.62	5.26-7.16	10.50-12.77
	<i>Difference</i>	2.26	2.82	3.21	2.25	4.6	3.29	4.29	1.48	1.8	1.77	2.62	2.8	1.9	2.27
13	HPD	7.8	8.91	10.65	11.27	14.93	6.69	14.62	7.62	8.43	10.83	11.67	12.02	7.66	13.73
	50%	6.53-9.31	7.38-10.75	8.90-12.75	10.12-12.55	11.48-19.88	4.82-9.26	11.42-19.22	6.80-8.54	7.39-9.61	9.91-11.84	10.35-13.15	10.61-13.62	6.55-8.95	11.56-17.16
	<i>Difference</i>	2.78	3.37	3.85	2.43	8.4	4.44	7.8	1.74	2.22	1.93	2.8	3.01	2.4	5.6
14	HPD	10.74	11.91	14.35	13.7	-	9.81	-	9.84	11.28	13.04	14.09	14.75	10.5	-
	50%	8.38-14.19	9.21-15.82	11.26-18.79	11.43-17.19	-	6.73-14.54	-	8.11-12.62	9.20-14.45	11.08-16.27	11.62-17.79	12.12-18.64	8.36-13.69	-
	<i>Difference</i>	5.81	6.61	7.53	5.76	-	7.81	-	4.51	5.25	5.19	6.17	6.52	5.33	-

Table 6: Male Highest Posterior Density (HPD), Deaths 2008 Prior

Phase	Tooth	UI1	UI2	UC	UPM1	UPM2	UM1	UM2	LI1	LI2	LC	LPM1	LPM2	LM1	LM2
5	HPD	-	-	-	-	-	-	4.37	-	-	-	-	-	-	-
	50%	-	-	-	-	-	-	3.68-5.19	-	-	-	-	-	-	-
	<i>Difference</i>	-	-	-	-	-	-	1.51	-	-	-	-	-	-	-
6	HPD	-	-	-	-	-	-	4.72	-	-	-	-	4.33	-	5.5
	50%	-	-	-	-	-	-	3.98-5.61	-	-	-	-	3.61-5.19	-	4.79-6.32
	<i>Difference</i>	-	-	-	-	-	-	1.63	-	-	-	-	1.58	-	1.53
7	HPD	-	-	-	5.94	6.61	-	5.77	-	-	-	6.14	5.62	-	7
	50%	-	-	-	5.21-6.78	5.90-7.42	-	4.80-6.96	-	-	-	5.45-6.92	4.60-6.87	-	6.13-8.01
	<i>Difference</i>	-	-	-	1.57	1.52	-	2.16	-	-	-	1.47	2.27	-	1.88
8	HPD	-	-	-	-	-	-	7.84	-	-	-	-	-	-	8.18
	50%	-	-	-	-	-	-	6.56-9.37	-	-	-	-	-	-	7.19-9.30
	<i>Difference</i>	-	-	-	-	-	-	2.81	-	-	-	-	-	-	2.11
9	HPD	-	-	5.55	7.91	8.4	-	9.57	-	-	5.4	7.97	7.71	-	9.1
	50%	-	-	4.73-6.52	7.03-8.90	7.55-9.35	-	8.05-11.39	-	-	4.54-6.44	7.16-8.87	6.43-9.23	-	7.99-10.36
	<i>Difference</i>	-	-	1.79	1.87	1.8	-	3.34	-	-	1.9	1.71	2.8	-	2.37
10	HPD	-	5.05	7.42	9.32	9.91	4.11	11.1	-	-	7.25	9.24	8.91	-	10.25
	50%	-	4.11-6.20	6.38-8.63	8.32-10.44	8.94-10.99	3.36-5.02	9.35-13.18	-	-	6.16-8.54	8.34-10.25	7.43-10.69	-	9.01-11.67
	<i>Difference</i>	-	2.09	2.25	2.12	2.05	1.66	3.83	-	-	2.38	1.91	3.26	-	2.66
11	HPD	4.33	6.77	8.95	10.26	10.79	5.26	12.64	-	6.14	8.63	10.17	10.16	6.32	11.64
	50%	3.47-5.40	5.59-8.21	7.73-10.37	9.18-11.47	9.76-11.92	4.30-6.45	10.63-15.05	-	5.39-7.00	7.35-10.14	9.18-11.27	8.50-12.15	5.72-6.98	10.21-13.27
	<i>Difference</i>	1.93	2.62	2.64	2.29	2.16	2.15	4.42	-	1.61	2.79	2.09	3.65	1.26	3.06
12	HPD	6.3	7.78	10.37	10.92	11.31	6.93	16.16	6.07	7.3	9.94	10.85	10.91	7.35	14.95
	50%	5.13-7.75	6.43-9.42	8.95-12.02	9.77-12.21	10.22-12.51	5.66-8.50	12.73-21.11	5.31-6.94	6.46-8.26	8.46-11.68	9.80-12.01	9.14-13.01	6.68-8.08	12.20-19.06
	<i>Difference</i>	2.62	2.99	3.07	2.44	2.29	2.84	8.38	1.63	1.8	3.22	2.21	3.87	1.4	6.86
13	HPD	7.91	9.38	13.48	13.21	13.64	8.89	-	7.29	8.47	11.89	11.85	13.63	8.4	-
	50%	6.46-9.70	7.70-11.45	10.80-17.47	10.92-16.81	11.41-17.23	7.29-10.85	-	6.39-8.32	7.44-9.65	10.08-14.05	10.67-13.17	10.59-18.08	7.61-9.27	-
	<i>Difference</i>	3.24	3.75	6.67	5.89	5.82	3.56	-	1.93	2.21	3.97	2.5	7.49	1.66	-
14	HPD	10.7	13.62	-	-	-	12.16	-	9.54	11.39	16.24	14.92	-	10.7	-
	50%	8.09-14.59	10.49-18.21	-	-	-	9.27-16.42	-	7.68-12.49	9.27-14.70	12.94-21.01	12.50-18.75	-	8.96-13.69	-
	<i>Difference</i>	6.5	7.72	-	-	-	7.15	-	4.81	5.43	8.07	6.25	-	4.73	-

Table 7: Male Highest Posterior Density (HPD), Violent Deaths 2010 Prior

Phase	Tooth	UI1	UI2	UC	UPM1	UPM2	UM1	UM2	LI1	LI2	LC	LPM1	LPM2	LM1	LM2
5	HPD	-	-	-	-	-	-	4.46	-	-	-	-	-	-	-
	50%	-	-	-	-	-	-	3.76-5.29	-	-	-	-	-	-	-
	<i>Difference</i>	-	-	-	-	-	-	1.53	-	-	-	-	-	-	-
6	HPD	-	-	-	-	-	-	4.83	-	-	-	-	4.43	-	5.58
	50%	-	-	-	-	-	-	4.06-5.73	-	-	-	-	3.69-5.31	-	4.86-6.41
	<i>Difference</i>	-	-	-	-	-	-	1.67	-	-	-	-	1.62	-	1.55
7	HPD	-	-	-	6.01	6.68	-	5.92	-	-	-	6.2	5.78	-	7.09
	50%	-	-	-	5.28-6.86	5.96-7.49	-	4.92-7.13	-	-	-	5.51-7.00	4.74-7.06	-	6.21-8.11
	<i>Difference</i>	-	-	-	1.58	1.53	-	2.21	-	-	-	1.49	2.32	-	1.9
8	HPD	-	-	-	-	-	-	8.01	-	-	-	-	-	-	8.27
	50%	-	-	-	-	-	-	6.71-9.55	-	-	-	-	-	-	7.27-9.40
	<i>Difference</i>	-	-	-	-	-	-	2.84	-	-	-	-	-	-	2.13
9	HPD	-	-	5.66	7.98	8.47	-	9.74	-	-	5.53	8.03	7.87	-	9.19
	50%	-	-	4.82-6.64	7.10-8.98	7.61-9.42	-	8.20-11.56	-	-	4.64-6.58	7.22-8.94	6.58-9.42	-	8.08-10.46
	<i>Difference</i>	-	-	1.82	1.88	1.81	-	3.36	-	-	1.94	1.72	2.84	-	2.38
10	HPD	-	5.2	7.54	9.39	9.97	4.22	11.26	-	-	7.39	9.31	9.09	-	10.34
	50%	-	4.23-6.39	6.49-8.76	8.39-10.51	8.99-11.05	3.45-5.16	9.47-13.39	-	-	6.28-8.69	8.40-10.31	7.59-10.88	-	9.09-11.77
	<i>Difference</i>	-	2.16	2.27	2.12	2.06	1.71	3.92	-	-	2.41	1.91	3.29	-	2.68
11	HPD	4.47	6.95	9.07	10.33	10.84	5.42	12.78	-	6.22	8.77	10.23	10.33	6.37	11.73
	50%	3.59-5.59	5.74-8.41	7.84-10.50	9.25-11.55	9.81-11.98	4.42-6.64	10.76-15.18	-	5.46-7.09	7.48-10.29	9.24-11.33	8.66-12.33	5.76-7.04	10.30-13.35
	<i>Difference</i>	2	2.67	2.66	2.3	2.17	2.22	4.42	-	1.63	2.81	2.09	3.67	1.28	3.05
12	HPD	6.49	7.97	10.49	10.99	11.36	7.13	16.27	6.15	7.38	10.08	10.9	11.07	7.4	15.04
	50%	5.29-7.98	6.60-9.63	9.07-12.14	9.84-12.28	10.28-12.56	5.83-8.73	12.81-21.25	5.39-7.03	6.53-8.35	8.59-11.83	9.86-12.06	9.31-13.18	6.73-8.13	12.28-19.19
	<i>Difference</i>	2.69	3.03	3.07	2.44	2.28	2.9	8.44	1.64	1.82	3.24	2.2	3.87	1.4	6.91
13	HPD	8.13	9.6	13.62	13.31	13.72	9.1	-	7.38	8.56	12.03	11.91	13.82	8.45	-
	50%	6.65-9.93	7.90-11.67	10.92-17.66	11.00-16.97	11.47-17.37	7.48-11.08	-	6.47-8.42	7.52-9.75	10.21-14.18	10.73-13.22	10.75-18.30	7.66-9.33	-
	<i>Difference</i>	3.28	3.77	6.74	5.97	5.9	3.6	-	1.95	2.23	3.97	2.49	7.55	1.67	-
14	HPD	11	13.83	-	-	-	12.42	-	9.71	11.53	16.33	14.98	-	10.81	-
	50%	8.34-14.96	10.66-18.45	-	-	-	9.49-16.72	-	7.80-12.79	9.38-14.93	13.01-21.14	12.55-18.86	-	9.04-13.91	-
	<i>Difference</i>	6.62	7.79	-	-	-	7.23	-	4.99	5.55	8.13	6.31	-	4.87	-

Discussion

The four highest posterior density tables (Tables 4-7) presented in the previous chapter can be used to estimate age in other studies. Although Bayes' theorem is effective in estimating individual age in a forensic context, and in some cases an archaeological context, these tables are most appropriate for use in forensic cases. The informative priors used to develop the age ranges in Tables 4-7 are based on modern populations. Of course, age estimates in forensic cases should be based on multiple age indicators whenever possible, due to the possibility of advanced or delayed development of skeletal indicators in some areas relative to others (Schulz R, *et al.* 2008, Langley-Shirley and Jantz 2010, Klepinger L 2001).

As expected, each prior probability produces different age ranges for males and females. Although the widths of the age ranges vary, they are each an exact 50% probability, and thus have the same likelihood for reliability. For each tooth with any prior, the widths increase as the stages progress. Due to the low frequency of older individuals in this sample, Bayes' theorem attempts to compensate by increasing the width of the age range to remain an exact 50% probability. Thus, the age estimates of younger individuals using these ranges will have more precise age estimates than older children.

Since these age ranges differ depending on the prior that was incorporated, it is important to choose the prior that is appropriate for the sex of the individual, which presents a new issue. No tested method has been found to provide an accurate estimation of sex of a subadult, who has not reached puberty (and therefore have not developed primary and secondary sexual characteristics) (Boucher 1955, Boucher 1957, Rösing 1983, Thomson 1899). Although many authors have studied the timing of permanent tooth formation in relation to sex, dental development does not differ between the sexes in a predictable manner. Therefore, determining

the correct prior to use in determination of age for an unknown individual becomes much more complicated. For this reason, it is recommended that an overall prior that does not rely on the sex of the individual be incorporated into the model.

As shown in the survivorship models, the Gompertz-Makeham model is less stable for the younger individuals in this sample. This could be corrected by adjusting the age ranges for these younger individuals, such as changing them to monthly intervals for the first two to four years of life, rather than by year. This could create a confidence curve with a tighter fit, allowing for more accurate priors for individuals under the age of four. In addition, a Siler model of mortality better represents the mortality patterns of younger individuals than the Gompertz-Makeham (Wood *et al.* 2002), and should be used in future applications. The Siler model is preferred because it adds a third component to the Gompertz-Makeham model in order to represent the earliest segment of life. This early segment is when the risk of death declines rapidly (Wood *et al.* 2002). These early stages of life are where the Gompertz-Makeham model struggled to create a confidence curve with a tight match in this sample.

This project is still in its infant stages, and there are several adjustments that should be made when conducting similar future research. As discussed, the sample I derived from the Maxwell Museum was relatively small and only included an age range of five to twelve. To create more precise age ranges, a wider distribution of ages, particularly earlier than five years, is necessary. It could also be beneficial to have information of age by month. Incorporating children under the age of five will allow for the initial stages of tooth development to be observed and recorded. Unfortunately in this case, those age groups were not available and therefore the final age ranges were nonexistent or omitted for inaccuracy. Another suggestion is to increase the maximum age. Although tooth development usually ceases after the age of

twelve, this addition would permit for Bayes' theorem to factor in these stages, allowing for smaller posterior probability age ranges for the later stages of development. This would prevent the compensation for a lack of information, which is seen in the results of this study.

In addition, a method for simultaneously using multiple teeth to generate an overall HPD for an individual would provide a more useful means for generating unbiased age estimates of a subadult individual. This would alleviate the issue of determining the sex of a subadult, allowing for the investigator to rely on an overall prior, rather than one based on the sex of the individual.

Finally, the methods described here require the use of the statistical software program R. This necessitates researchers to learn coding for R in order to reproduce these methods. I have provided the codes for each step of these analyses in the Appendix, to alleviate some of these computational burdens. Also, computer simulation methods and related software have been developed that can aid in the recreation of these methods (Geyer 1992, Gilks *et al.* 1996, Gamerman 1997, Lunn *et al.* 2000, 2009, Brooks *et al.* 2011, Konigsberg and Frankenberg 2013).

There is always room for improvement, but the outcomes of this research are very encouraging. Even with the small and narrow sample that was utilized in this research, the methods employed here do make reliable predictions for the data. The posterior probabilities that resulted from this study can be applied to future forensic cases by providing age ranges of an exact 50% probability. If the appropriate adjustments are made in future research, the methods discussed here will create very accurate predictions of age based on dental eruption. Incorporating appropriate prior probabilities allow for these predictions, and therefore anthropology should seek to employ Bayes' theorem whenever possible.

Conclusion

Bayesian statistics is a compelling tool that should be used more often in anthropological investigations. The ability to incorporate prior probabilities into our formulae has proven to be a new advantage in anthropology, and has aided in alleviating the biases we struggle with. The age mimicry bias continues to haunt anthropologists as we strive to improve our methods and develop new ways to control for it. Although there have been few successful attempts, Bayesian statistics presents a strong case for alleviating the stress of this bias, and providing the prior information needed to improve our standard aging methods.

Although there are several limitations to this study, future research on this topic looks promising. With larger sample sizes incorporating older and younger children, we can expect smaller, more precise age ranges that cover the entire spectrum of Moorrees' dental developmental stages. These larger samples will provide more information on each stage and the younger individuals will provide data on the stages that were missing from this study. The incorporation of older individuals provides evidence regarding the ceasing of dental development, when individuals reach the final, fourteenth stage. Even with the small and narrow sample that was utilized in this research, the methods employed here do make reliable predictions for the data. With this narrow sample, I was able to create age ranges with an exact 50% probability that is applicable to any forensic case concerning the estimation of age through dental development. However, the development of a stronger sample can only produce superior results.

Throughout the process of this project, it has been determined that other than sample size, other adjustments should also be made in future studies. Although the Gompertz-Makeham model is acceptable, the Siler model provides a stronger correlation for younger individuals, and is therefore more appropriate for this study (Wood *et al.* 2002). This would provide more

meaningful priors, with more accurate results. Also, since there is a shift in the age ranges dependent on the prior used, it would be beneficial if an overall prior were developed. This would allow researchers to use a single prior, rather than attempting to estimate the sex of a subadult, which we know is a precarious mission. An overall HPD table would be applicable to all unidentified subadults, which would be more suitable and realistic on a case-by-case basis.

This research project has provided successful results to the questions I sought to answer, and produced persuasive evidence for the next steps in the fight to control for the age mimicry bias. With the methods in this thesis, there is a better understanding of why this bias exists, and some of the ways it can be alleviated. Bayesian statistics has proven to be a significant tool within anthropological research, and I think it has the potential to depress this bias in future research.

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Appendix 1 – R code for transition analysis

```
{  
  age=seq(0.1,20,.1)  
  plot(age,dlnorm(age,mus[1],SD),type='l',xlab='Age',ylab='Density')  
  for(i in 2:8) lines(age,dlnorm(age,mus[i],SD))  
}
```

Appendix 2 - R code for Gompertz-Makeham survivorship model using the Violent Deaths in 2010 prior

```
gompviolent=function ()
{
  library(survival)
  makeham<-function(t,a3,b3)
  {

    a2<-0
    shift<-5
    h.t<-a2+a3*exp(b3*(t-shift))
    S.t<-exp(-a2*(t-shift)+a3/b3*(1-exp(b3*(t-shift))))
    return(S.t)
  }

  ages=violentdeathfemale$age

  plot(survfit(Surv(ages)~1),lty=c(0,1),lwd=c(1,1),xlim=c(0,15),
       main='US Violent Death Mortality 2010 Females',xlab='Age',ylab='Survivorship')

  #####

  jfs.optim<-function (x)
  {
```

```

makeham2<-function(t,x)
{
  a2<-0
  a3<-x[1]
  b3<-x[2]
  shift<-5
  h.t<-a2+a3*exp(b3*(t-shift))
  S.t<-exp(-a2*(t-shift)+a3/b3*(1-exp(b3*(t-shift))))
  return(S.t*h.t)
}

```

```

t<-ages
lnlk<-0
for(i in 1:1700){
  lnlk<-lnlk+log(makeham2(t[i],x))
}

```

```

return(lnlk)
}

```

```
#####
```

```

sto<-optim(c(.01,.05),jfs.optim,control=list(fnscale=-1),hessian=T)
print(sto)

library(MASS)

myenv <- new.env()

assign("a3",sto$par[1], env = myenv)

assign("b3", sto$par[2], env = myenv)

assign("t", seq(5:15), env = myenv)

dt<-numericDeriv(quote(makeham(seq(5:15), a3, b3)), c("a3", "b3"), myenv)

est<-dt[1:150]

gr<-attr(dt,'gradient')

V<-ginv(-sto$hess)

lo<-0

hi<-0

for(i in 1:150){

  se<-sqrt(as.vector(gr[i,]%*%V*%gr[i,]))

  lo[i]<-est[i]-1.96*se

  hi[i]<-est[i]+1.96*se

  if(lo[i]<0) lo[i]<-0

  if(hi[i]>1) hi[i]<-1

}

lines(seq(5:15),hi,lwd=2)

lines(seq(5:15),lo,lwd=2)

}
gompviolent()

```

Appendix 3 – R code for Gompertz-Makeham survivorship model using the Normal US mortality in 2008 prior

```
gompUSmort=function ()
{
  library(survival)
  makeham<-function(t,a3,b3)
  {

    a2<-0
    shift<-0
    h.t<-a2+a3*exp(b3*(t-shift))
    S.t<-exp(-a2*(t-shift)+a3/b3*(1-exp(b3*(t-shift))))
    return(S.t)
  }

  ages=USmort2010female$Age

  plot(survfit(Surv(ages)~1),lty=c(0,1),lwd=c(1,1),xlim=c(0,15),
       main='US Mortality 2008 Females',xlab='Age',ylab='Survivorship')

  #####

  jfs.optim<-function (x)
  {
```

```

makeham2<-function(t,x)
{
  a2<-0
  a3<-x[1]
  b3<-x[2]
  shift<-0
  h.t<-a2+a3*exp(b3*(t-shift))
  S.t<-exp(-a2*(t-shift)+a3/b3*(1-exp(b3*(t-shift))))
  return(S.t*h.t)
}

```

```

t<-ages
lnlk<-0
for(i in 1:817){
  lnlk<-lnlk+log(makeham2(t[i],x))
}

```

```

return(lnlk)
}

```

```
#####
```

```

sto<-optim(c(.01,.05),jfs.optim,control=list(fnscale=-1),hessian=T)
print(sto)

library(MASS)

myenv <- new.env()

assign("a3",sto$par[1], env = myenv)

assign("b3", sto$par[2], env = myenv)

assign("t", seq(0,14.9,.1), env = myenv)

dt<-numericDeriv(quote(makeham(seq(0,14.9,.1), a3, b3)), c("a3", "b3"), myenv)

est<-dt[1:150]

gr<-attr(dt,'gradient')

V<-ginv(-sto$hess)

lo<-0

hi<-0

for(i in 1:150){

  se<-sqrt(as.vector(gr[i,]%*%V*%gr[i,]))

  lo[i]<-est[i]-1.96*se

  hi[i]<-est[i]+1.96*se

  if(lo[i]<0) lo[i]<-0

  if(hi[i]>1) hi[i]<-1

}

lines(seq(0,14.9,.1),hi,lwd=2)

lines(seq(0,14.9,.1),lo,lwd=2)

}
gompUSmort()

```

Appendix 4 – R code for the Highest Posterior Denisty (HPD) analysis

```
lrange.viewer=function (ip=1,top=20,area=.50)

{
  # Prior age-at-death from Gompertz fit to death data - change according to prior used

  bot=0

  a2=0.0

  a3=0.35706326

  b3=-0.05985166

  # "Transition analysis" parameters - change according to tooth

  mu=c(1.653948, 1.936380, 2.148368, 2.244497, 2.374791, 2.491333, 2.642698)

  sdev=0.18947

  #####
  probit=function (t)
  {
    imax = 8
    if(ip==1) return(1-pnorm(log(t),mu[1],sdev))
    if(ip==imax) return(pnorm(log(t),mu[imax-1],sdev))
    return(pnorm(log(t),mu[ip-1],sdev)-pnorm(log(t),mu[ip],sdev))
  }
  #####
  # pia() #
  # This function finds the unnormalized posterior density of age #
  # conditional on the observed phase and the Gompertz-Makeham #
  # #
  #####

  pia<-function(t)
  {
    p<-probit(t)
    h.t<-a2+a3*exp(b3*(t-bot))
    S.t<-exp(-a2*(t-bot)+a3/b3*(1-exp(b3*(t-bot))))
    return(S.t*h.t*p)
  }
  #####
  # pia2() #
  # This function finds the normalized posterior density of age #
```



```

# conditional on the observed phase and the Gompertz-Makeham #
#
#####
pia2<-function(t)
{
  p<-probit(t)
  shift<-bot
  h.t<-a2+a3*exp(b3*(t-bot))
  S.t<-exp(-a2*(t-bot)+a3/b3*(1-exp(b3*(t-bot))))
  return(S.t*h.t*p/denom)
}
#####
# pia5() #
# Searches left and right to find cut points, such that the #
# integral between cut points less the desired area is near #
# zero #
# #
#####
pia5<-function(divid)
{
  pia4<-function(tt)
  {
    cd<-pia2(tt)-prob*divid
    return(cd)
  }
  low<-uniroot(pia4,c(bot,hpd))$root
  hi<-uniroot(pia4,c(hpd,top))$root
  return(integrate(pia2,low,hi)$value-area)
}
#####
# pia6() #
# Returns the cut points after they have been found by pia5() #
# #
#####
pia6<-function(divid)
{
  pia4<-function(tt)
  {
    cd<-pia2(tt)-prob*divid
    return(cd)
  }
  low<-uniroot(pia4,c(bot,hpd))$root
  hi<-uniroot(pia4,c(hpd,top))$root
  return(c(low,hi))
}
#####

```

```

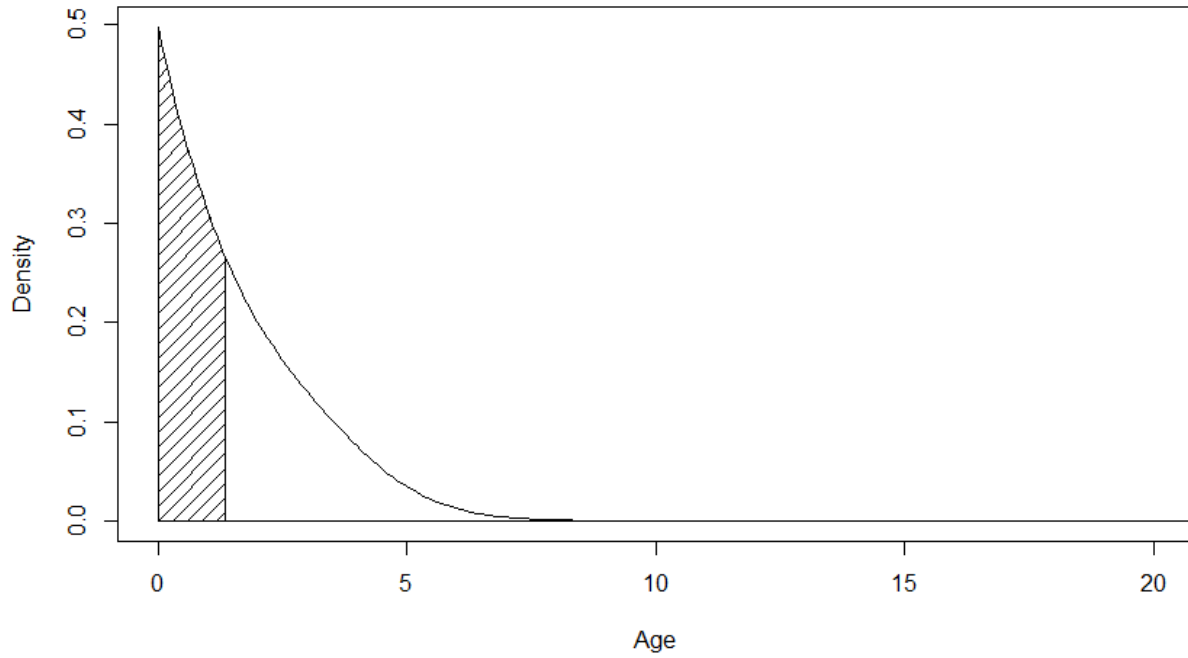
pia7<-function(t)
{ return(integrate(pia2,bot,t)$value-area) }
#####END OF FUNCTIONS#####

plot.at=seq(bot,top,.1)
denom<-integrate(pia,bot,top)$value
hpd<-optimize(pia,c(bot,top),maximum=T)$maximum
prob<-pia2(hpd)
prob2<-pia2(bot)
tunebot<-1.00001*prob2/prob
tunebot=max(.1,tunebot)
tune<-c(tunebot,.99999)
plot(c(bot,plot.at),c(0,pia2(plot.at)),
     type='l',main='HPD male molar 2 stage 12 (Rc), US Violent Deaths 2010 prior',
xlab='Age',ylab='Density',xlim=c(bot,top))
cat('\n\n Maximum density occurs at ',round(hpd,digits=2),'\n\n')
lines(c(0,100),c(0,0))
hi<-uniroot(pia7,c(bot,top))$root
if(pia2(hi)<prob2)
{
  x<-c(plot.at[plot.at<hi],hi)
  polygon(c(bot,x,x[NROW(x)]),c(0,pia2(x),0),density=10)
  cat(area*100,'% HPD from bottom [' ,bot,'],' ,round(hi,digits=2),'\n\n')
  return(c(round(bot,digits=2),round(hpd,digits=2),round(hi,digits=2)))
}
if(pia2(hi)>prob2)
{
  divid<-uniroot(pia5,tune)$root
  x<-pia6(divid)
  x<-c(x[1],plot.at[plot.at>x[1] & plot.at<x[2]],x[2])
  polygon(c(x[1],x,x[NROW(x)]),c(0,pia2(x),0),density=10)
  cat(area*100,'% HPD \n')
  return(c(round(x[1],digits=2),round(hpd,digits=2),round(x[NROW(x)],digits=2)))
}
}

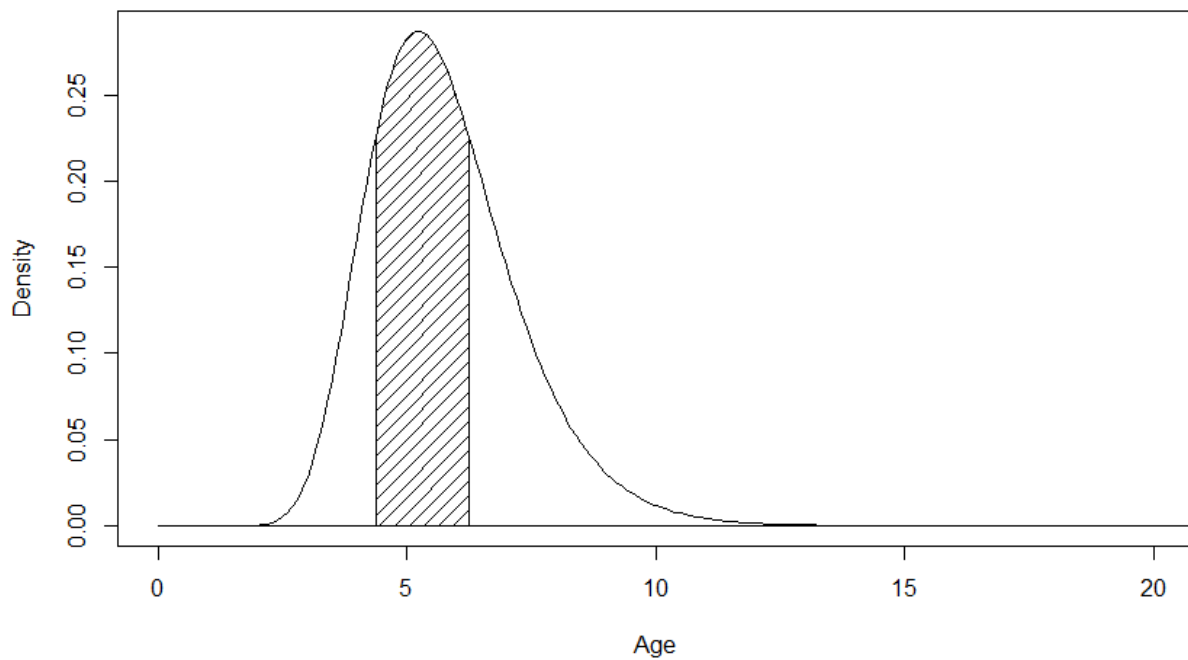
```

Appendix 5 – Highest posterior density graphs for all teeth at various recorded stages for males and females

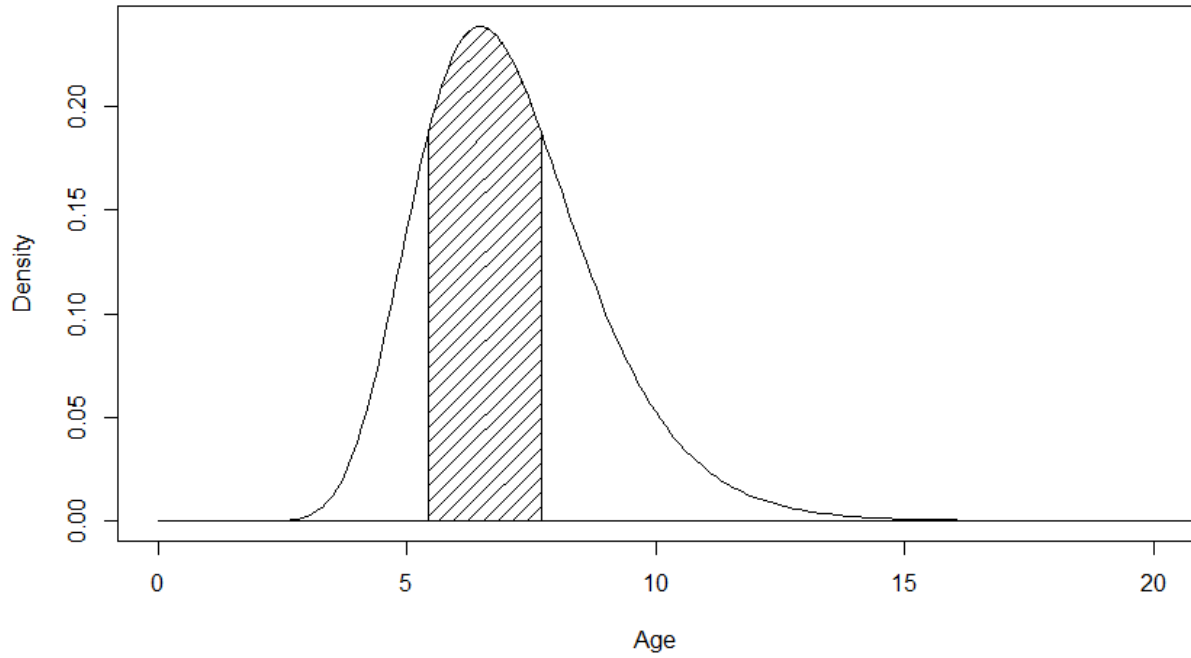
HPD female upper incisor 1 stage 10 (R1/2), US Violent Deaths 2010 prior



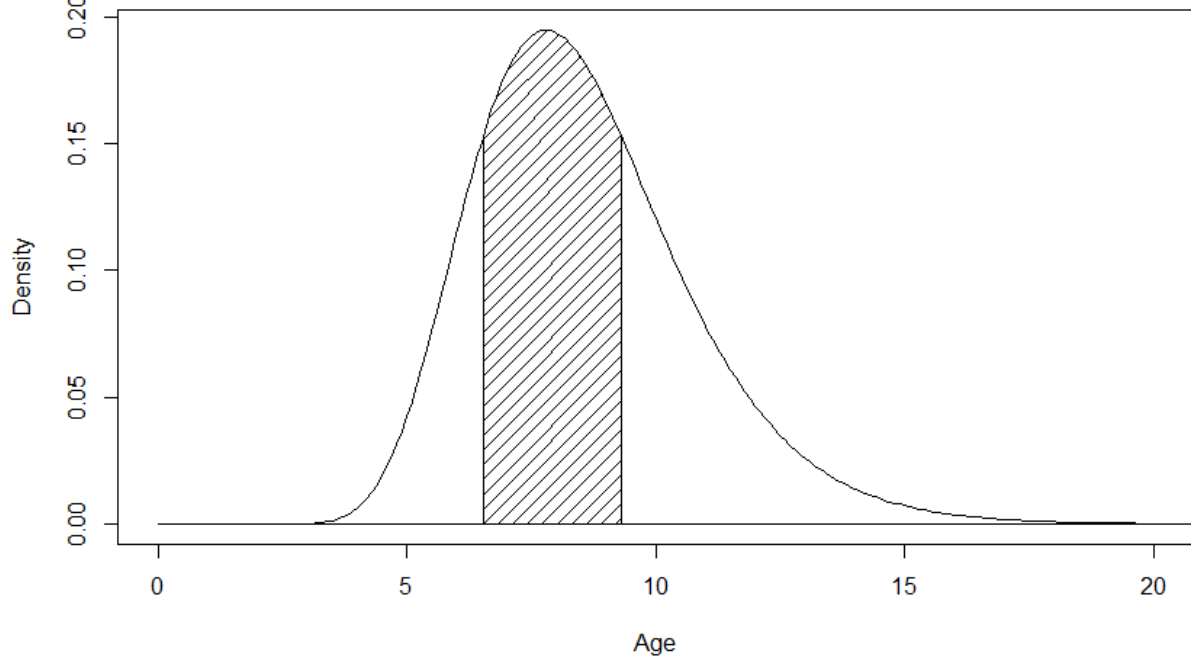
HPD female upper incisor 1 stage 11 (R3/4), US Violent Deaths 2010 prior



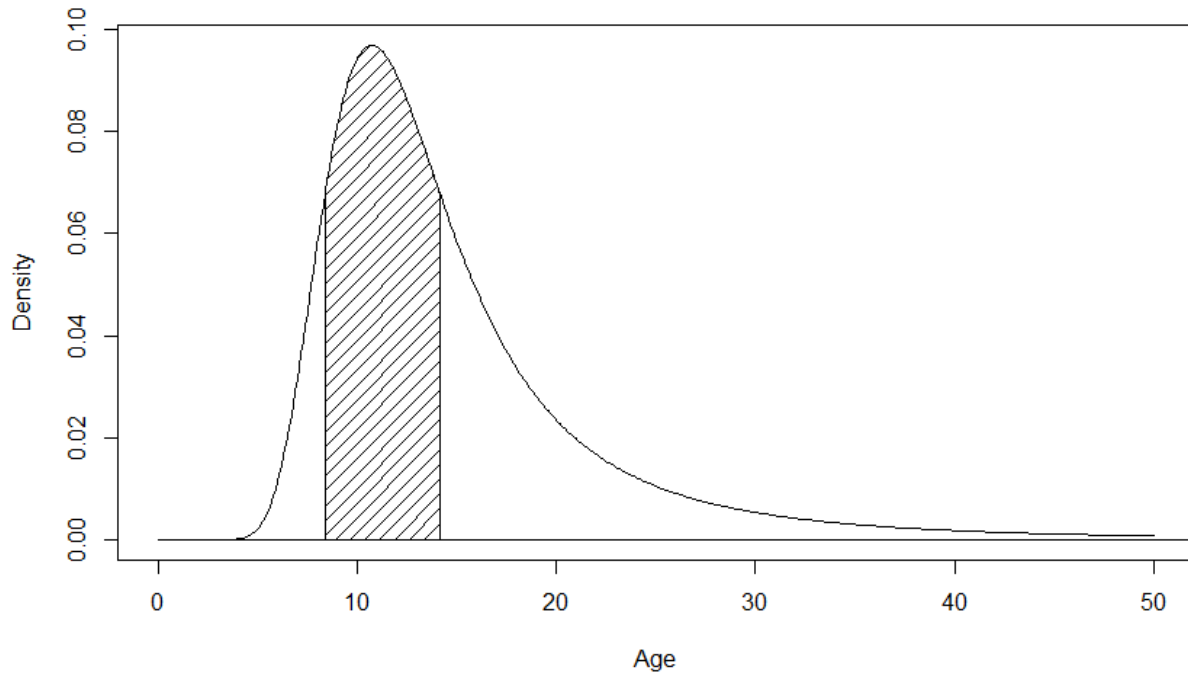
HPD female upper incisor 1 stage 12 (Rc), US Violent Deaths 2010 prior



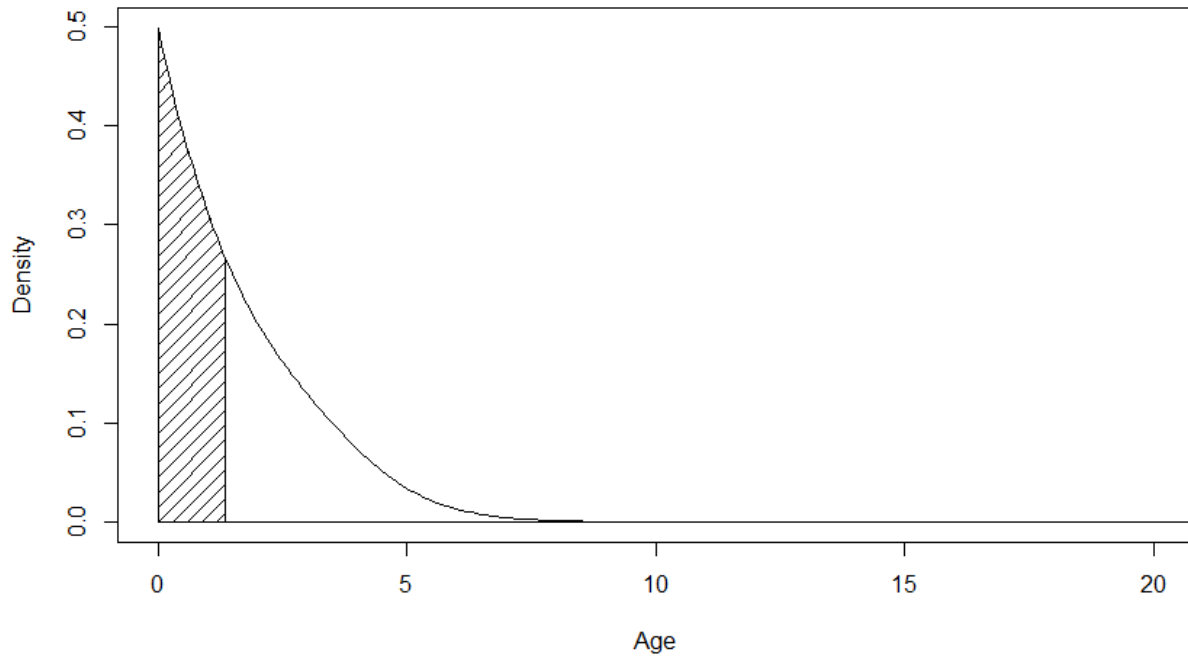
HPD female upper incisor 1 stage 13 (A1/2), US Violent Deaths 2010 prior



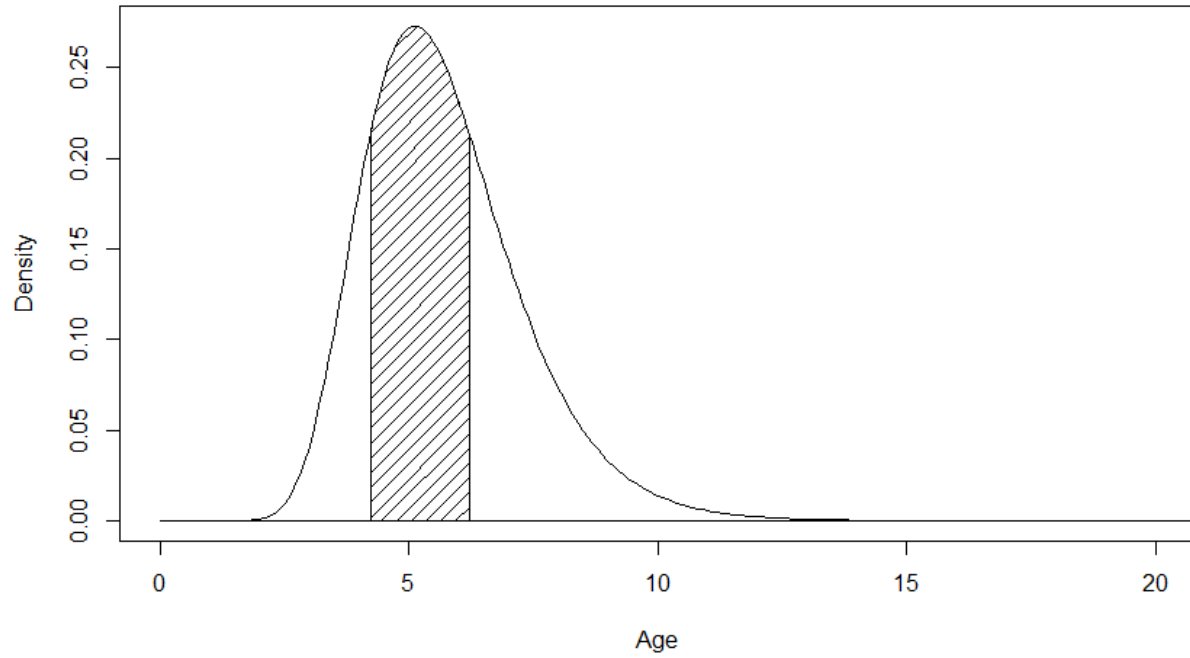
HPD female upper incisor 1 stage 14 (Ac), US Violent Deaths 2010 prior



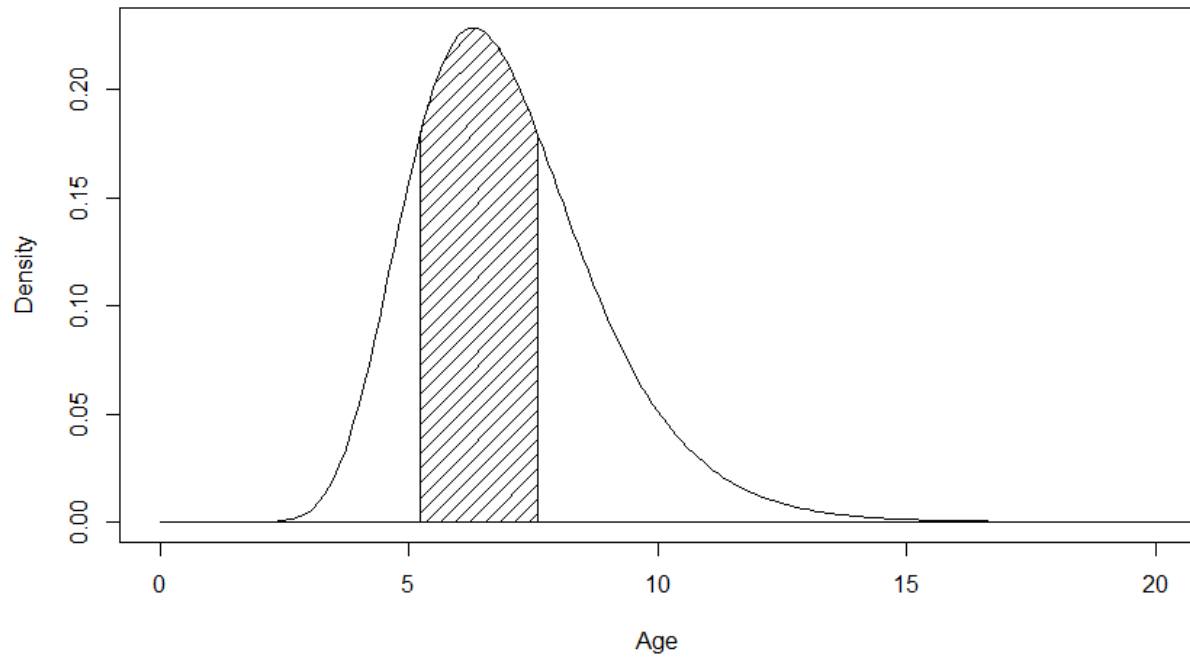
HPD female upper incisor 2 stage 9 (R1/4), US Violent Deaths 2010 prior



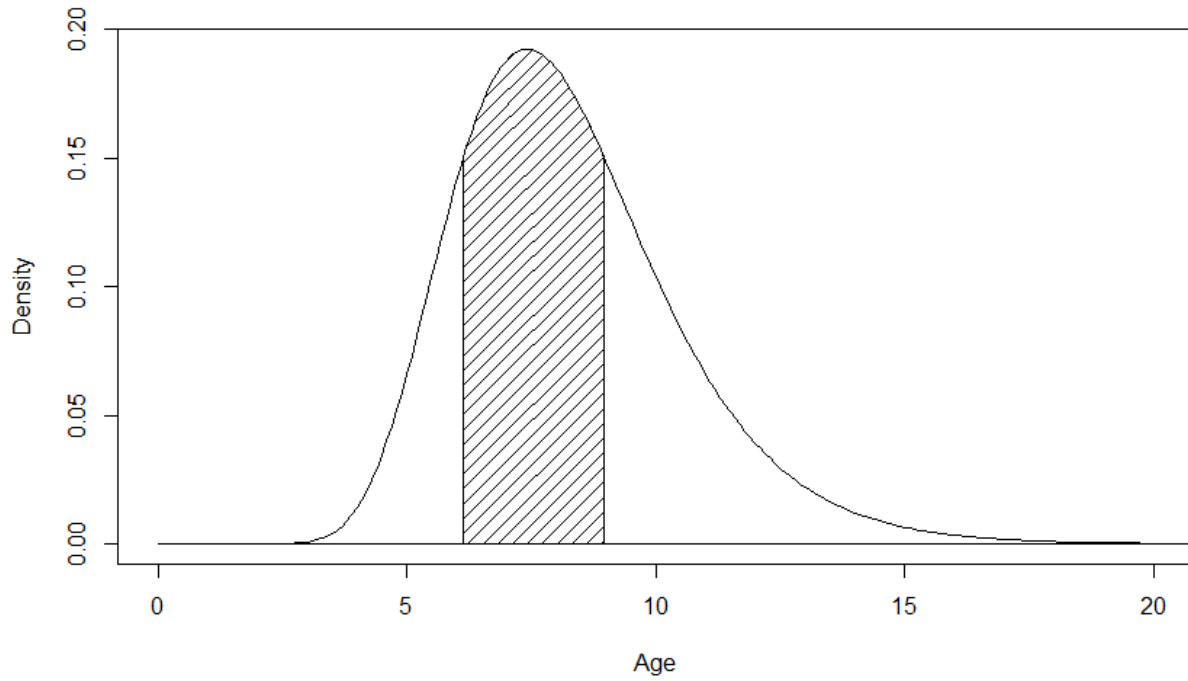
HPD female upper incisor 2 stage 10 (R1/2), US Violent Deaths 2010 prior



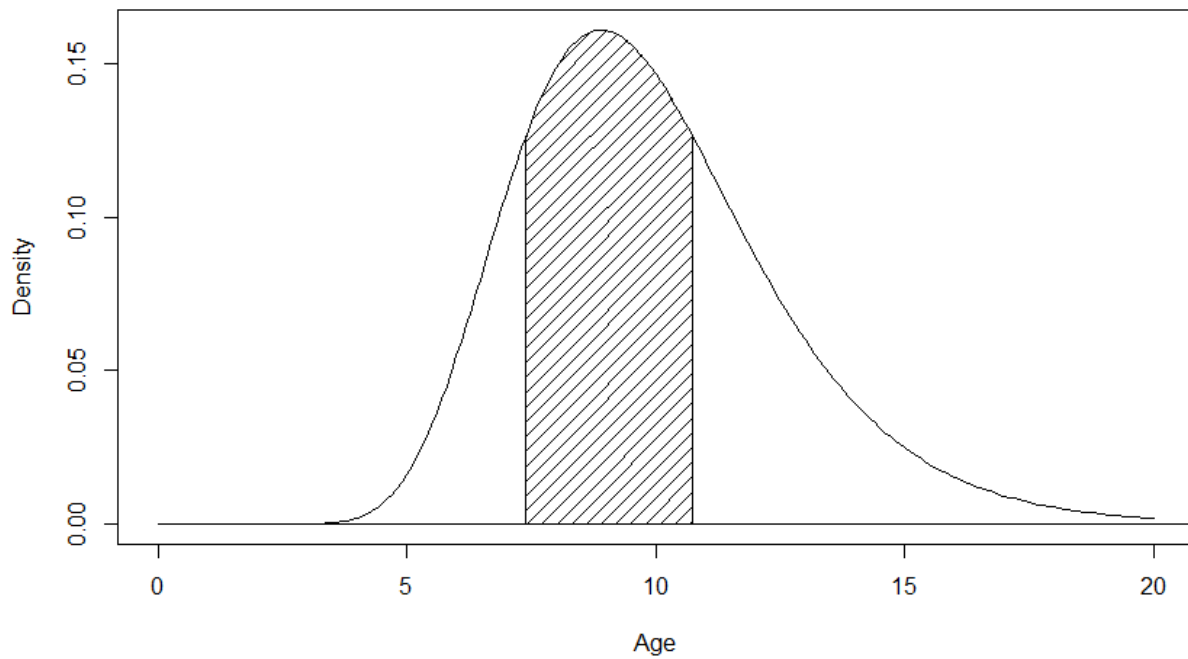
HPD female upper incisor 2 stage 11 (R3/4), US Violent Deaths 2010 prior



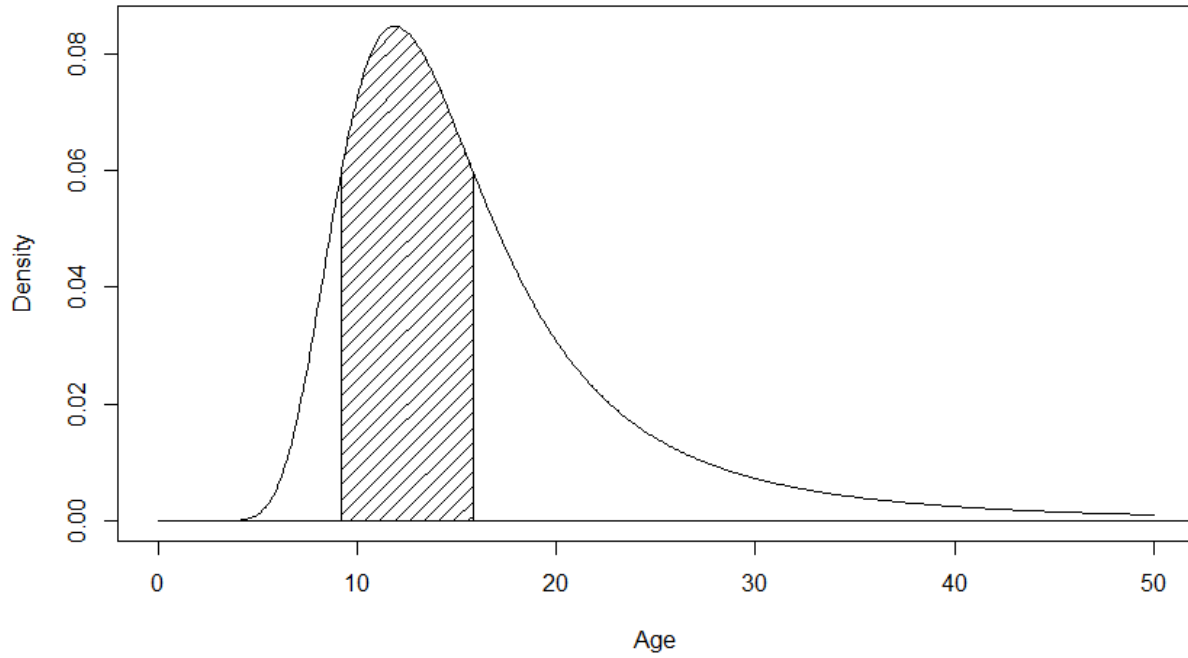
HPD female upper incisor 2 stage 12 (Rc), US Violent Deaths 2010 prior



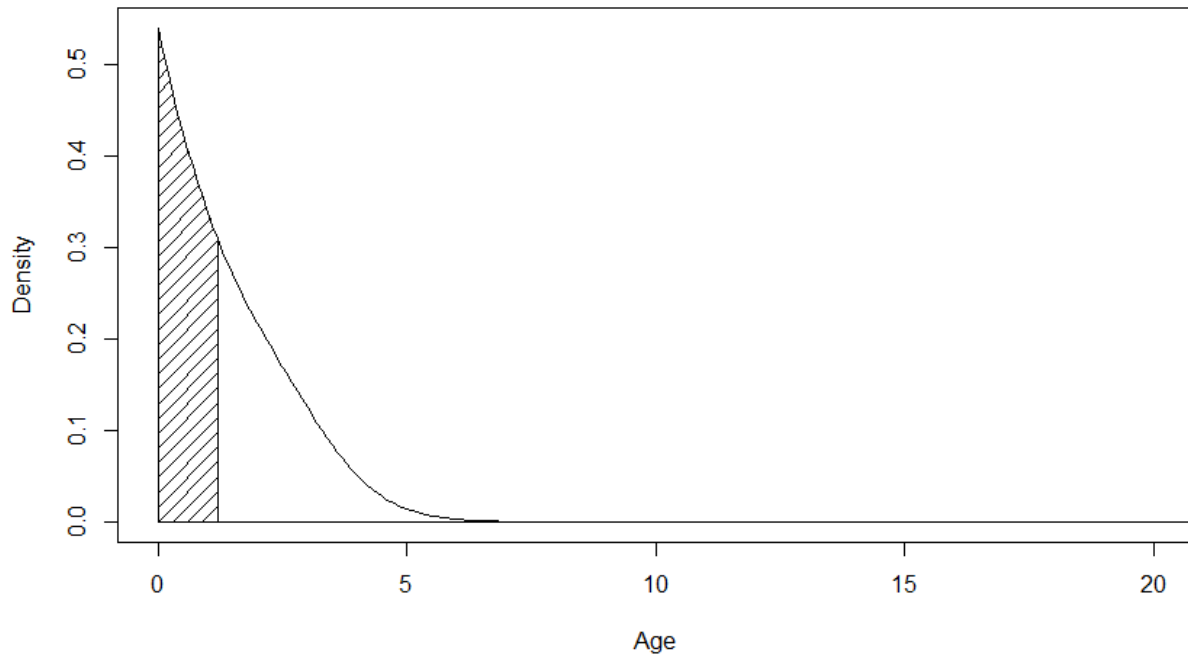
HPD female upper incisor 2 stage 13 (A1/2), US Violent Deaths 2010 prior



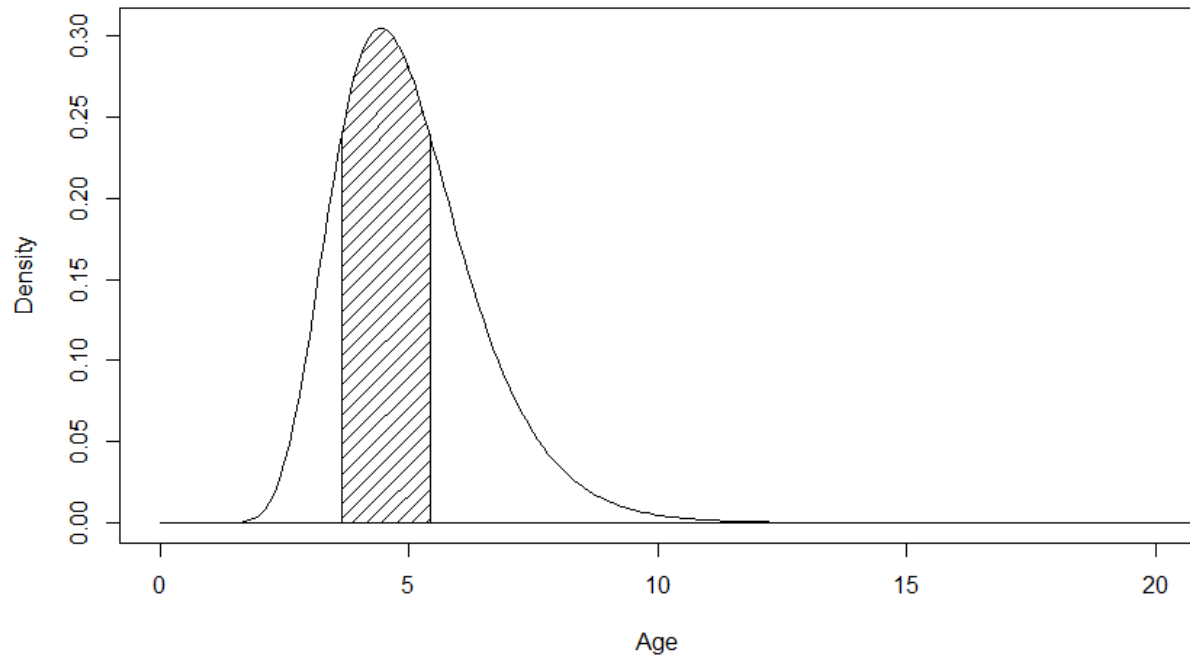
HPD female upper incisor 2 stage 14 (Ac), US Violent Deaths 2010 prior



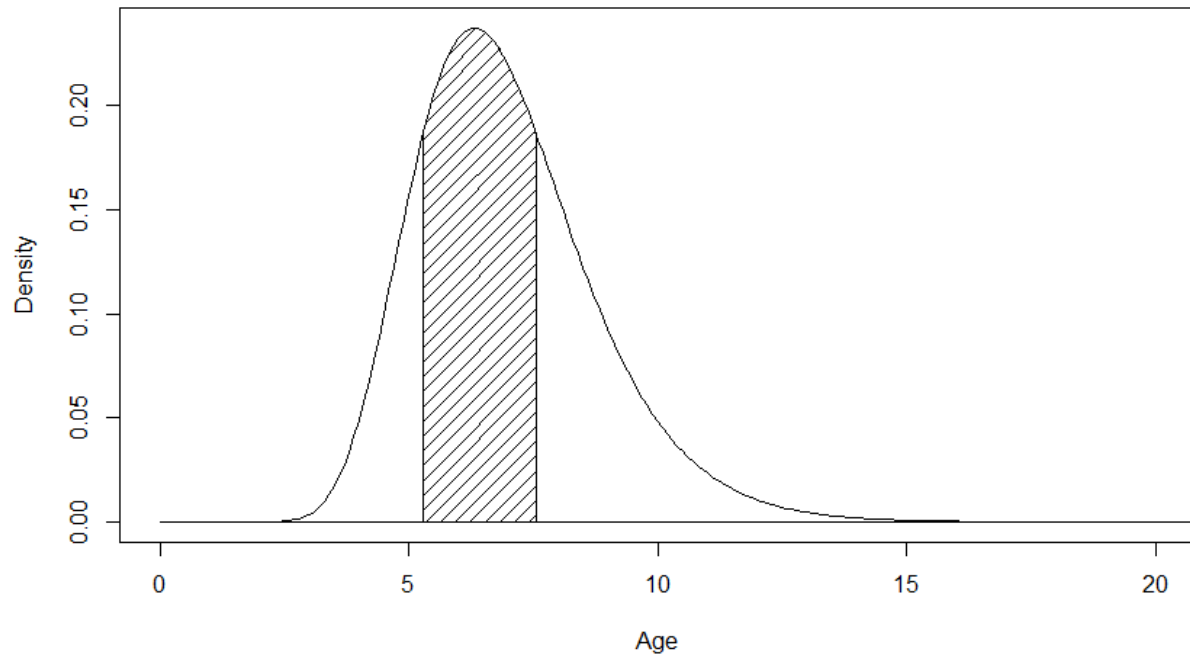
HPD female upper canine stage 7 (Ri), US Violent Deaths 2010 prior



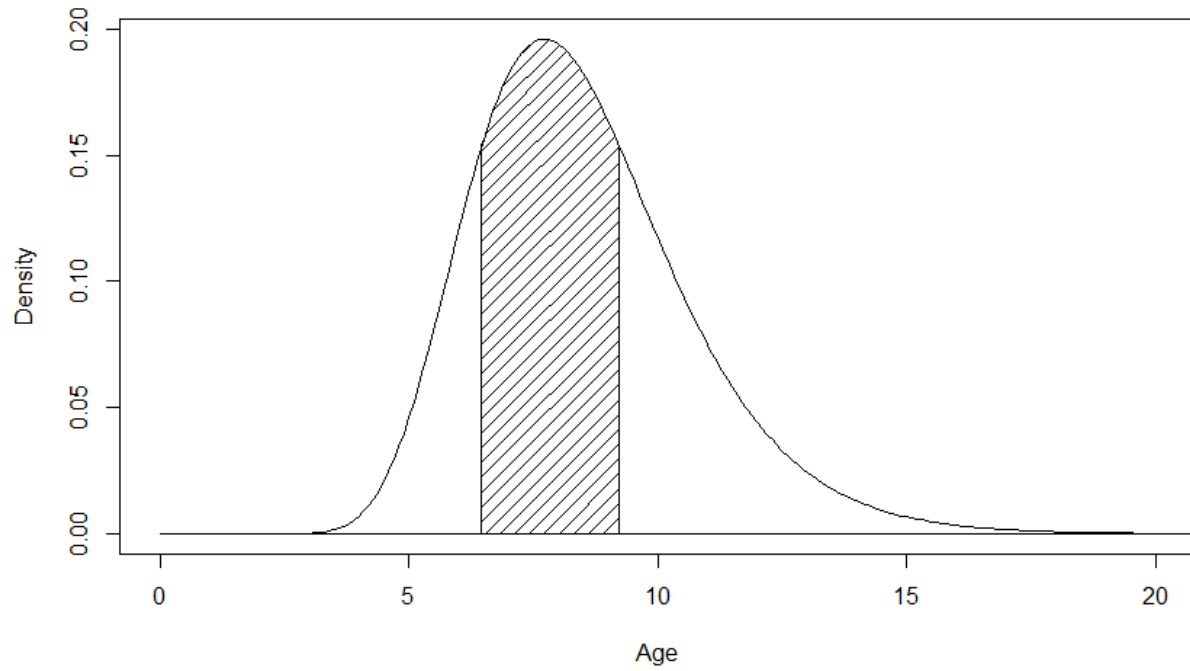
HPD female upper canine stage 9 (R1/4), US Violent Deaths 2010 prior



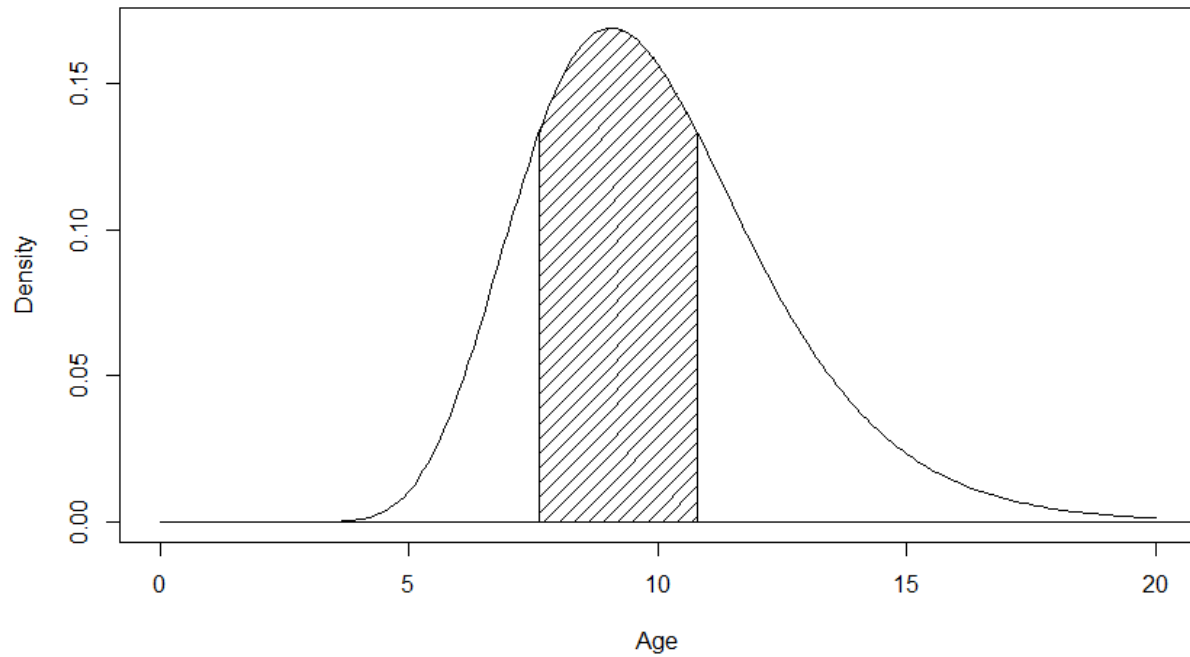
HPD female upper canine stage 10 (R1/2), US Violent Deaths 2010 prior



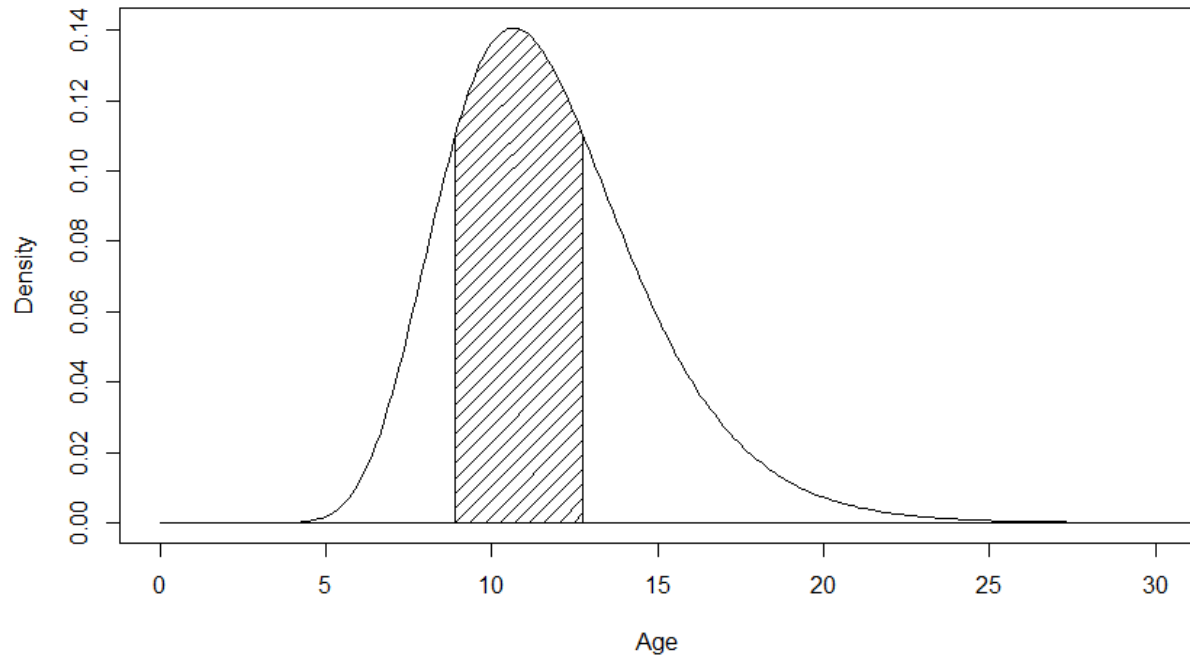
HPD female upper canine stage 11 (R3/4), US Violent Deaths 2010 prior



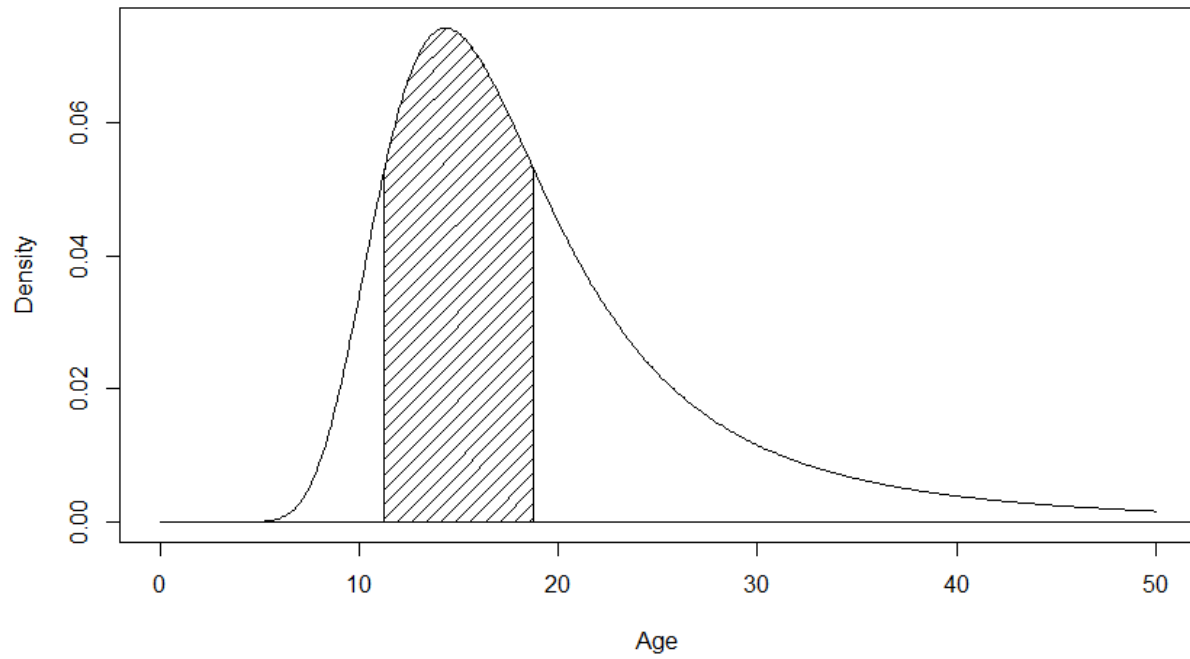
HPD female upper canine stage 12 (Rc), US Violent Deaths 2010 prior



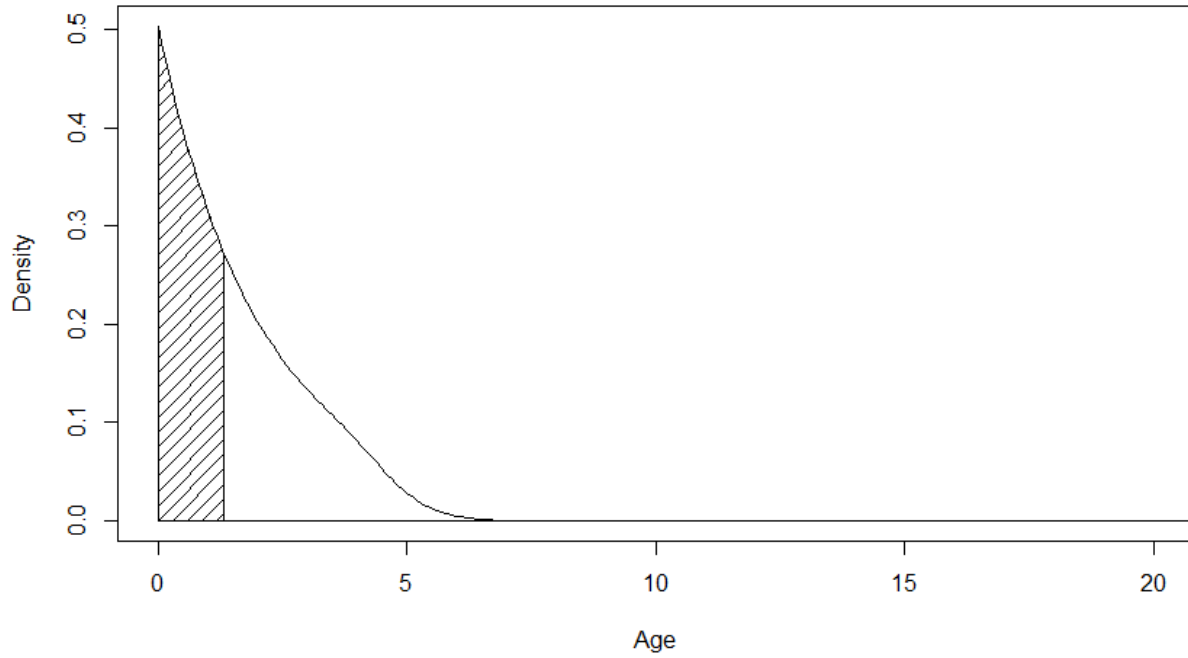
HPD female upper canine stage 13 (A1/2), US Violent Deaths 2010 prior



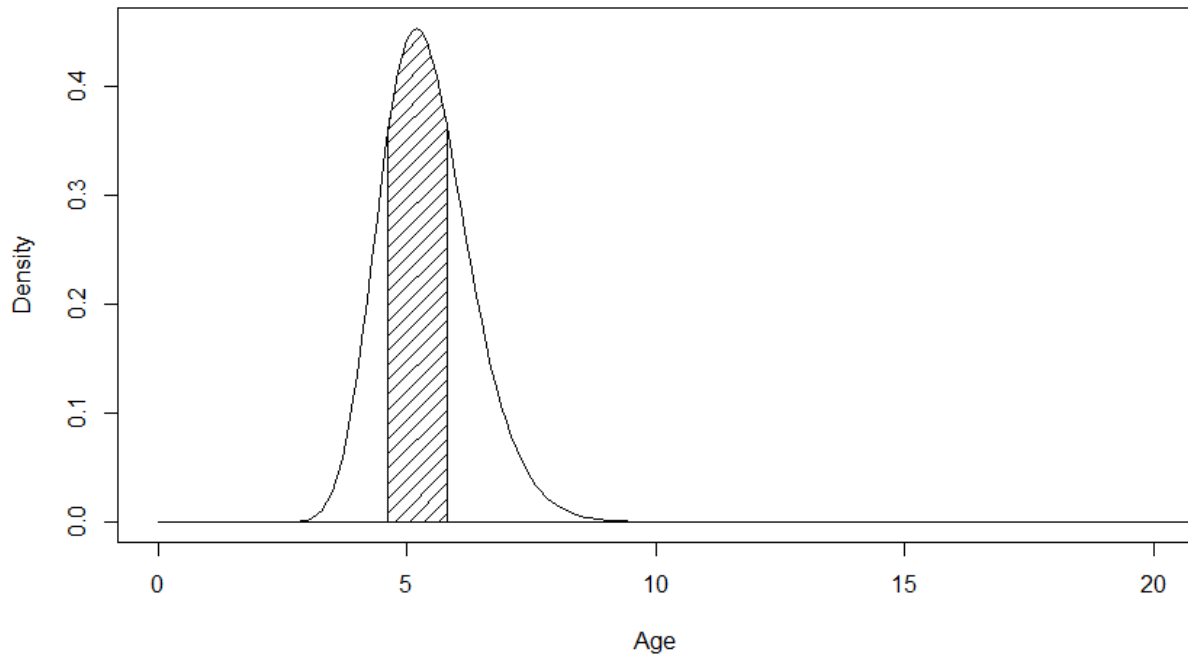
HPD female upper canine stage 14 (Ac), US Violent Deaths 2010 prior



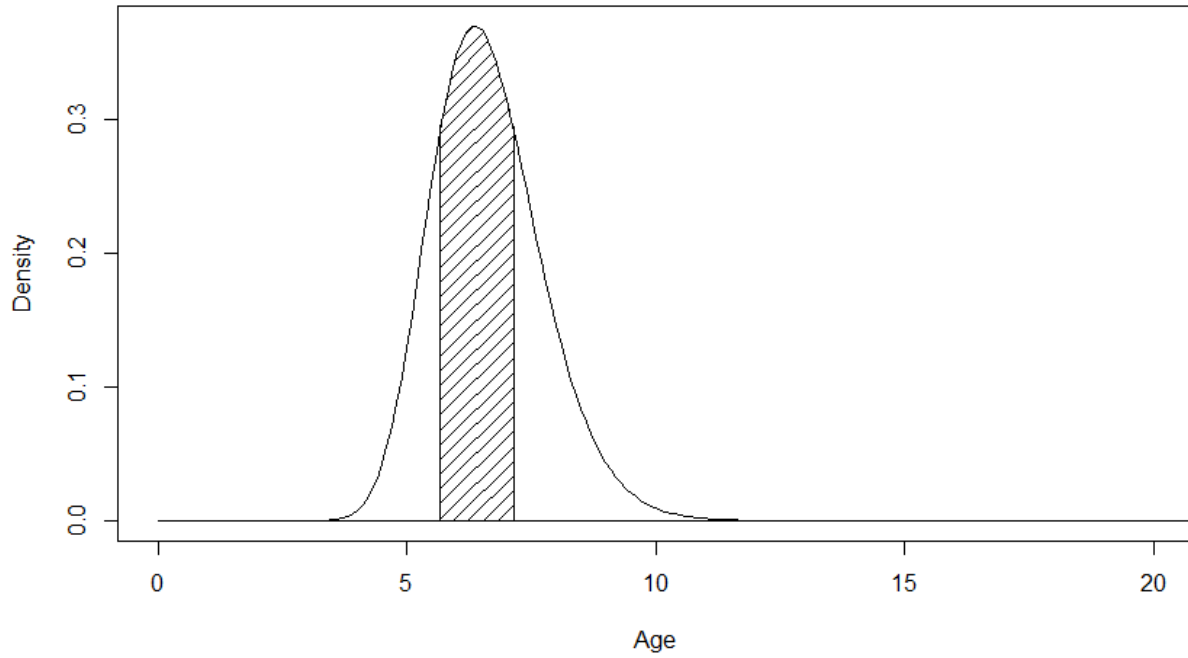
HPD female upper premolar 1 stage 5 (Cr3/4), US Violent Deaths 2010 prior



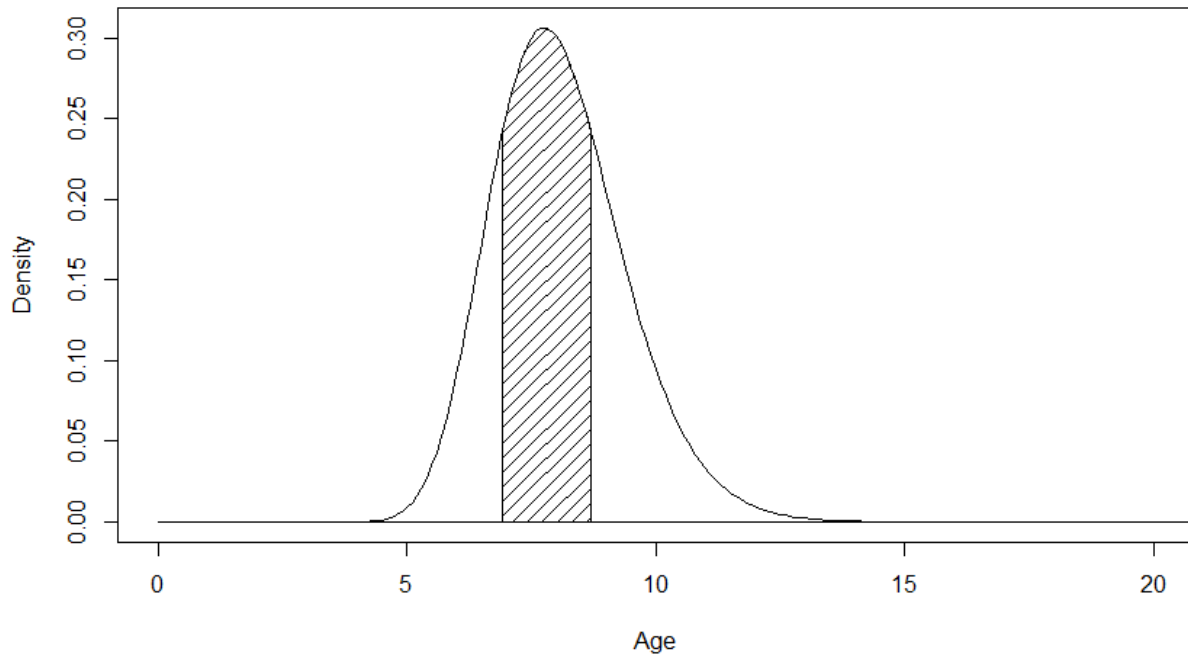
HPD female upper premolar 1 stage 6 (Crc), US Violent Deaths 2010 prior



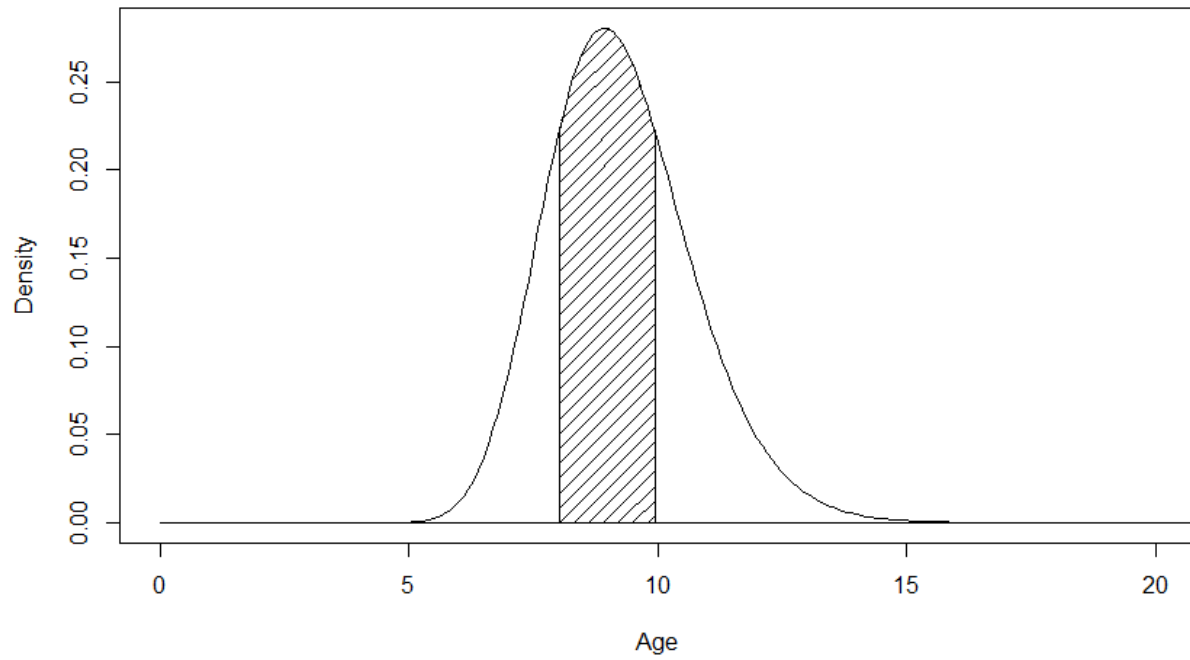
HPD female upper premolar 1 stage 7 (Ri), US Violent Deaths 2010 prior



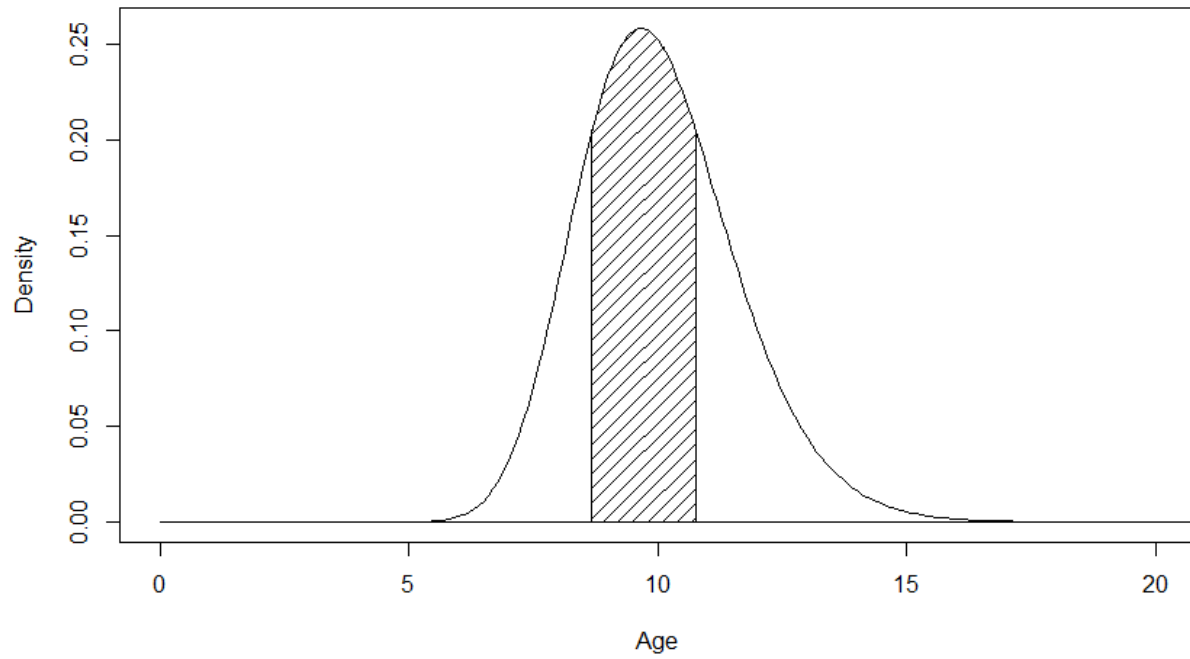
HPD female upper premolar 1 stage 9 (R1/4), US Violent Deaths 2010 prior



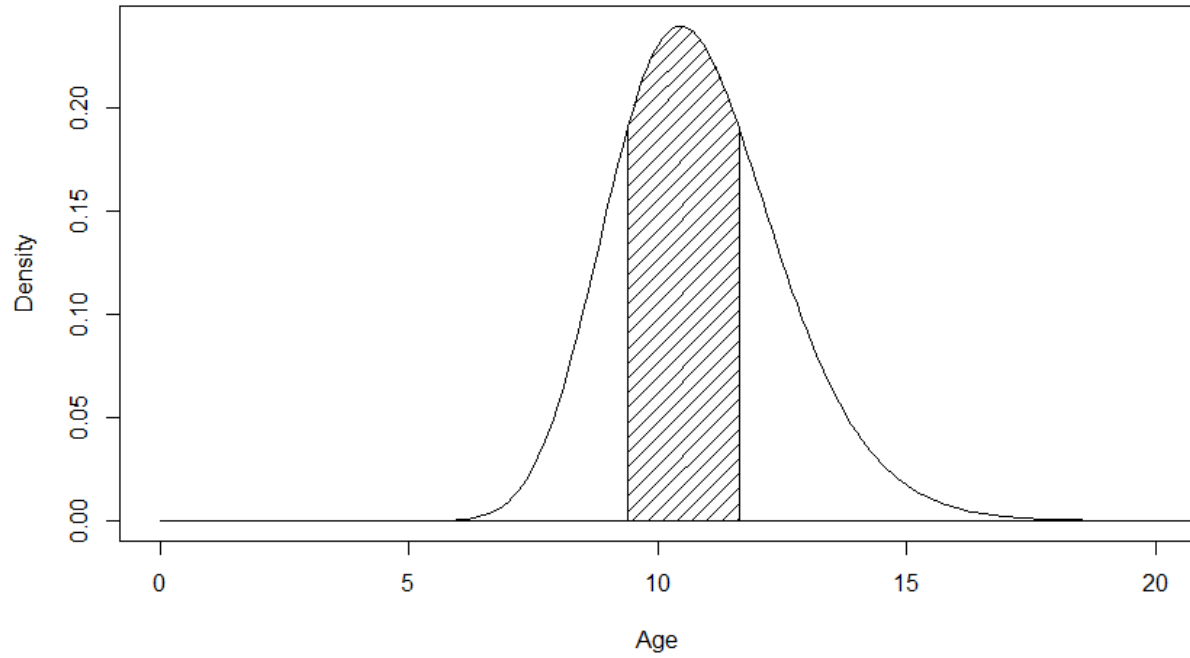
HPD female upper premolar 1 stage 10 (R1/2), US Violent Deaths 2010 prior



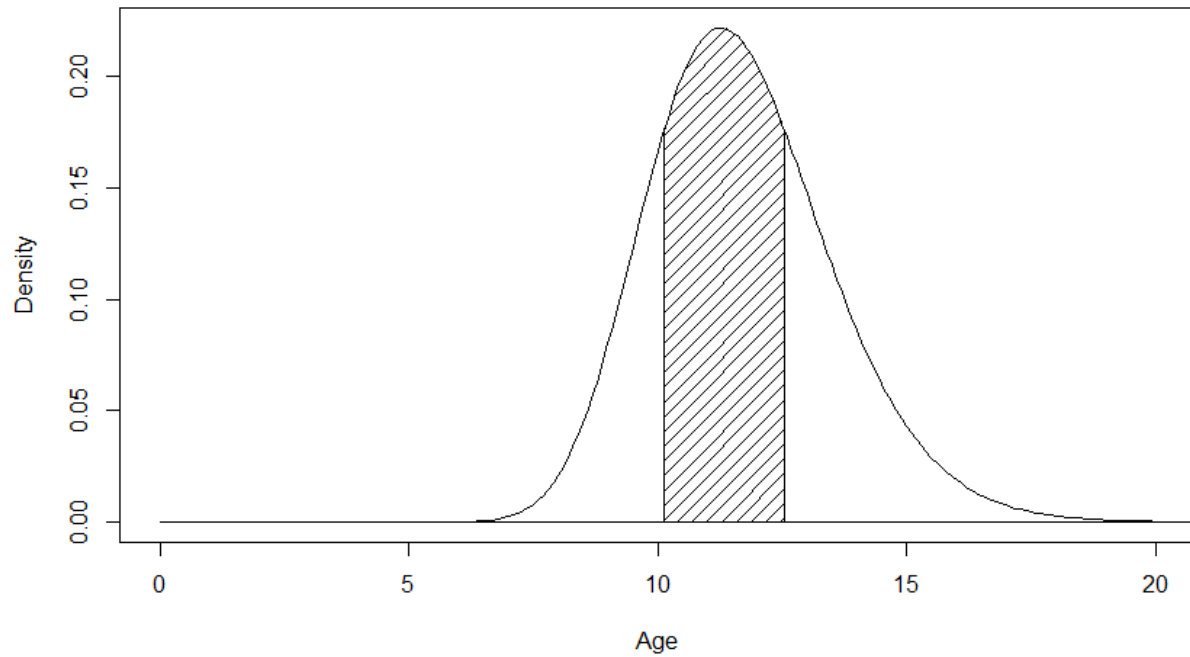
HPD female upper premolar 1 stage 11 (R3/4), US Violent Deaths 2010 prior



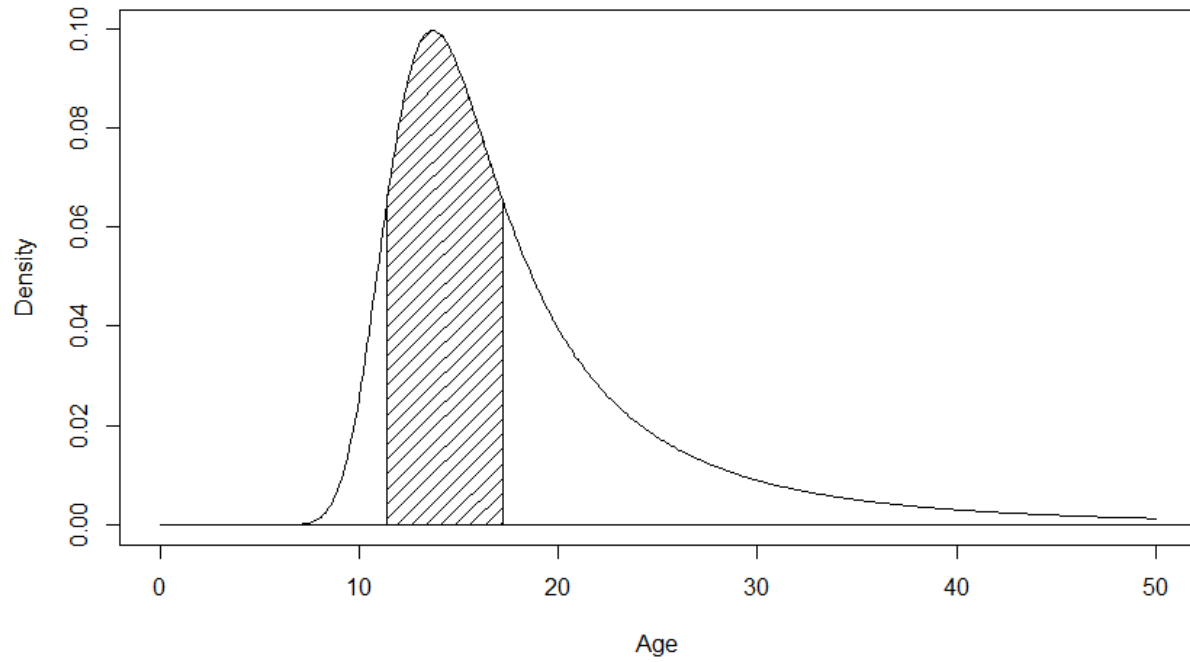
HPD female upper premolar 1 stage 12 (Rc), US Violent Deaths 2010 prior



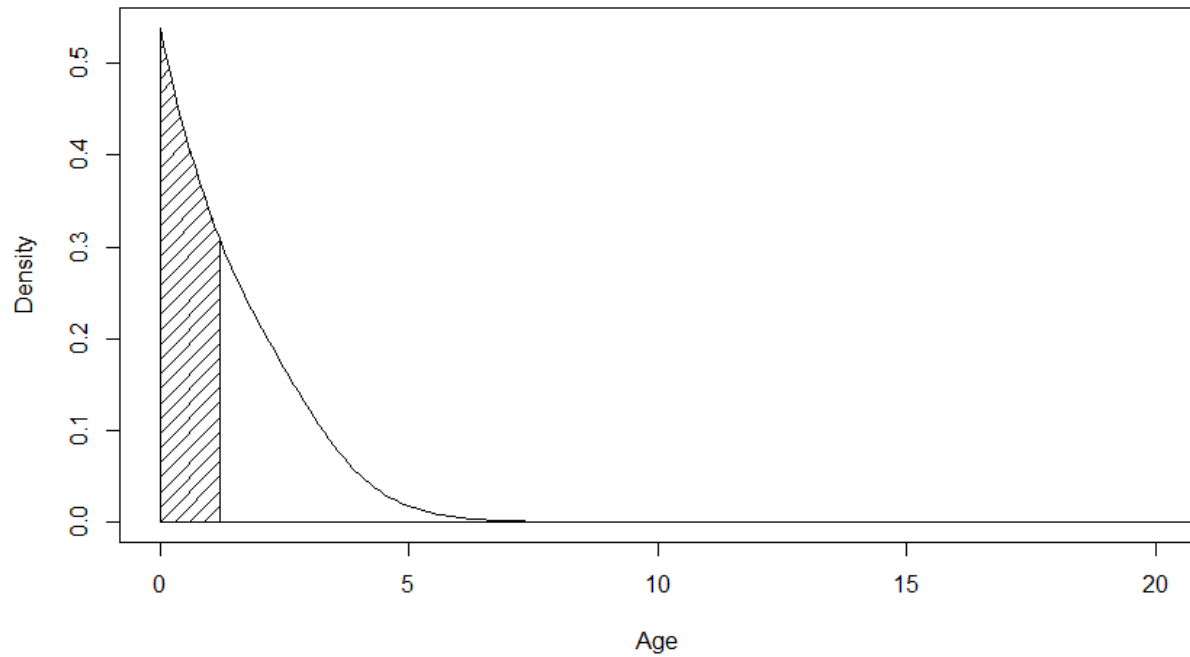
HPD female upper premolar 1 stage 13 (A1/2), US Violent Deaths 2010 prior



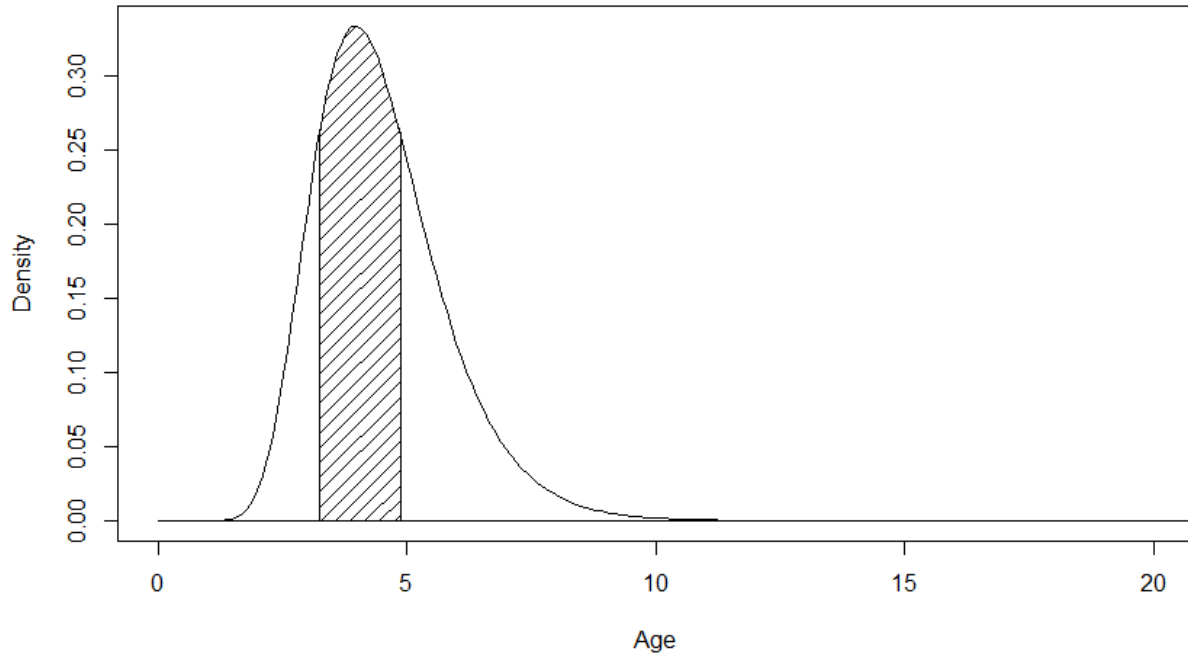
HPD female upper premolar 1 stage 14 (Ac), US Violent Deaths 2010 prior



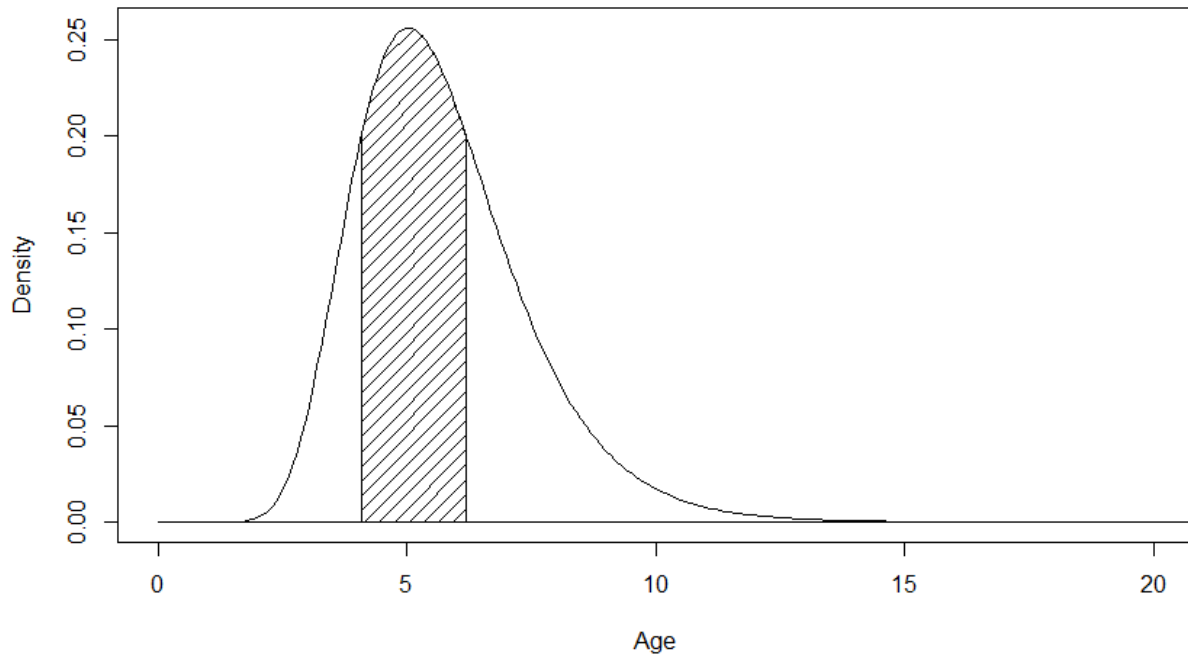
HPD female upper premolar 2 stage 5 (Cr3/4), US Violent Deaths 2010 prior



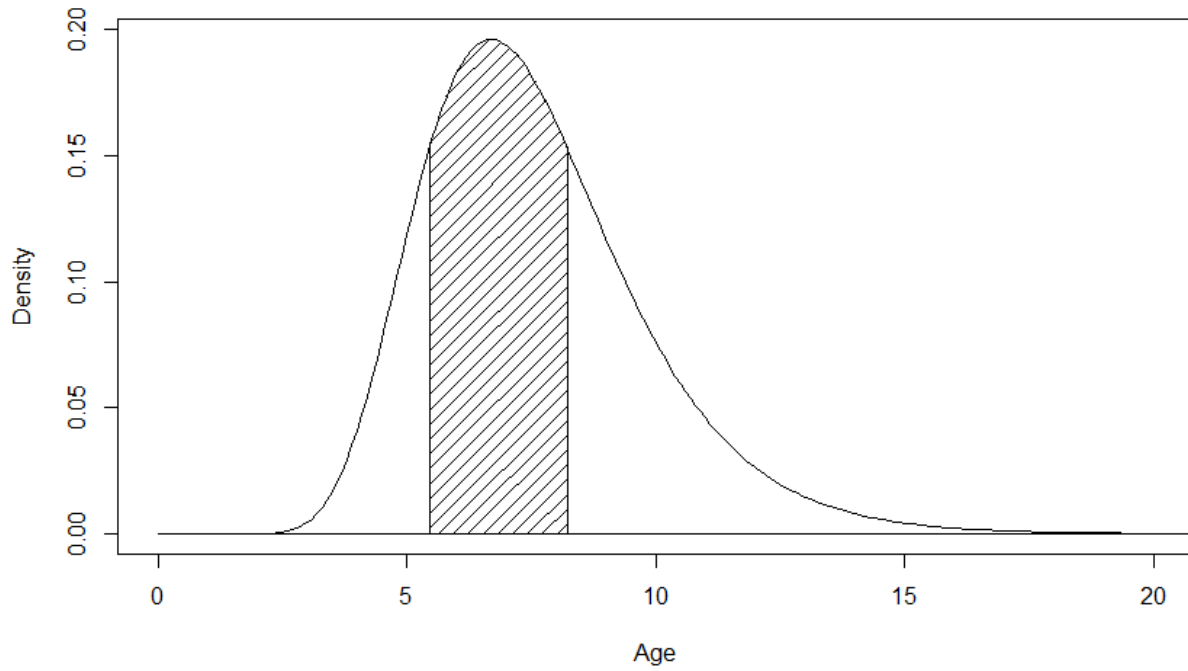
HPD female upper premolar 2 stage 6 (Crc), US Violent Deaths 2010 prior



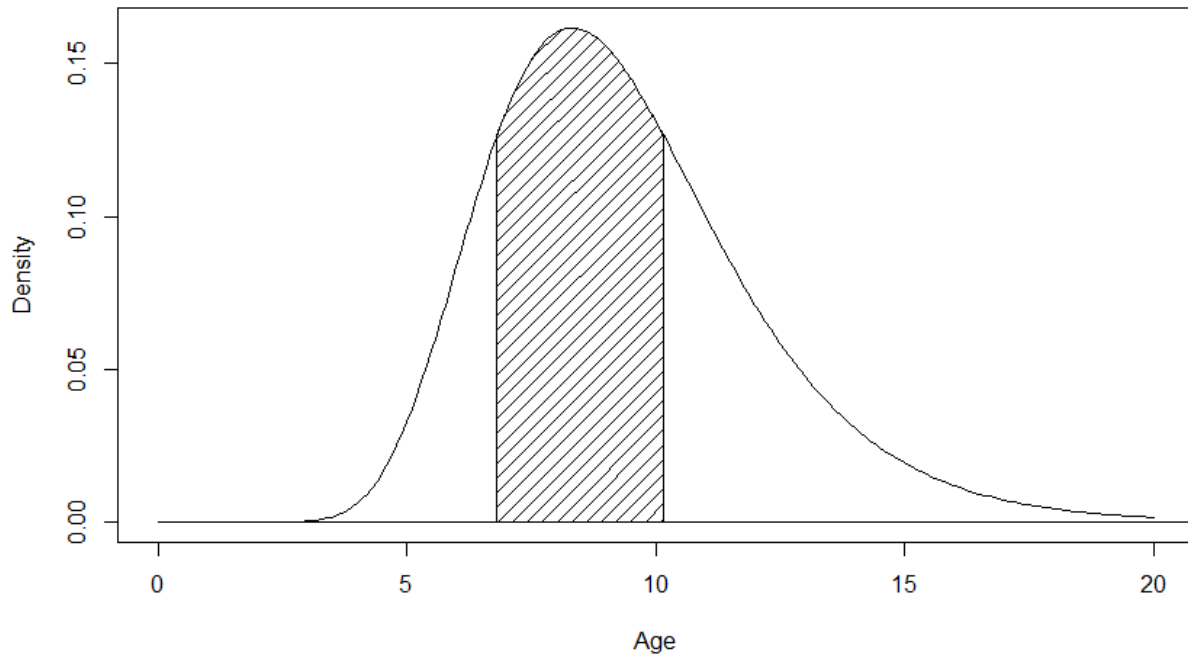
HPD female upper premolar 2 stage 7 (Ri), US Violent Deaths 2010 prior



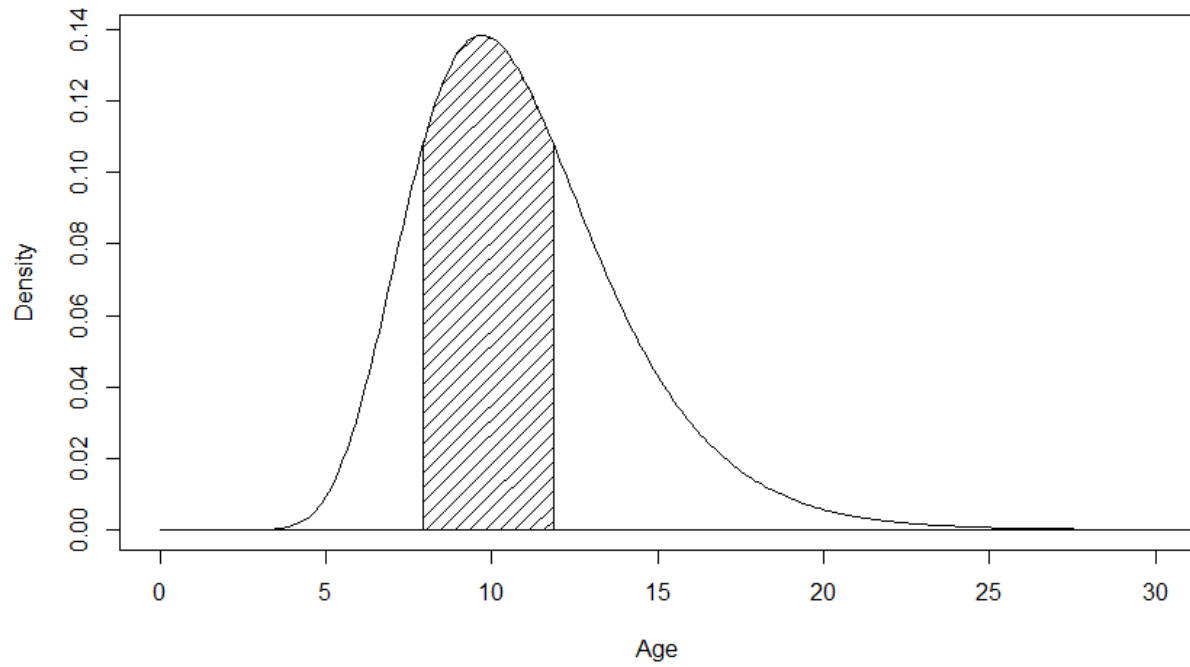
HPD female upper premolar 2 stage 9 (R1/4), US Violent Deaths 2010 prior



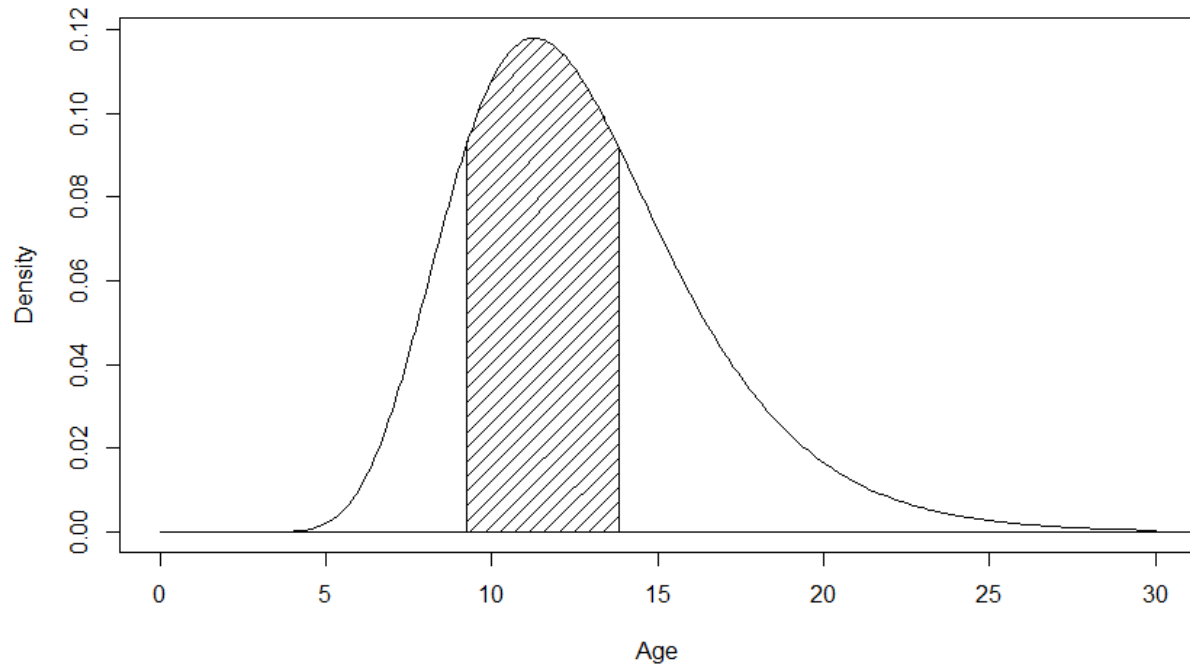
HPD female upper premolar 2 stage 10 (R1/2), US Violent Deaths 2010 prior



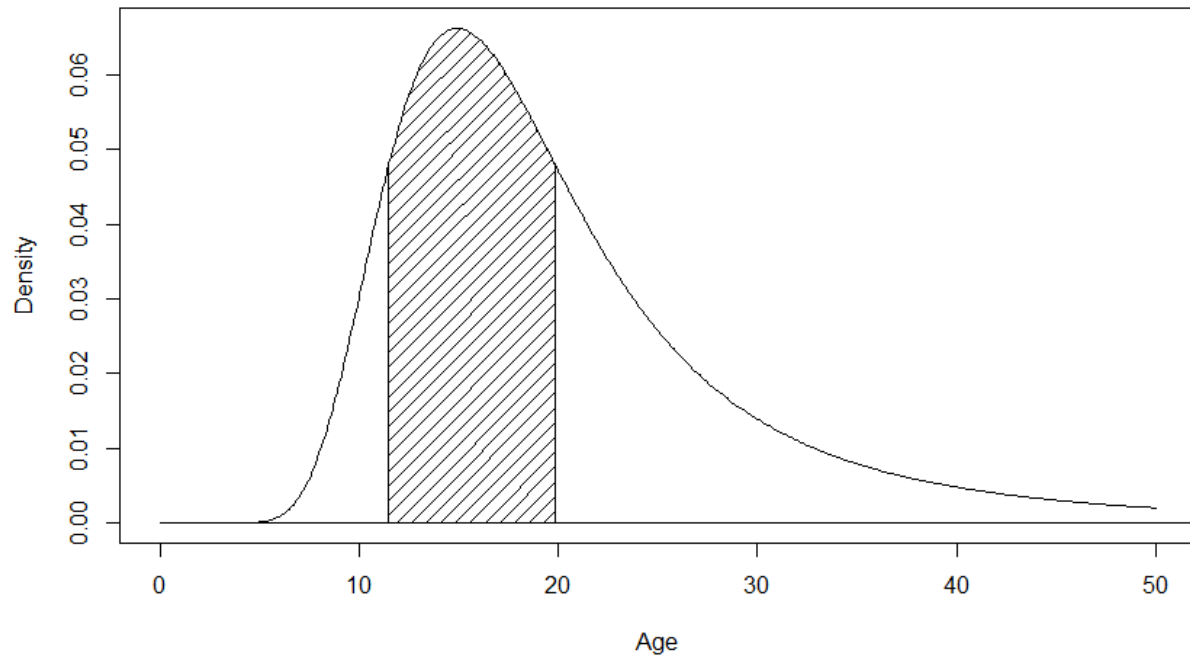
HPD female upper premolar 2 stage 11 (R3/4), US Violent Deaths 2010 prior



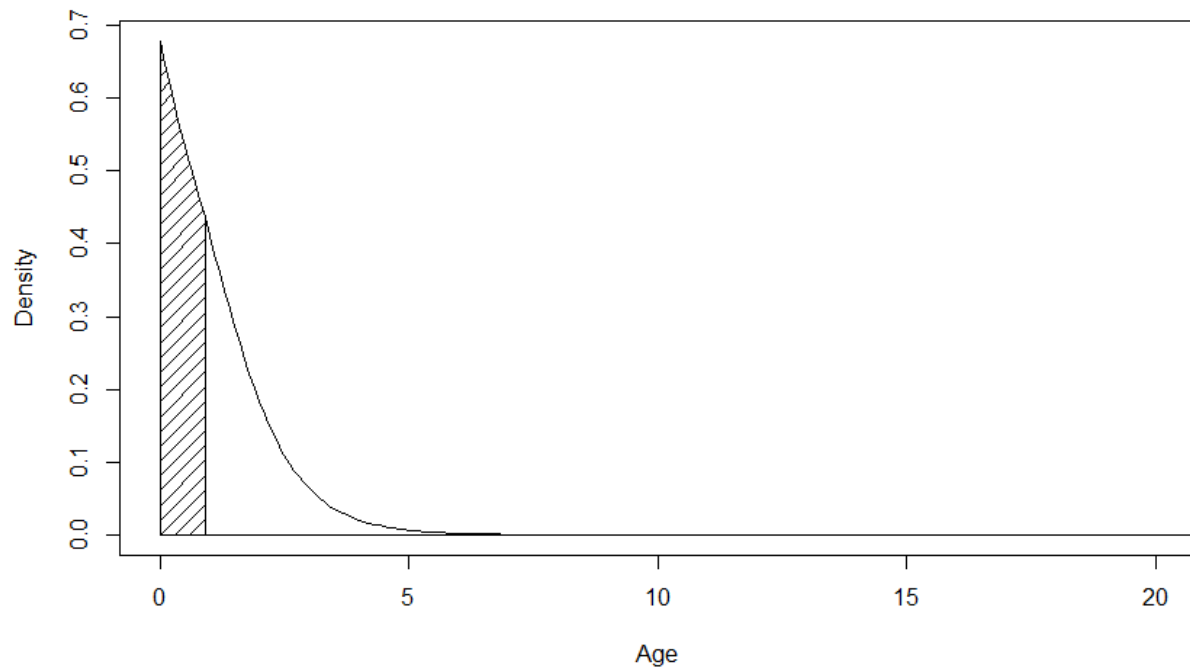
HPD female upper premolar 2 stage 12 (Rc), US Violent Deaths 2010 prior



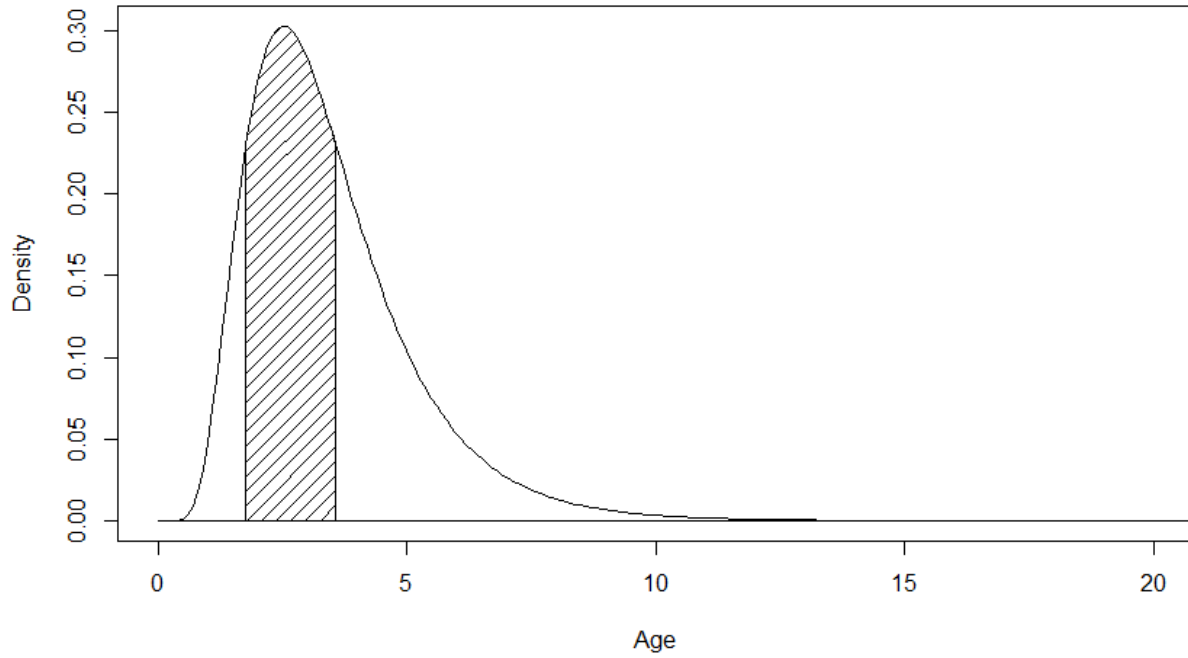
HPD female upper premolar 2 stage 13 (A1/2), US Violent Deaths 2010 prior



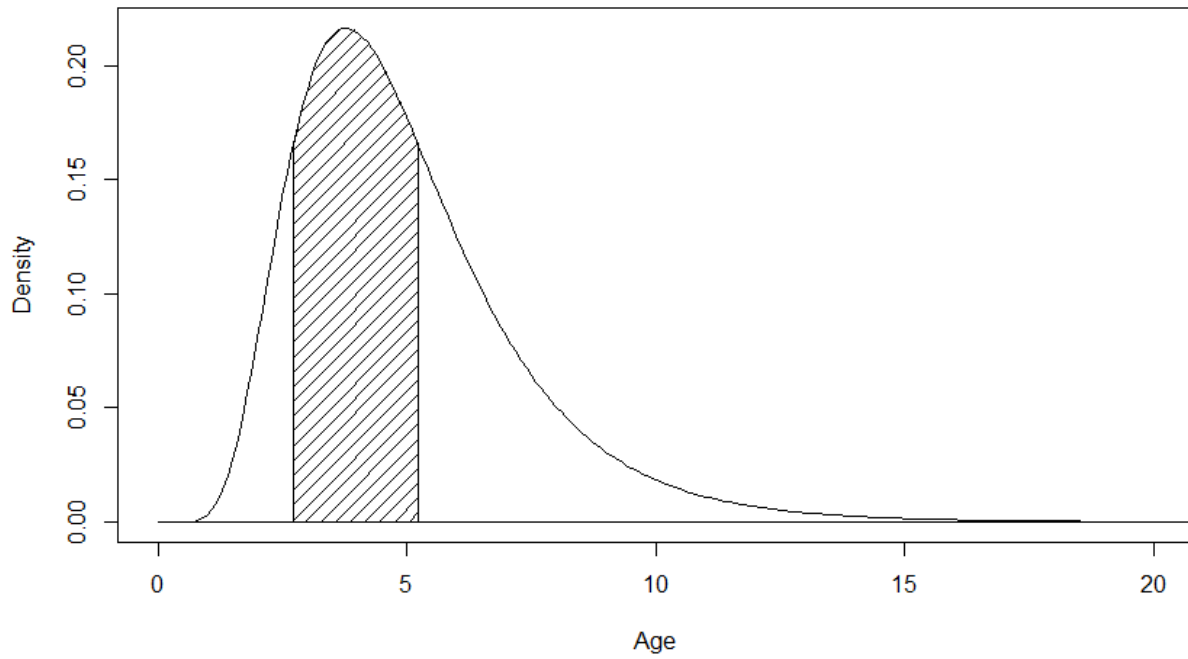
HPD female upper molar 1 stage 9 (R1/4), US Violent Deaths 2010 prior



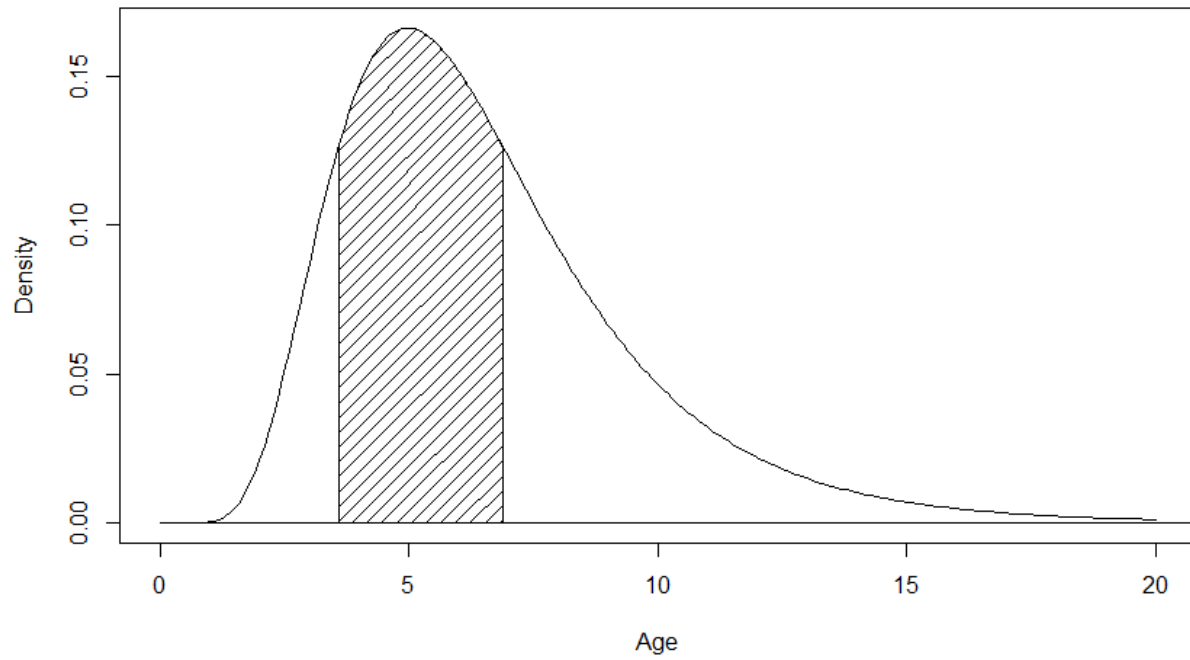
HPD female upper molar 1 stage 10 (R1/2), US Violent Deaths 2010 prior



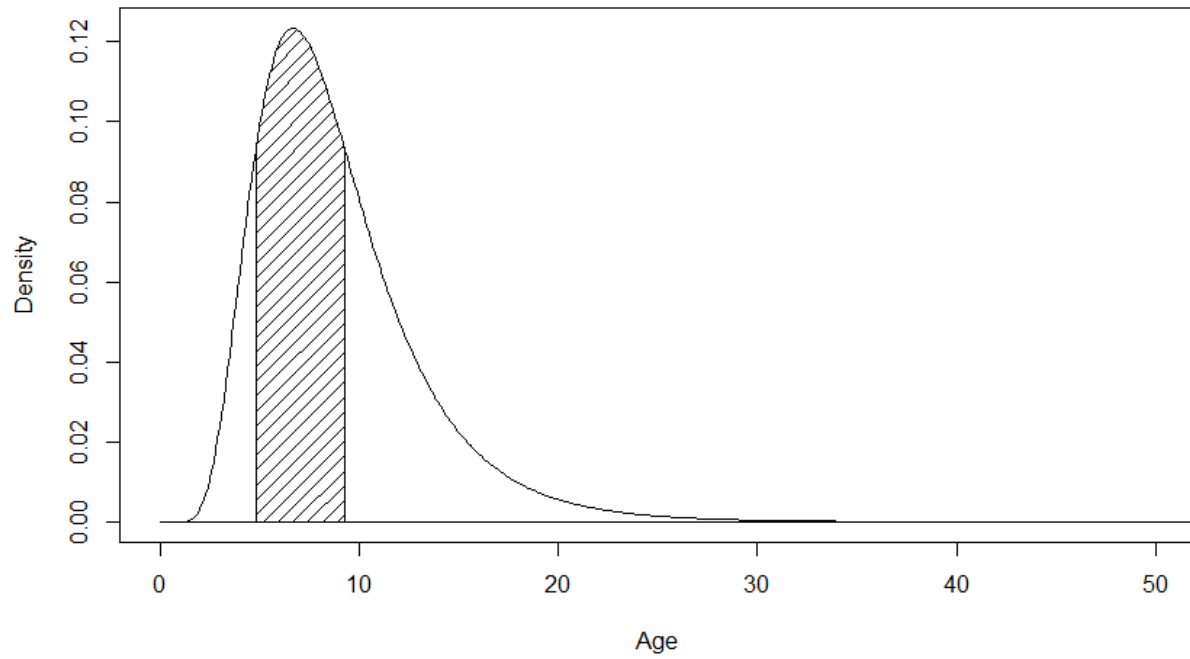
HPD female upper molar 1 stage 11 (R3/4), US Violent Deaths 2010 prior



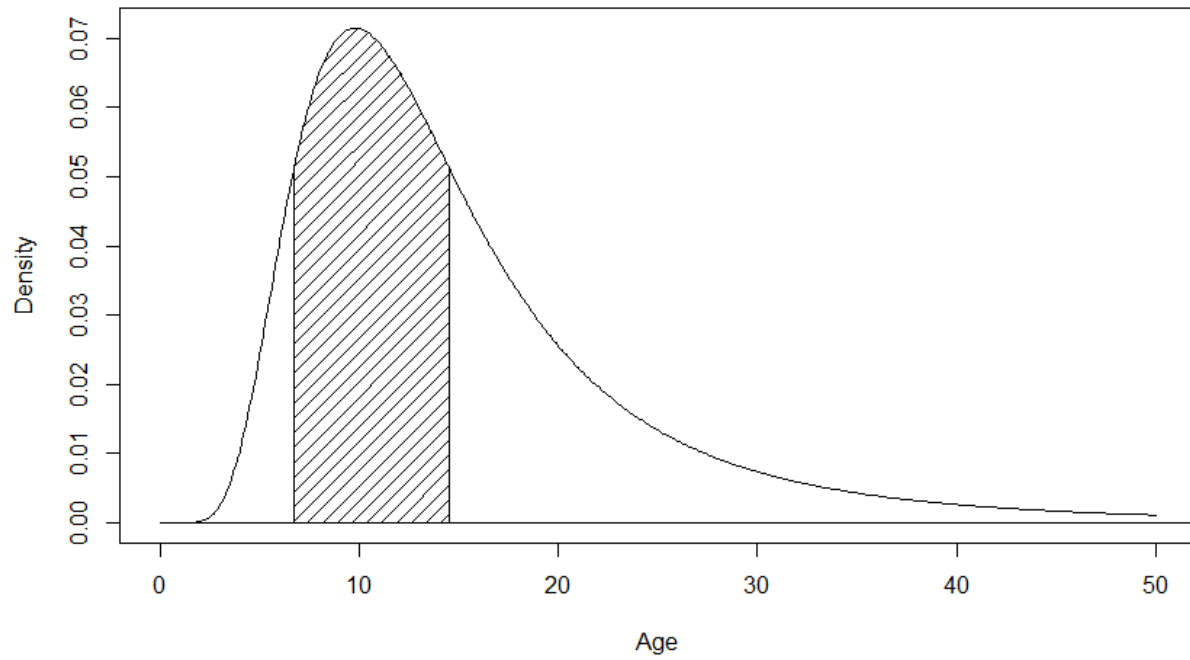
HPD female upper molar 1 stage 12 (Rc), US Violent Deaths 2010 prior



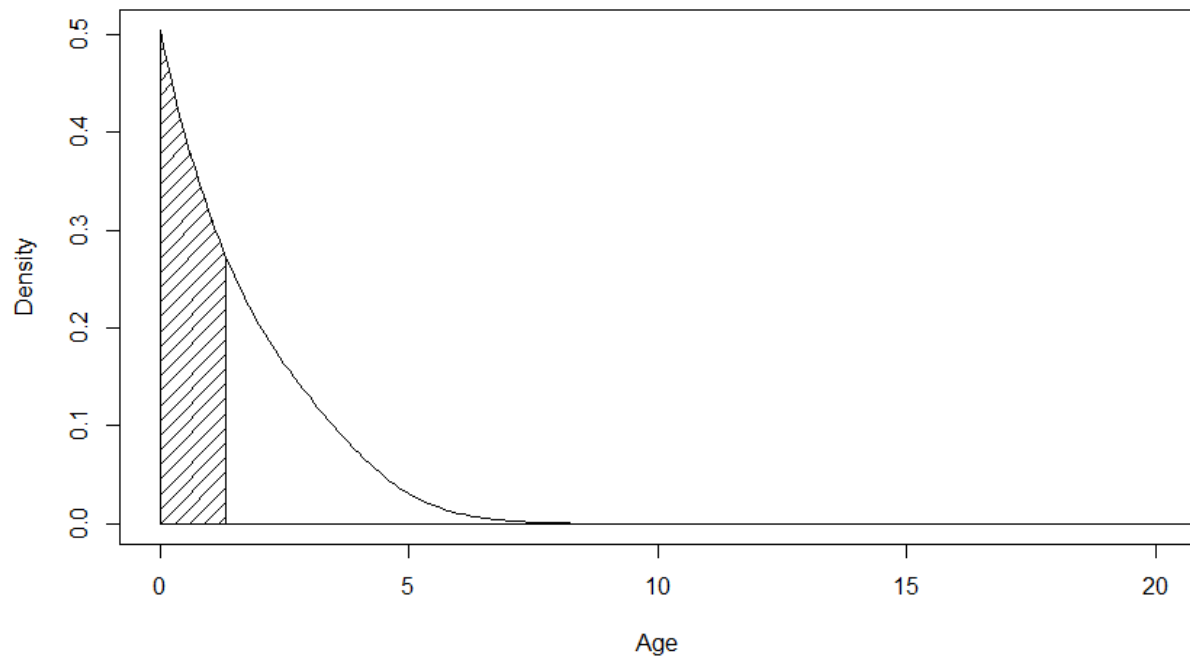
HPD female upper molar 1 stage 13 (A1/2), US Violent Deaths 2010 prior



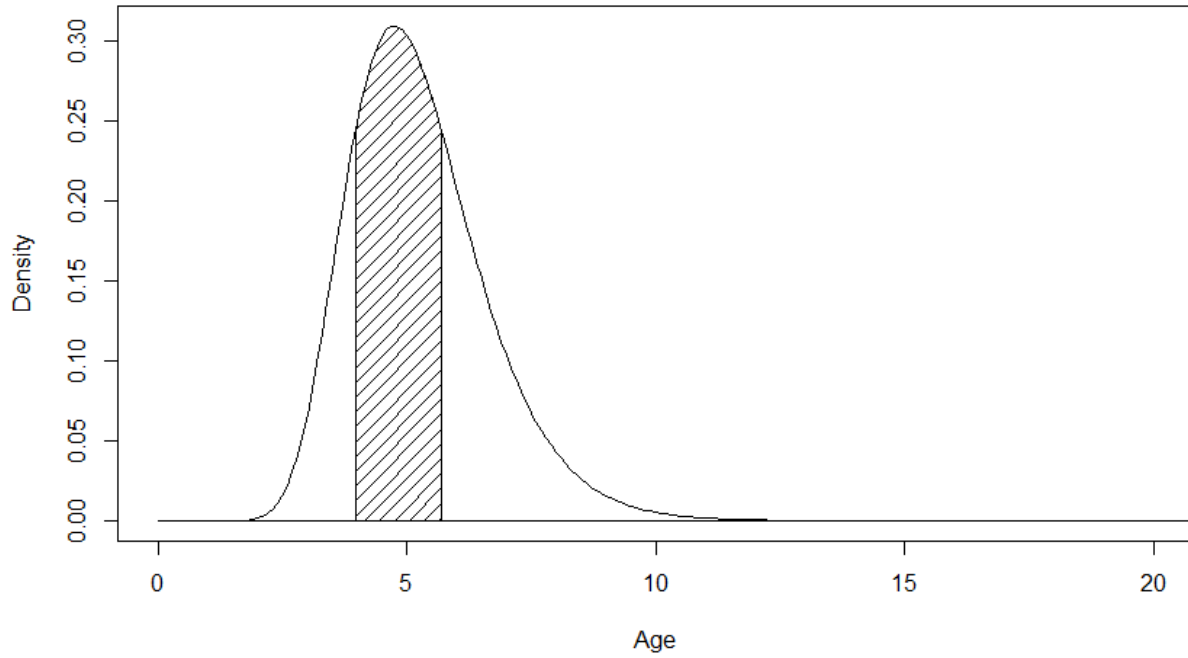
HPD female upper molar 1 stage 14 (Ac), US Violent Deaths 2010 prior



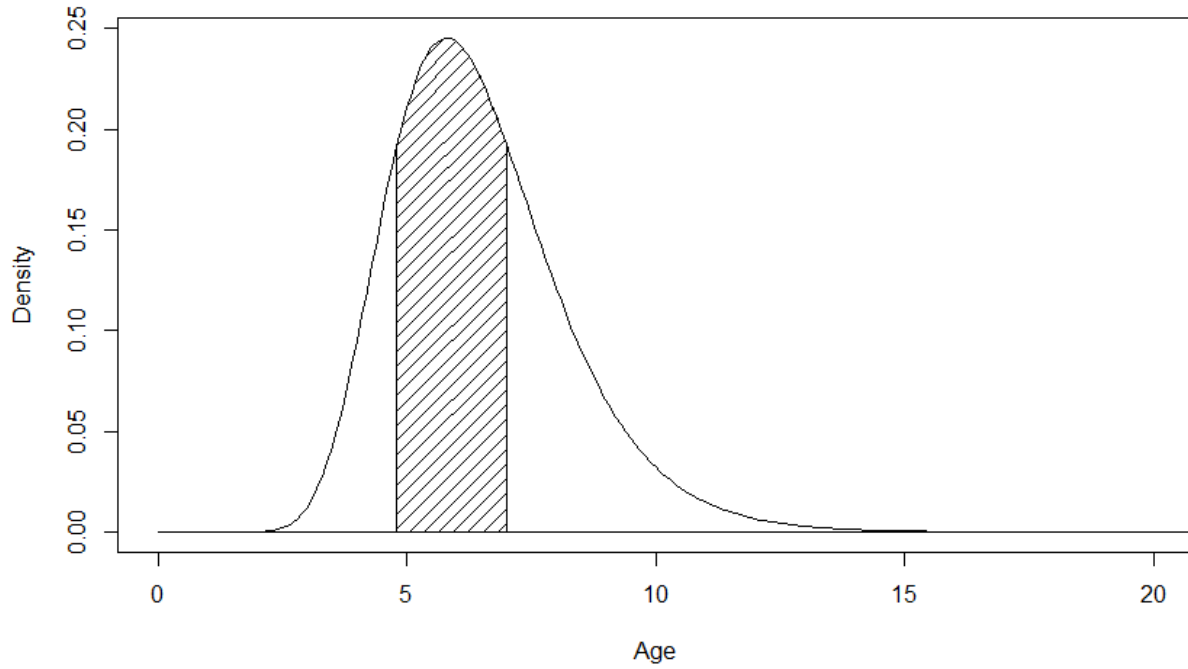
HPD female upper molar 2 stage 5 (Cr3/4), US Violent Deaths 2010 prior



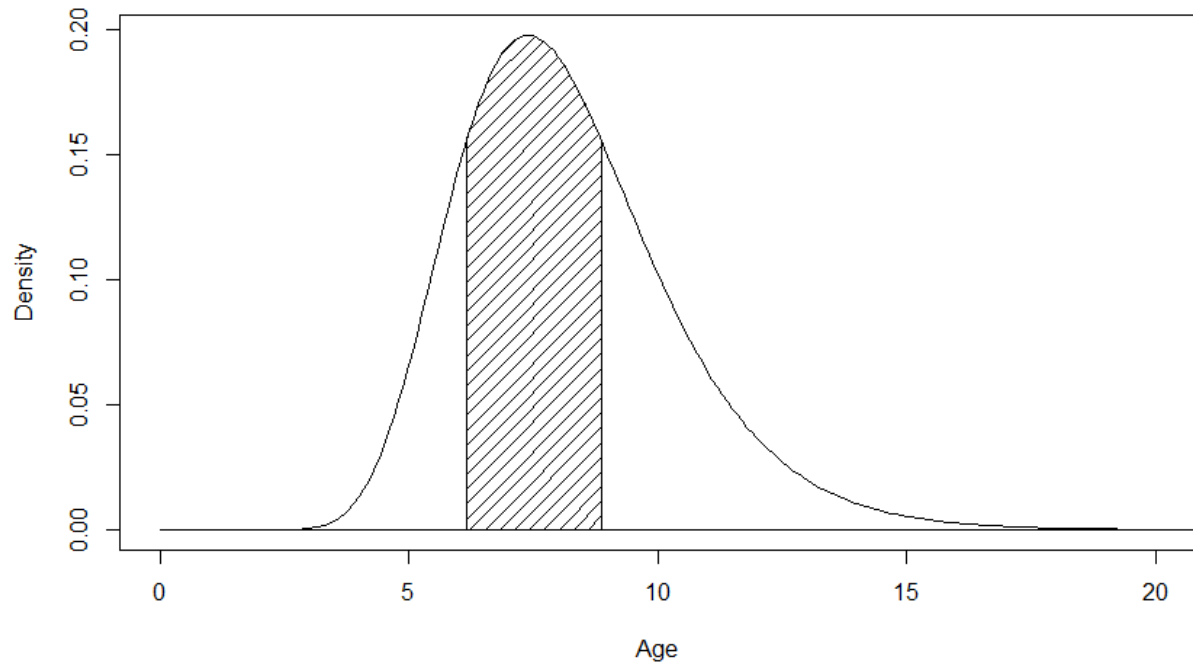
HPD female upper molar 2 stage 6 (Crc), US Violent Deaths 2010 prior



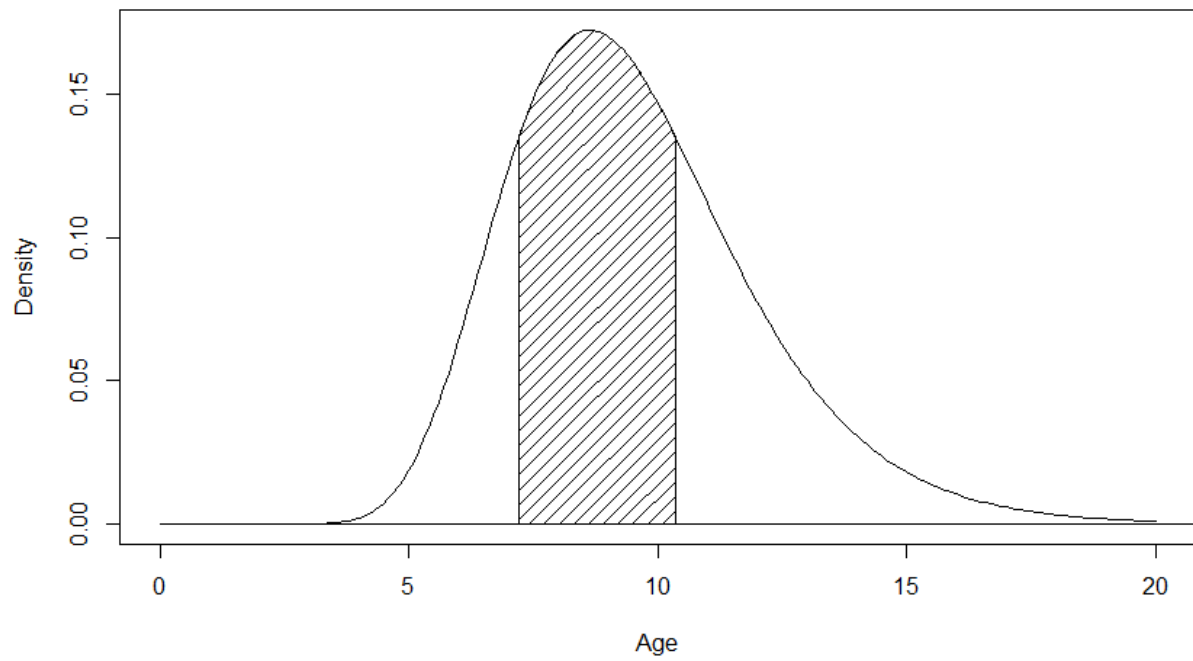
HPD female upper molar 2 stage 7 (Ri), US Violent Deaths 2010 prior



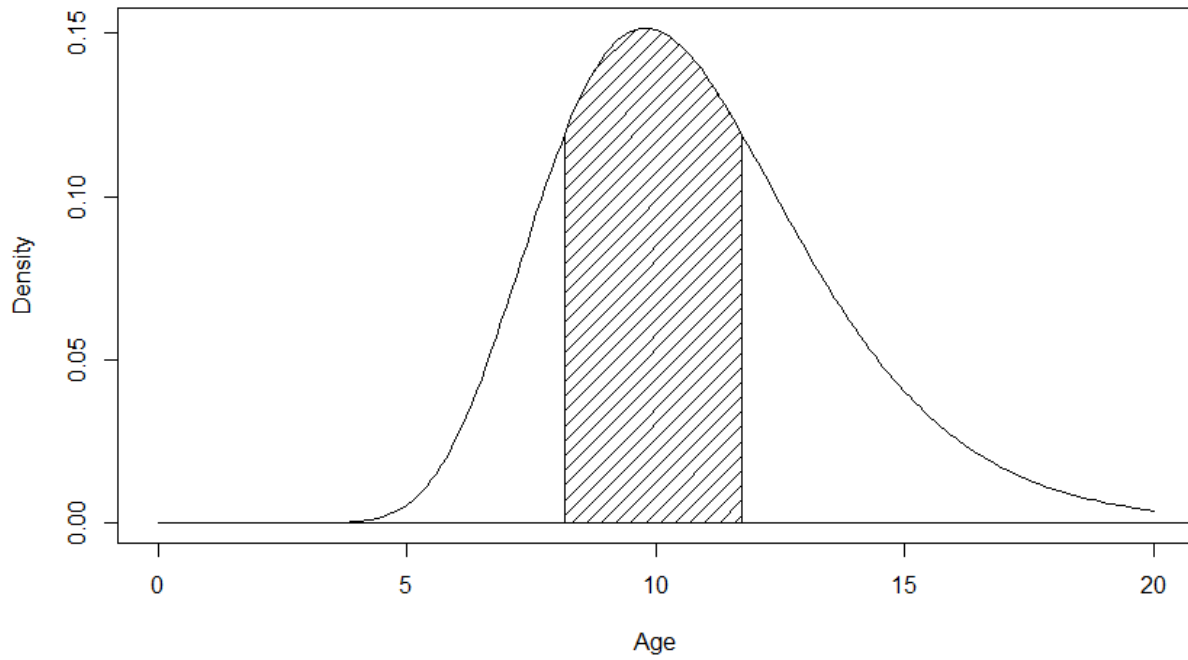
HPD female upper molar 2 stage 8 (C1i), US Violent Deaths 2010 prior



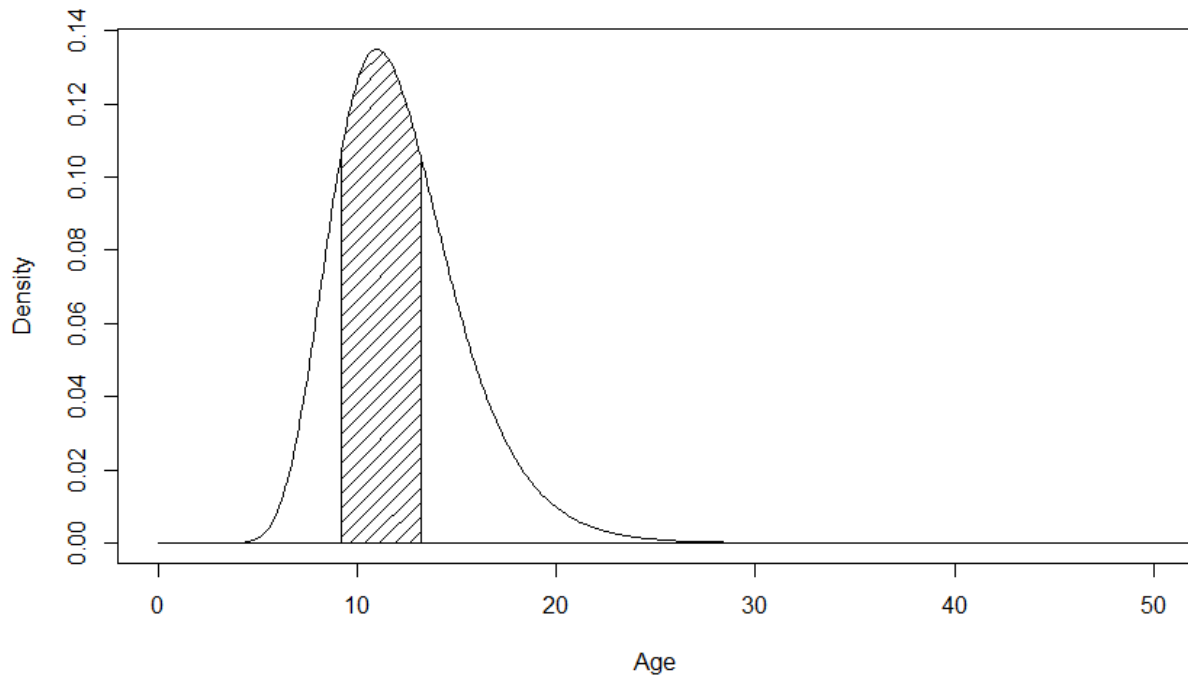
HPD female upper molar 2 stage 9 (R1/4), US Violent Deaths 2010 prior



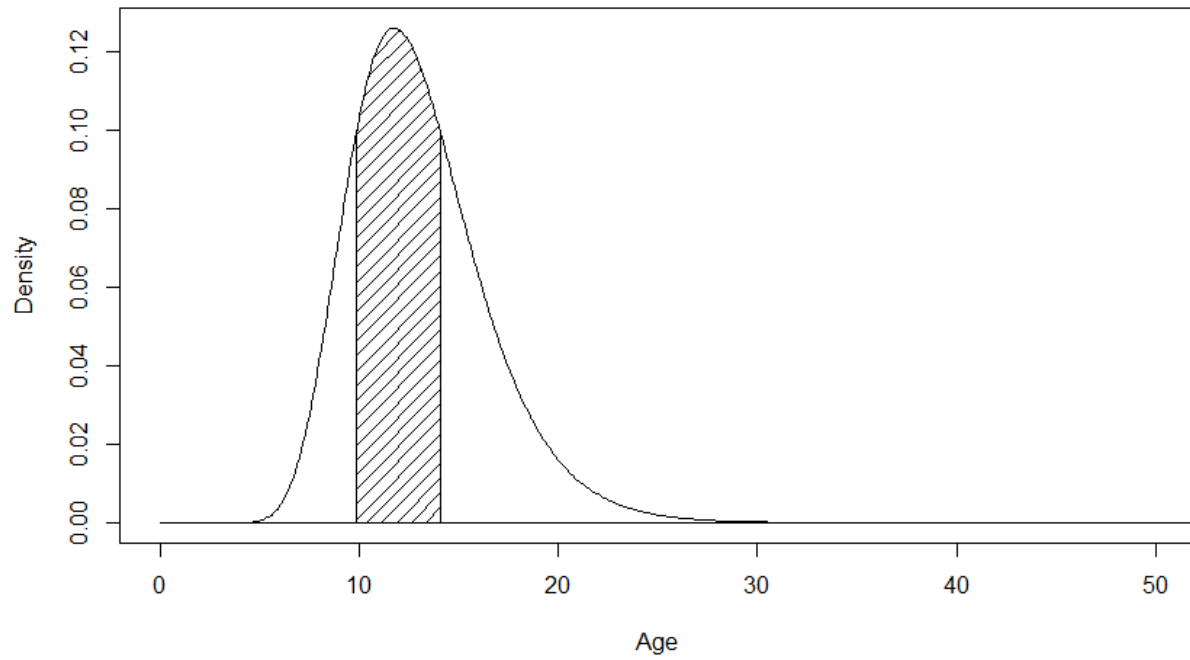
HPD female upper molar 2 stage 10 (R1/2), US Violent Deaths 2010 prior



HPD female upper molar 2 stage 11 (R3/4), US Violent Deaths 2010 prior



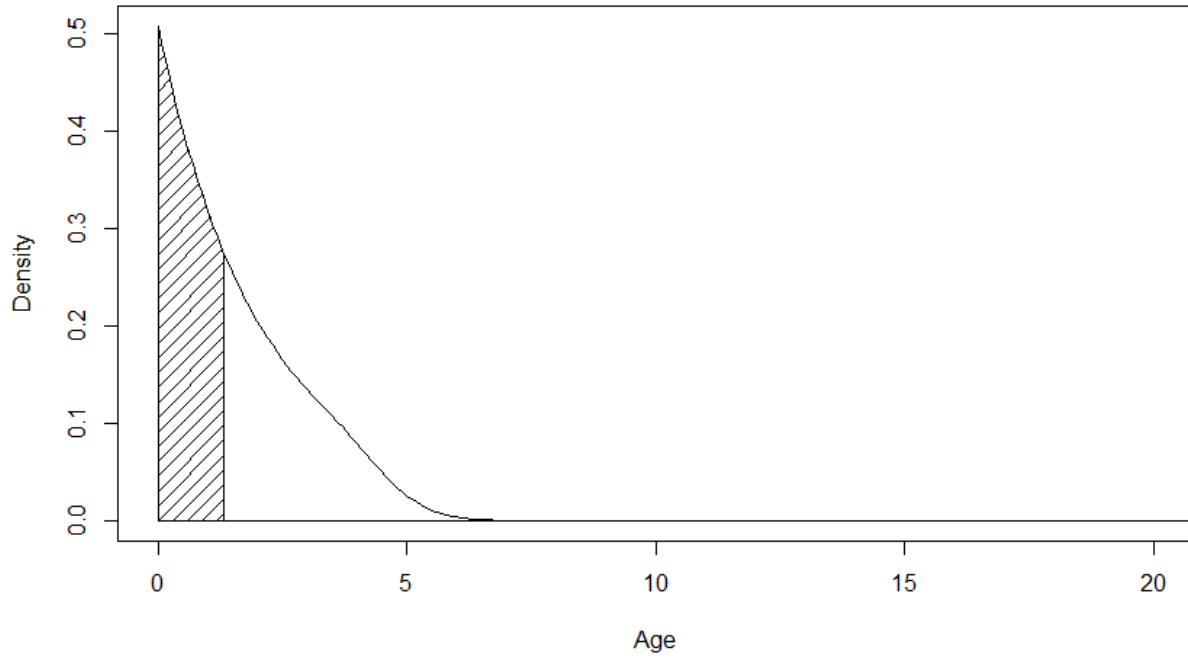
HPD female upper molar 2 stage 12 (Rc), US Violent Deaths 2010 prior



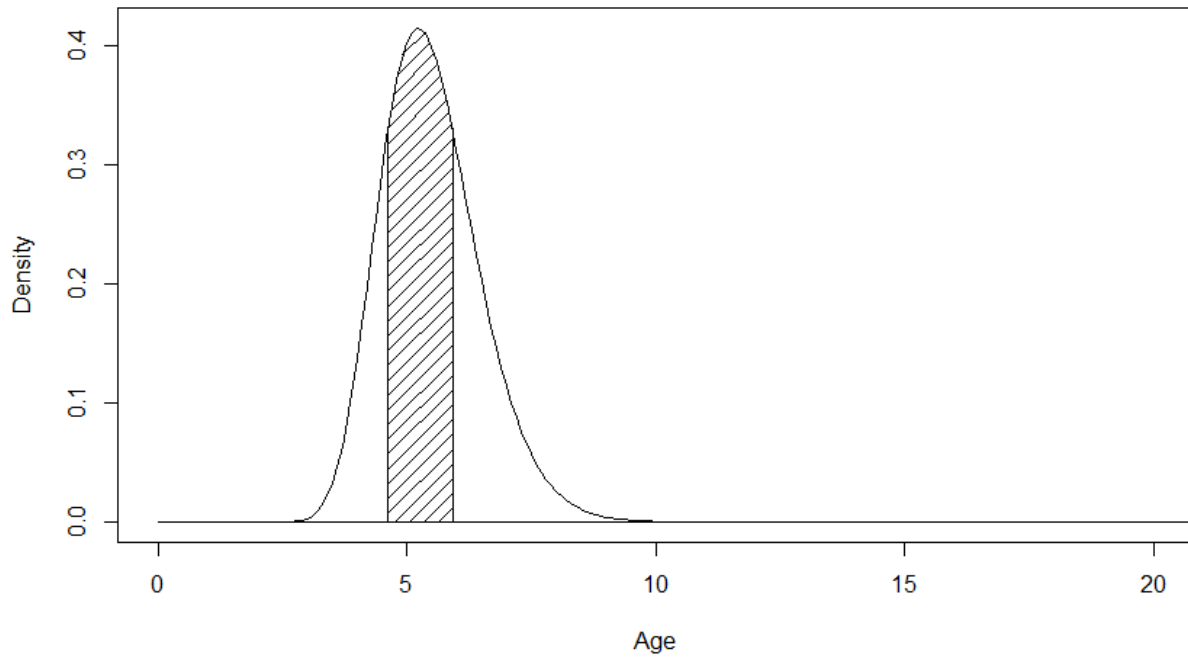
HPD female upper molar 2 stage 13 (A1/2), US Violent Deaths 2010 prior



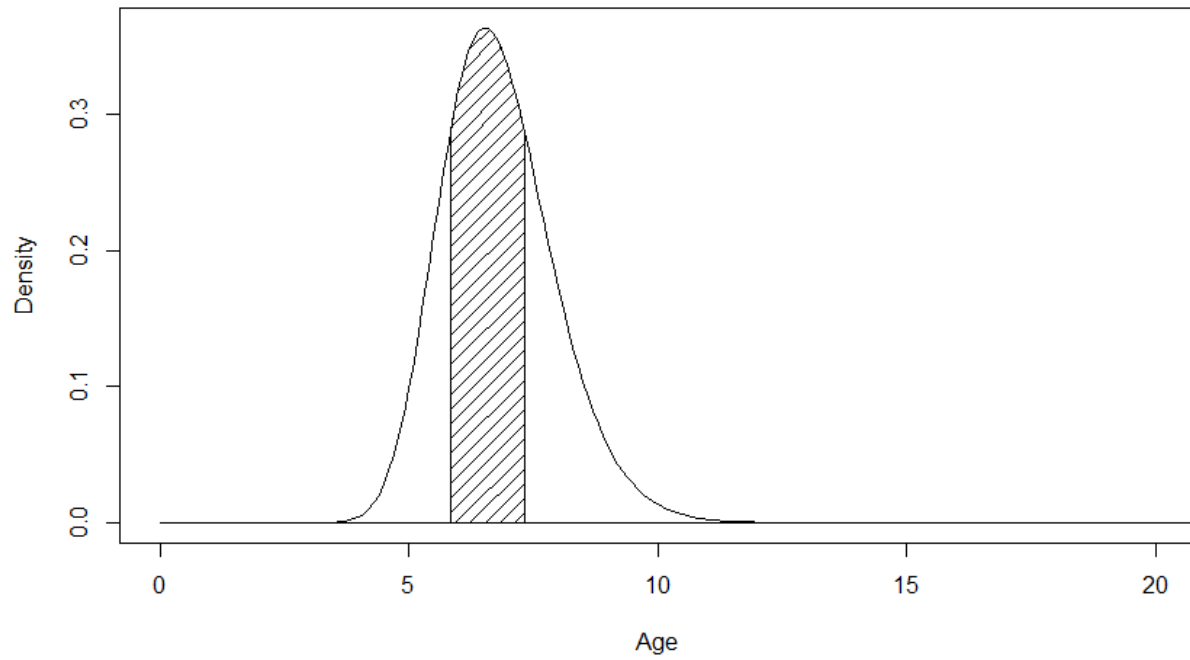
HPD female lower incisor 1 stage 9 (R1/4), US Violent Deaths 2010 prior



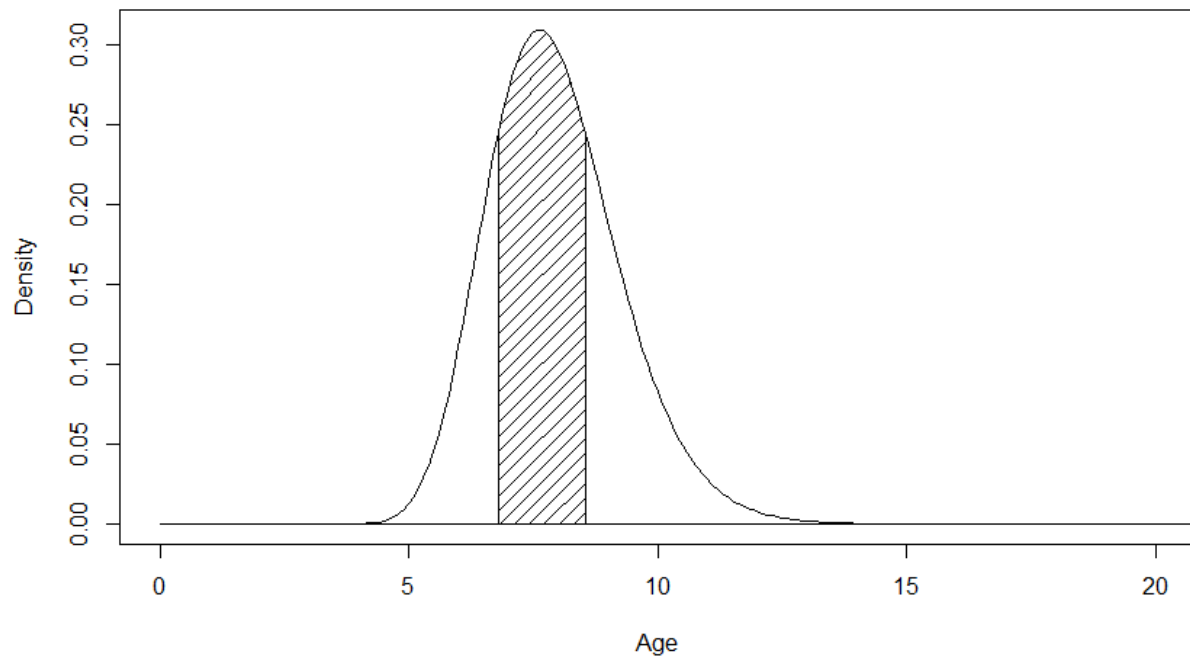
HPD female lower incisor 1 stage 11 (R3/4), US Violent Deaths 2010 prior



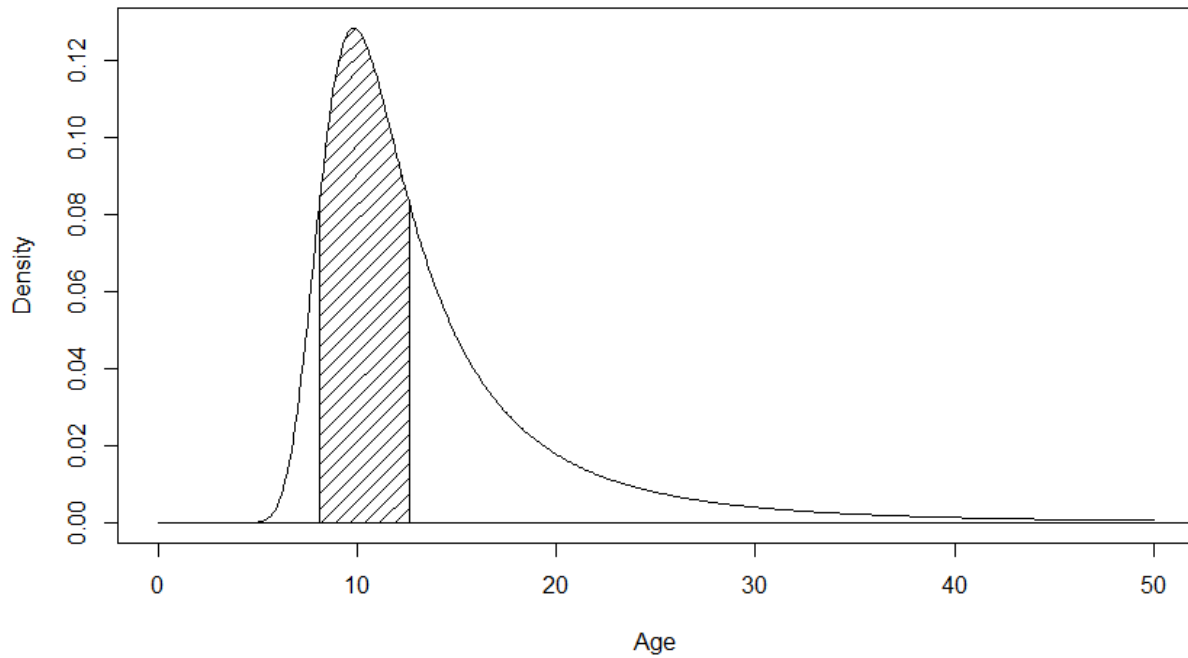
HPD female lower incisor 1 stage 12 (Rc), US Violent Deaths 2010 prior



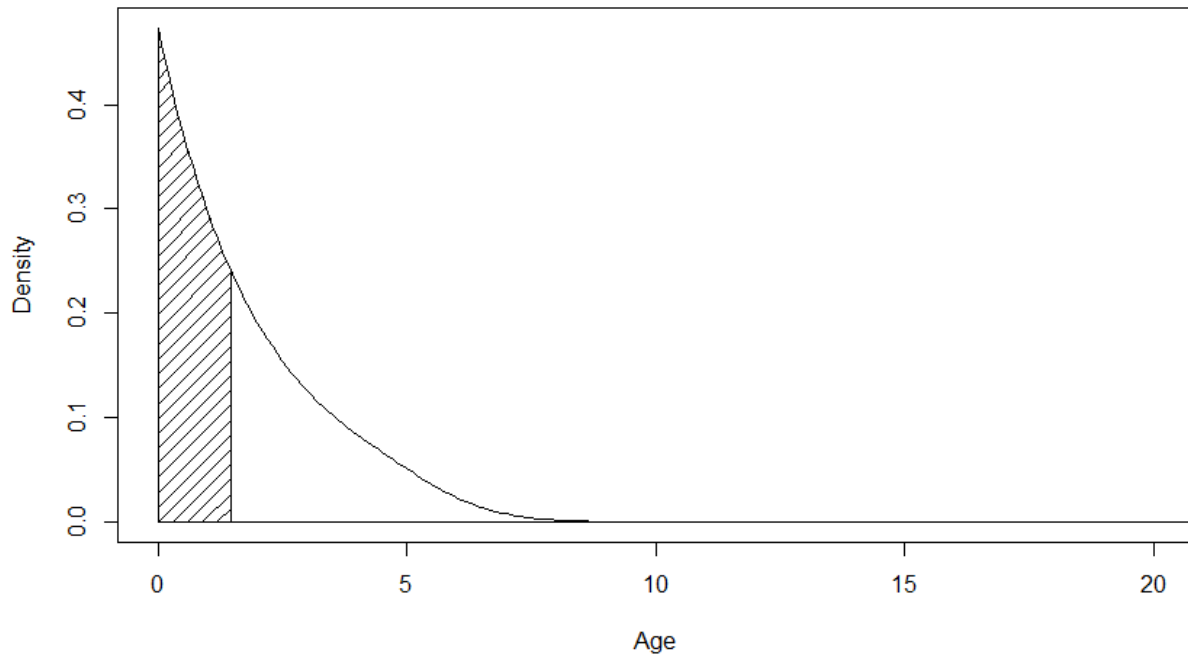
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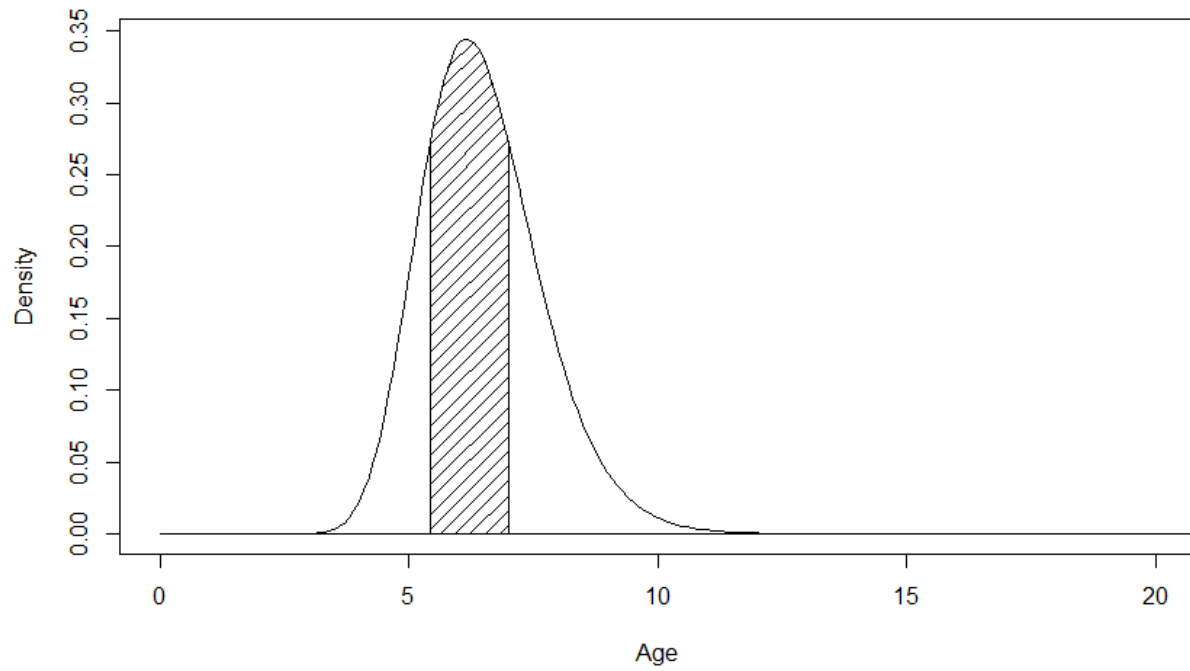
HPD female lower incisor 1 stage 14 (Ac), US Violent Deaths 2010 prior



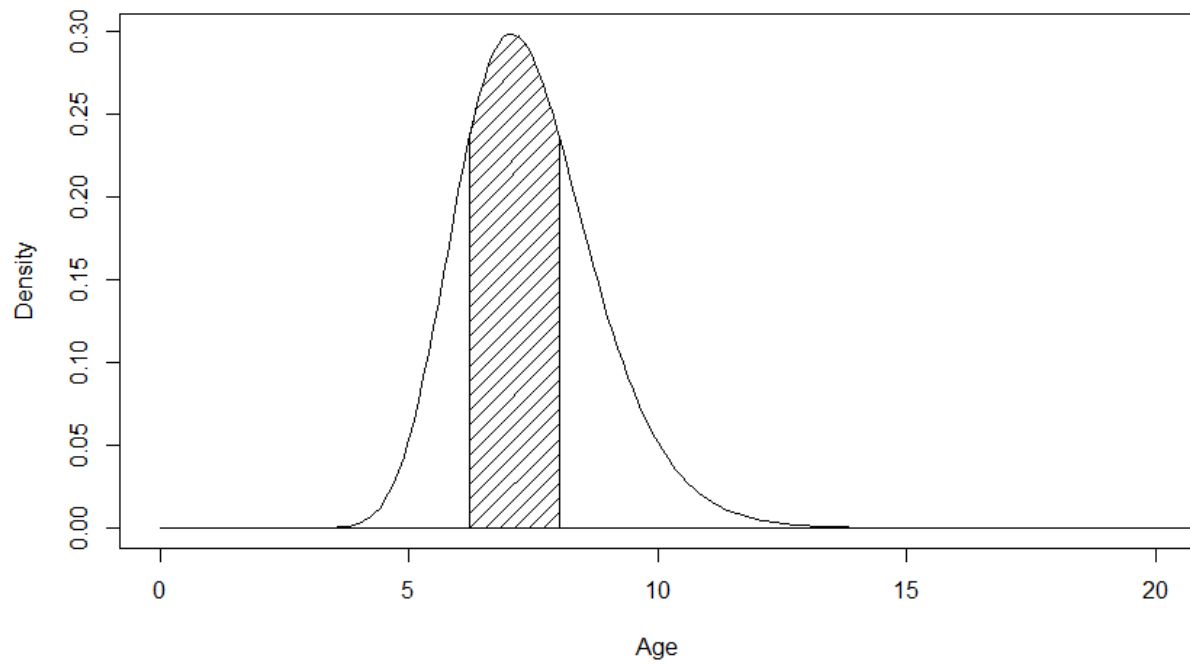
HPD female lower incisor 1 stage 10 (R 1/2), US Violent Deaths 2010 prior



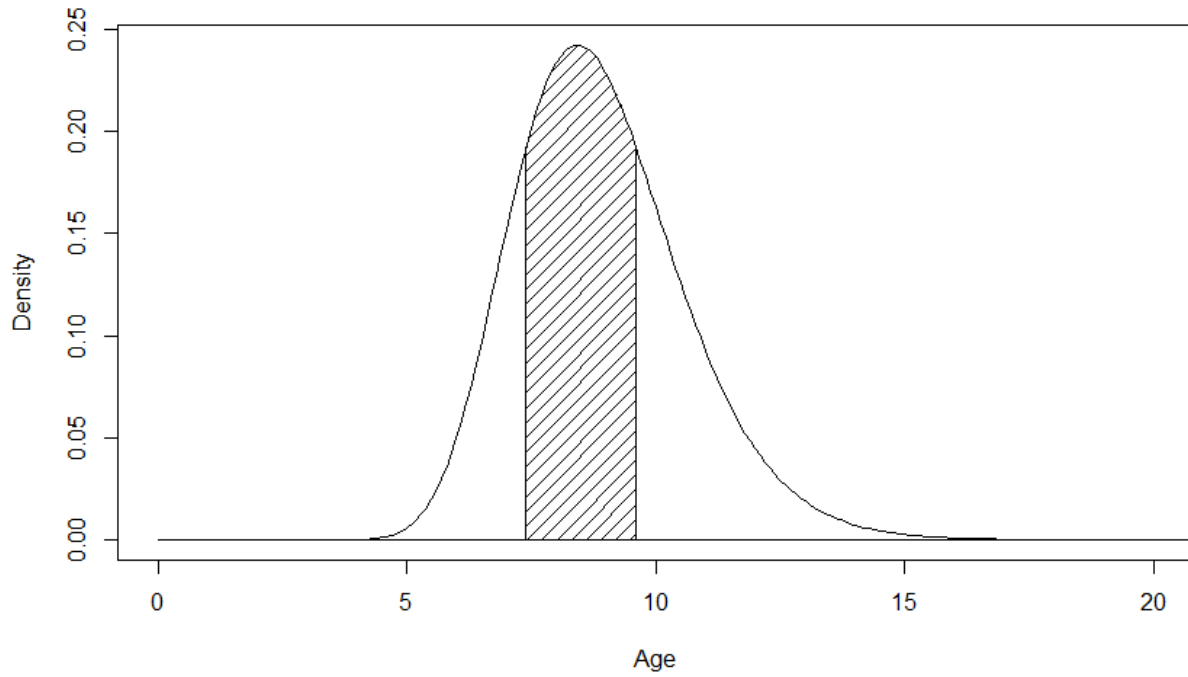
HPD female lower incisor 1 stage 11 (R 3/4), US Violent Deaths 2010 prior



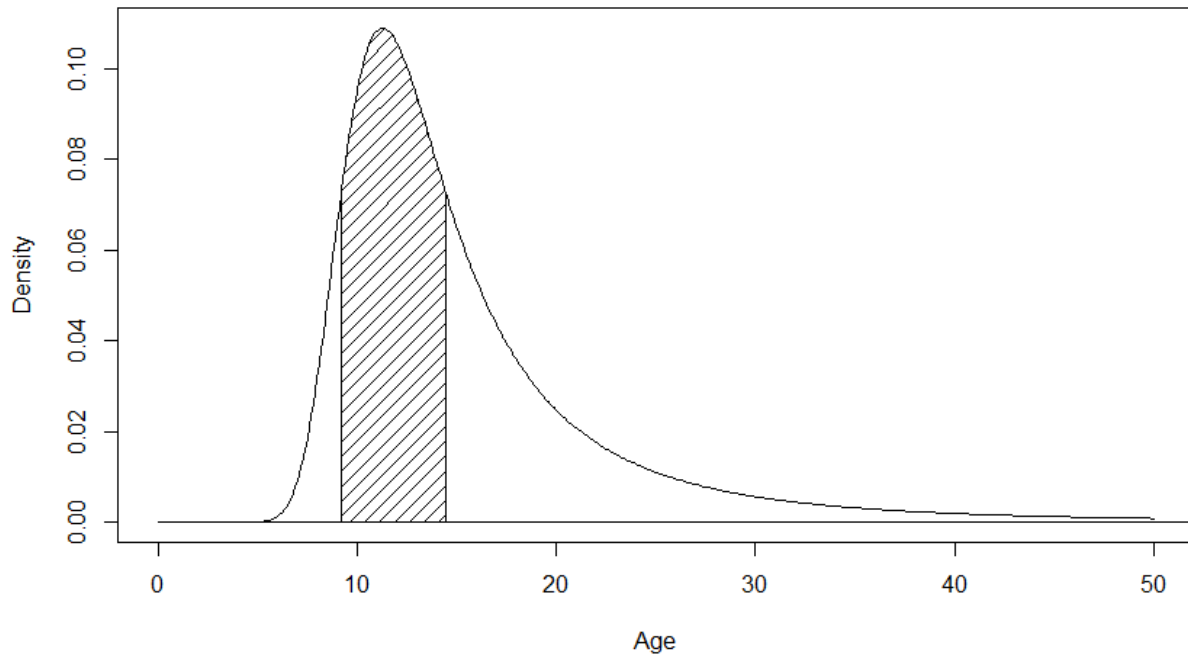
HPD female lower incisor 1 stage 12 (Rc), US Violent Deaths 2010 prior



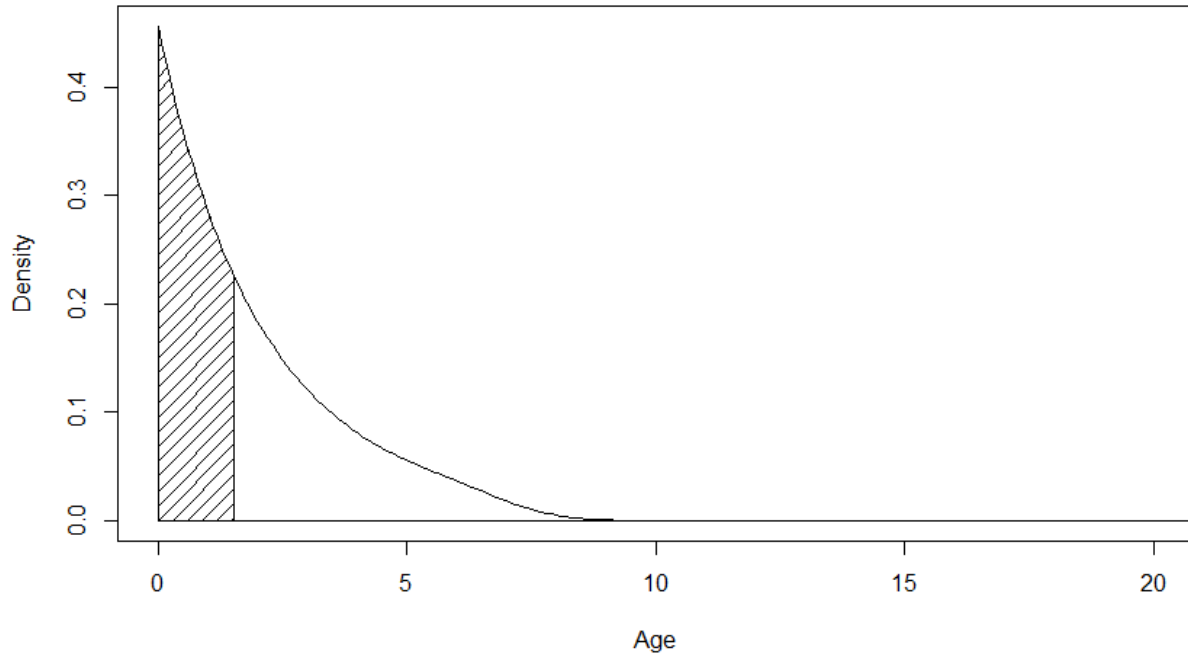
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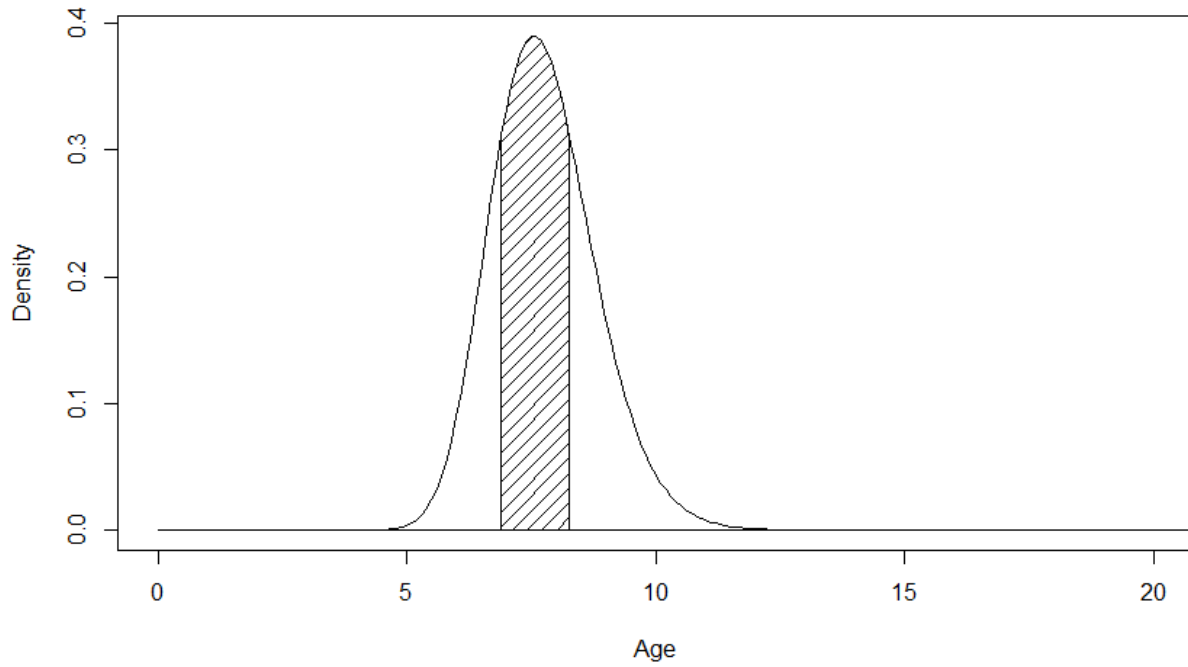
HPD female lower incisor 1 stage 14 (Ac), US Violent Deaths 2010 prior



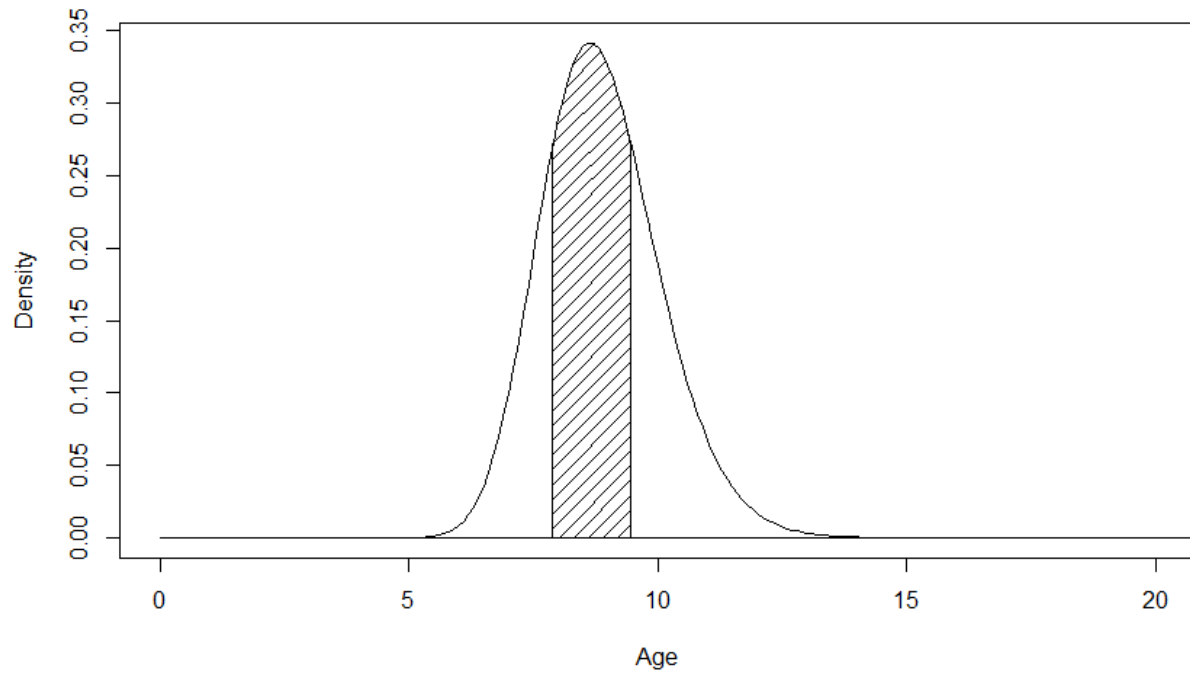
HPD female lower canine stage 9 (R 1/4), US Violent Deaths 2010 prior



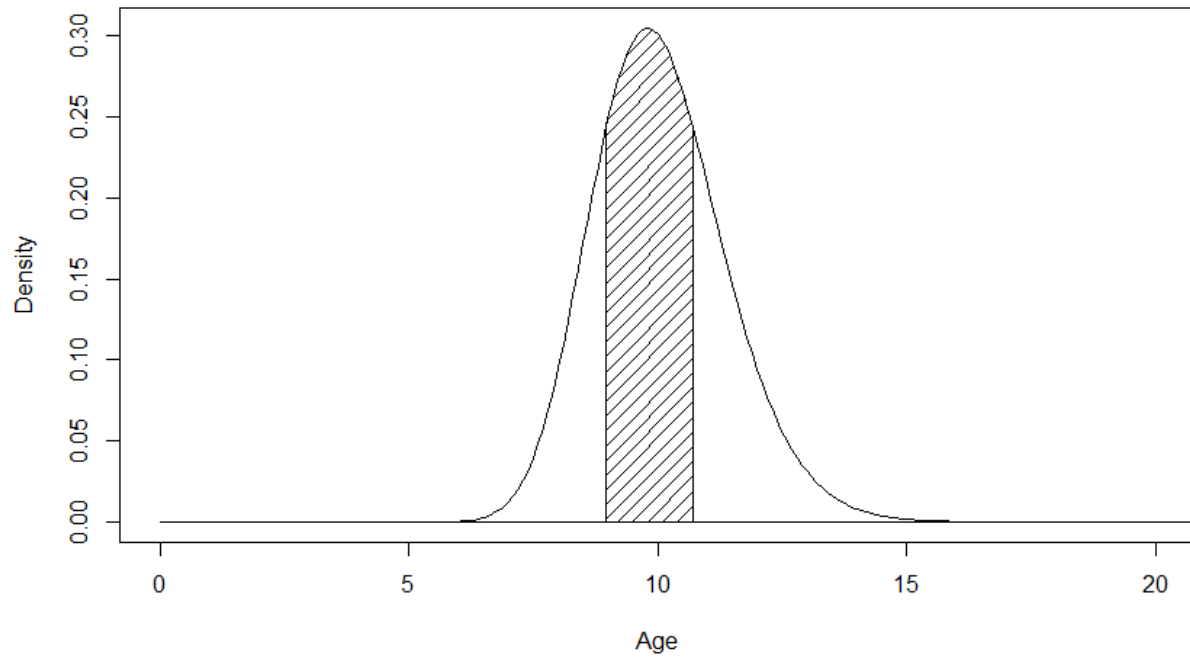
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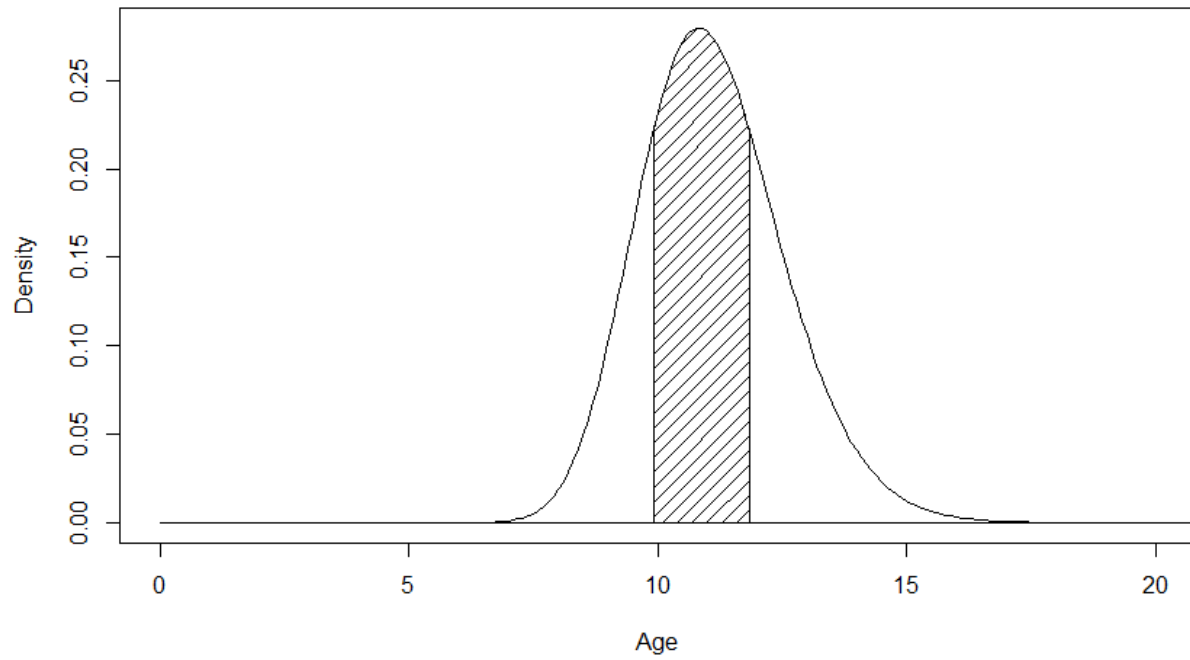
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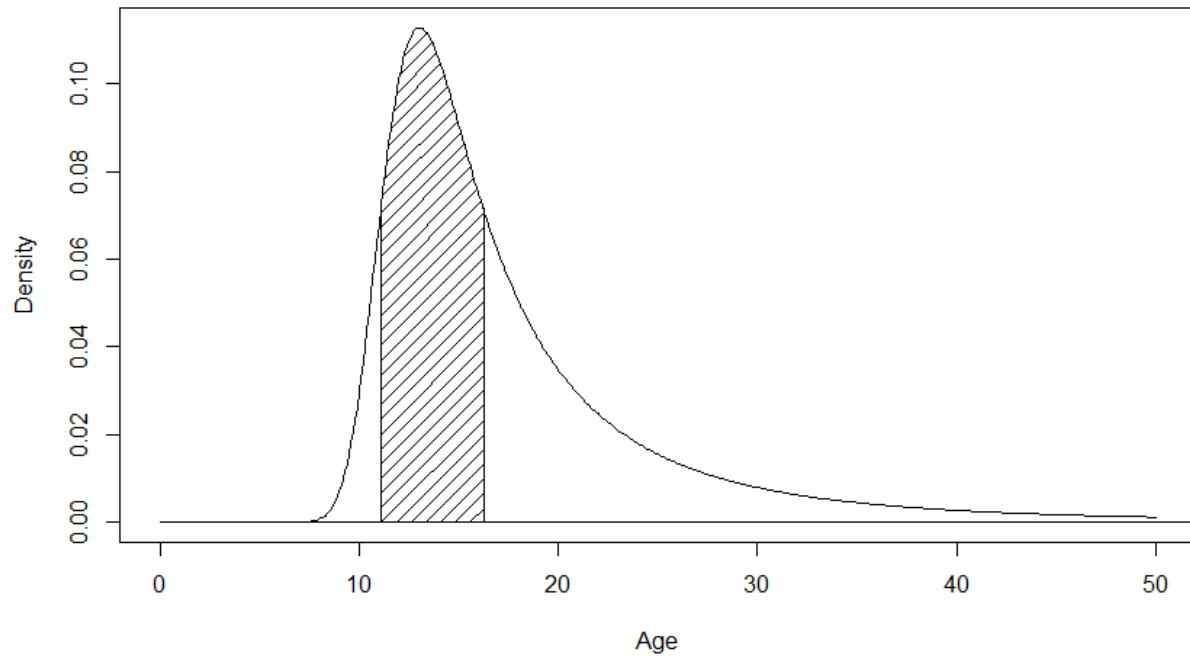
HPD female lower canine stage 12 (Rc), US Violent Deaths 2010 prior



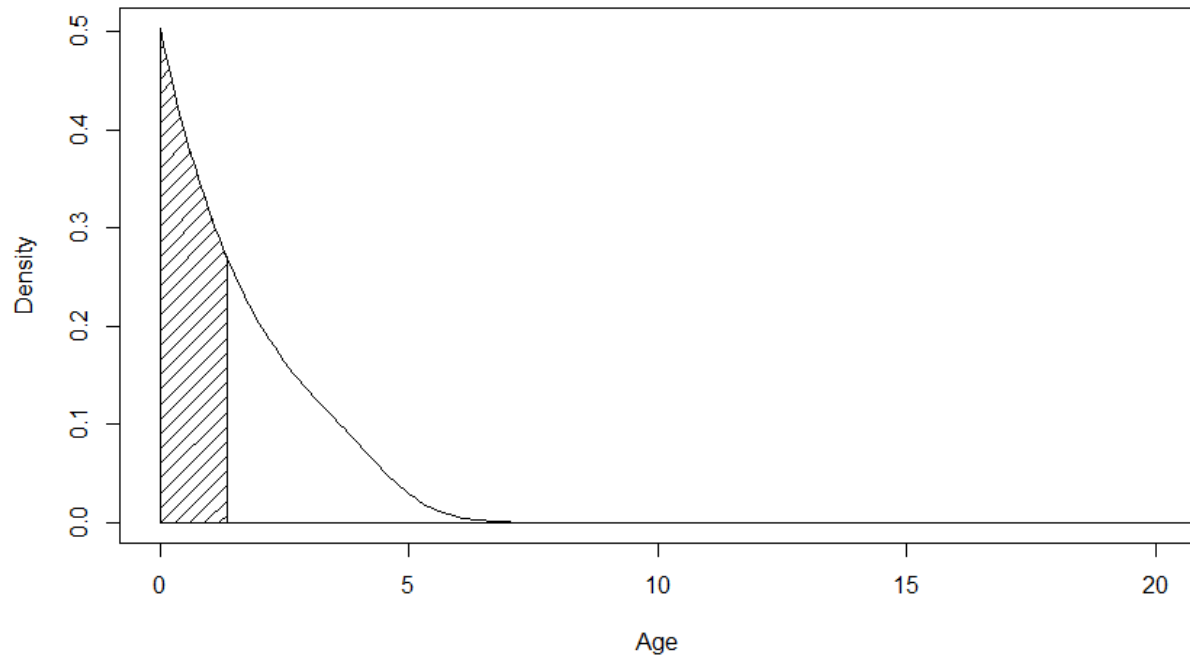
HPD female lower canine stage 13 (A 1/2), US Violent Deaths 2010 prior



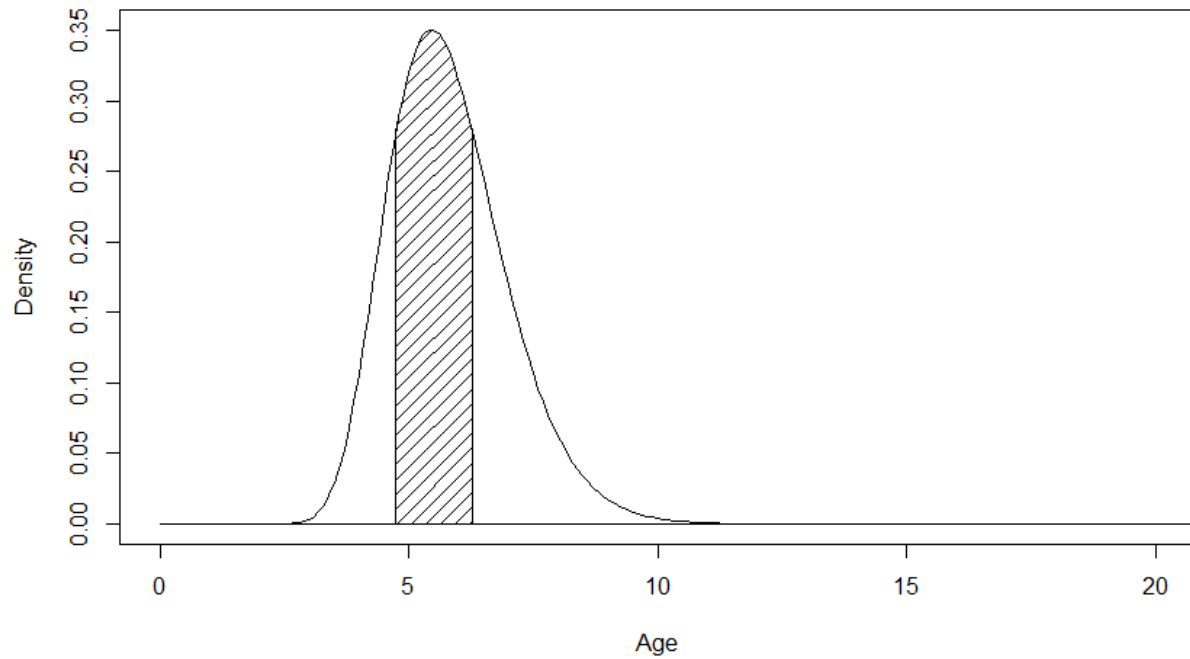
HPD female lower canine stage 14 (Ac), US Violent Deaths 2010 prior



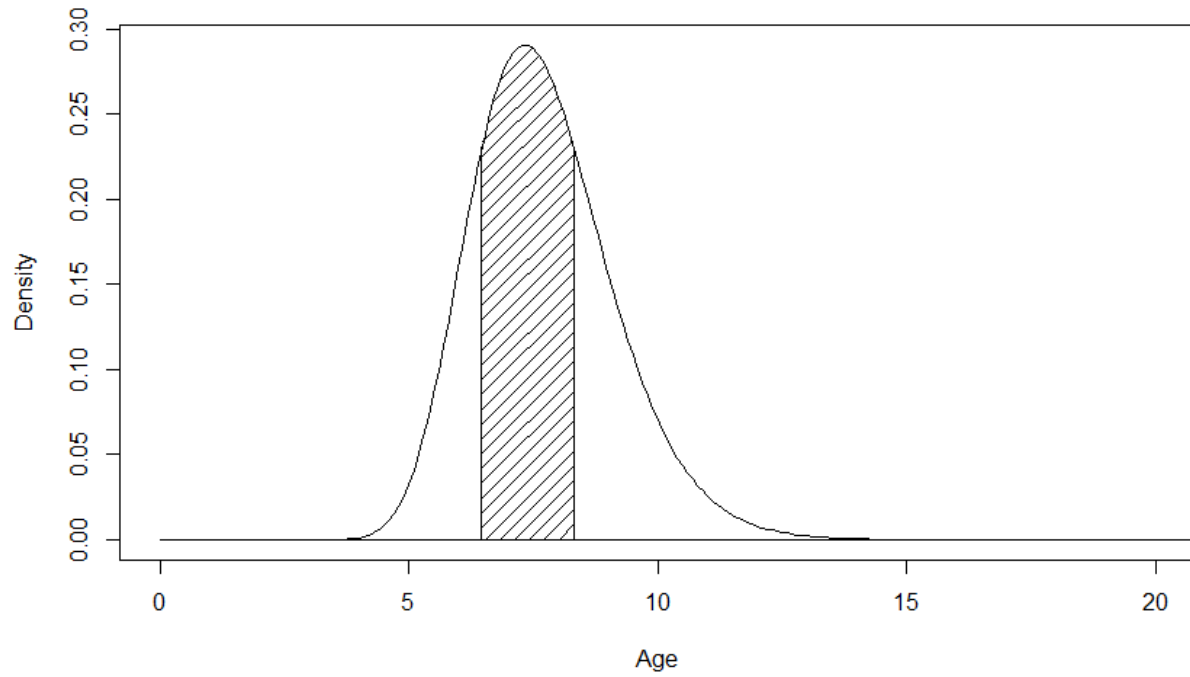
HPD female lower premolar 1 stage 6 (Crc), US Violent Deaths 2010 prior



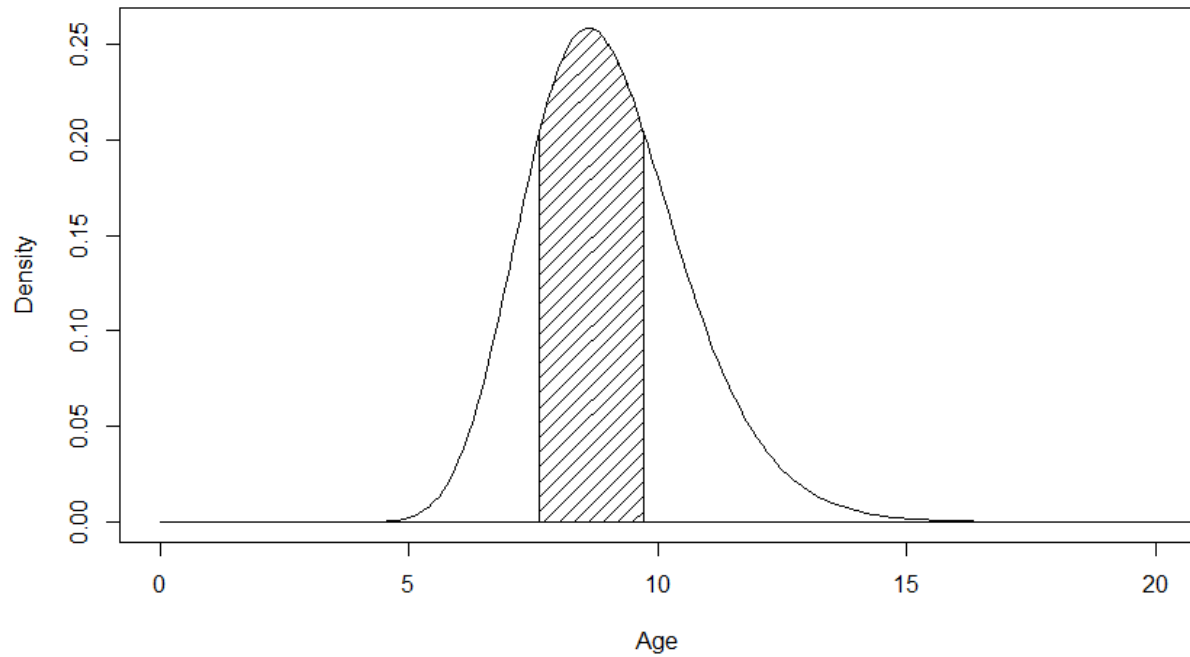
HPD female lower premolar 1 stage 7 (Ri), US Violent Deaths 2010 prior



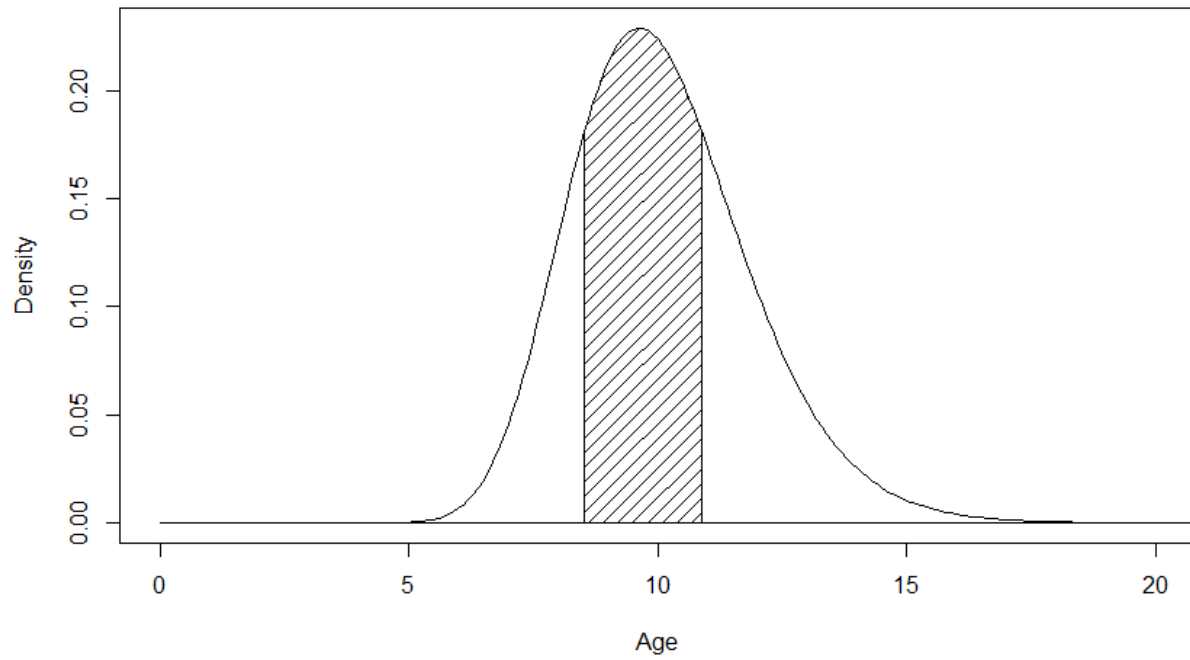
HPD female lower premolar 1 stage 9 (R 1/4), US Violent Deaths 2010 prior



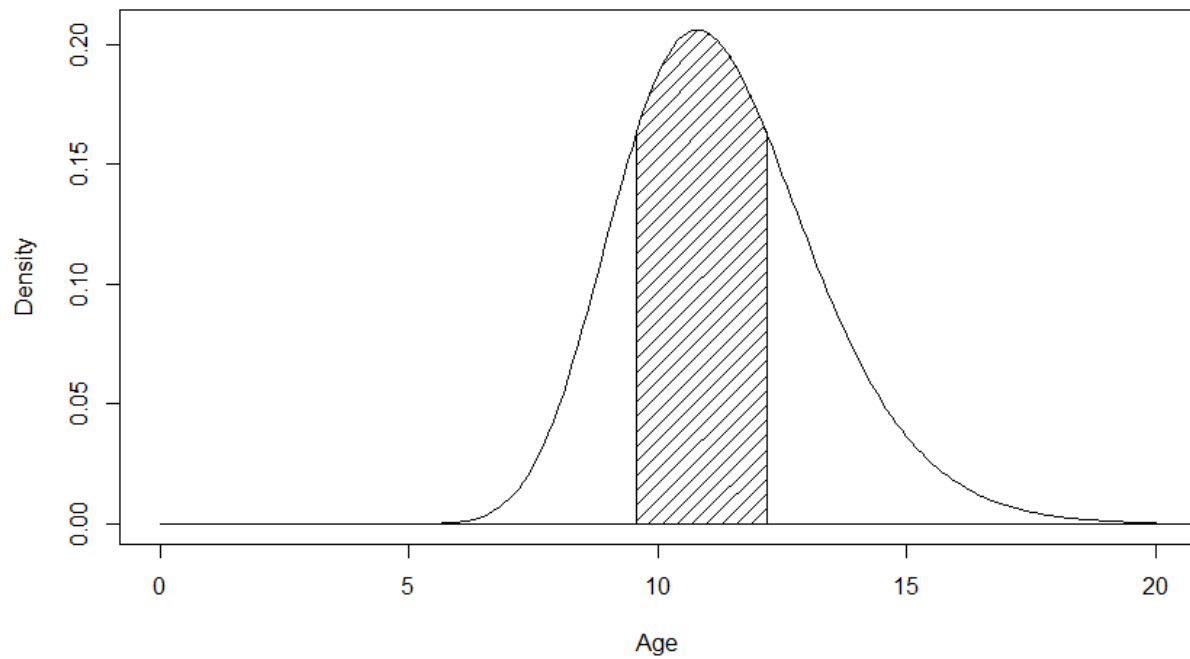
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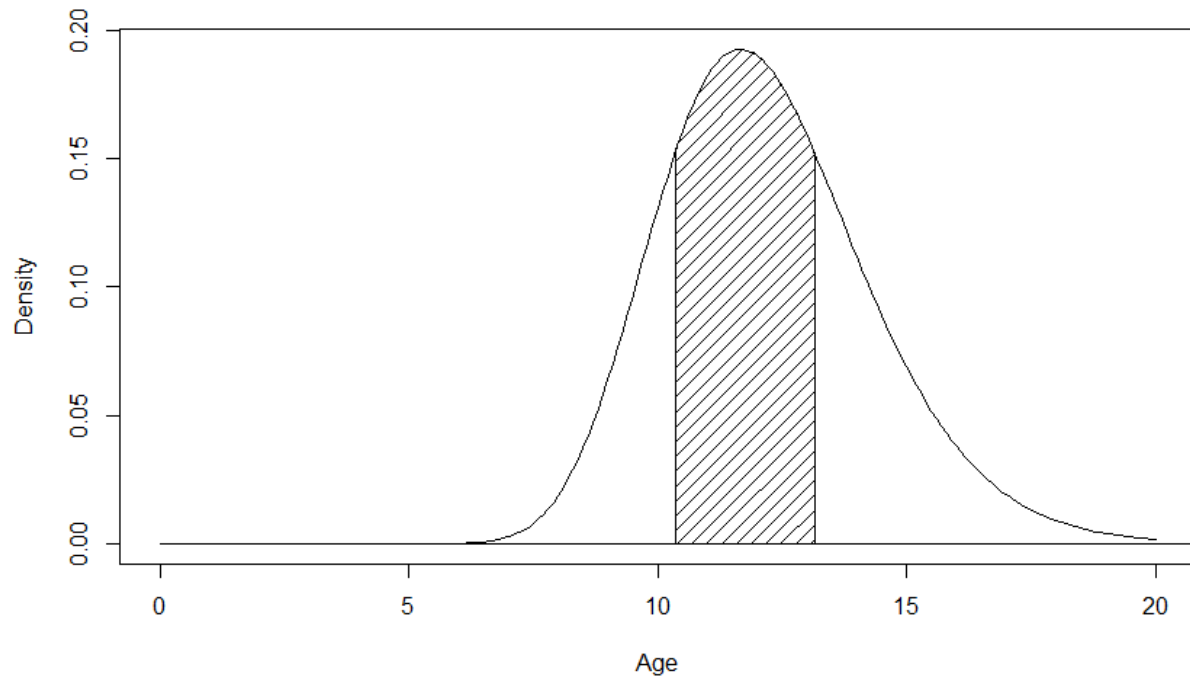
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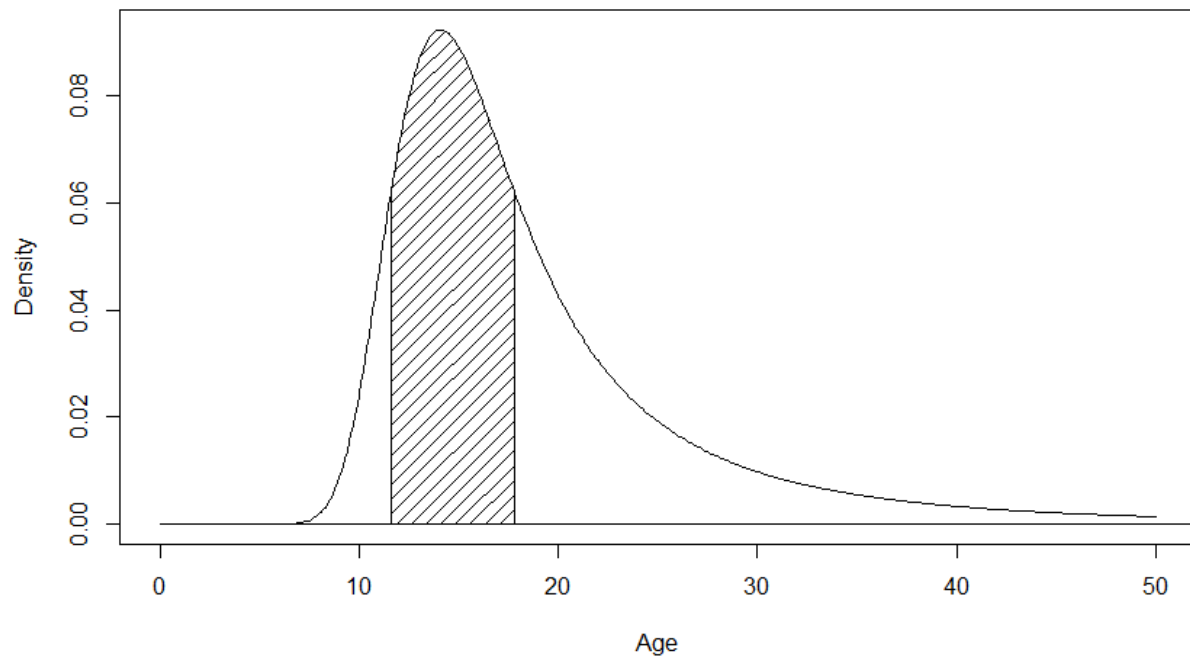
HPD female lower premolar 1 stage 12 (Rc), US Violent Deaths 2010 prior



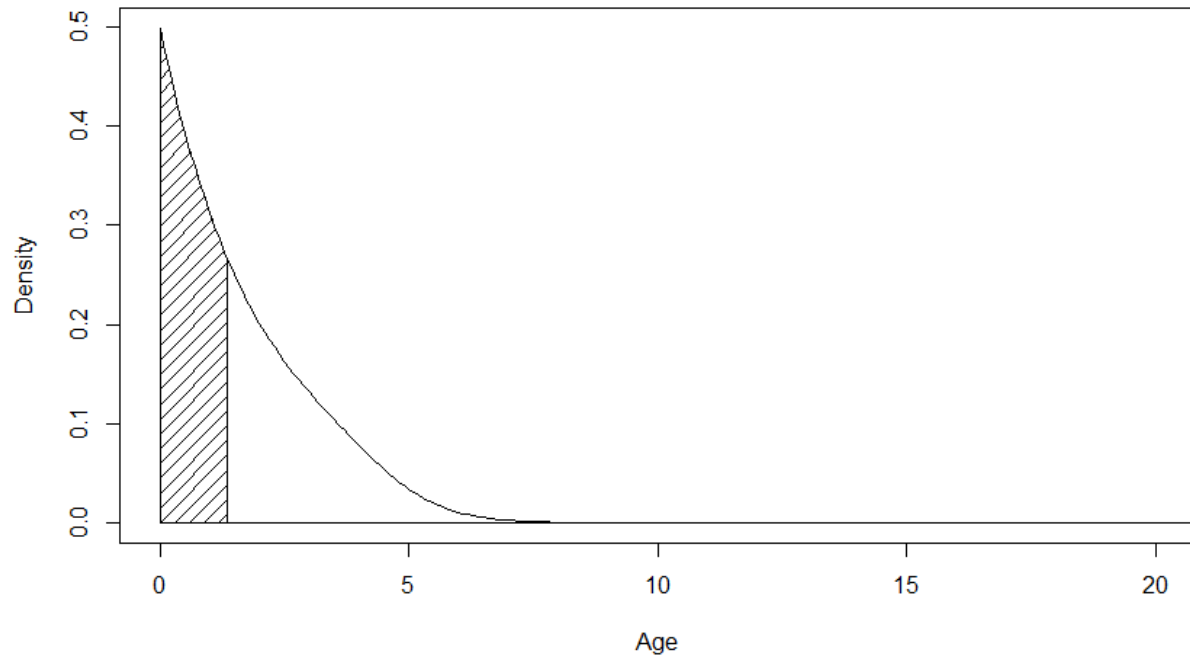
HPD female lower premolar 1 stage 13 (A 1/2), US Violent Deaths 2010 prior



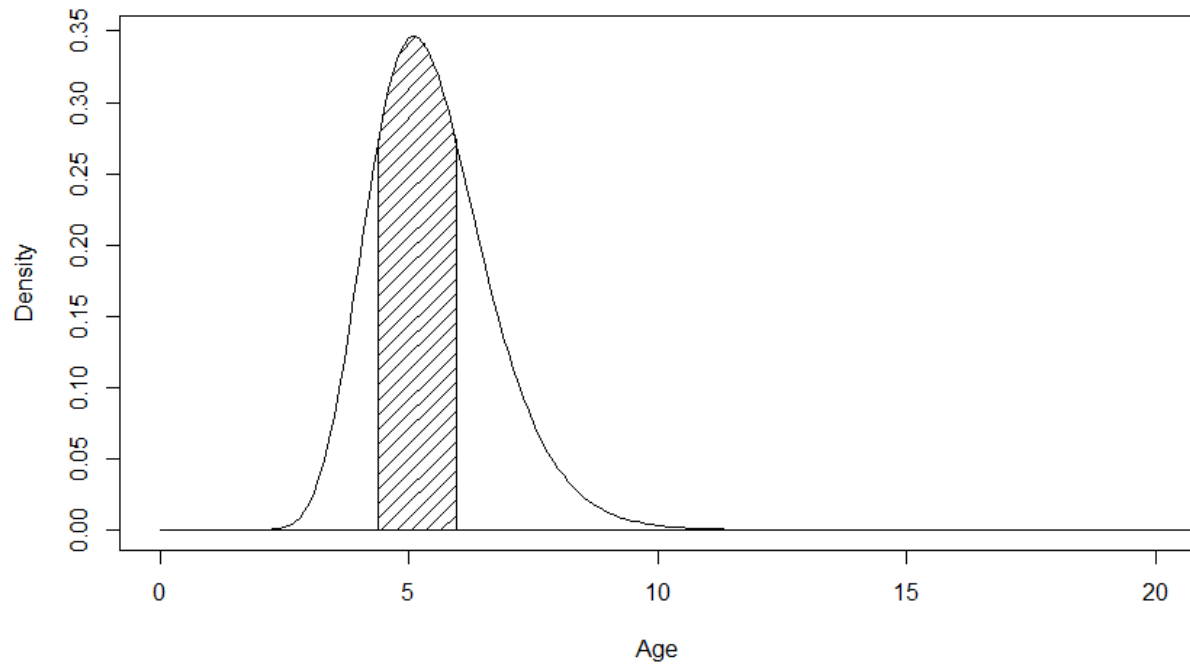
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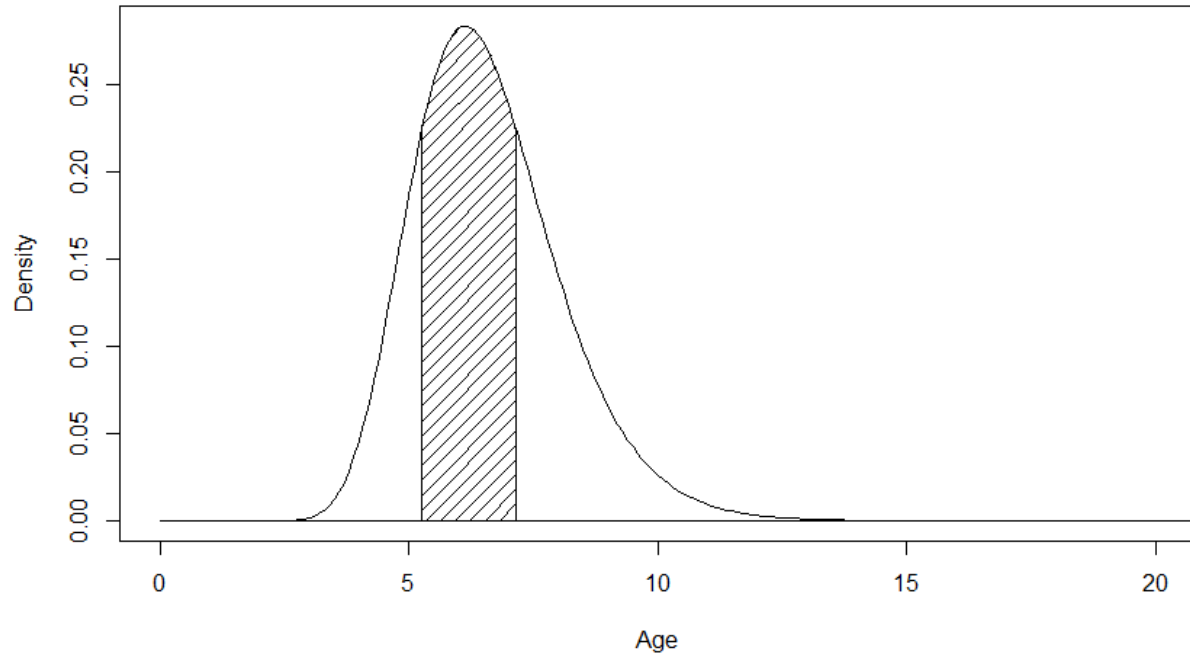
HPD female lower molar 1 stage 10 (R 1/2), US Violent Deaths 2010 prior



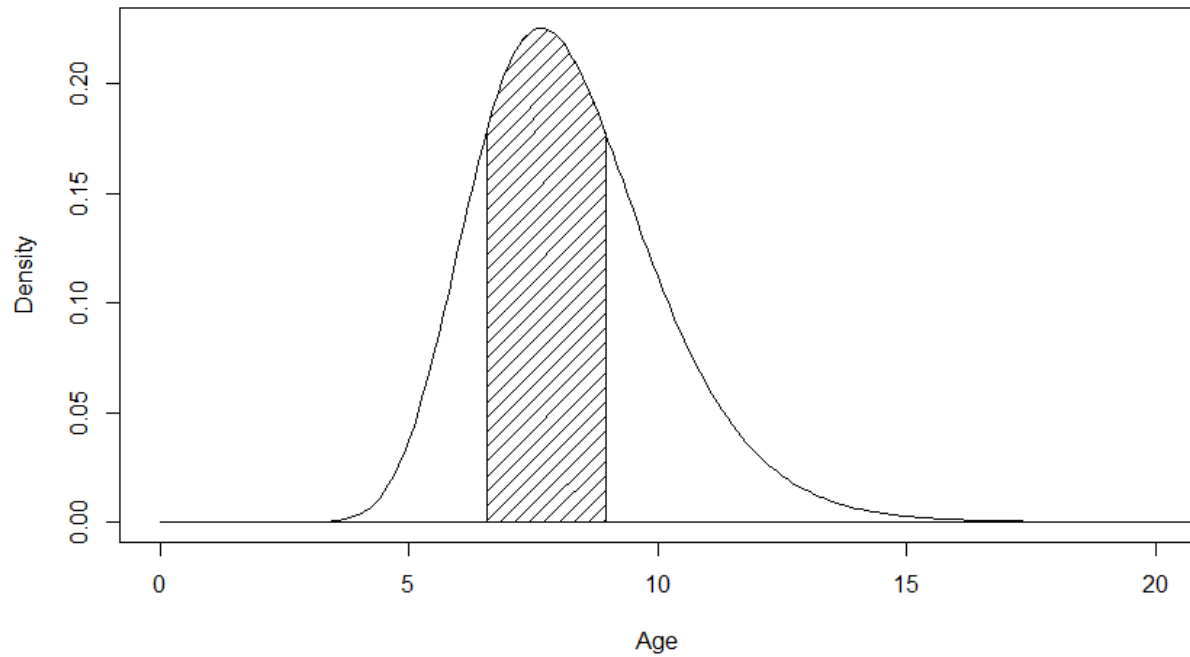
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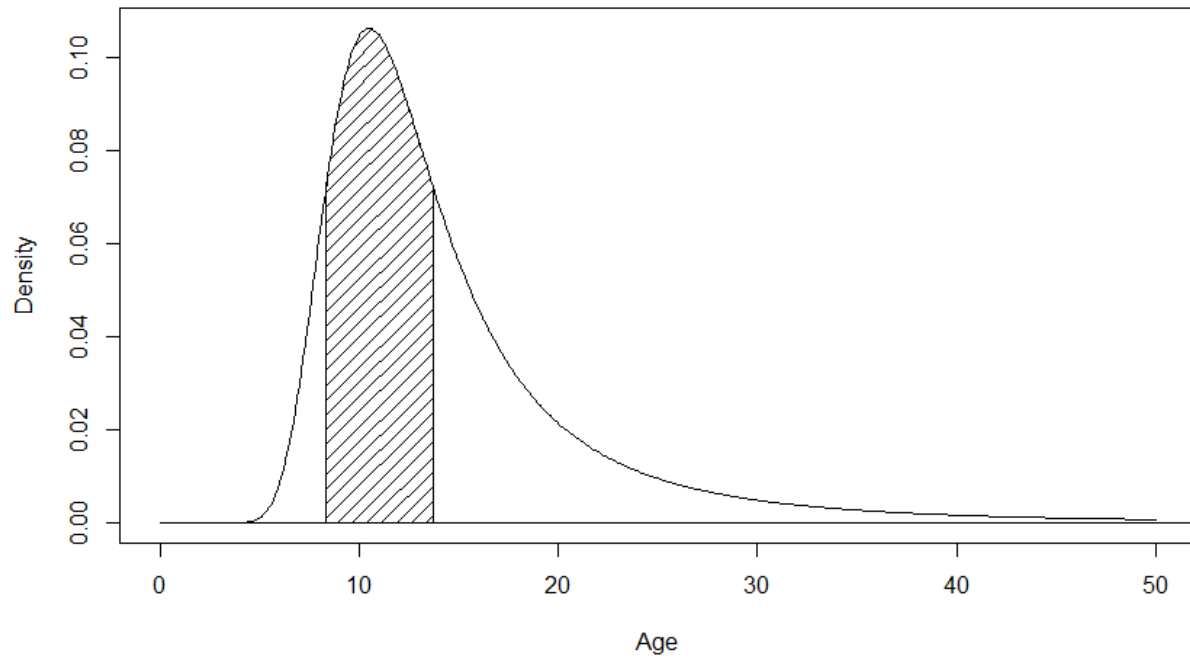
HPD female lower molar 1 stage 12 (Rc), US Violent Deaths 2010 prior



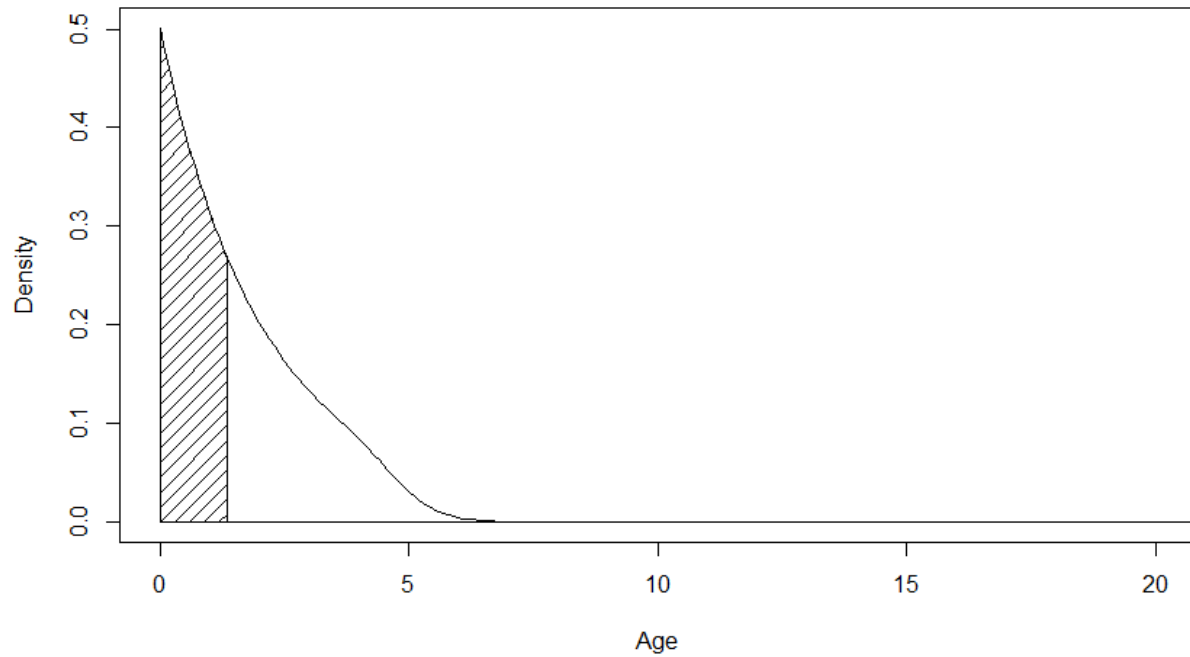
HPD female lower molar 1 stage 13 (A 1/2), US Violent Deaths 2010 prior



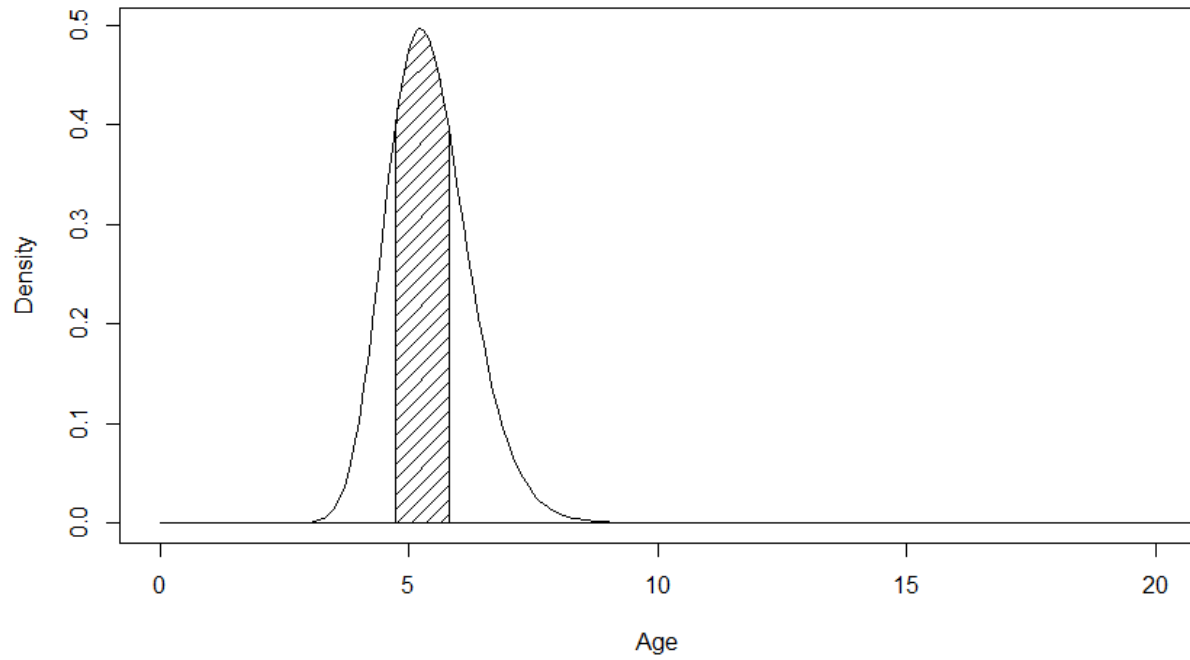
HPD female lower molar 1 stage 14 (Ac), US Violent Deaths 2010 prior



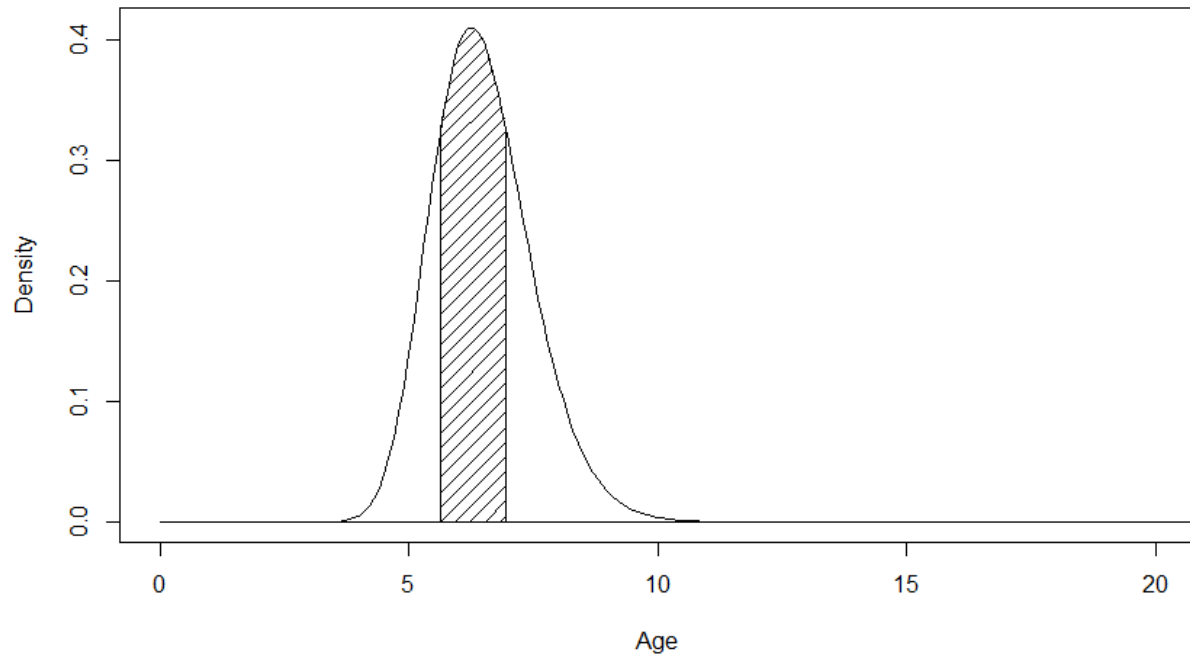
HPD female lower molar 2 stage 3 (Coc), US Violent Deaths 2010 prior



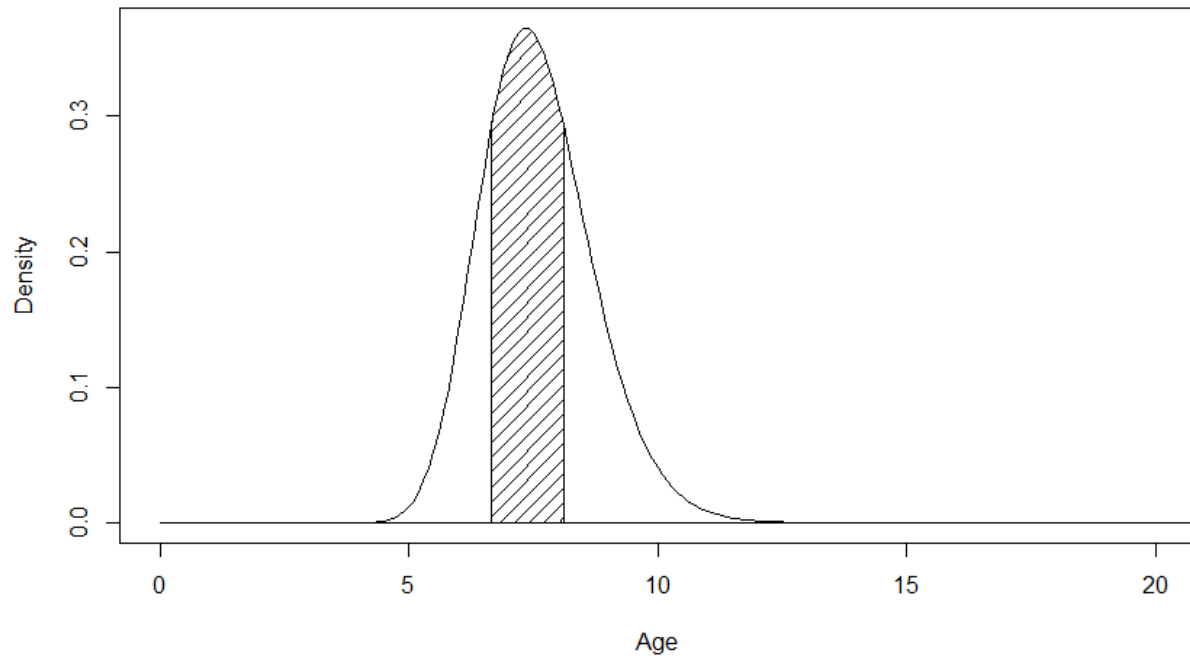
HPD female lower molar 2 stage 5 (Cr 3/4), US Violent Deaths 2010 prior



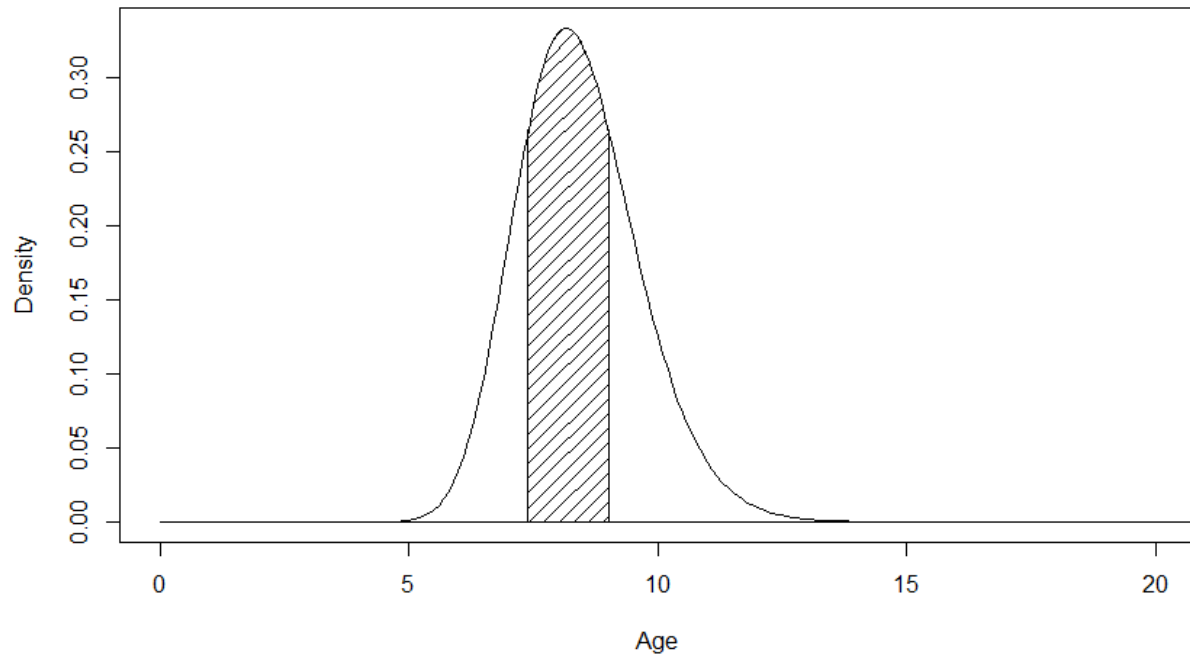
HPD female lower molar 2 stage 6 (Crc), US Violent Deaths 2010 prior



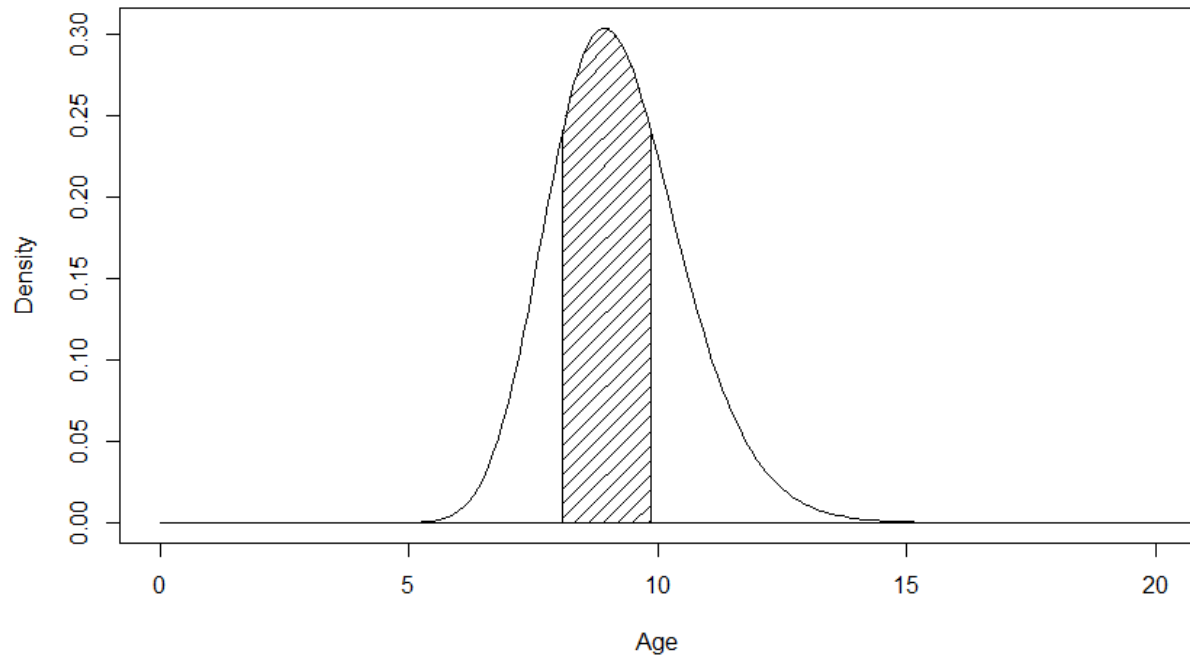
HPD female lower molar 2 stage 7 (Ri), US Violent Deaths 2010 prior



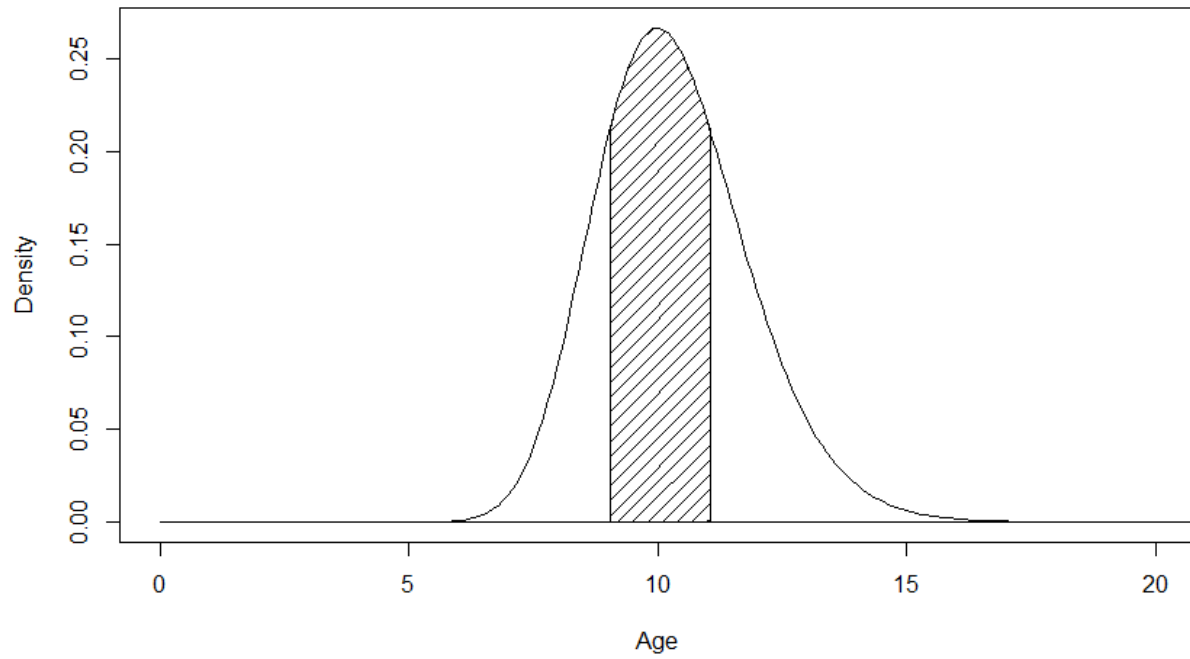
HPD female lower molar 2 stage 8 (C1i), US Violent Deaths 2010 prior



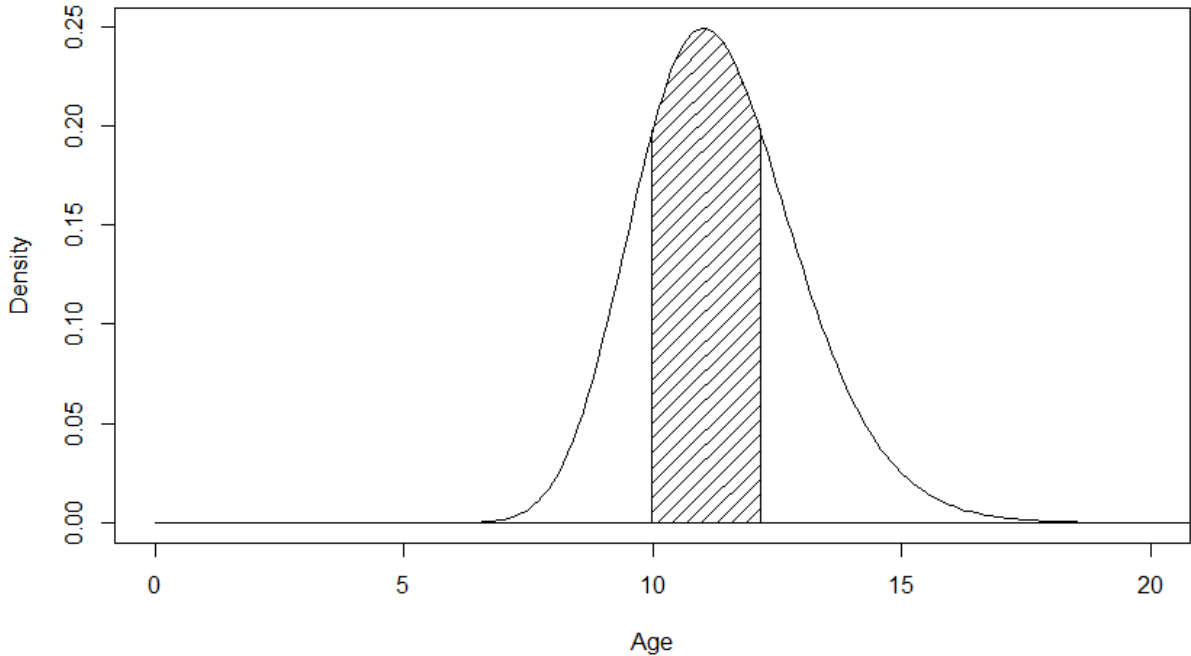
HPD female lower molar 2 stage 9 (R 1/4), US Violent Deaths 2010 prior



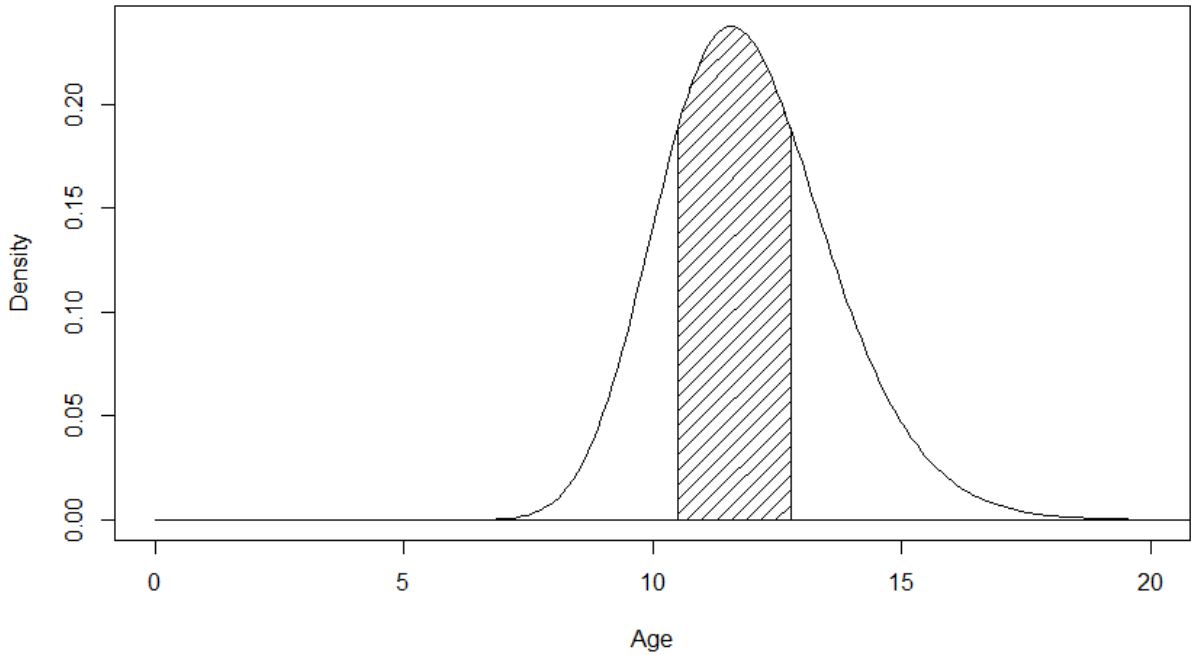
HPD female lower molar 2 stage 10 (R 1/2), US Violent Deaths 2010 prior



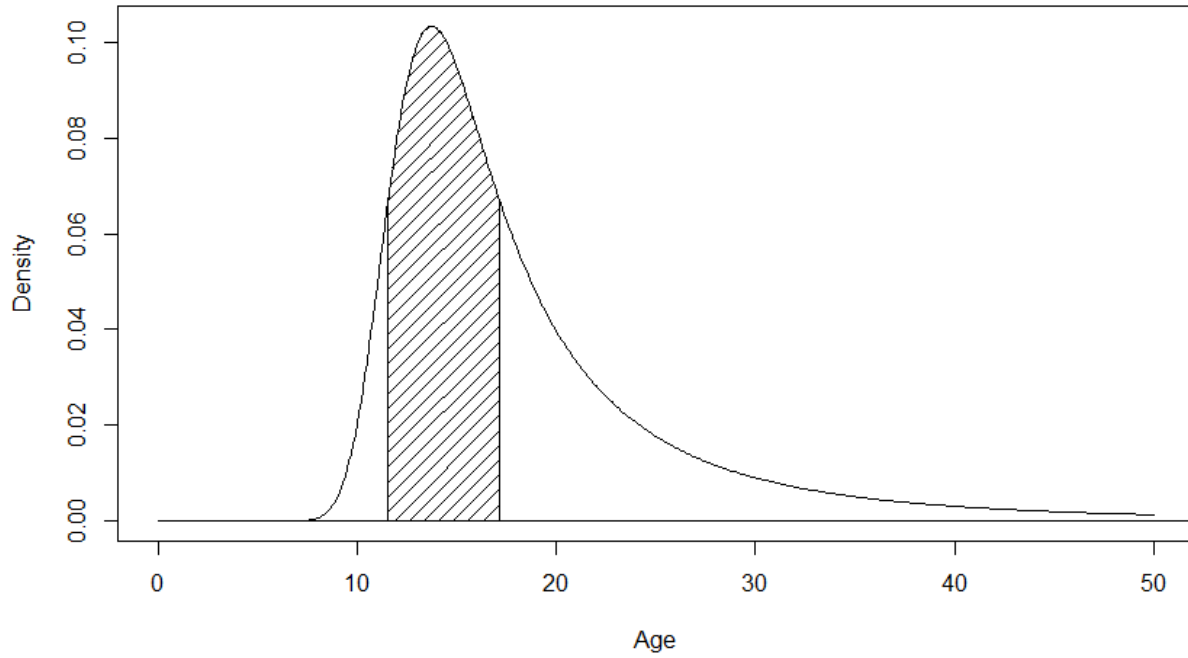
HPD female lower molar 2 stage 11 (R 3/4), US Violent Deaths 2010 prior



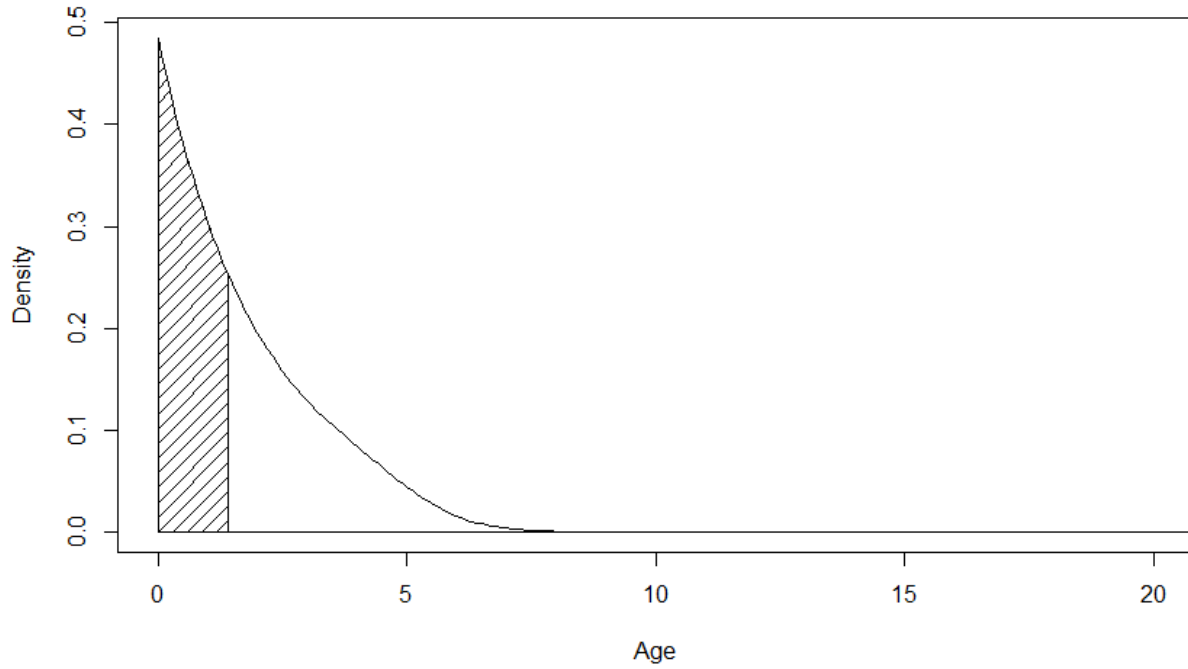
HPD female lower molar 2 stage 12 (Rc), US Violent Deaths 2010 prior



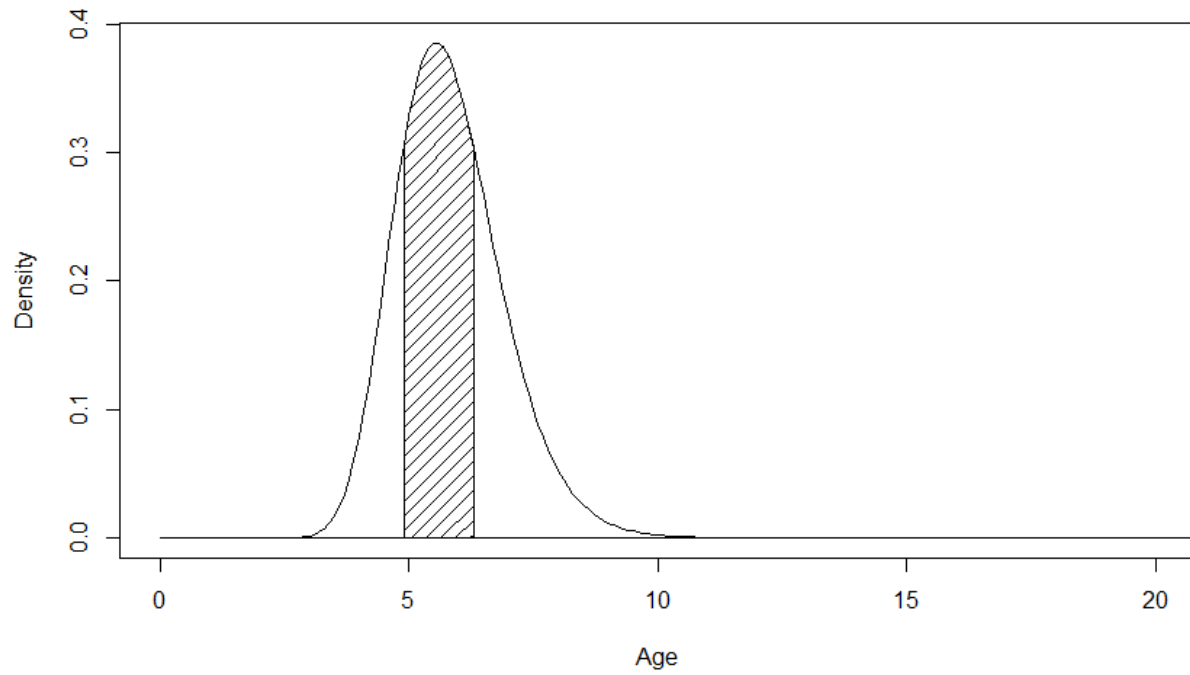
HPD female lower molar 2 stage 13 (A 1/2), US Violent Deaths 2010 prior



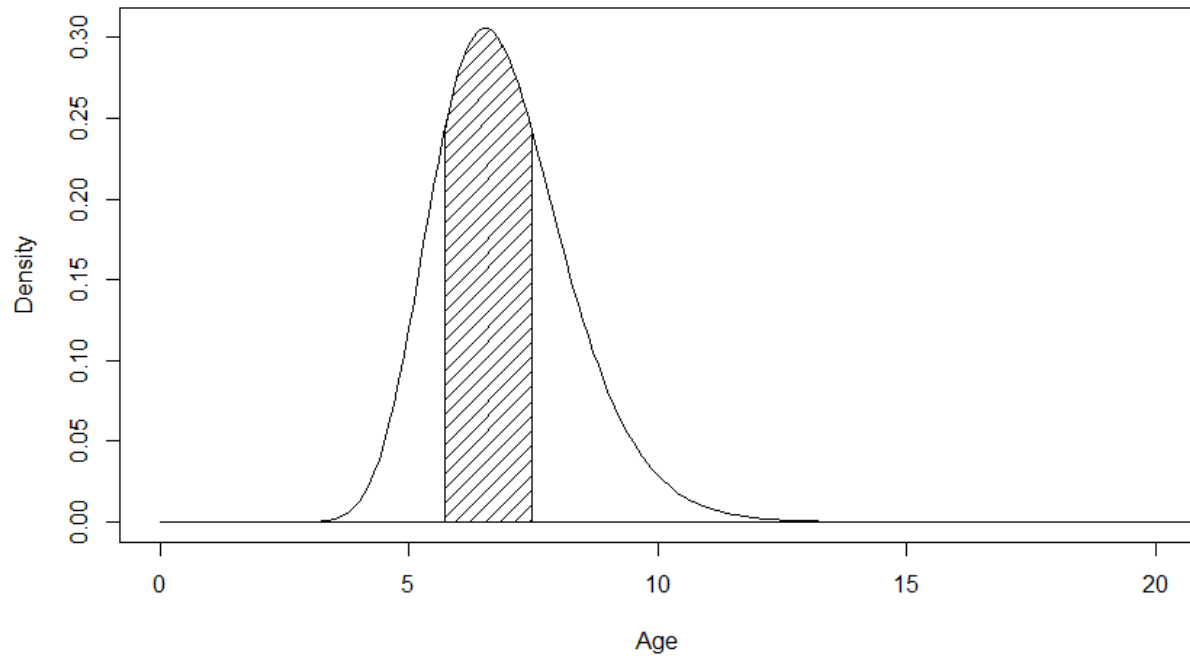
HPD female lower premolar 2 stage 5 (Cr 3/4), US Violent Deaths 2010 prior



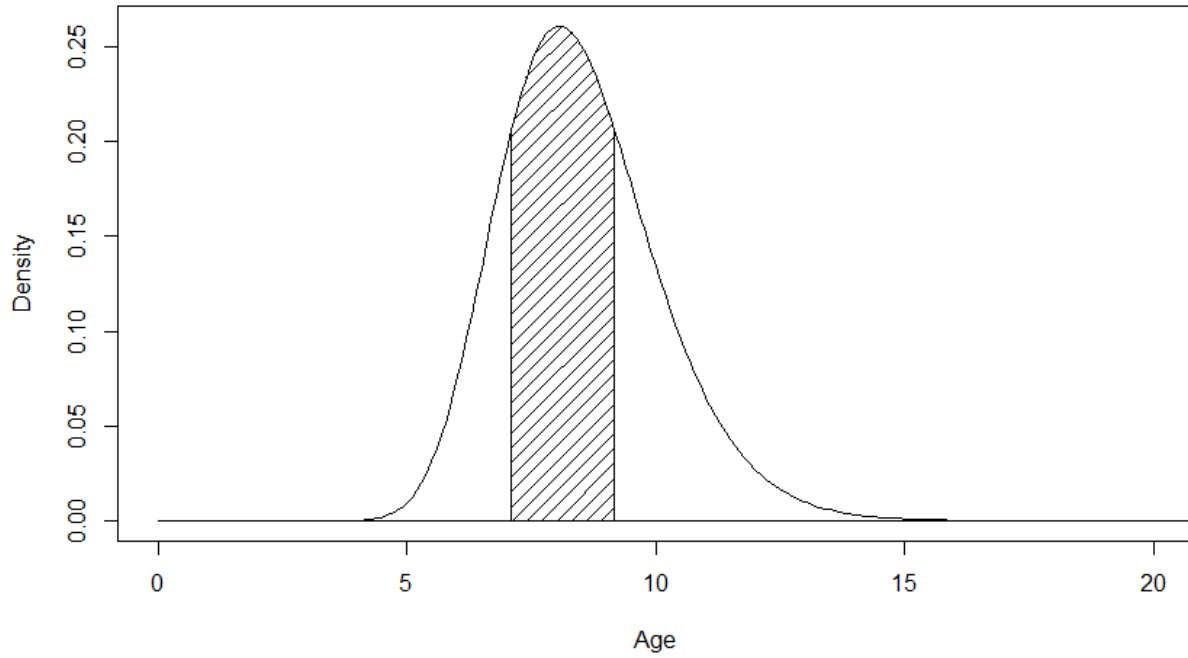
HPD female lower premolar 2 stage 6 (Crc), US Violent Deaths 2010 prior



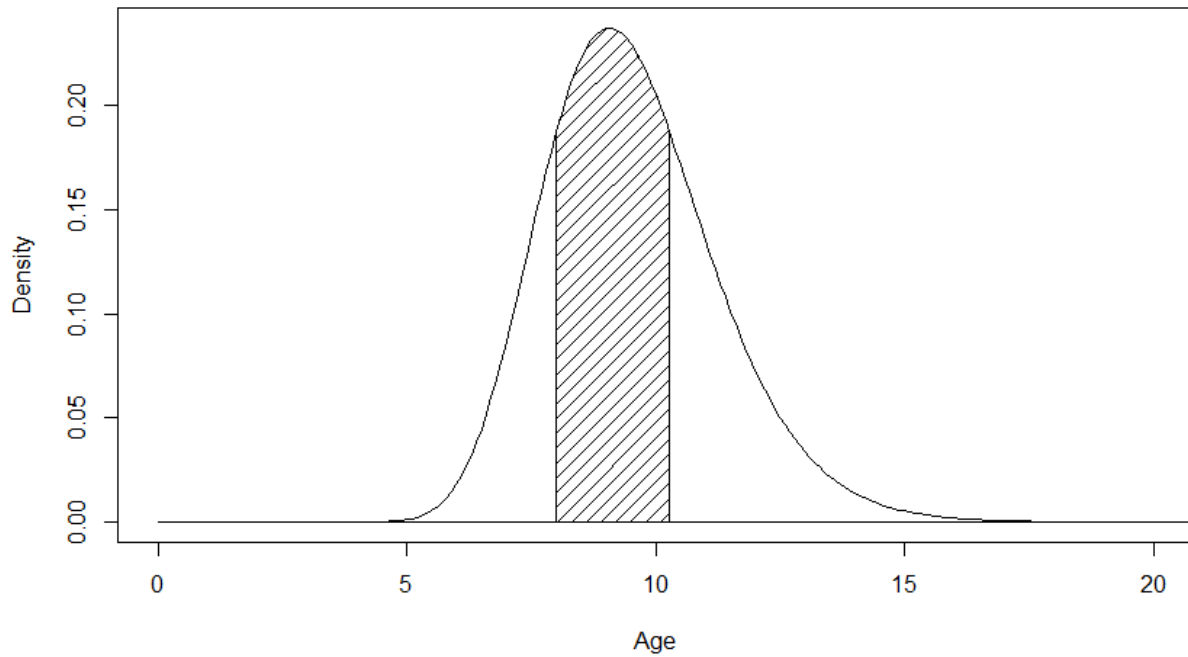
HPD female lower premolar 2 stage 7 (Ri), US Violent Deaths 2010 prior



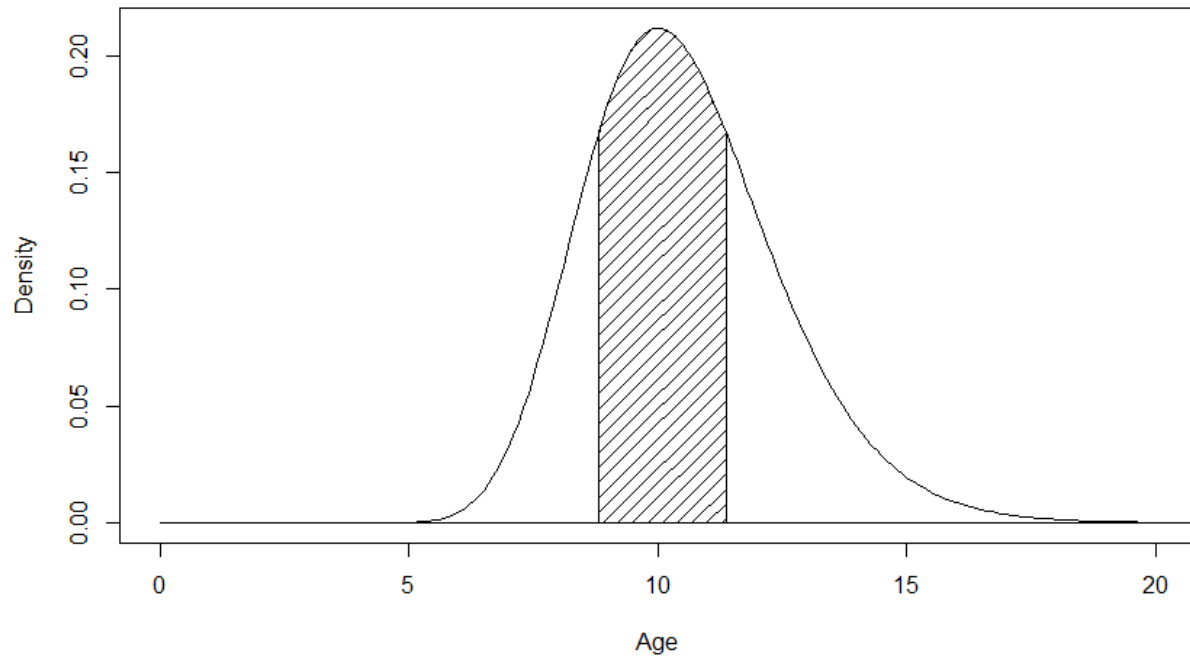
HPD female lower premolar 2 stage 9 (R 1/4), US Violent Deaths 2010 prior



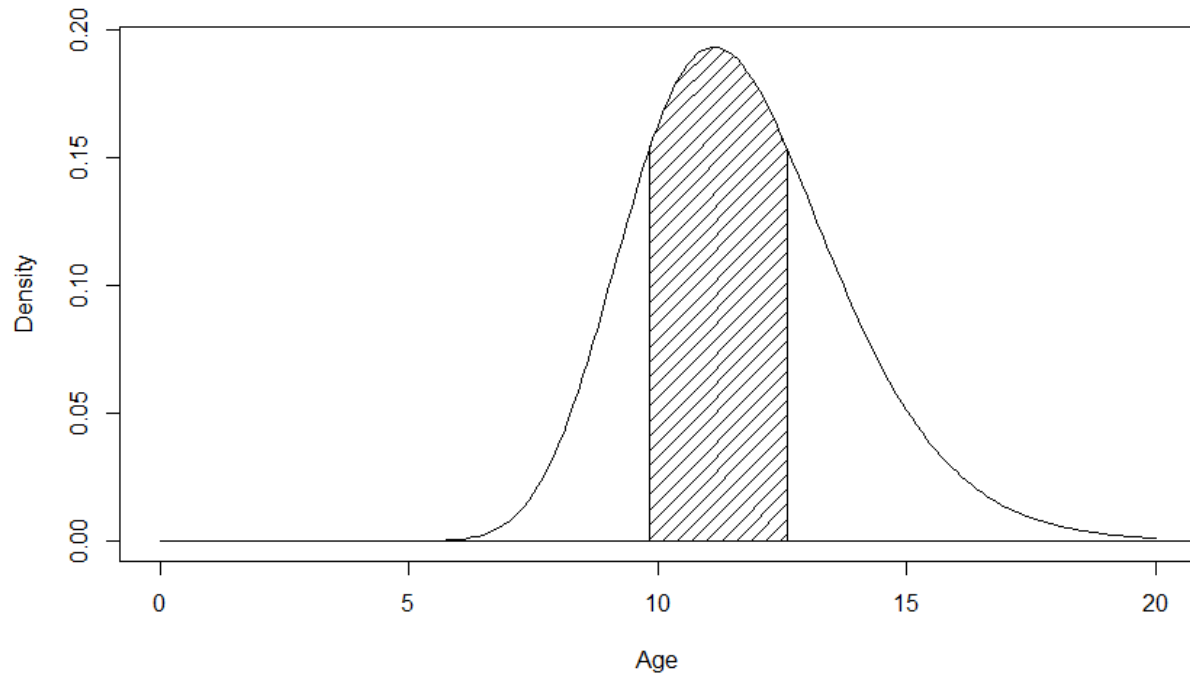
HPD female lower premolar 2 stage 10 (R 1/2), US Violent Deaths 2010 prior



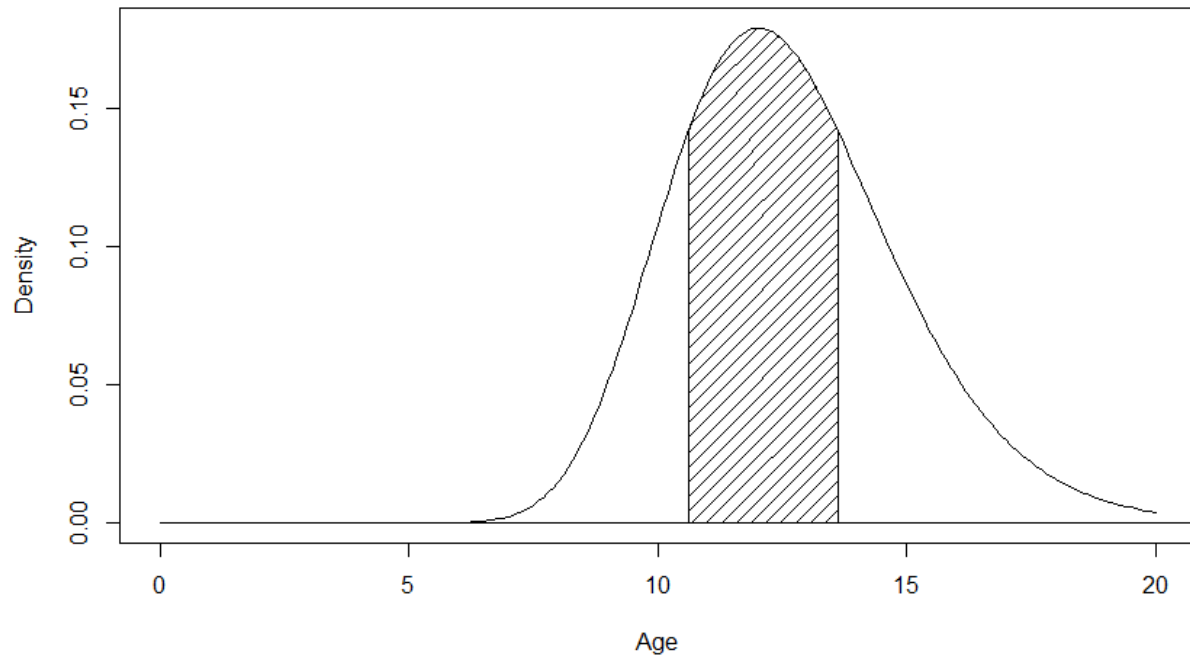
HPD female lower premolar 2 stage 11 (R 3/4), US Violent Deaths 2010 prior



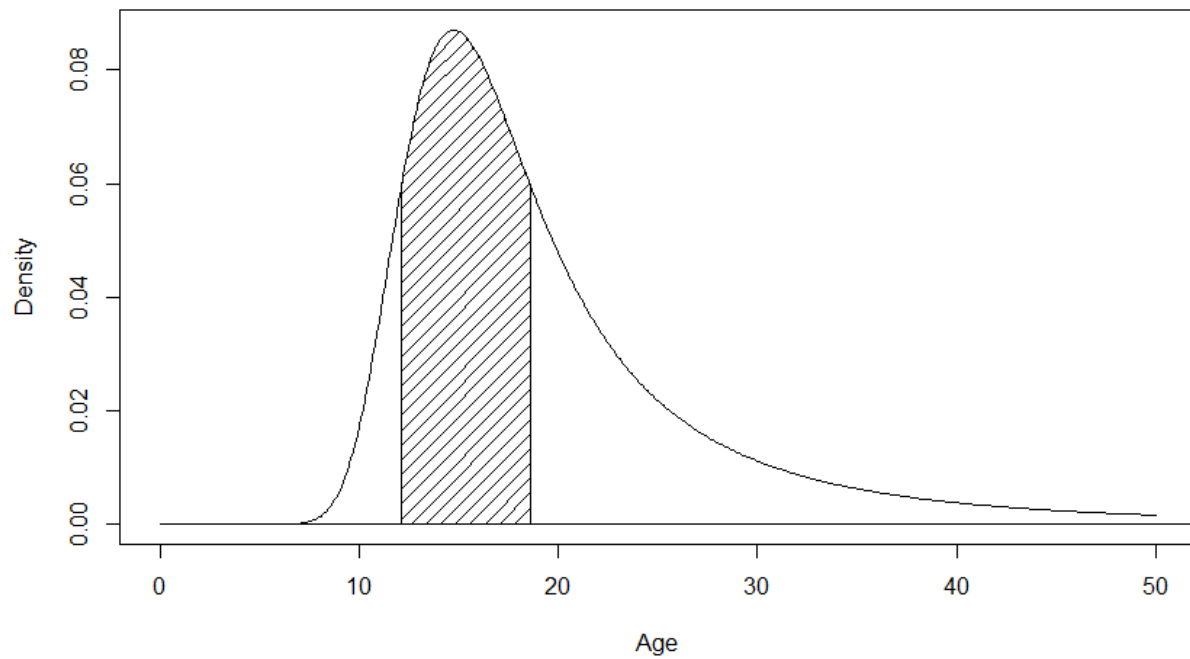
HPD female lower premolar 2 stage 12 (Rc), US Violent Deaths 2010 prior



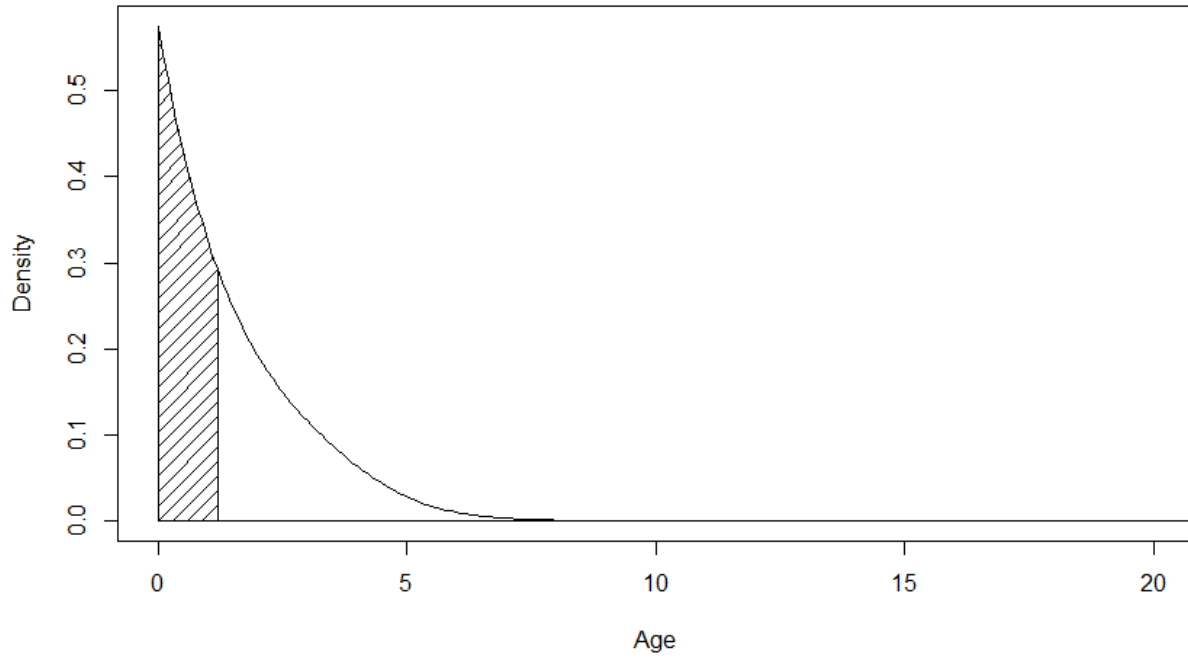
HPD female lower premolar 2 stage 13 (A 1/2), US Violent Deaths 2010 prior



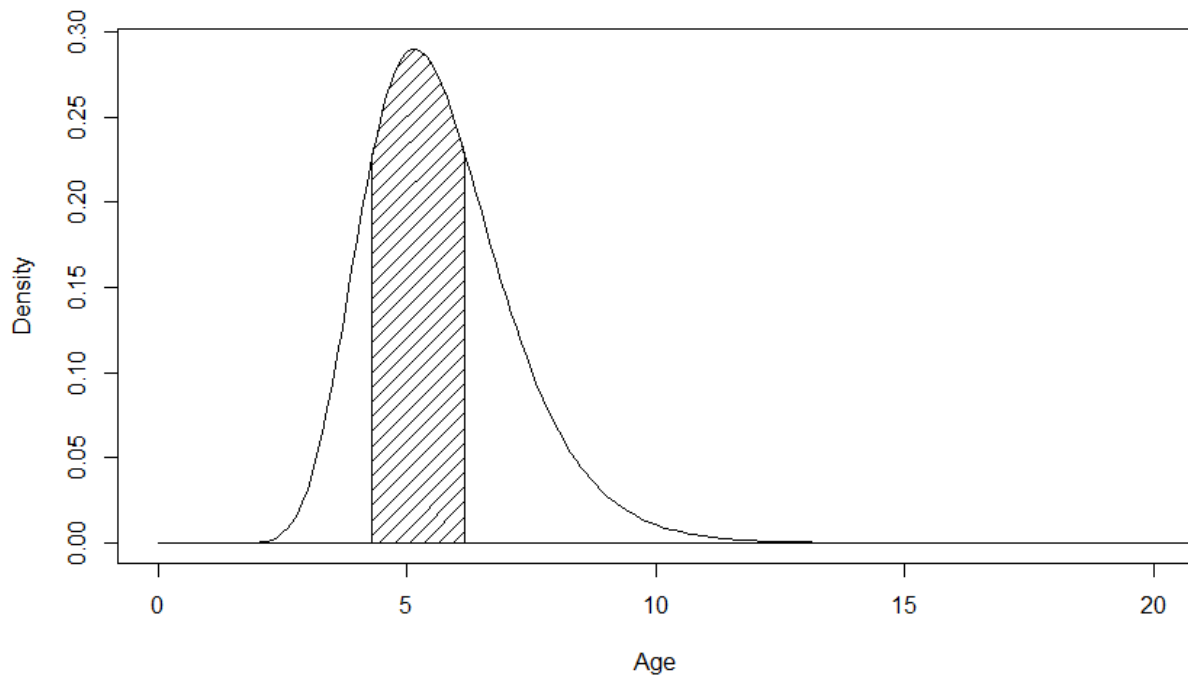
HPD female lower premolar 2 stage 14 (Ac), US Violent Deaths 2010 prior



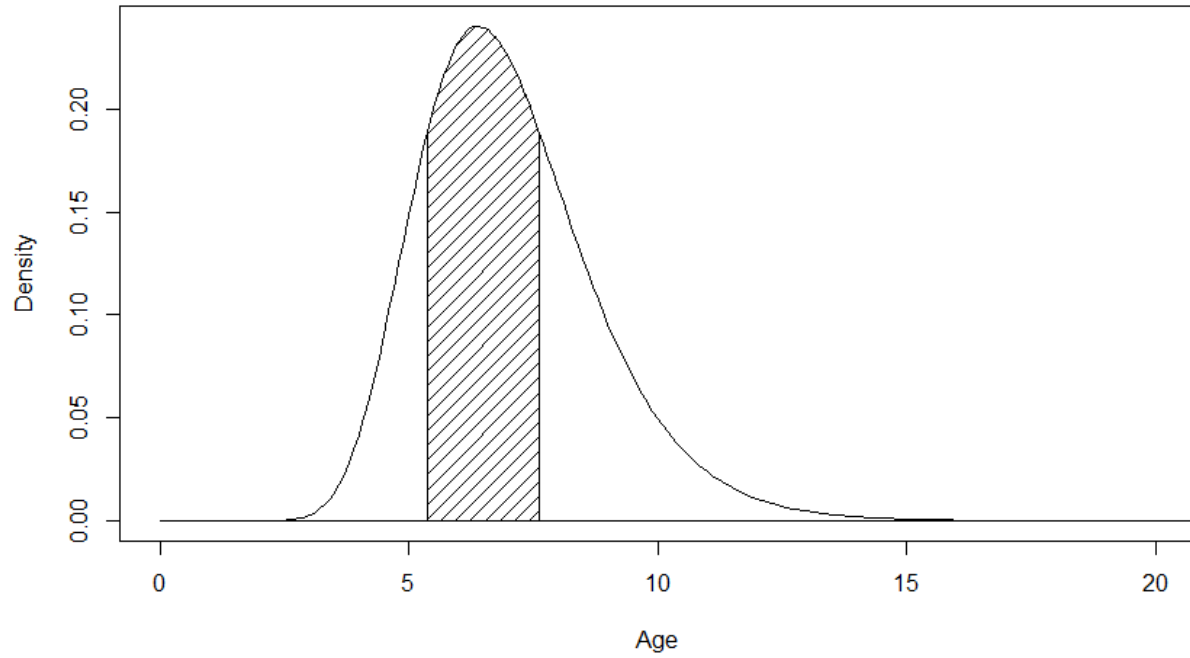
HPD female upper incisor 1 stage 10 (R 1/2), US Deaths 2008 prior



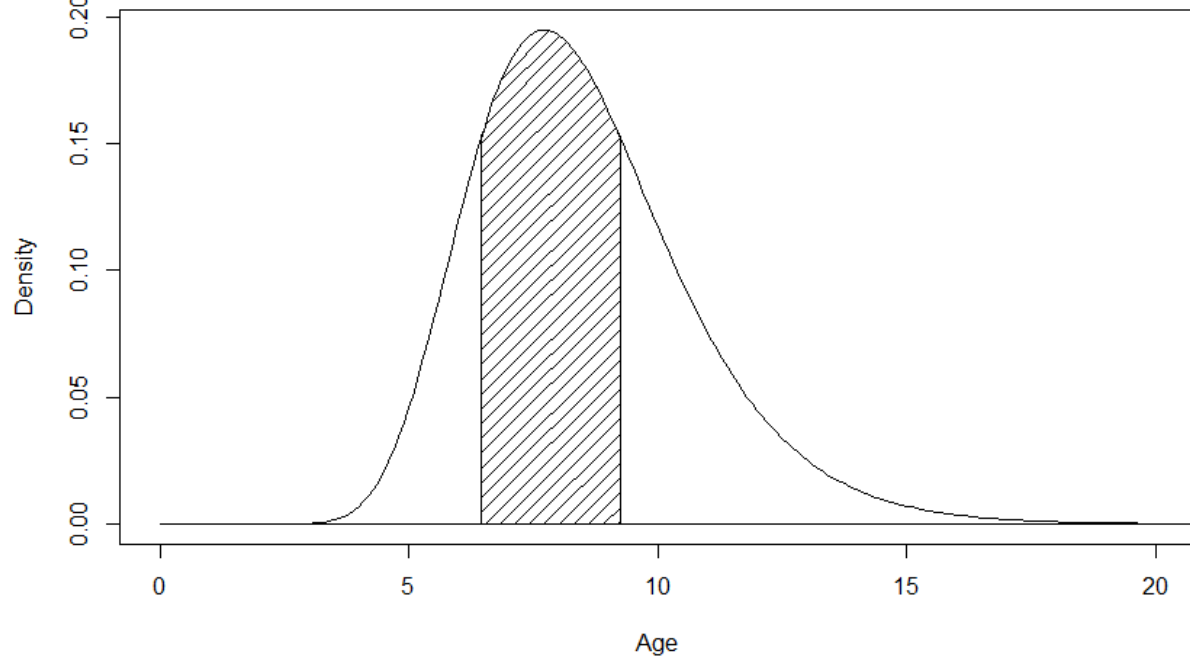
HPD female upper incisor 1 stage 11 (R 3/4), US Deaths 2008 prior



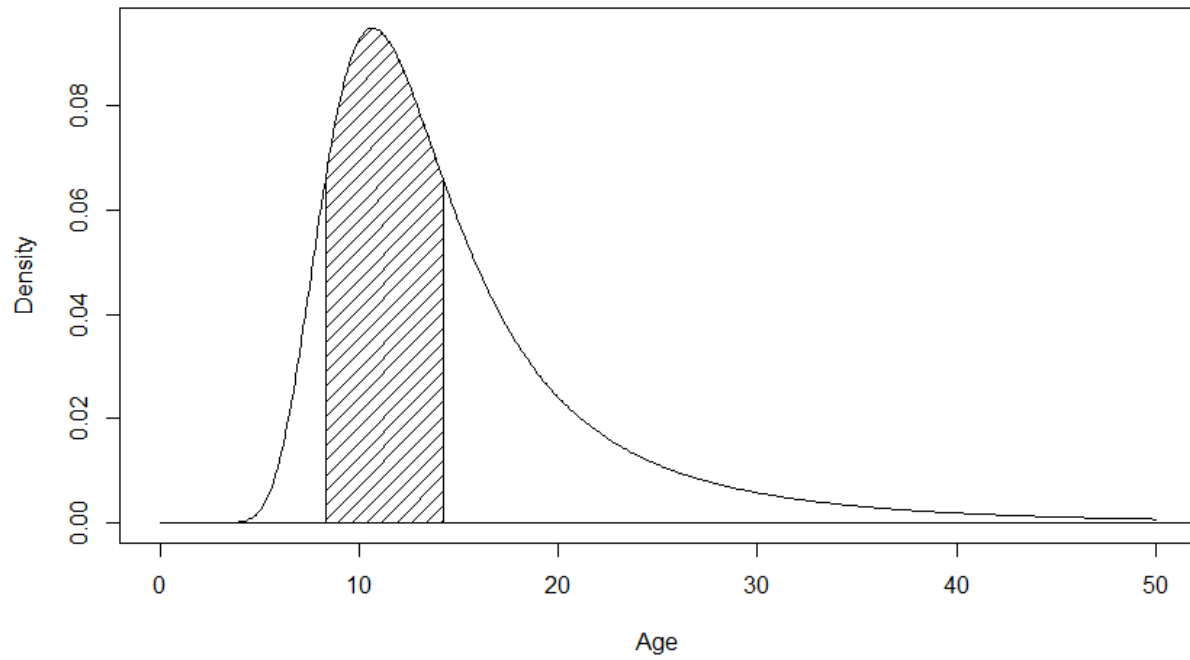
HPD female upper incisor 1 stage 12 (Rc), US Deaths 2008 prior



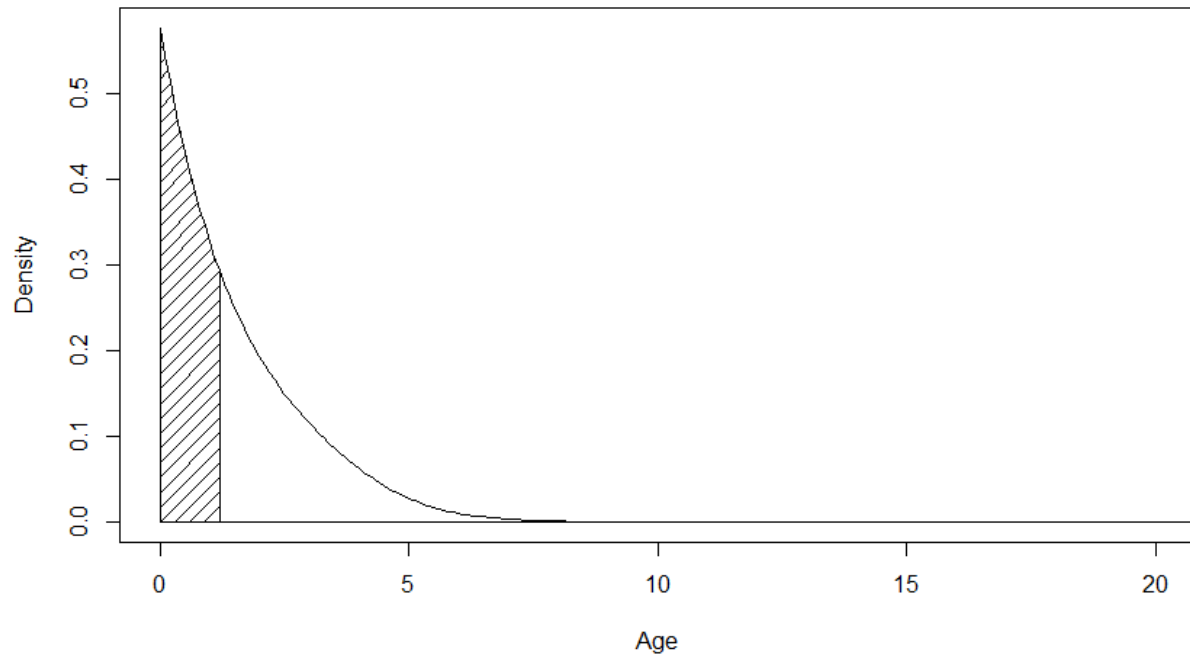
HPD female upper incisor 1 stage 13 (A 1/2), US Deaths 2008 prior



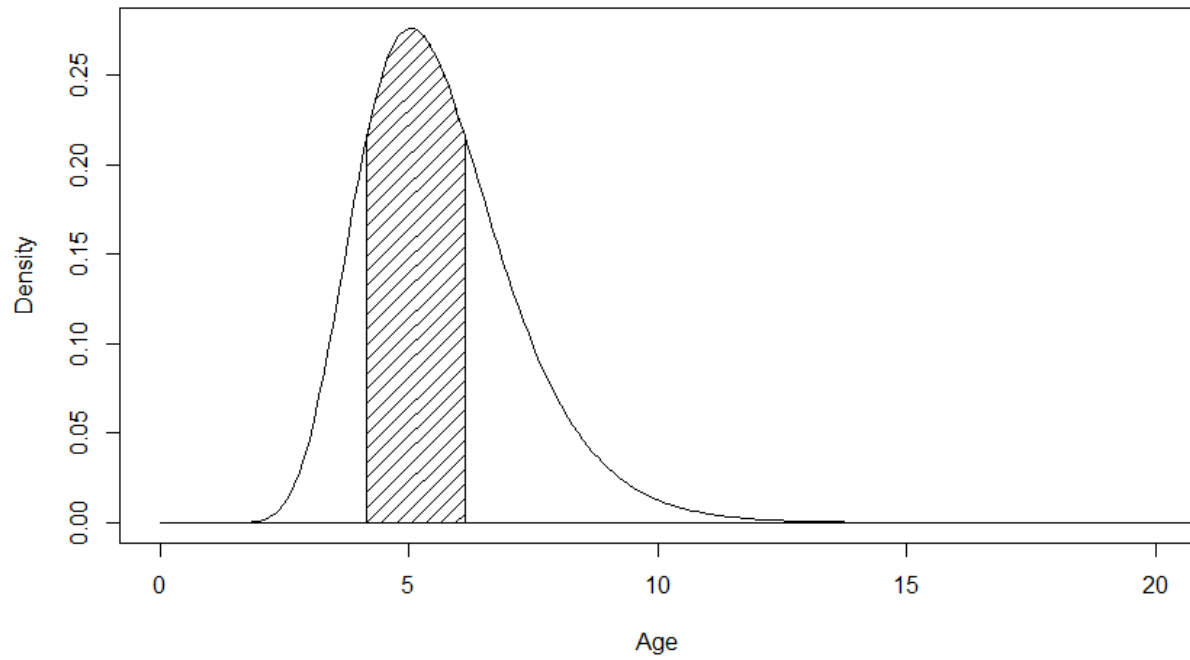
HPD female upper incisor 1 stage 14 (Ac), US Deaths 2008 prior



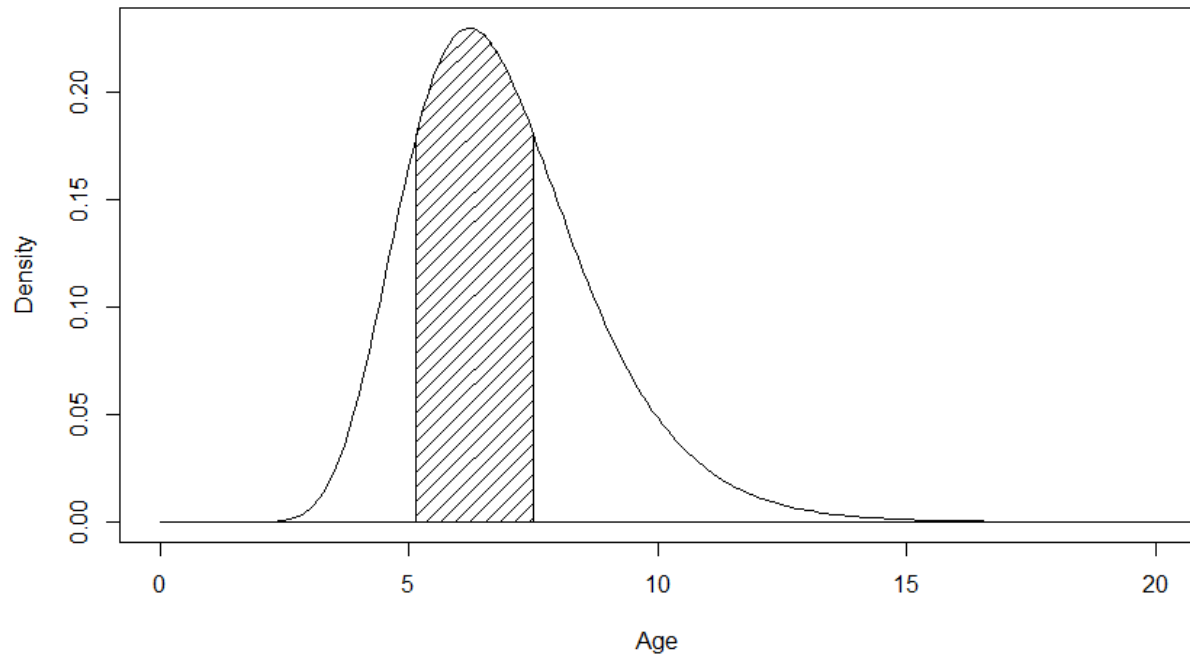
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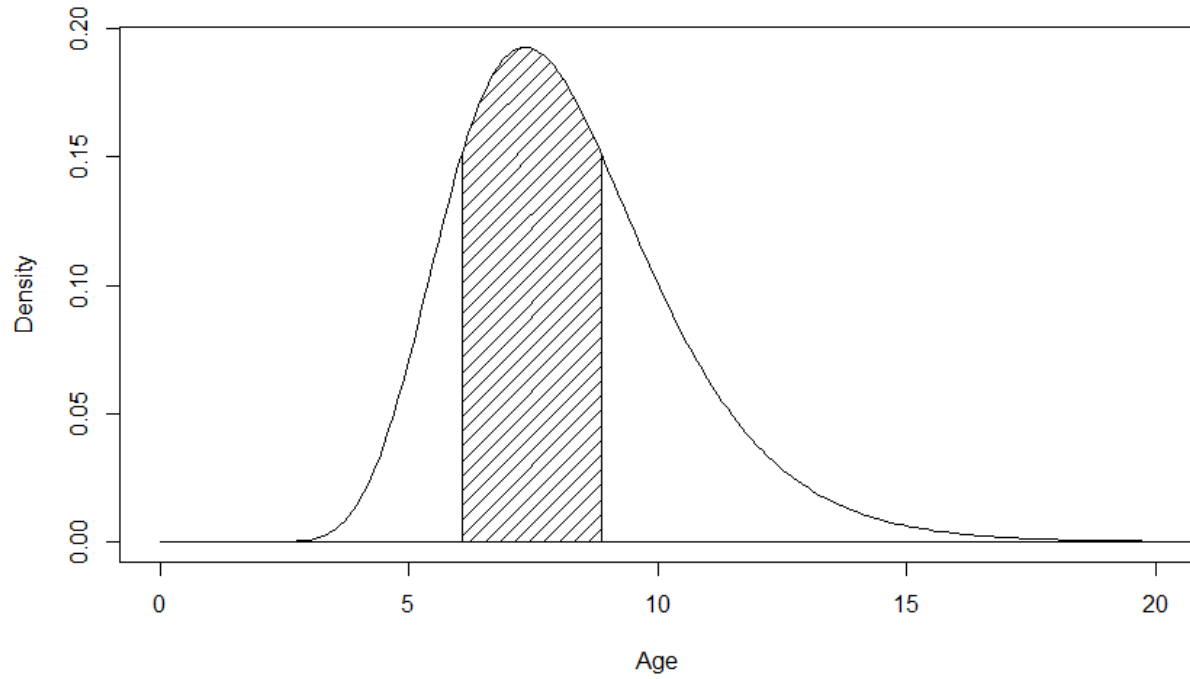
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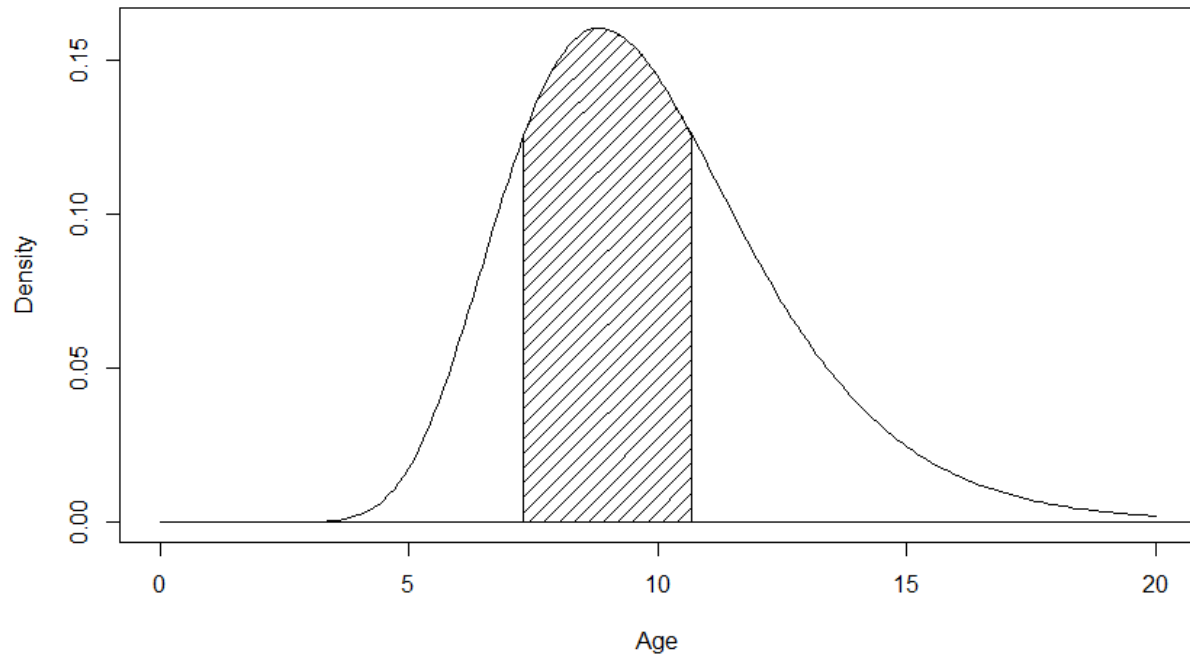
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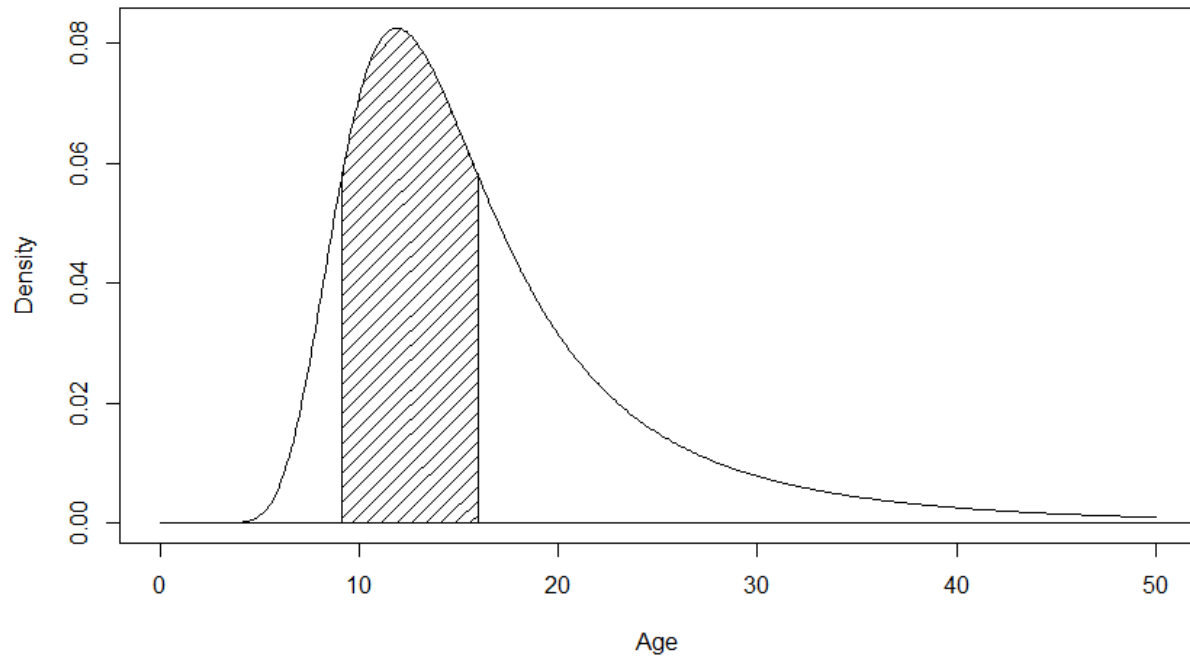
HPD female upper incisor 2 stage 12 (Rc), US Deaths 2008 prior



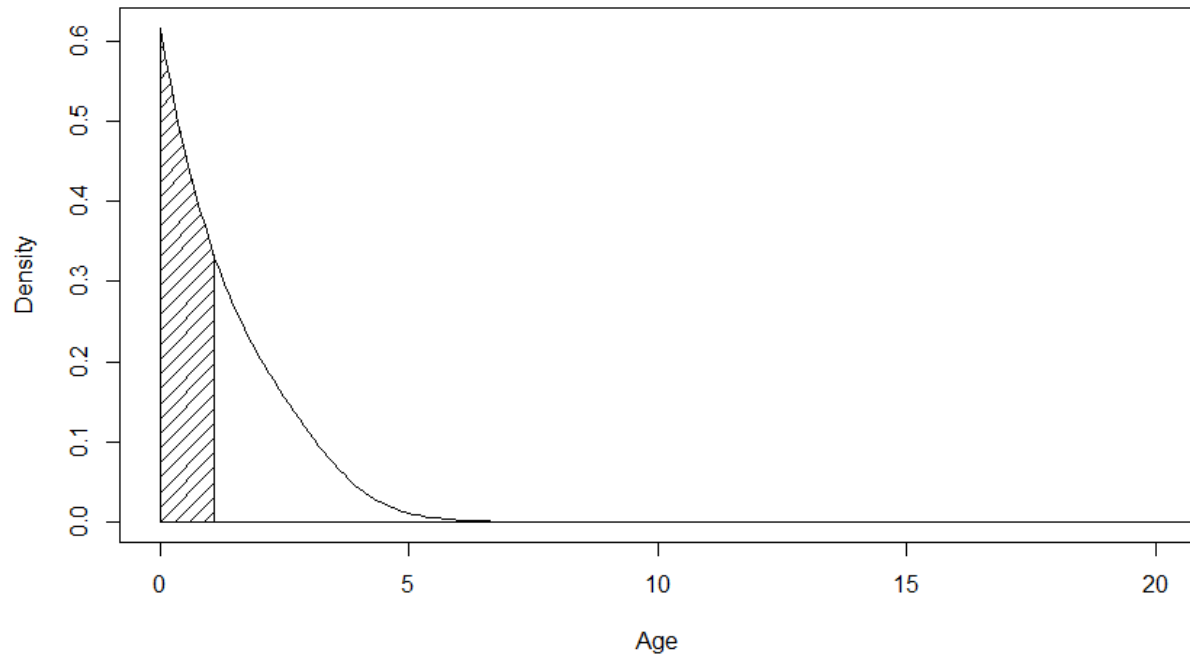
HPD female upper incisor 2 stage 13 (A 1/2), US Deaths 2008 prior



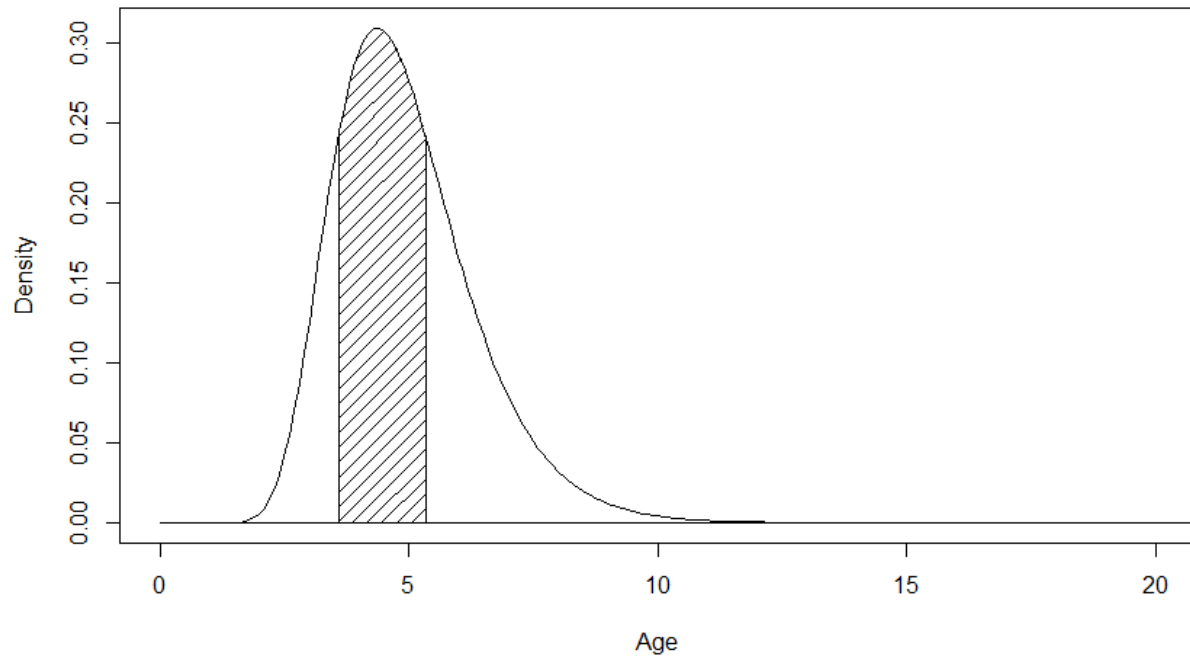
HPD female upper incisor 2 stage 14 (Ac), US Deaths 2008 prior



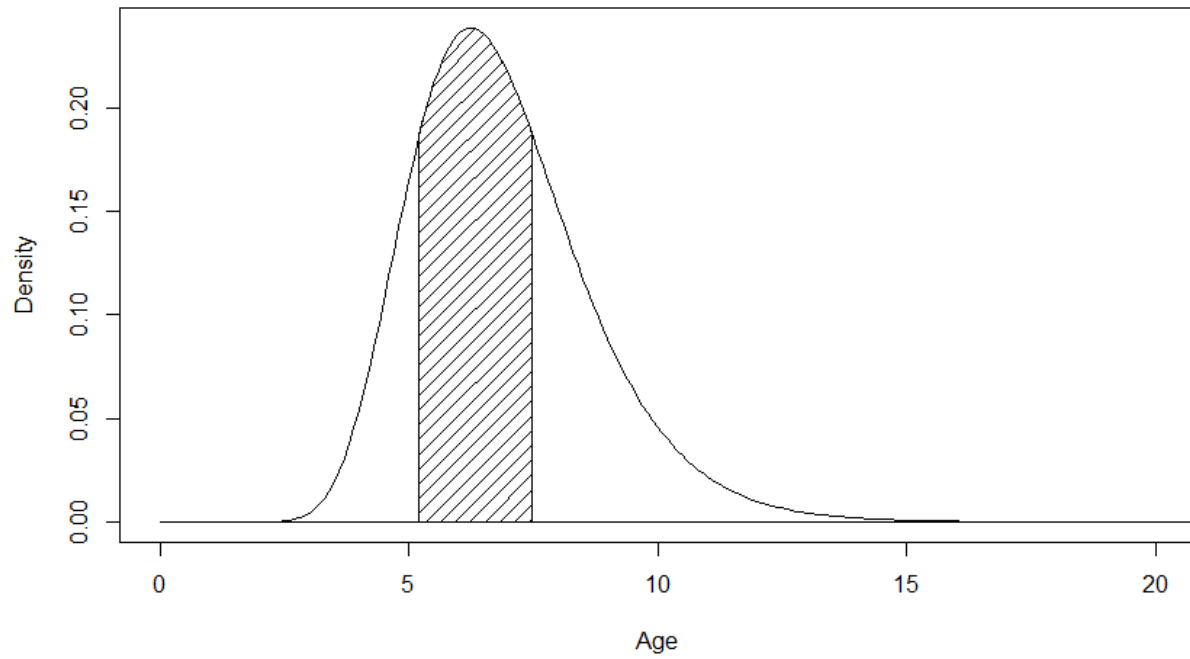
HPD female upper canine stage 7 (Ri), US Deaths 2008 prior



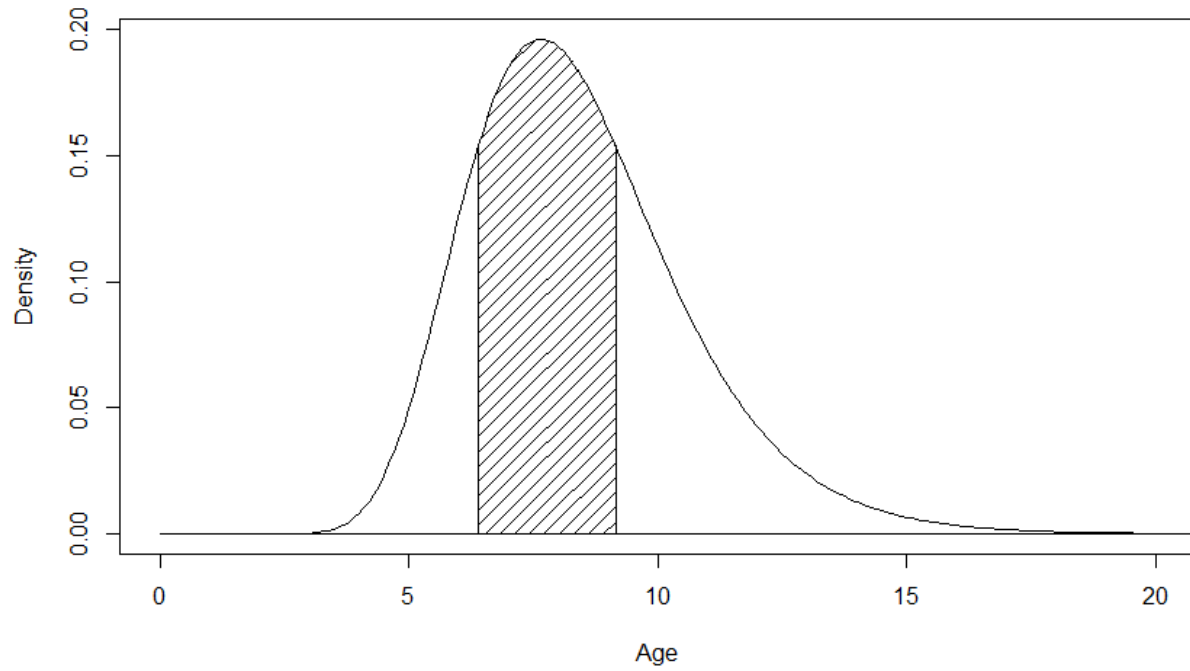
HPD female upper canine stage 9 (R 1/4), US Deaths 2008 prior



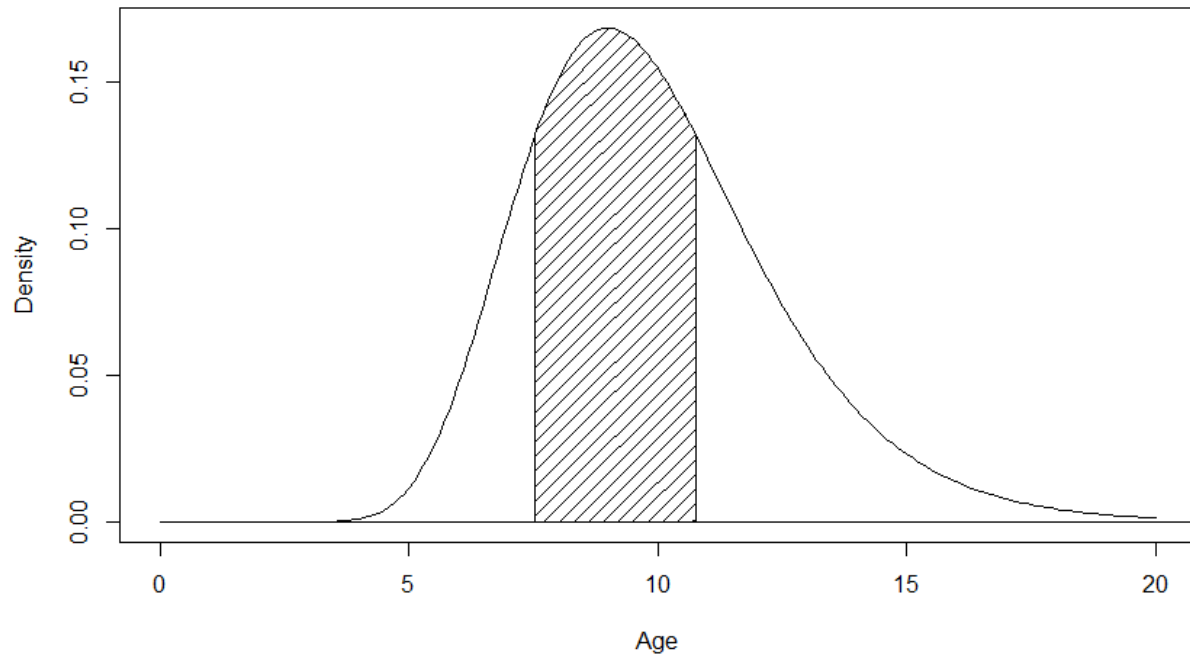
HPD female upper canine stage 10 (R 1/2), US Deaths 2008 prior



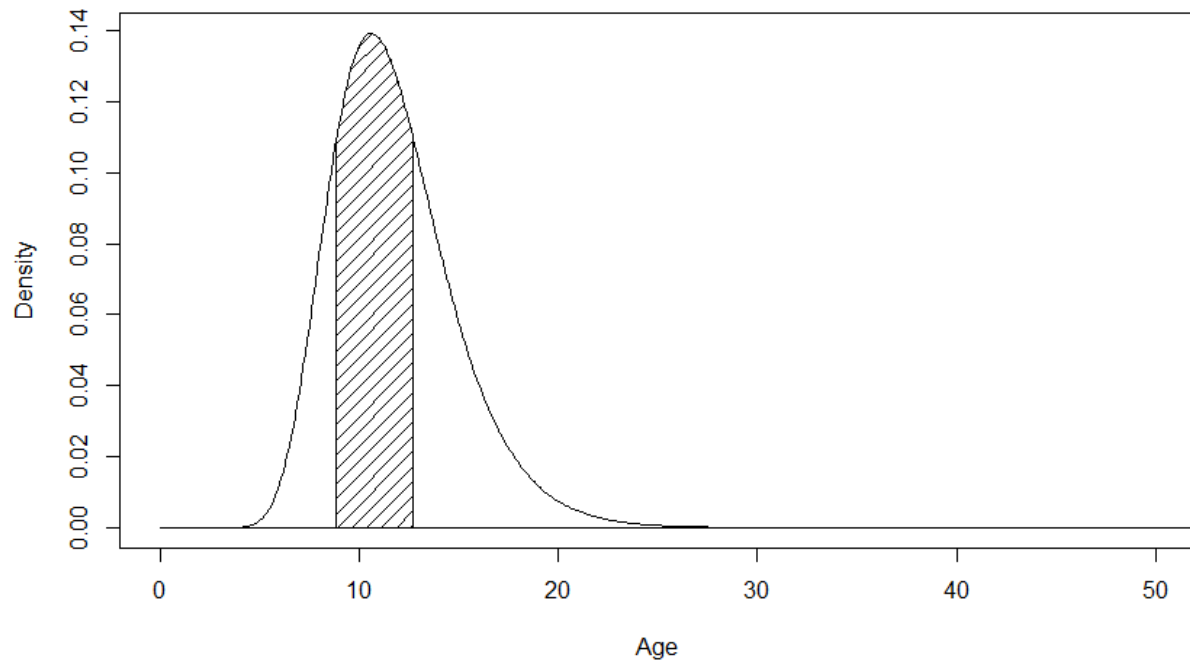
HPD female upper canine stage 11 (R 3/4), US Deaths 2008 prior



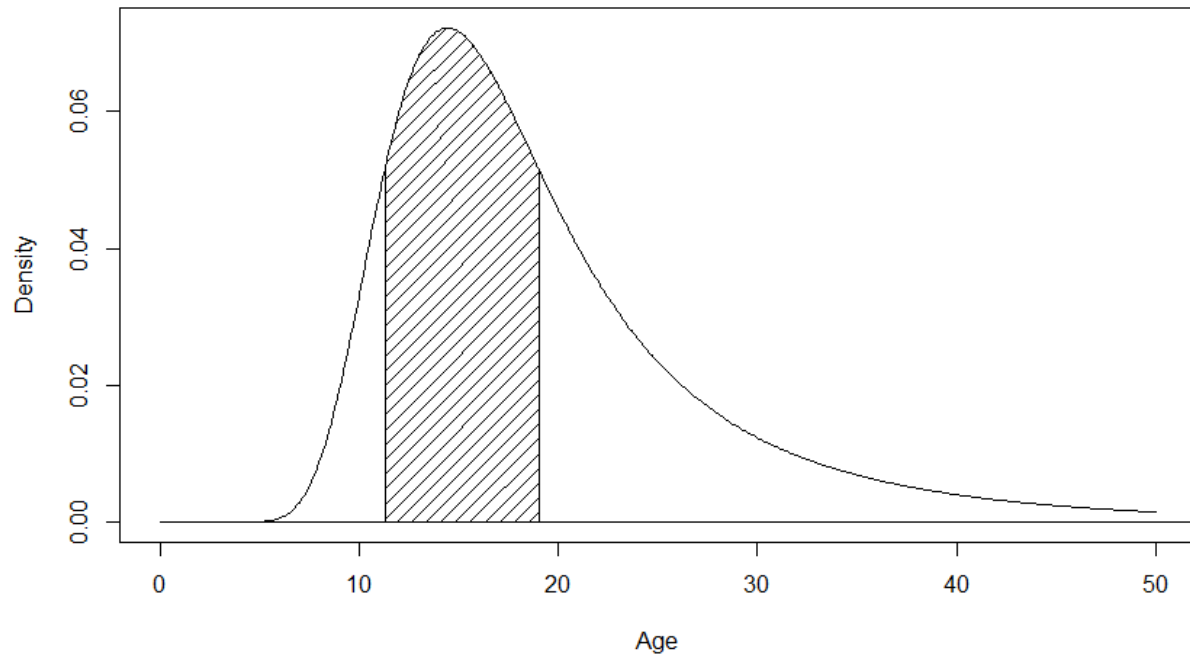
HPD female upper canine stage 12 (Rc), US Deaths 2008 prior



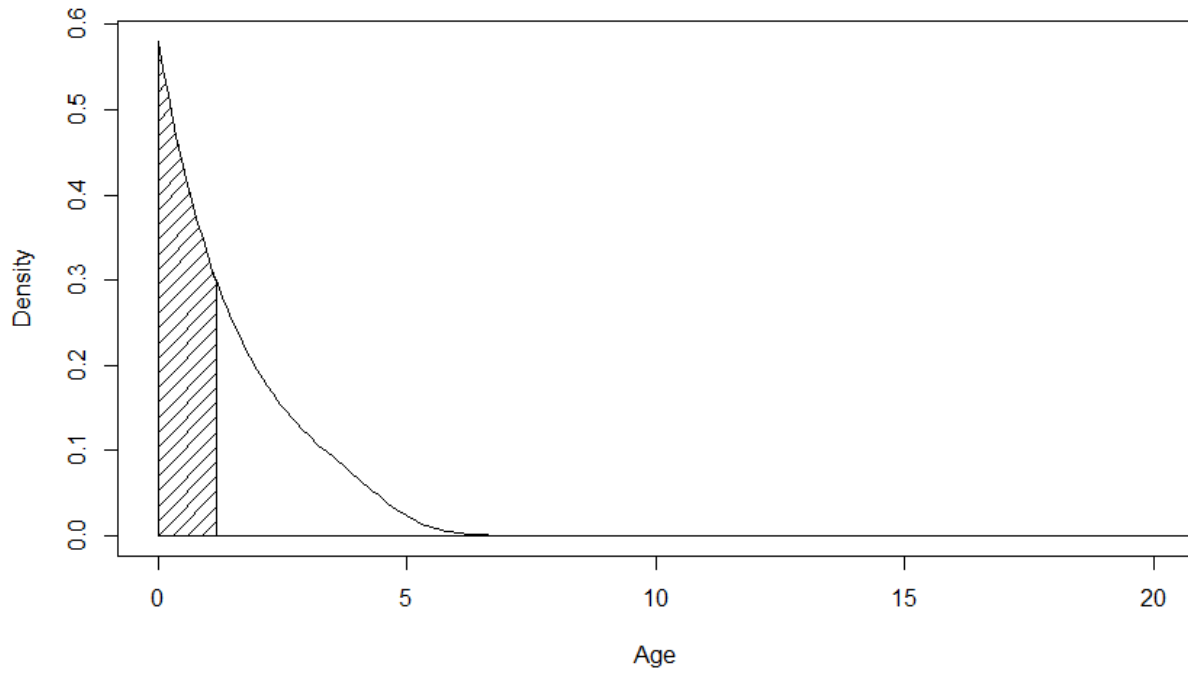
HPD female upper canine stage 13 (A 1/2), US Deaths 2008 prior



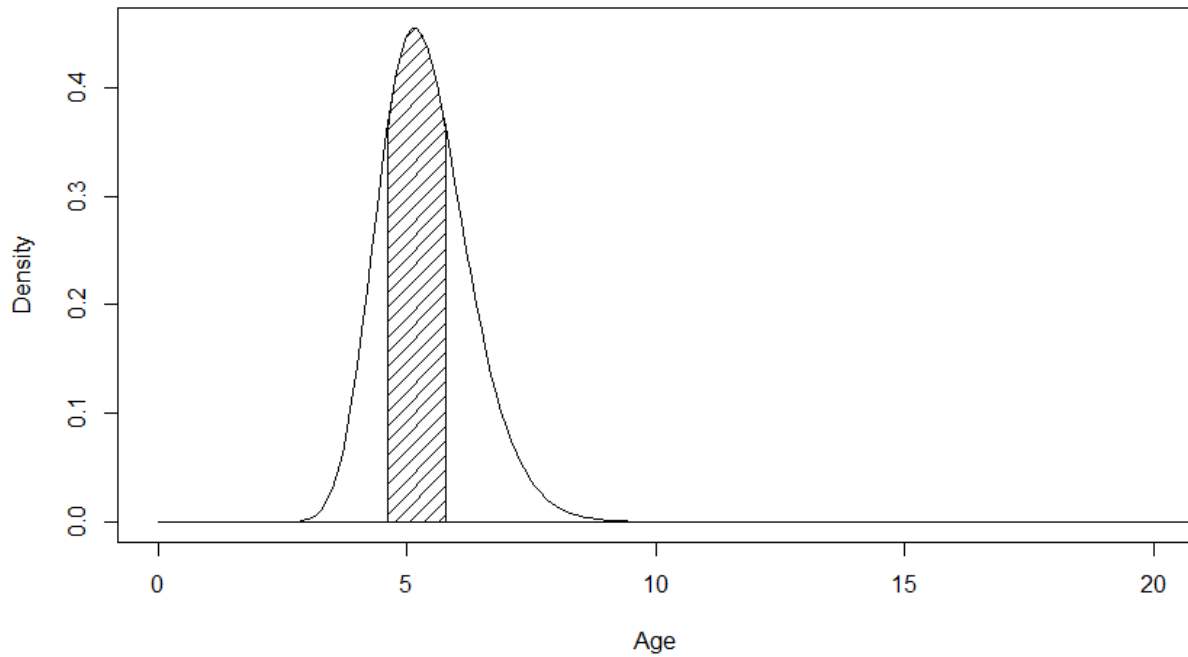
HPD female upper canine stage 14 (Ac), US Deaths 2008 prior



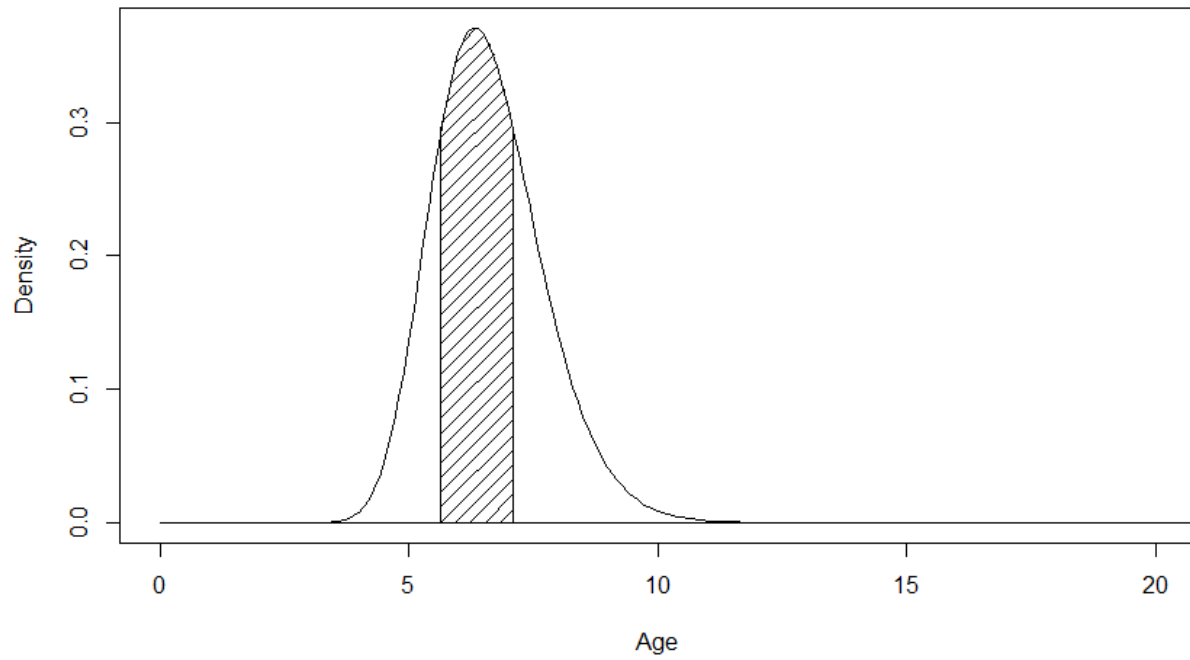
HPD female upper premolar 1 stage 5 (Cr 3/4), US Deaths 2008 prior



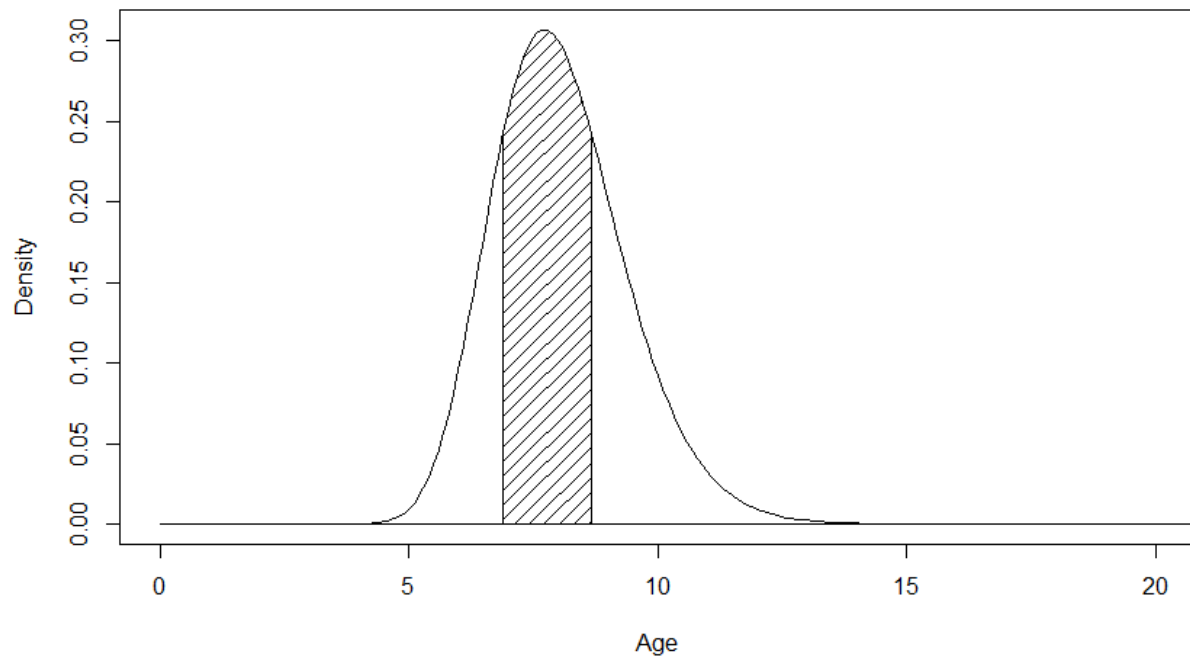
HPD female upper premolar 1 stage 6 (Crc), US Deaths 2008 prior



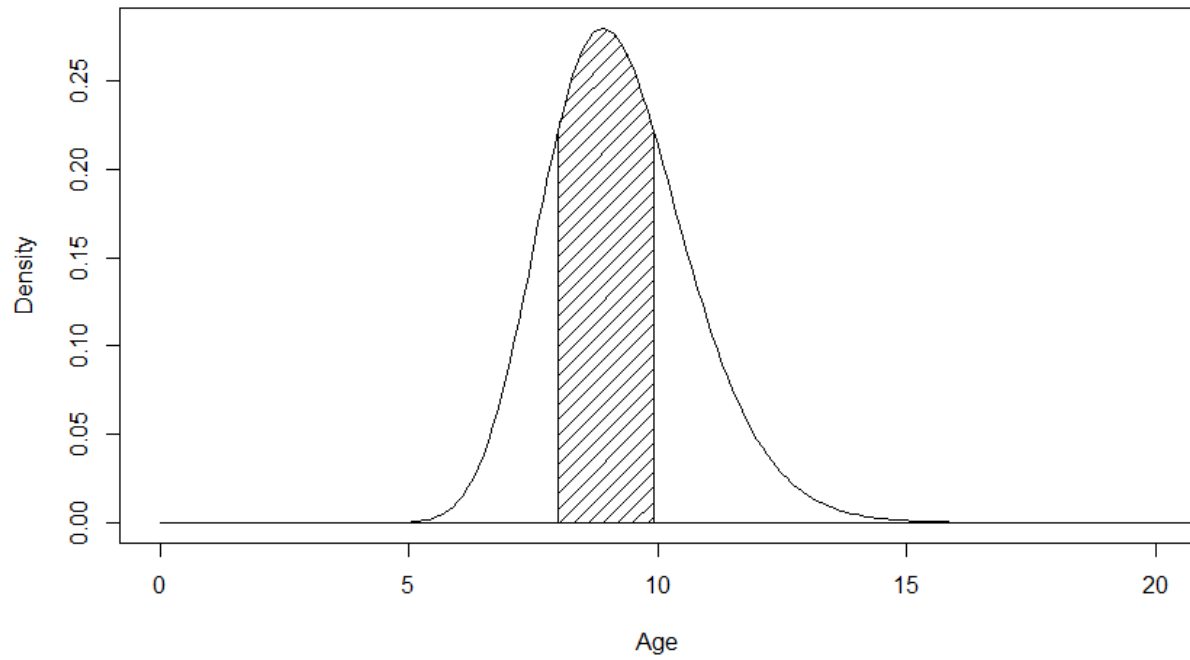
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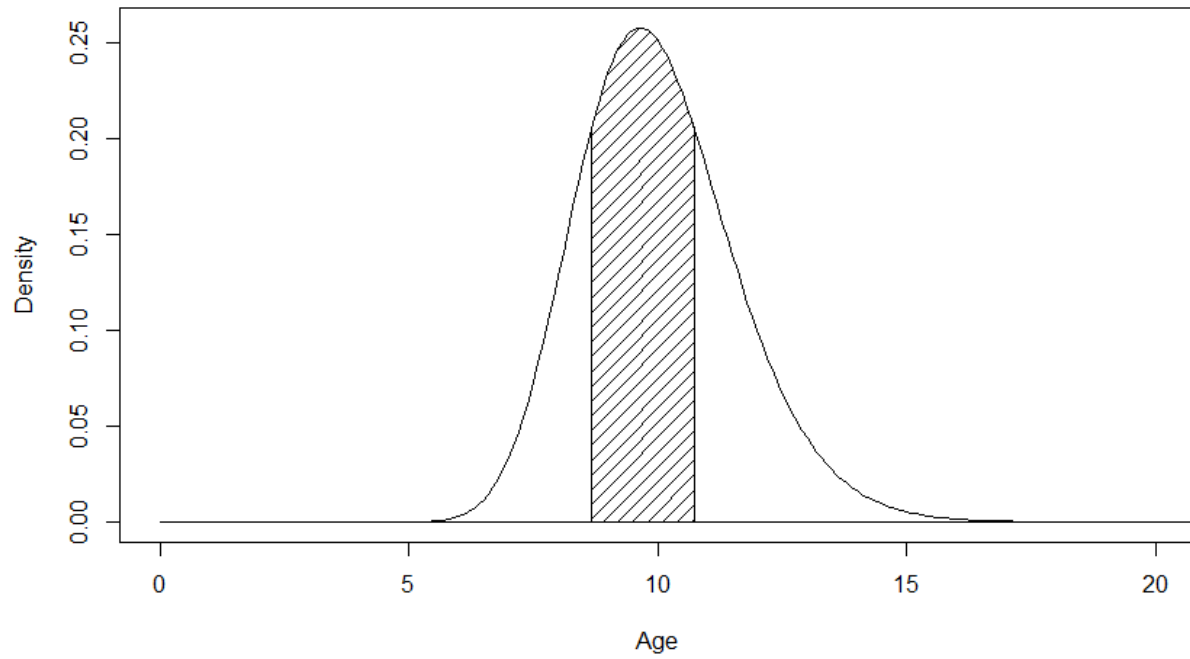
HPD female upper premolar 1 stage 9 (R 1/4), US Deaths 2008 prior



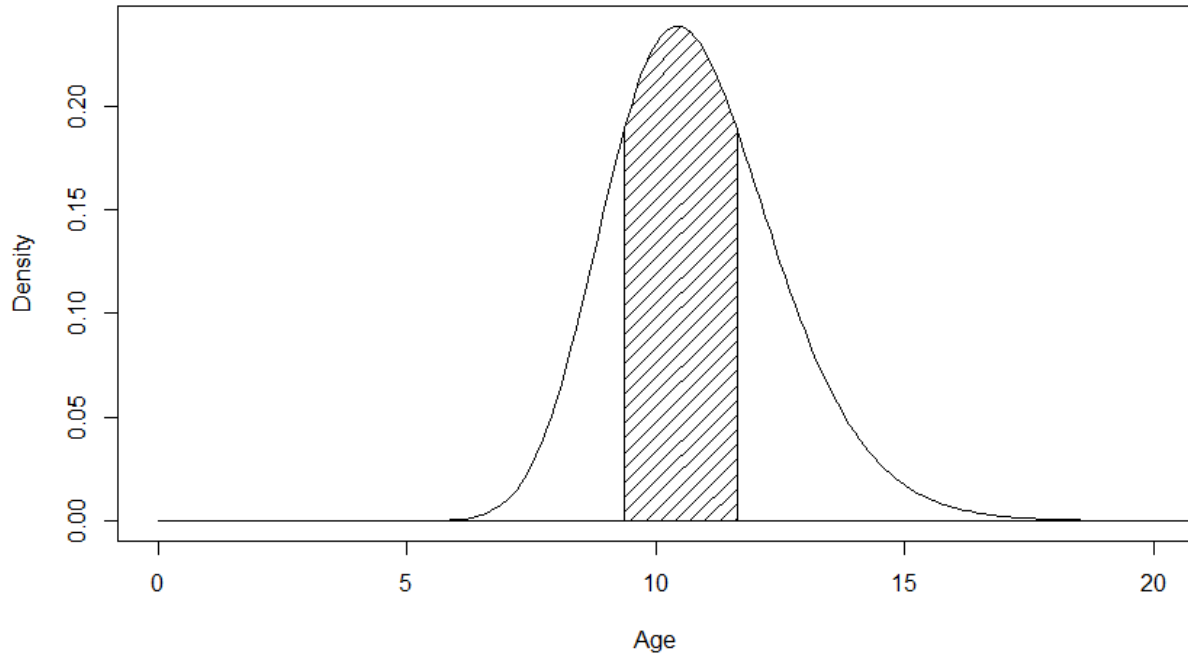
HPD female upper premolar 1 stage 10 (R 1/2), US Deaths 2008 prior



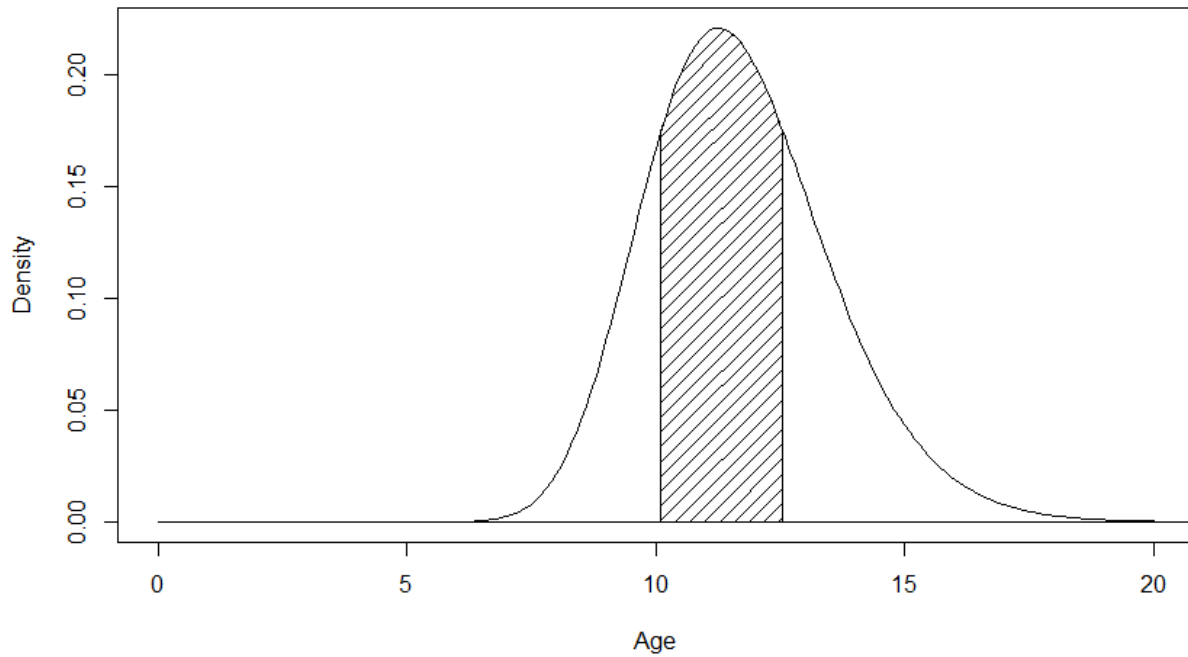
HPD female upper premolar 1 stage 11 (R 3/4), US Deaths 2008 prior



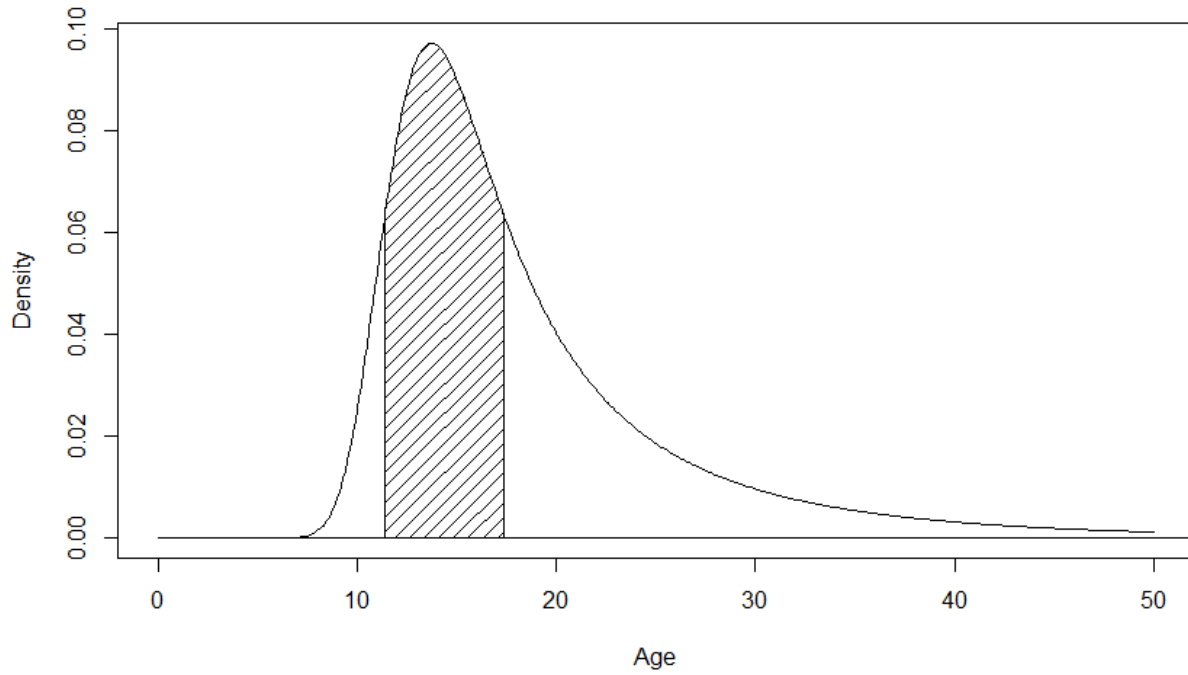
HPD female upper premolar 1 stage 12 (Rc), US Deaths 2008 prior



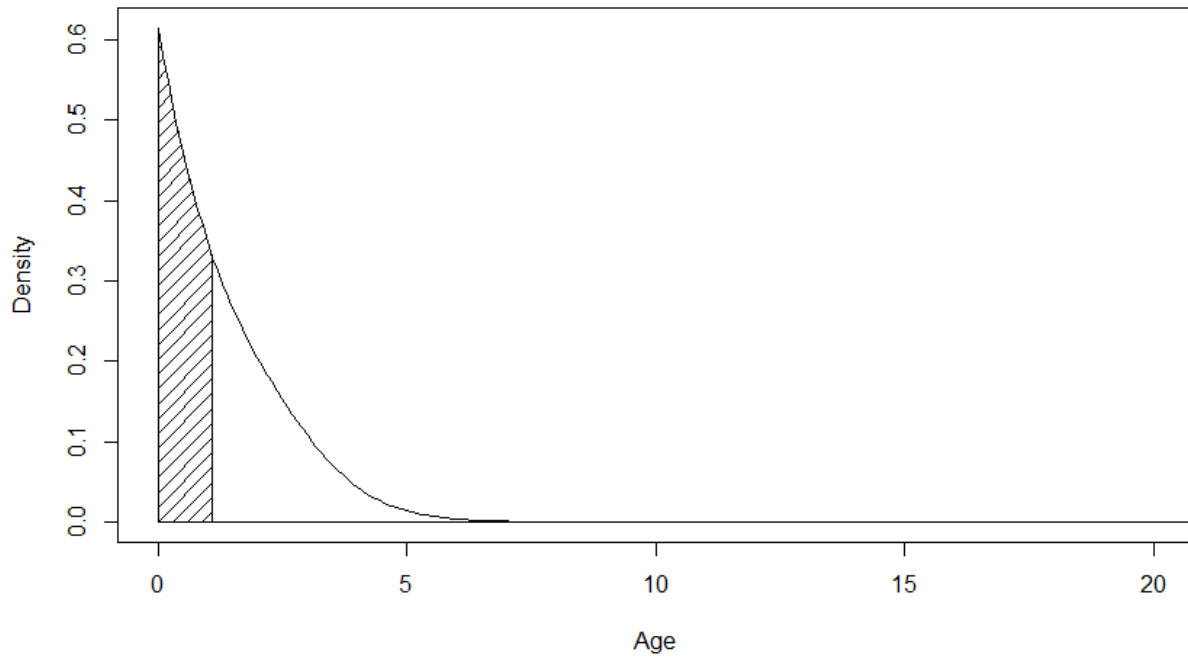
HPD female upper premolar 1 stage 13 (A 1/2), US Deaths 2008 prior



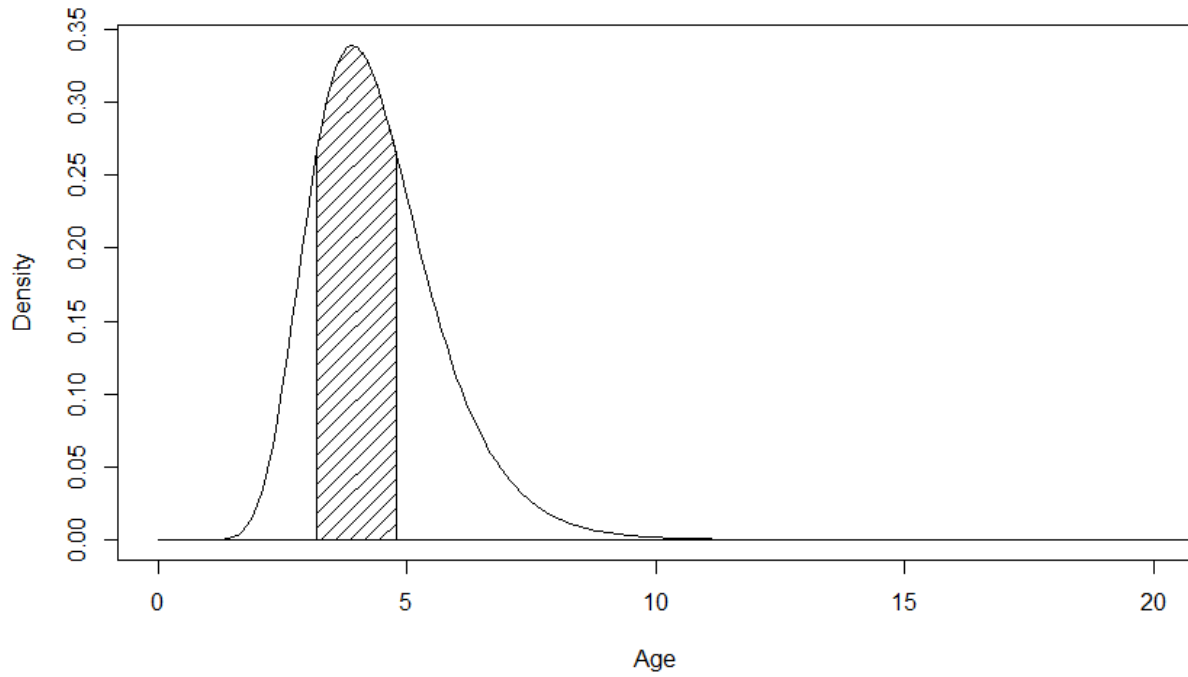
HPD female upper premolar 1 stage 14 (Ac), US Deaths 2008 prior



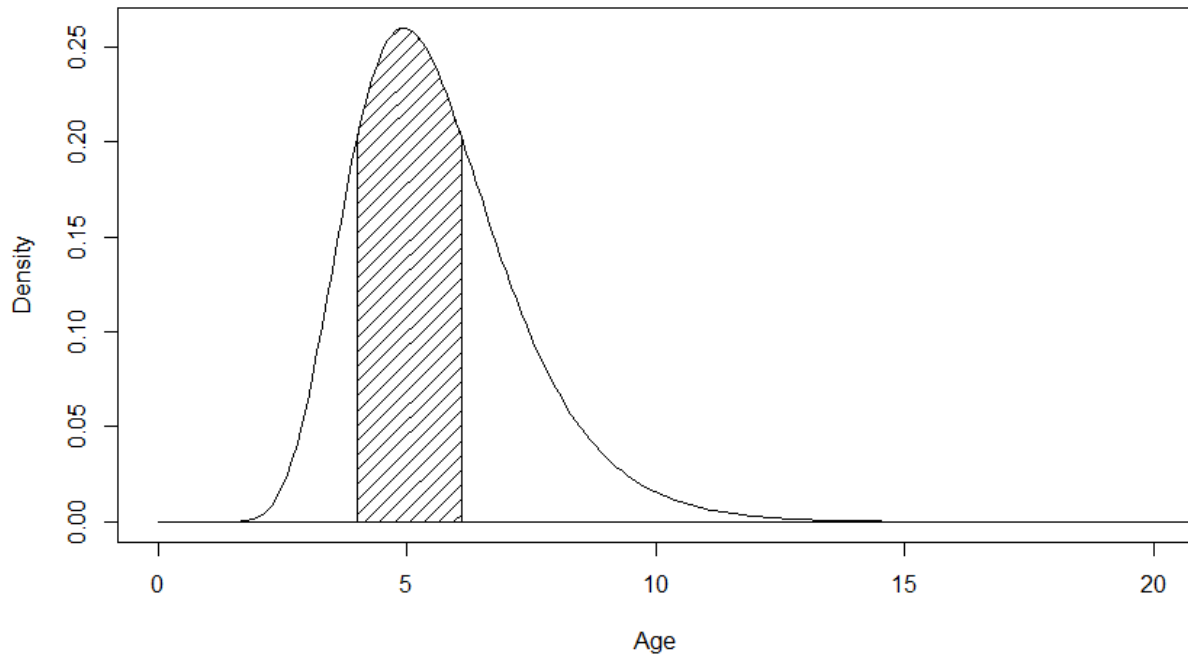
HPD female upper premolar 2 stage 5 (Cr 3/4), US Deaths 2008 prior



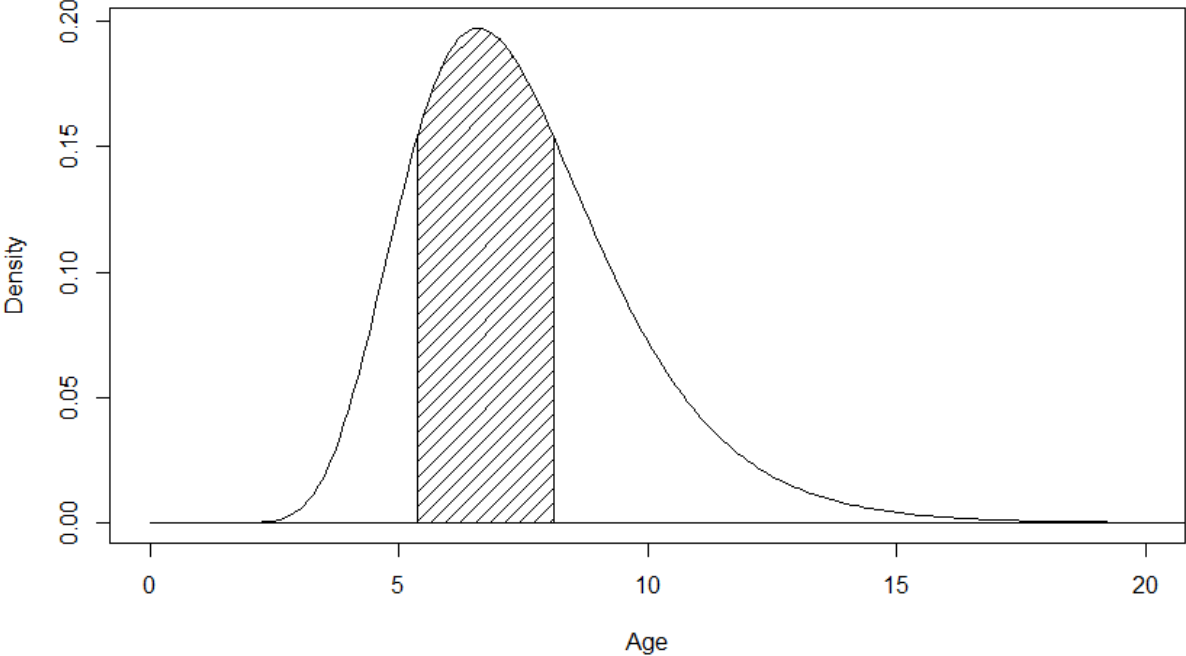
HPD female upper premolar 2 stage 6 (Crc), US Deaths 2008 prior



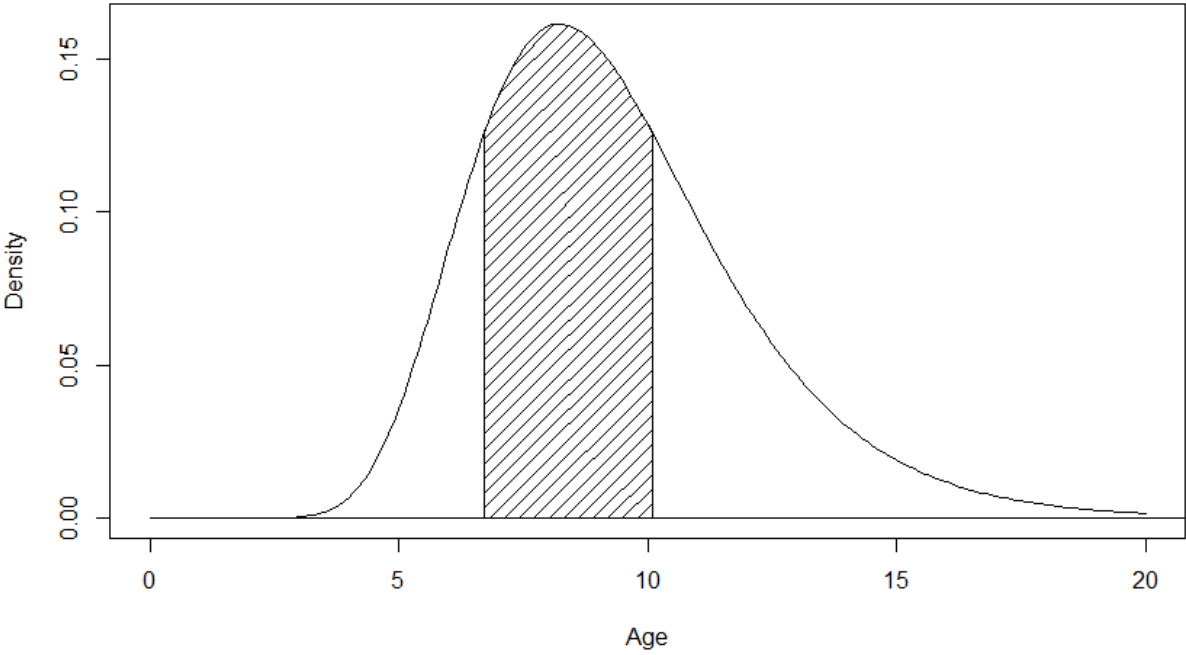
HPD female upper premolar 2 stage 7 (Ri), US Deaths 2008 prior



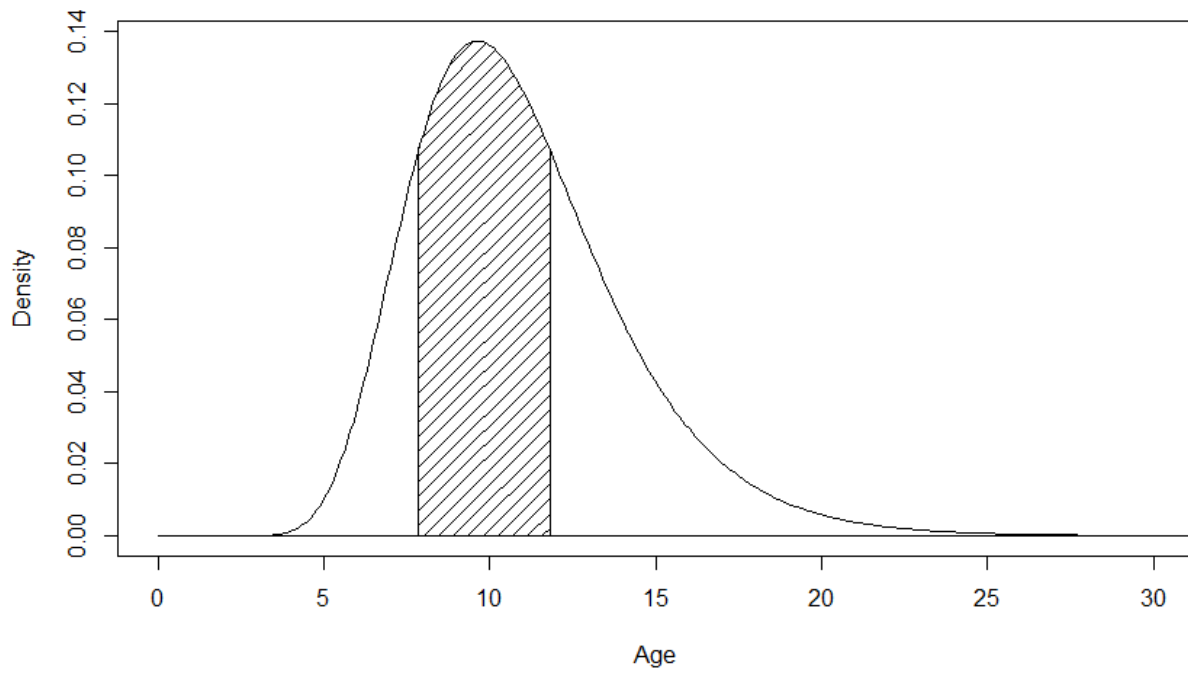
HPD female upper premolar 2 stage 9 (R 1/4), US Deaths 2008 prior



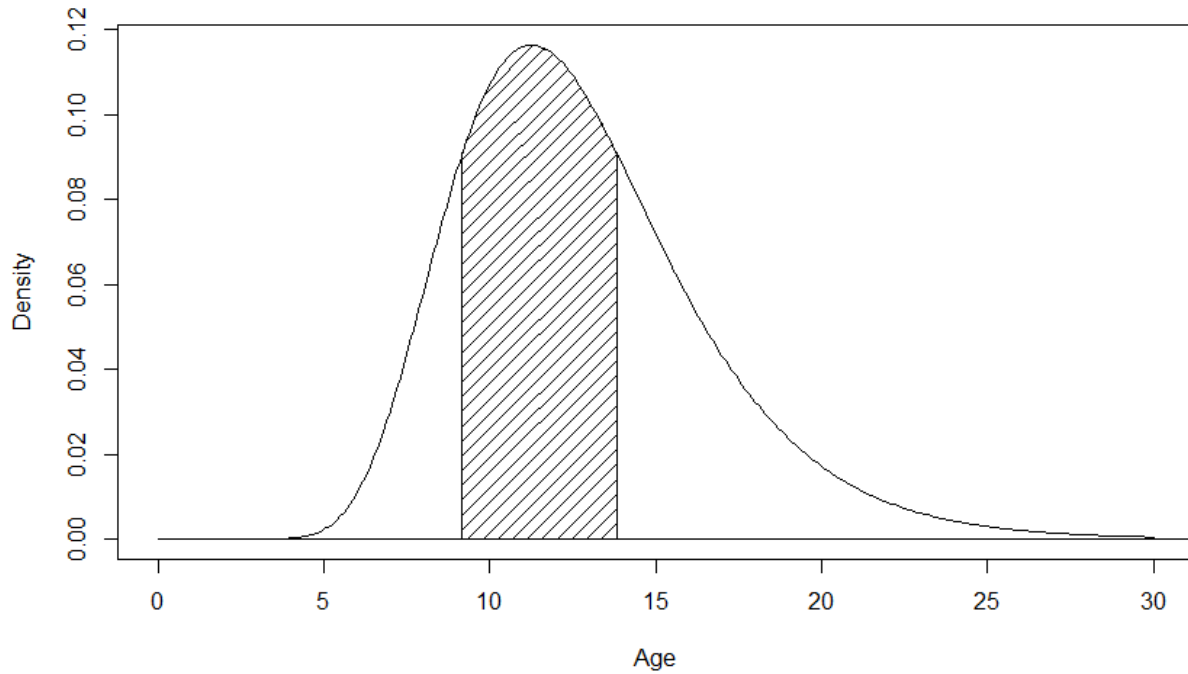
HPD female upper premolar 2 stage 10 (R 1/2), US Deaths 2008 prior



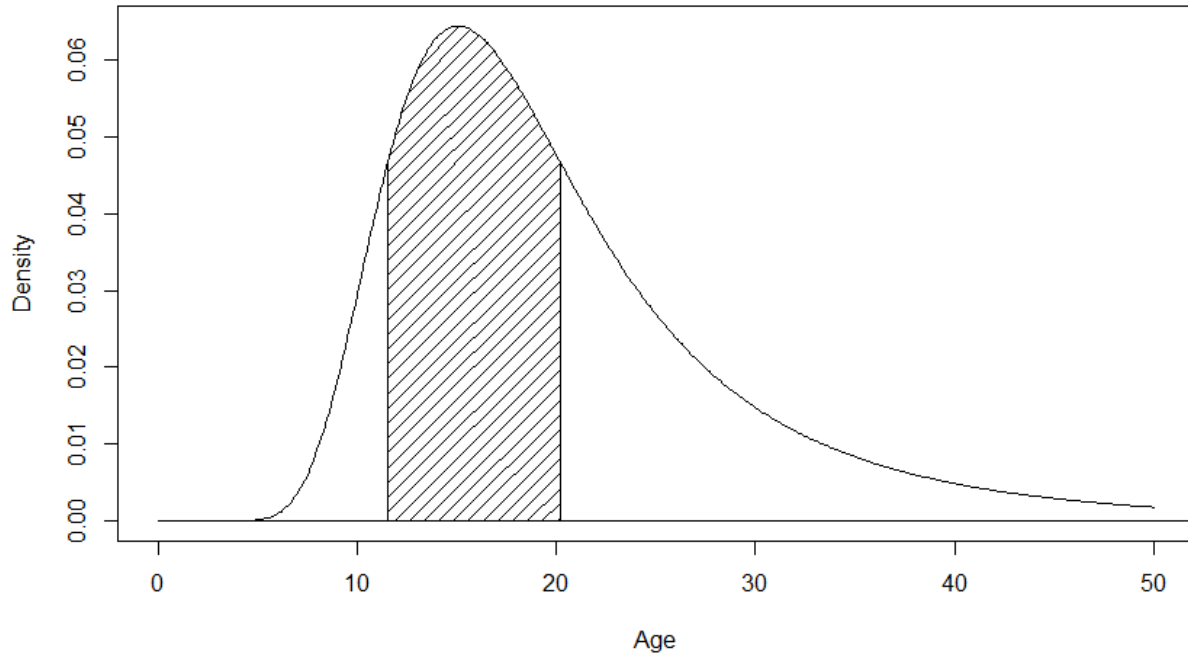
HPD female upper premolar 2 stage 11 (R 3/4), US Deaths 2008 prior



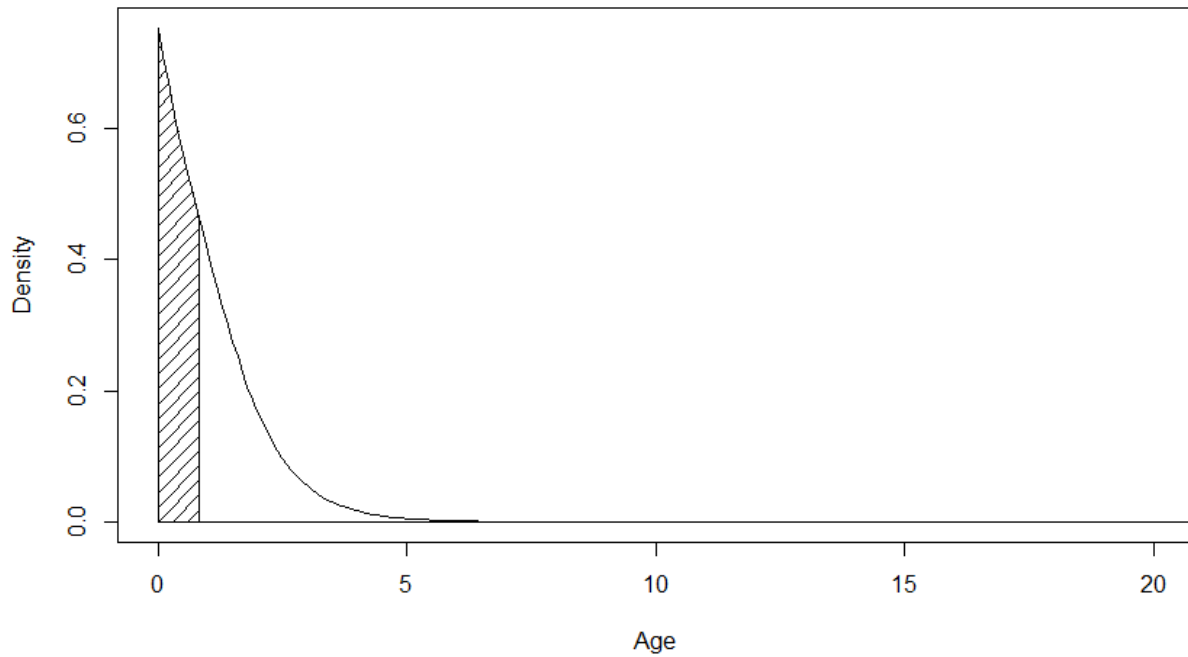
HPD female upper premolar 2 stage 12 (Rc), US Deaths 2008 prior



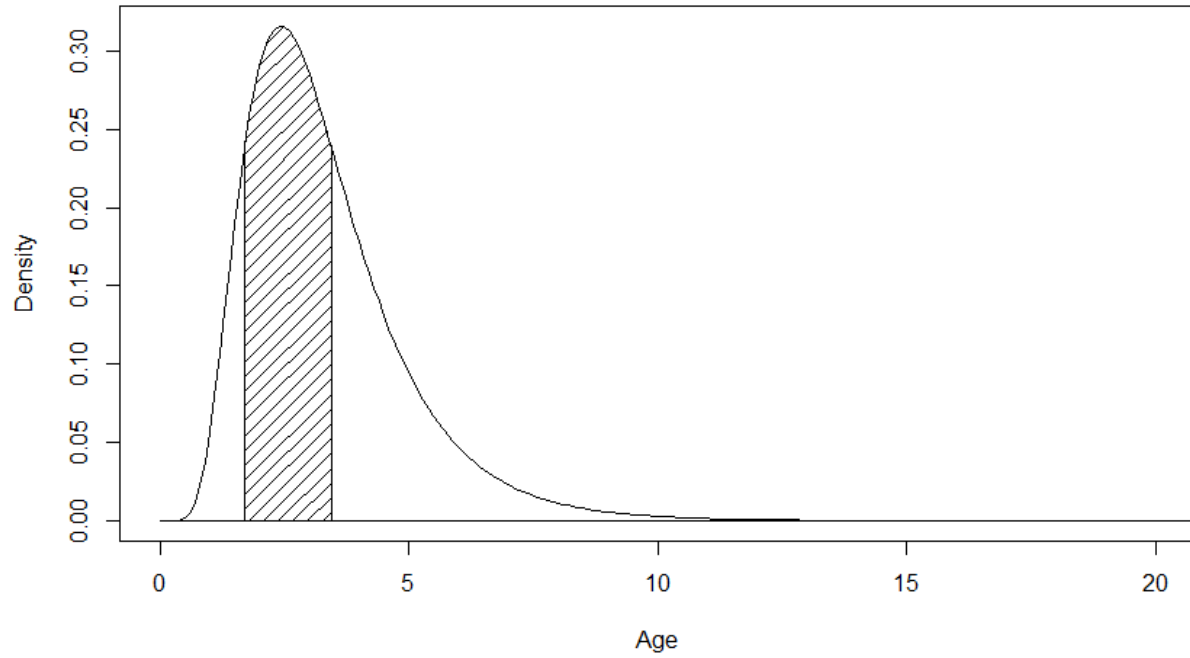
HPD female upper premolar 2 stage 13 (A 1/2), US Deaths 2008 prior



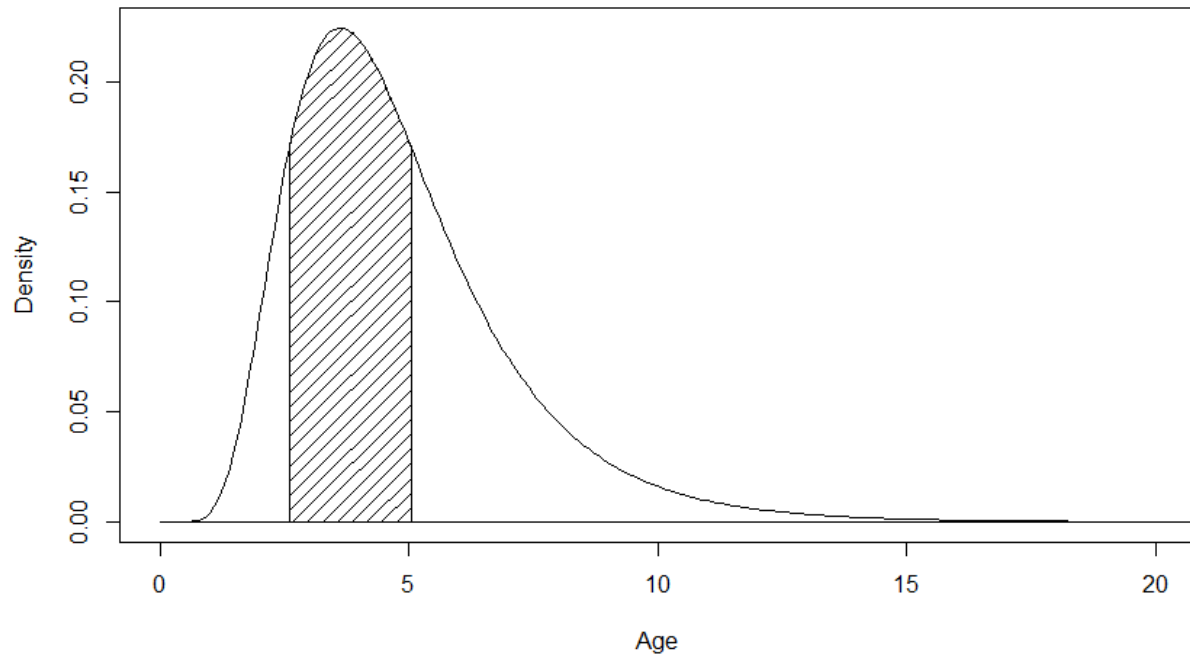
HPD female upper molar 1 stage 9 (R 1/4), US Deaths 2008 prior



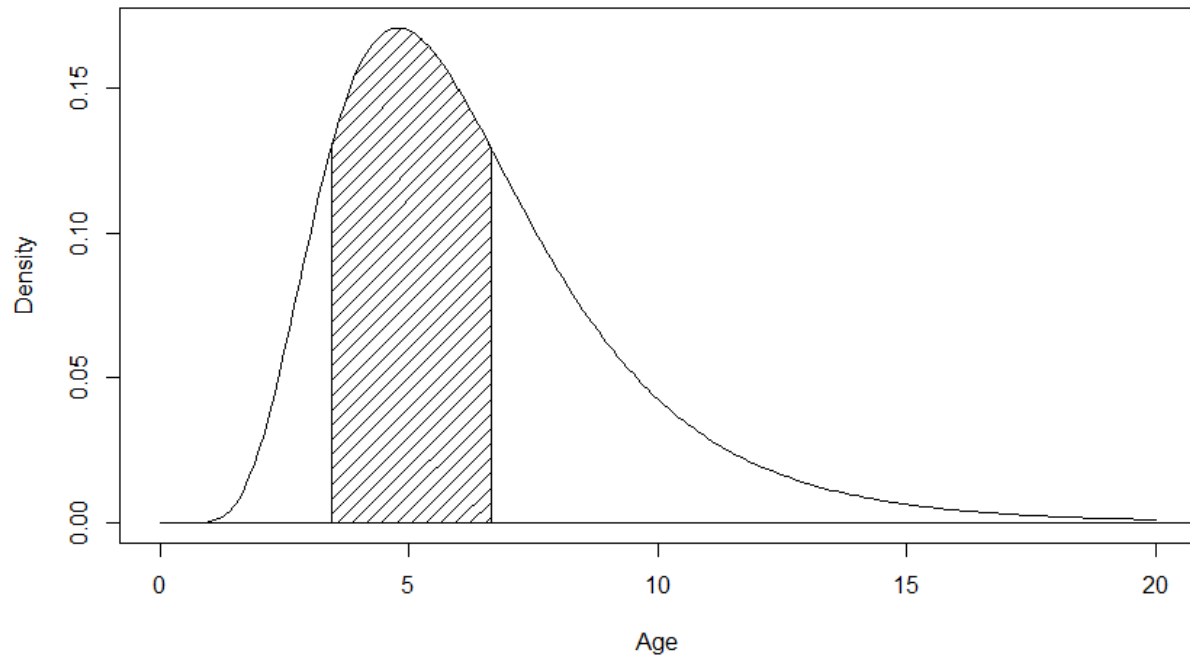
HPD female upper molar 1 stage 10 (R 1/2), US Deaths 2008 prior



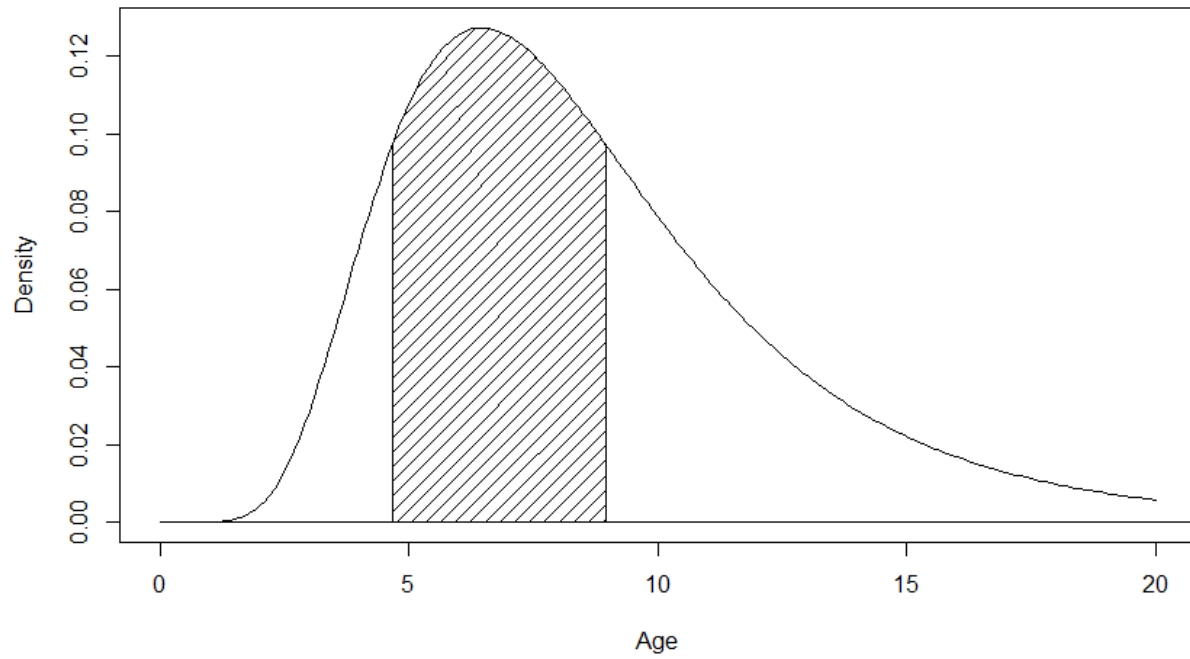
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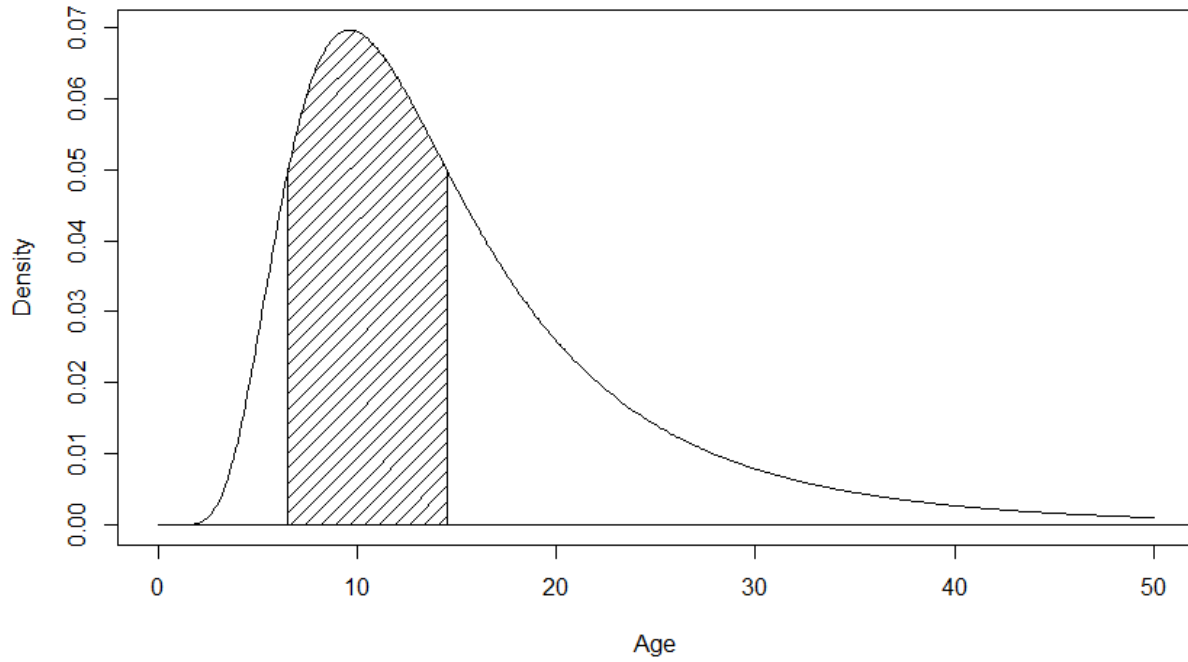
HPD female upper molar 1 stage 12 (Rc), US Deaths 2008 prior



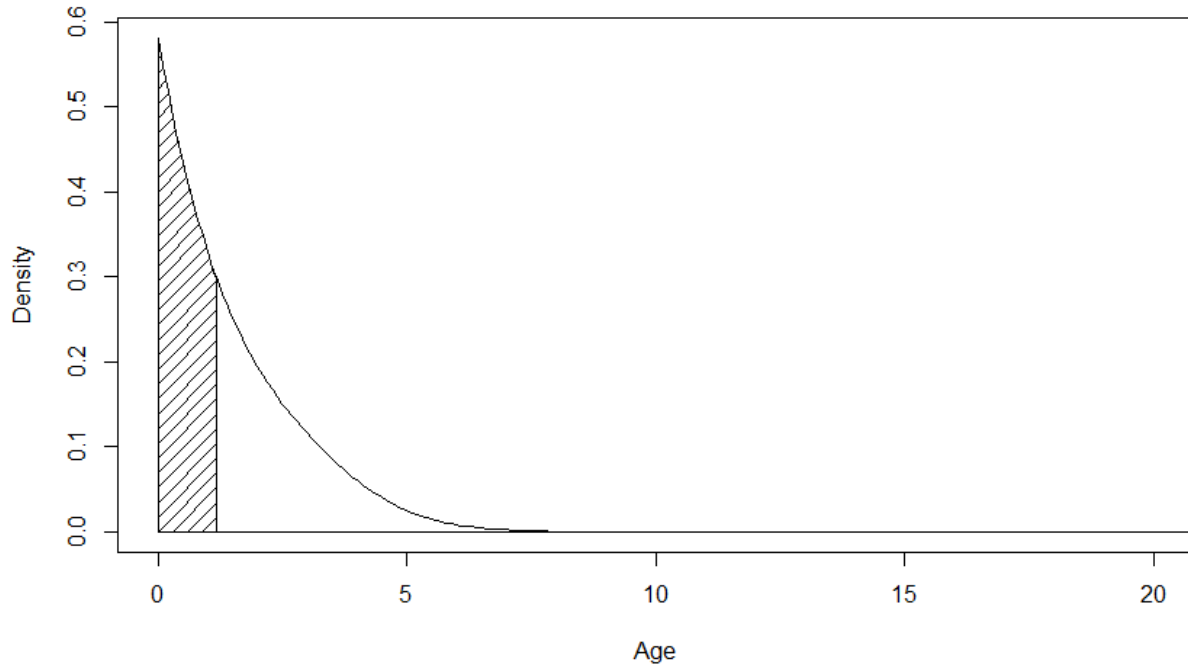
HPD female upper molar 1 stage 13 (A 1/2), US Deaths 2008 prior



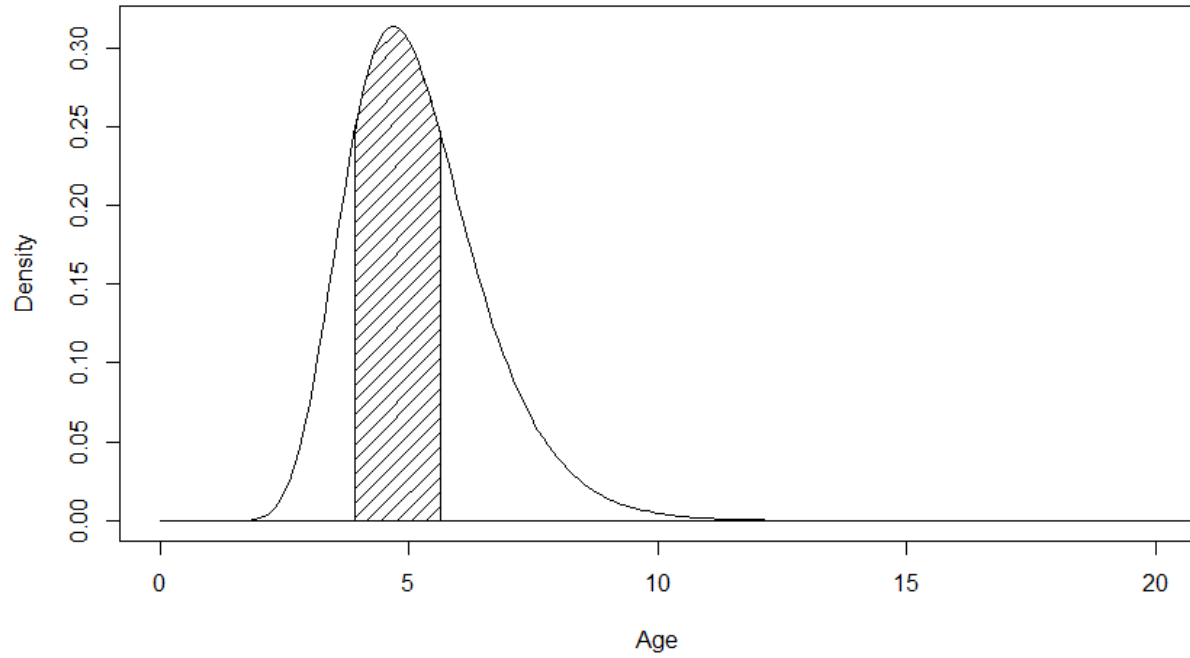
HPD female upper molar 1 stage 14 (Ac), US Deaths 2008 prior



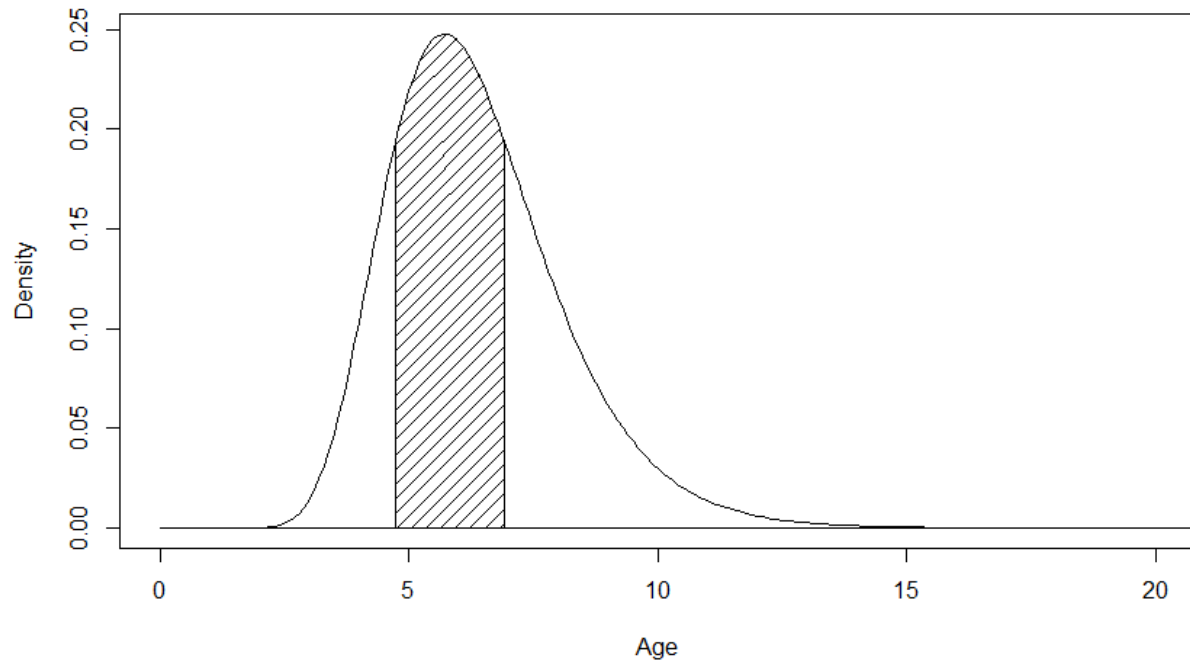
HPD female upper molar 2 stage 5 (Cr 3/4), US Deaths 2008 prior



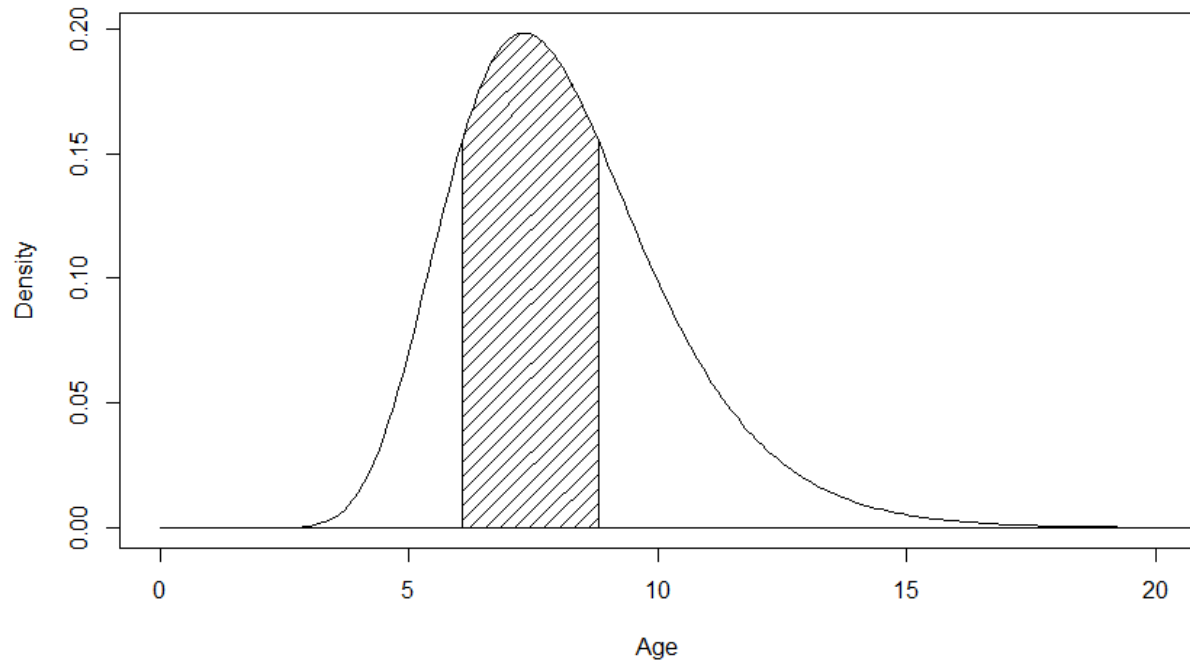
HPD female upper molar 2 stage 6 (Crc), US Deaths 2008 prior



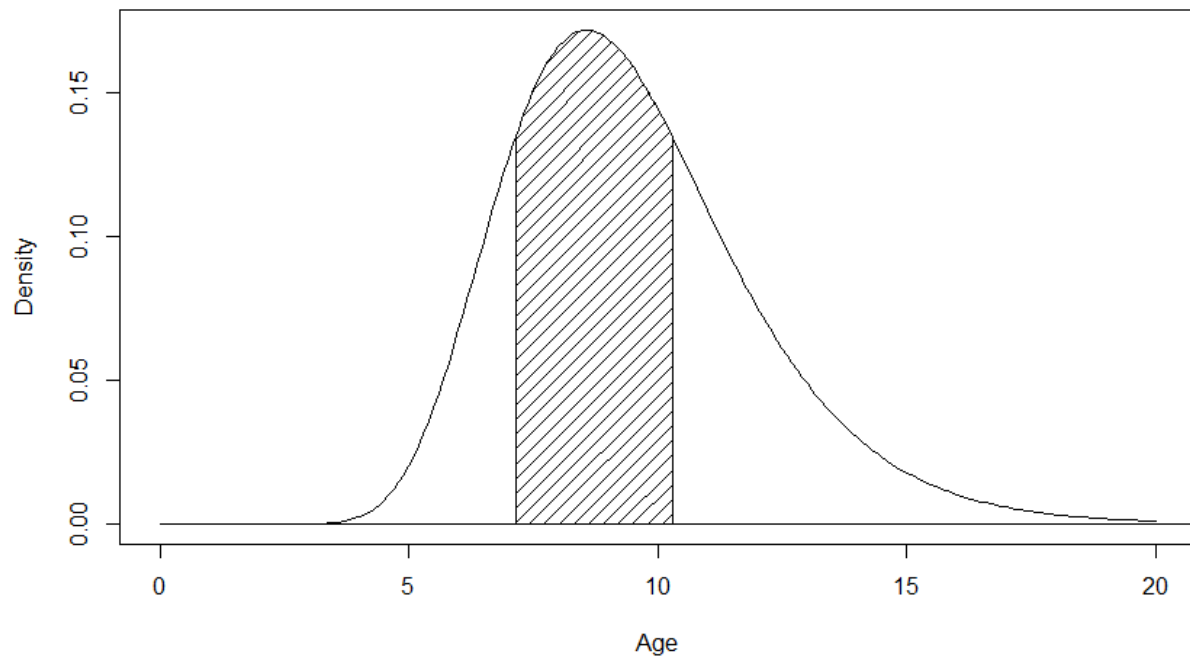
HPD female upper molar 2 stage 7 (Ri), US Deaths 2008 prior



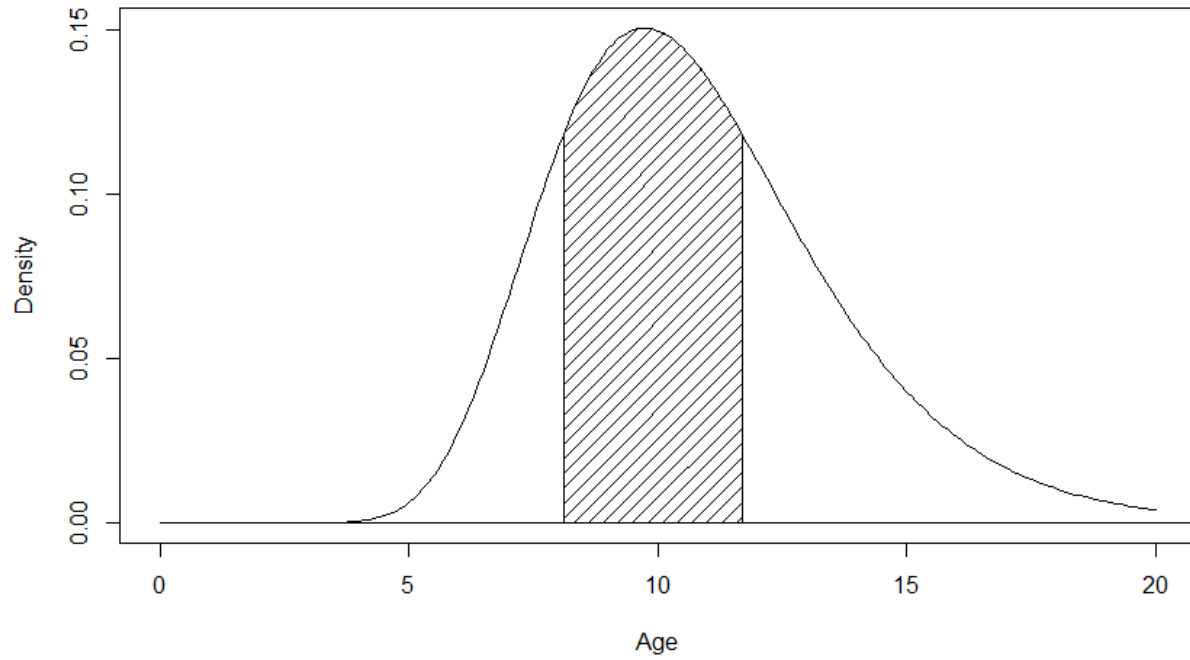
HPD female upper molar 2 stage 8 (C1i), US Deaths 2008 prior



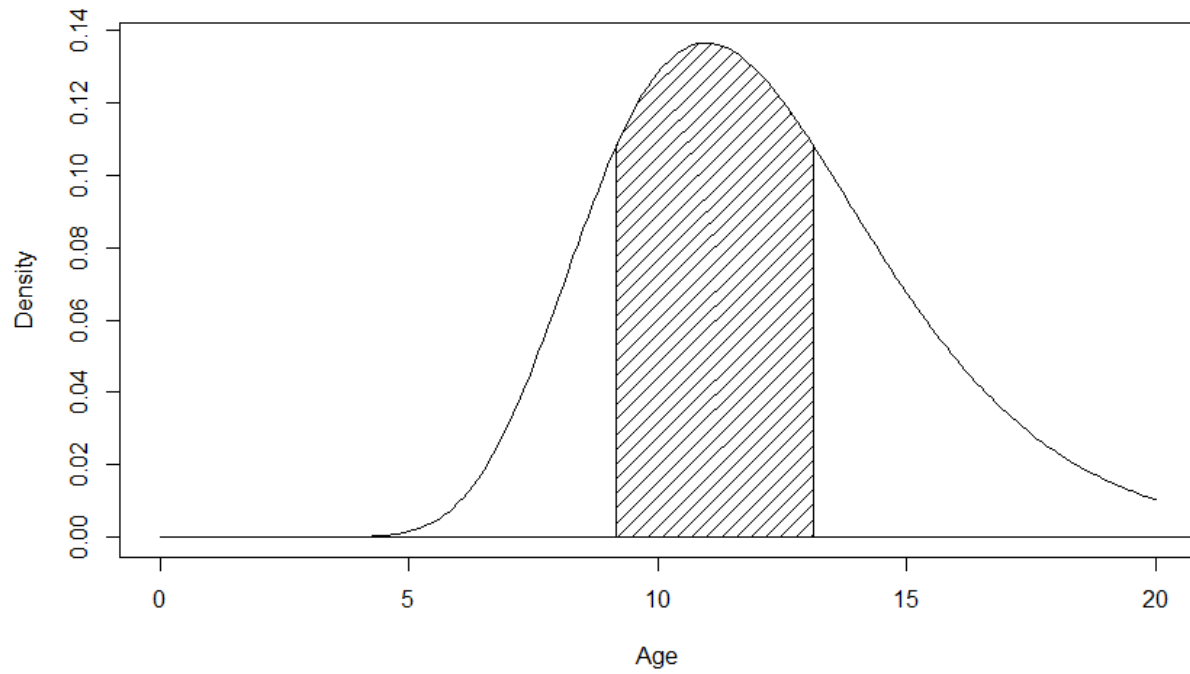
HPD female upper molar 2 stage 9 (R 1/4), US Deaths 2008 prior



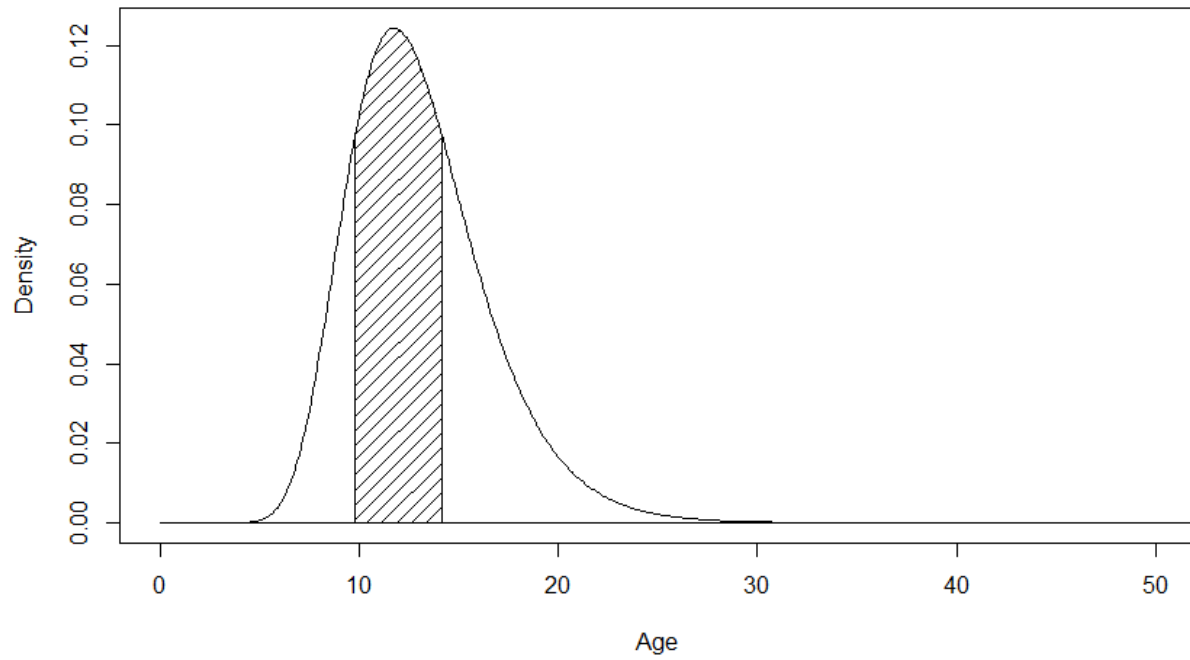
HPD female upper molar 2 stage 10 (R 1/2), US Deaths 2008 prior



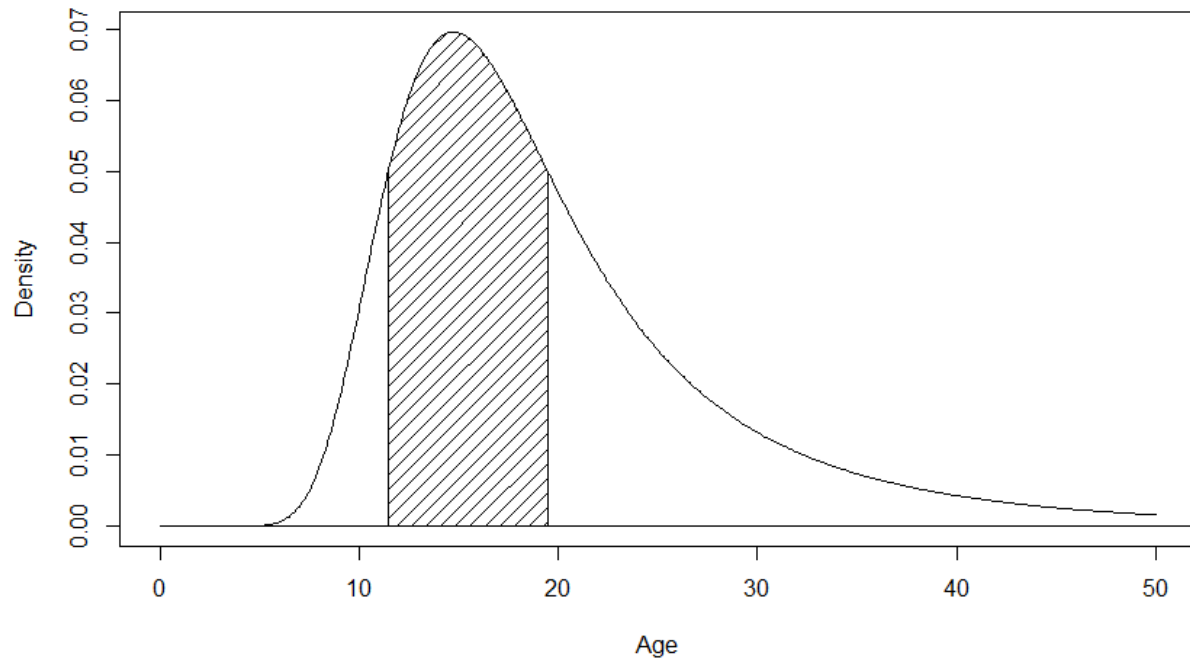
HPD female upper molar 2 stage 11 (R 3/4), US Deaths 2008 prior



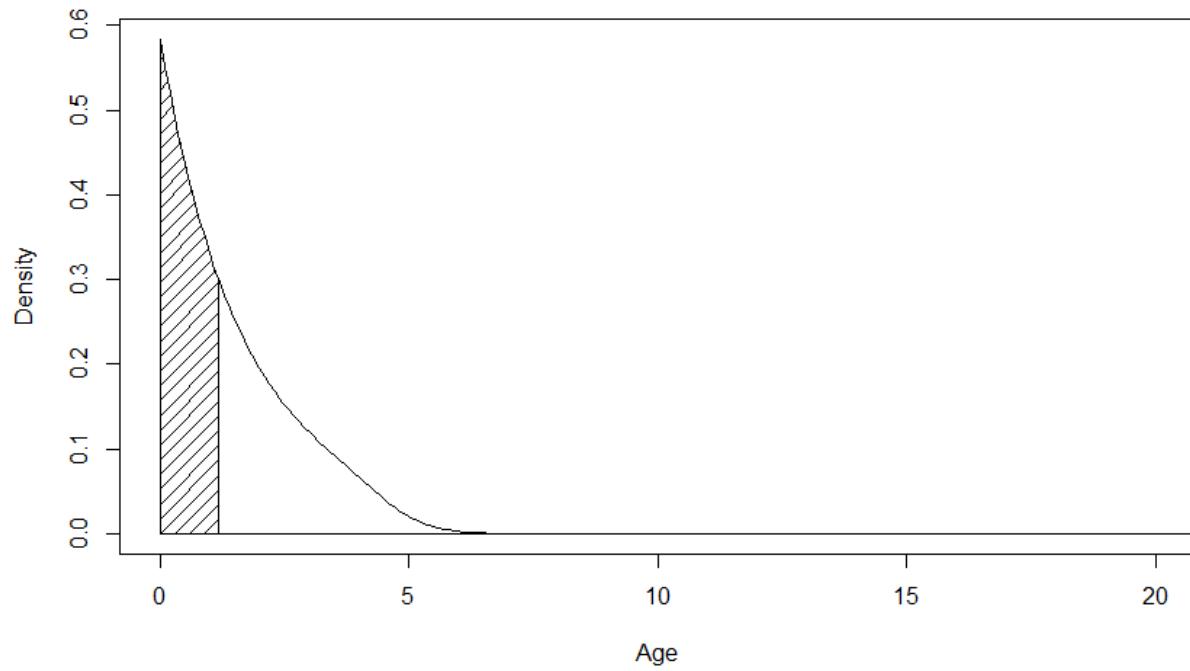
HPD female upper molar 2 stage 12 (Rc), US Deaths 2008 prior



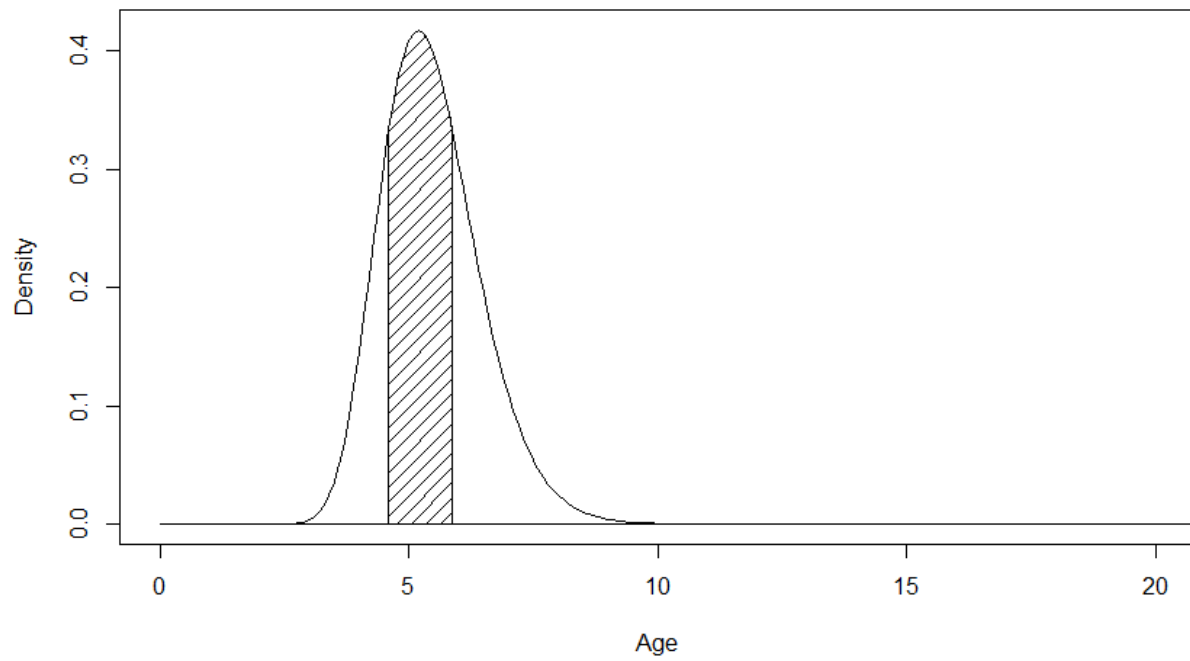
HPD female upper molar 2 stage 13 (A 1/2), US Deaths 2008 prior



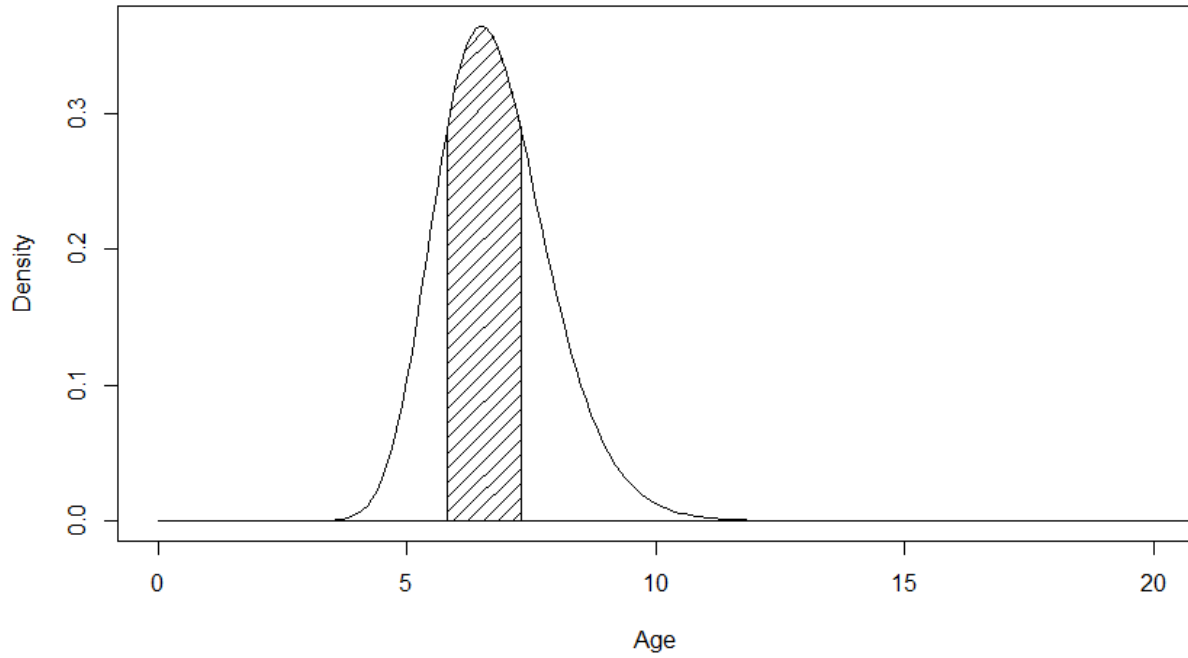
HPD female lower incisor 1 stage 9 (R 1/4), US Deaths 2008 prior



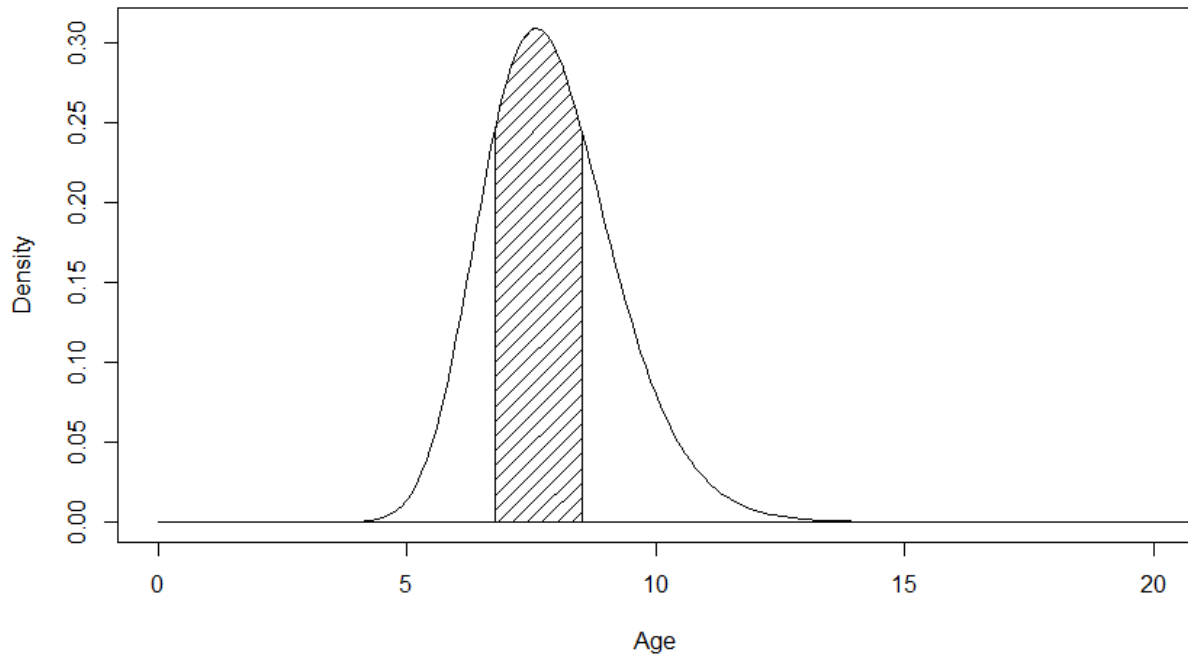
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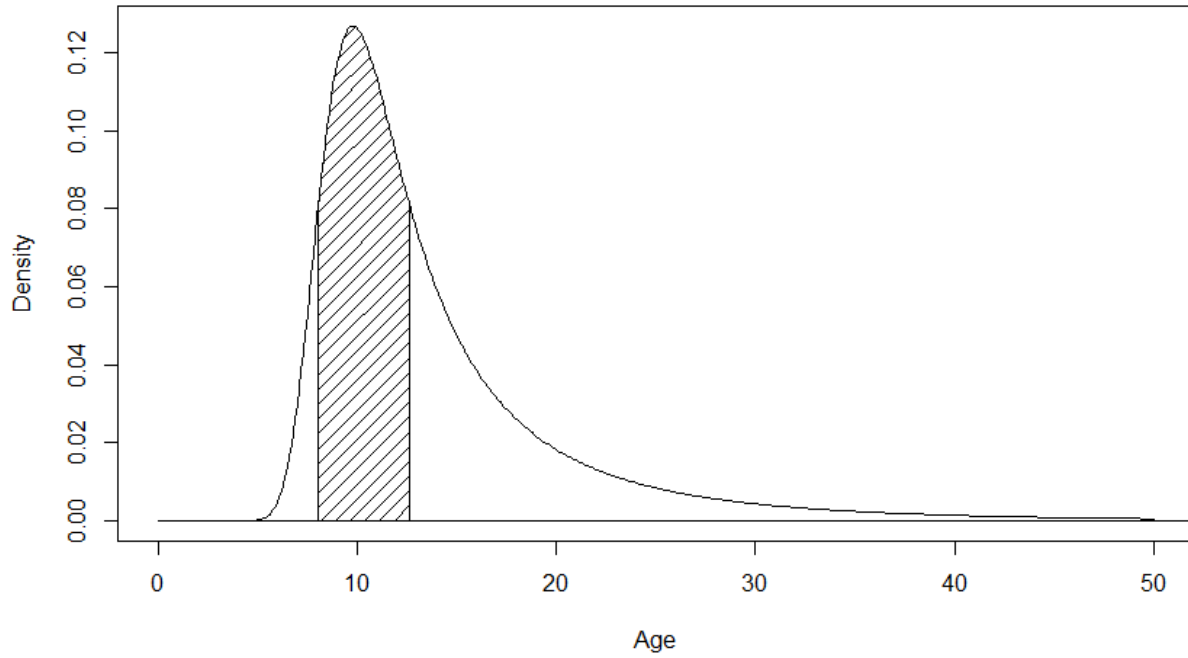
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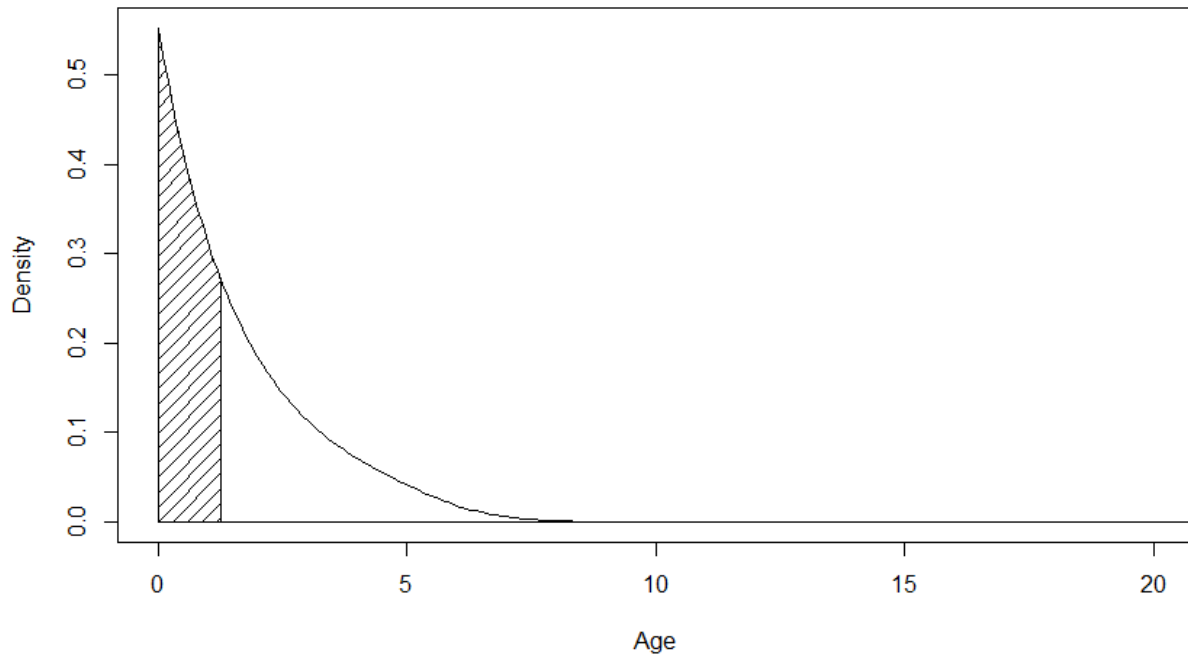
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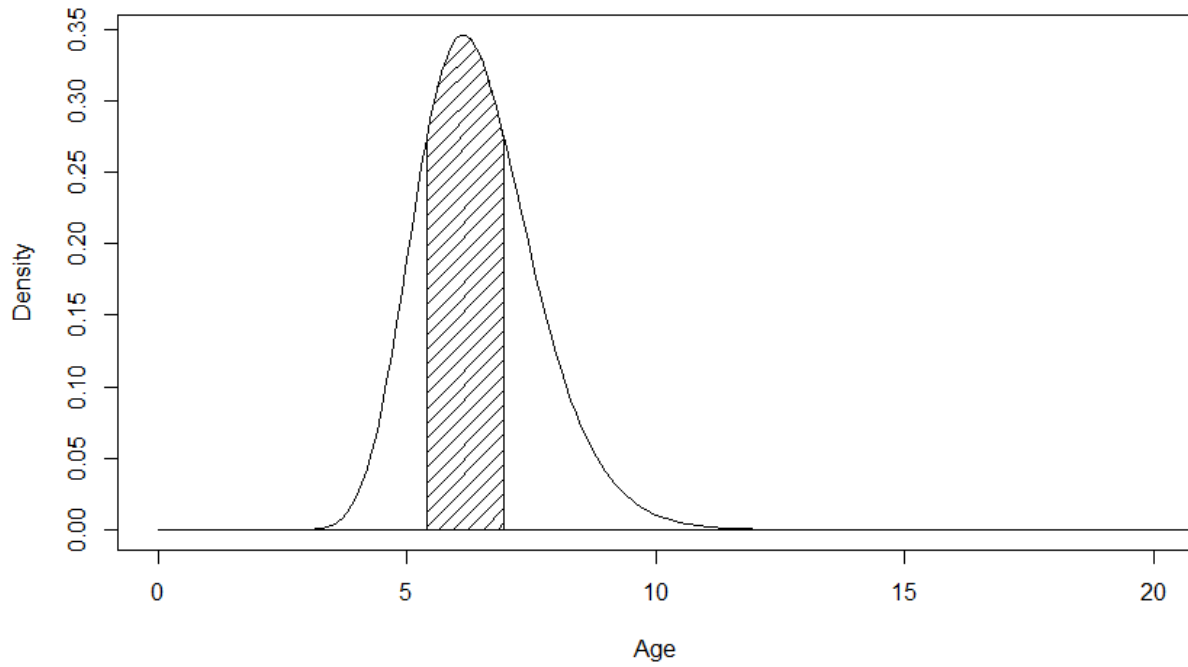
HPD female lower incisor 1 stage 14 (Ac), US Deaths 2008 prior



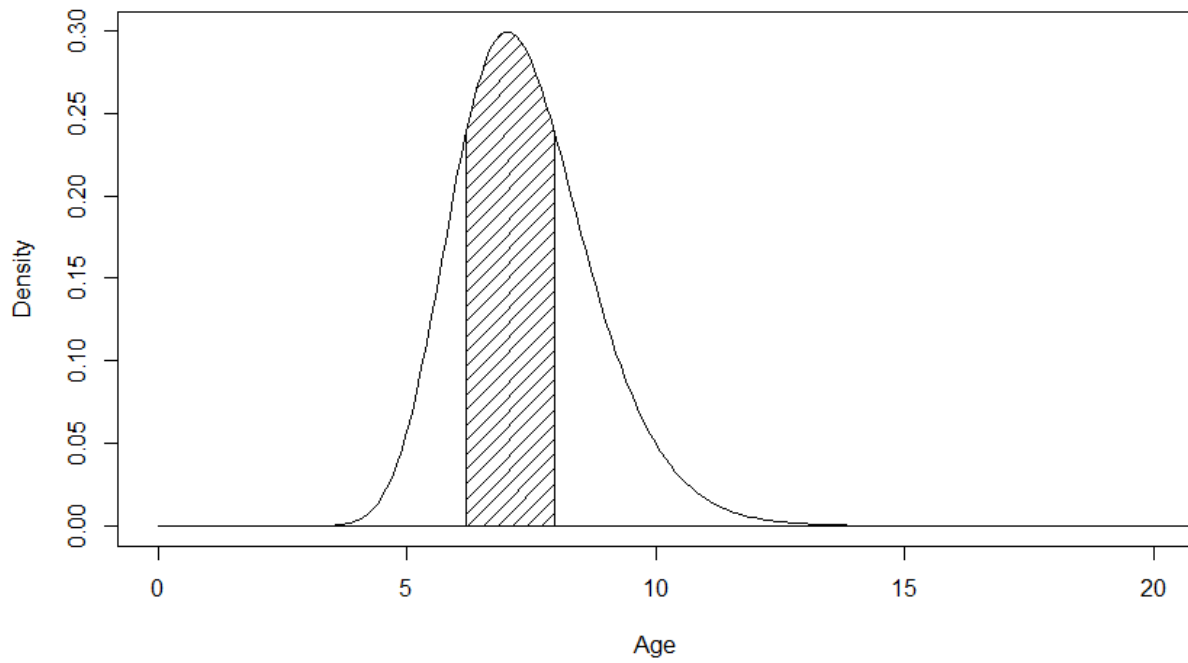
HPD female lower incisor 2 stage 10 (R 1/2), US Deaths 2008 prior



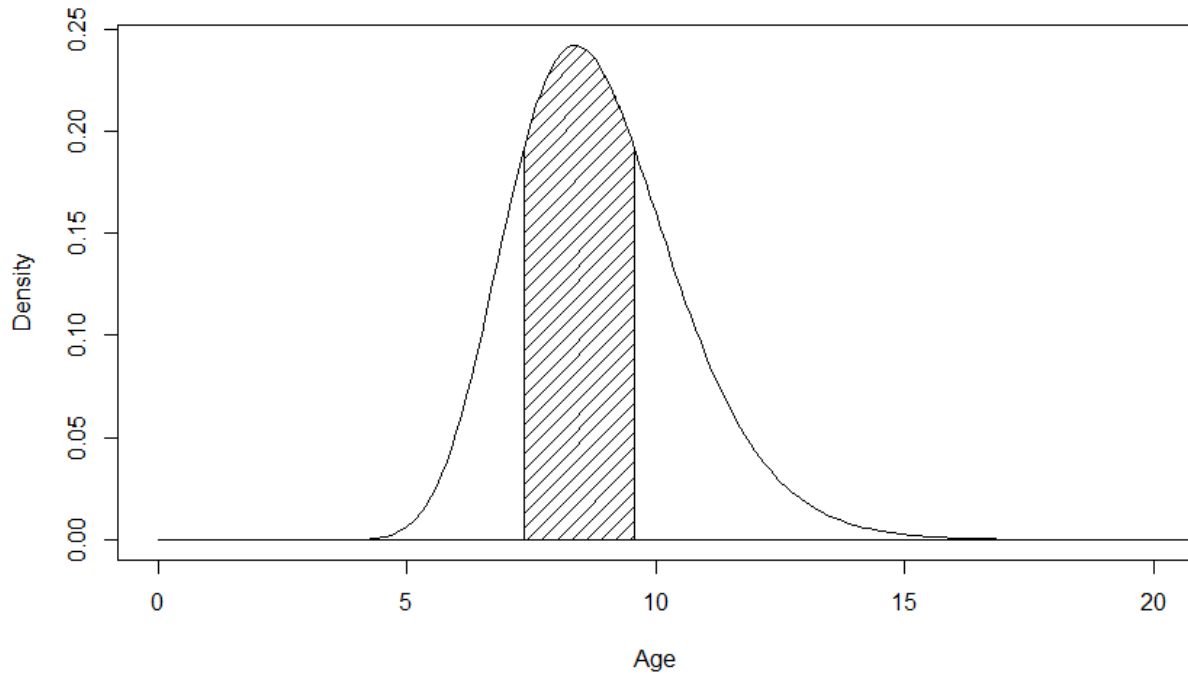
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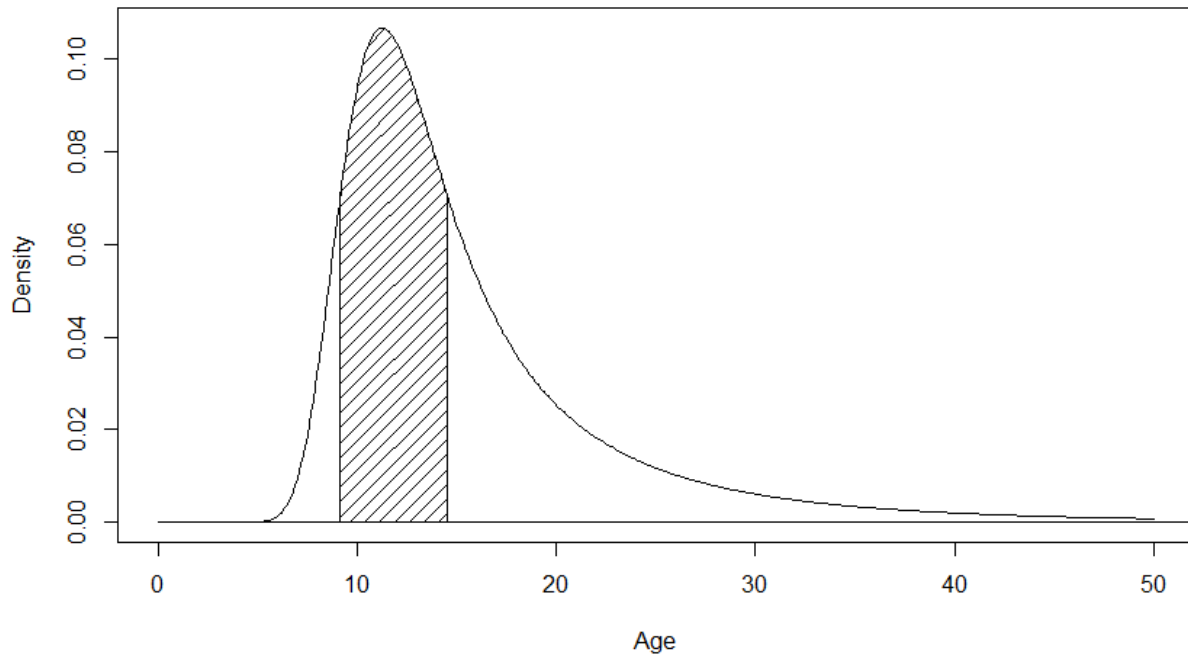
HPD female lower incisor 2 stage 12 (Rc), US Deaths 2008 prior



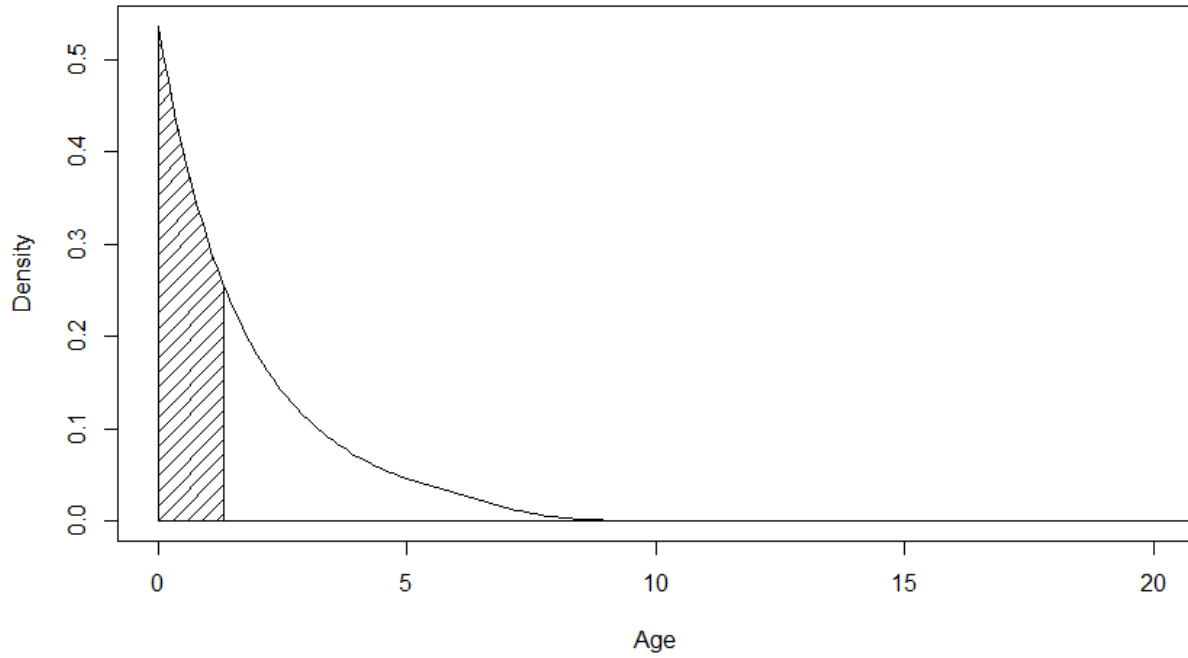
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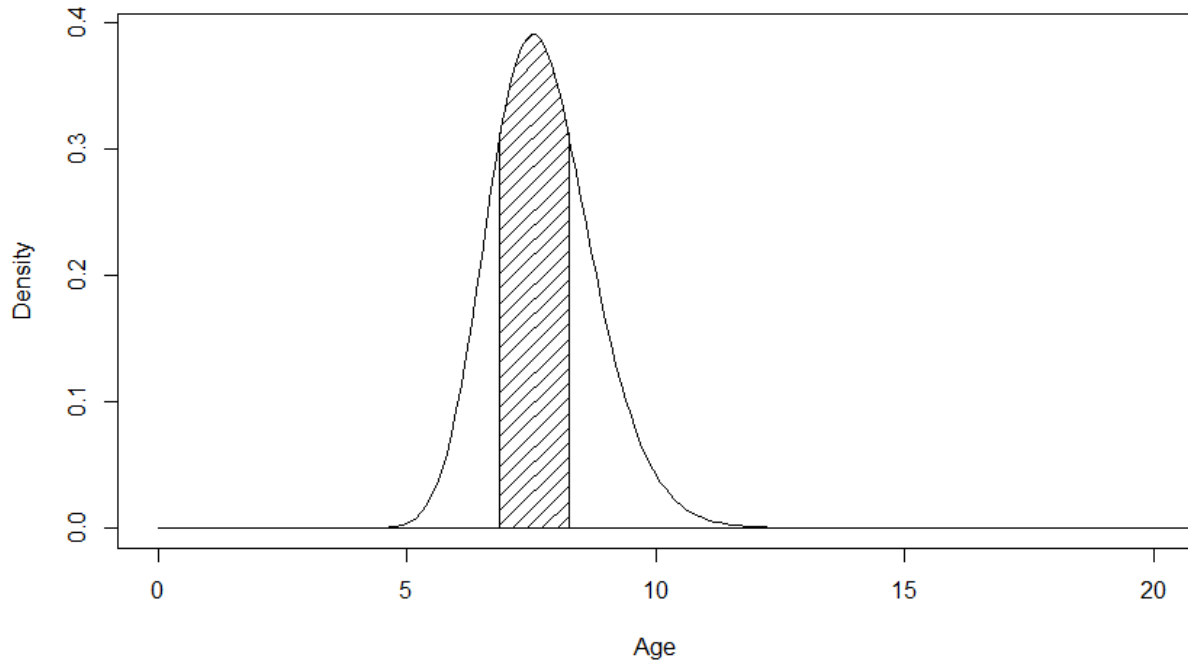
HPD female lower incisor 2 stage 14 (Ac), US Deaths 2008 prior



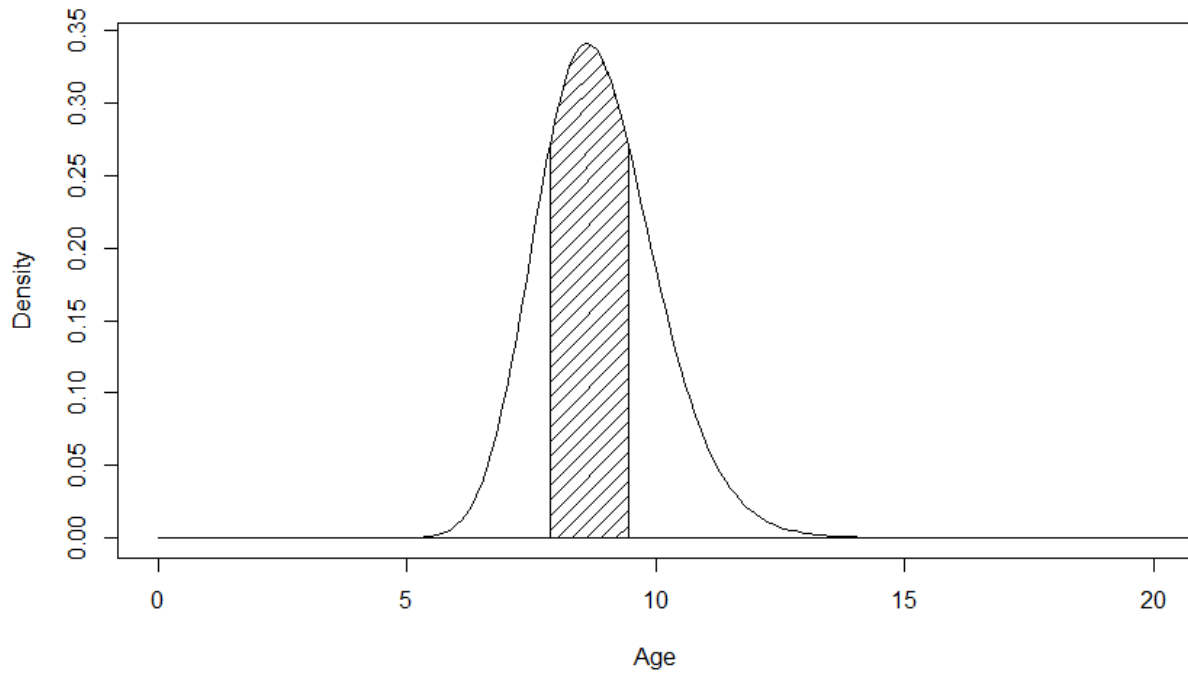
HPD female lower canine stage 9 (R 1/4), US Deaths 2008 prior



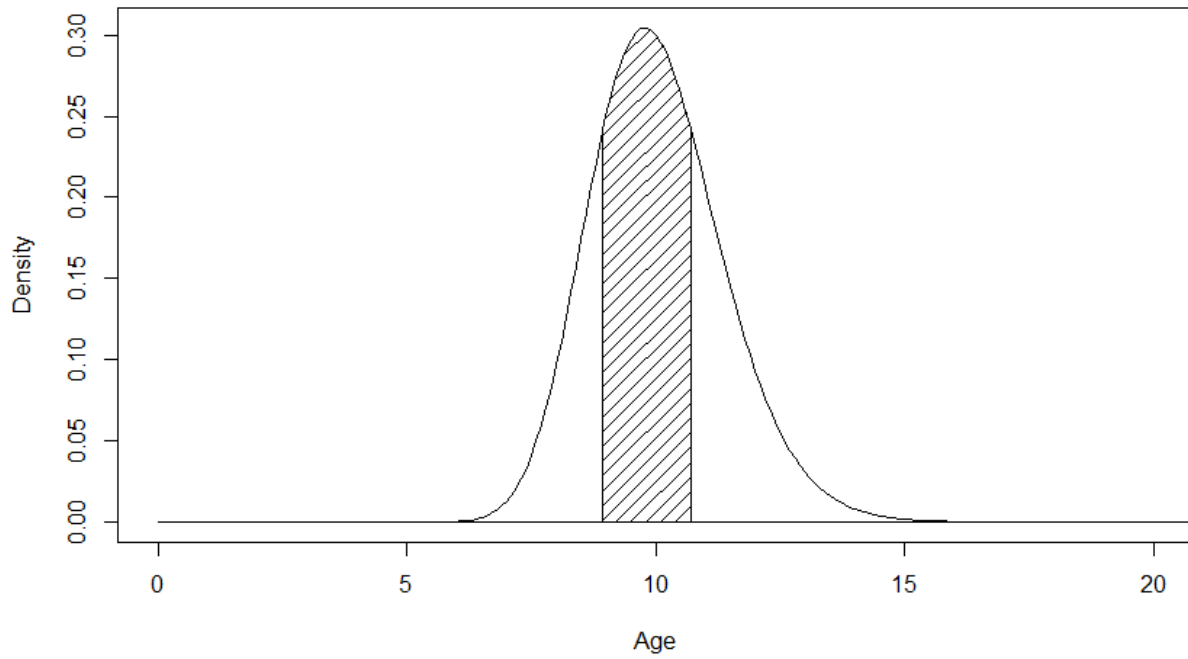
HPD female lower canine stage 10 (R 1/2), US Deaths 2008 prior



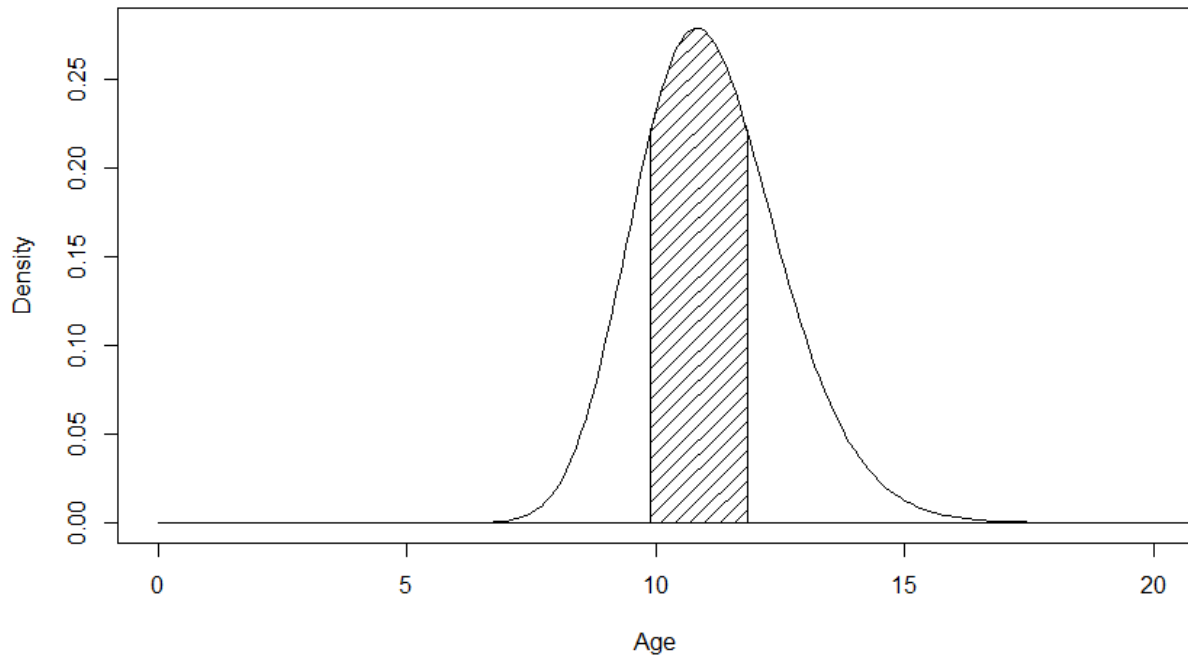
HPD female lower canine stage 11 (R 3/4), US Deaths 2008 prior



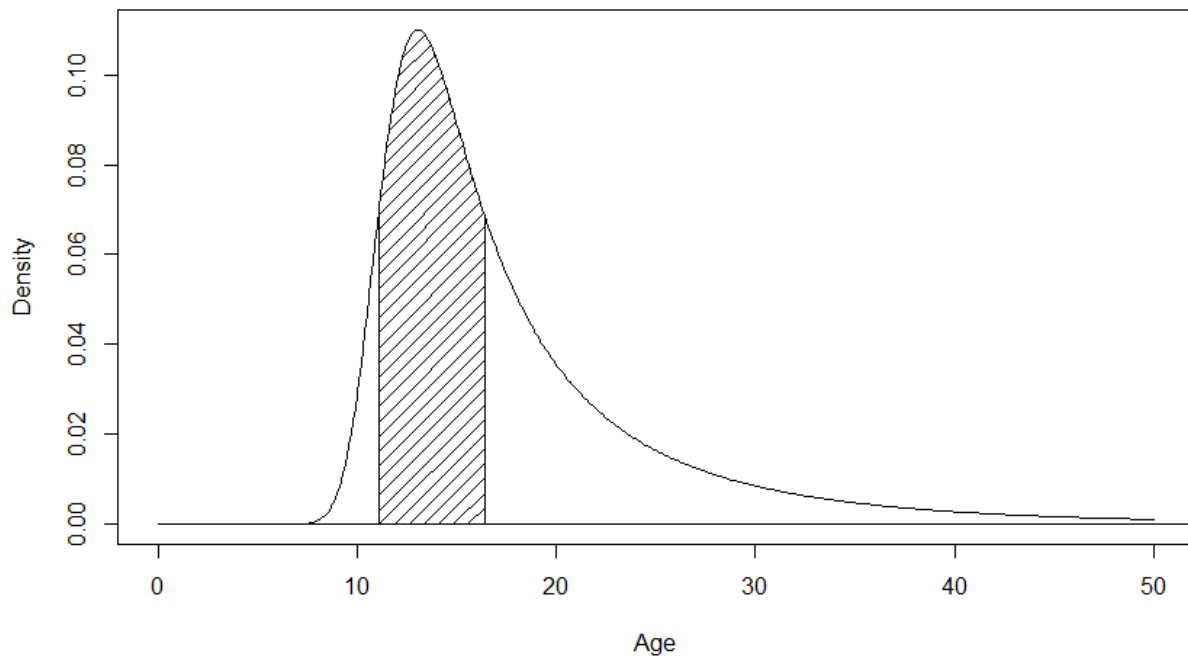
HPD female lower canine stage 12 (Rc), US Deaths 2008 prior



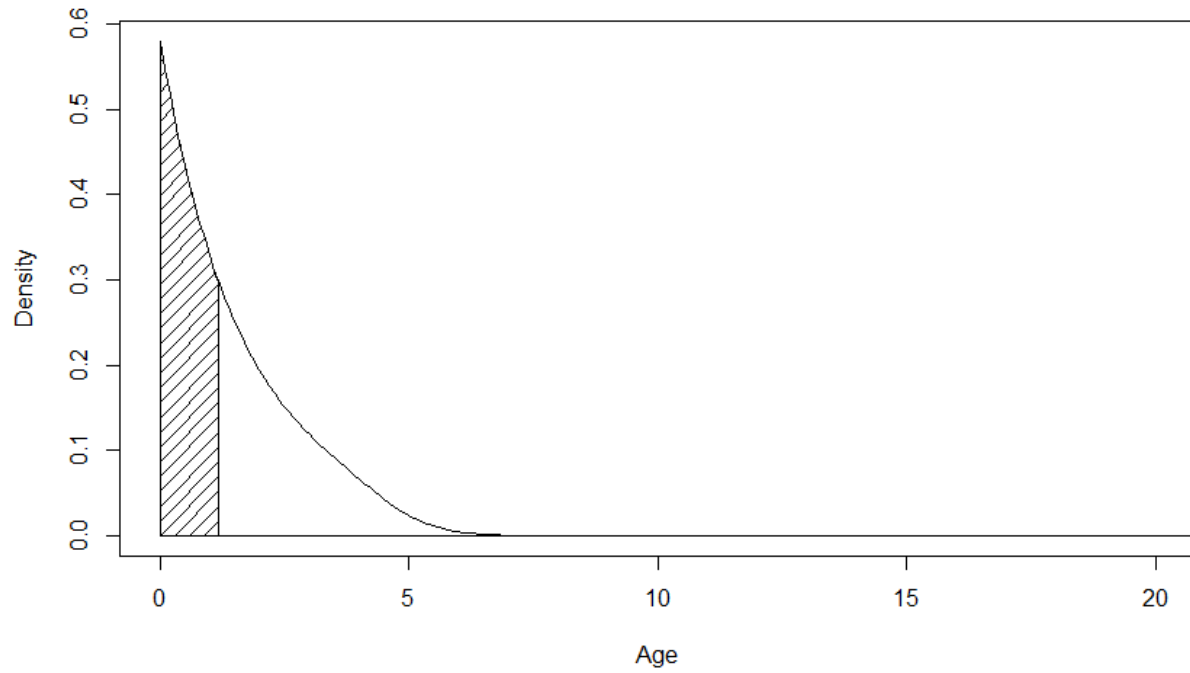
HPD female lower canine stage 13 (A 1/2), US Deaths 2008 prior



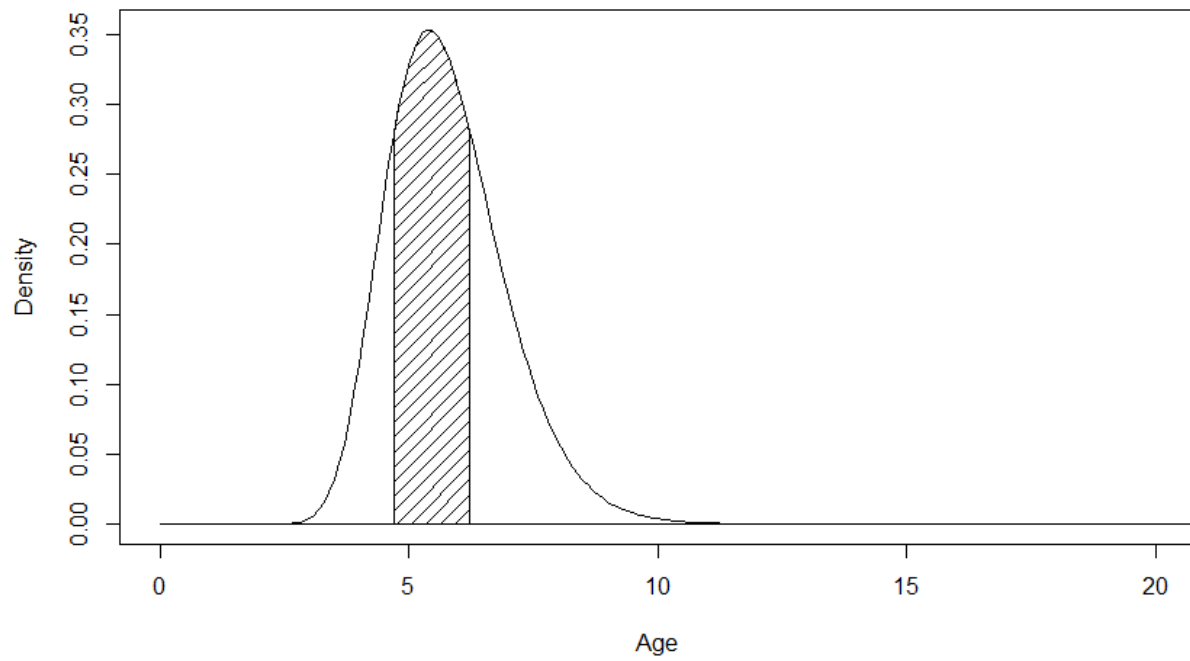
HPD female lower canine stage 14 (Ac), US Deaths 2008 prior



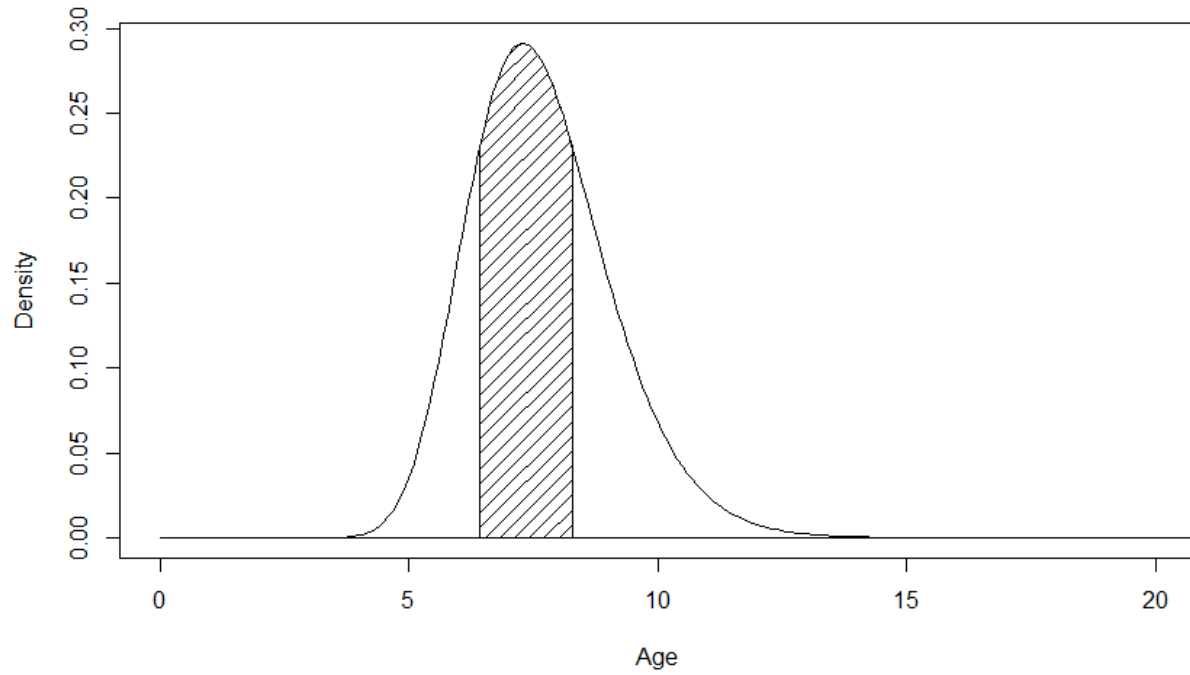
HPD female lower premolar 1 stage 6 (Crc), US Deaths 2008 prior



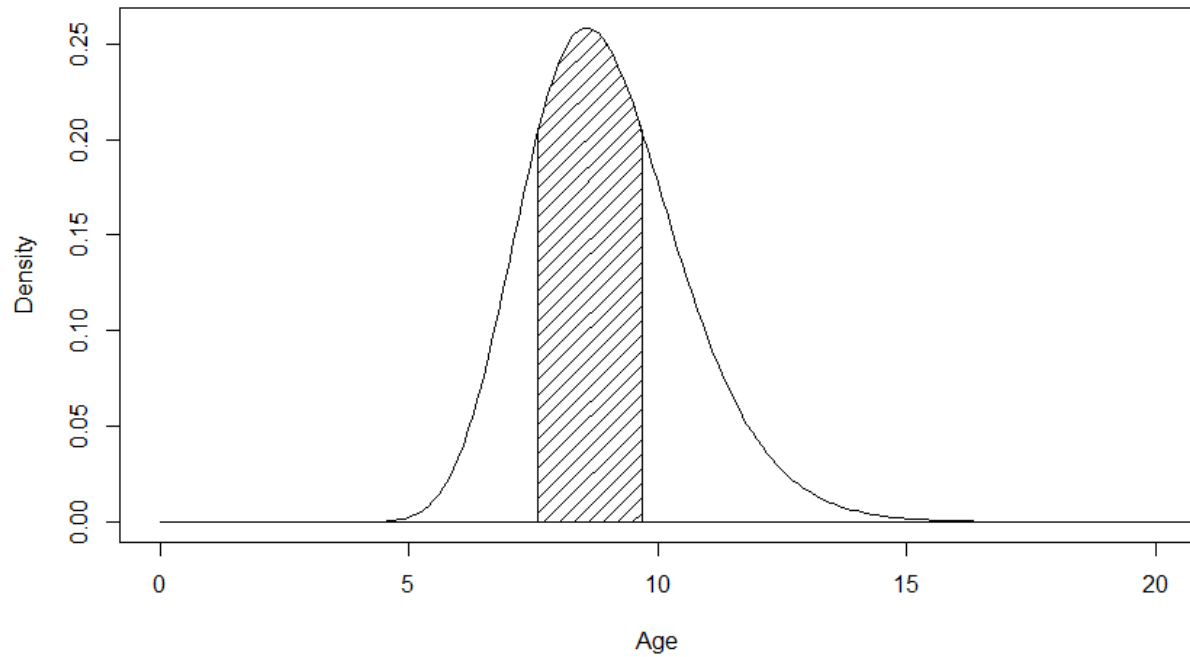
HPD female lower premolar 1 stage 7 (Ri), US Deaths 2008 prior



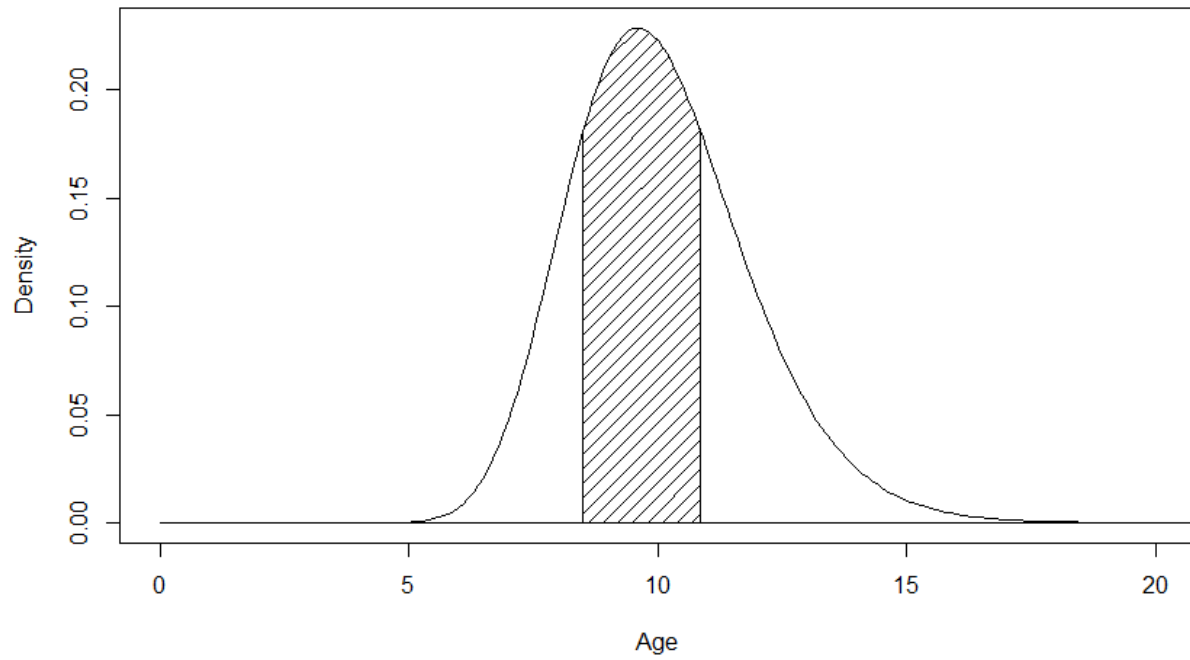
HPD female lower premolar 1 stage 9 (R 1/4), US Deaths 2008 prior



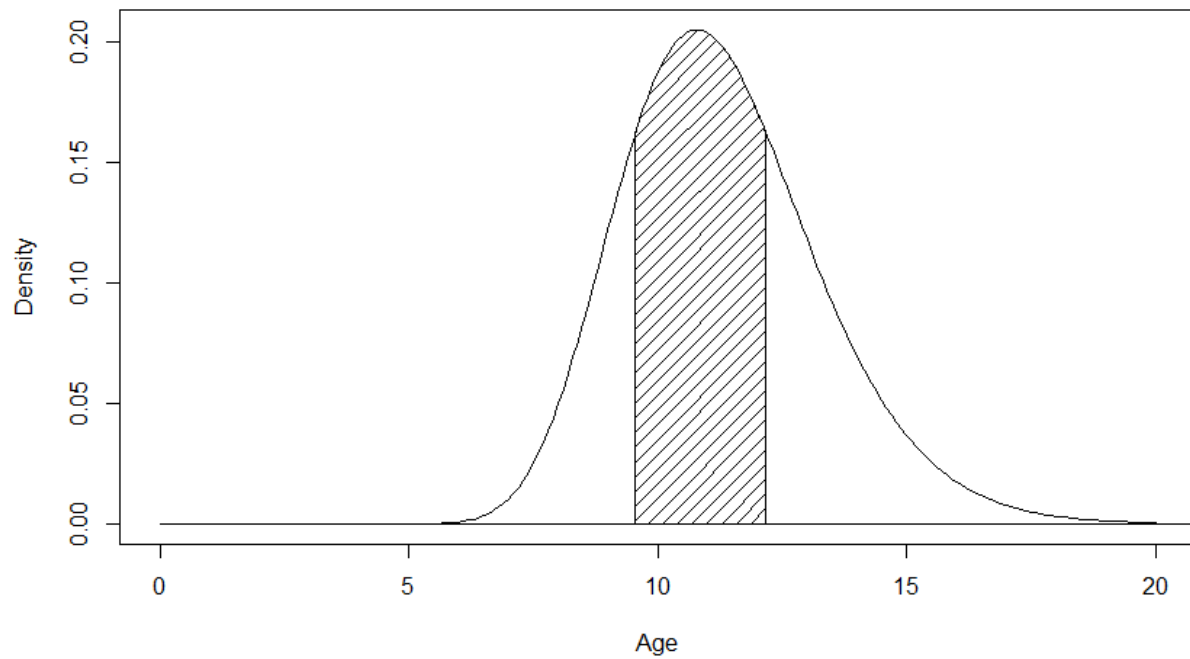
HPD female lower premolar 1 stage 10 (R 1/2), US Deaths 2008 prior



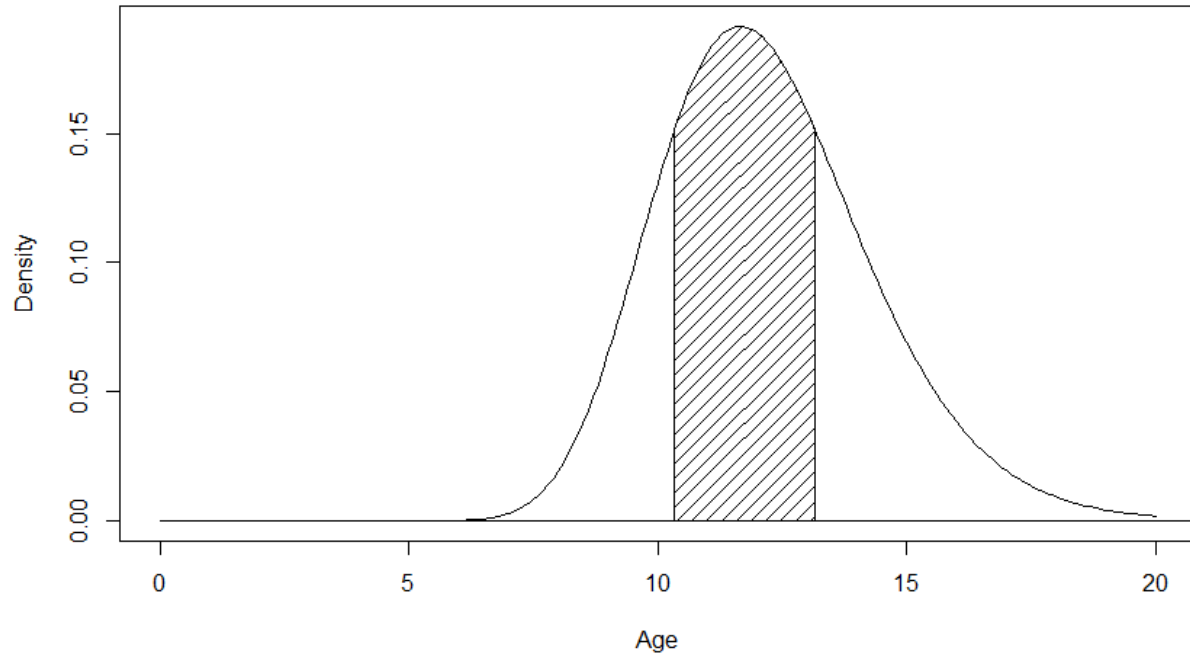
HPD female lower premolar 1 stage 11 (R 3/4), US Deaths 2008 prior



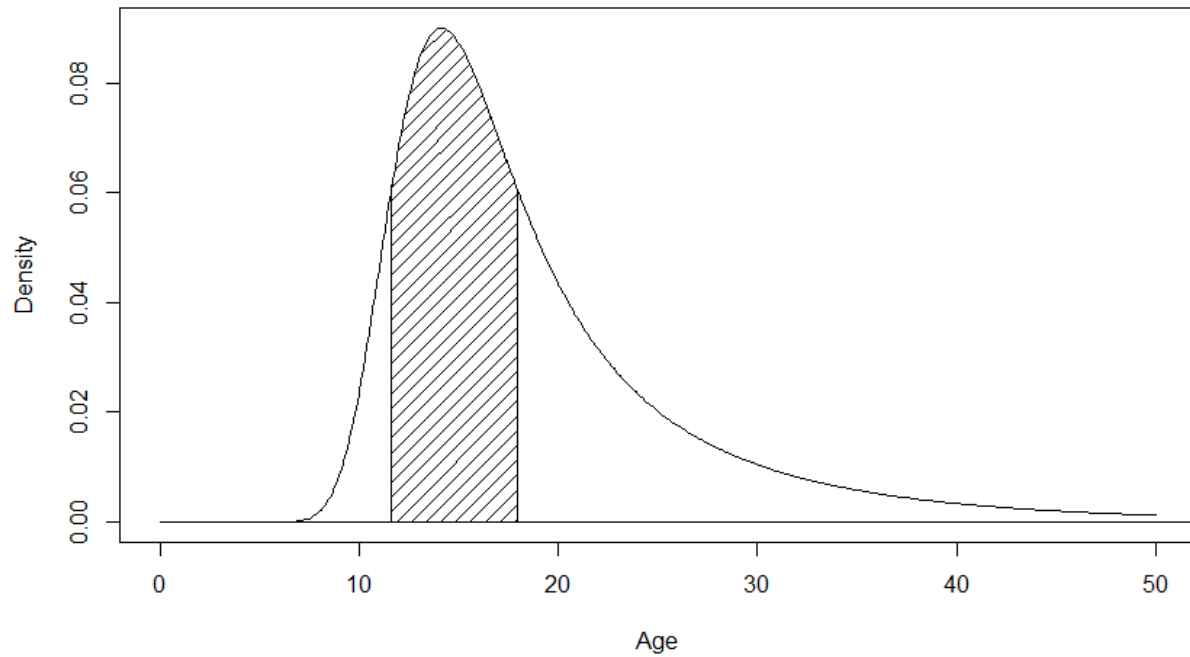
HPD female lower premolar 1 stage 12 (Rc), US Deaths 2008 prior



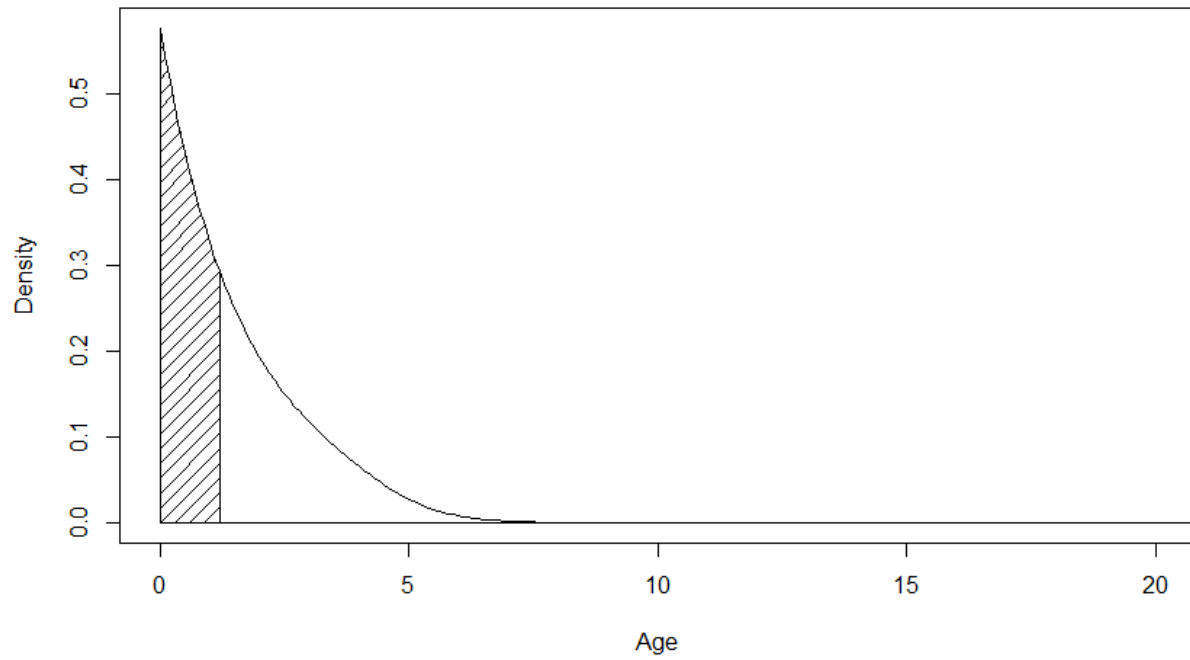
HPD female lower premolar 1 stage 13 (A 1/2), US Deaths 2008 prior



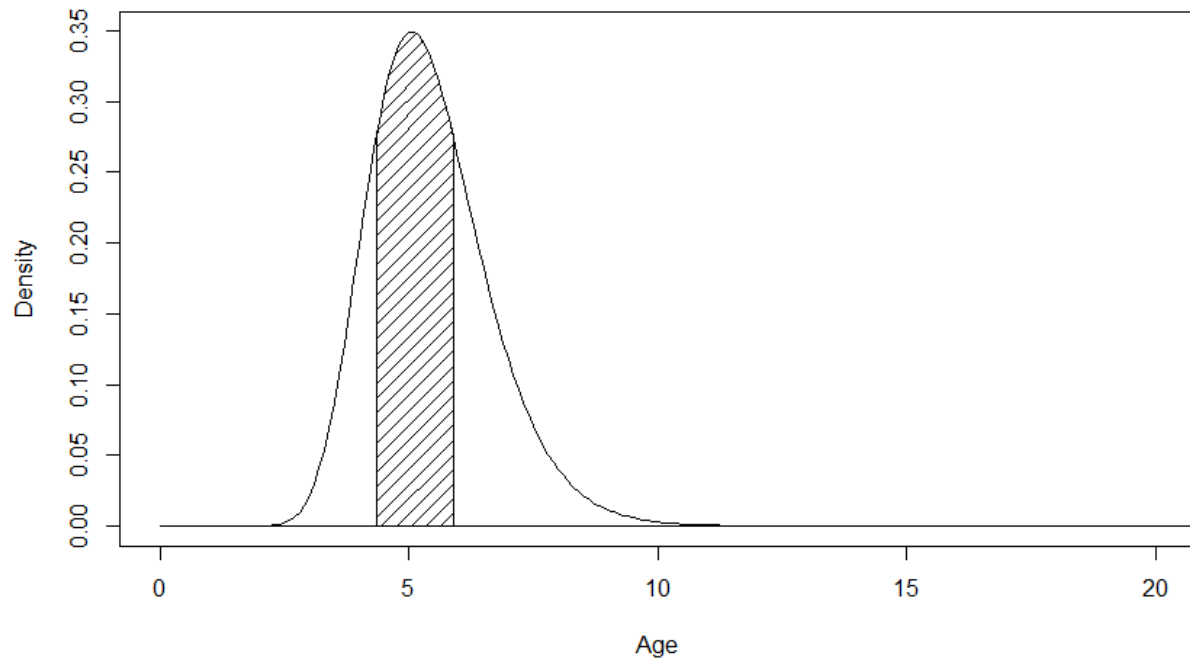
HPD female lower premolar 1 stage 14 (Ac), US Deaths 2008 prior



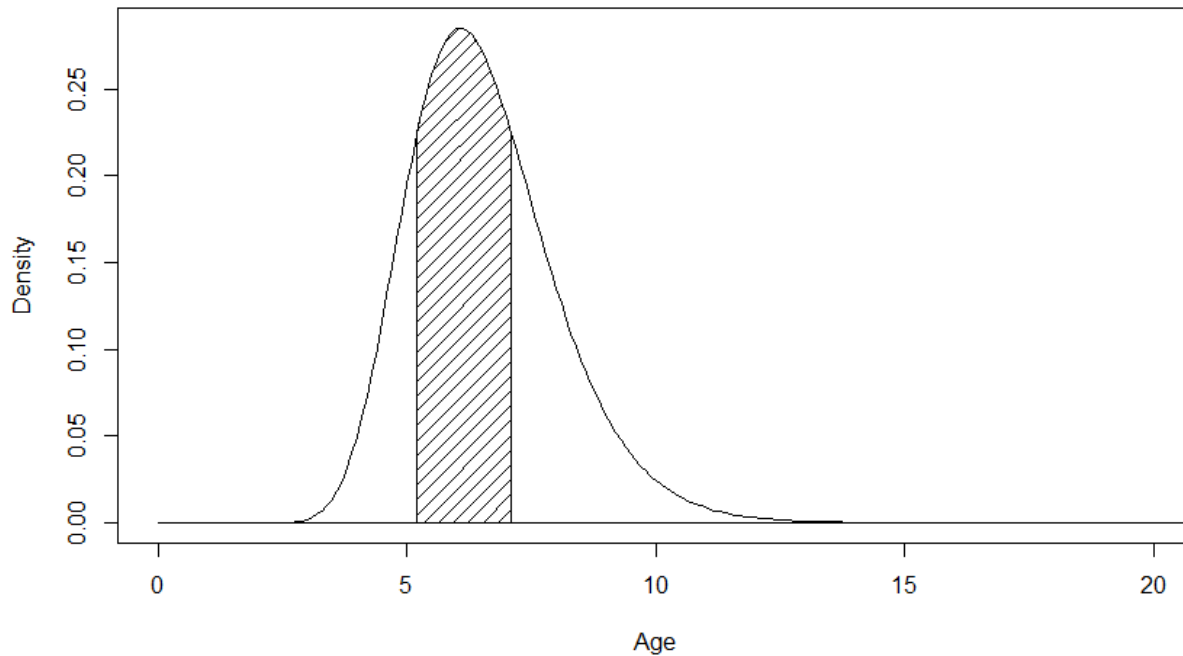
HPD female lower molar 1 stage 10 (R 1/2), US Deaths 2008 prior



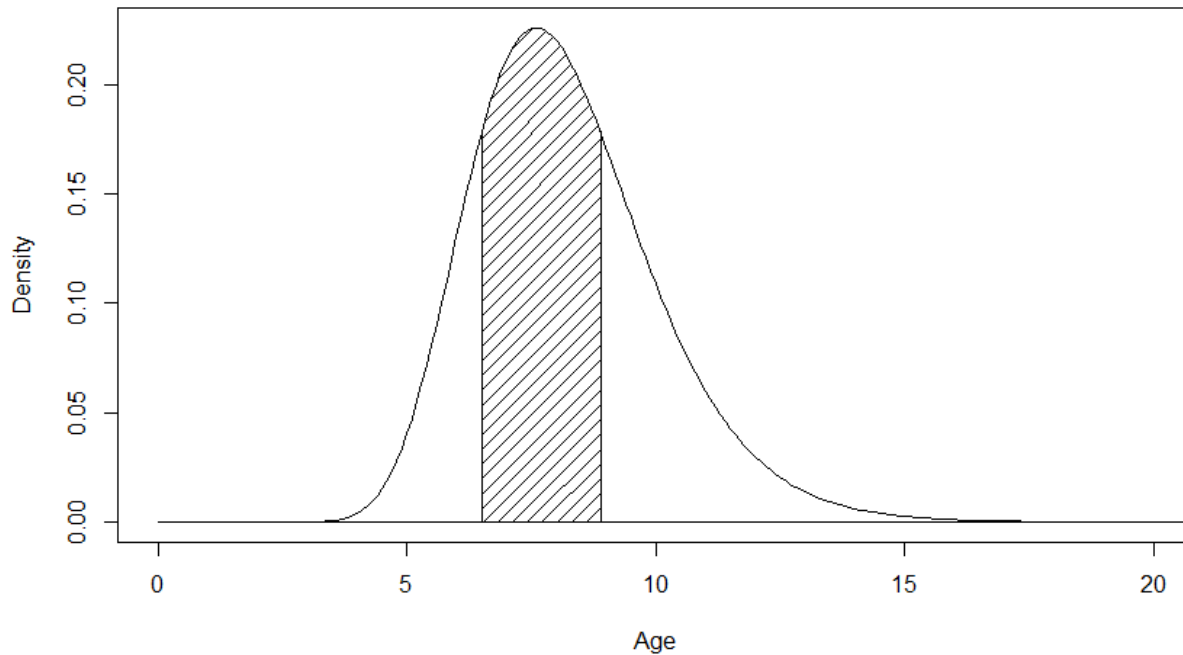
HPD female lower molar 1 stage 11 (R 3/4), US Deaths 2008 prior



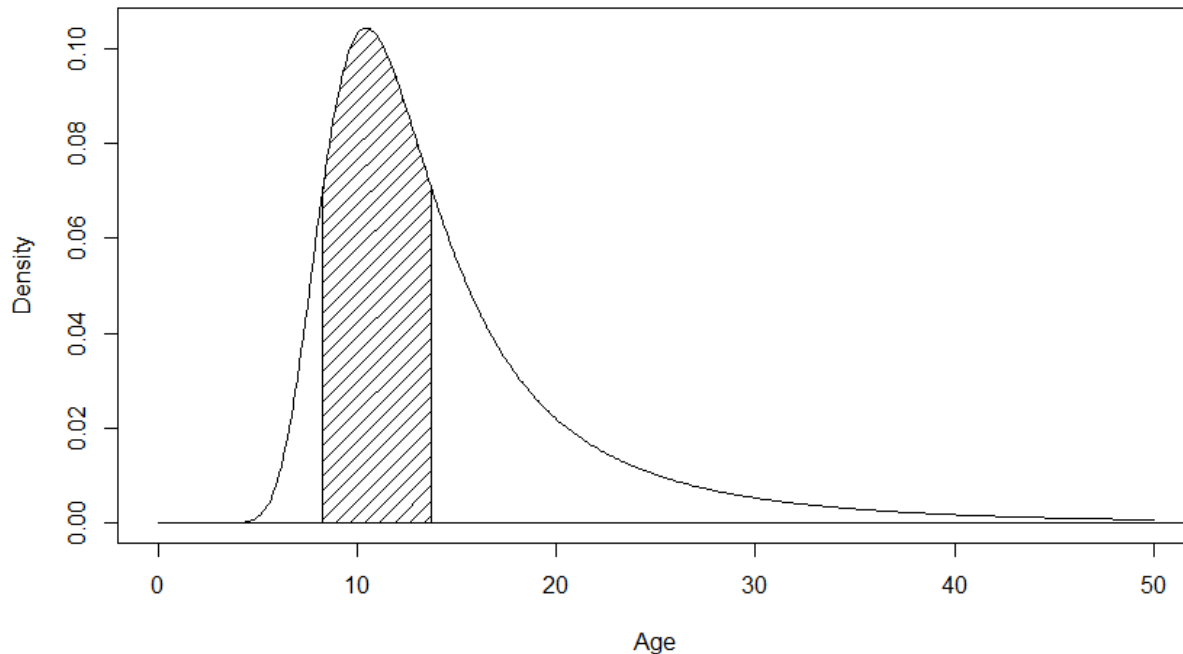
HPD female lower molar 1 stage 12 (Rc), US Deaths 2008 prior



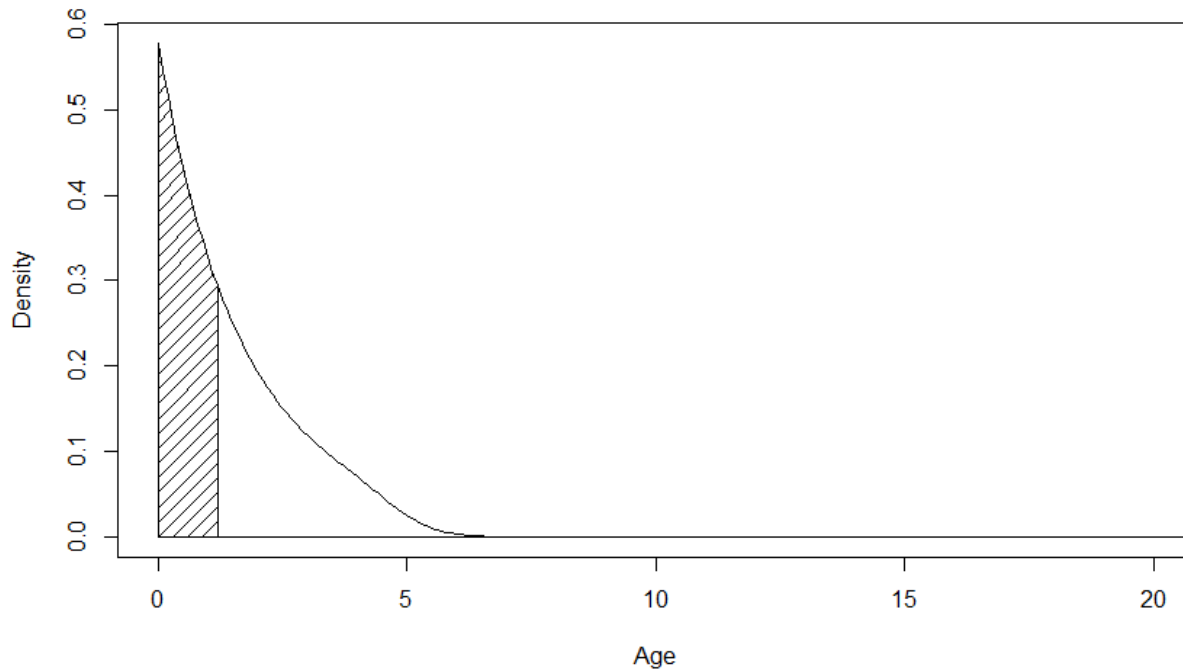
HPD female lower molar 1 stage 13 (A 1/2), US Deaths 2008 prior



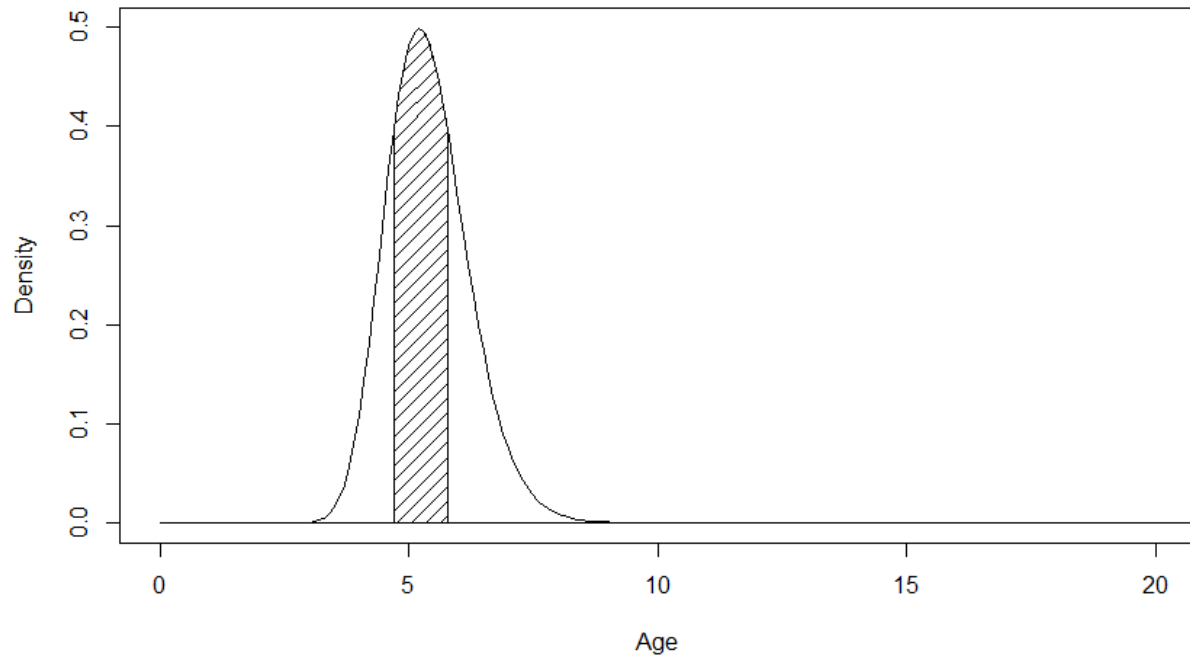
HPD female lower molar 1 stage 14 (Ac), US Deaths 2008 prior



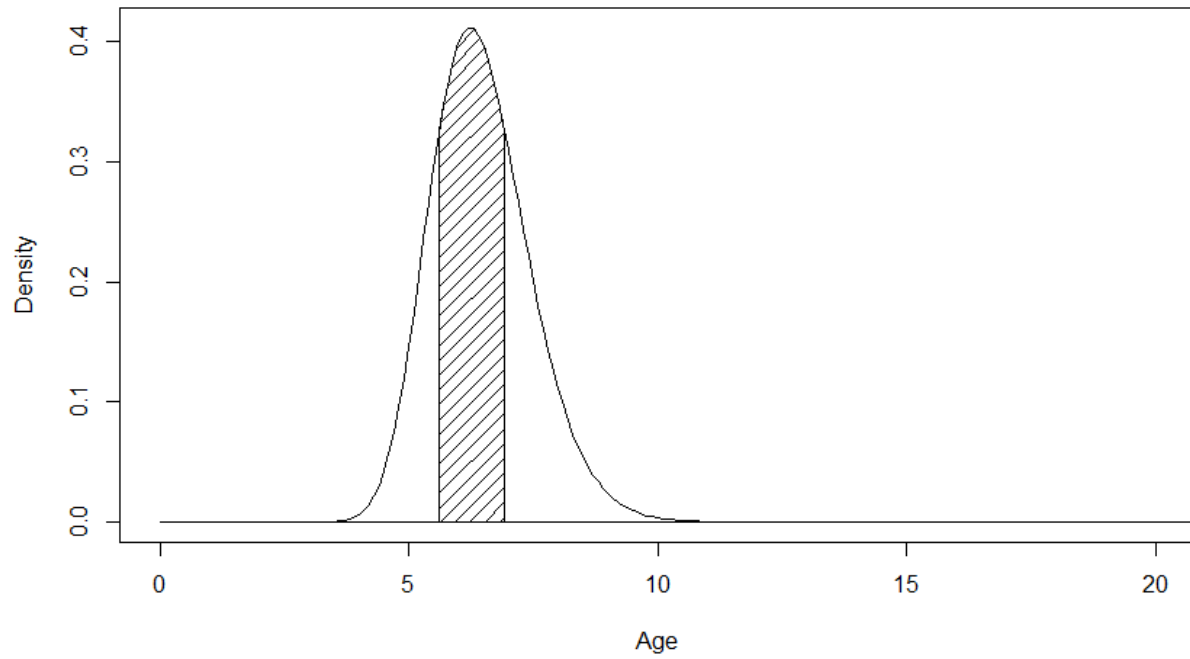
HPD female lower molar 2 stage 3 (Coc), US Deaths 2008 prior



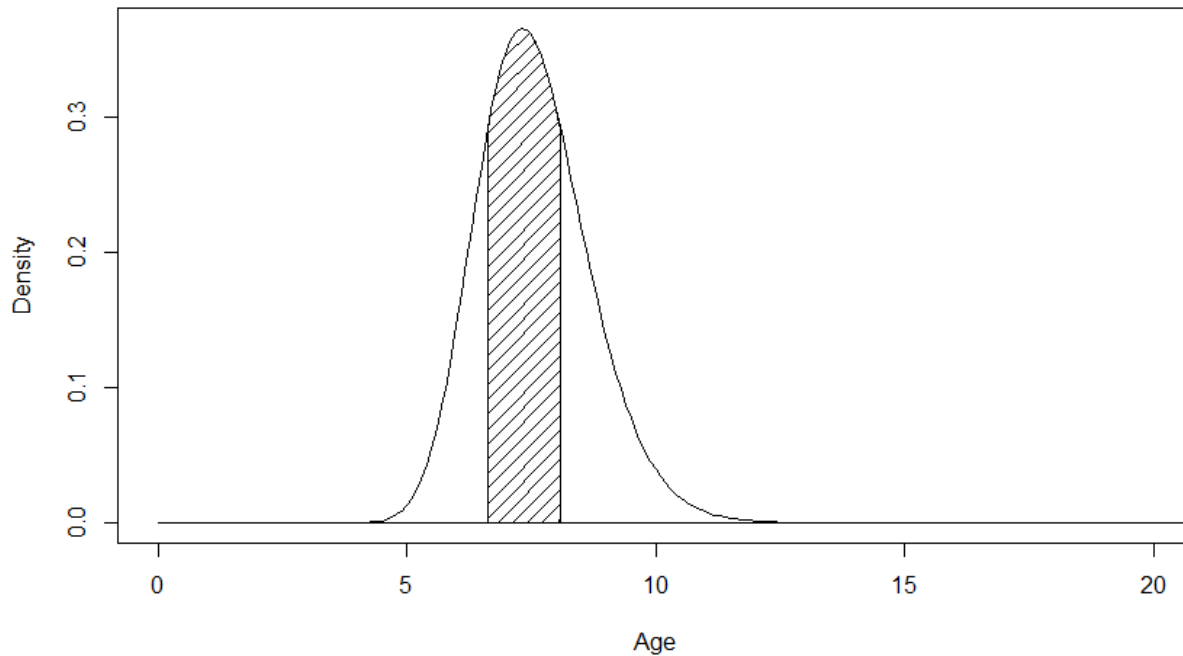
HPD female lower molar 2 stage 5 (Cr 3/4), US Deaths 2008 prior



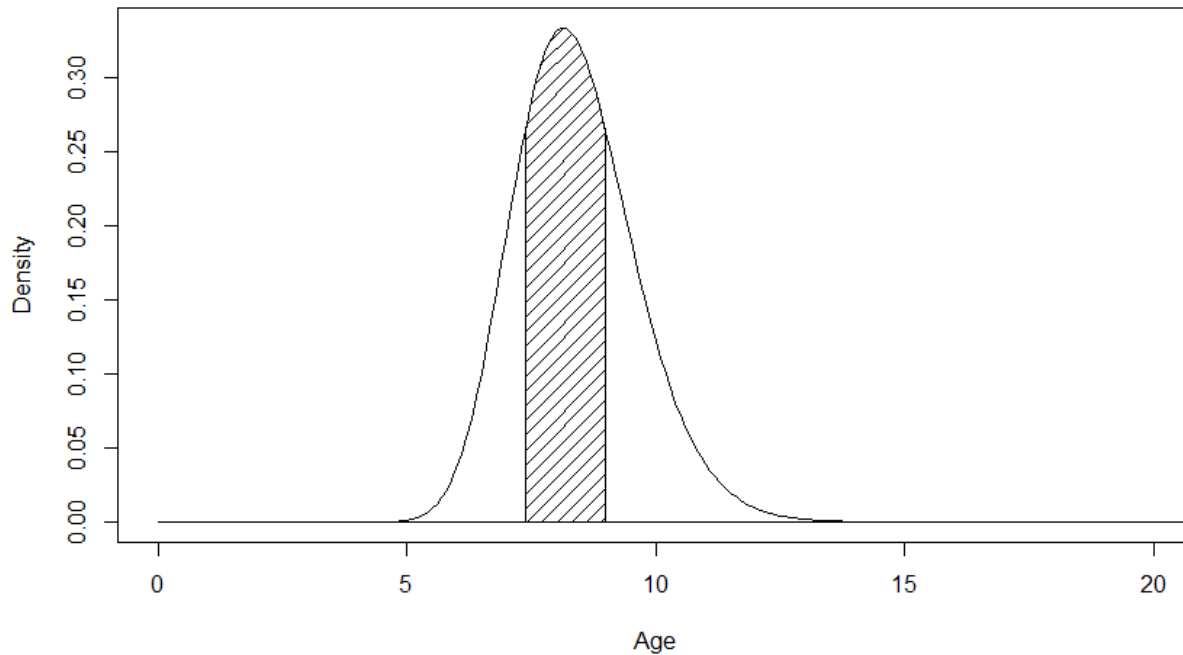
HPD female lower molar 2 stage 6 (Crc), US Deaths 2008 prior



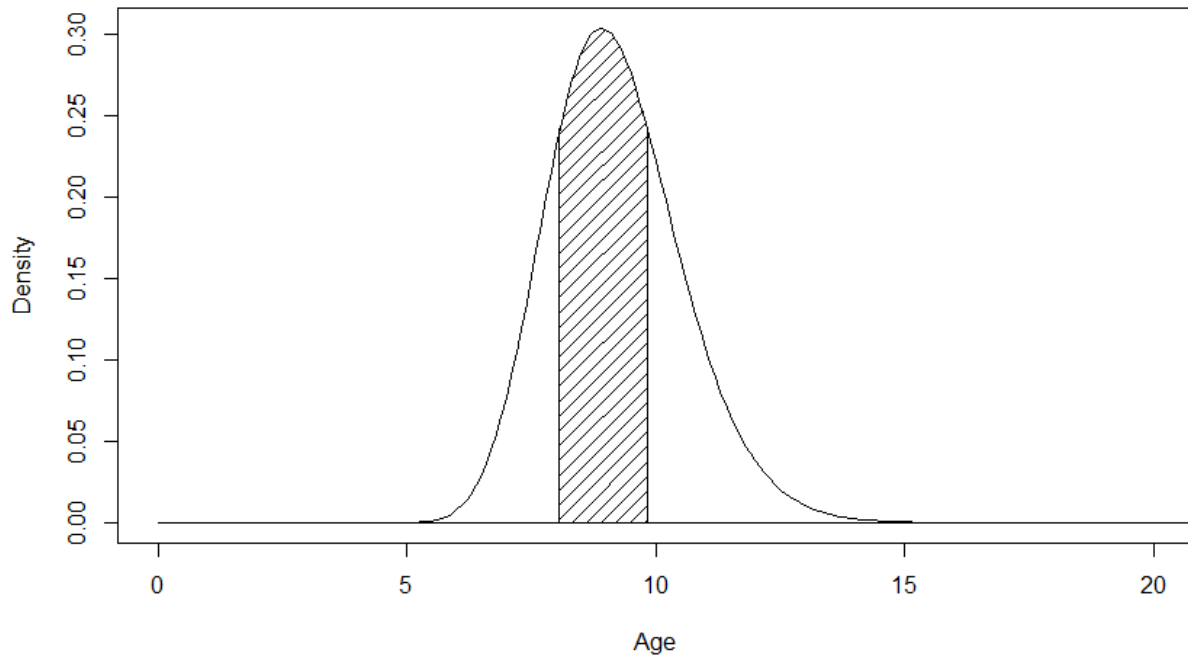
HPD female lower molar 2 stage 7 (Ri), US Deaths 2008 prior



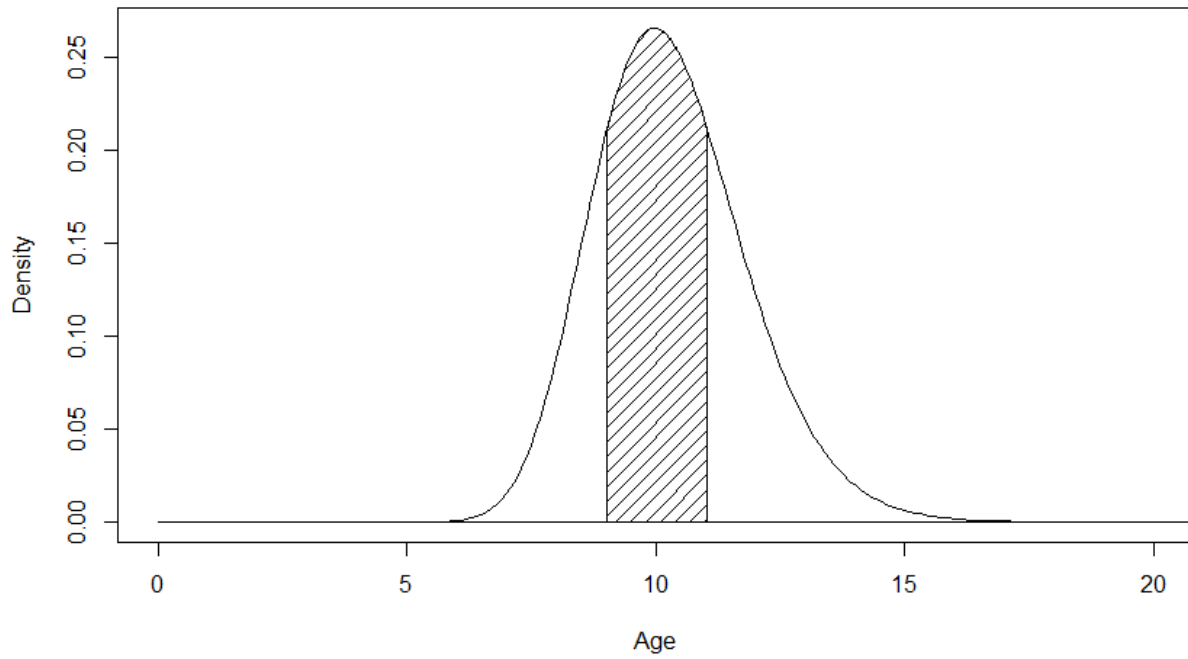
HPD female lower molar 2 stage 8 (C1i), US Deaths 2008 prior



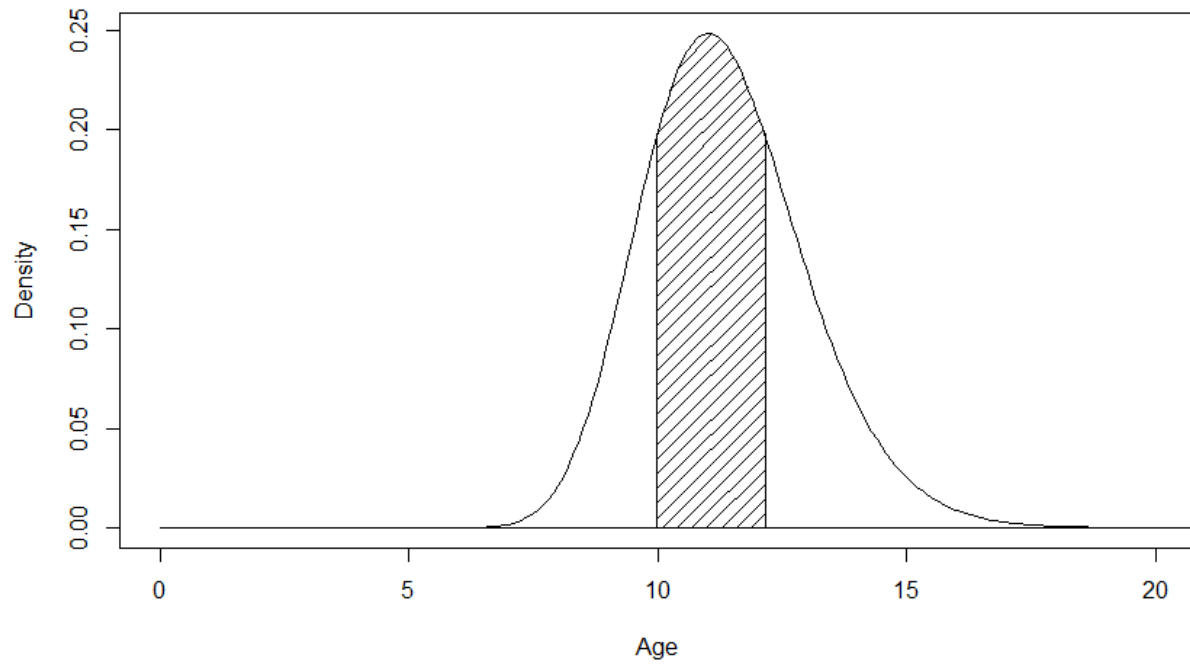
HPD female lower molar 2 stage 9 (R 1/4), US Deaths 2008 prior



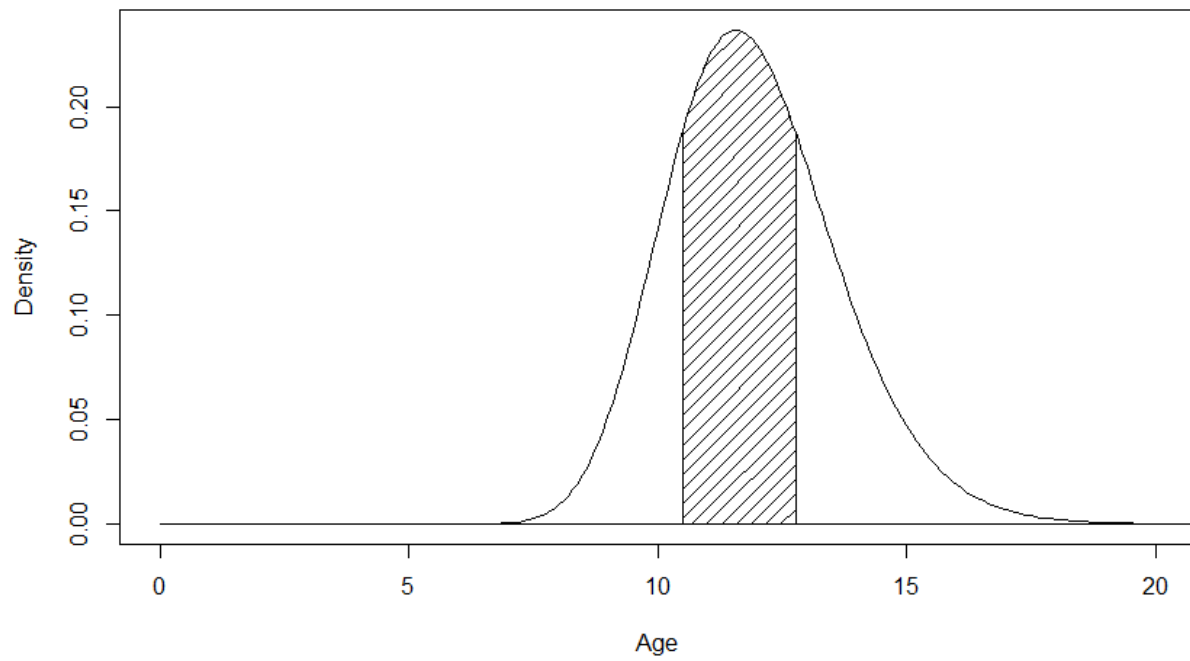
HPD female lower molar 2 stage 10 (R 1/2), US Deaths 2008 prior



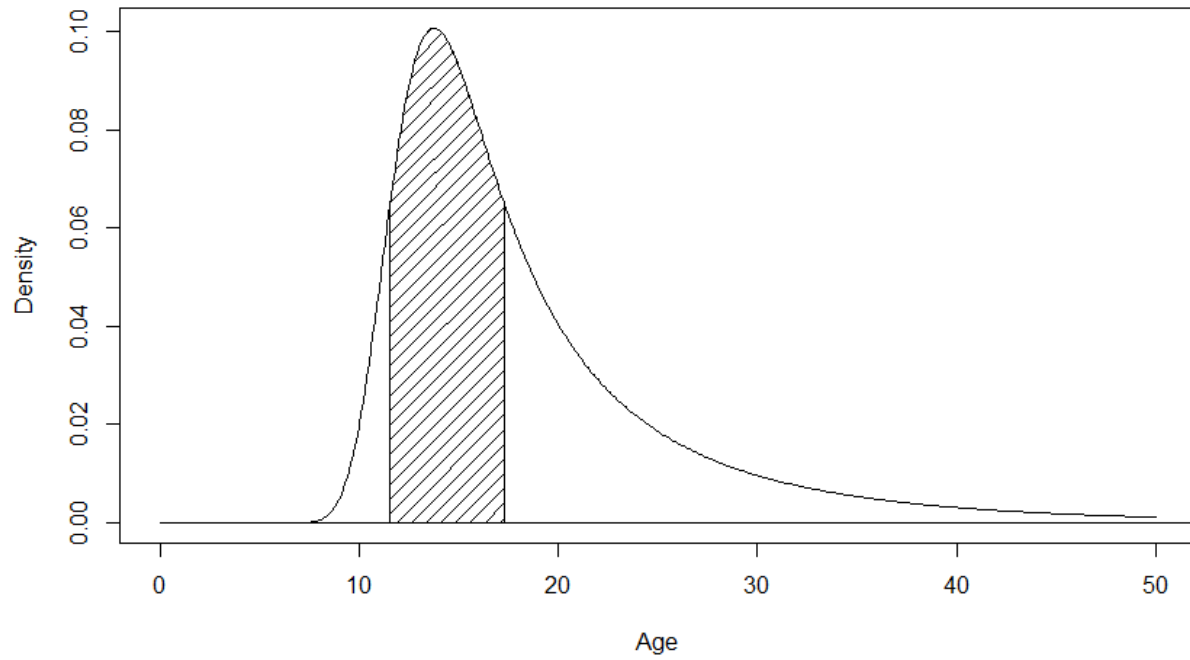
HPD female lower molar 2 stage 11 (R 3/4), US Deaths 2008 prior



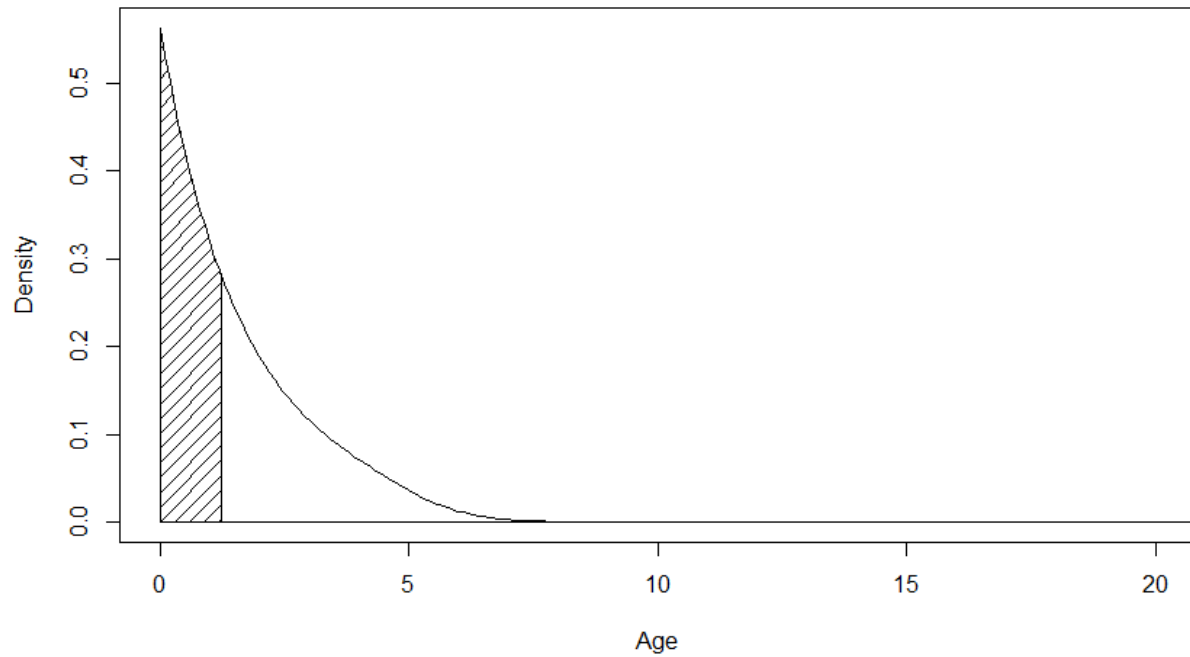
HPD female lower molar 2 stage 12 (Rc), US Deaths 2008 prior



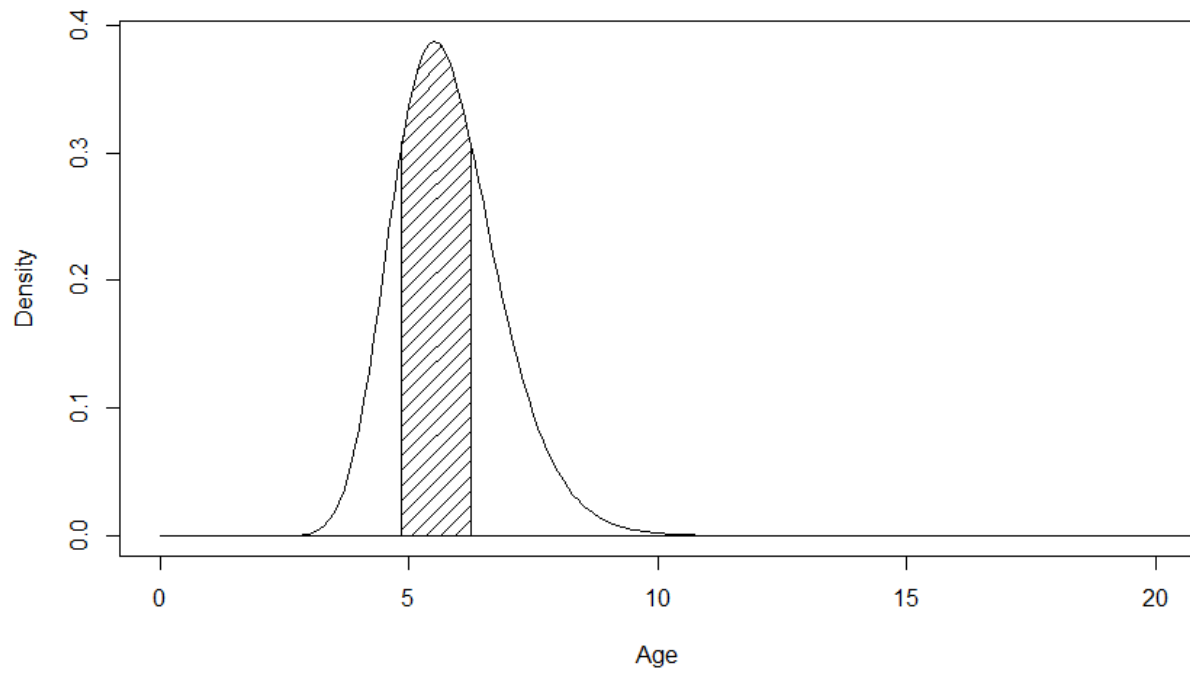
HPD female lower molar 2 stage 13 (A 1/2), US Deaths 2008 prior



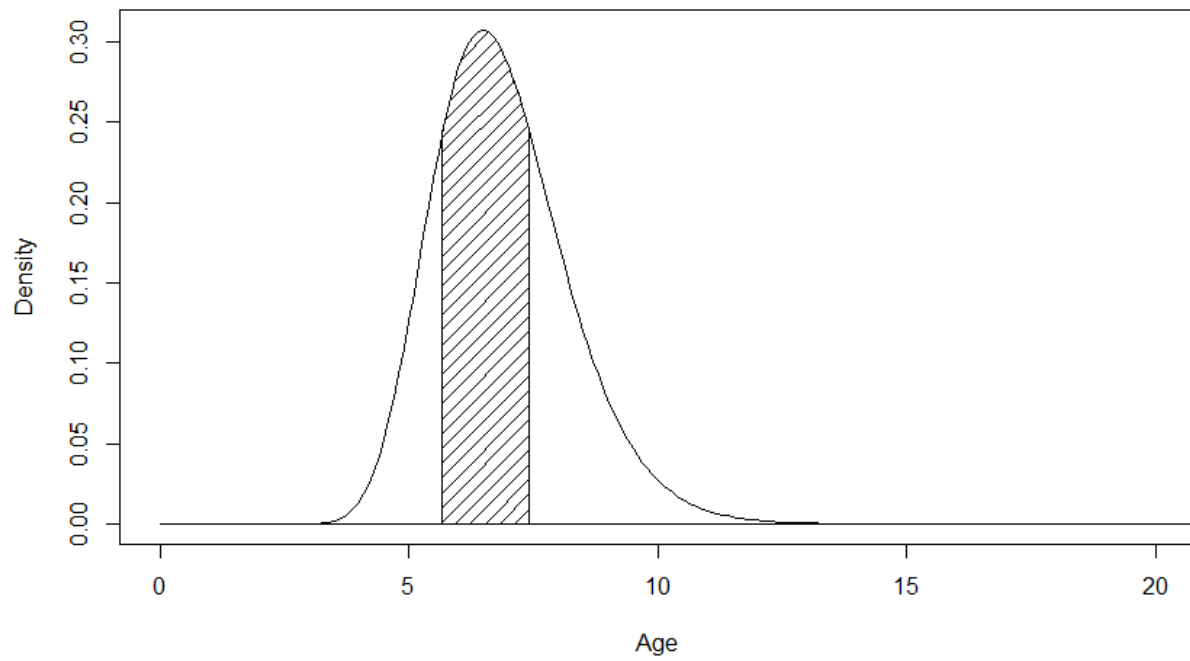
HPD female lower premolar 2 stage 5 (Cr 3/4), US Deaths 2008 prior



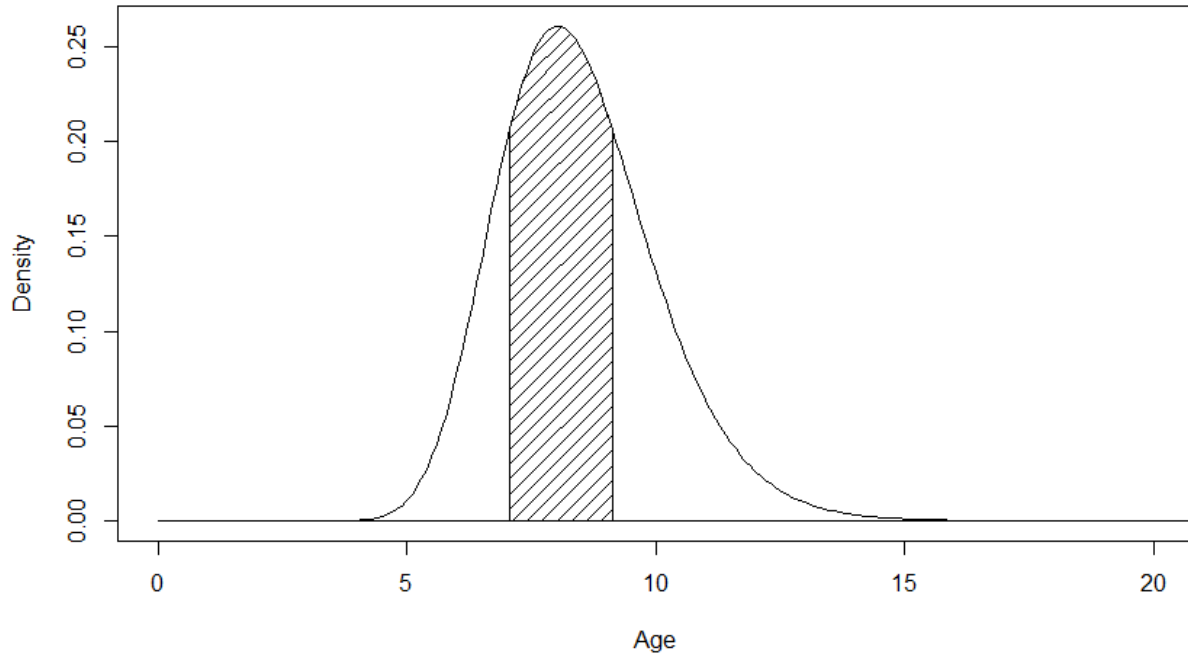
HPD female lower premolar 2 stage 6 (Crc), US Deaths 2008 prior



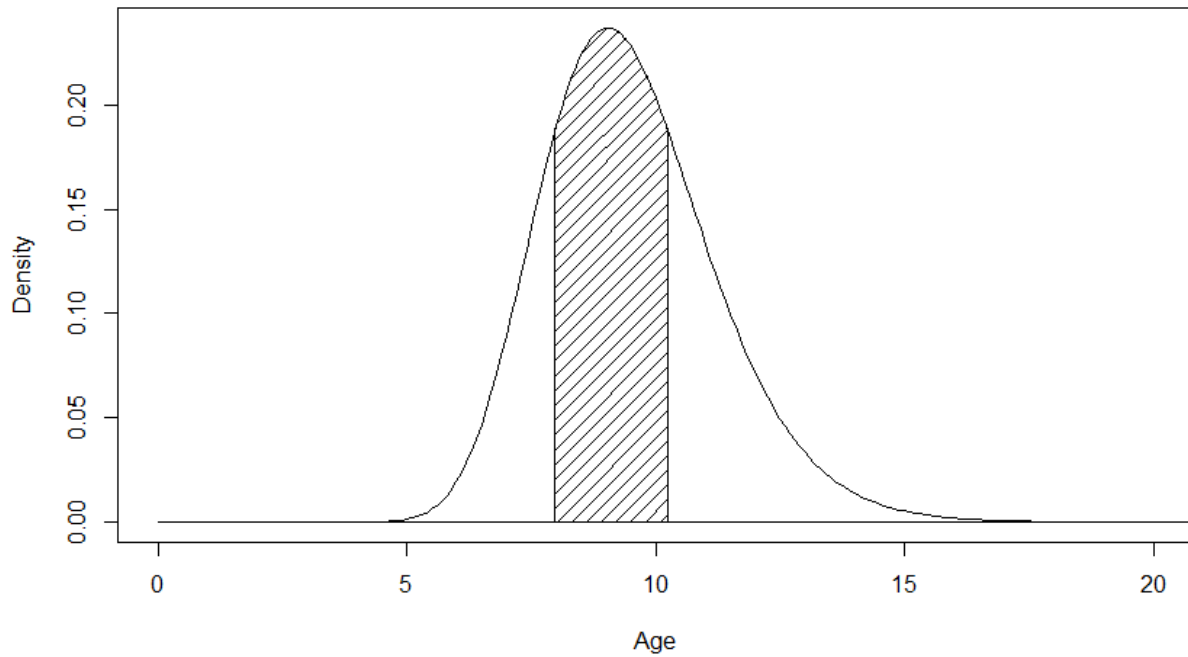
HPD female lower premolar 2 stage 7 (Ri), US Deaths 2008 prior



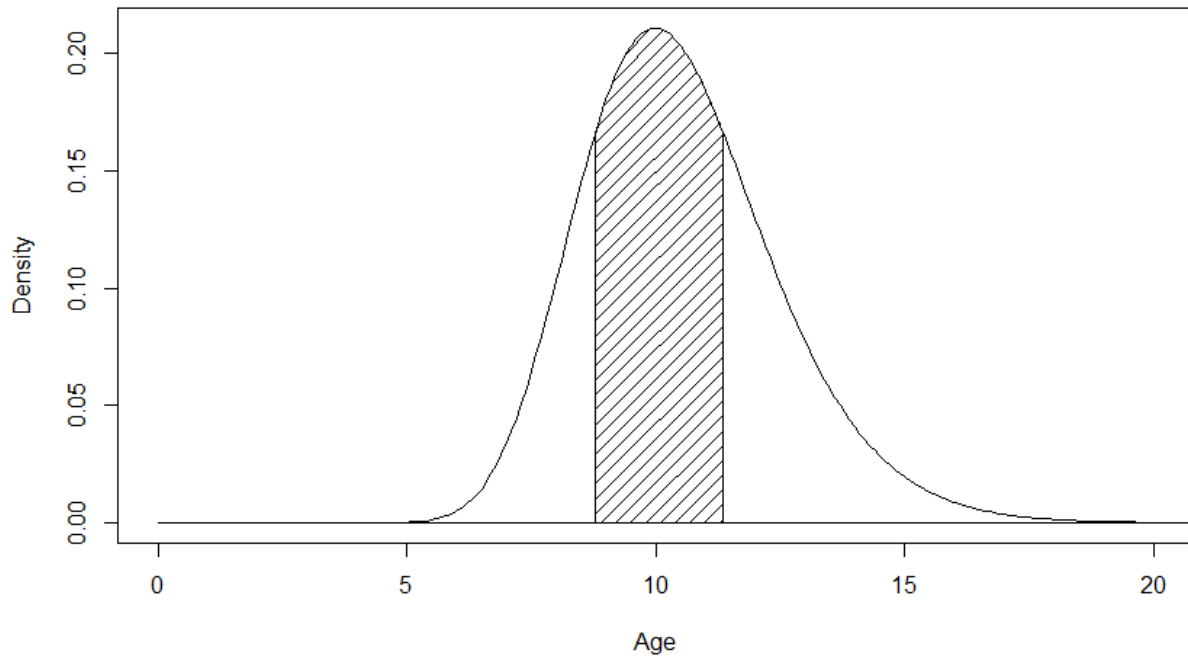
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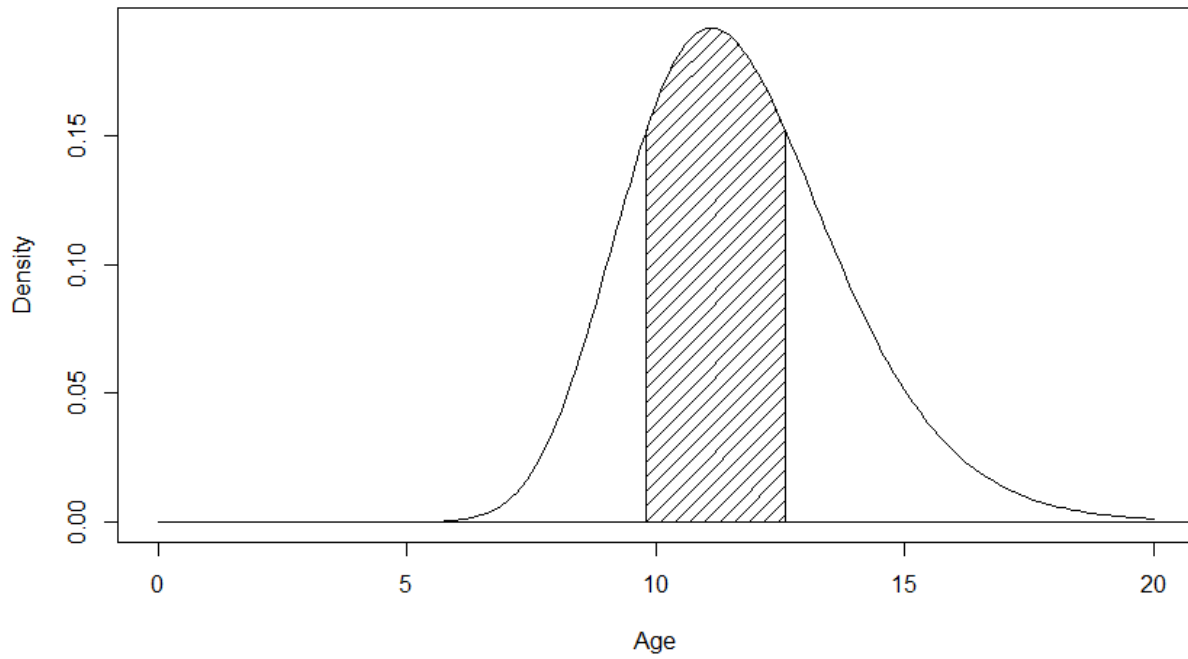
HPD female lower premolar 2 stage 10 (R 1/2), US Deaths 2008 prior



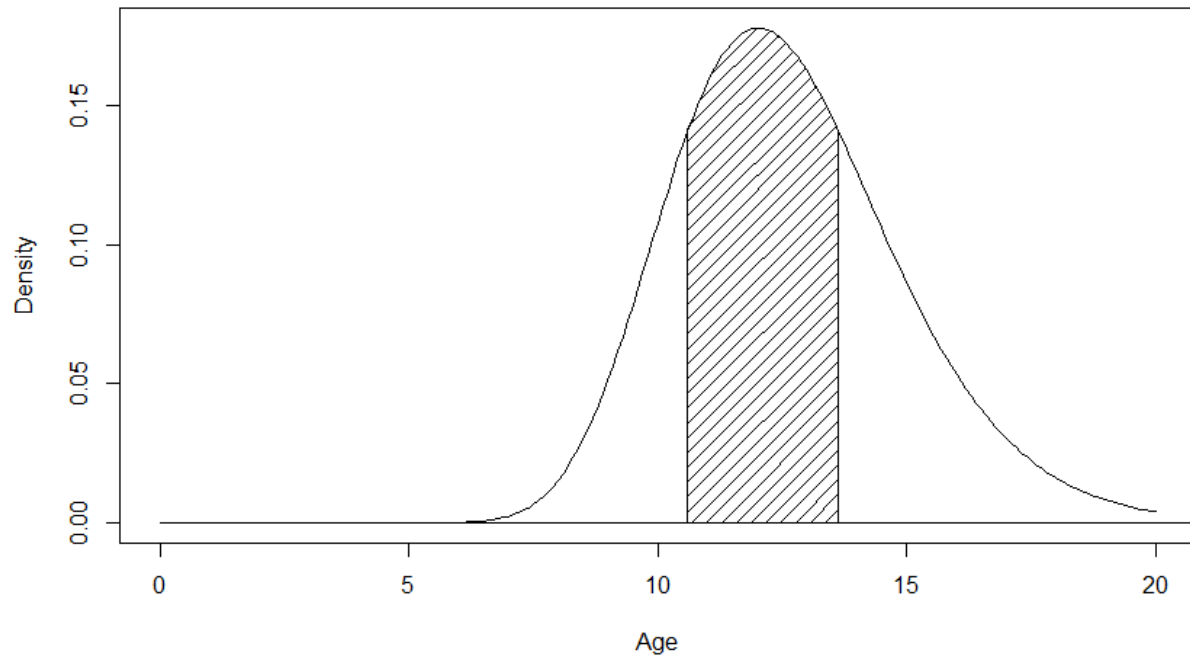
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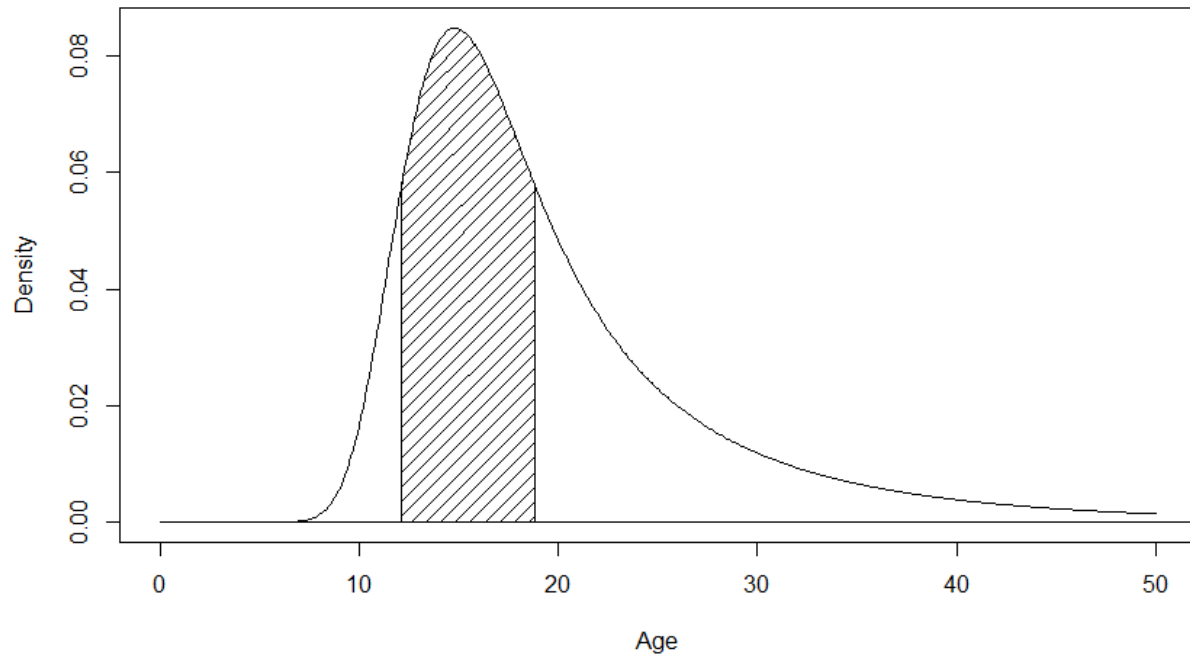
HPD female lower premolar 2 stage 12 (Rc), US Deaths 2008 prior



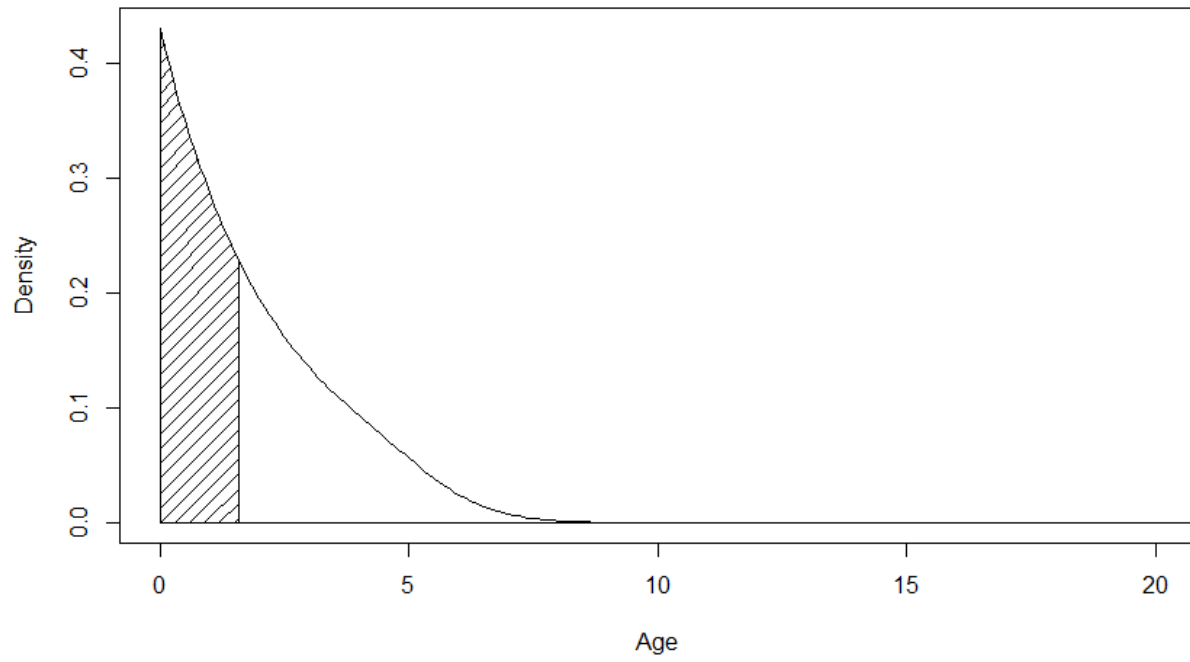
HPD female lower premolar 2 stage 13 (A 1/2), US Deaths 2008 prior



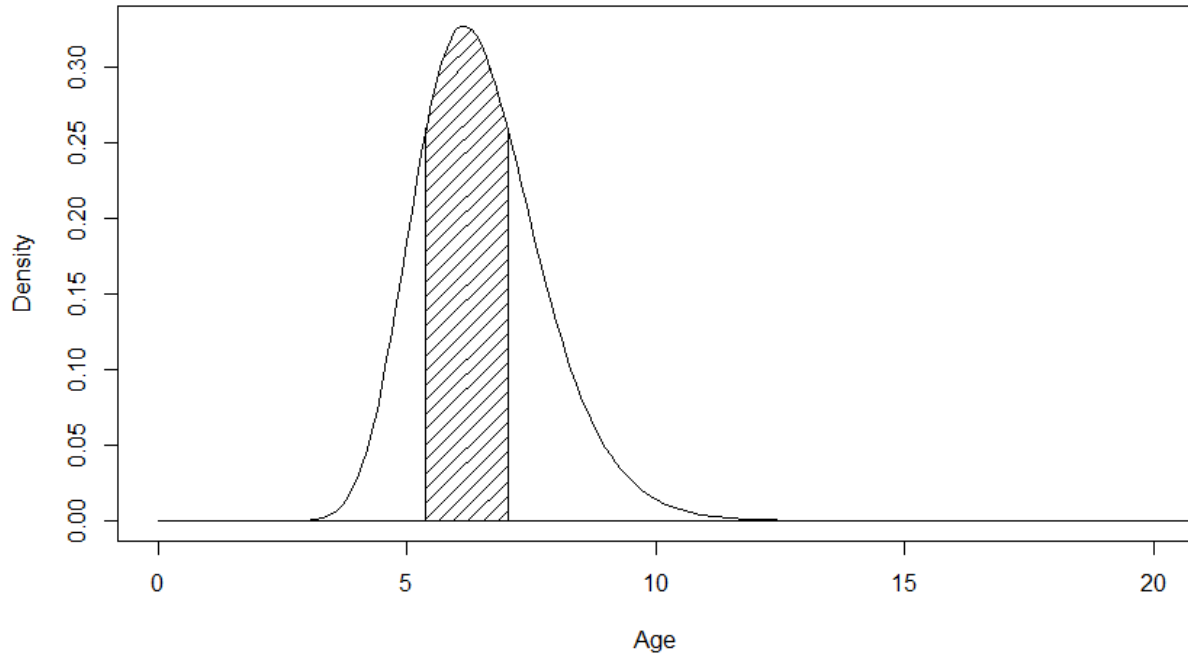
HPD female lower premolar 2 stage 14 (Ac), US Deaths 2008 prior



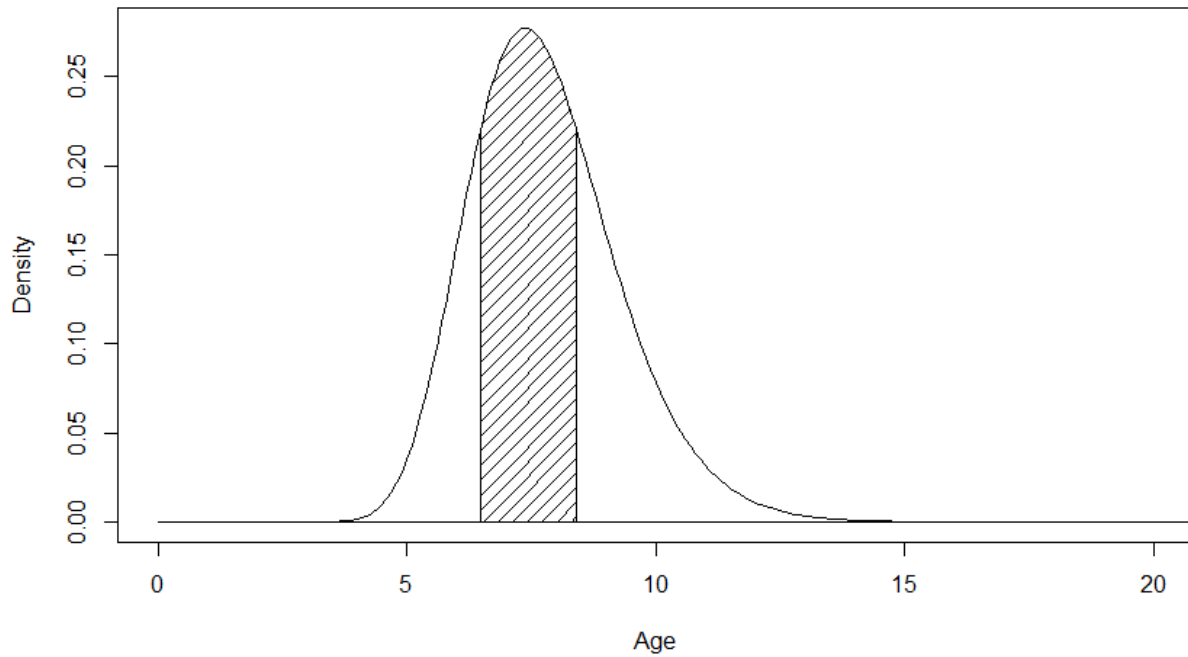
HPD male lower incisor 1 stage 11 (R3/4), US Violent Deaths 2010 prior



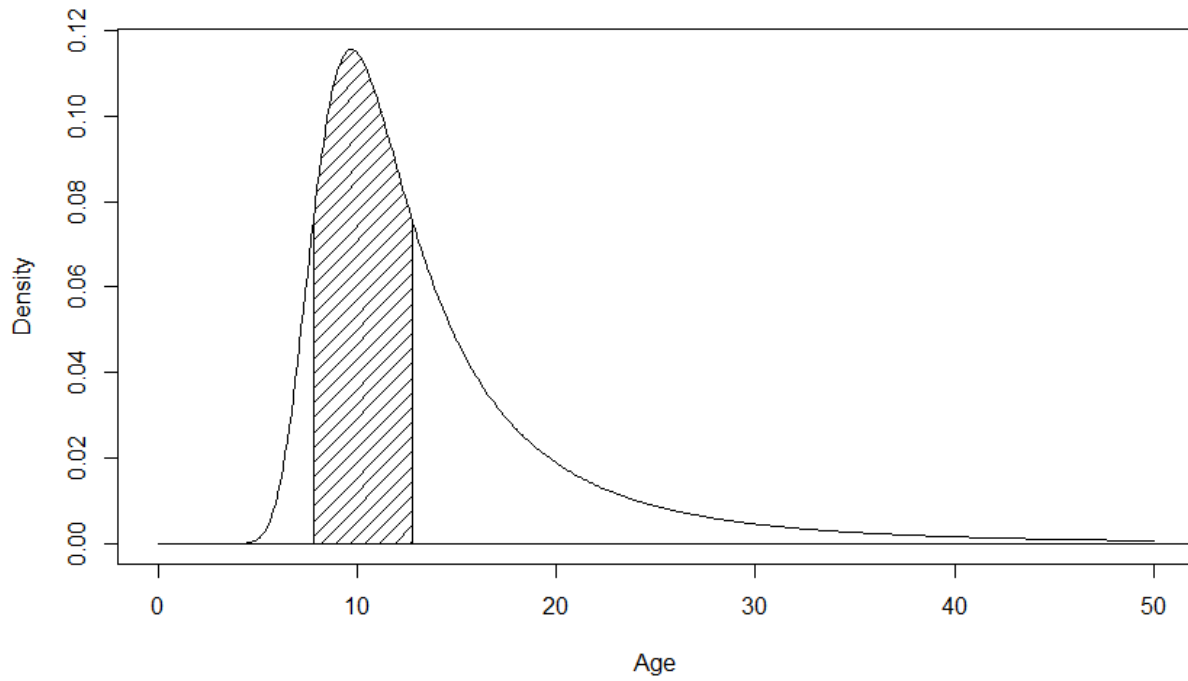
HPD male lower incisor 1 stage 12 (Rc), US Violent Deaths 2010 prior



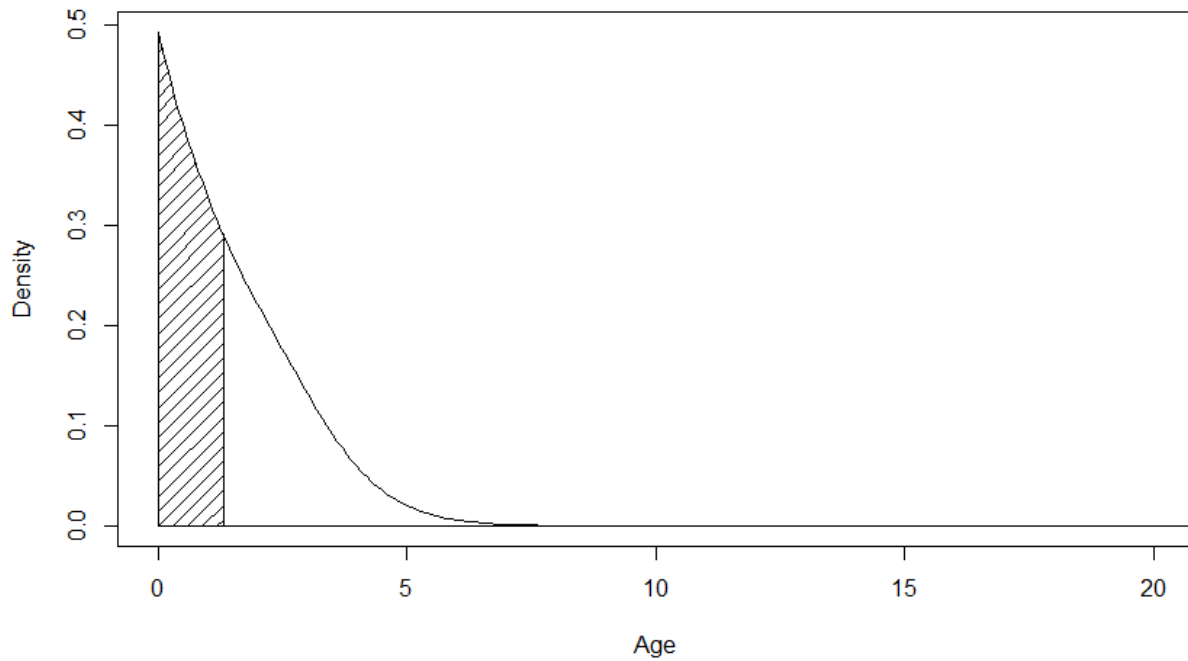
HPD male lower incisor 1 stage 13 (A1/2), US Violent Deaths 2010 prior



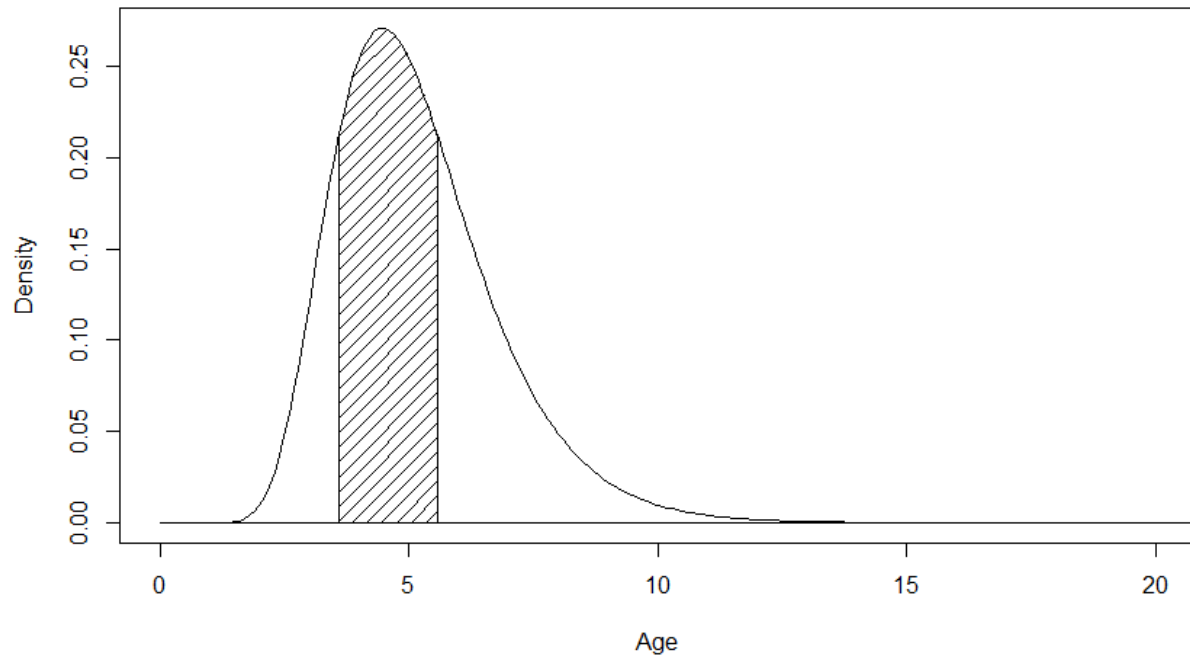
HPD male lower incisor 1 stage 14 (Ac), US Violent Deaths 2010 prior



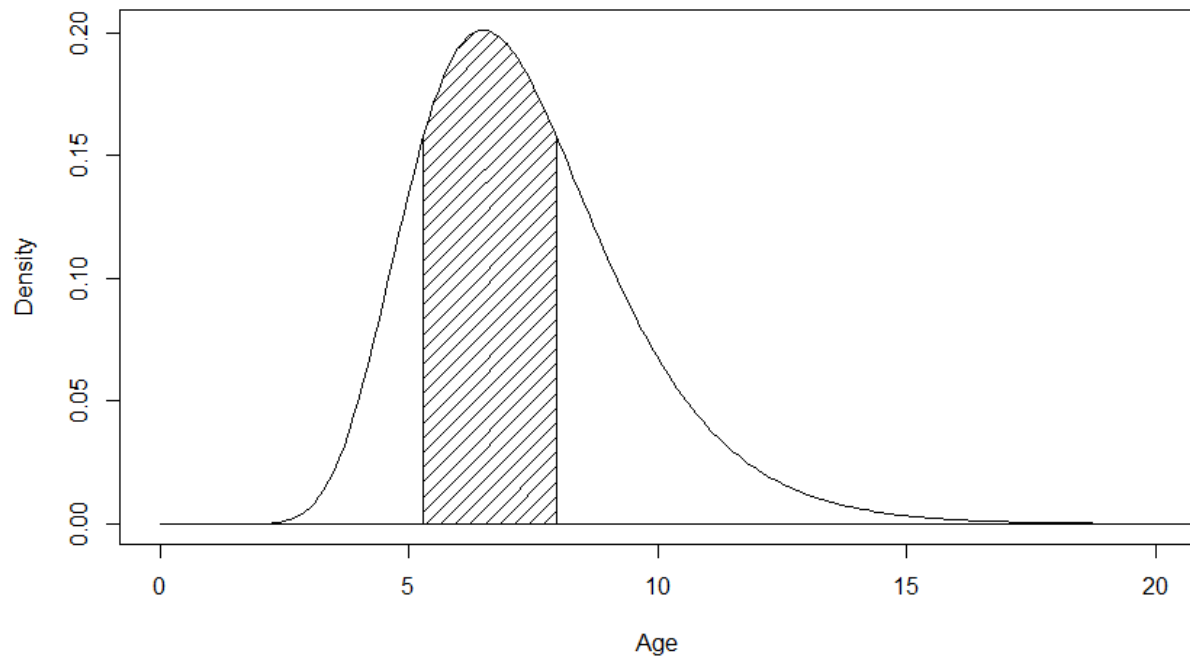
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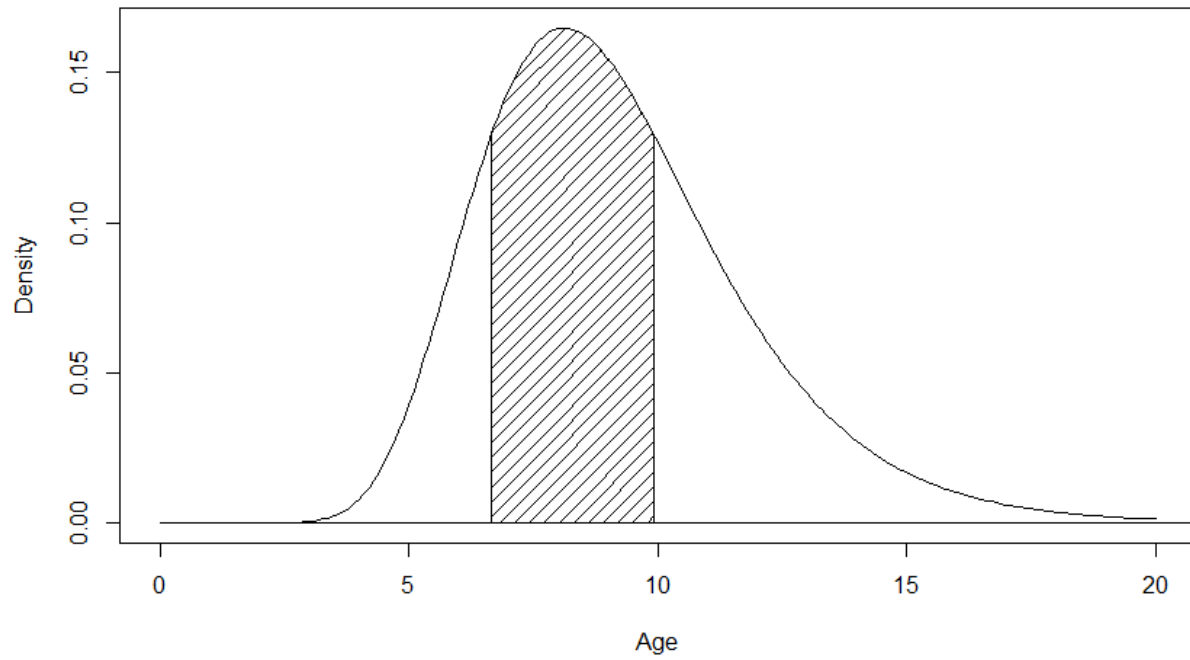
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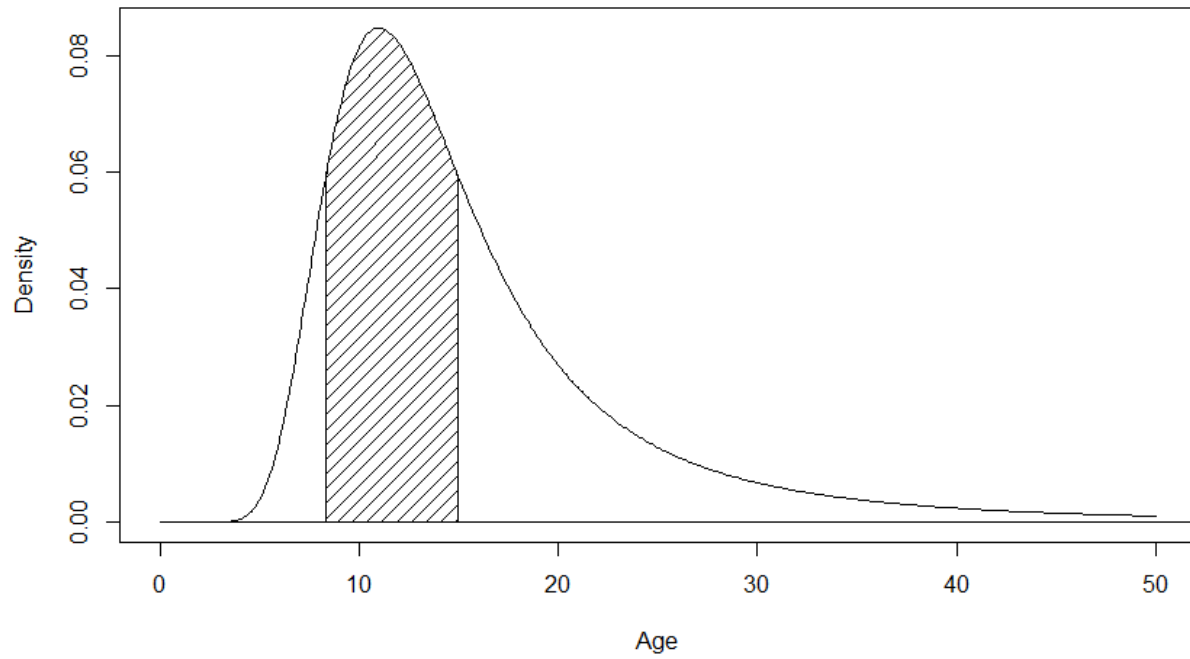
HPD male upper incisor 1 stage 12 (Rc), US Violent Deaths 2010 prior



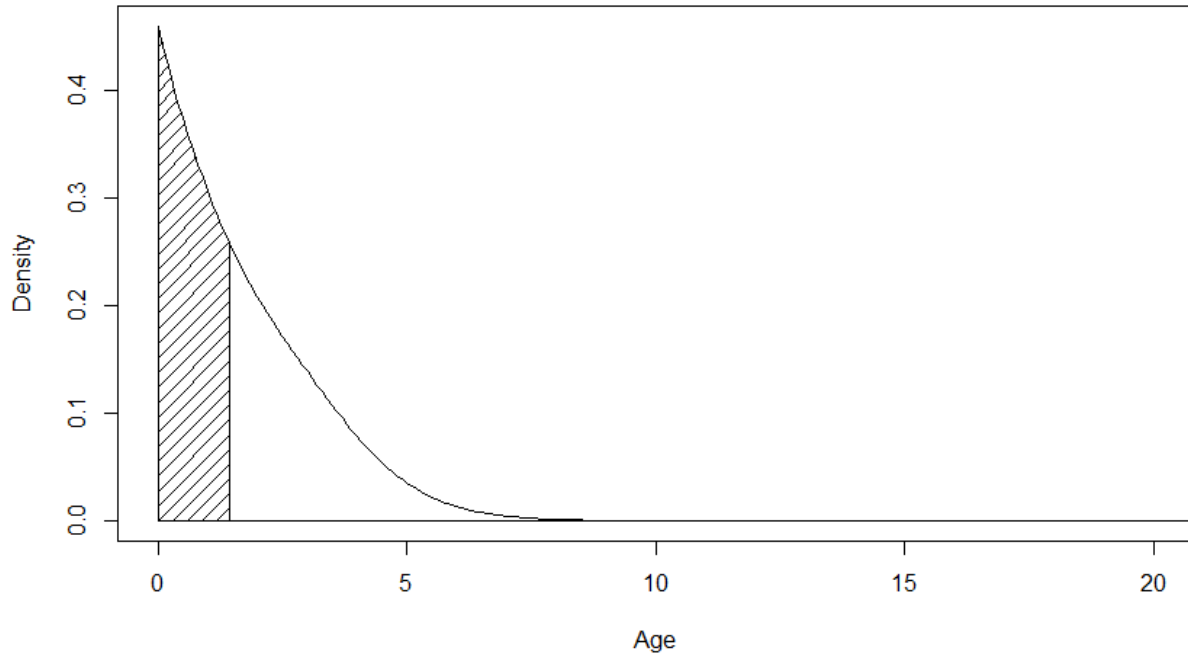
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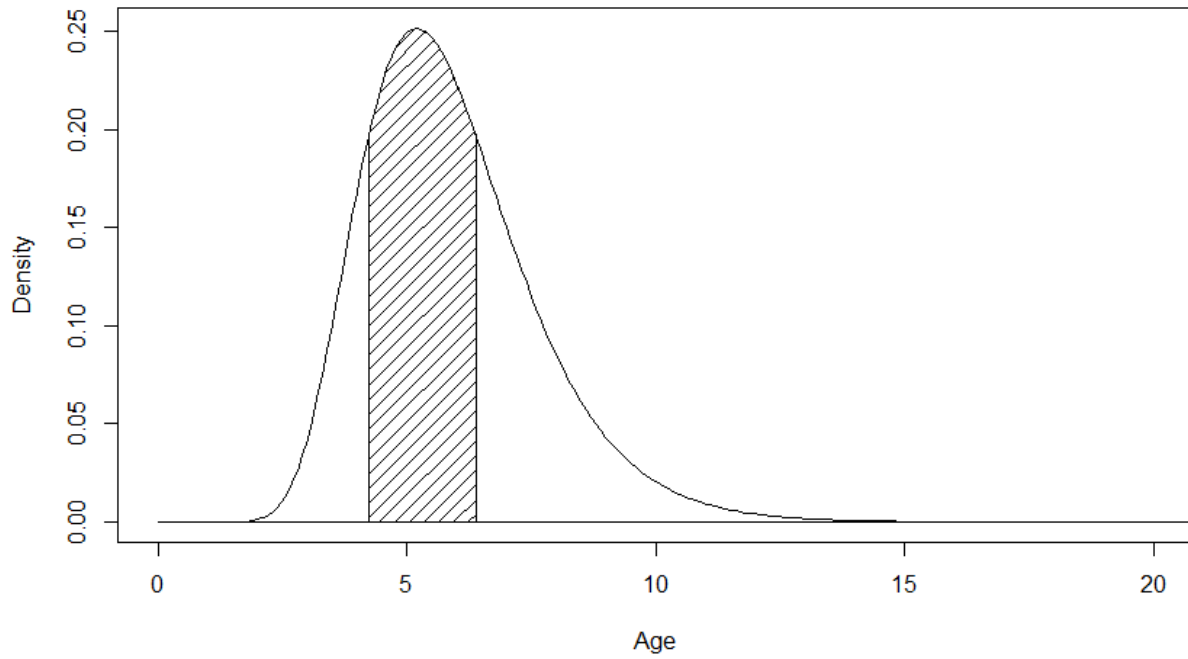
HPD male upper incisor 1 stage 14 (Ac), US Violent Deaths 2010 prior



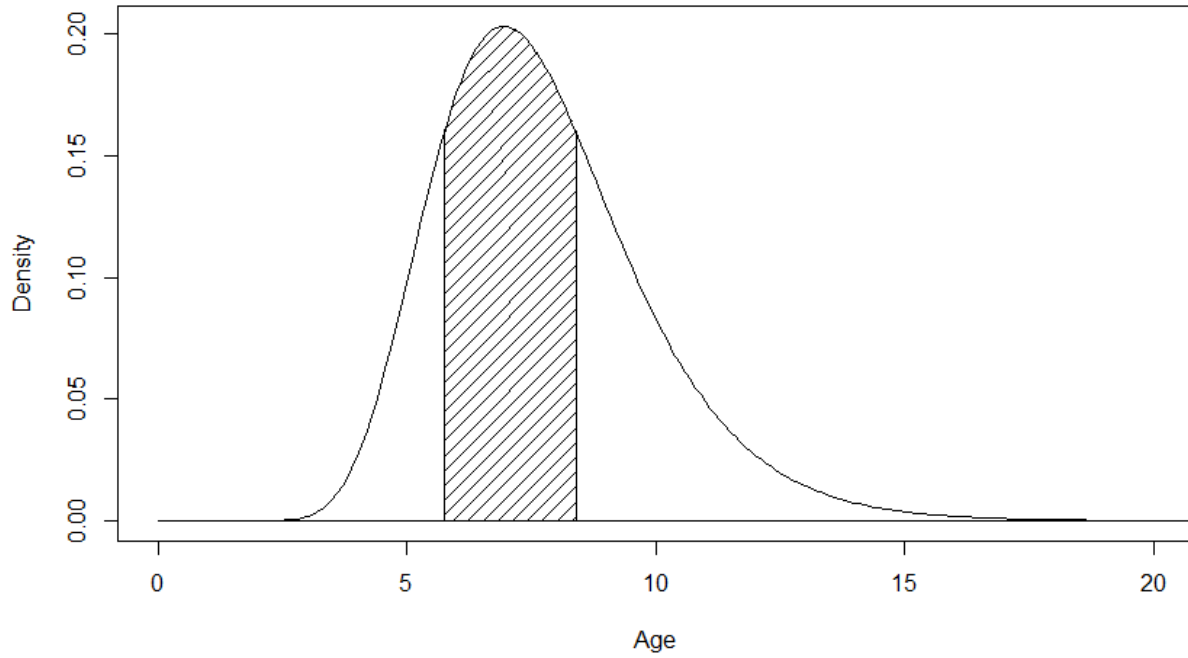
HPD male upper incisor 2 stage 9 (R1/4), US Violent Deaths 2010 prior



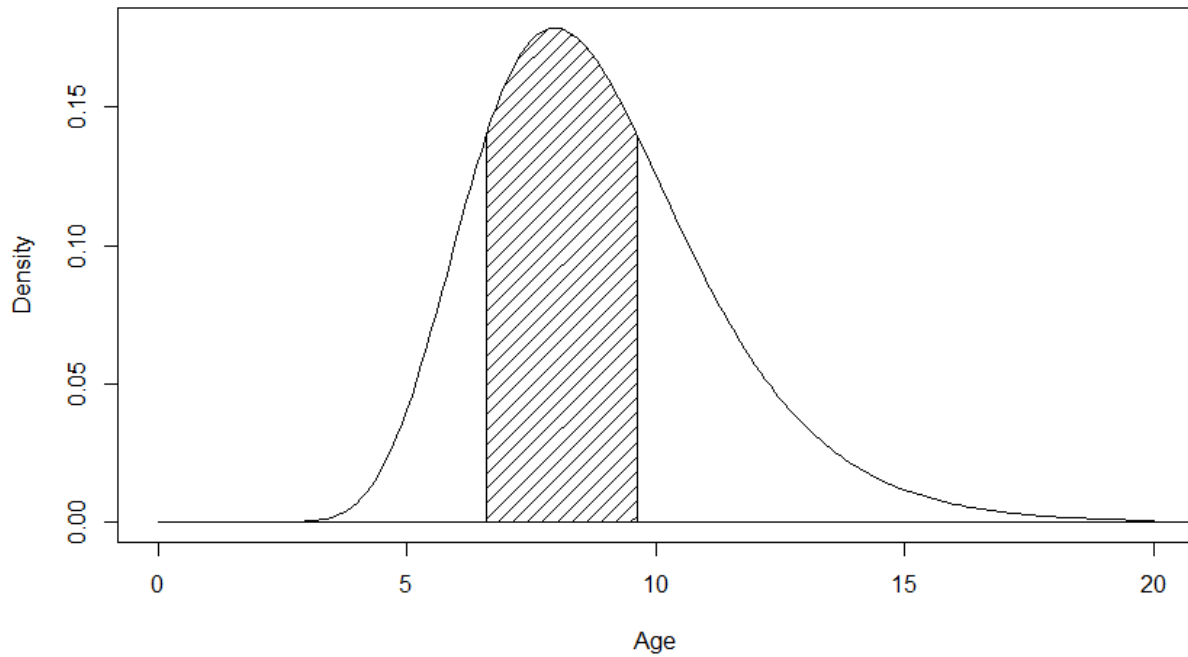
HPD male upper incisor 2 stage 10 (R1/2), US Violent Deaths 2010 prior



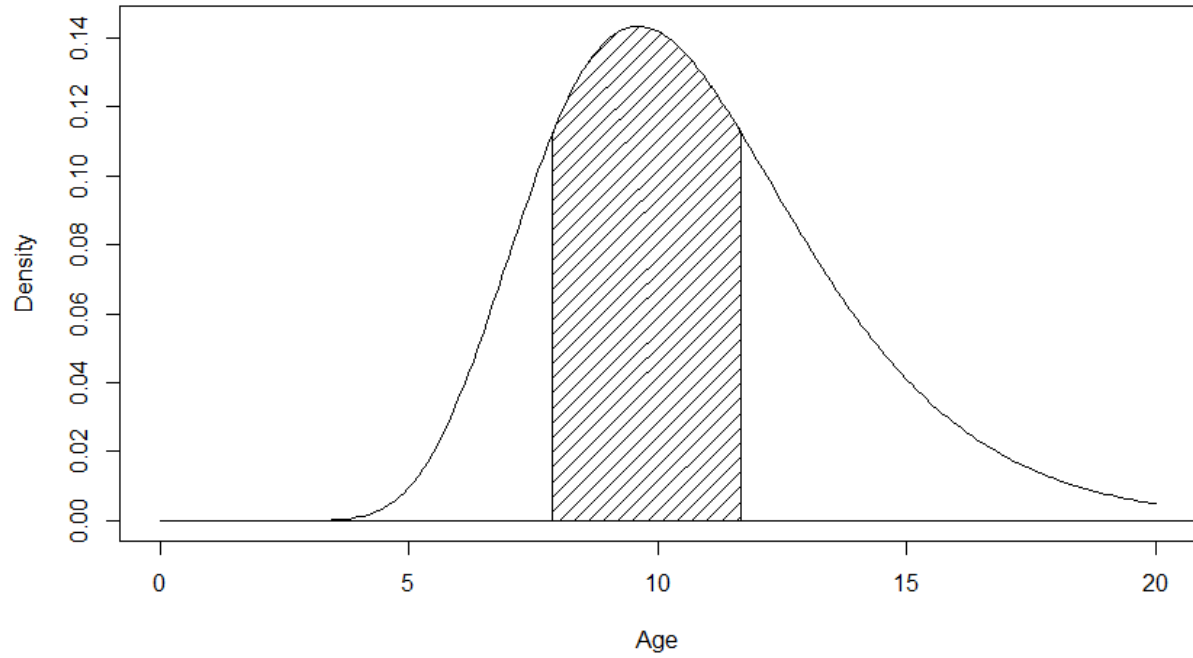
HPD male upper incisor 2 stage 11 (R3/4), US Violent Deaths 2010 prior



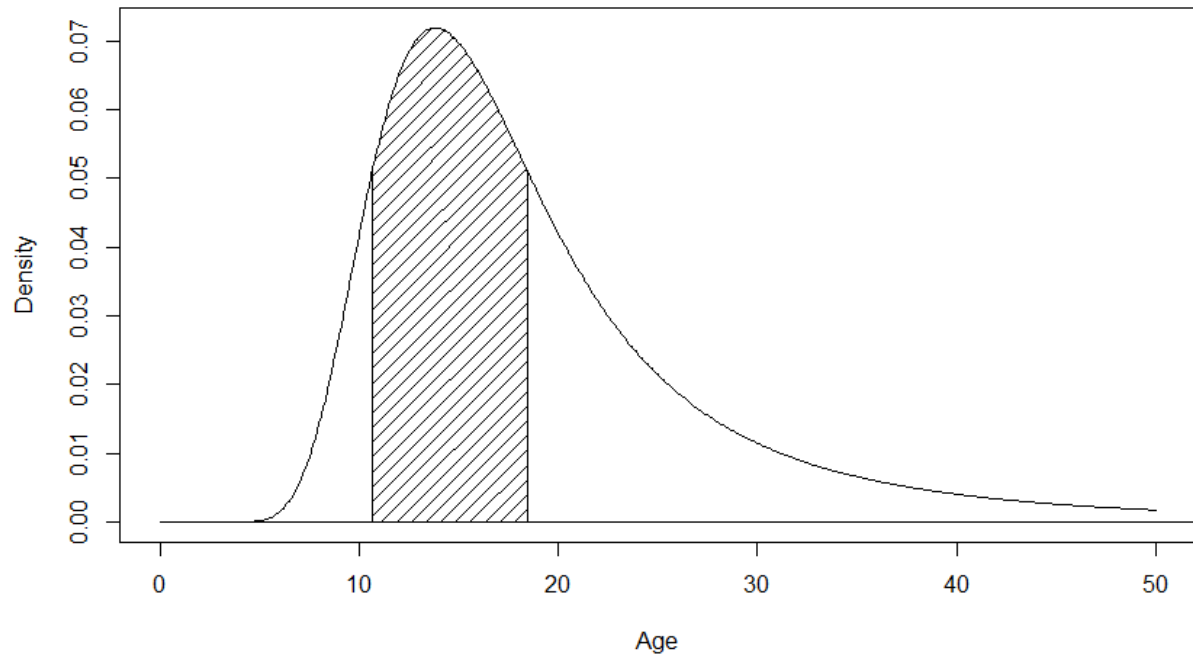
HPD male upper incisor 2 stage 12 (Rc), US Violent Deaths 2010 prior



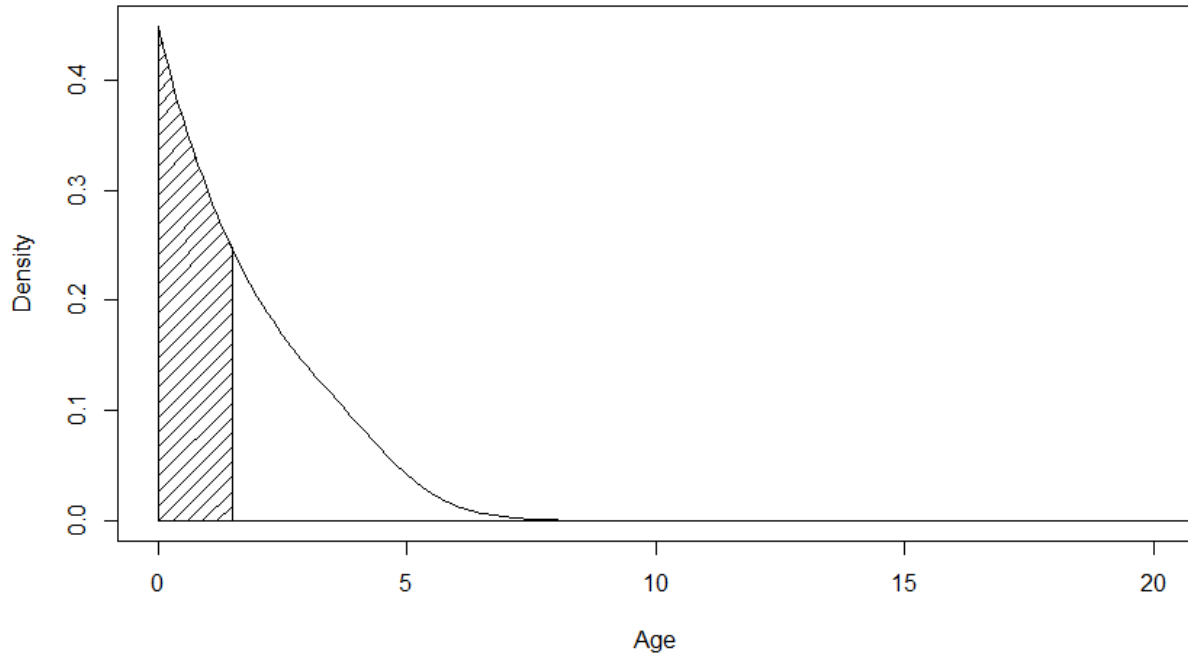
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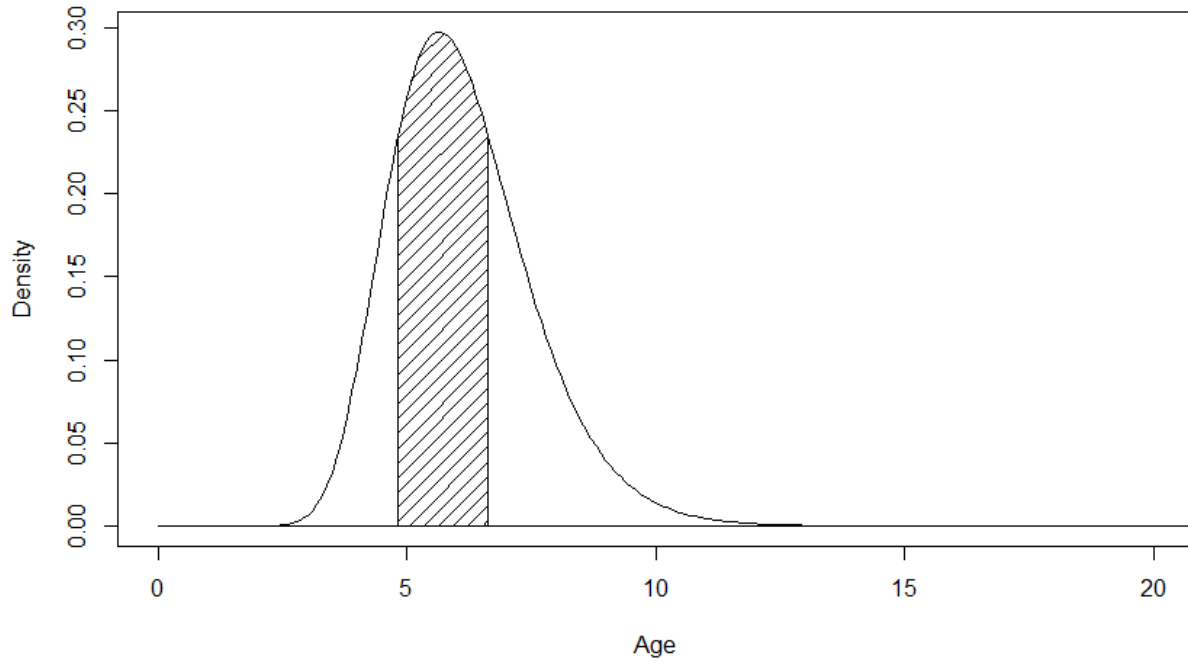
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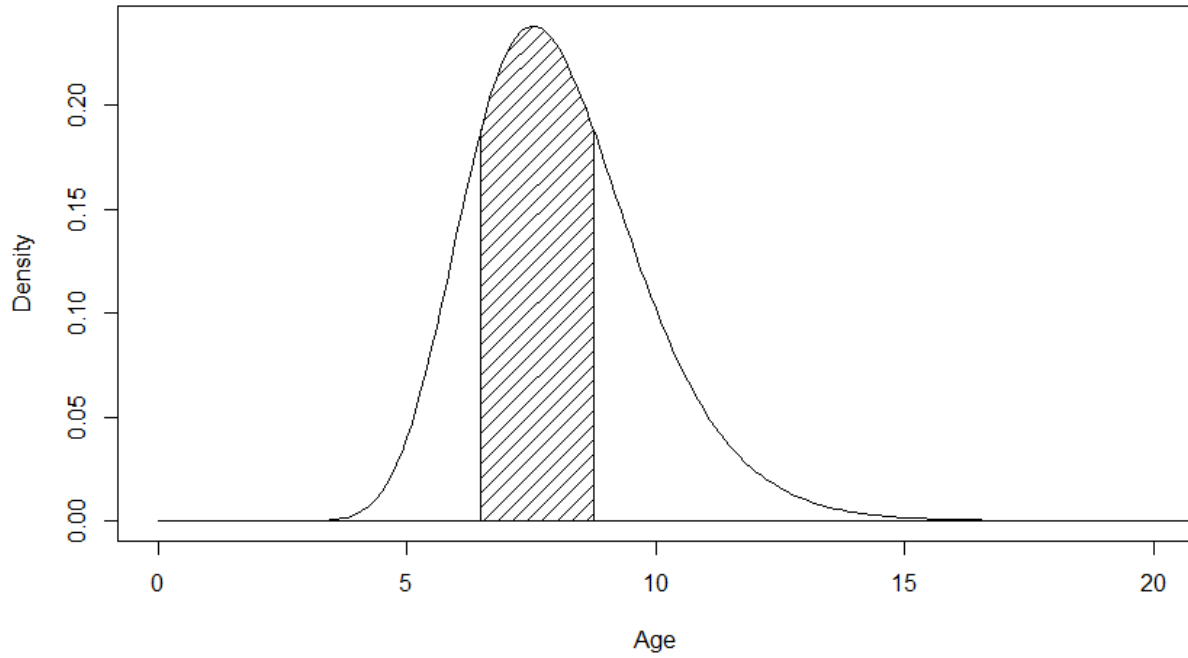
HPD male upper canine stage 7 (Ri), US Violent Deaths 2010 prior



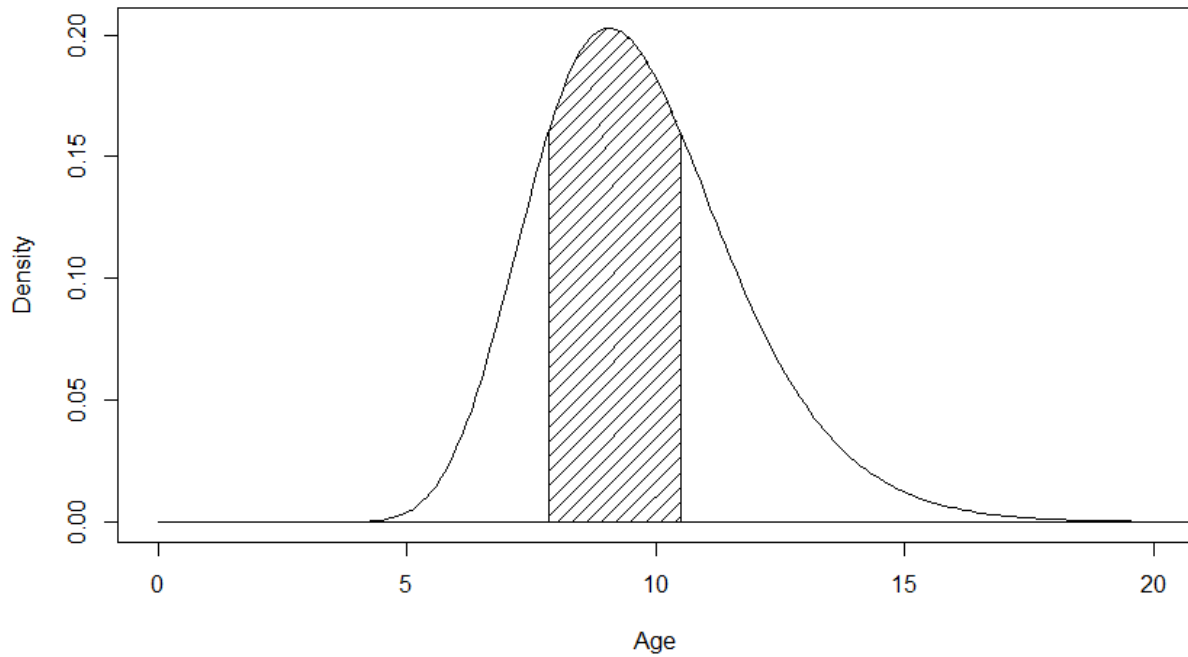
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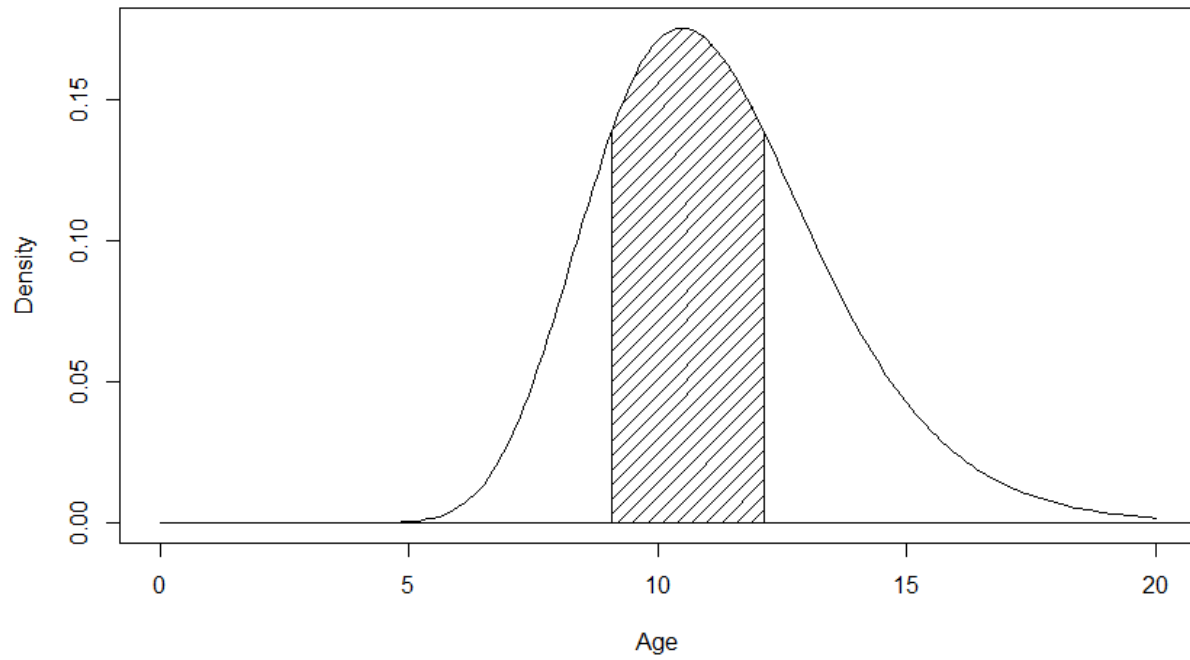
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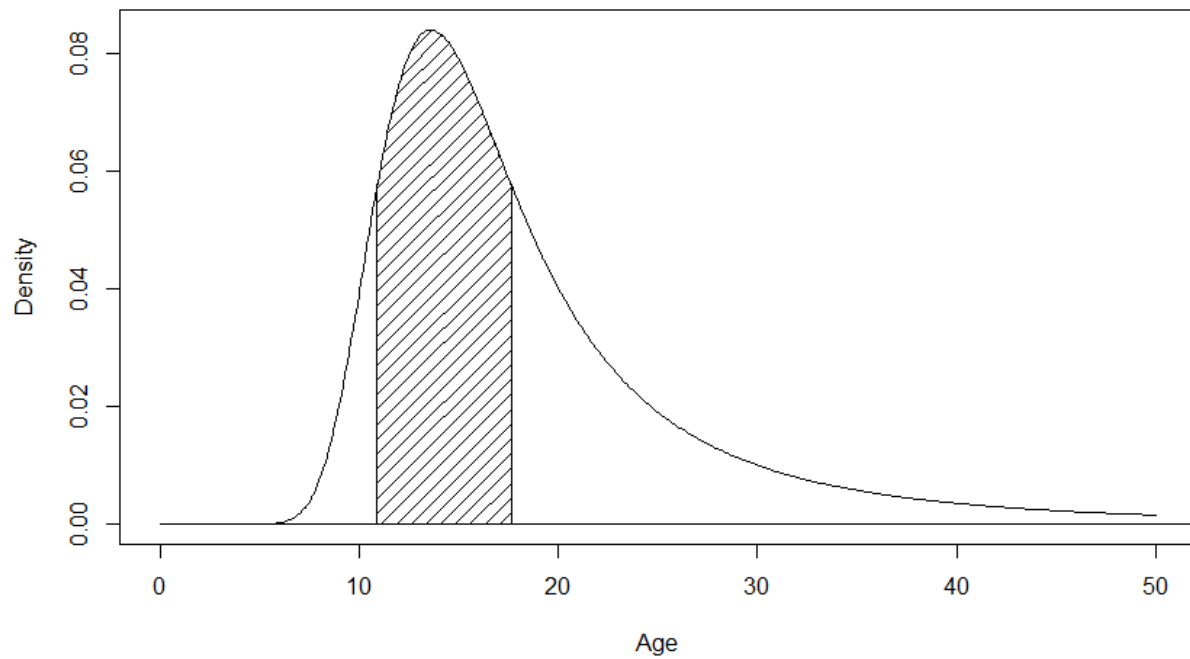
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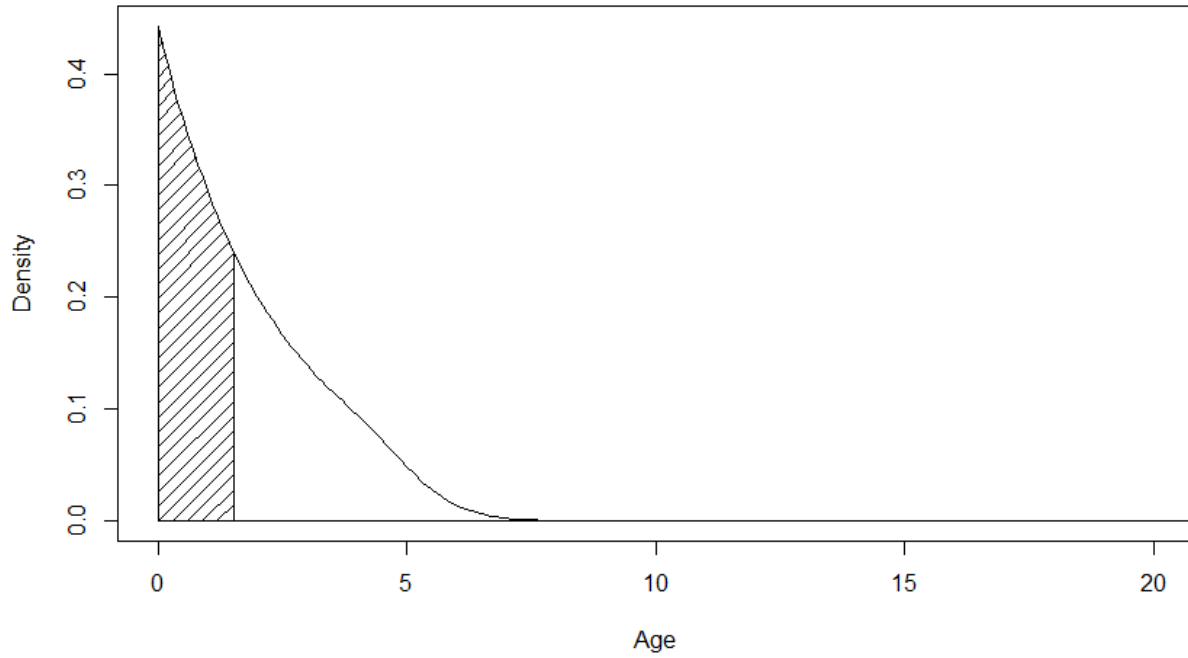
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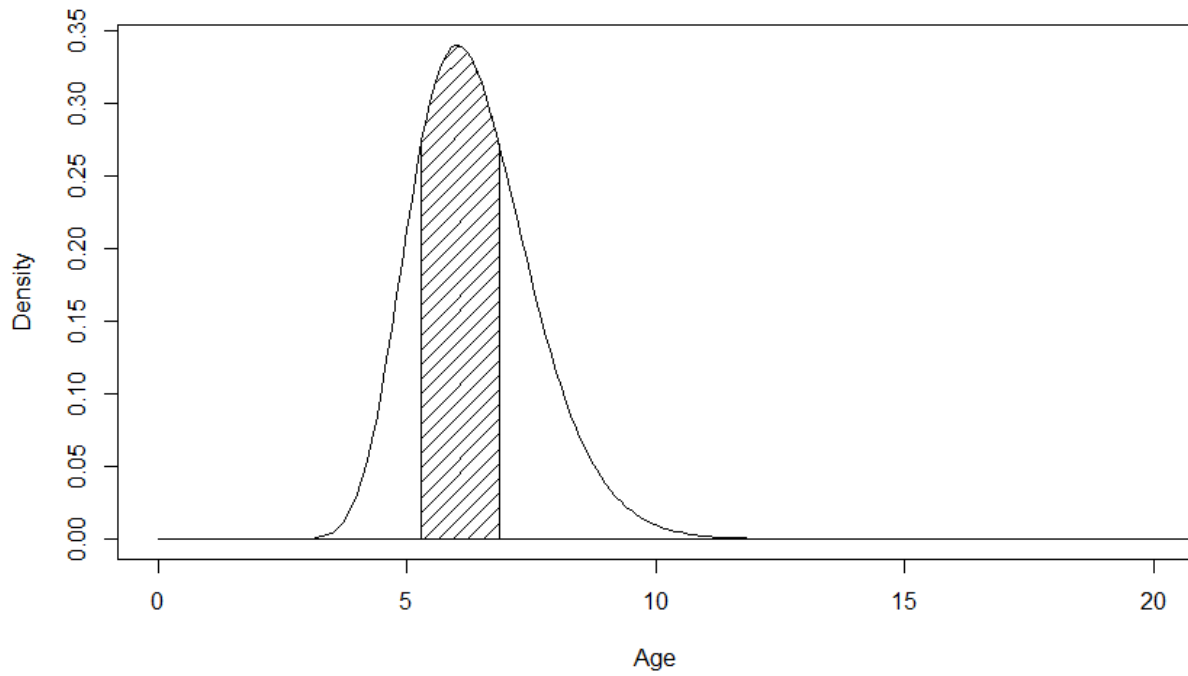
HPD male upper canine stage 13 (A1/2), US Violent Deaths 2010 prior



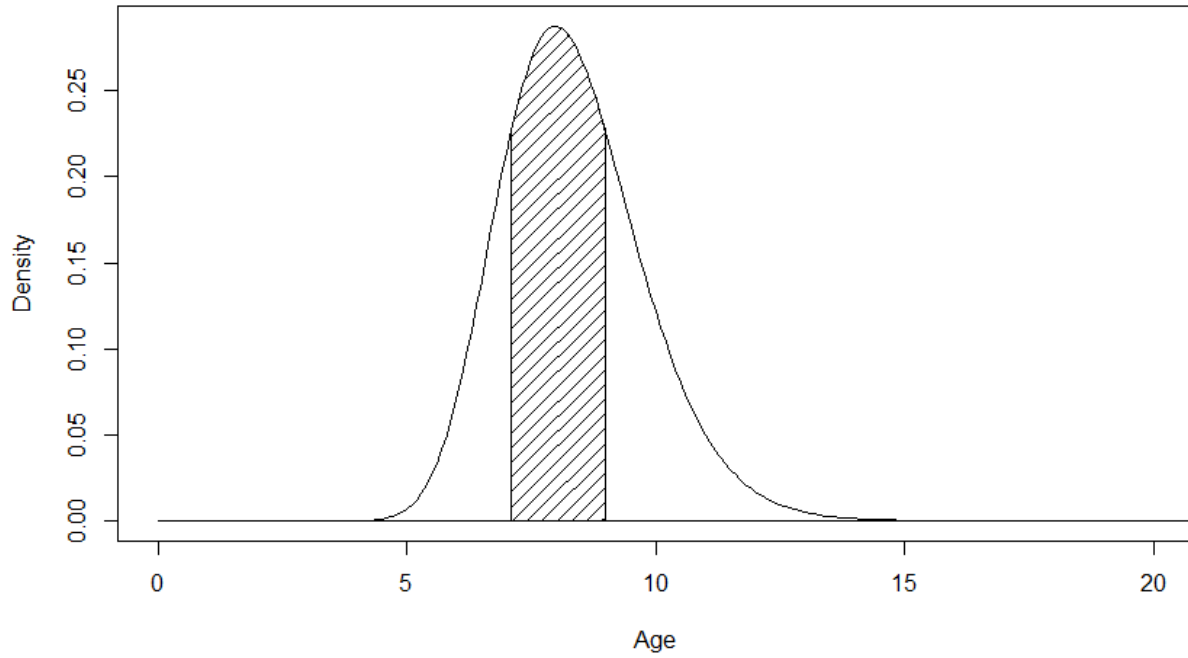
HPD male upper premolar 1 stage 6 (Crc), US Violent Deaths 2010 prior



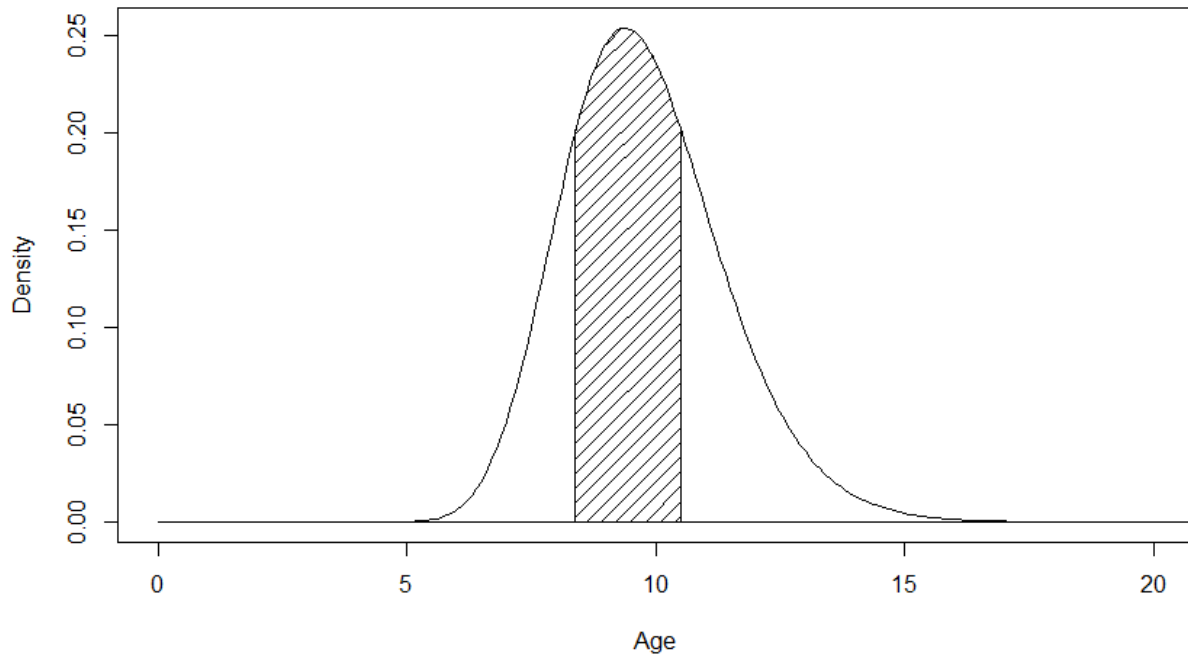
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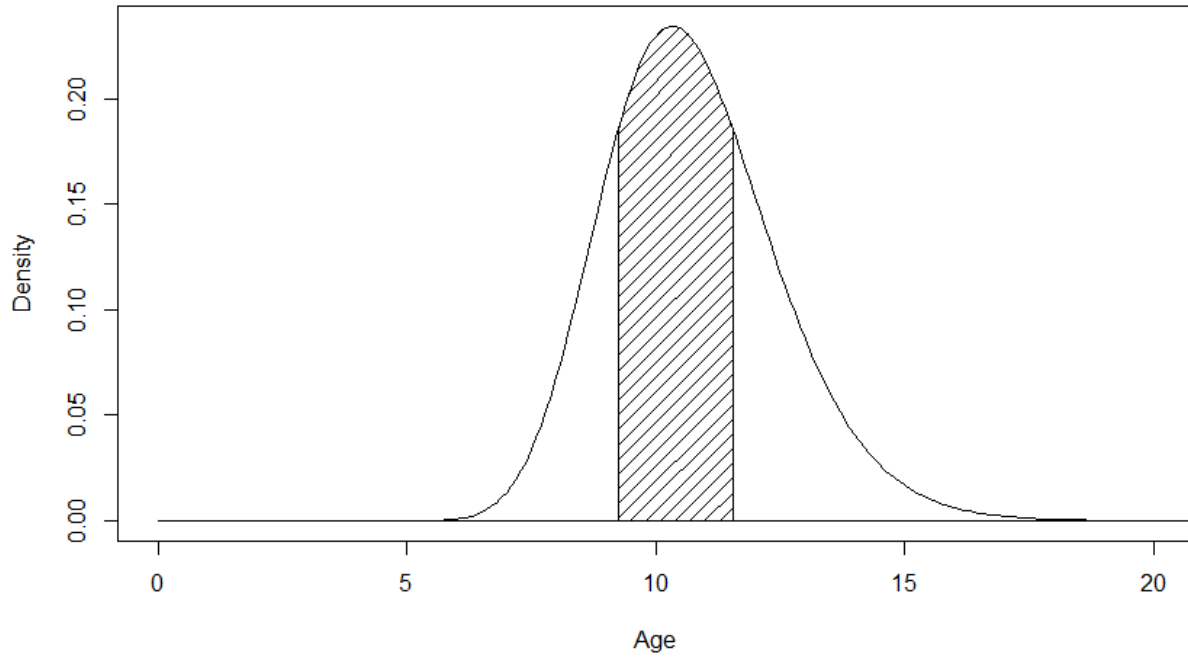
HPD male upper premolar 1 stage 9 (R1/4), US Violent Deaths 2010 prior



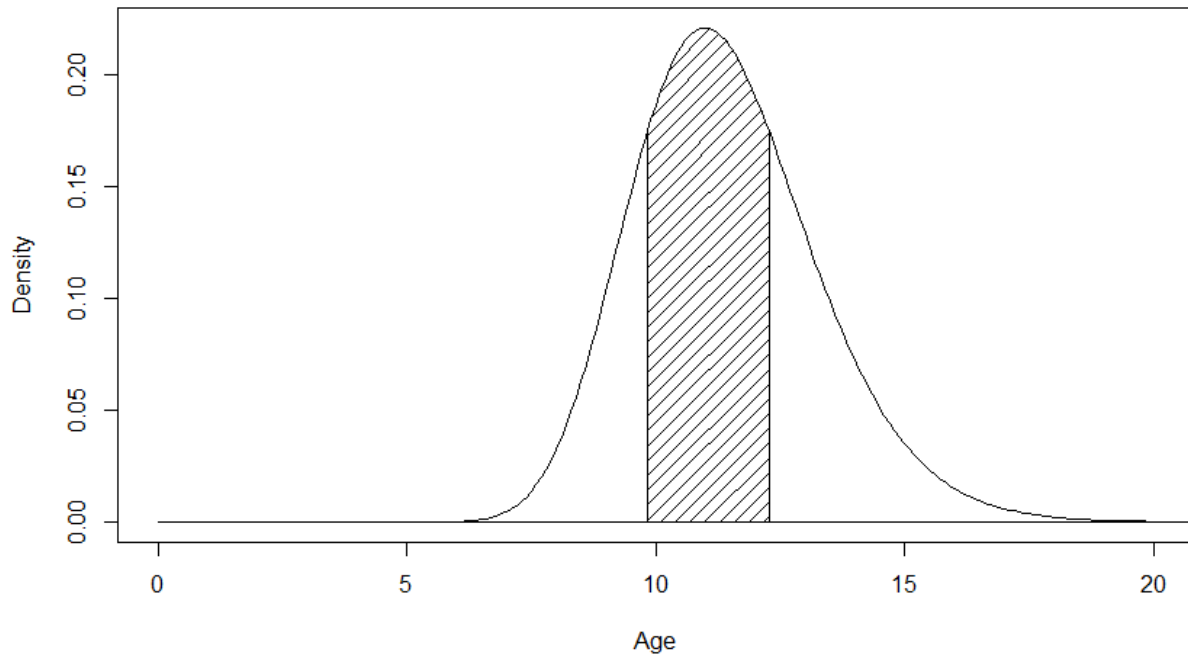
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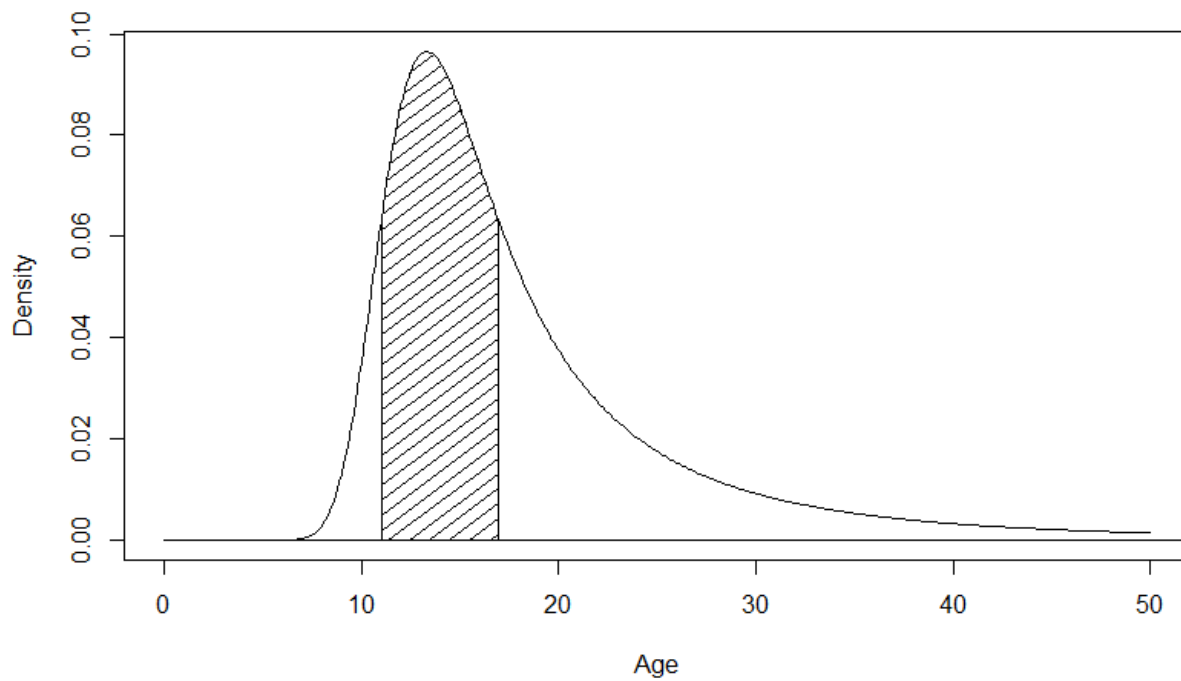
HPD male upper premolar 1 stage 11 (R3/4), US Violent Deaths 2010 prior



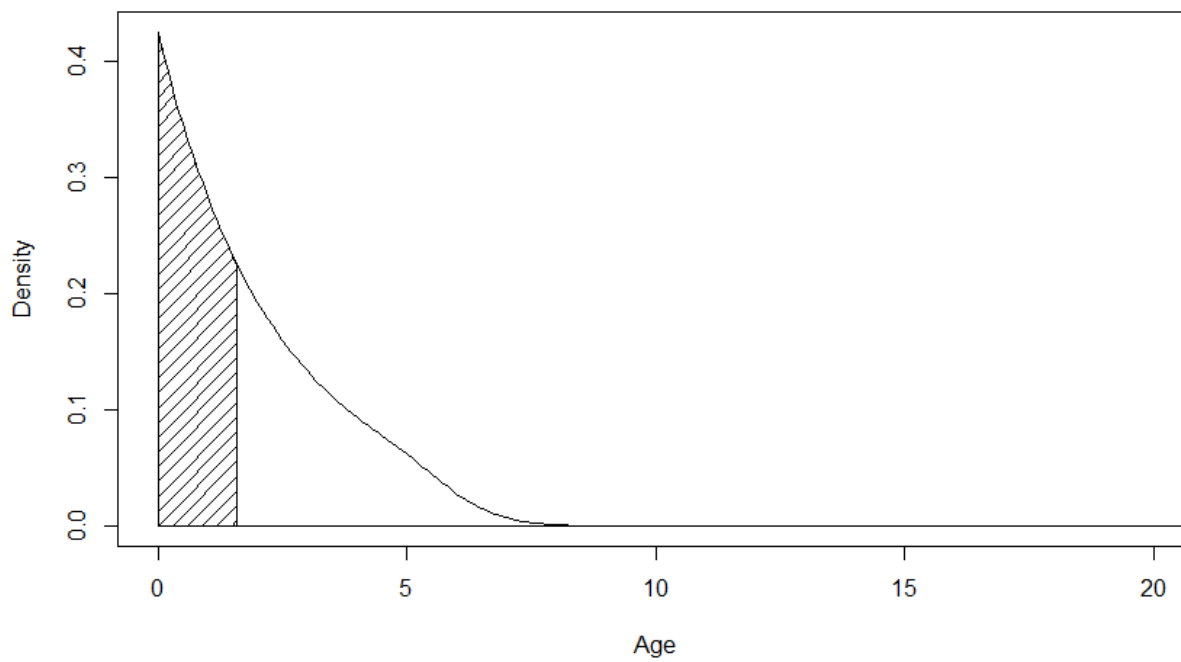
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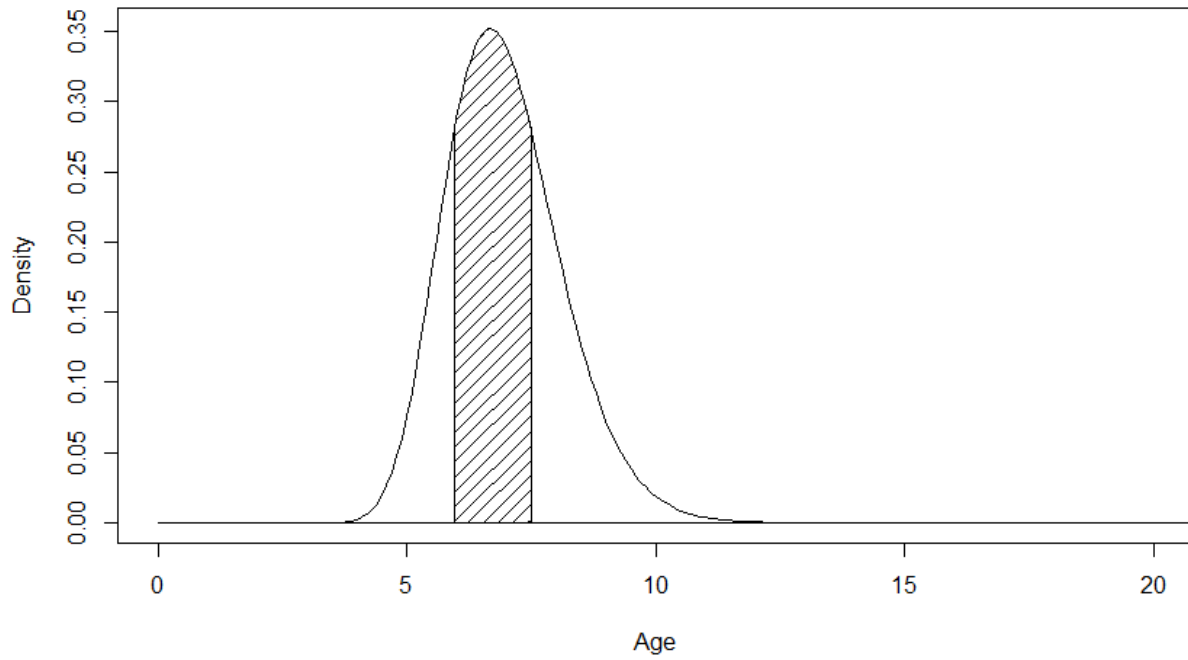
HPD male upper premolar 1 stage 13 (A 1/2), US Violent Deaths 2010 prior



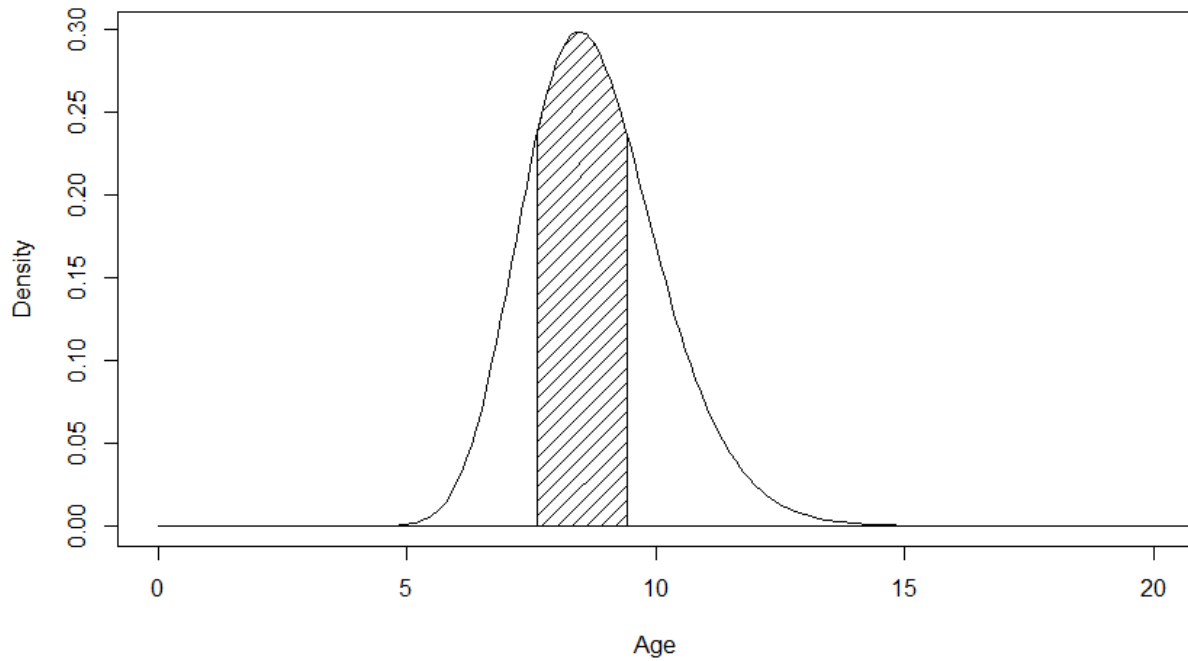
HPD male upper premolar 2 stage 6 (Crc), US Violent Deaths 2010 prior



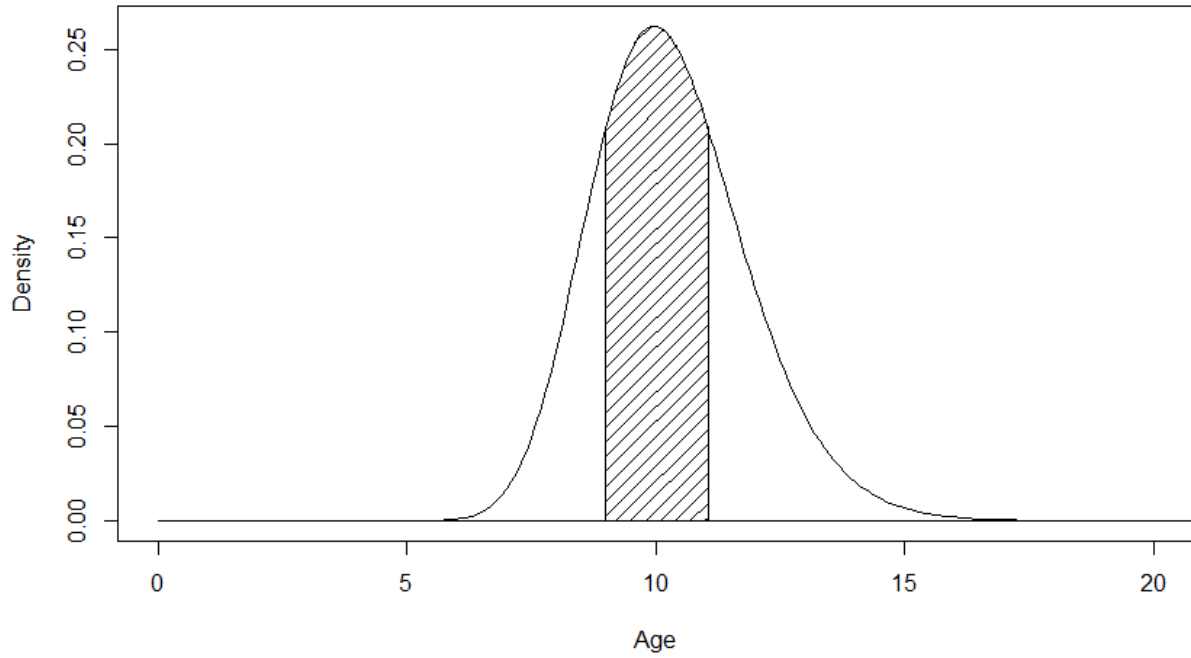
HPD male upper premolar 2 stage 7 (R1), US Violent Deaths 2010 prior



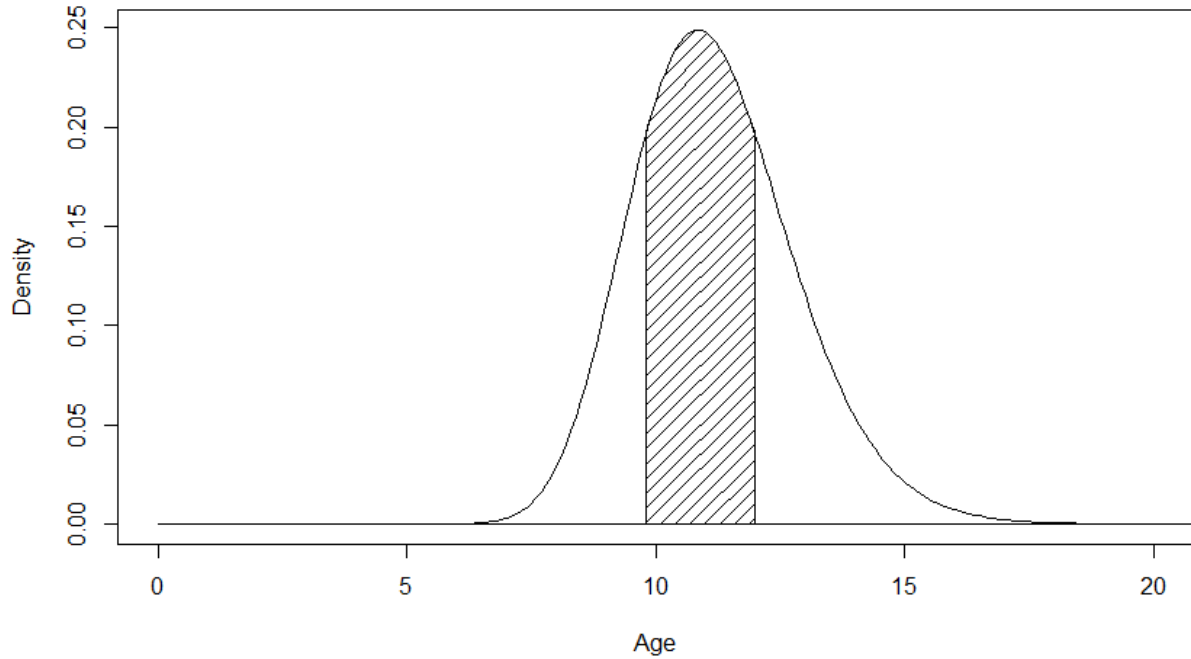
HPD male upper premolar 2 stage 9 (R1/4), US Violent Deaths 2010 prior



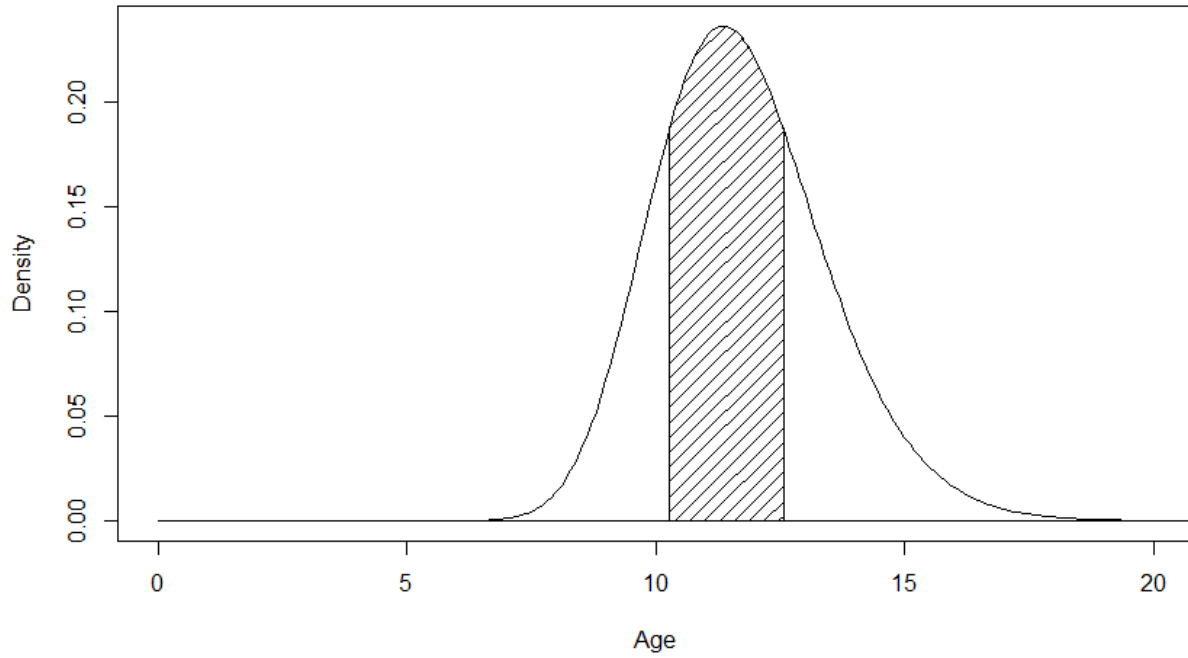
HPD male upper premolar 2 stage 10 (R1/2), US Violent Deaths 2010 prior



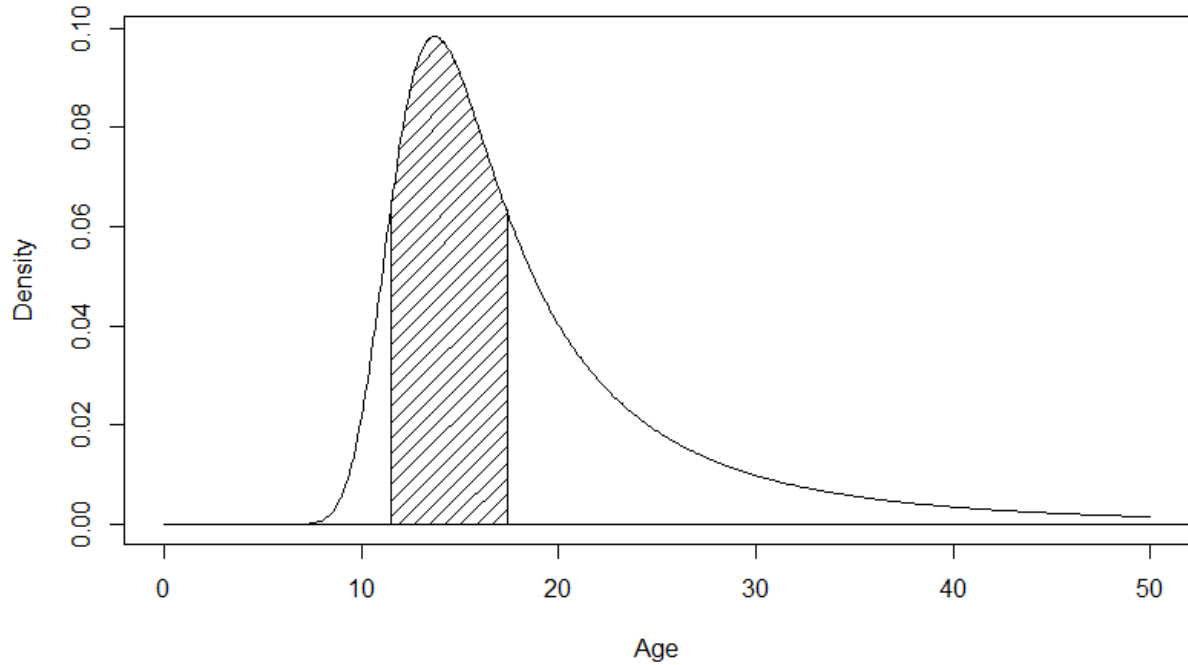
HPD male upper premolar 2 stage 11 (R3/4), US Violent Deaths 2010 prior



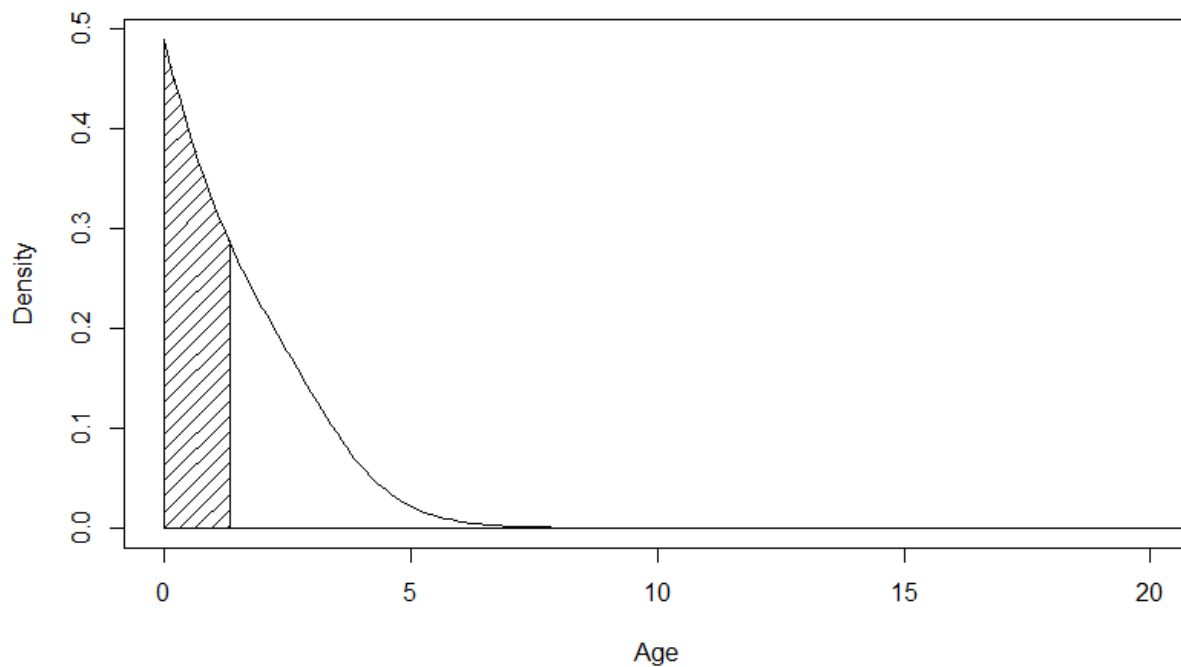
HPD male upper premolar 2 stage 12 (Rc), US Violent Deaths 2010 prior



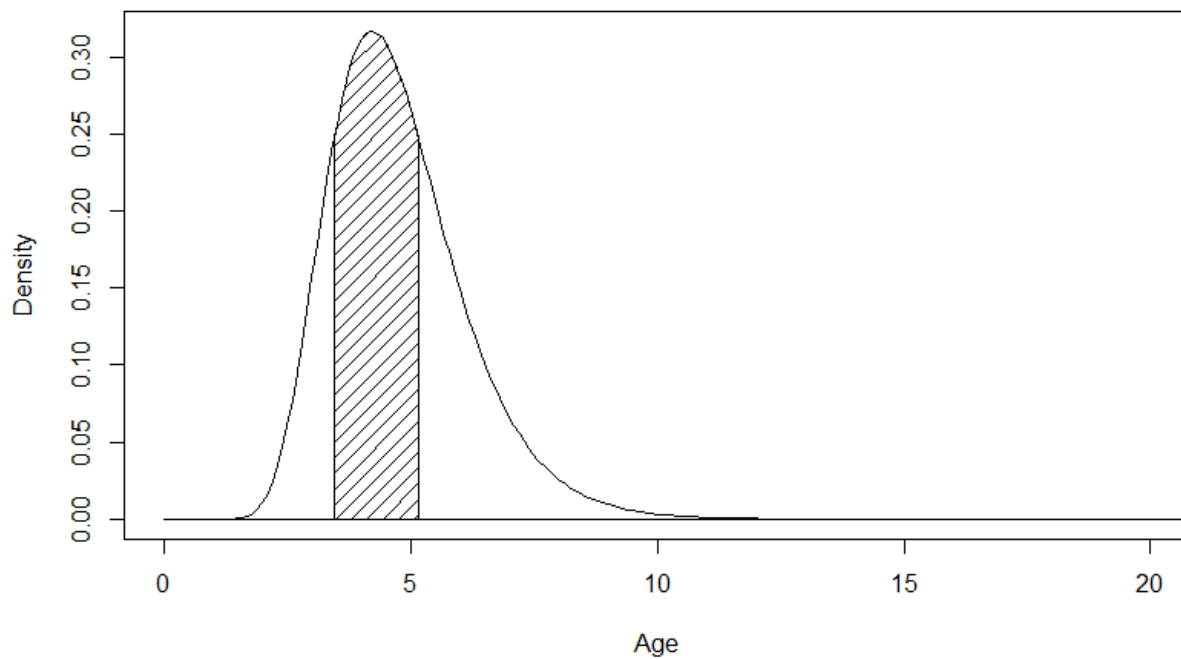
HPD male upper premolar 2 stage 13 (A 1/2), US Violent Deaths 2010 prior



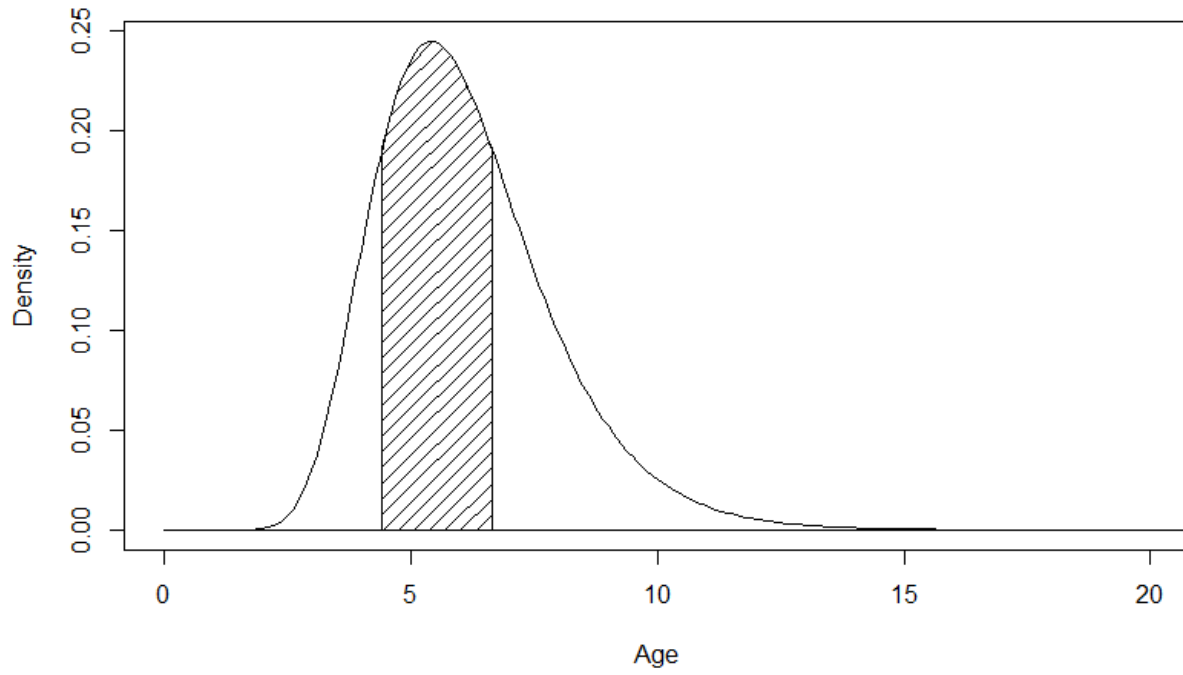
HPD male upper molar 1 stage 9 (R 1/4), US Violent Deaths 2010 prior



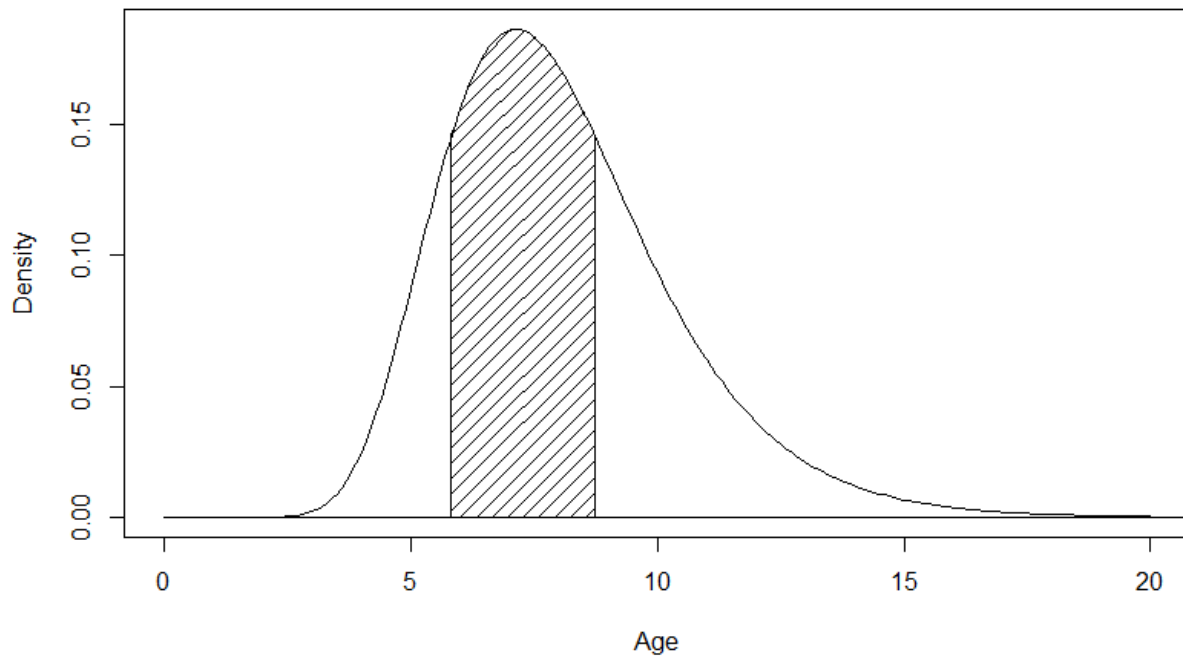
HPD male upper molar 1 stage 10 (R 1/2), US Violent Deaths 2010 prior



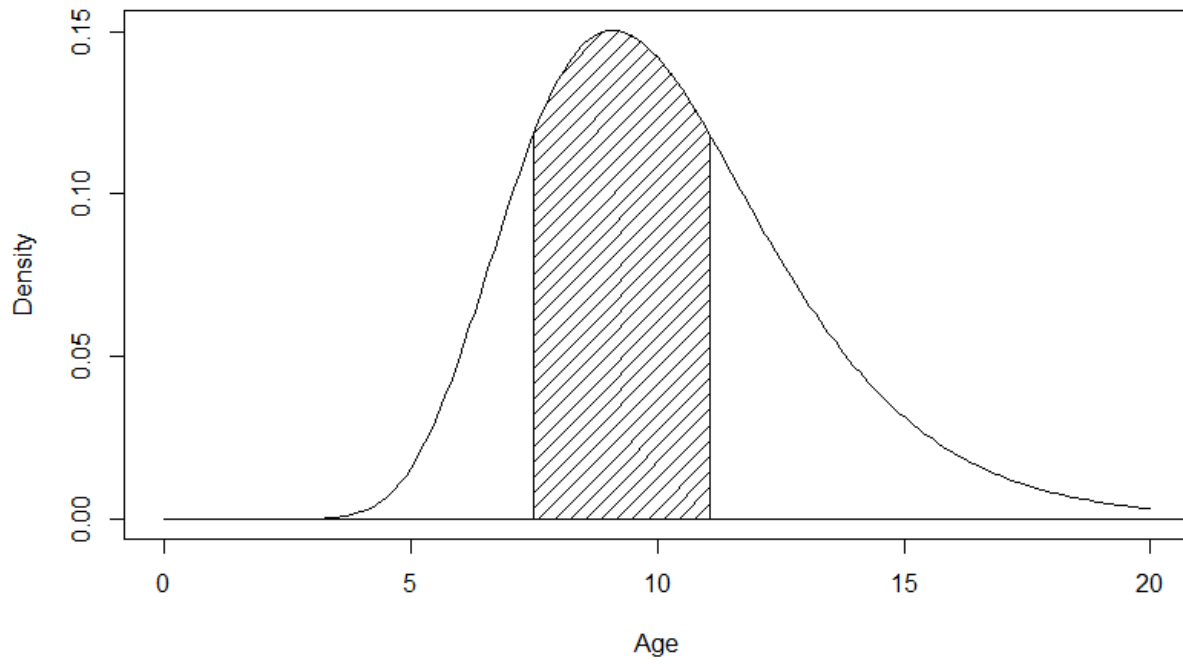
HPD male upper molar 1 stage 11 (R 3/4), US Violent Deaths 2010 prior



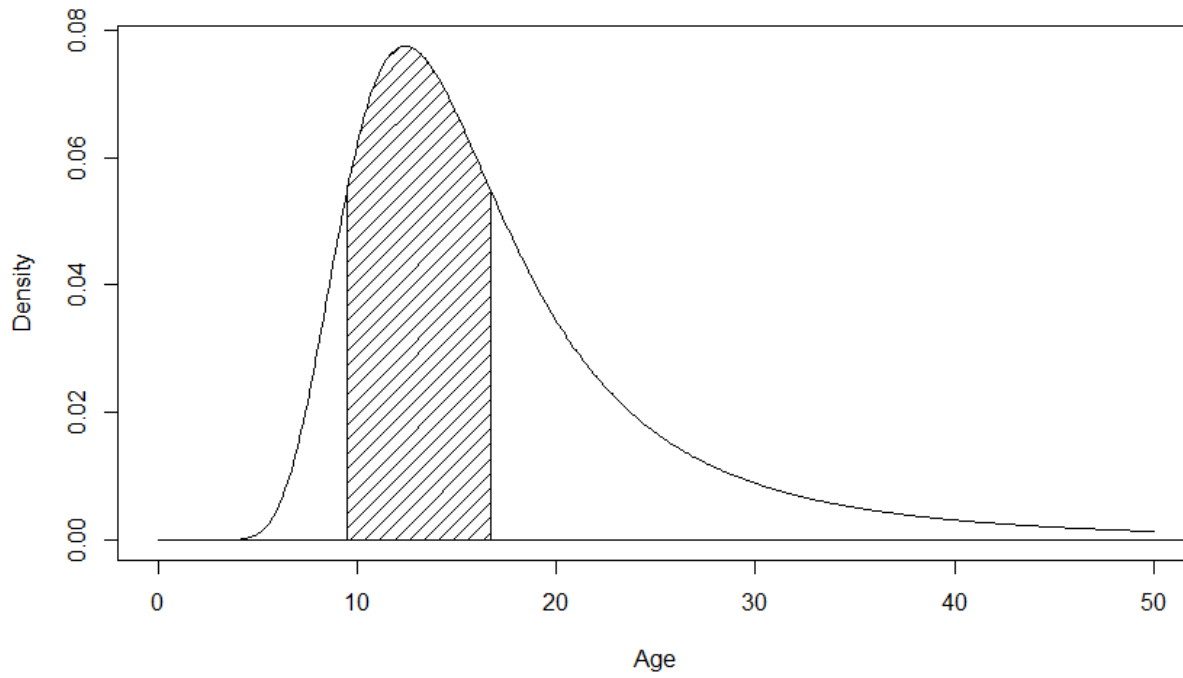
HPD male upper molar 1 stage 12 (Rc), US Violent Deaths 2010 prior



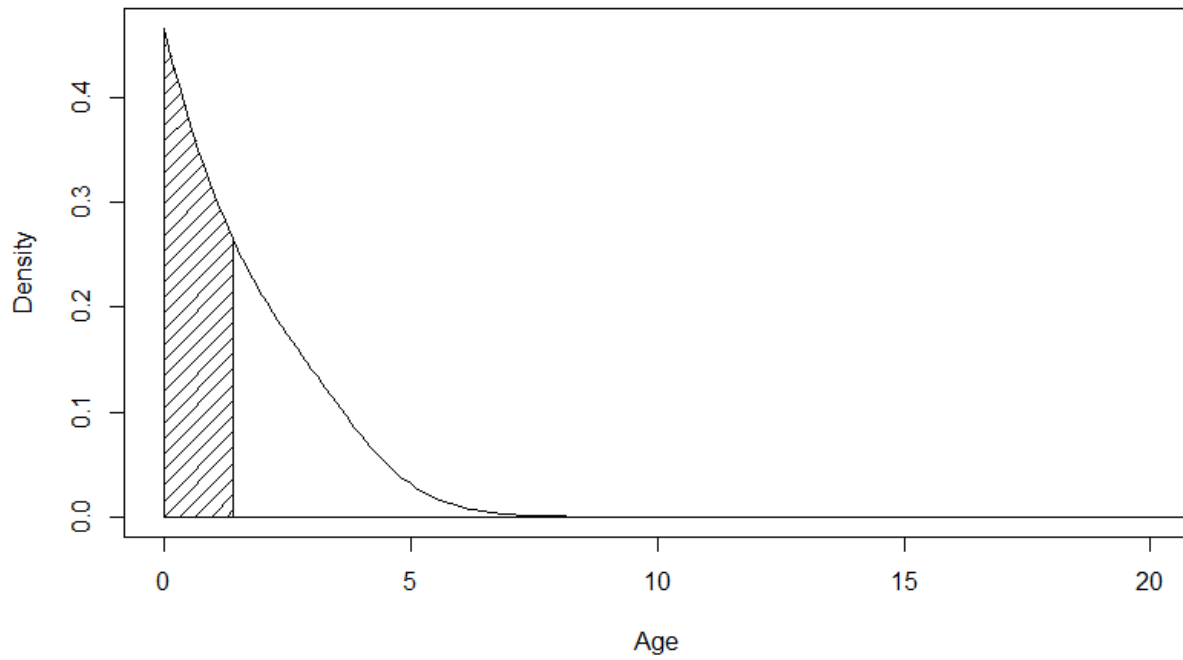
HPD male upper molar 1 stage 13 (A 1/2), US Violent Deaths 2010 prior



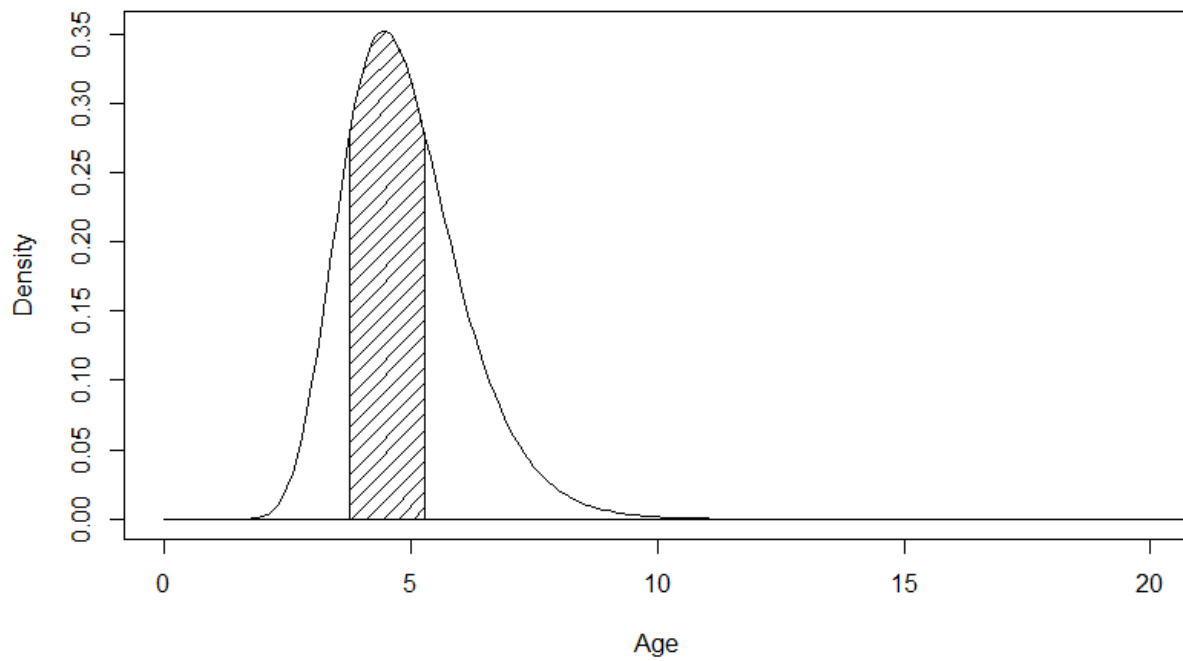
HPD male upper molar 1 stage 14 (Ac), US Violent Deaths 2010 prior



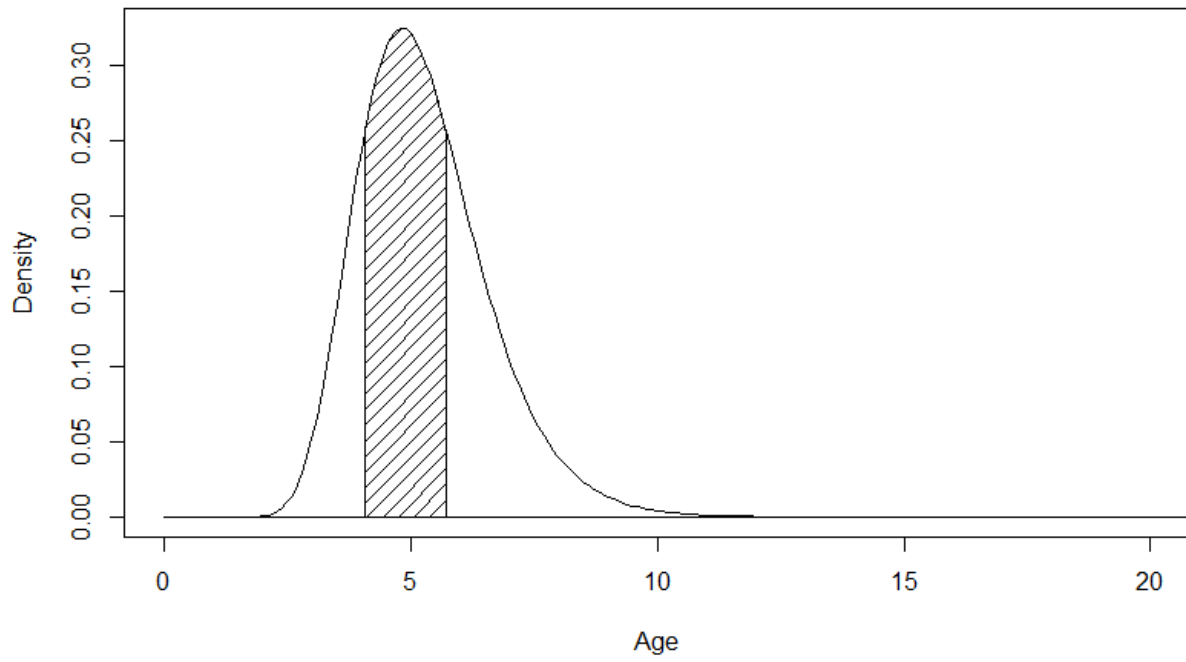
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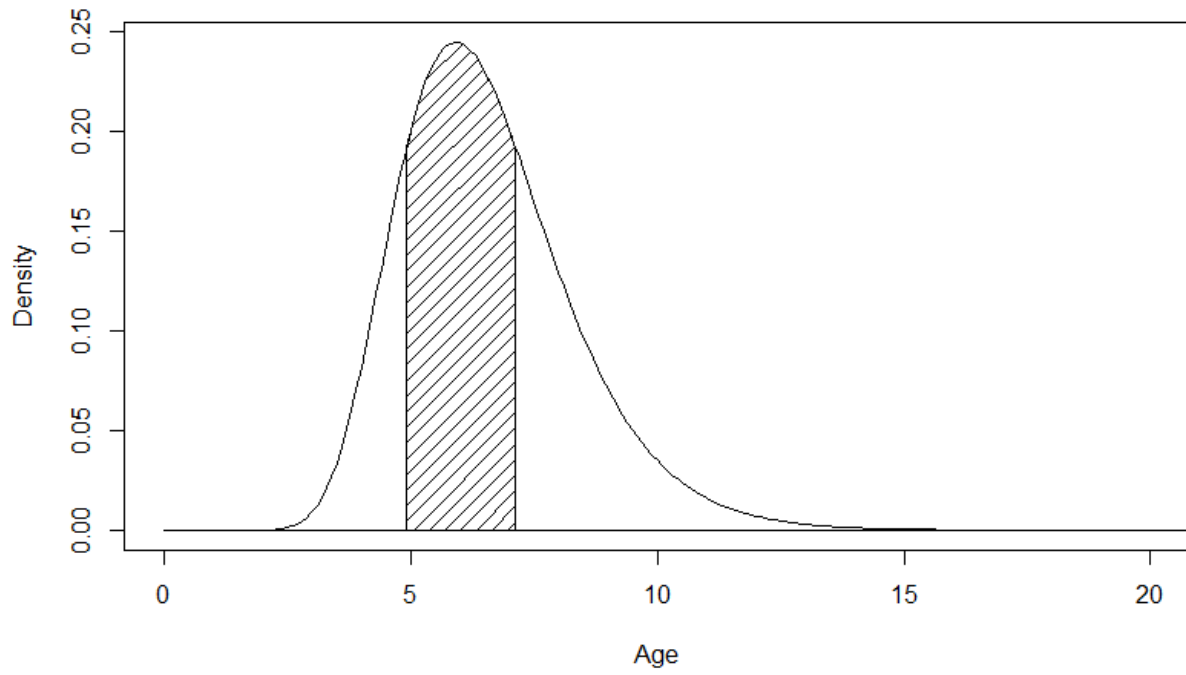
HPD male upper molar 2 stage 5 (Cr 3/4), US Violent Deaths 2010 prior



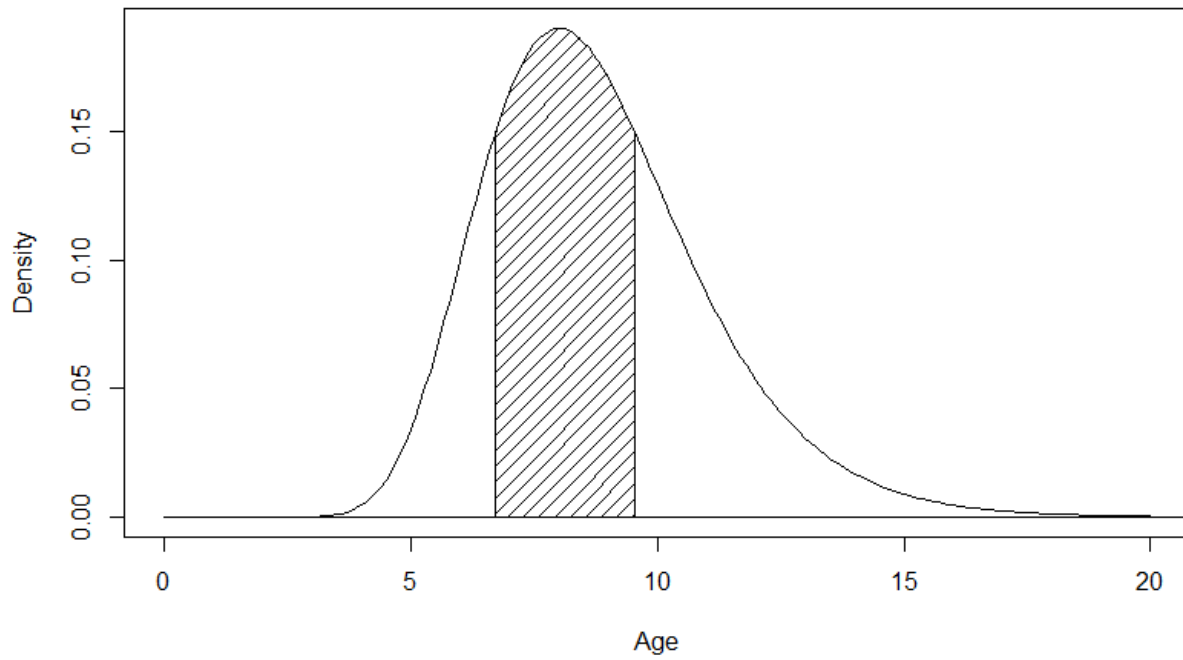
HPD male upper molar 2 stage 6 (Crc), US Violent Deaths 2010 prior



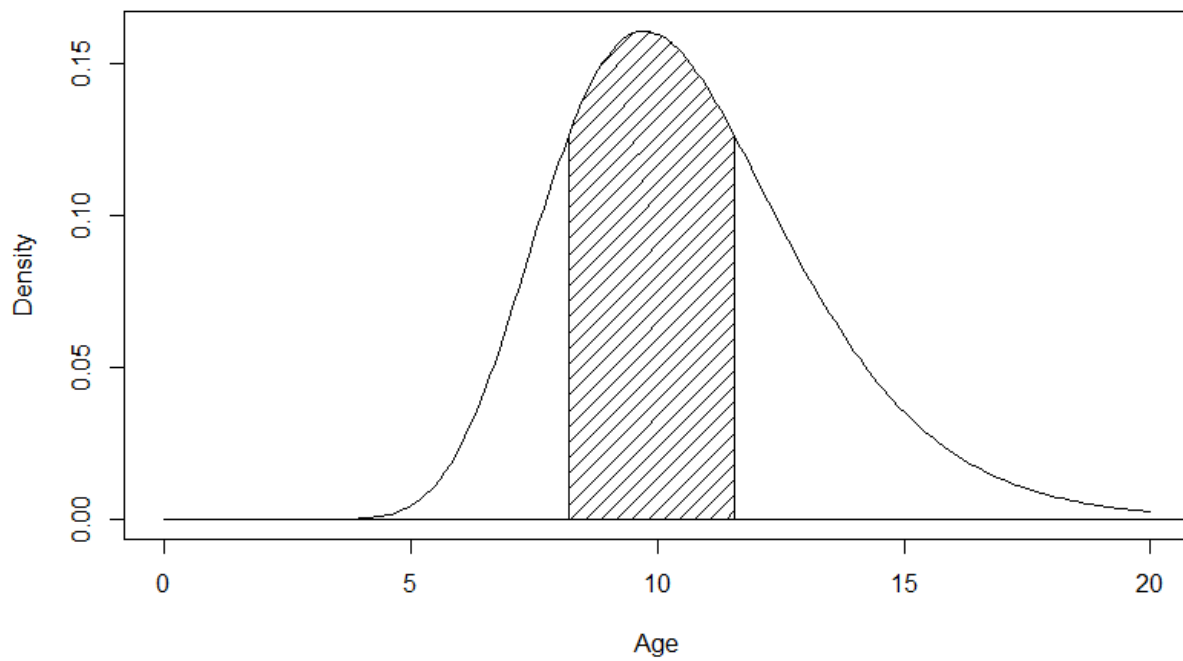
HPD male upper molar 2 stage 7 (Ri), US Violent Deaths 2010 prior



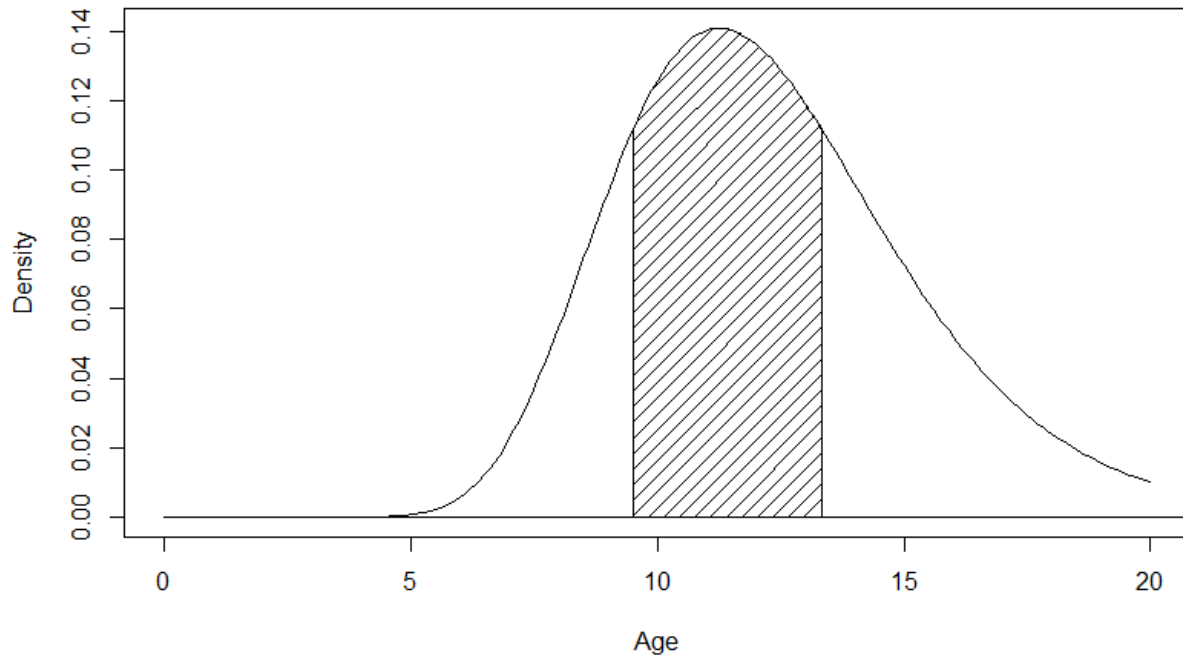
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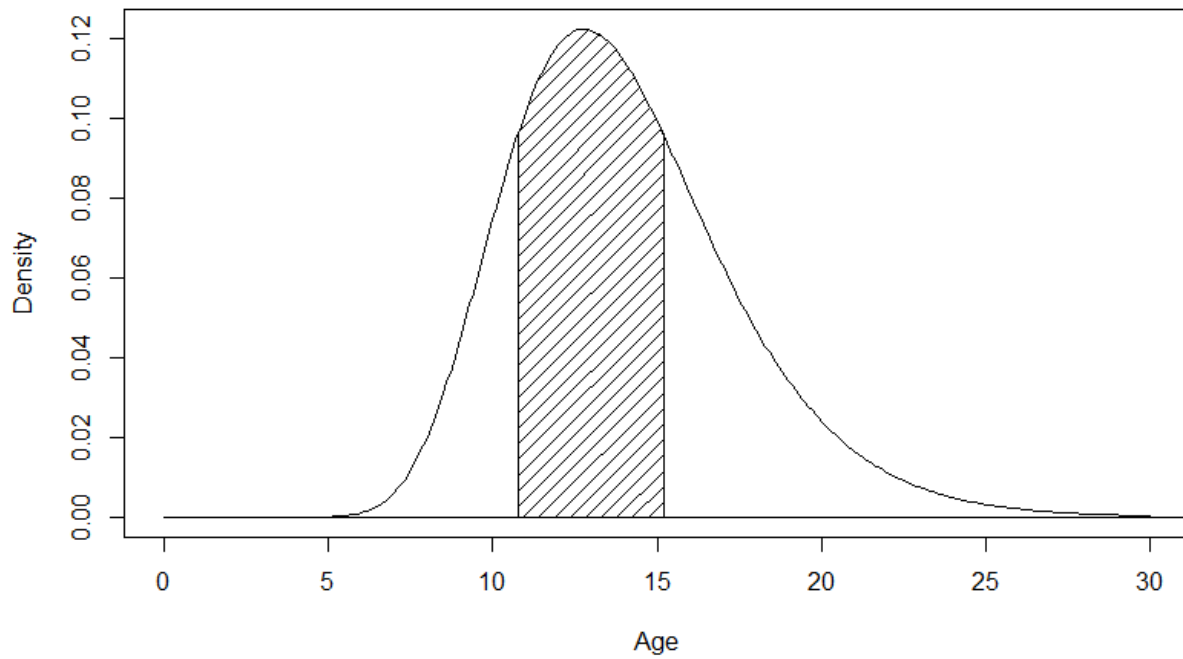
HPD male upper molar 2 stage 9 (R 1/4), US Violent Deaths 2010 prior



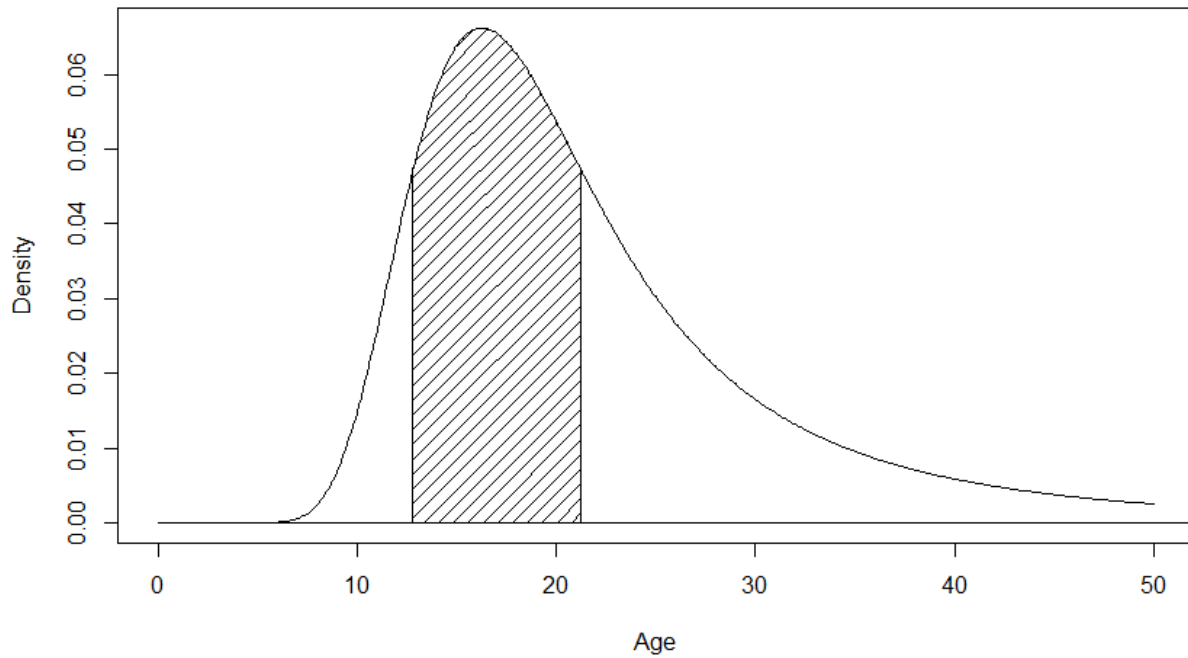
HPD male upper molar 2 stage 10 (R 1/2), US Violent Deaths 2010 prior



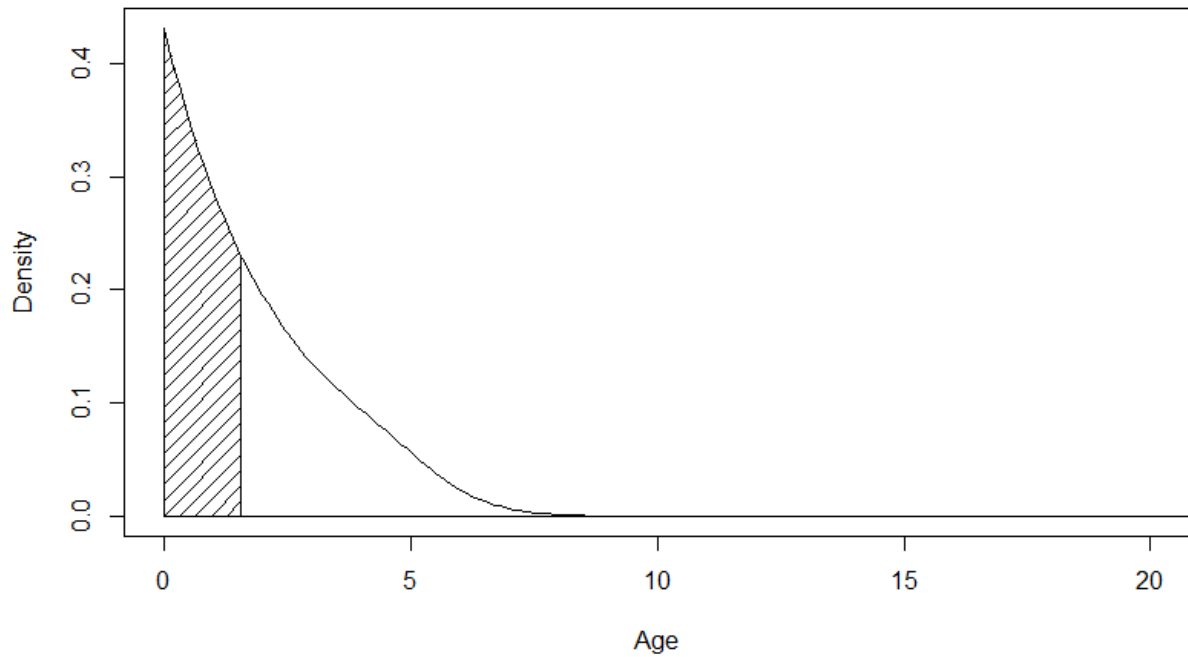
HPD male upper molar 2 stage 11 (R 3/4), US Violent Deaths 2010 prior



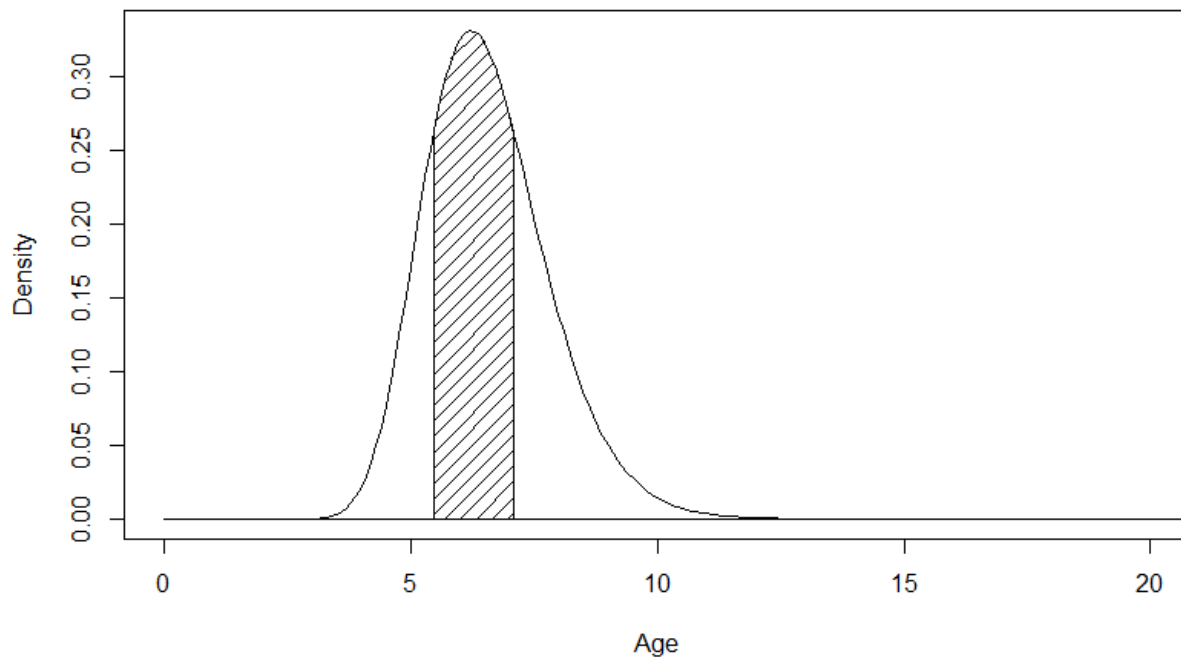
HPD male upper molar 2 stage 12 (Rc), US Violent Deaths 2010 prior



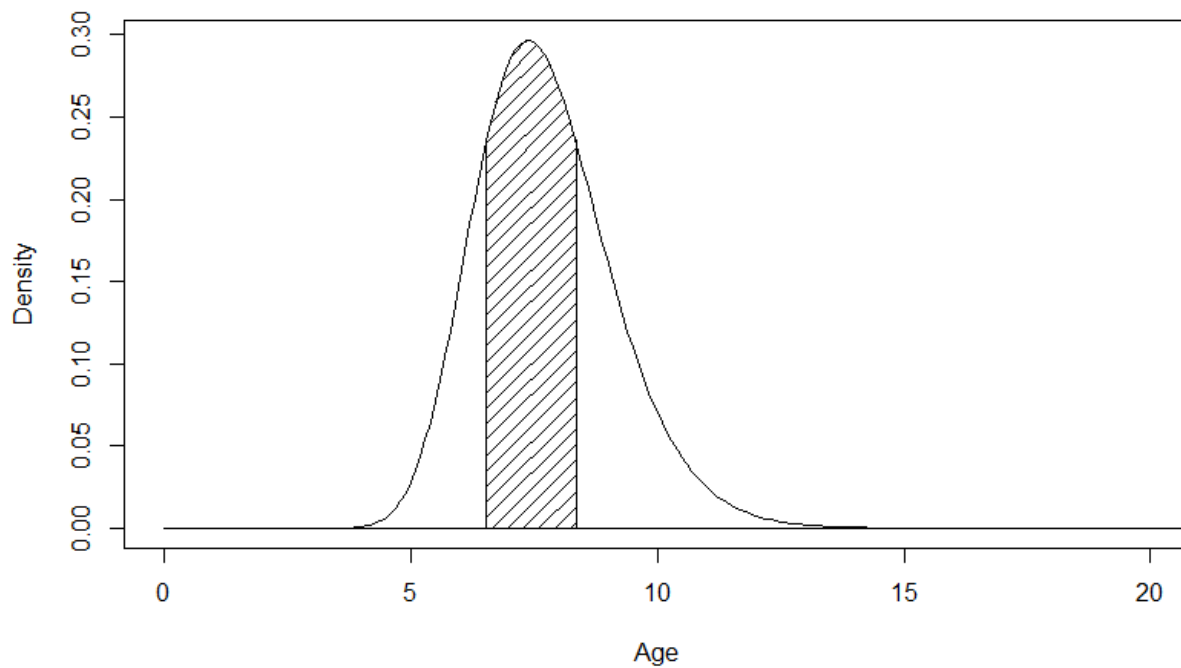
HPD male lower incisor 2 stage 10 (R 1/2), US Violent Deaths 2010 prior



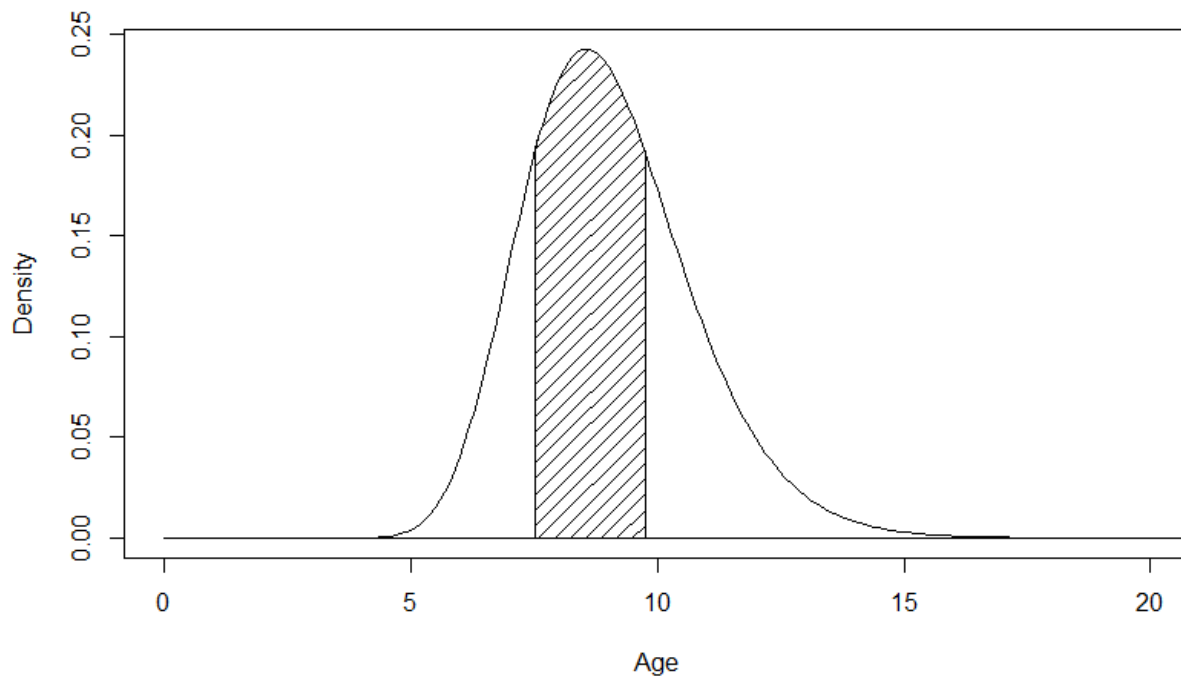
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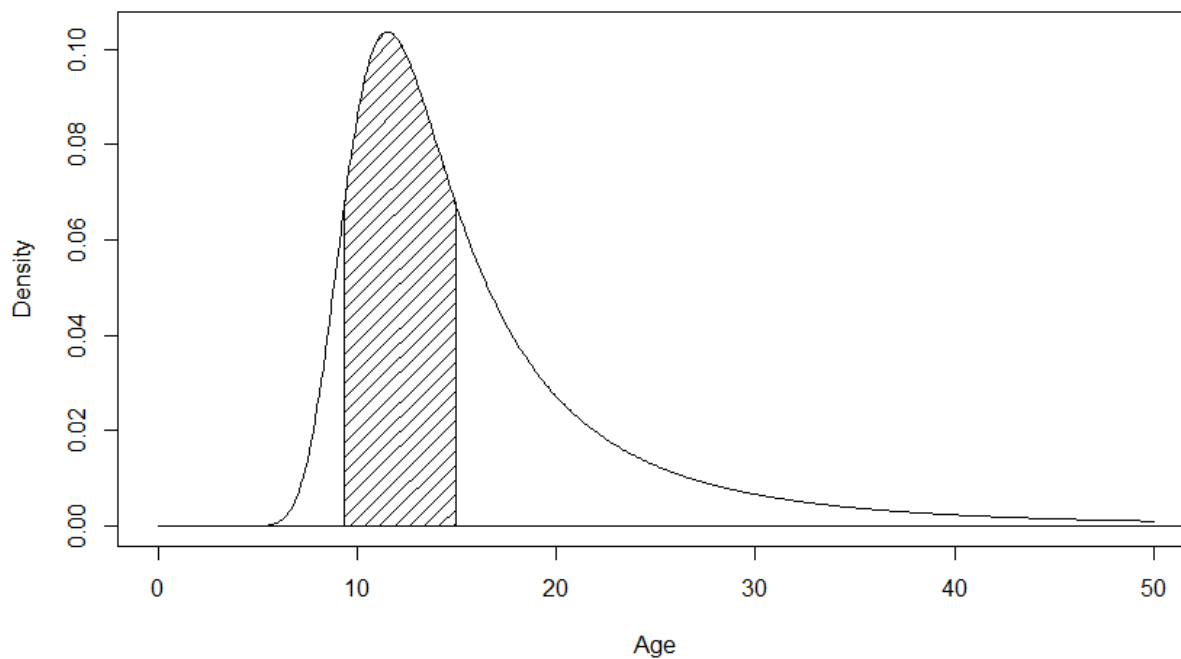
HPD male lower incisor 2 stage 12 (Rc), US Violent Deaths 2010 prior



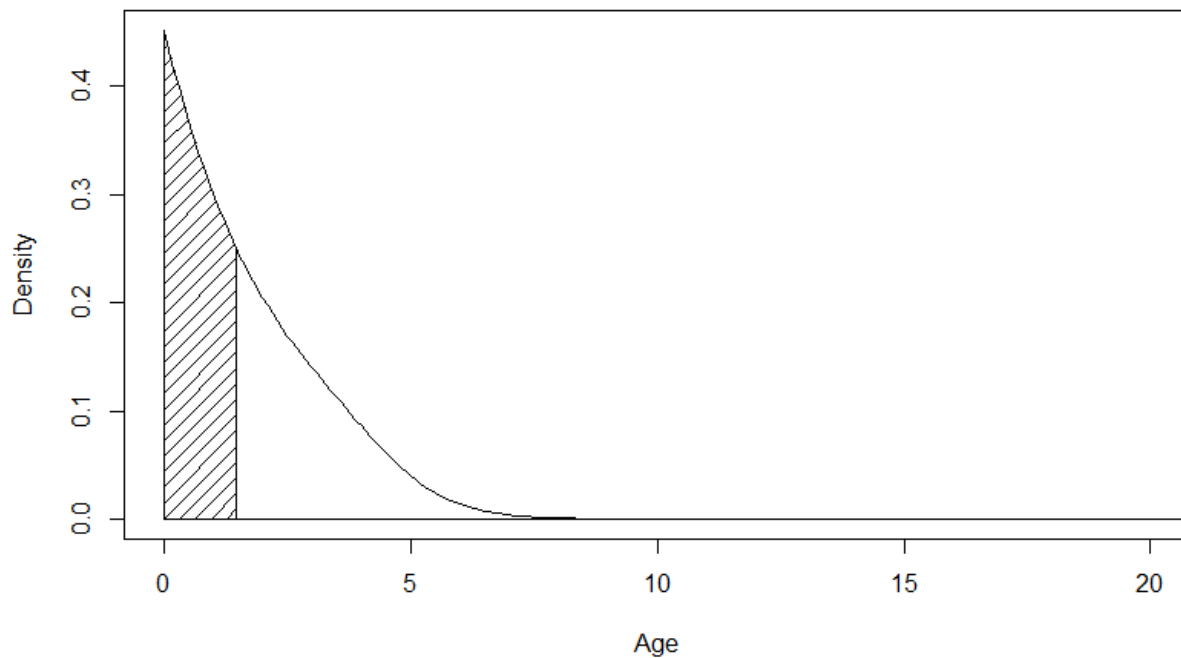
HPD male lower incisor 2 stage 13 (A 1/2), US Violent Deaths 2010 prior



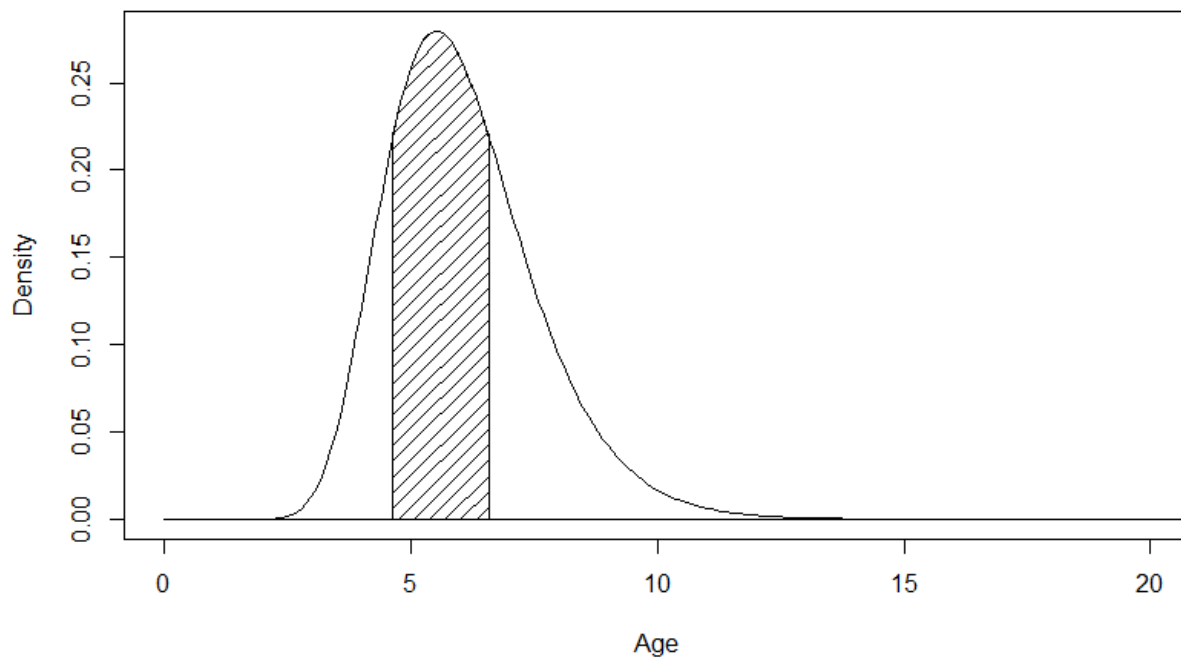
HPD male lower incisor 2 stage 14 (Ac), US Violent Deaths 2010 prior



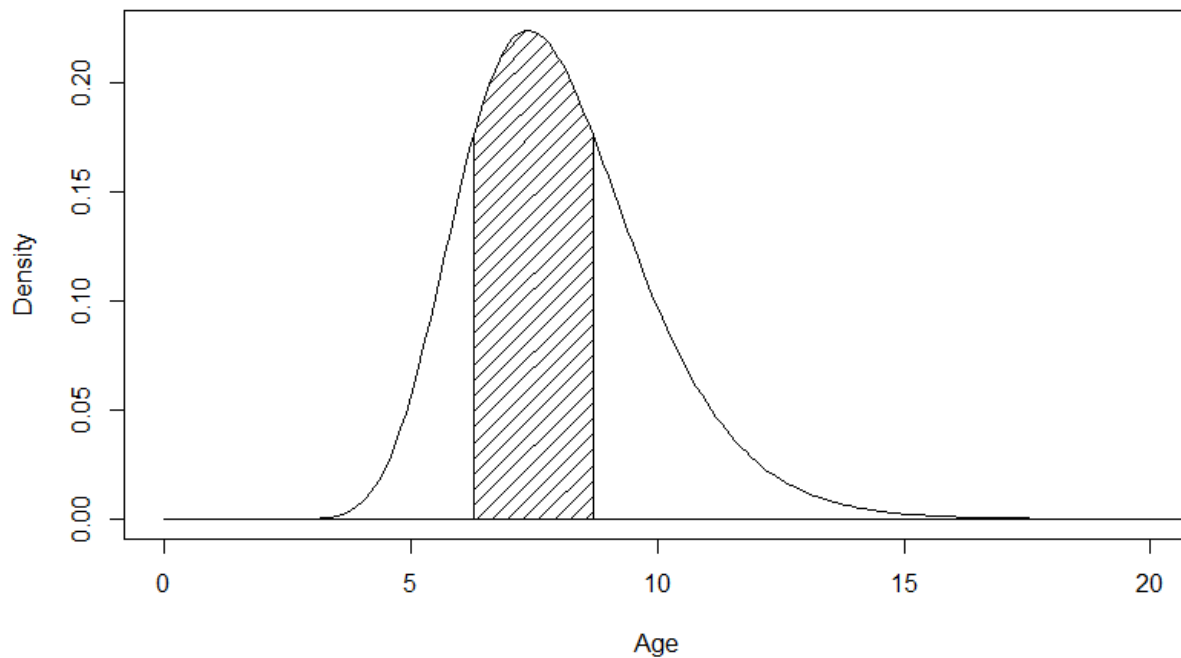
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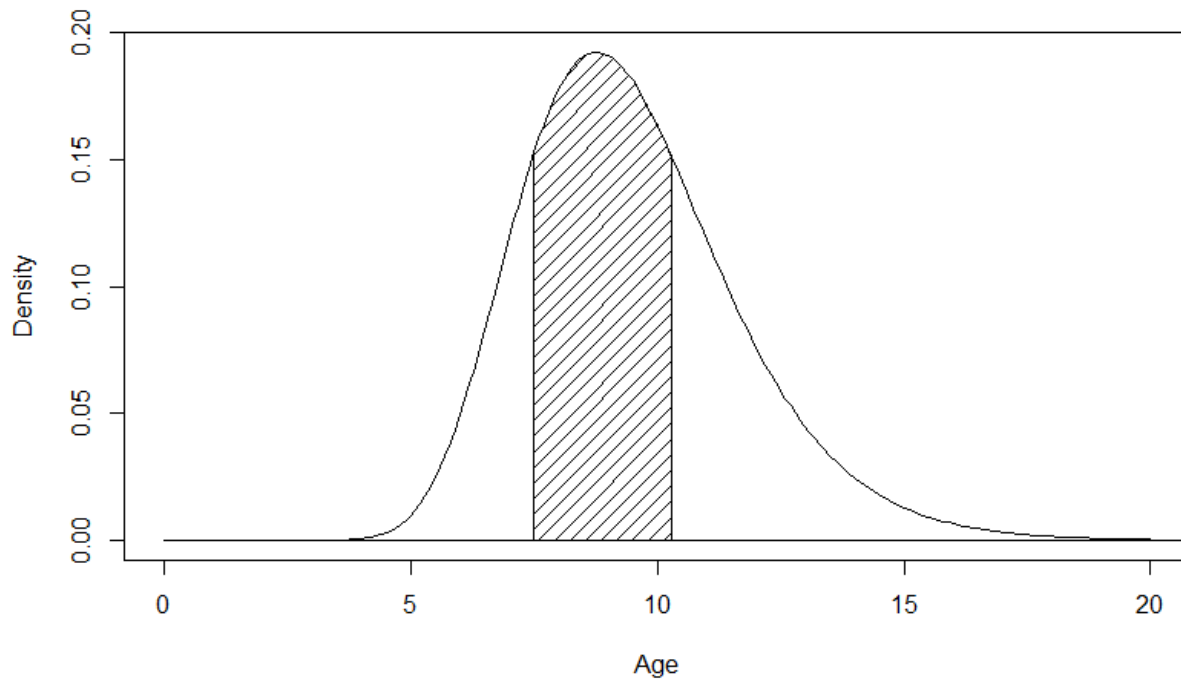
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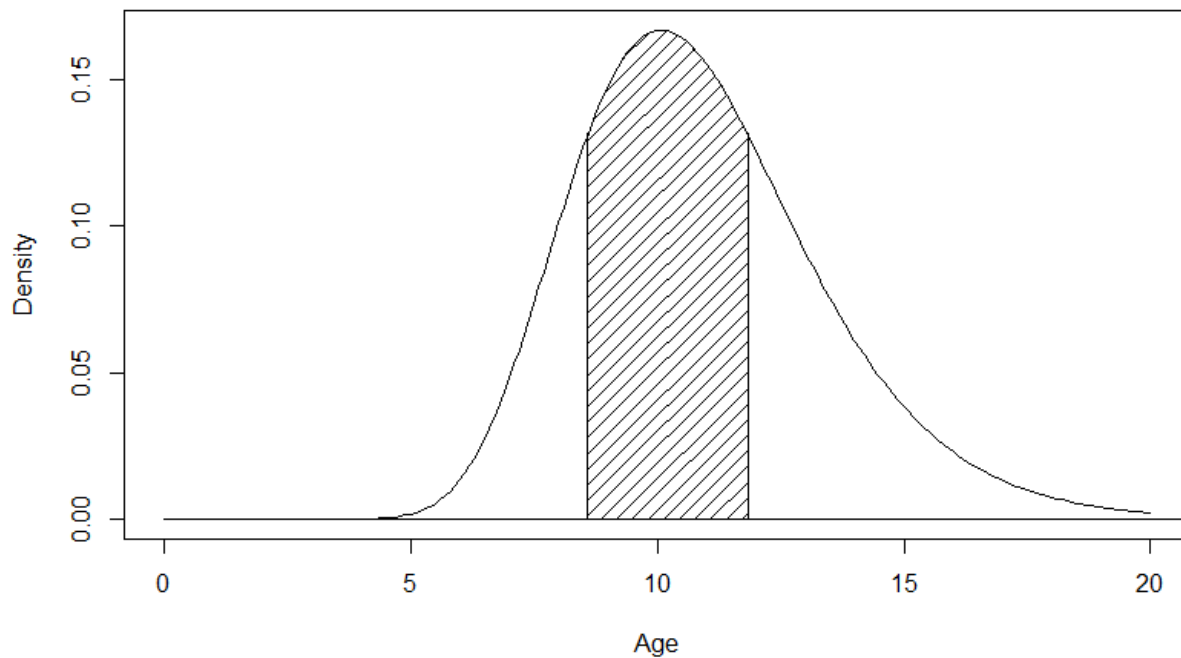
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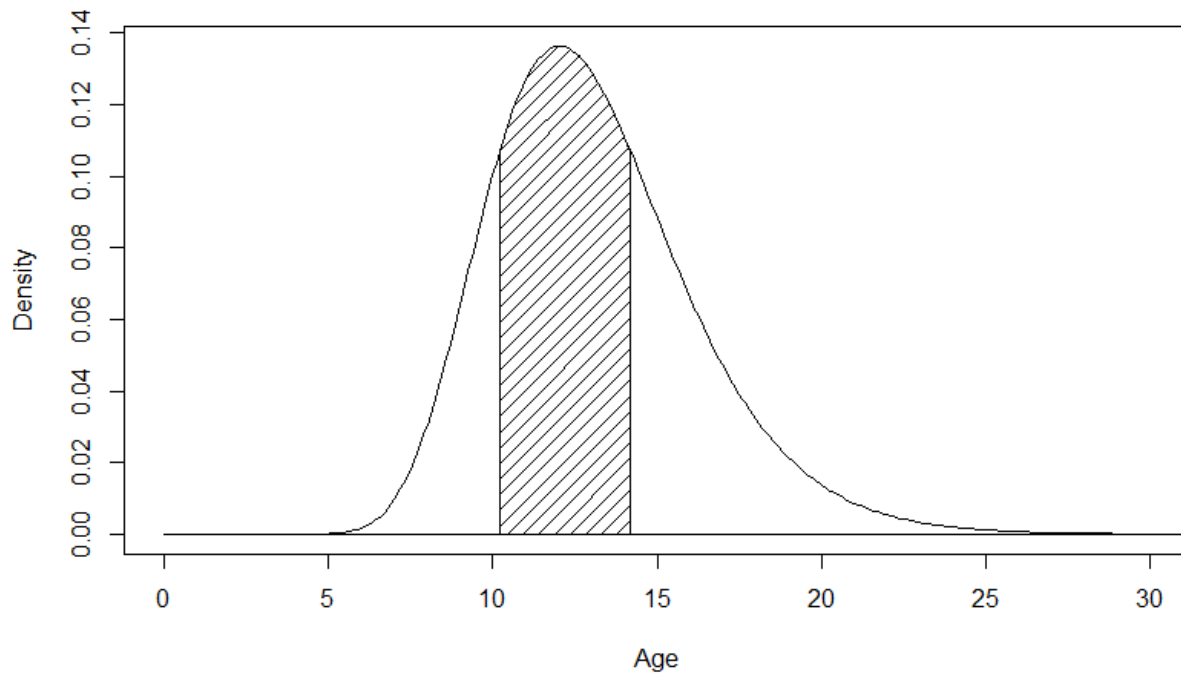
HPD male lower canine stage 11 (R 3/4), US Violent Deaths 2010 prior



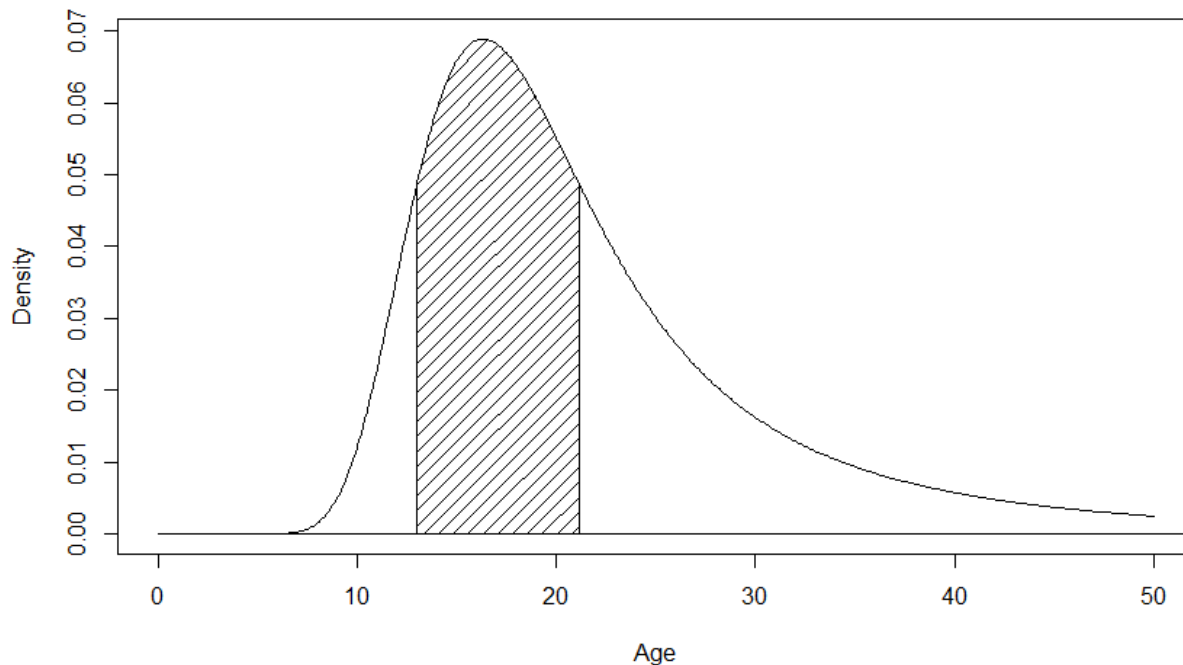
HPD male lower canine stage 12 (Rc), US Violent Deaths 2010 prior



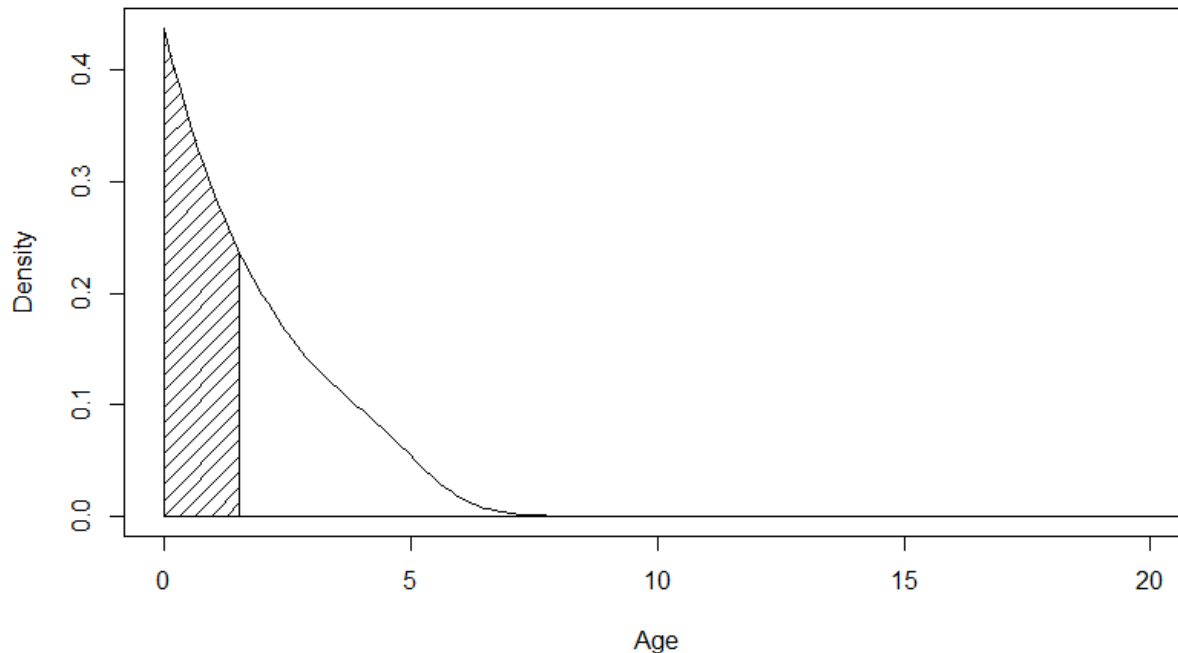
HPD male lower canine stage 13 (A 1/2), US Violent Deaths 2010 prior



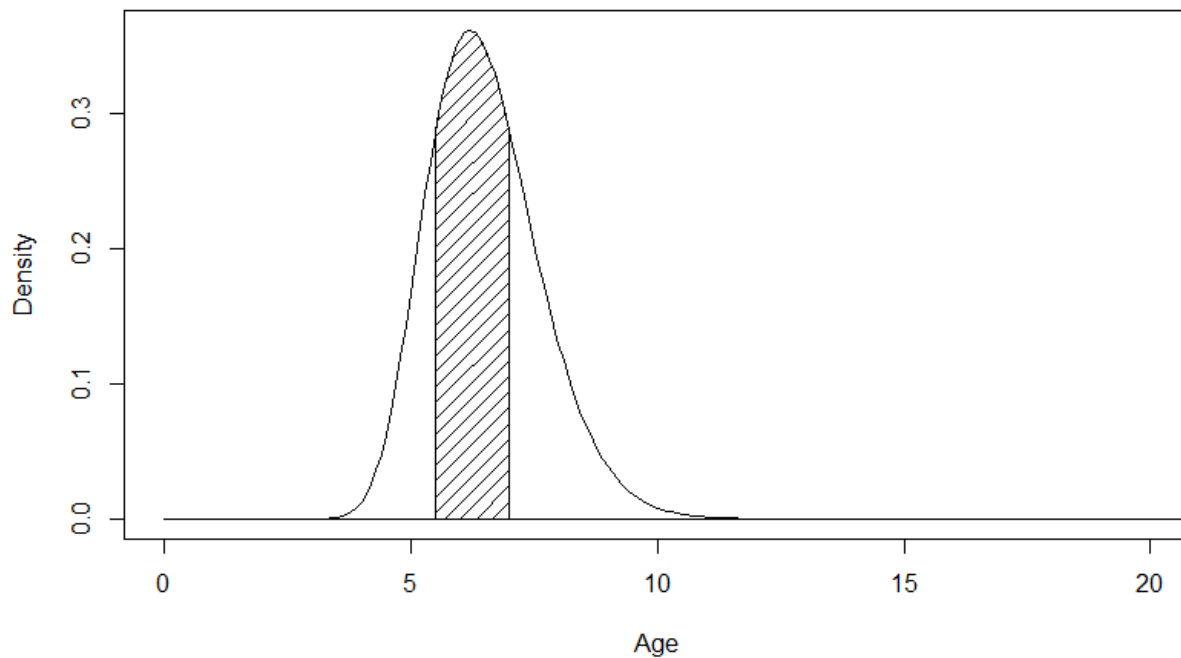
HPD male lower canine stage 14 (Ac), US Violent Deaths 2010 prior



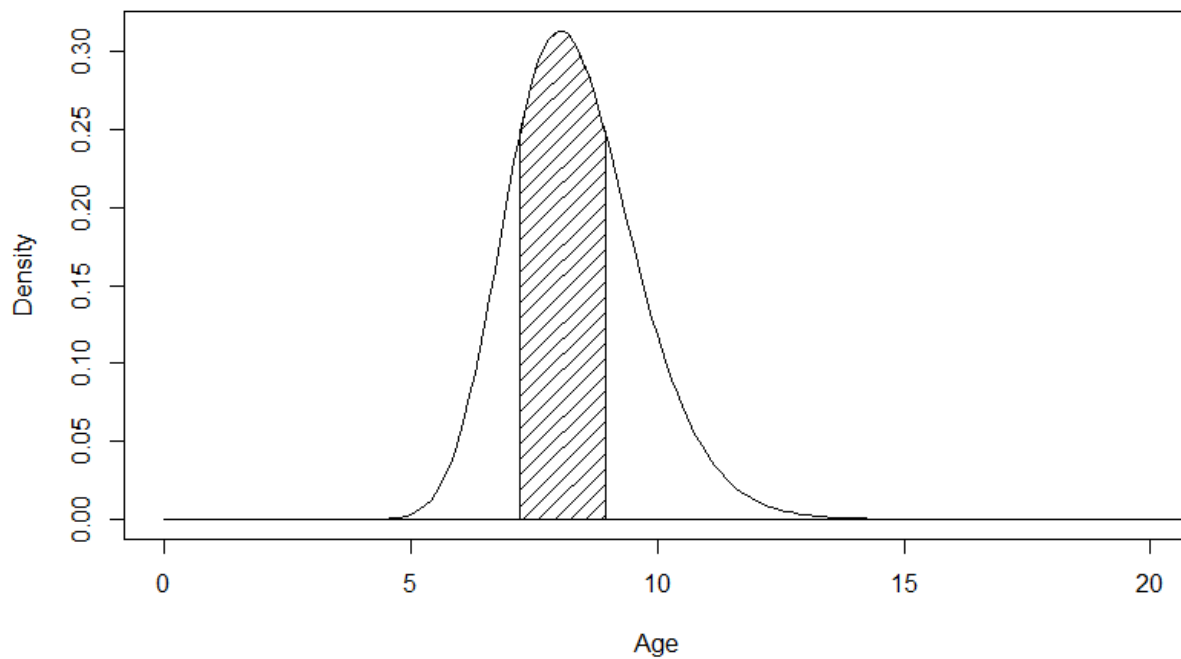
HPD male lower premolar 1 stage 6 (Crc), US Violent Deaths 2010 prior



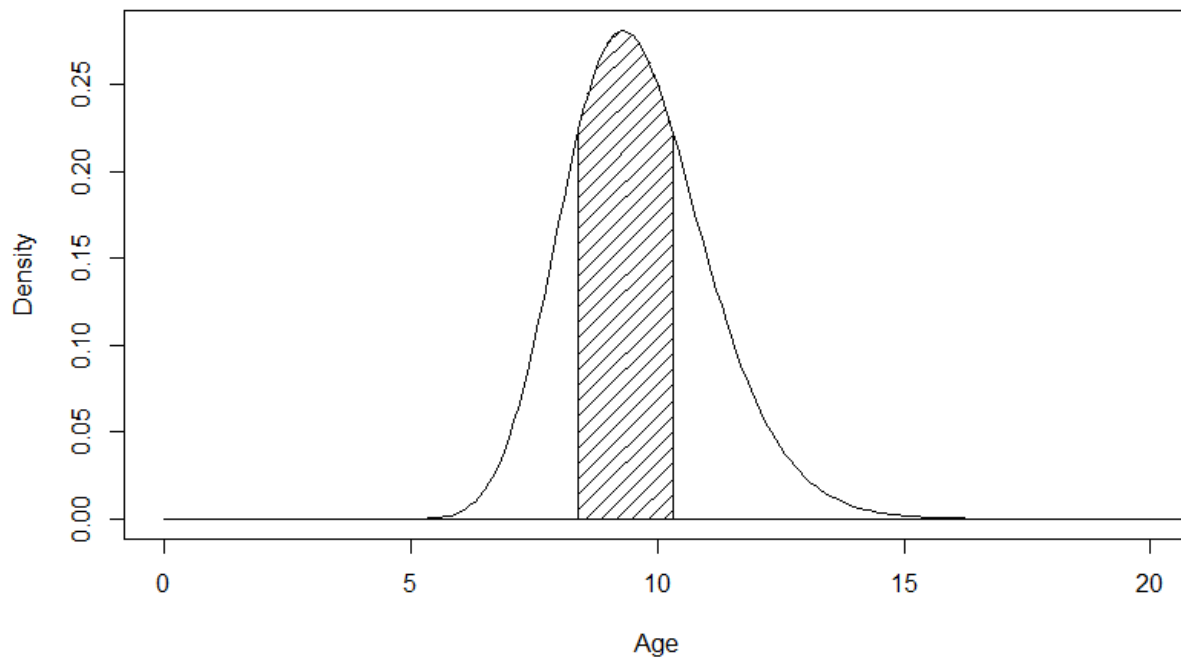
HPD male lower premolar 1 stage 7 (Ri), US Violent Deaths 2010 prior



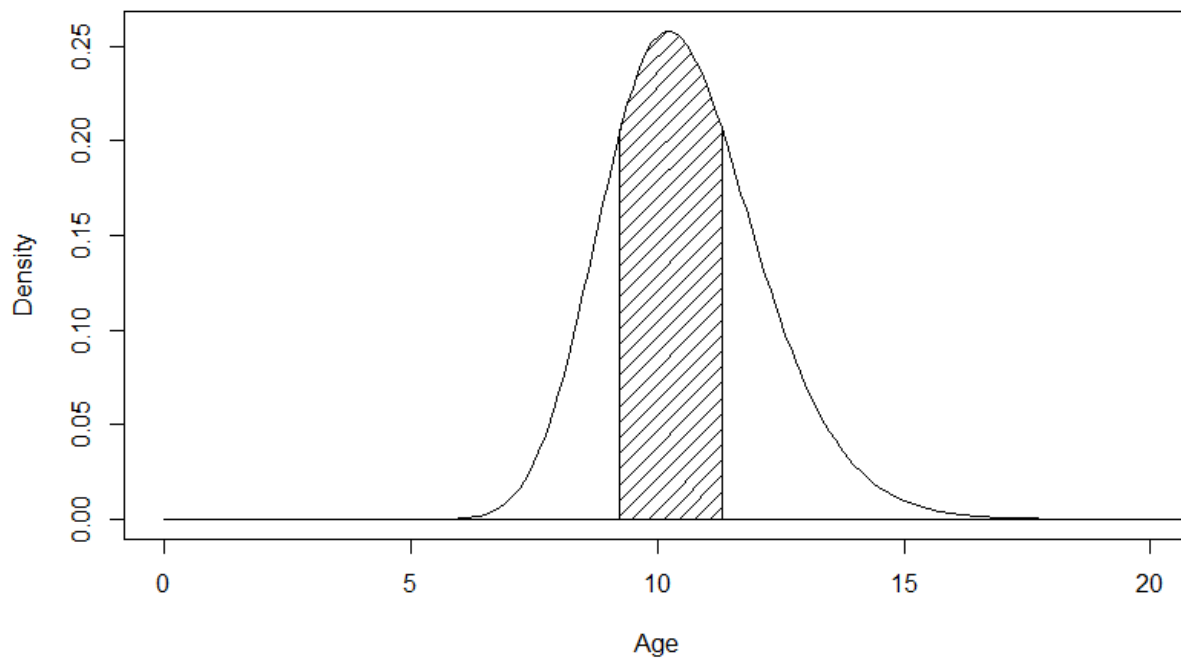
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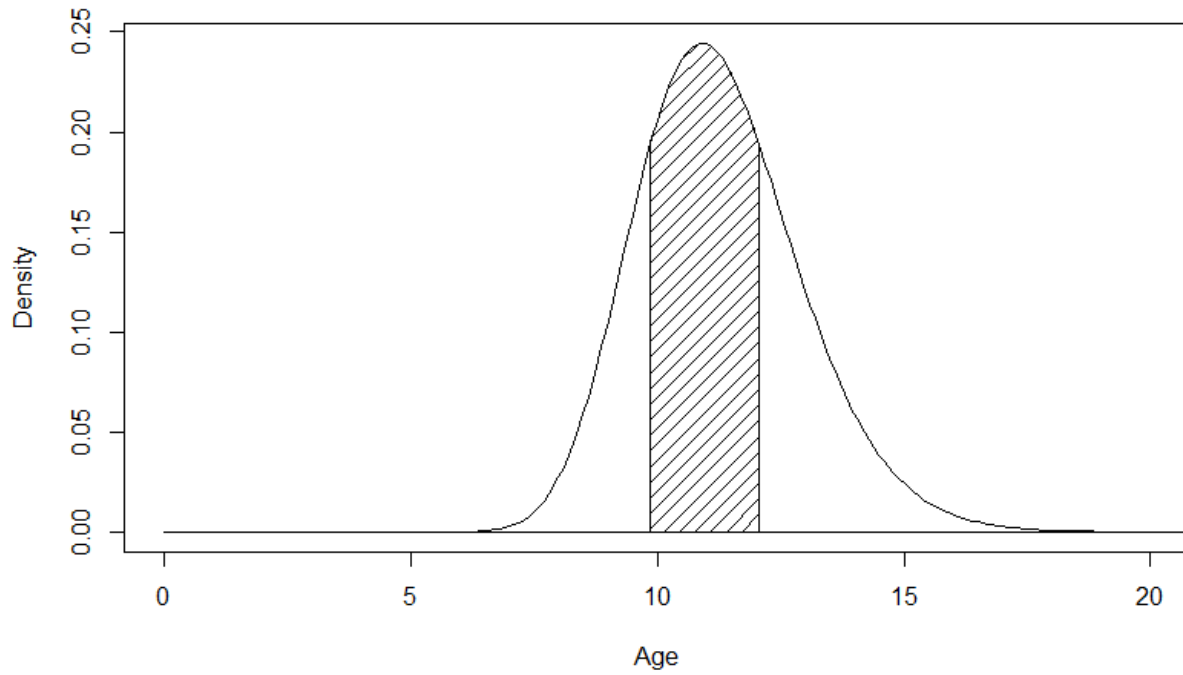
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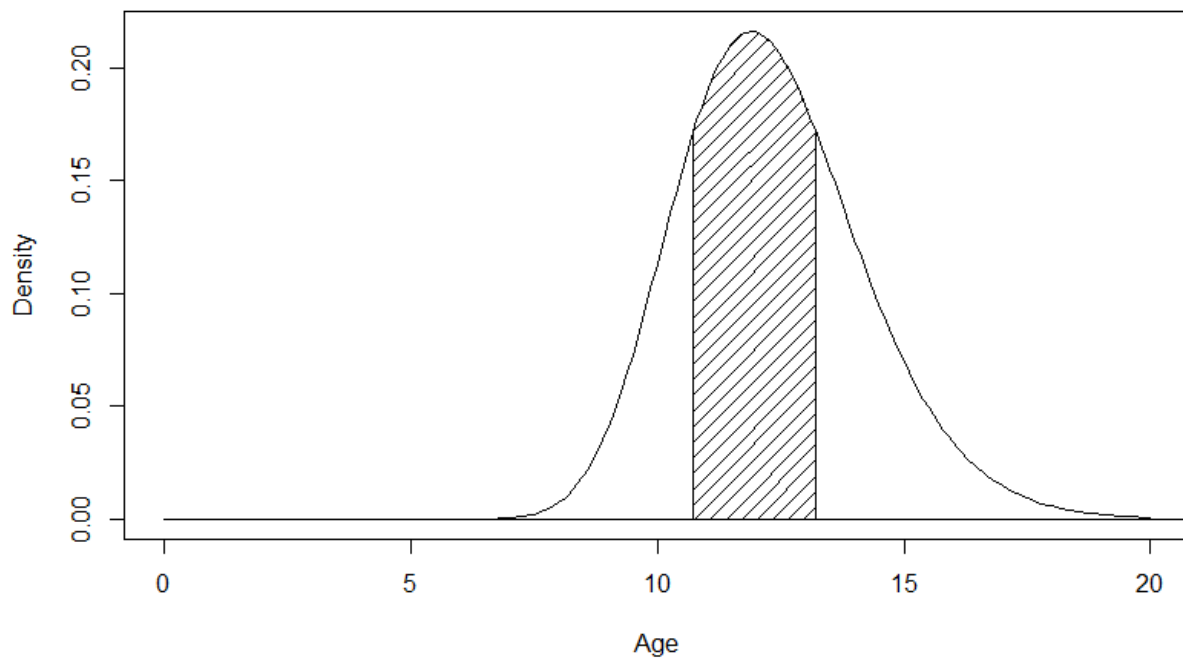
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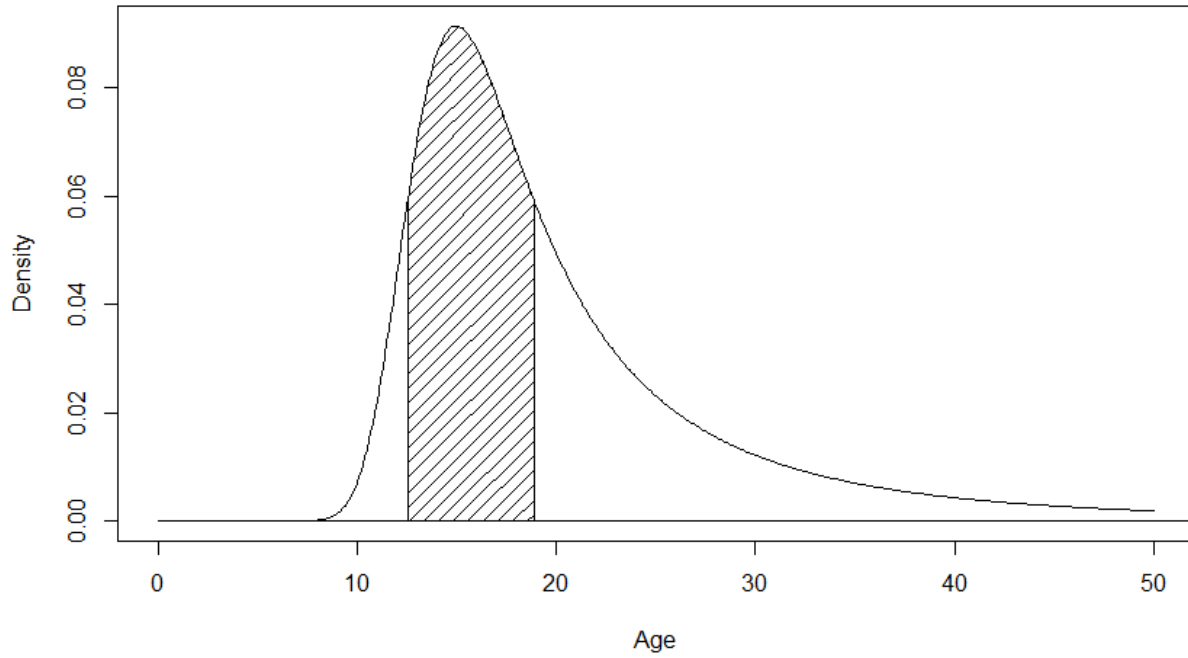
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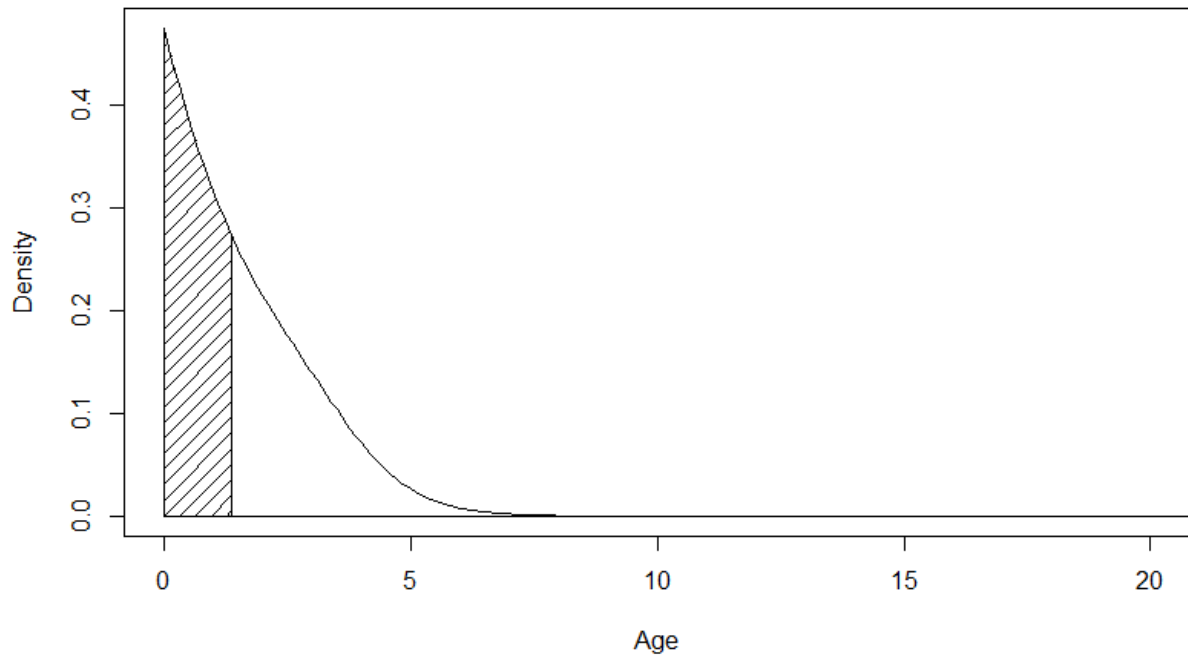
HPD male lower premolar 1 stage 13 (A 1/2), US Violent Deaths 2010 prior



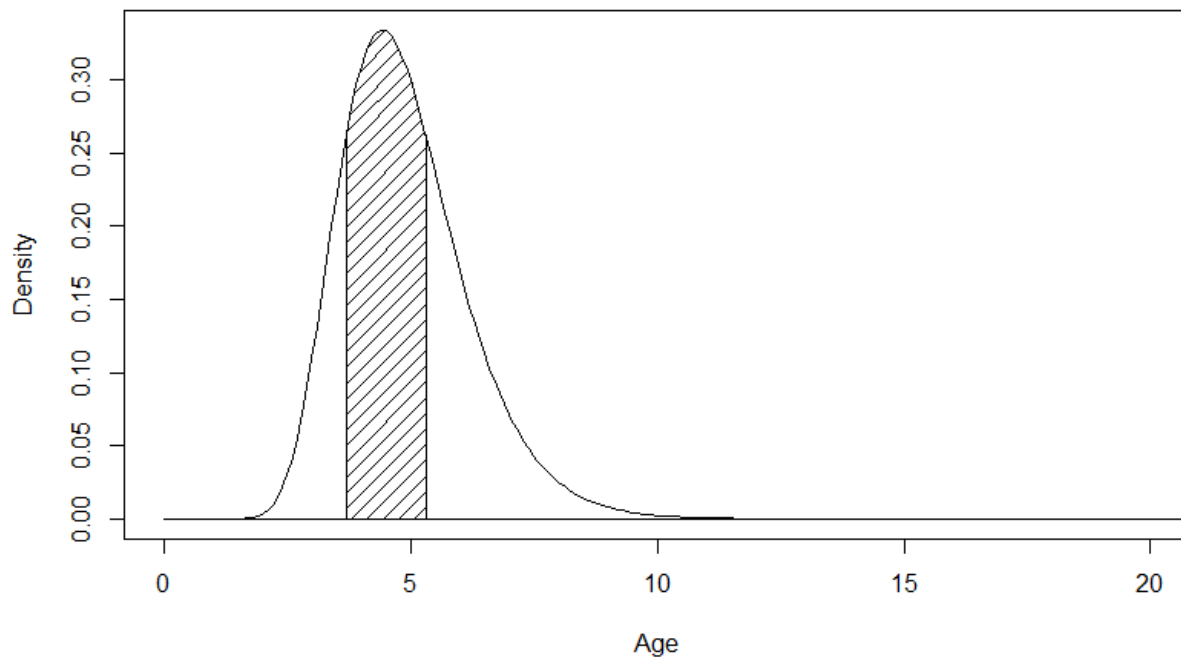
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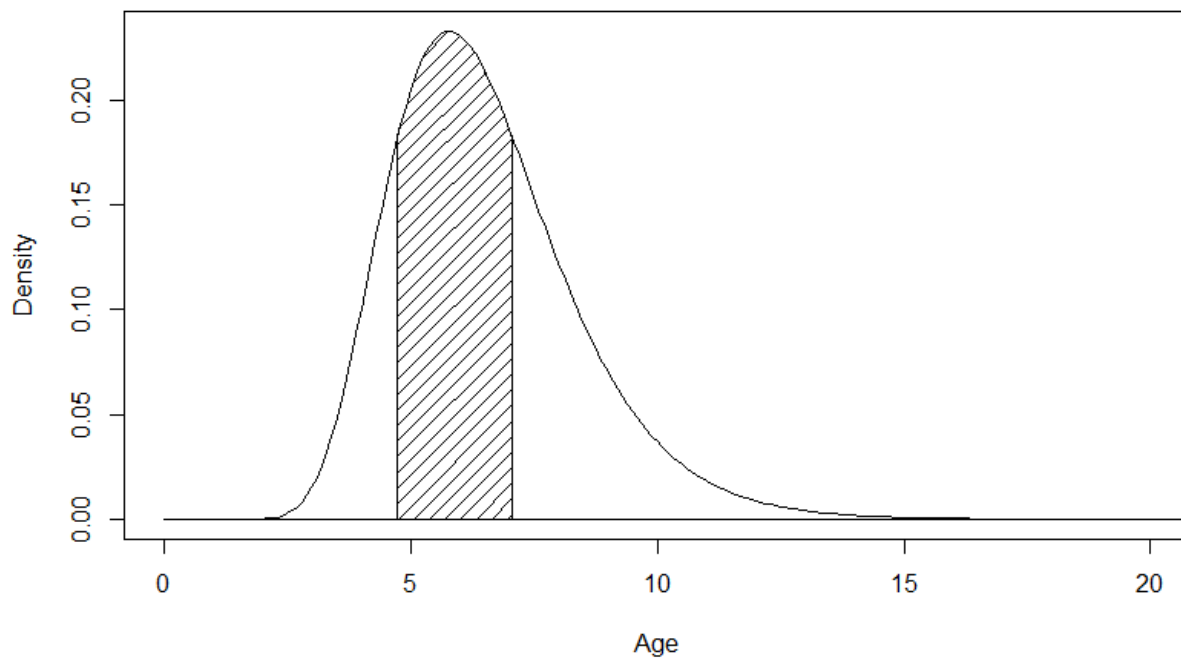
HPD male lower premolar 2 stage 5 (Cr 3/4), US Violent Deaths 2010 prior



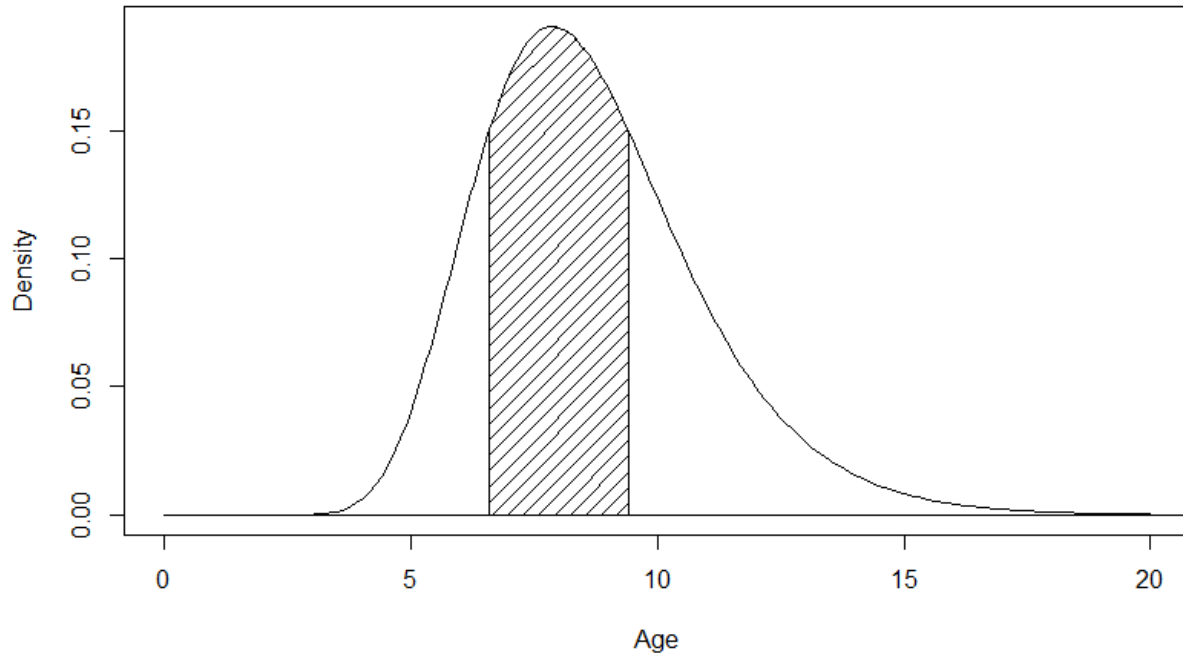
HPD male lower premolar 2 stage 6 (Crc), US Violent Deaths 2010 prior



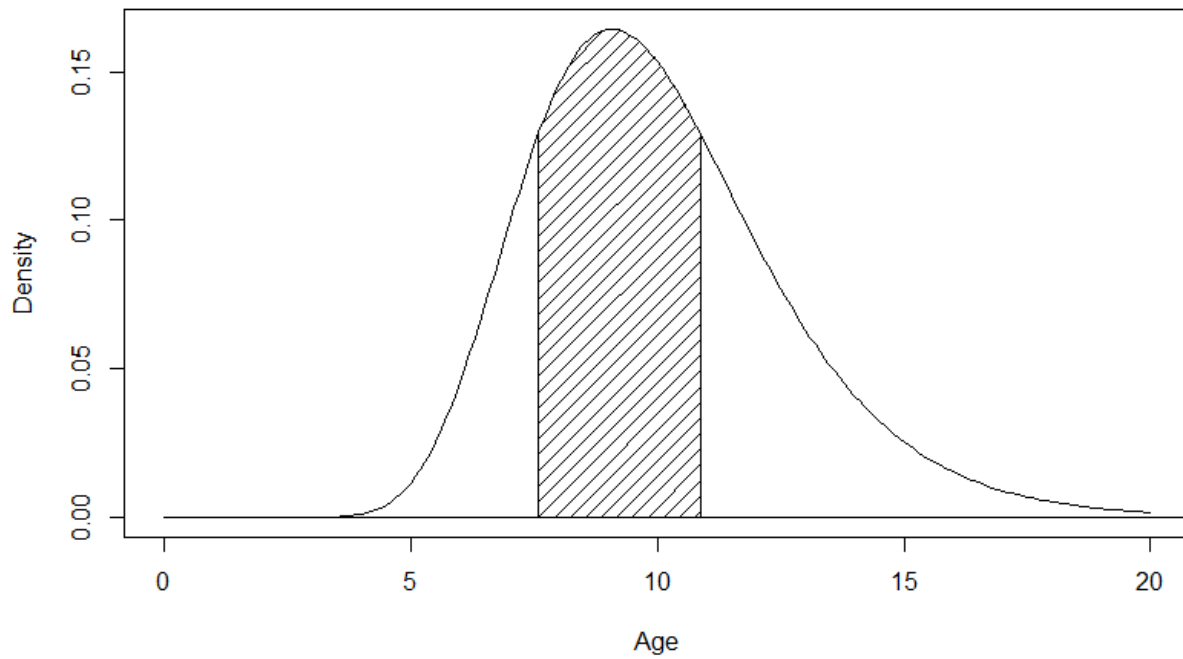
HPD male lower premolar 2 stage 7 (Ri), US Violent Deaths 2010 prior



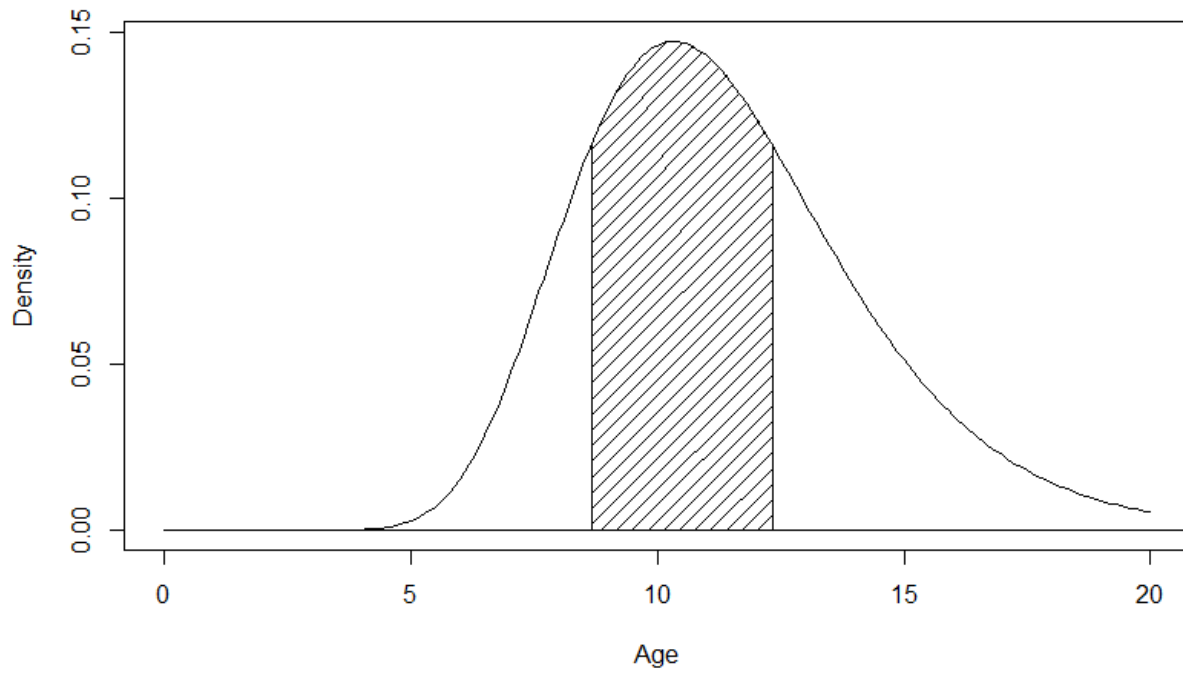
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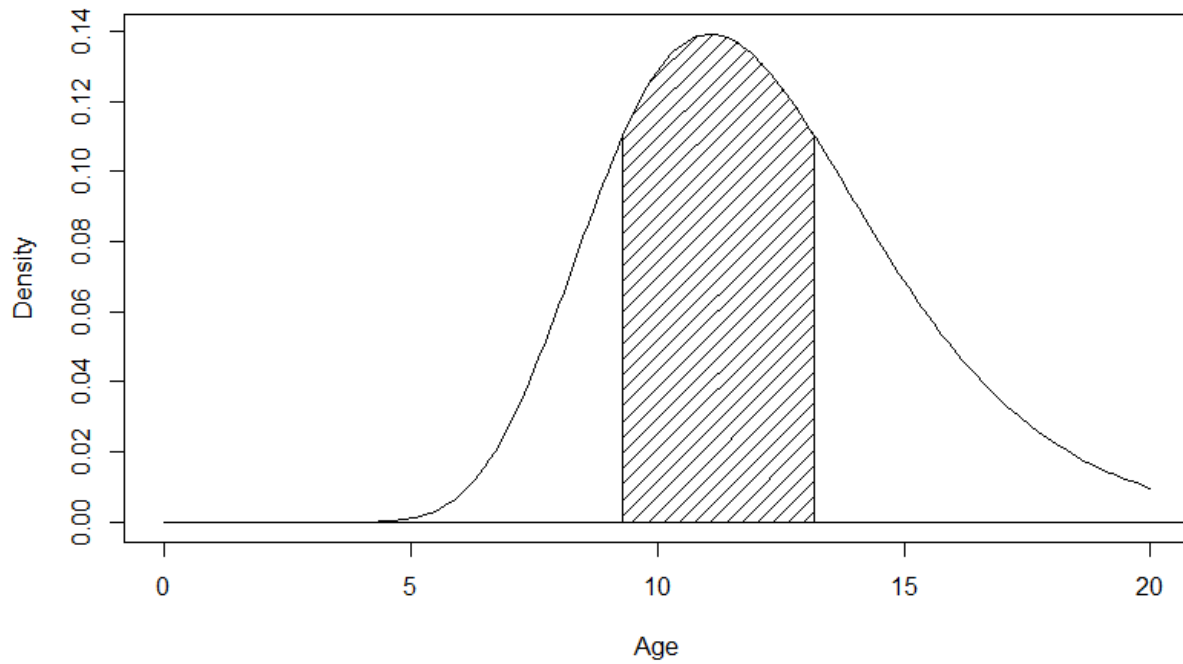
HPD male lower premolar 2 stage 10 (R 1/2), US Violent Deaths 2010 prior



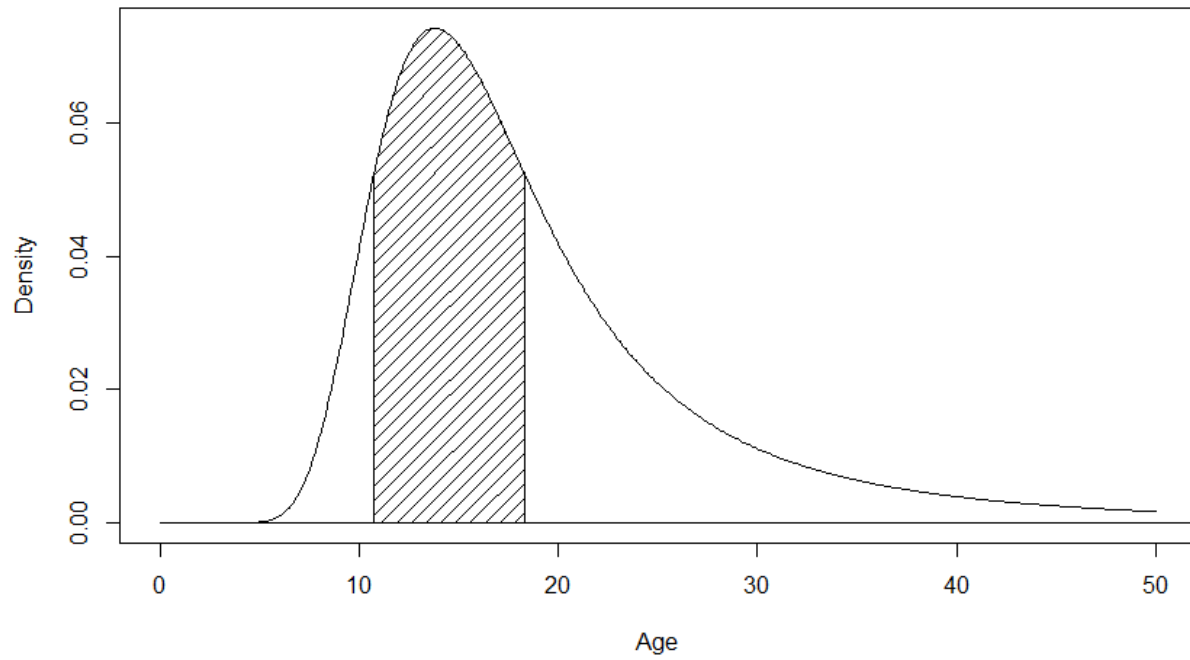
HPD male lower premolar 2 stage 11 (R 3/4), US Violent Deaths 2010 prior



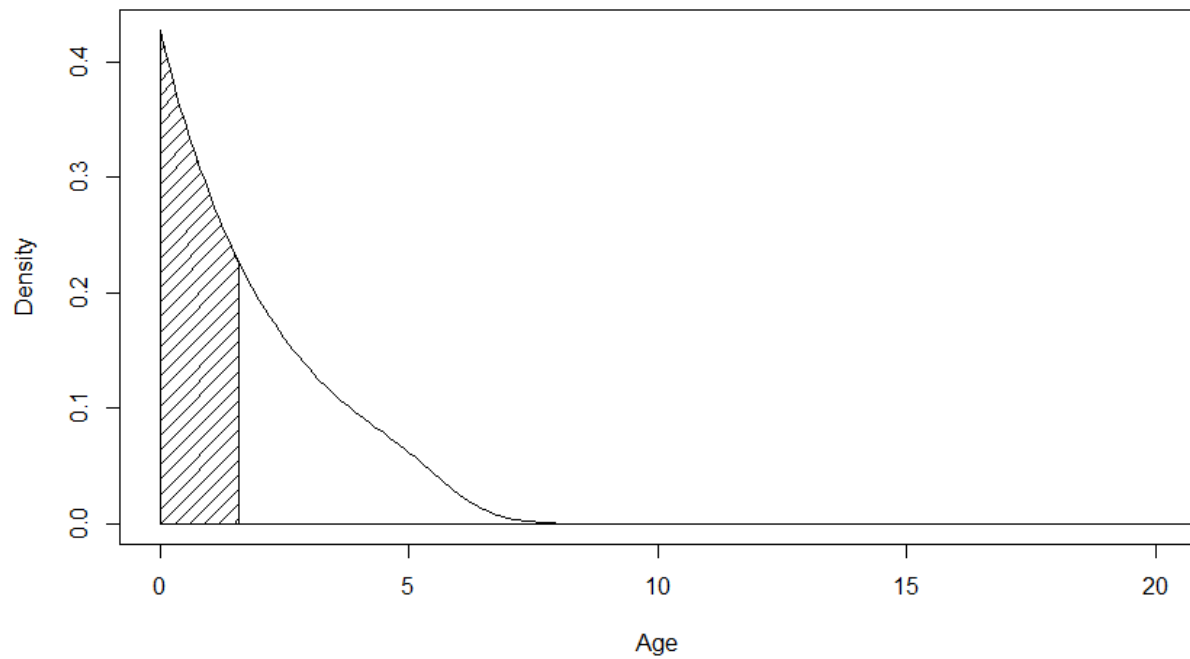
HPD male lower premolar 2 stage 12 (Rc), US Violent Deaths 2010 prior



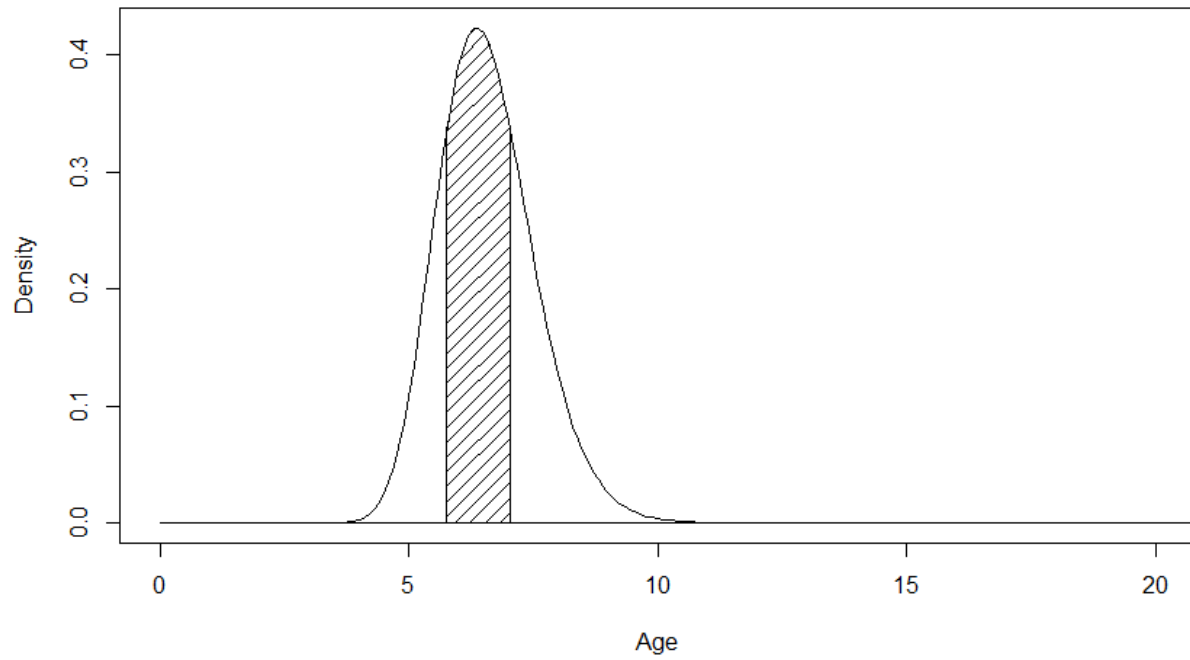
HPD male lower premolar 2 stage 13 (A1/2), US Violent Deaths 2010 prior



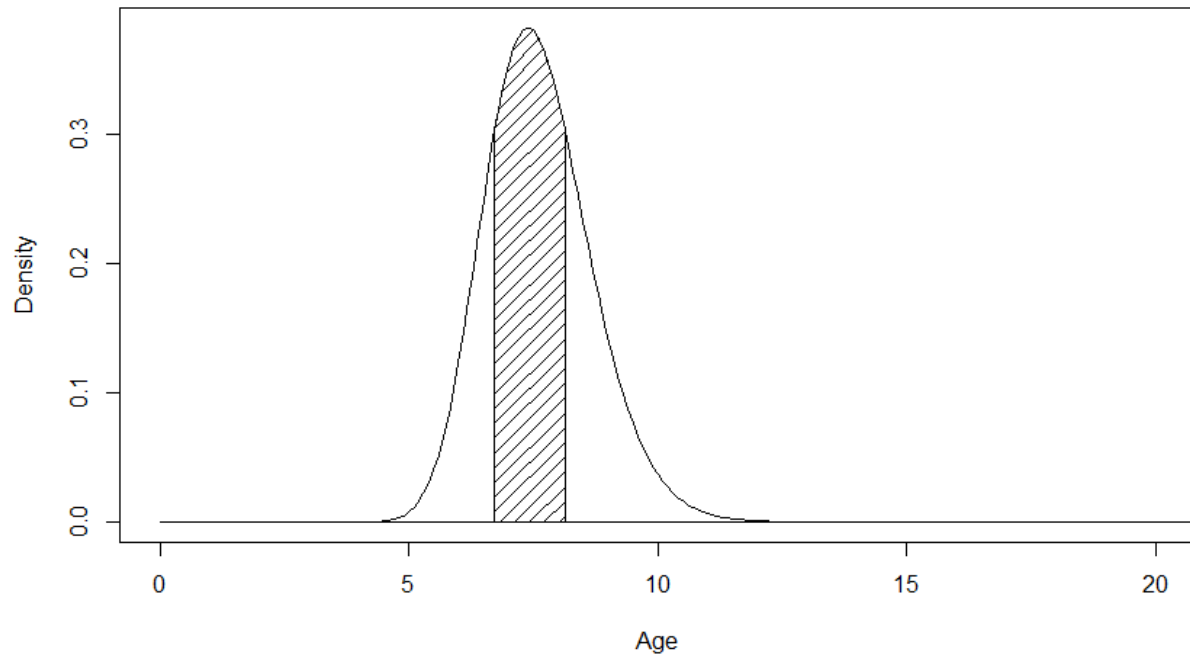
HPD male lower molar 1 stage 10 (R1/2), US Violent Deaths 2010 prior



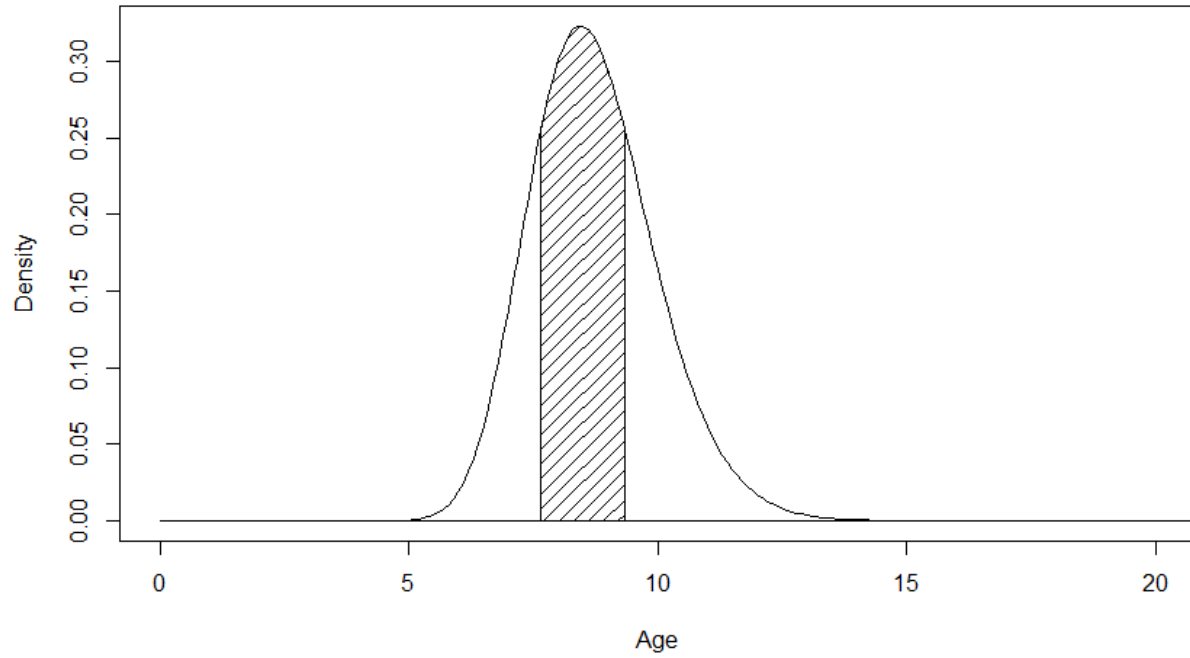
HPD male lower molar 1 stage 11 (R3/4), US Violent Deaths 2010 prior



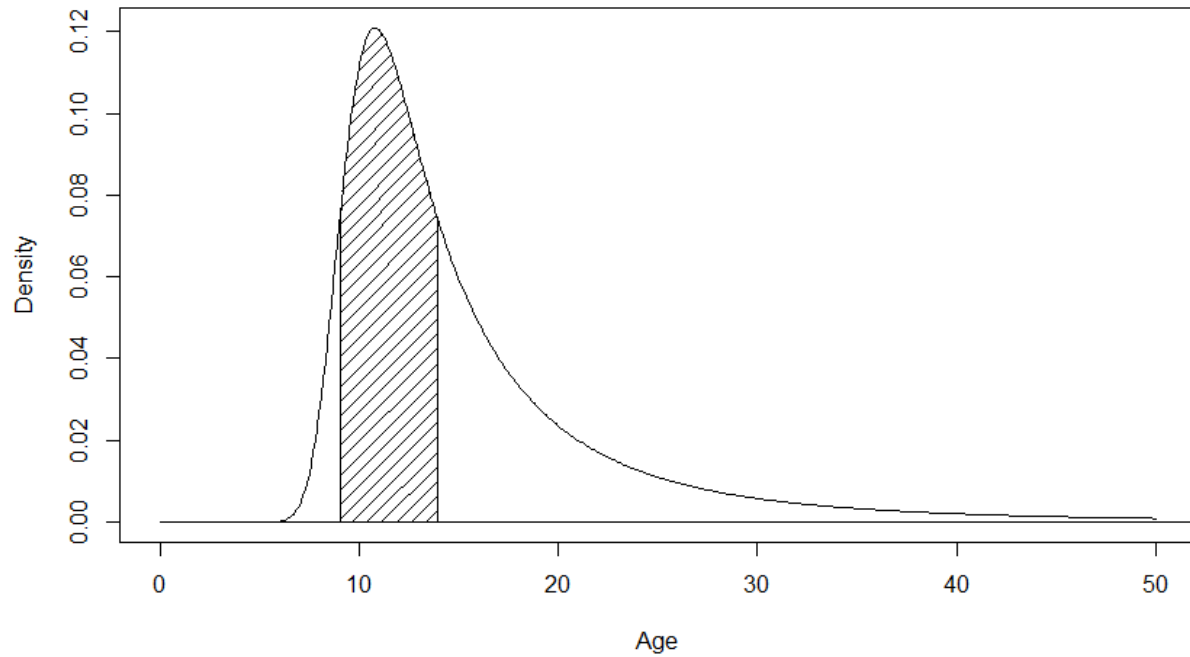
HPD male lower molar 1 stage 12 (Rc), US Violent Deaths 2010 prior



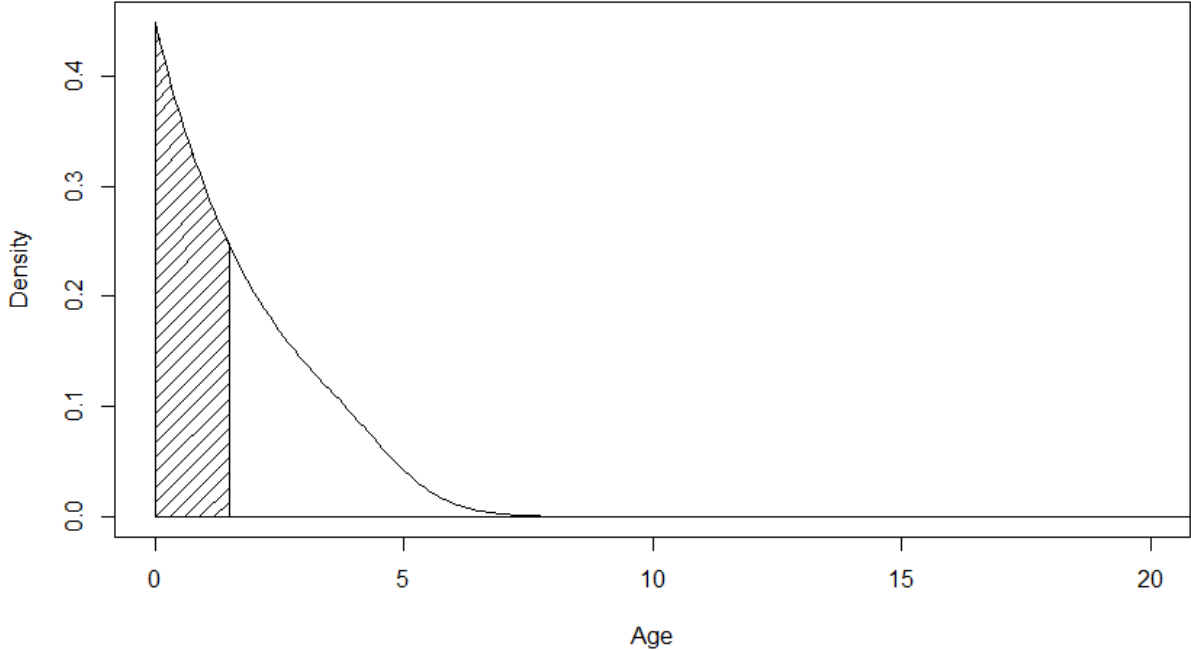
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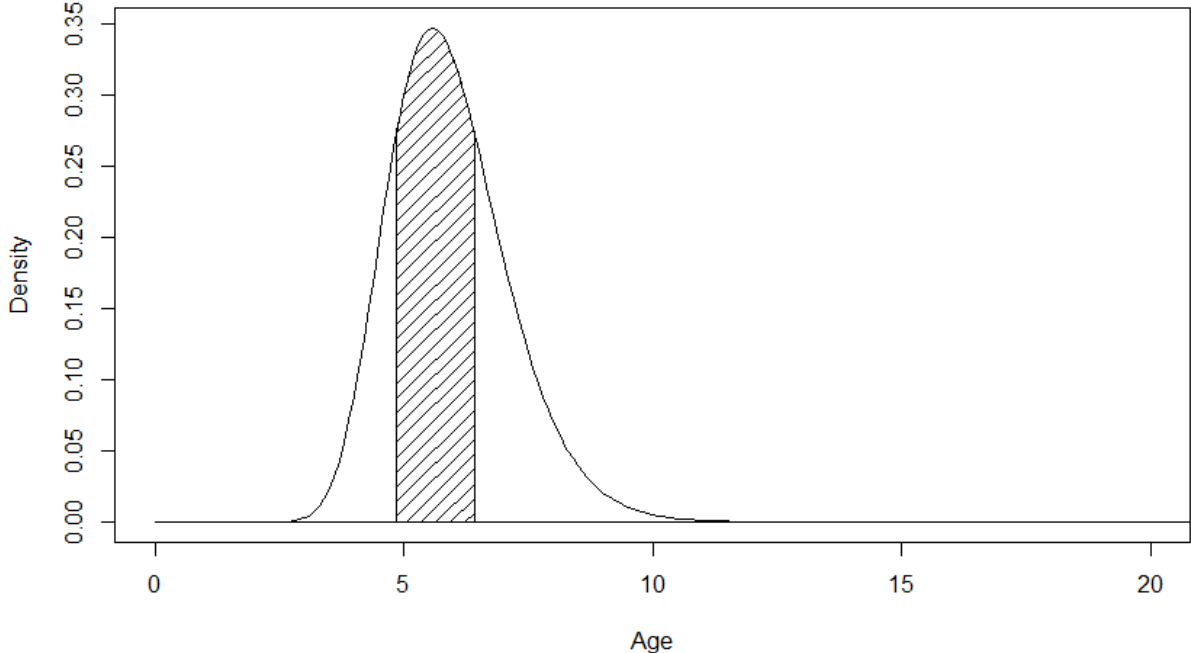
HPD male lower molar 1 stage 14 (Ac), US Violent Deaths 2010 prior



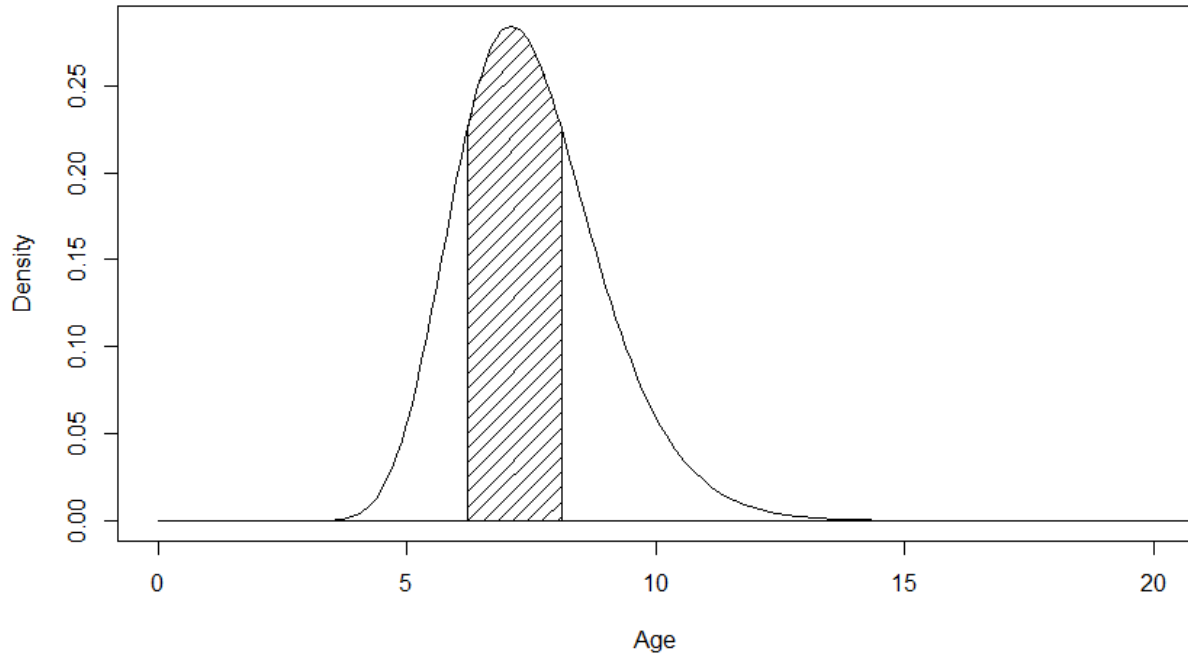
HPD male lower molar 2 stage 5 (Cr3/4), US Violent Deaths 2010 prior



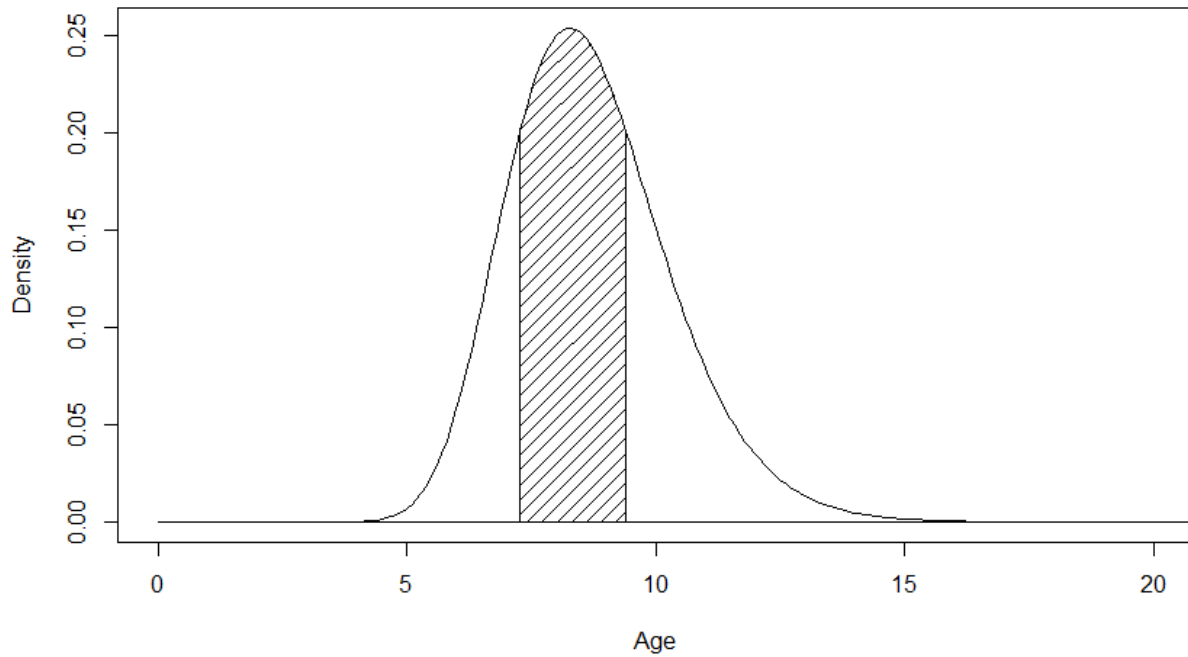
HPD male lower molar 2 stage 6 (Crc), US Violent Deaths 2010 prior



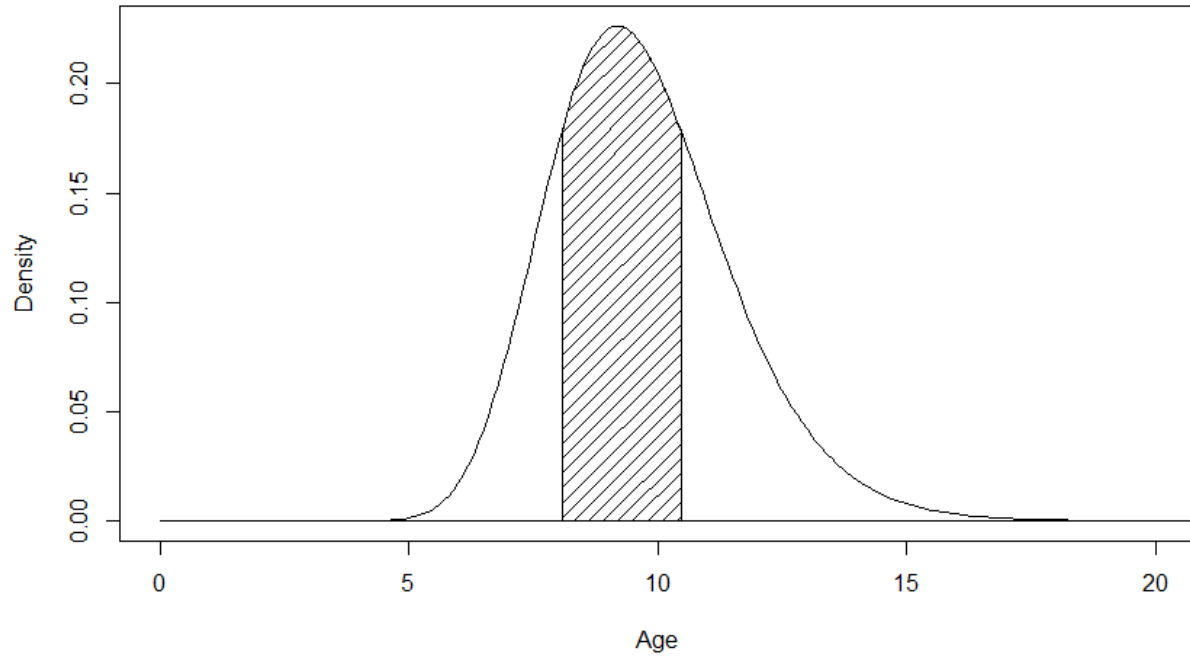
HPD male lower molar 2 stage 7 (Ri), US Violent Deaths 2010 prior



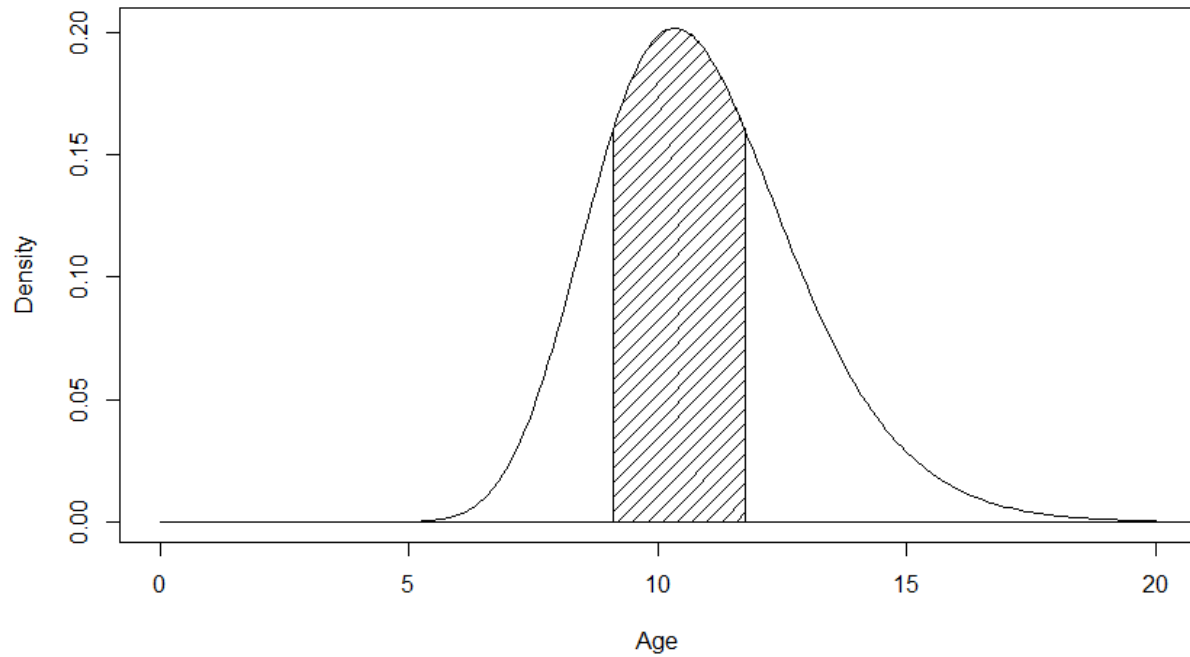
HPD male lower molar 2 stage 8 (C1i), US Violent Deaths 2010 prior



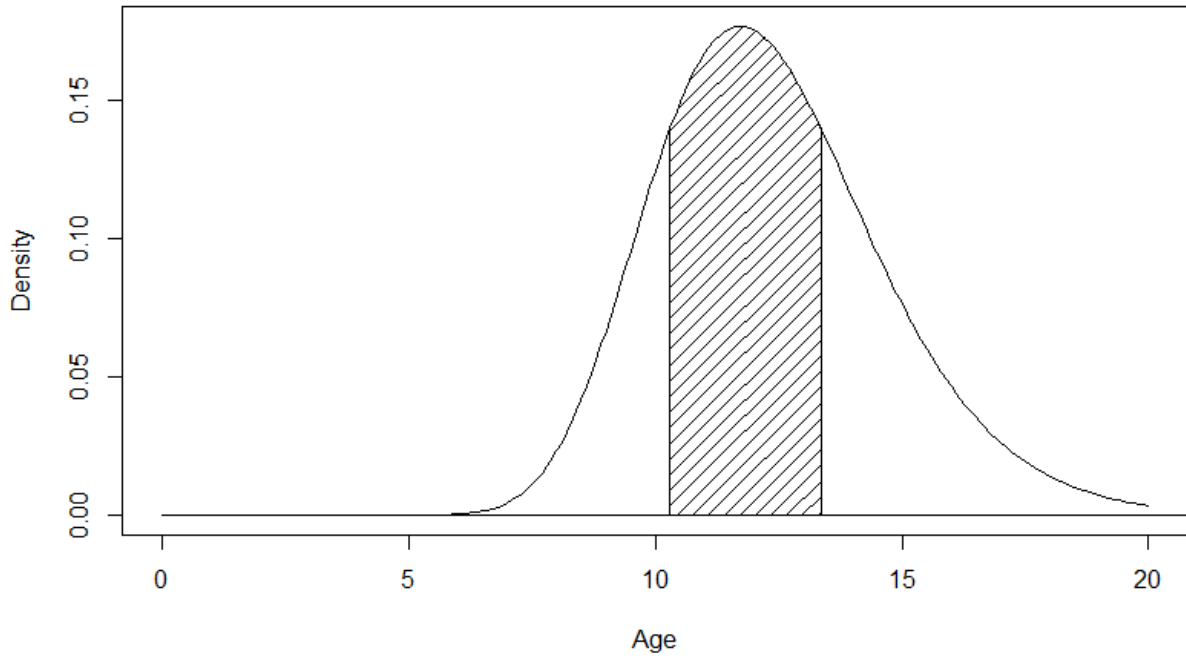
HPD male lower molar 2 stage 9 (R1/4), US Violent Deaths 2010 prior



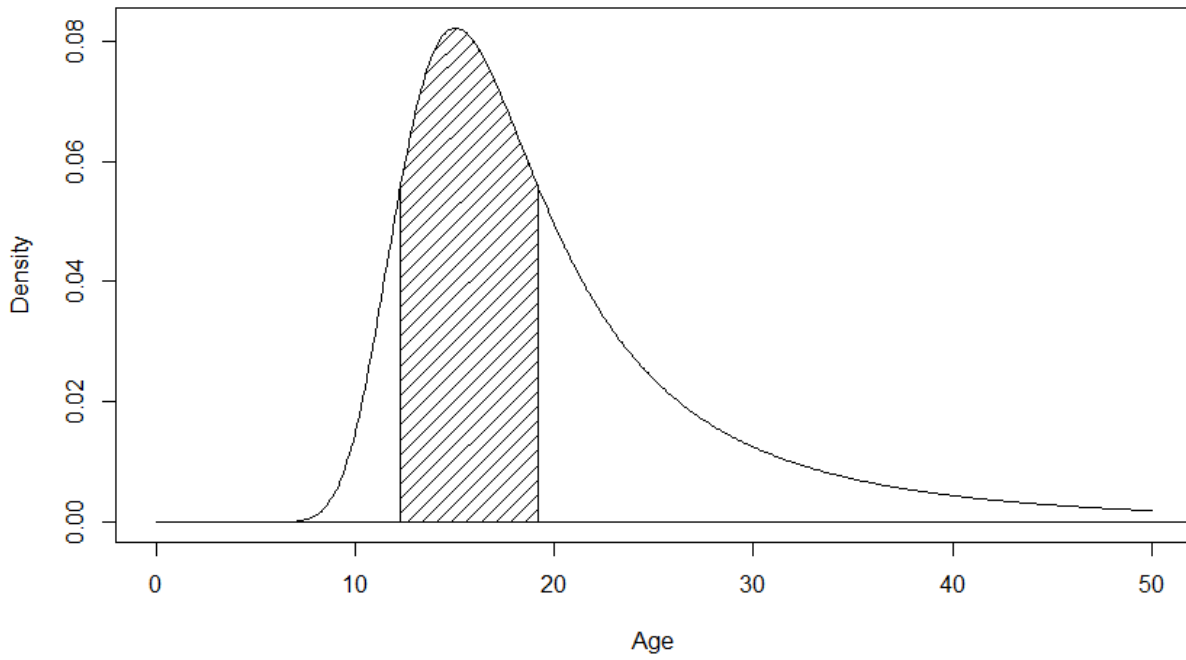
HPD male lower molar 2 stage 10 (R1/2), US Violent Deaths 2010 prior



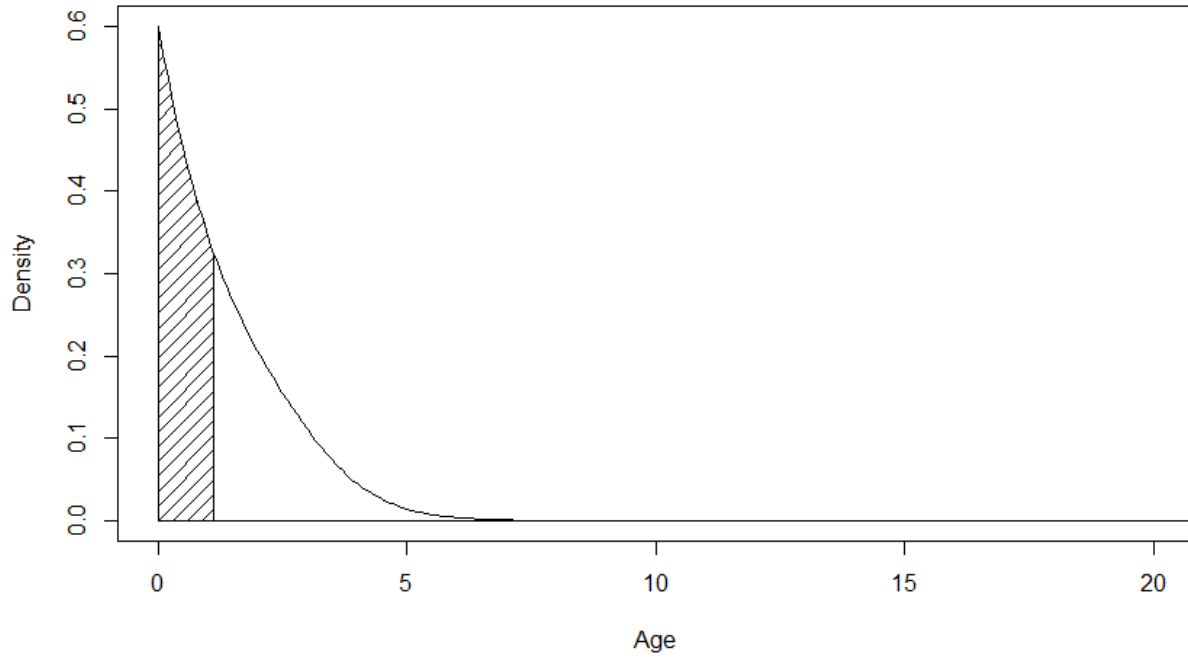
HPD male lower molar 2 stage 11 (R 3/4), US Violent Deaths 2010 prior



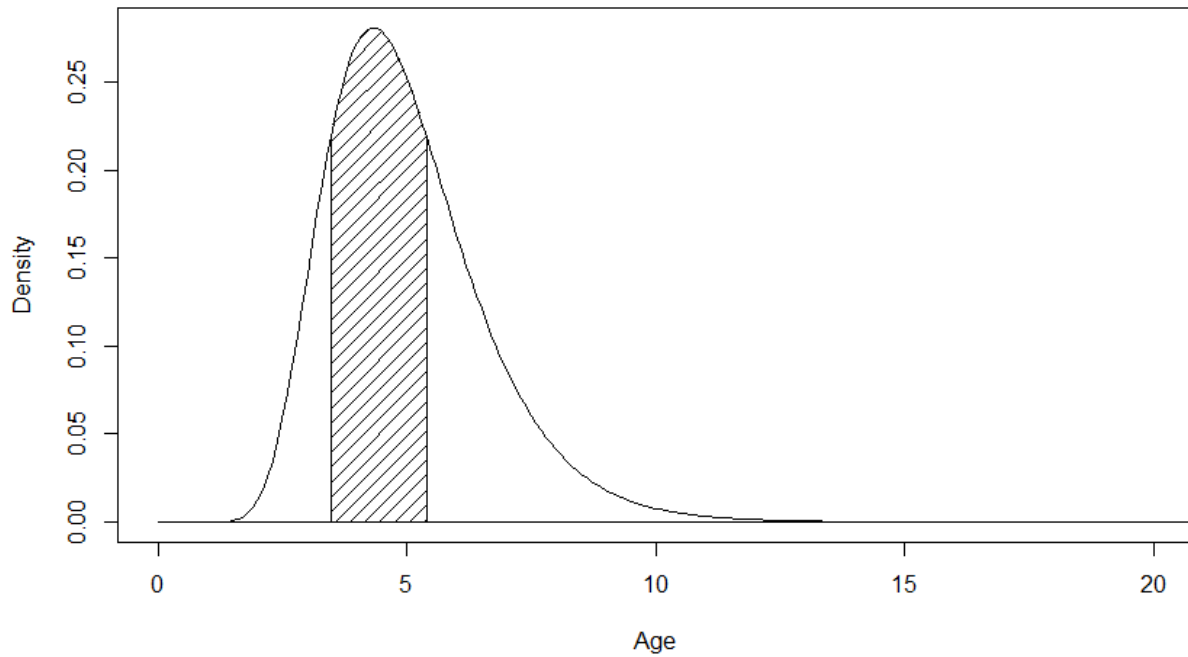
HPD male lower molar 2 stage 12 (Rc), US Violent Deaths 2010 prior



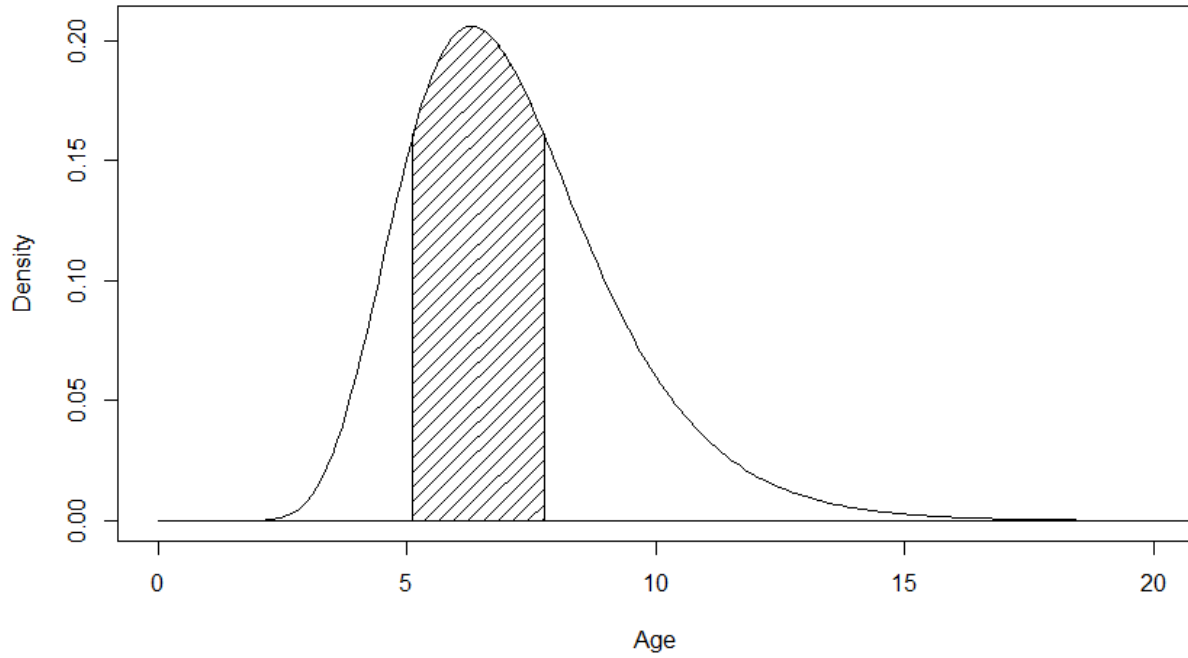
HPD male upper incisor 1 stage 10 (R 1/2), US Deaths 2008 prior



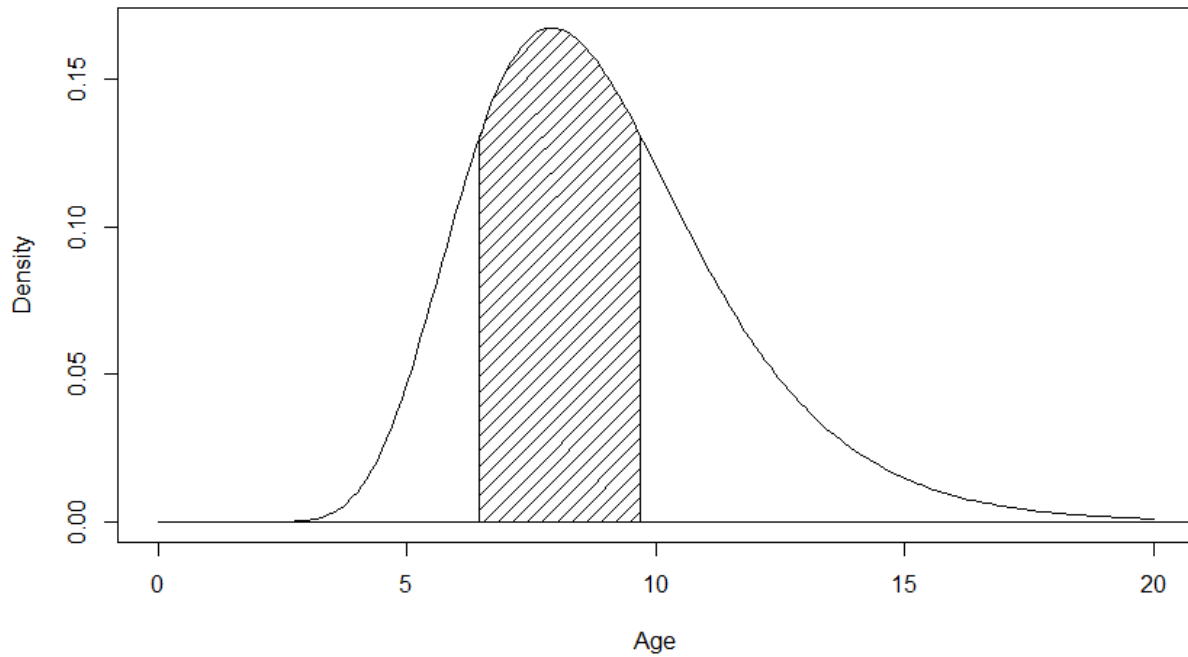
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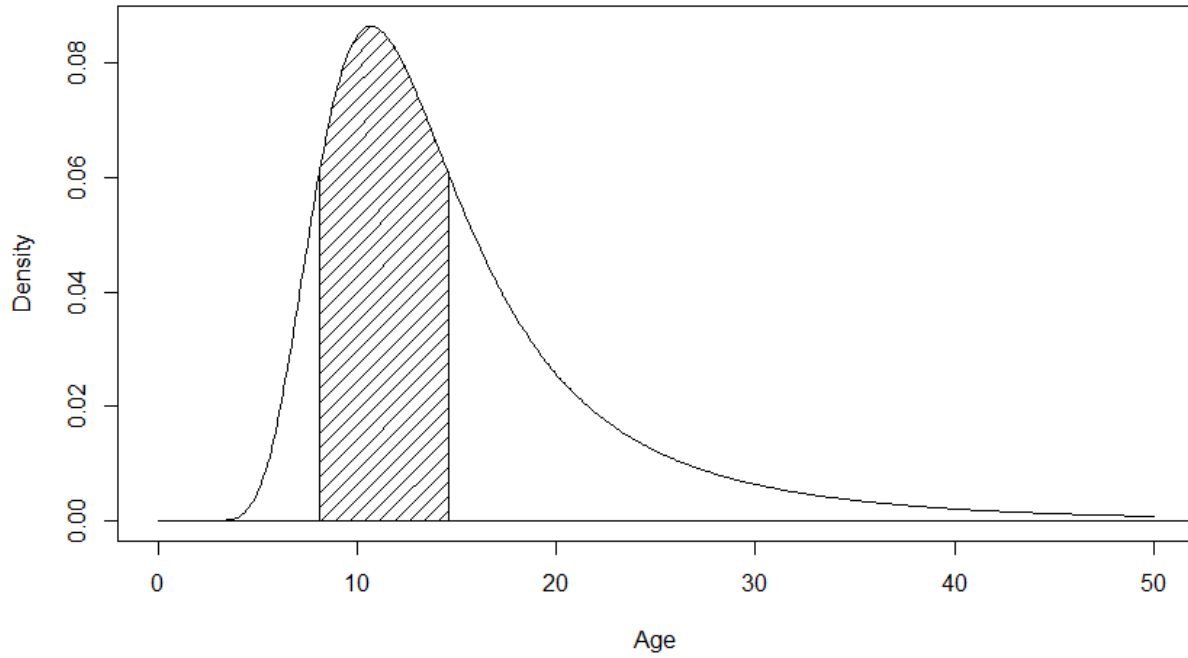
HPD male upper incisor 1 stage 12 (Rc), US Deaths 2008 prior



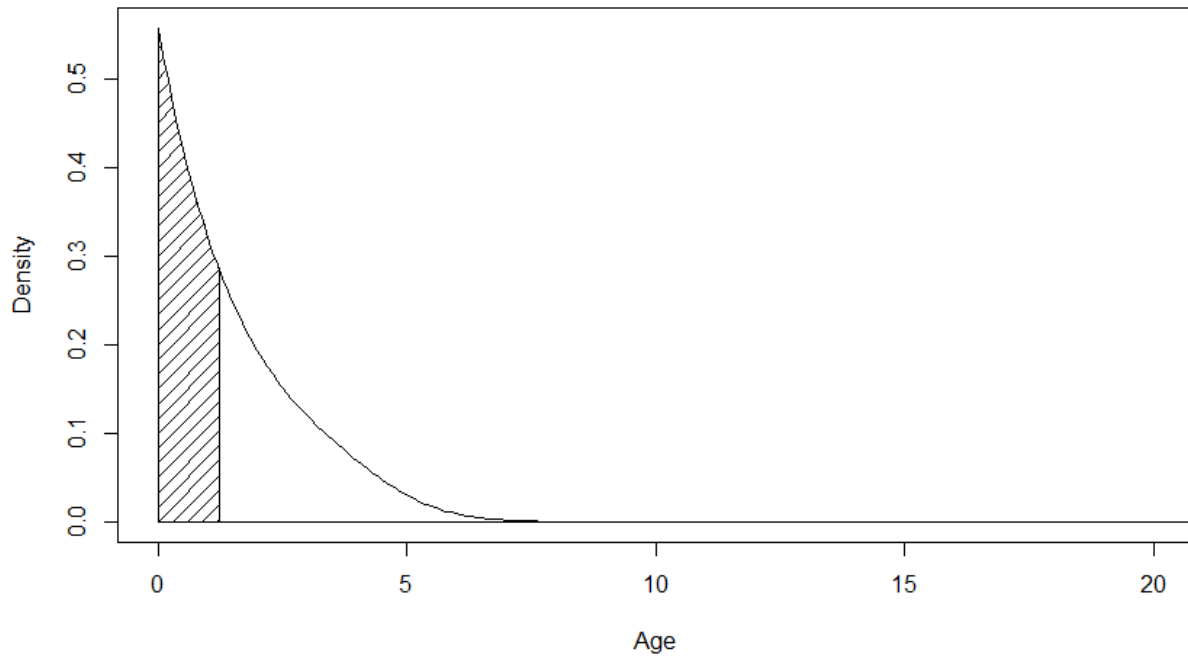
HPD male upper incisor 1 stage 13 (A 1/2), US Deaths 2008 prior



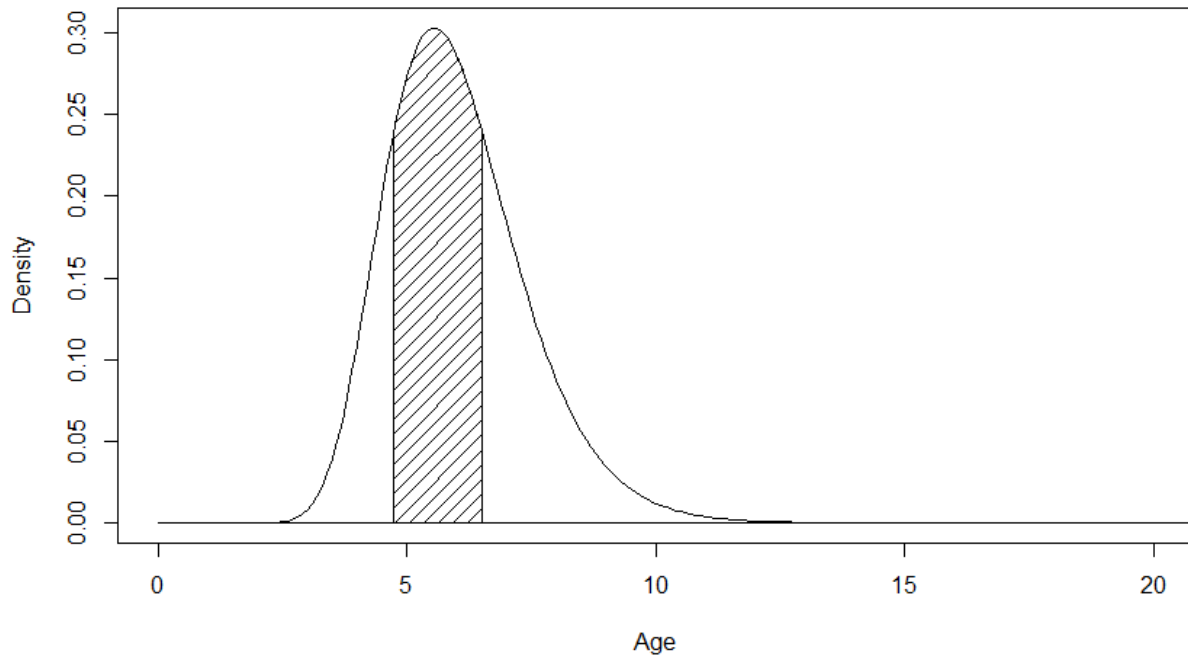
HPD male upper incisor 1 stage 14 (Ac), US Deaths 2008 prior



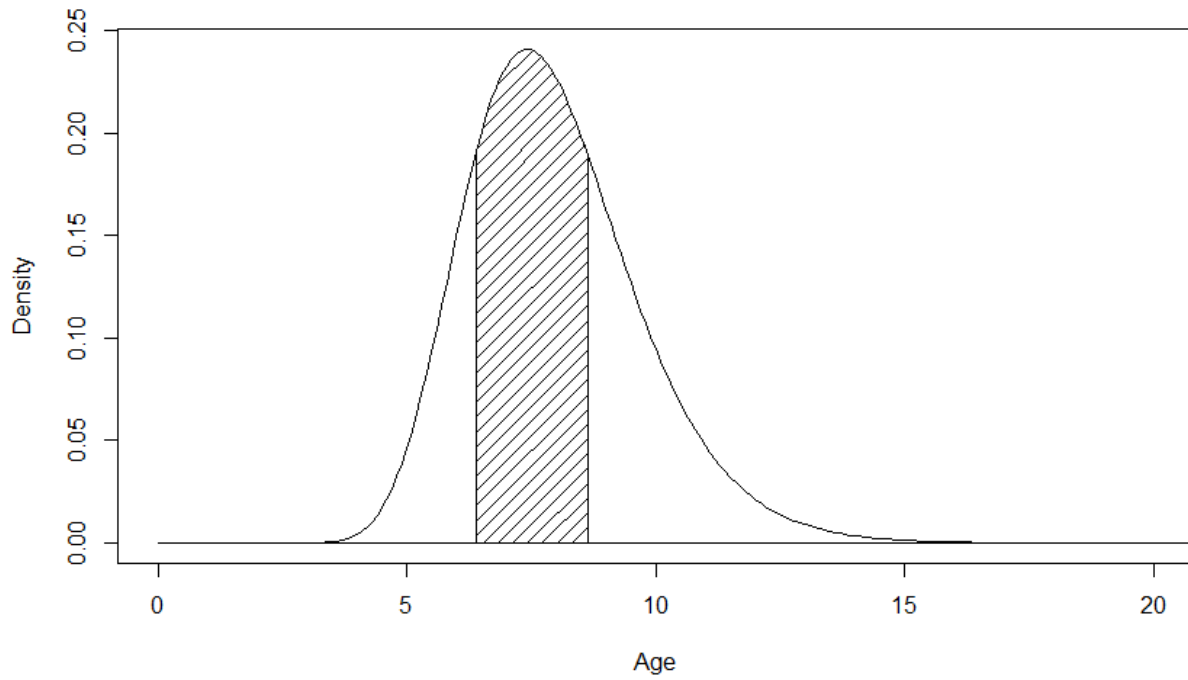
HPD male upper canine stage 7 (Ri), US Deaths 2008 prior



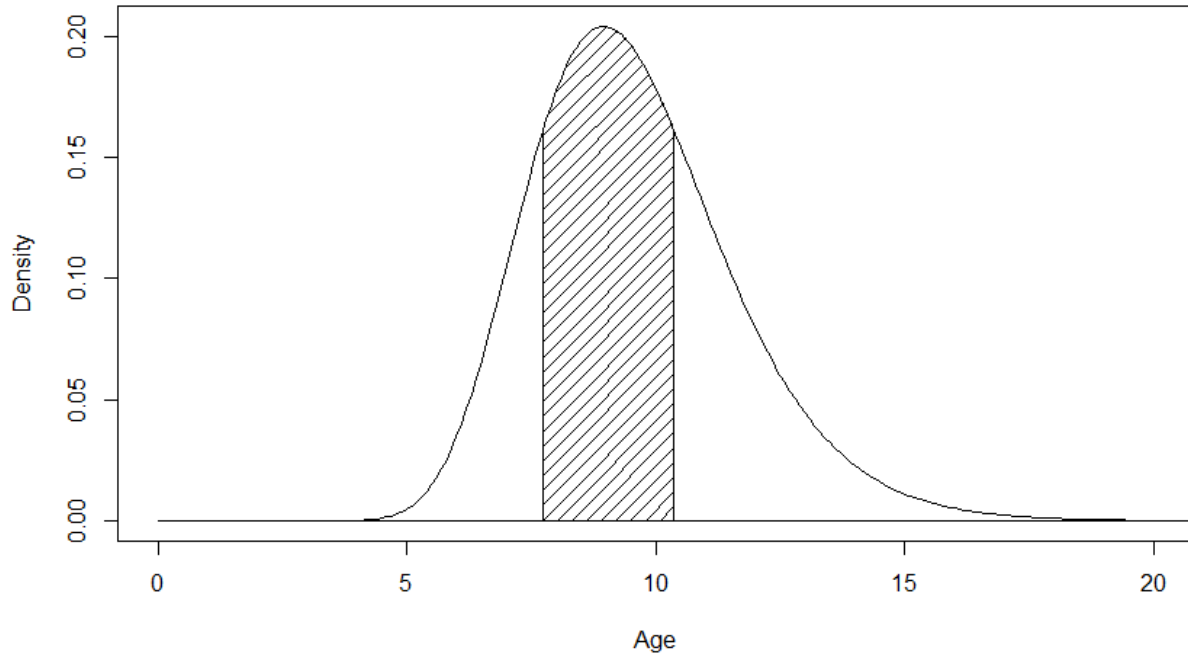
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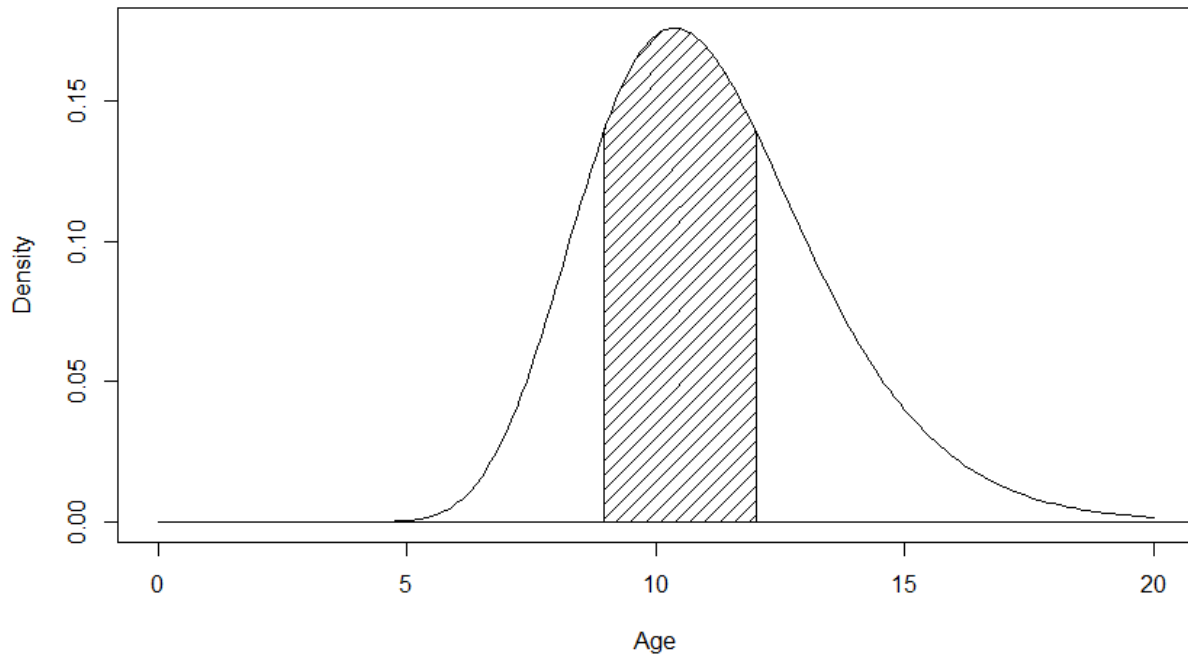
HPD male upper canine stage 10 (R 1/2), US Deaths 2008 prior



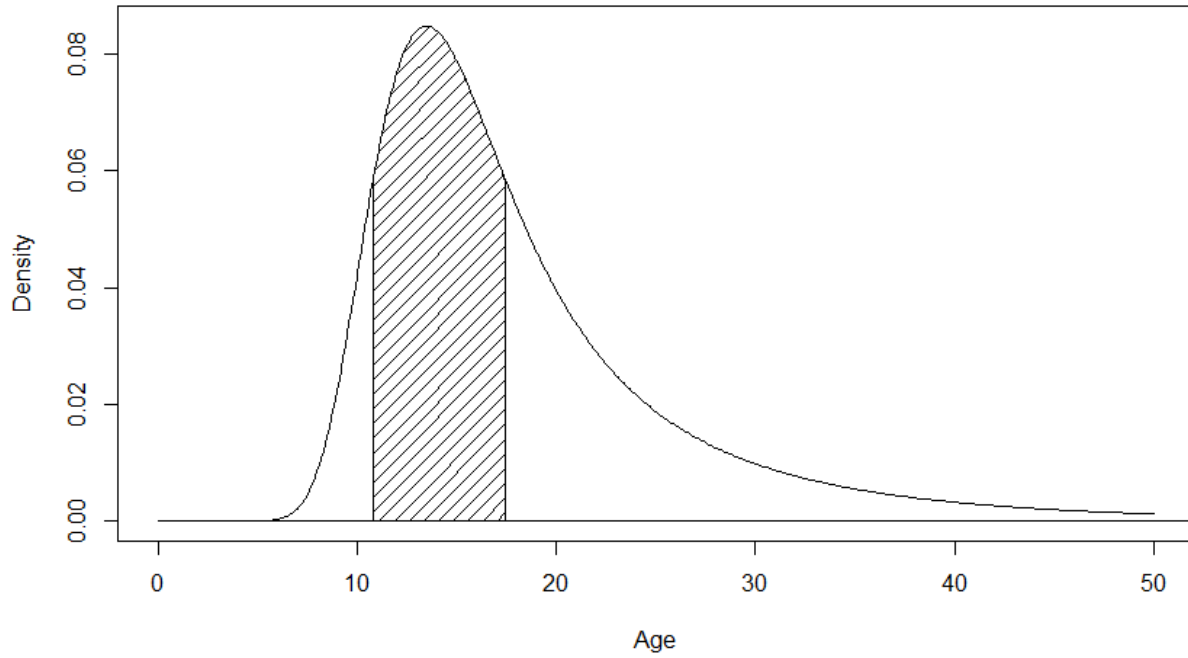
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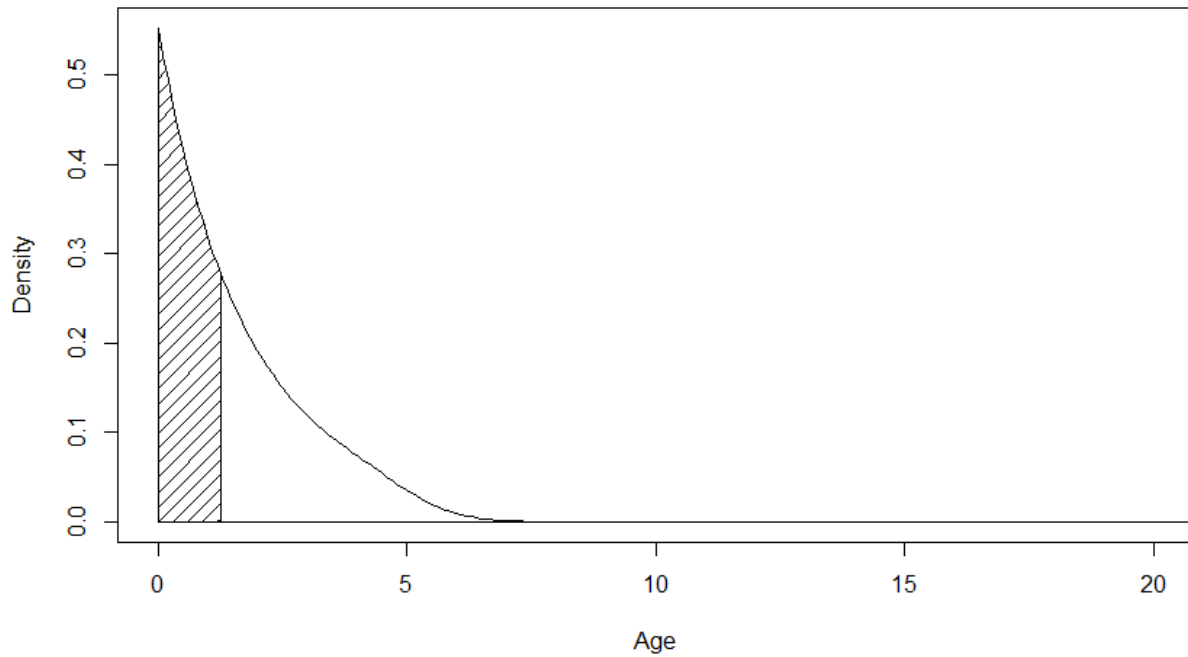
HPD male upper canine stage 12 (Rc), US Deaths 2008 prior



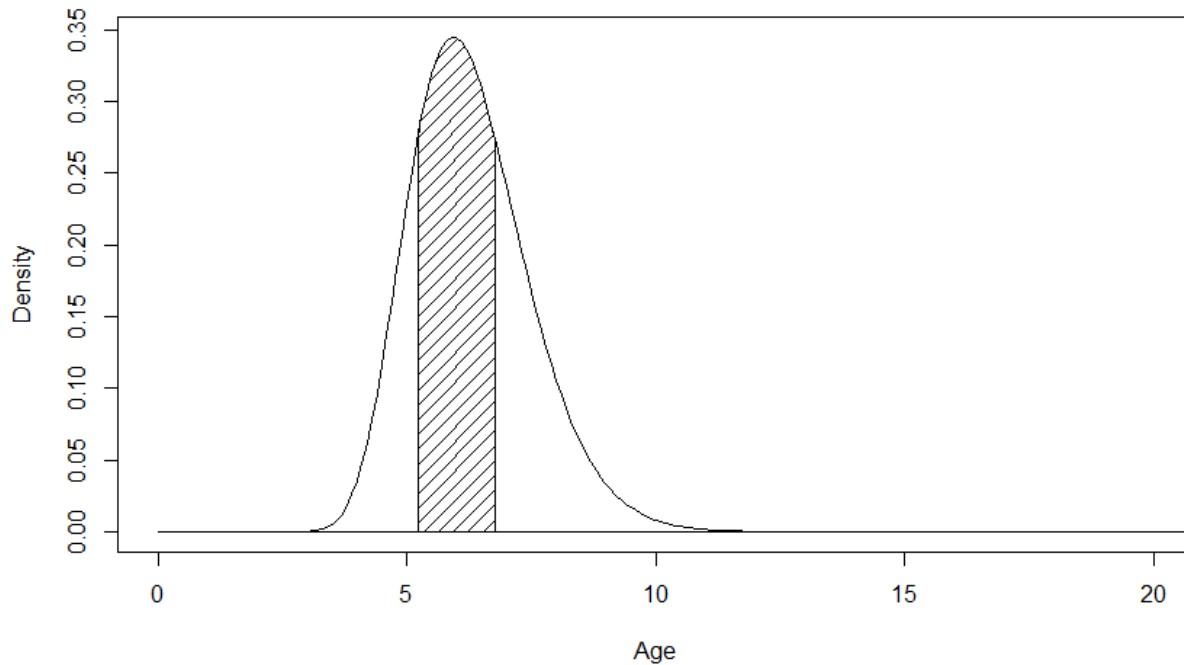
HPD male upper canine stage 13 (A 1/2), US Deaths 2008 prior



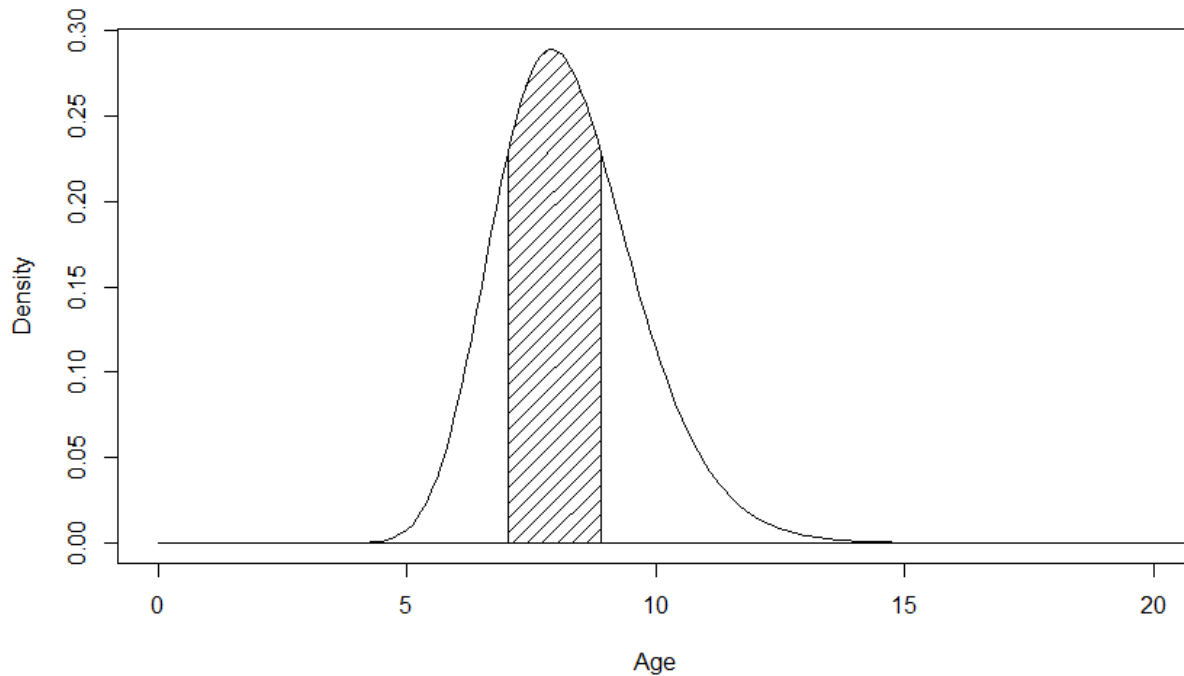
HPD male upper premolar 1 stage 6 (Crc), US Deaths 2008 prior



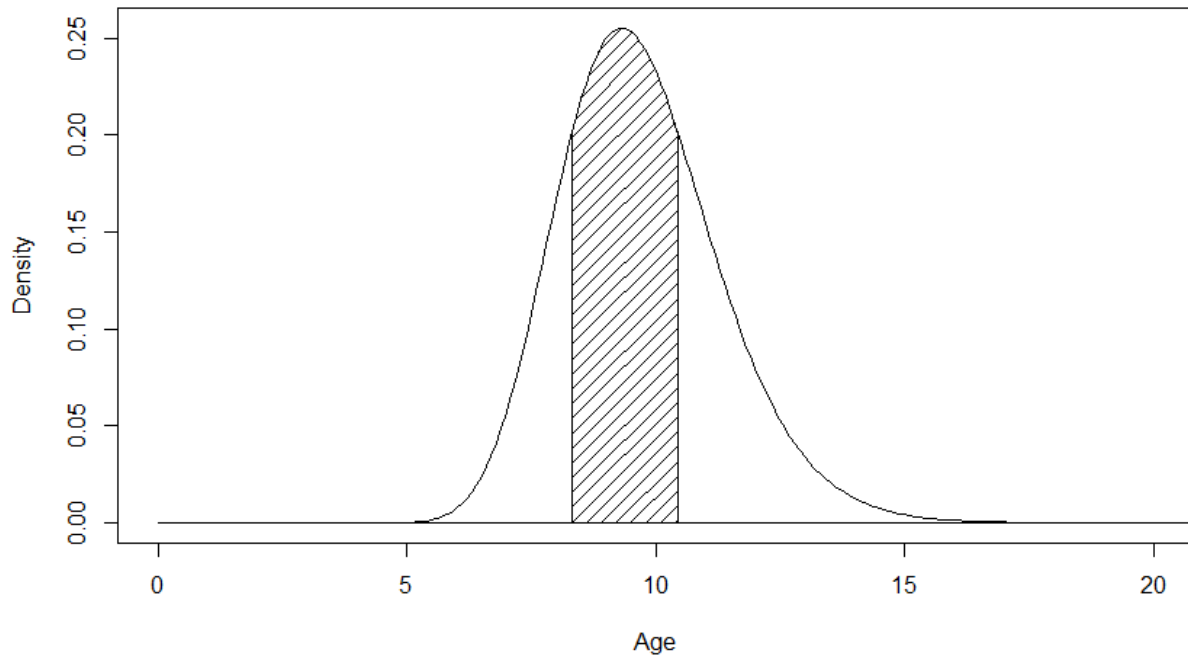
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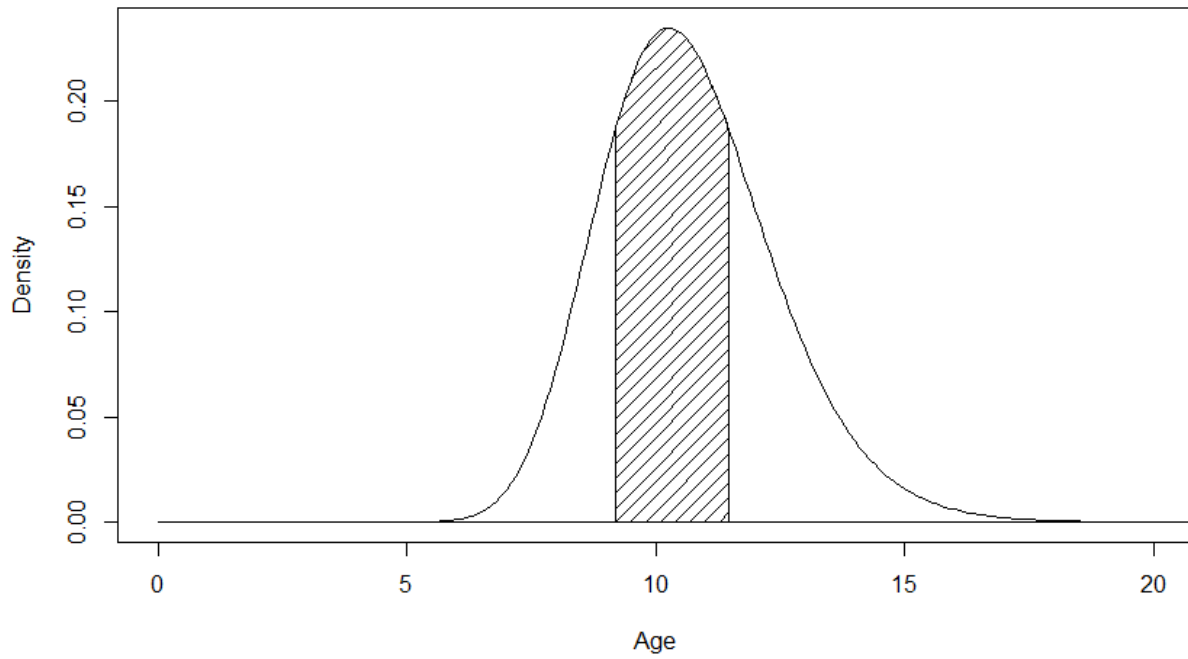
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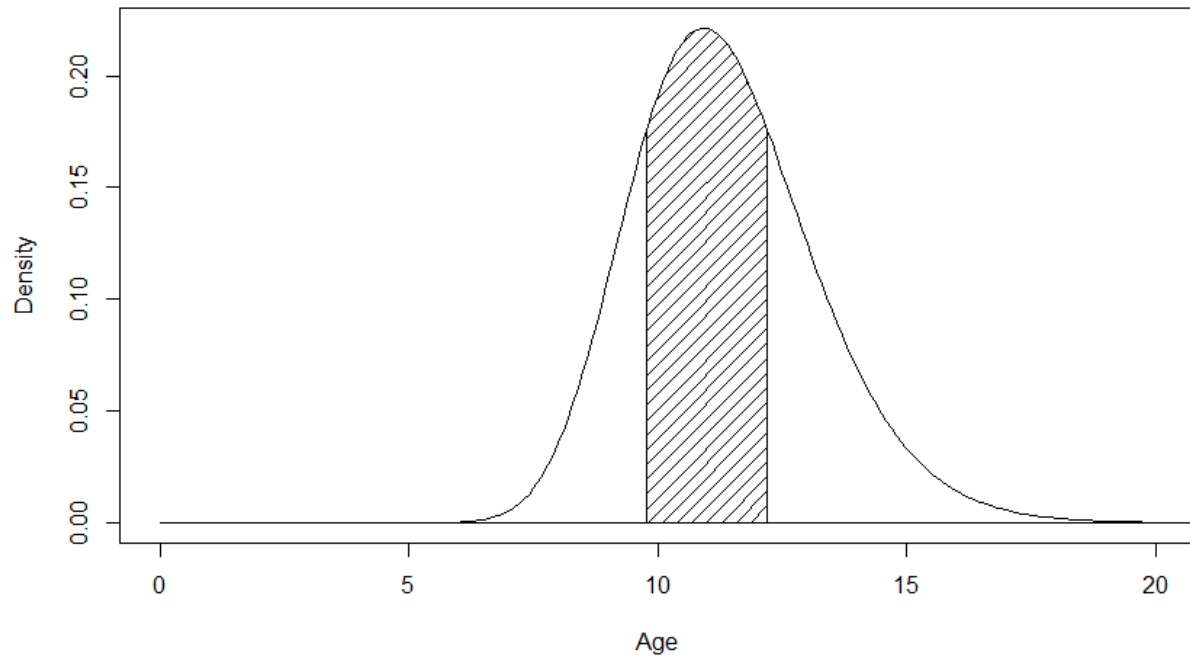
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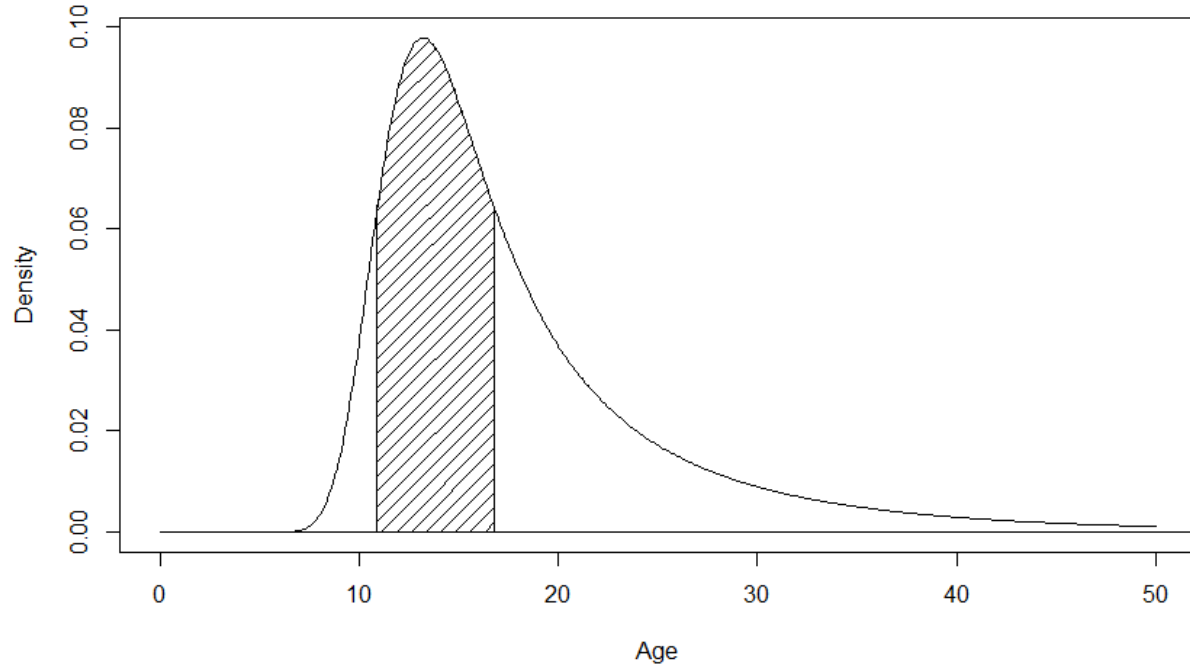
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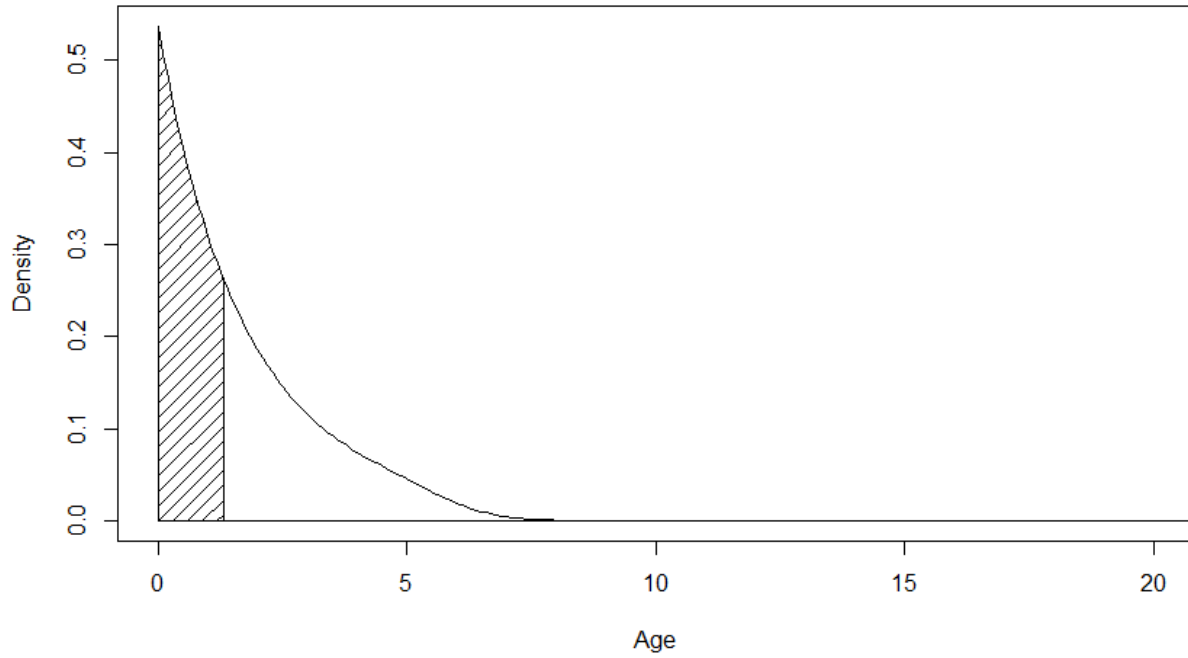
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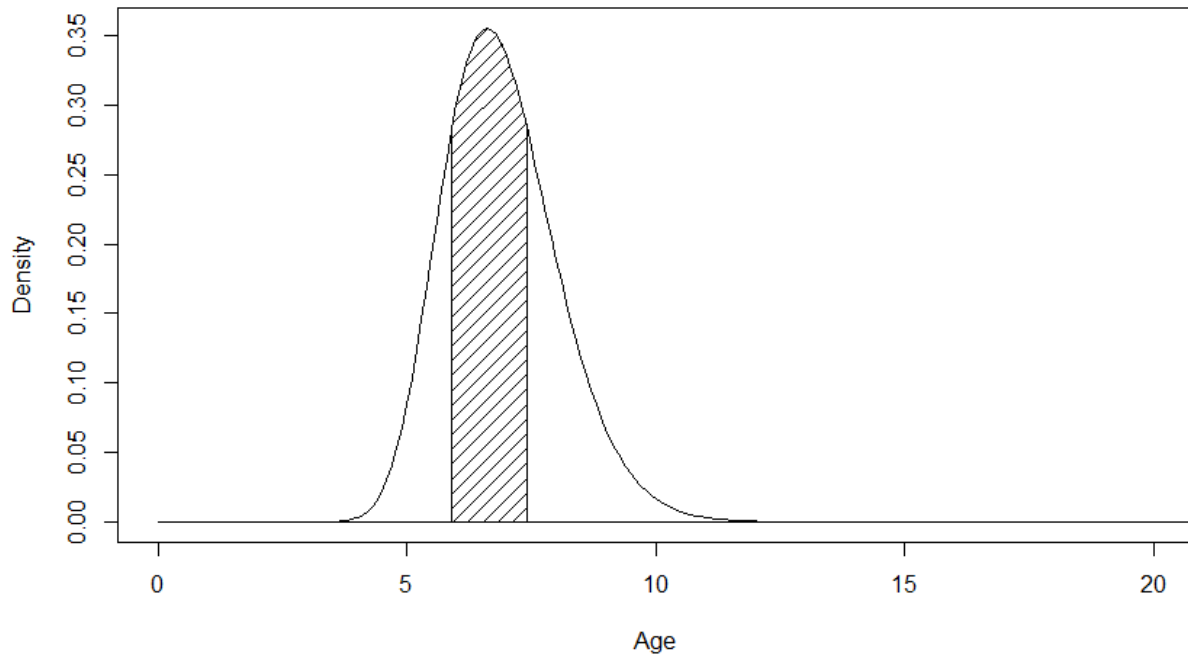
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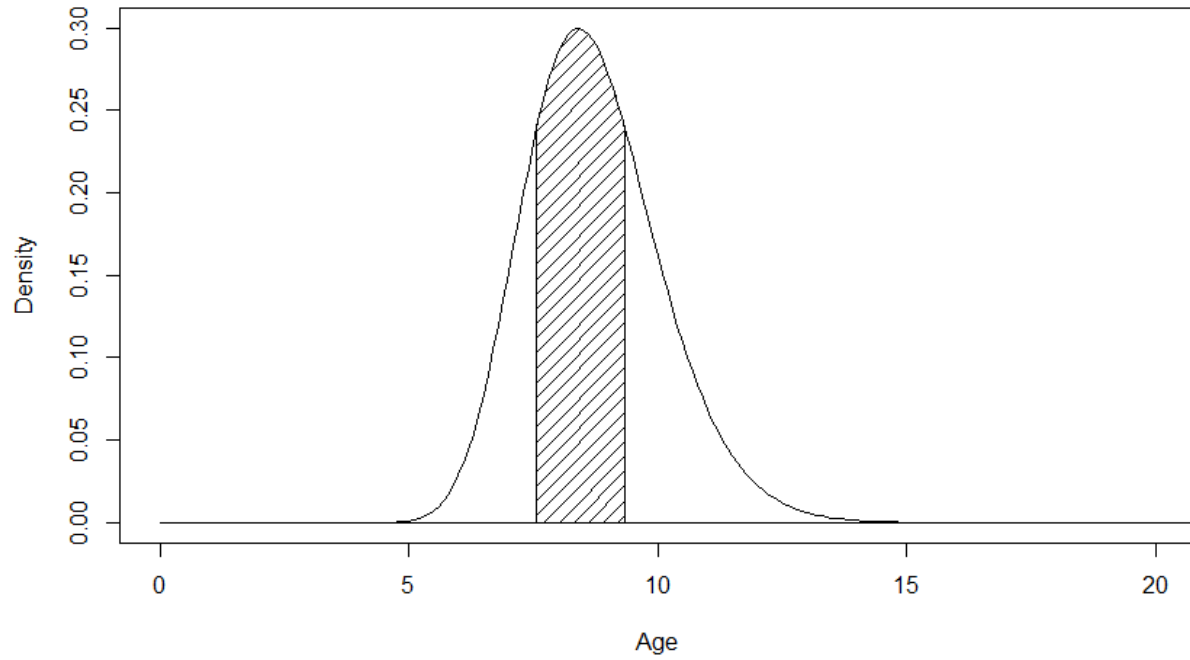
HPD male upper premolar 2 stage 6 (Crc), US Deaths 2008 prior



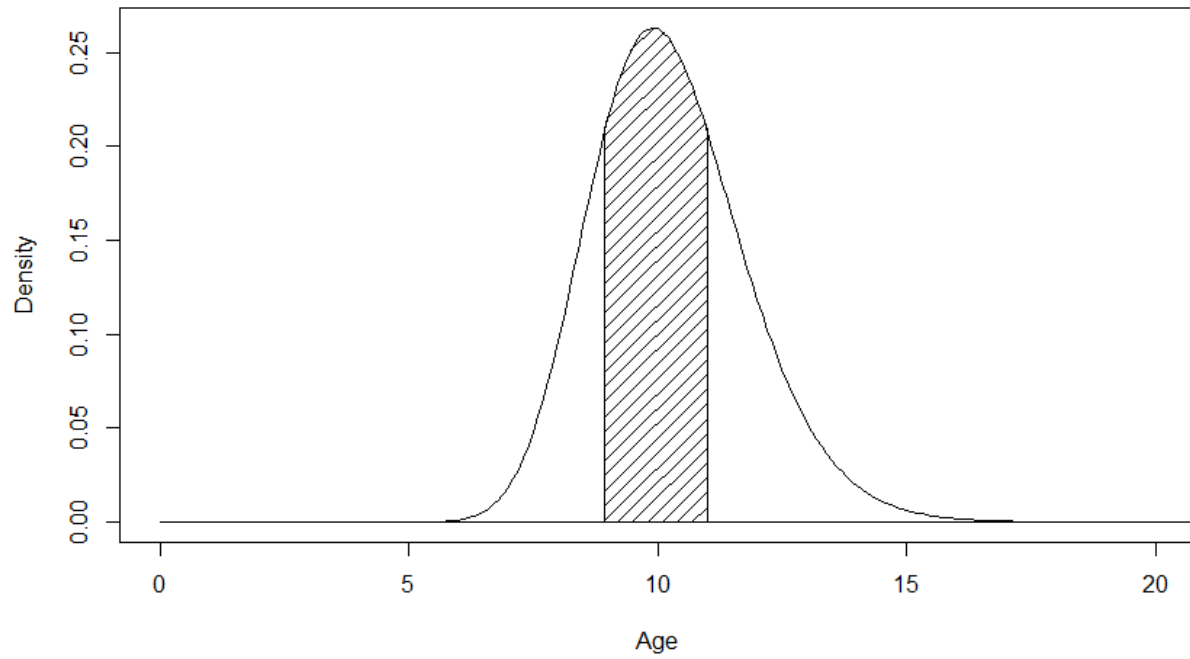
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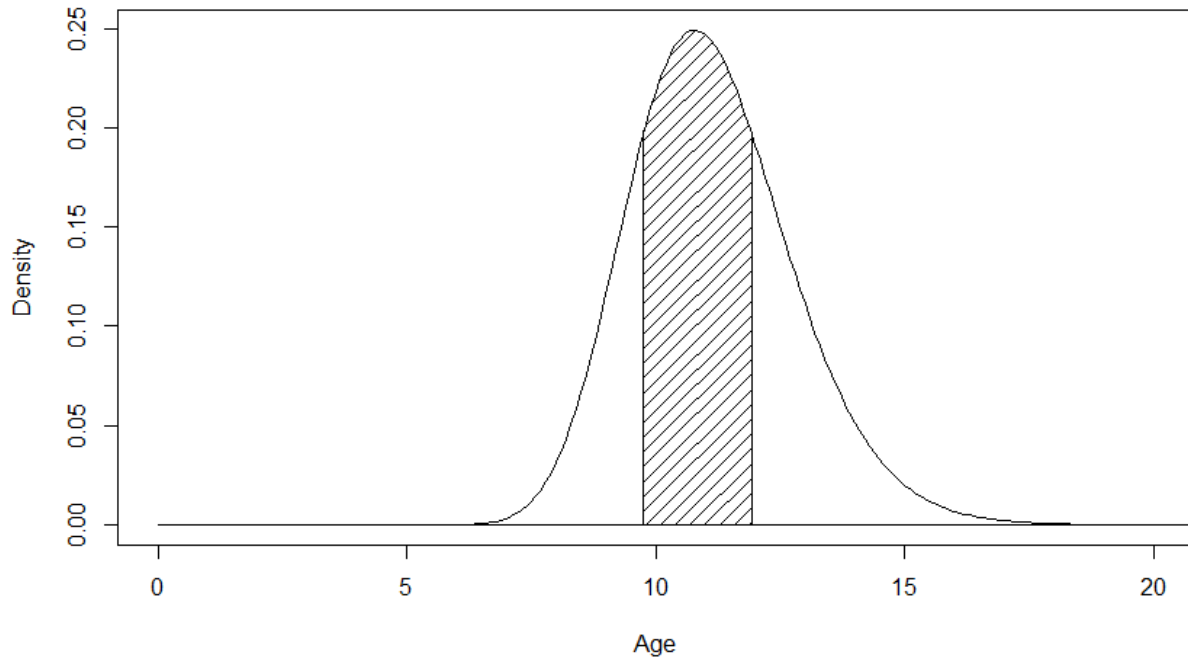
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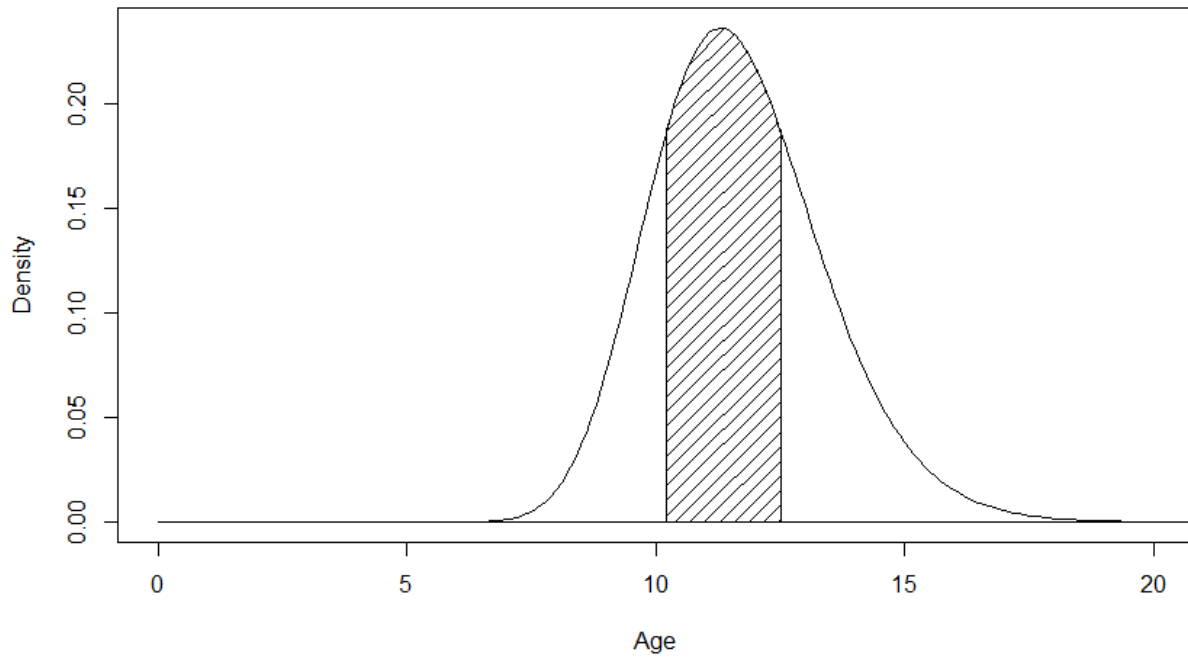
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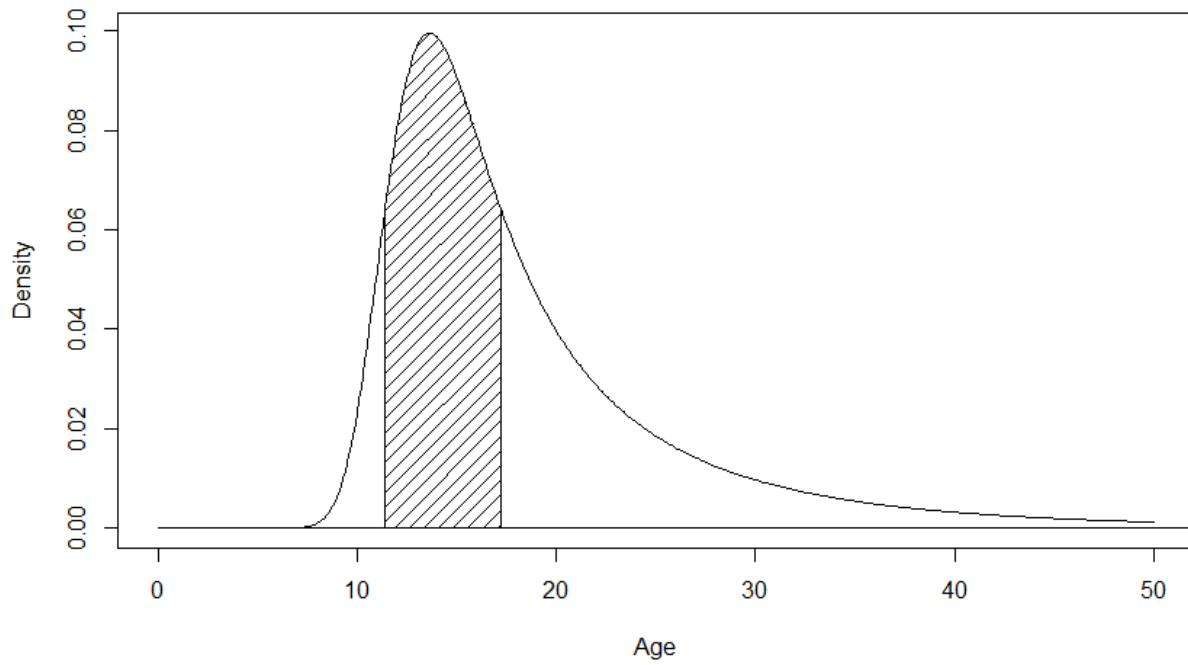
HPD male upper premolar 2 stage 11 (R 3/4), US Deaths 2008 prior



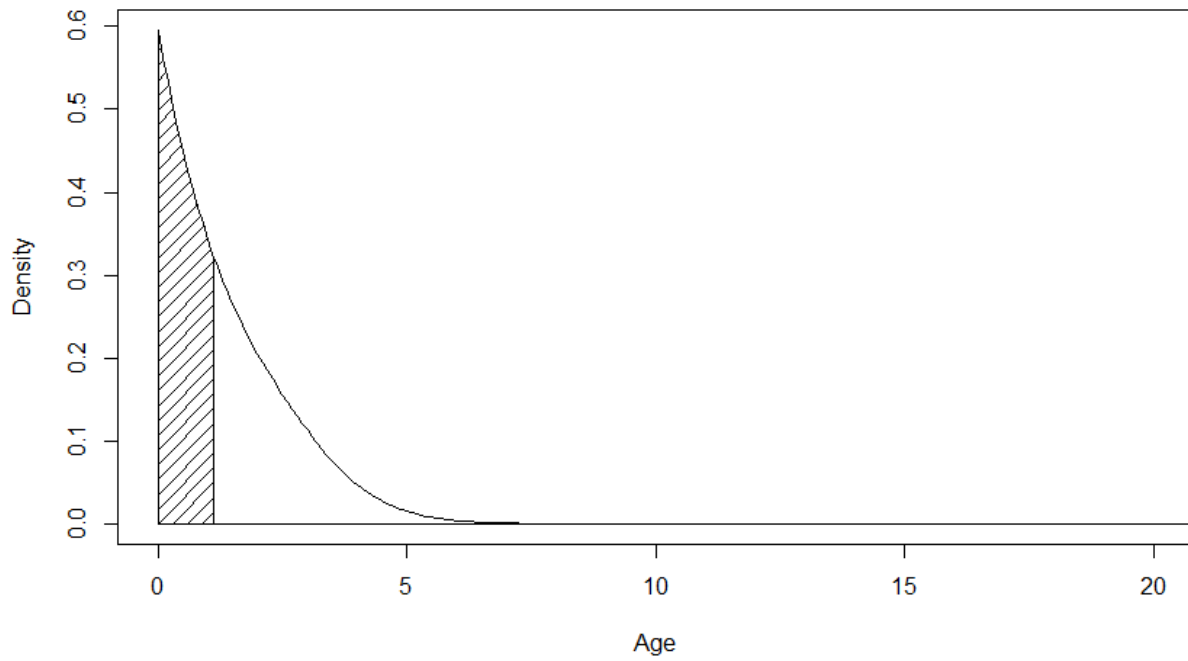
HPD male upper premolar 2 stage 12 (Rc), US Deaths 2008 prior



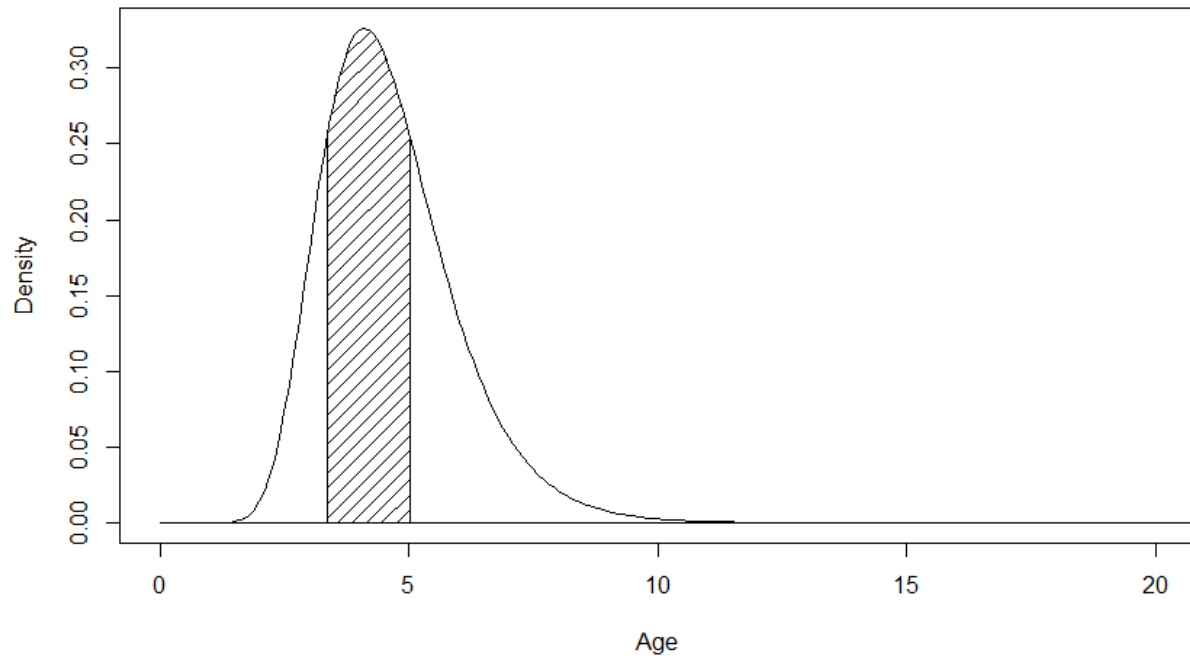
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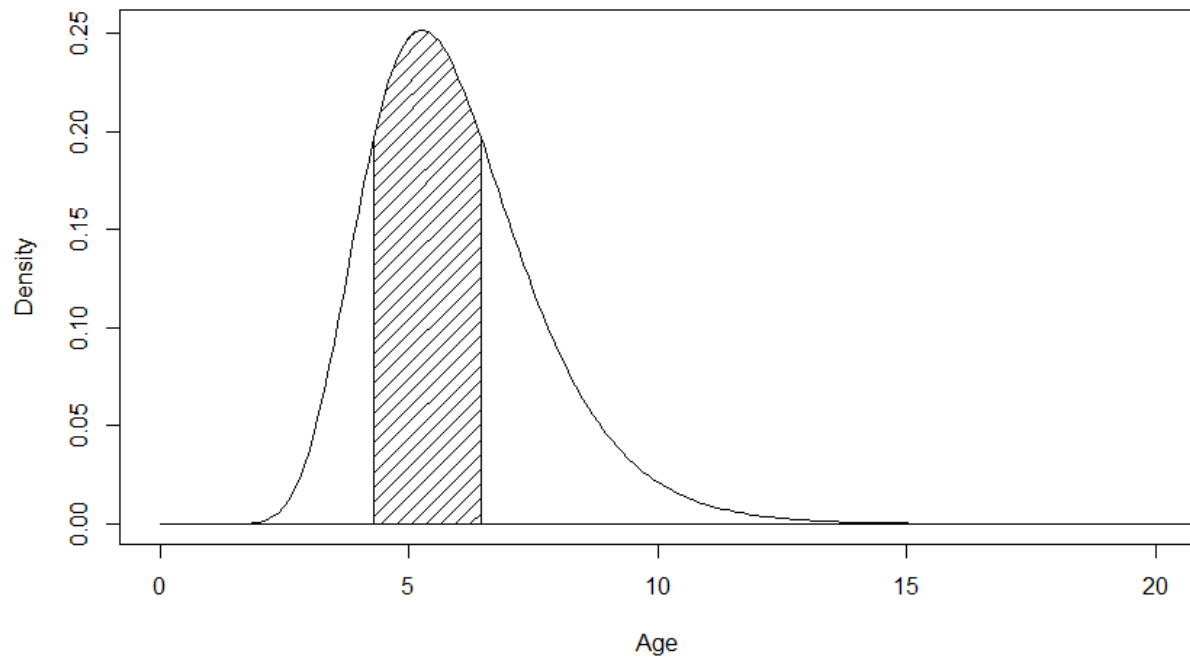
HPD male upper molar 1 stage 9 (R 1/4), US Deaths 2008 prior



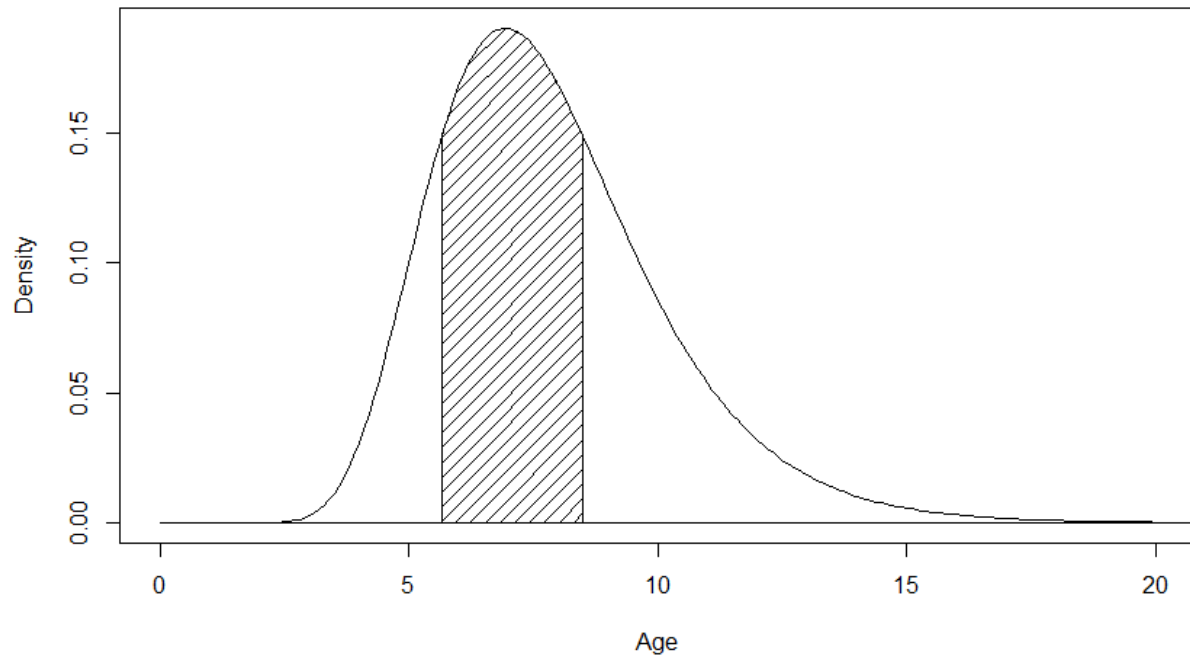
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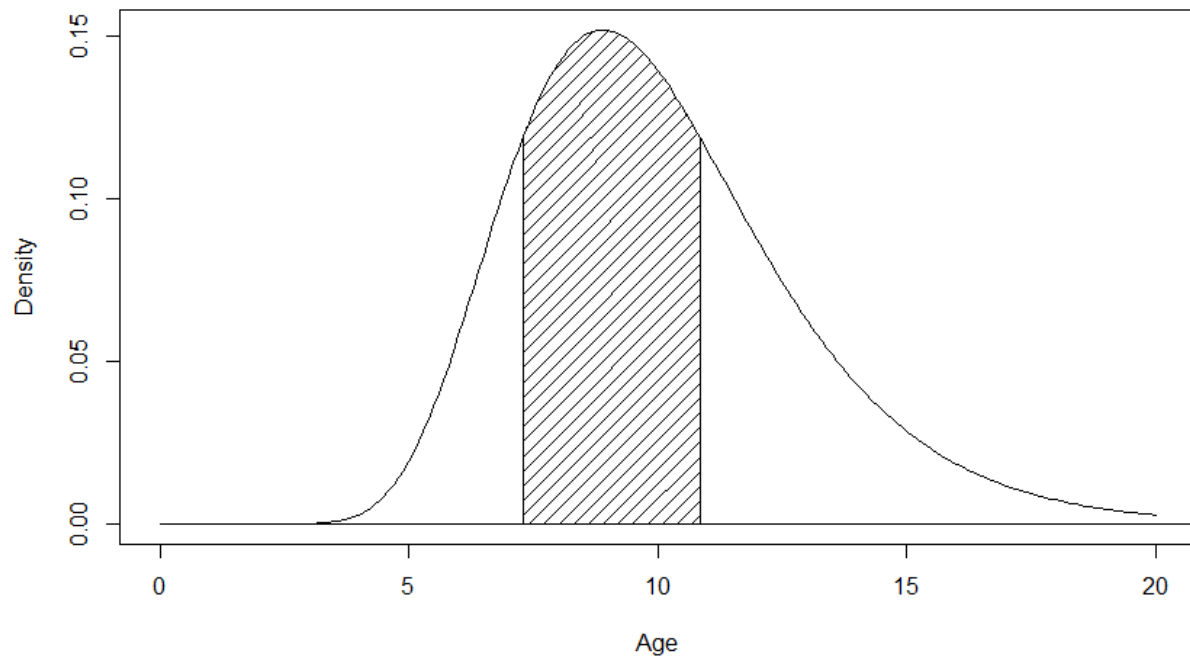
HPD male upper molar 1 stage 11 (R 3/4), US Deaths 2008 prior



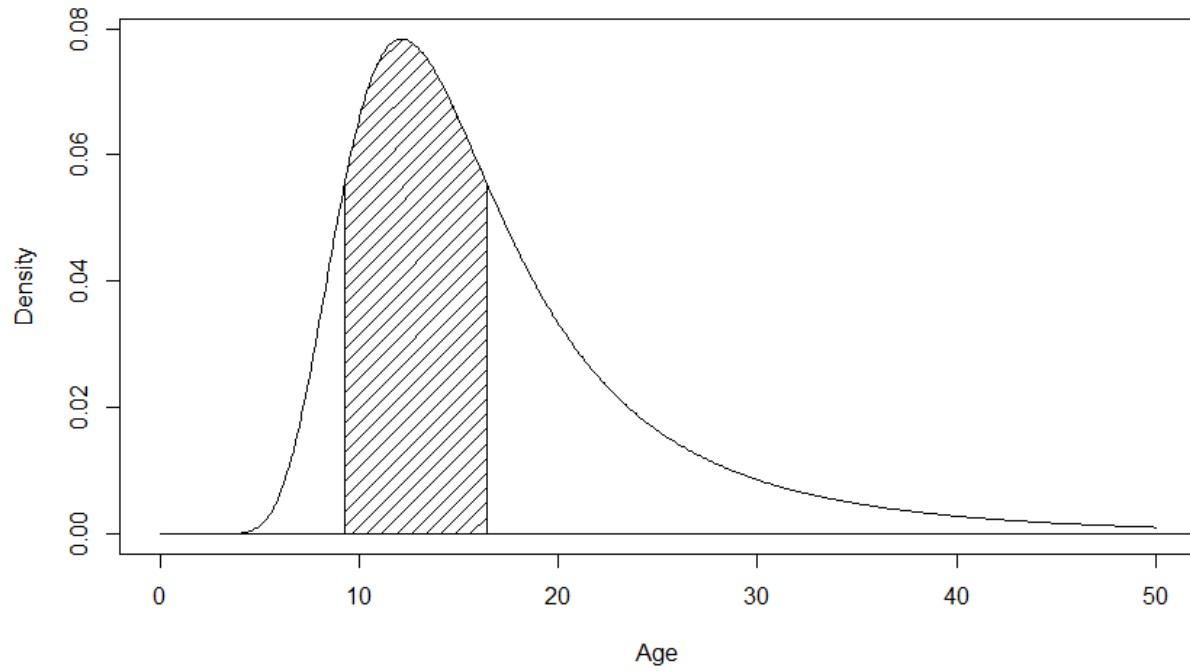
HPD male upper molar 1 stage 12 (Rc), US Deaths 2008 prior



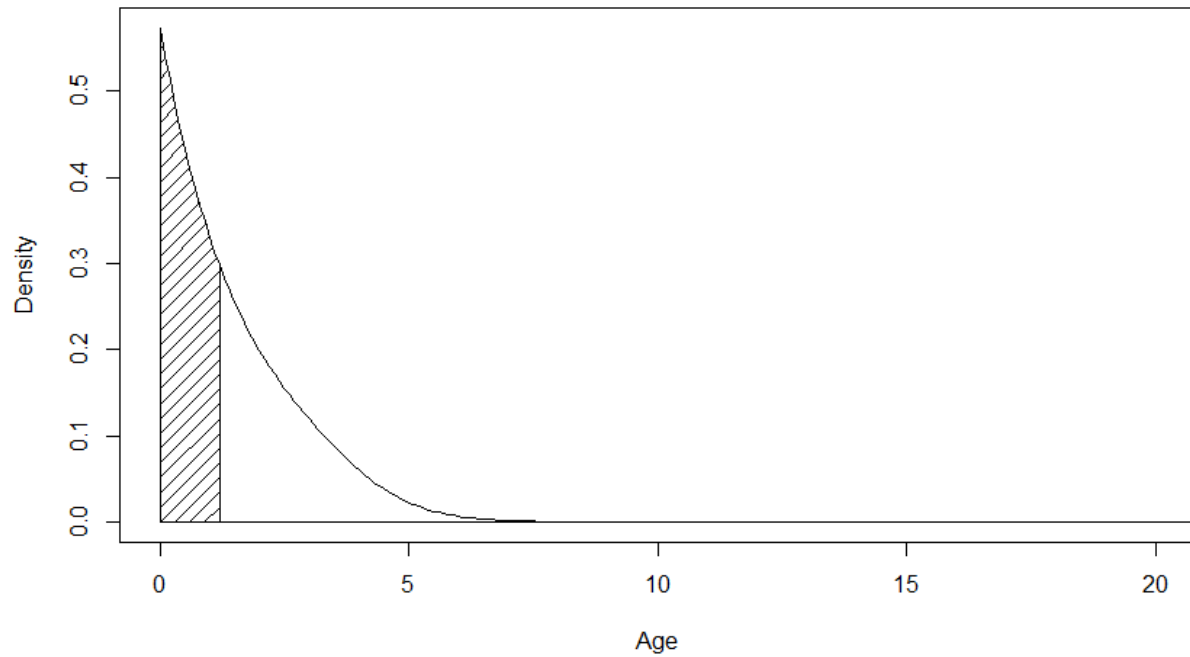
HPD male upper molar 1 stage 13 (A 1/2), US Deaths 2008 prior



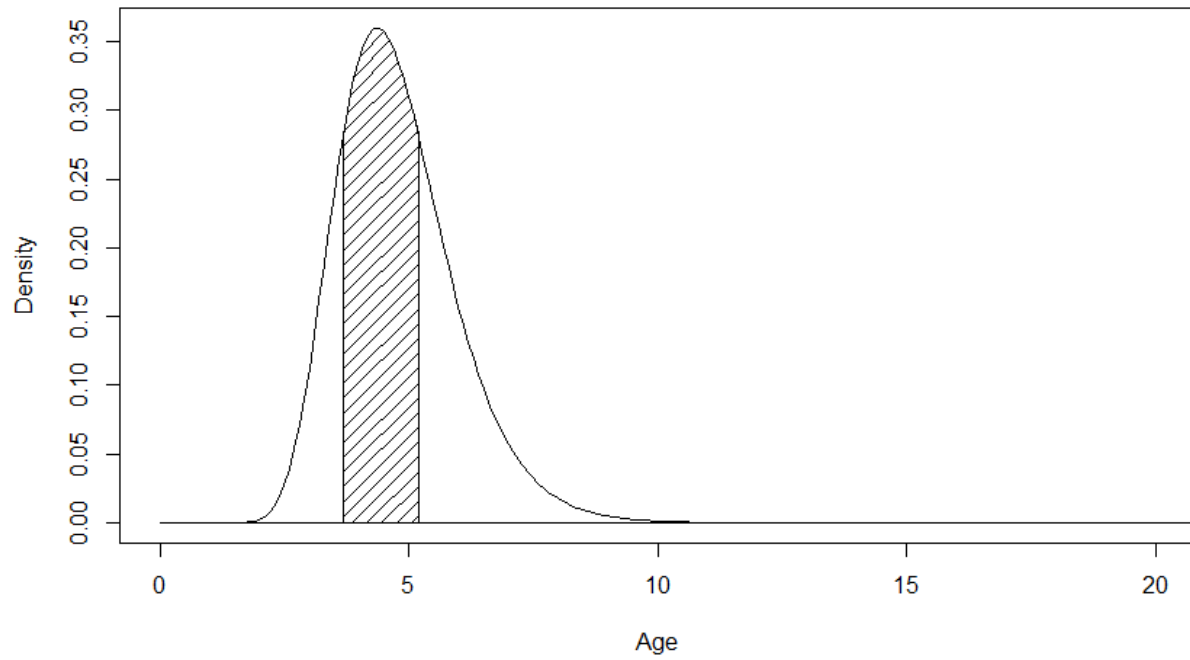
HPD male upper molar 1 stage 14 (Ac), US Deaths 2008 prior



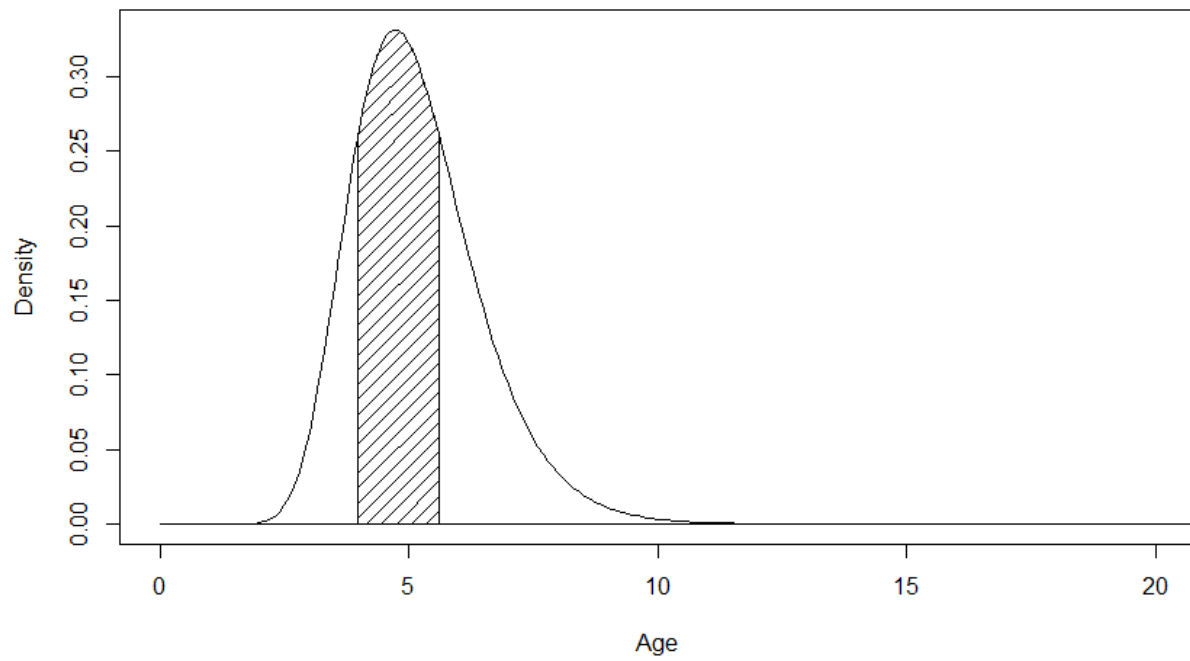
HPD male upper molar 2 stage 4 (Cr 1/2), US Deaths 2008 prior



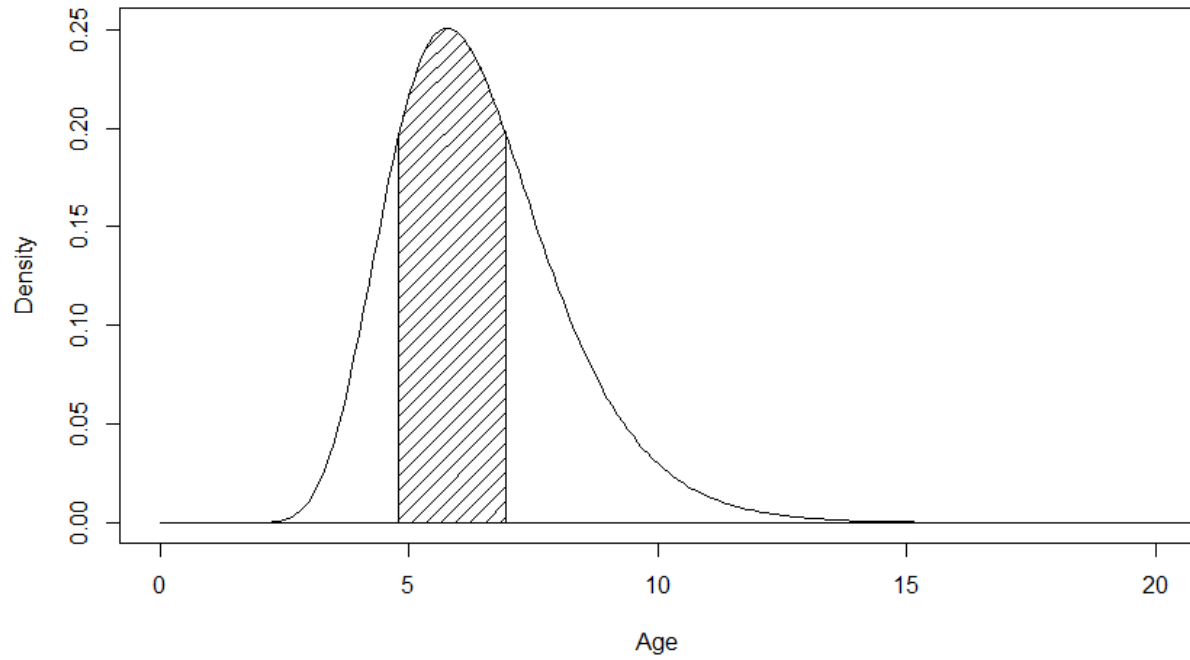
HPD male upper molar 2 stage 5 (Cr 3/4), US Deaths 2008 prior



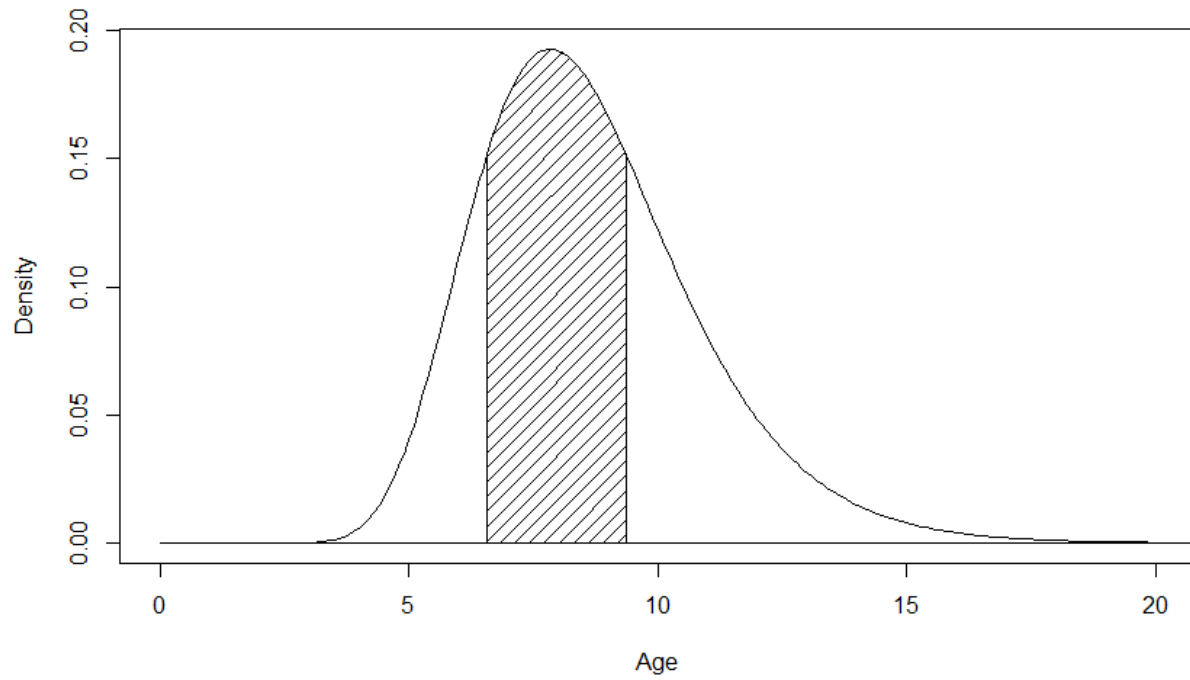
HPD male upper molar 2 stage 6 (Crc), US Deaths 2008 prior



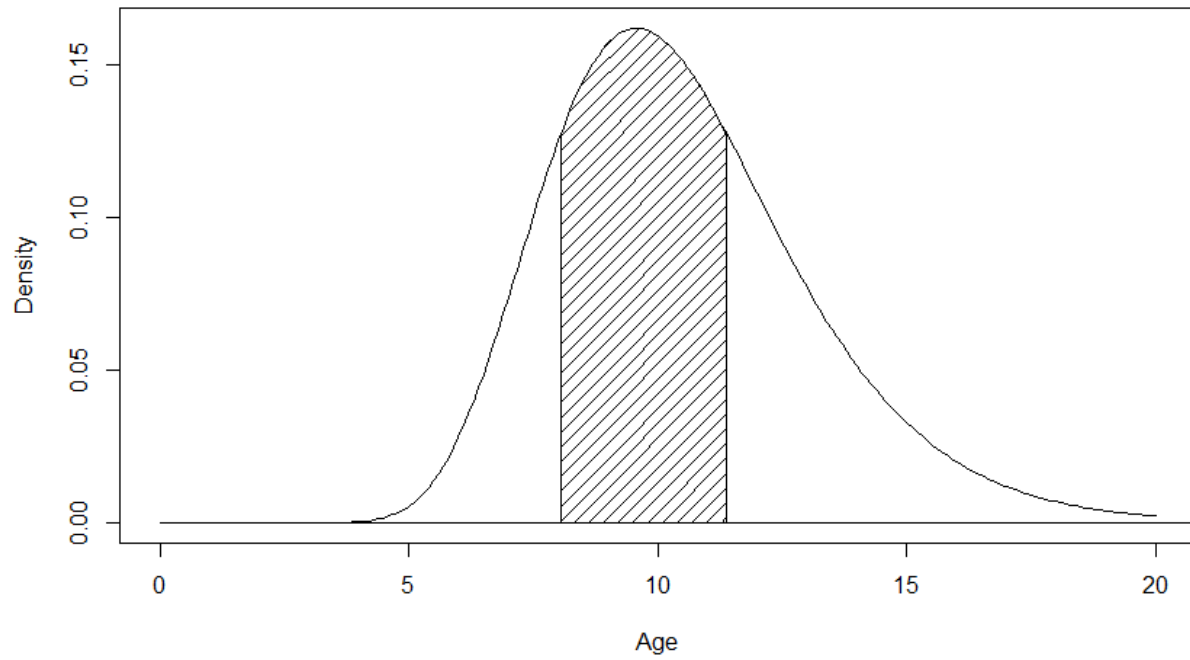
HPD male upper molar 2 stage 7 (Ri), US Deaths 2008 prior



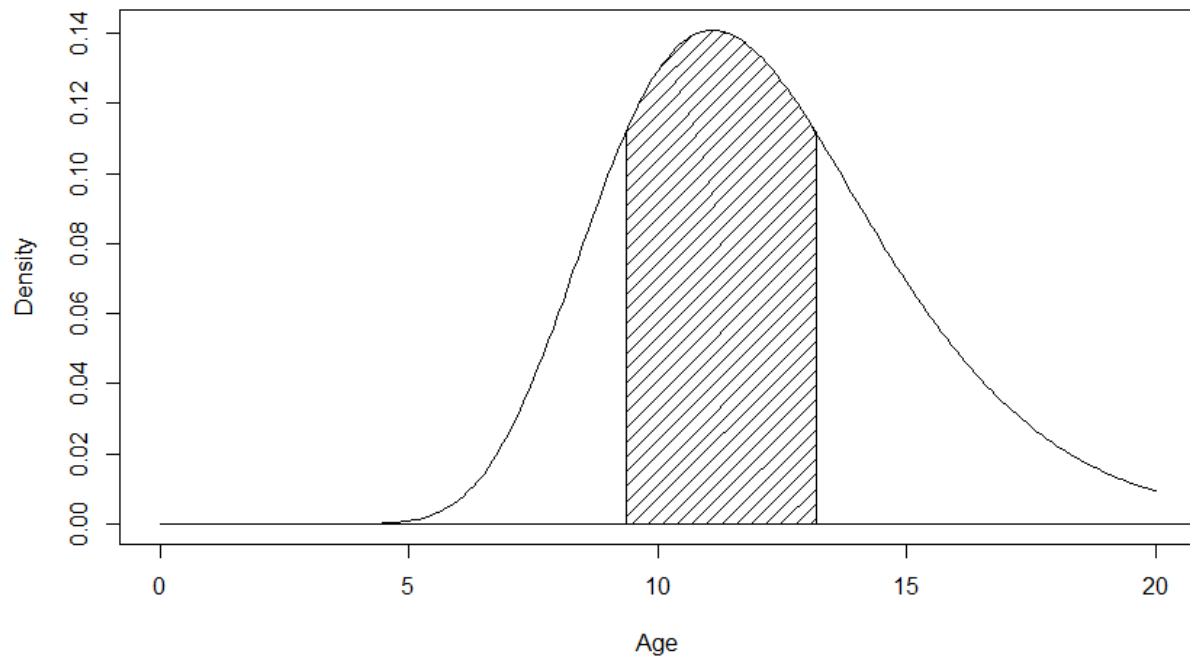
HPD male upper molar 2 stage 8 (C1i), US Deaths 2008 prior



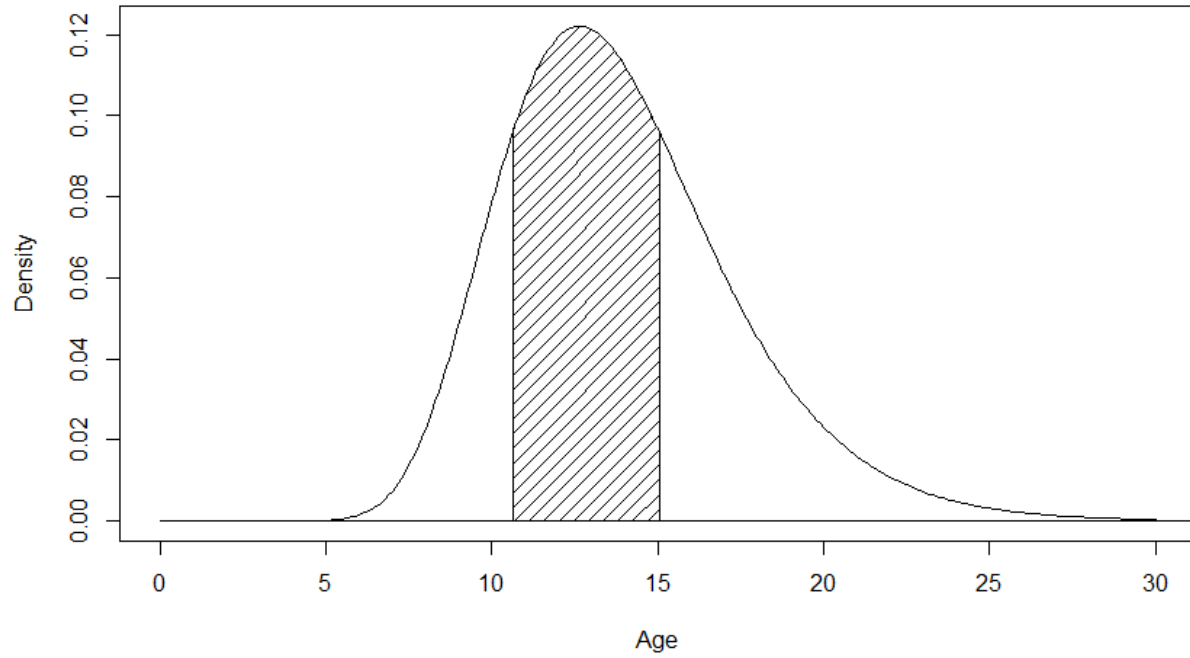
HPD male upper molar 2 stage 9 (R 1/4), US Deaths 2008 prior



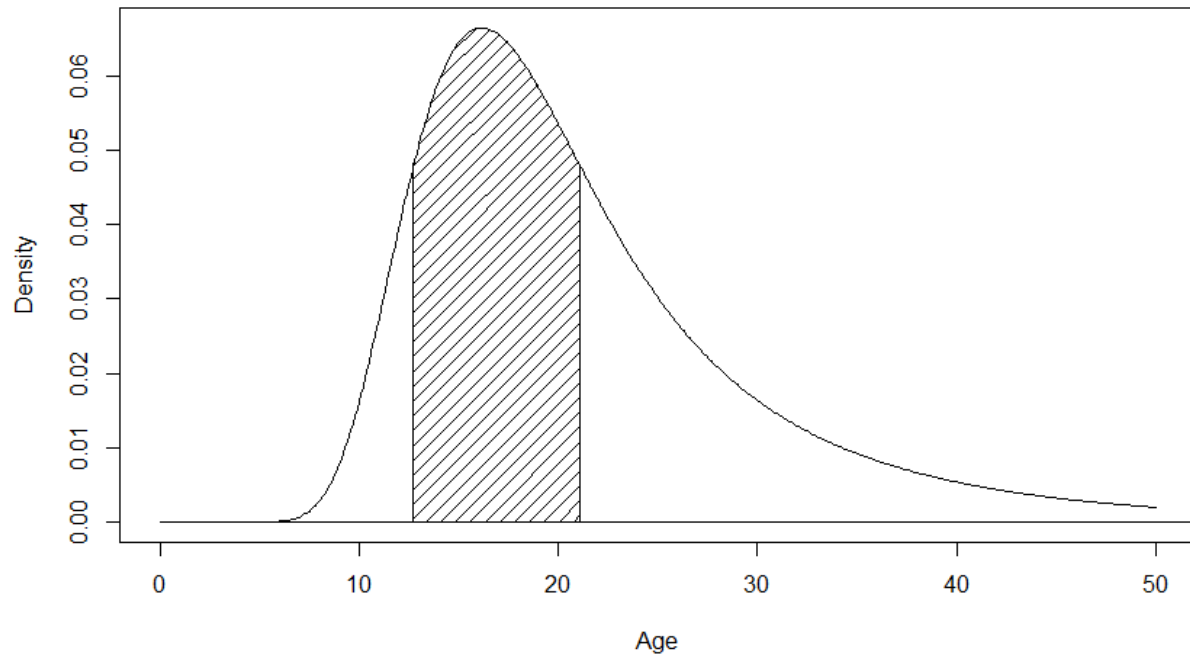
HPD male upper molar 2 stage 10 (R 1/2), US Deaths 2008 prior



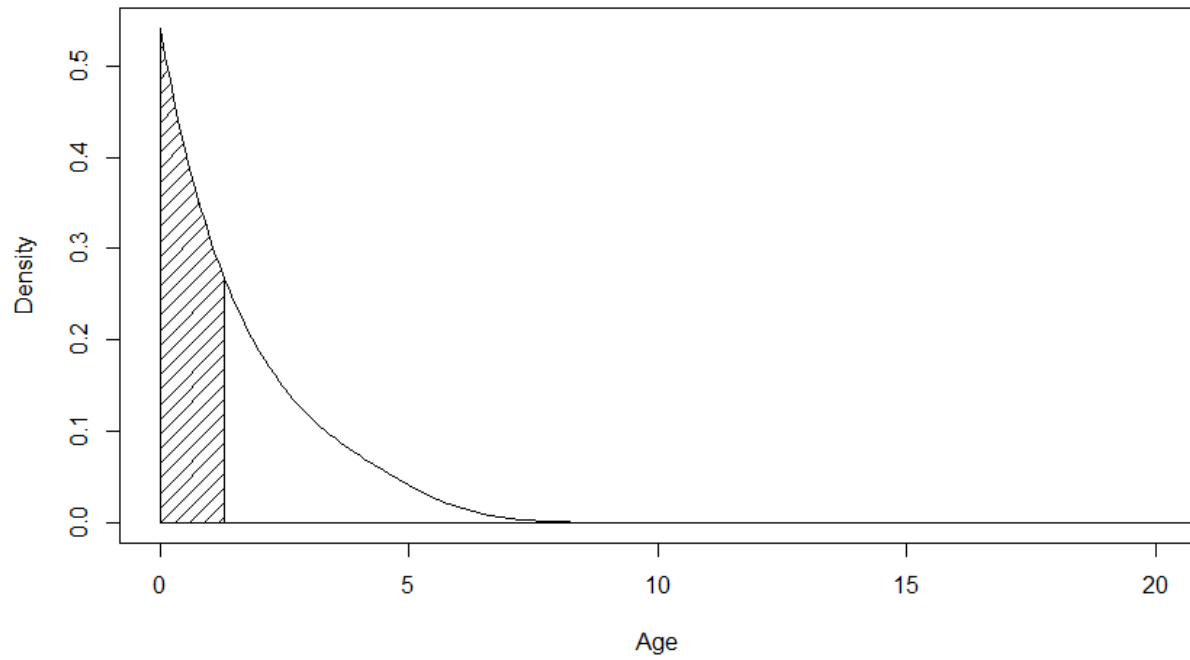
HPD male upper molar 2 stage 11 (R 3/4), US Deaths 2008 prior



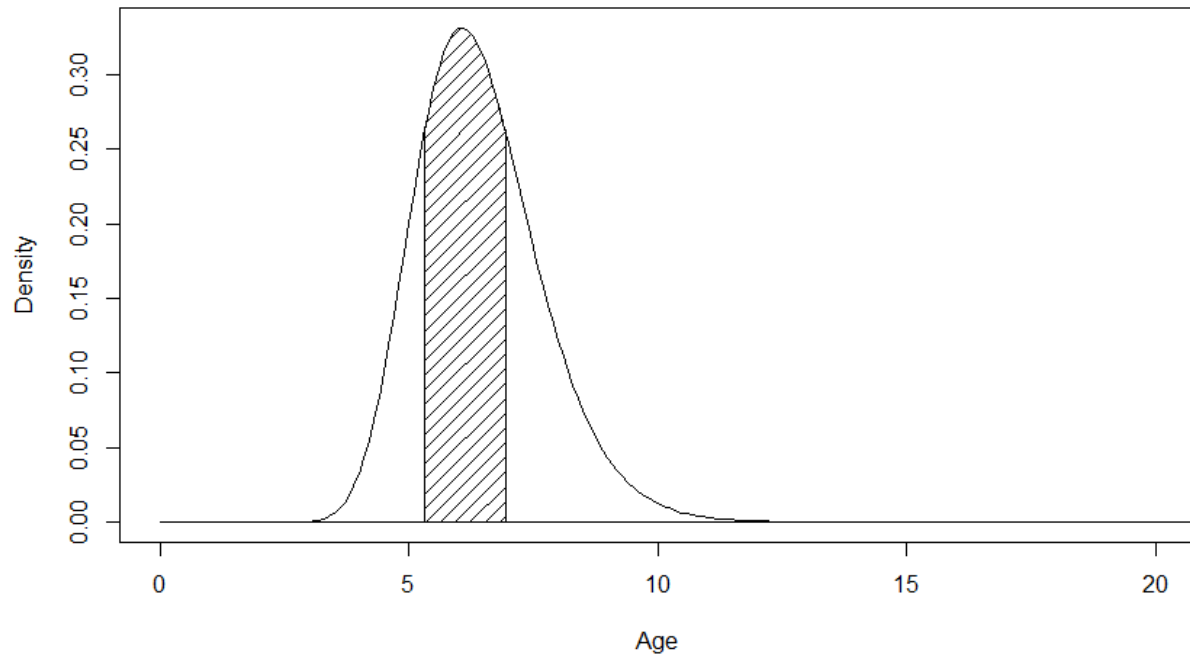
HPD male upper molar 2 stage 12 (Rc), US Deaths 2008 prior



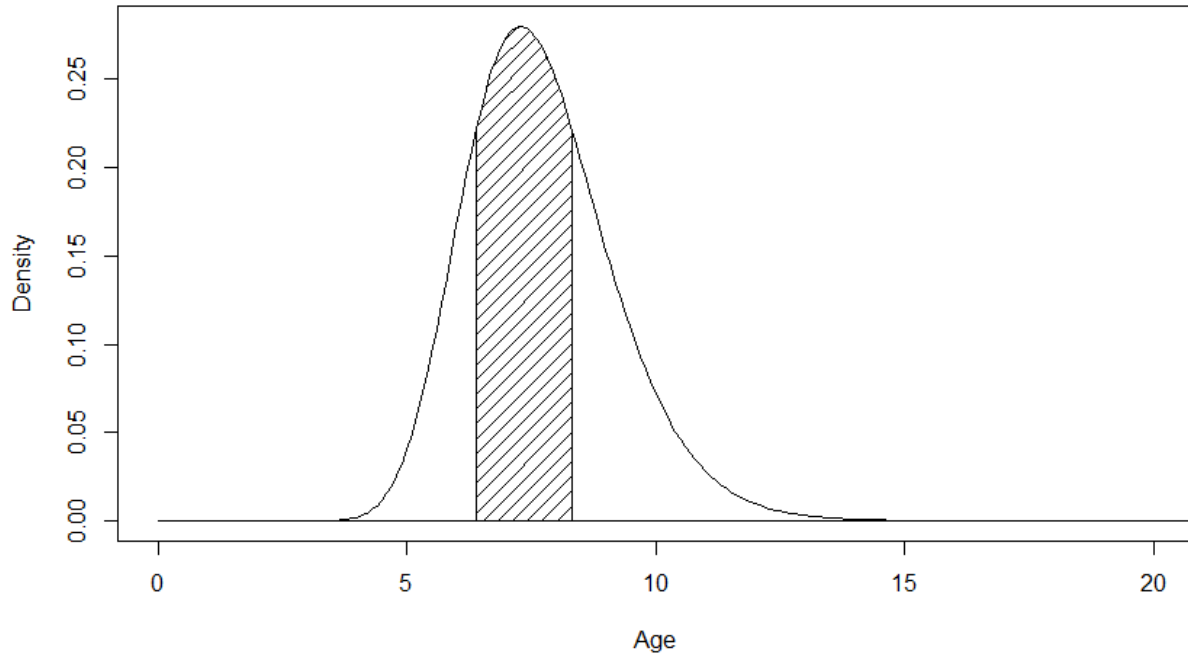
HPD male lower incisor 1 stage 11 (R 3/4), US Deaths 2008 prior



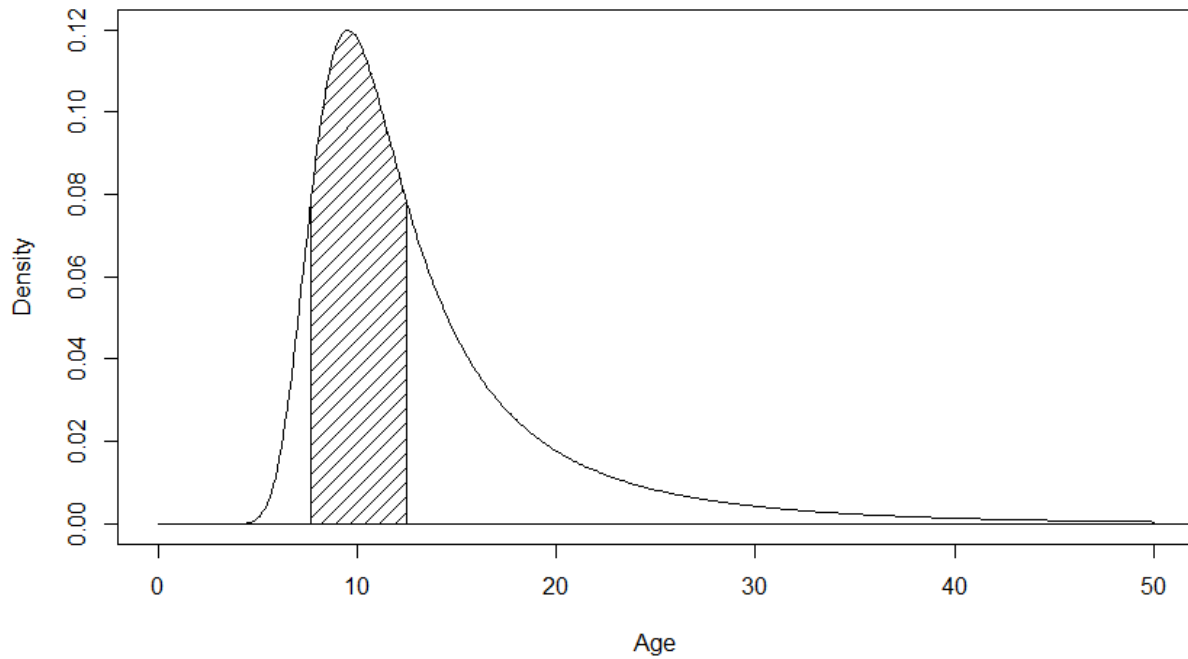
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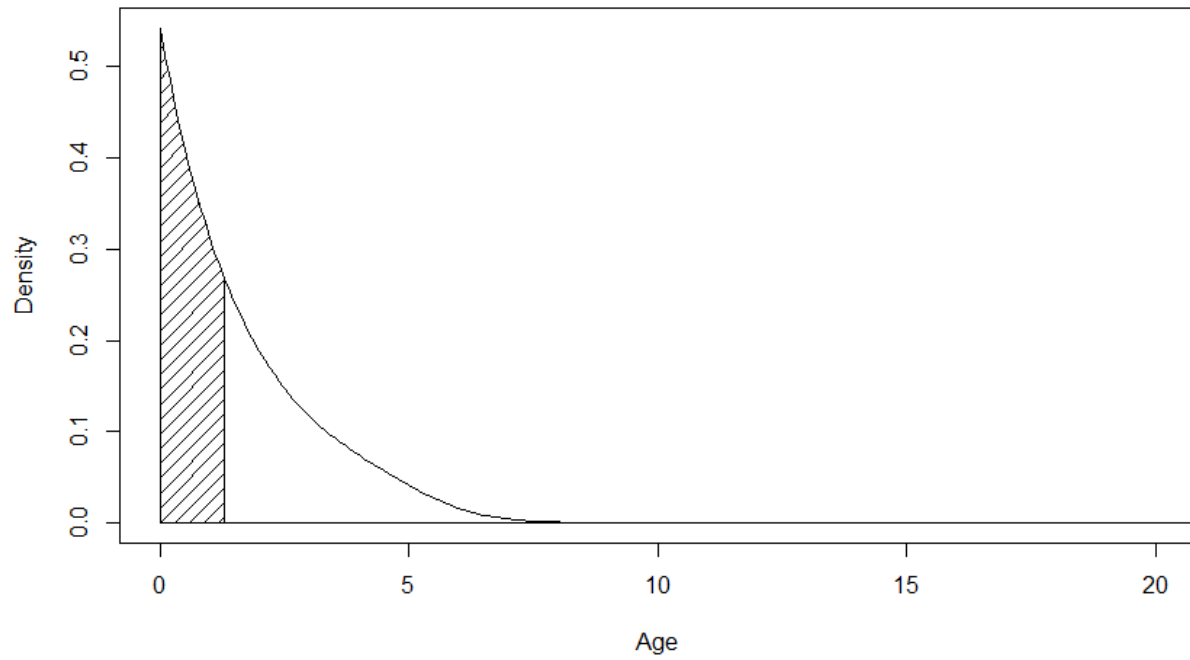
HPD male lower incisor 1 stage 13 (A 1/2), US Deaths 2008 prior



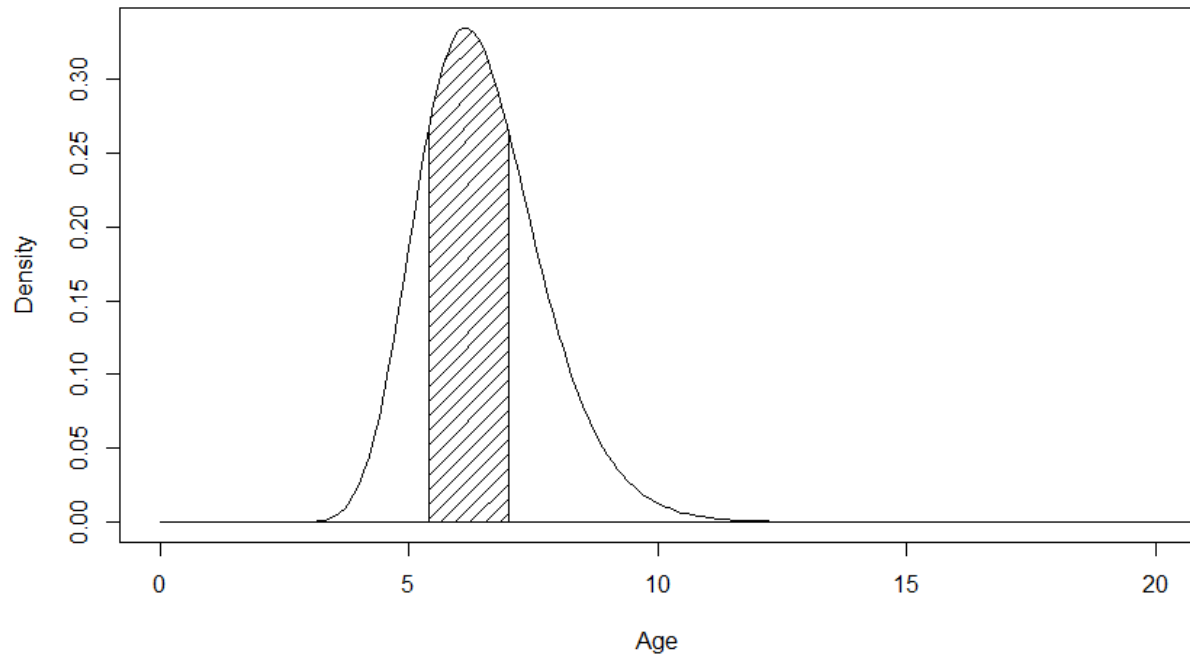
HPD male lower incisor 1 stage 14 (Ac), US Deaths 2008 prior



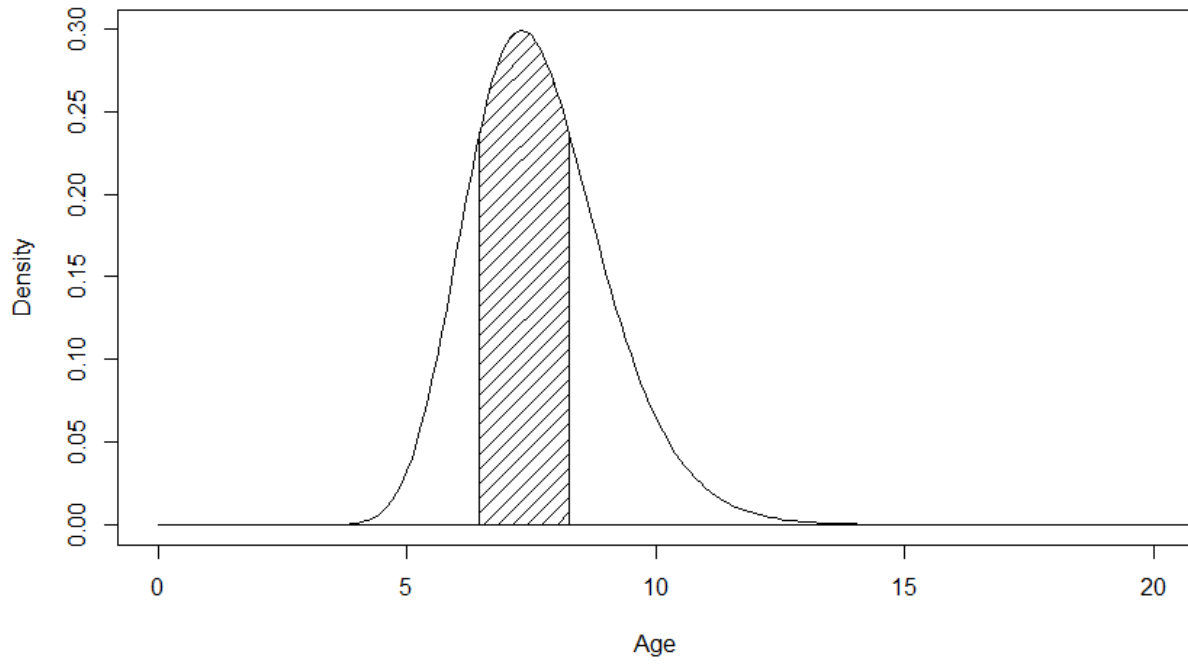
HPD male lower incisor 2 stage 10 (R 1/2), US Deaths 2008 prior



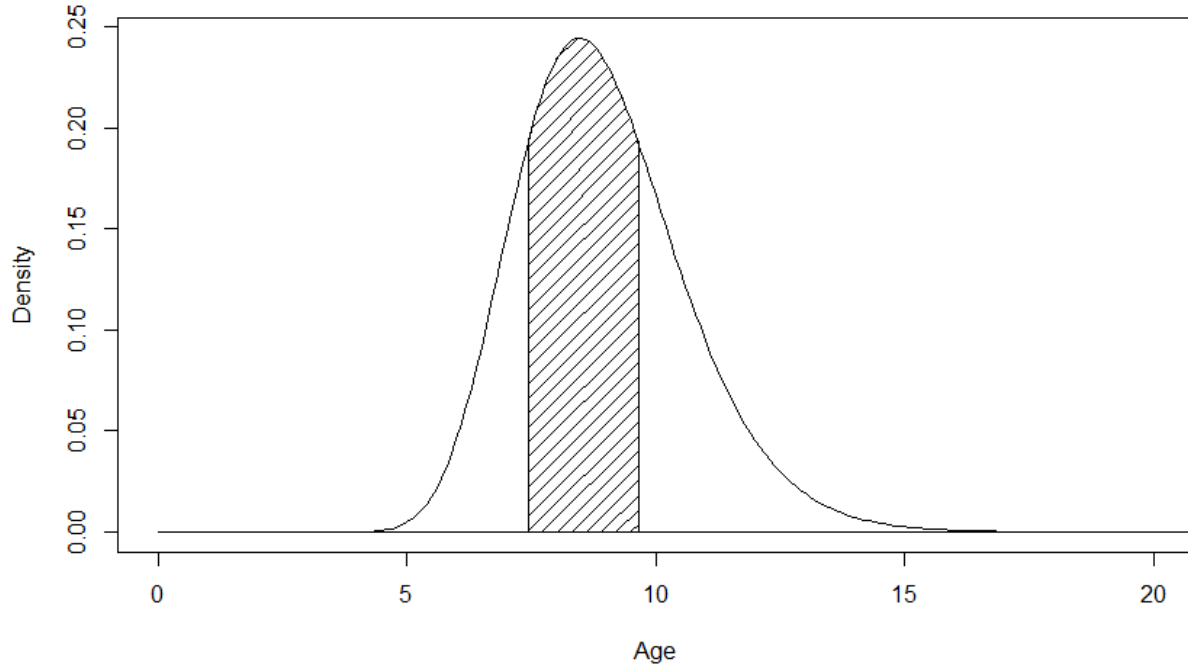
HPD male lower incisor 2 stage 11 (R 3/4), US Deaths 2008 prior



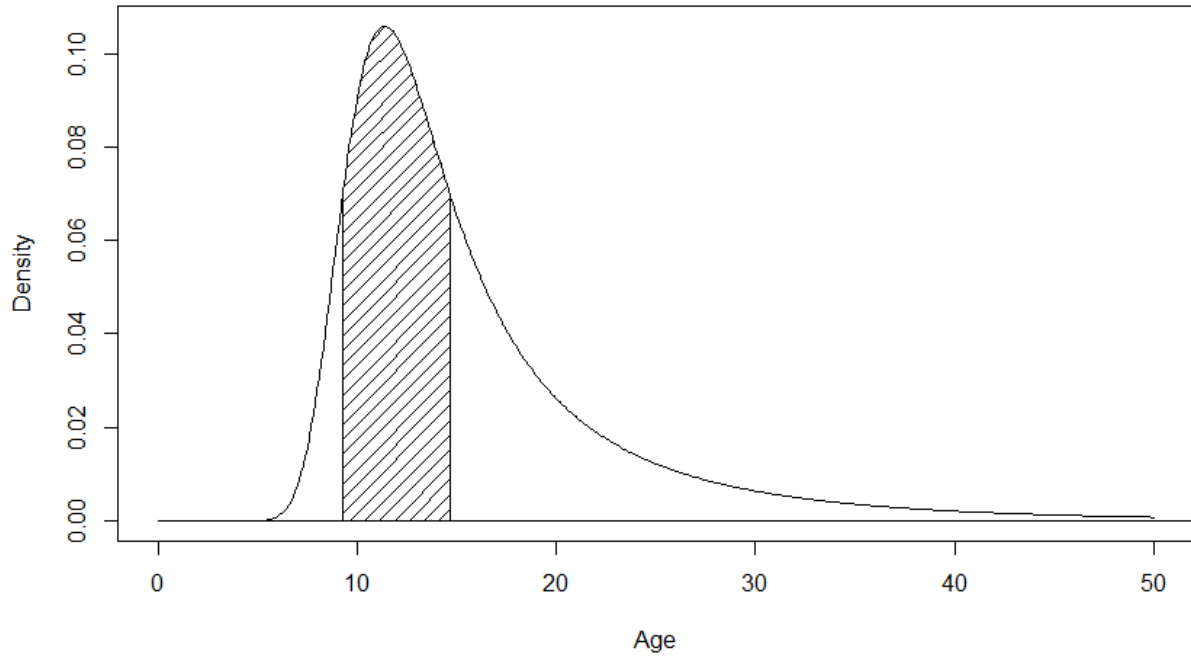
HPD male lower incisor 2 stage 12 (Rc), US Deaths 2008 prior



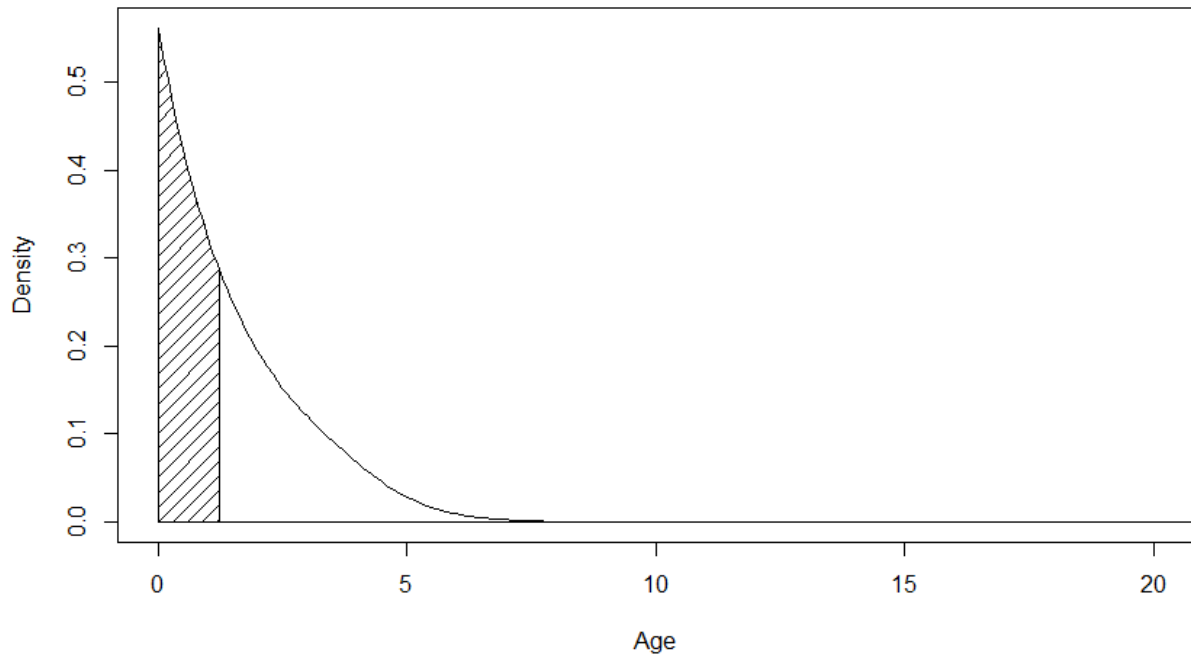
HPD male lower incisor 2 stage 13 (A 1/2), US Deaths 2008 prior



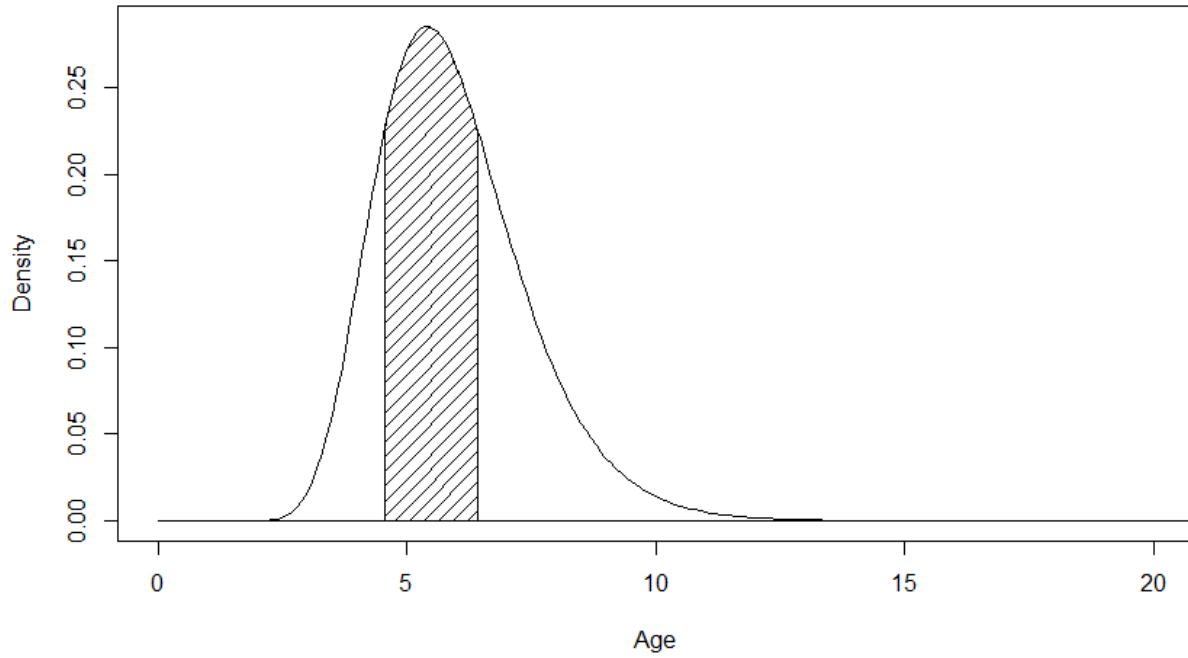
HPD male lower incisor 2 stage 14 (Ac), US Deaths 2008 prior



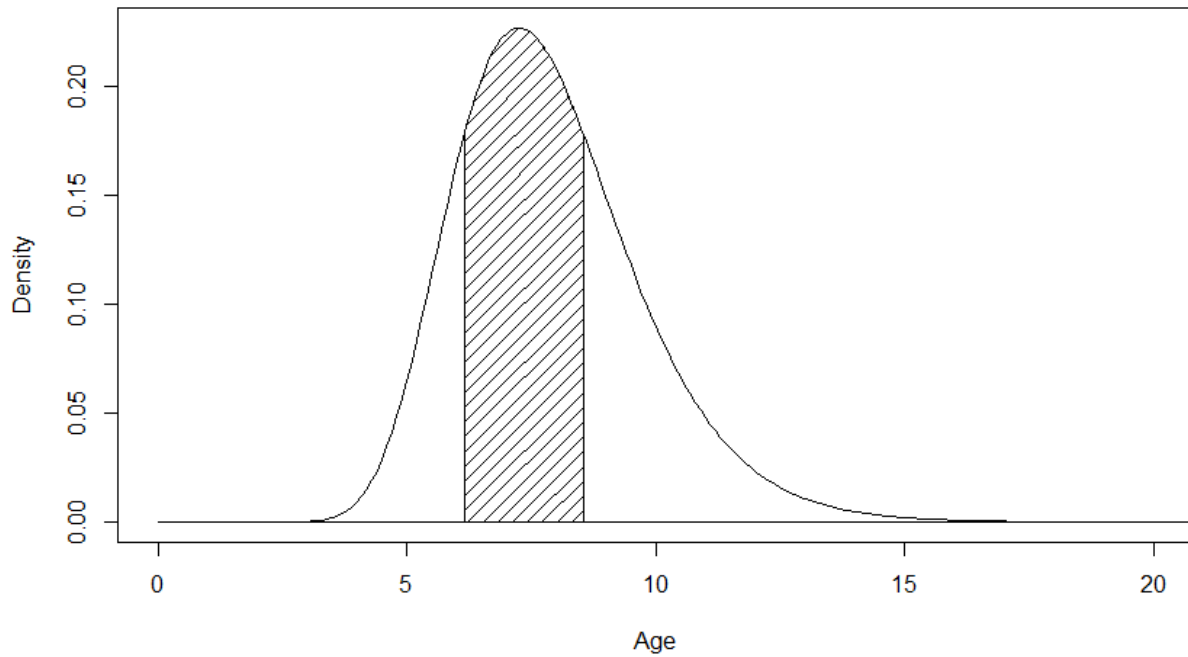
HPD male lower canine stage 7 (Ri), US Deaths 2008 prior



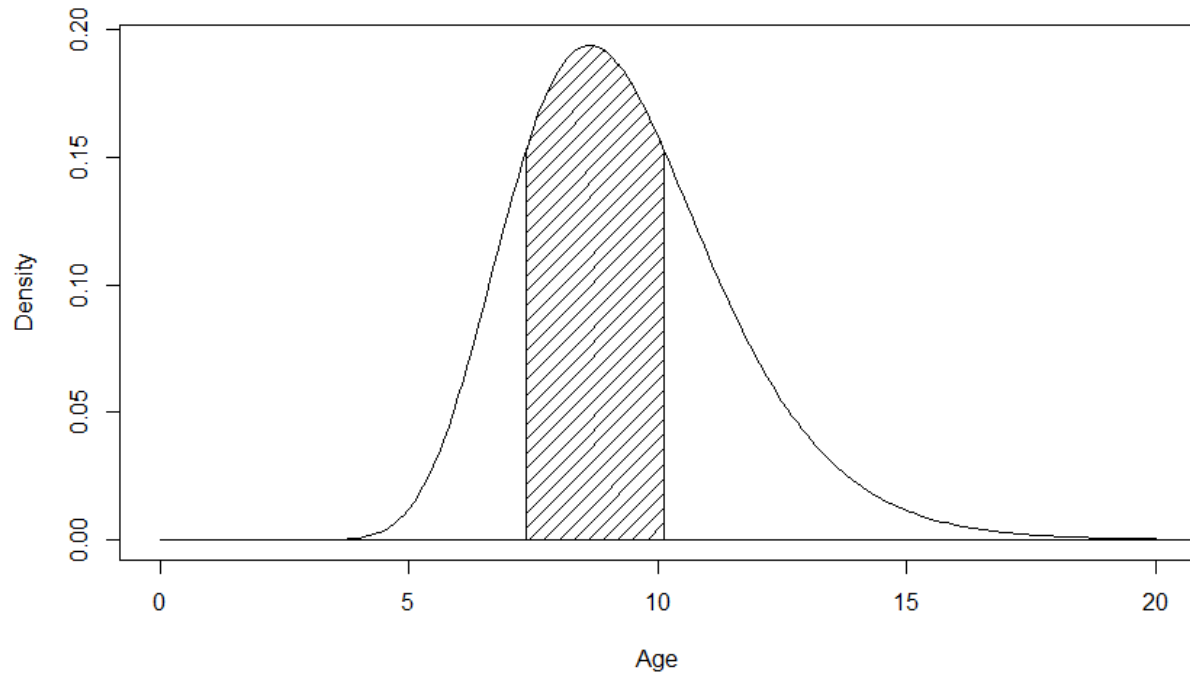
HPD male lower canine stage 9 (R 1/4), US Deaths 2008 prior



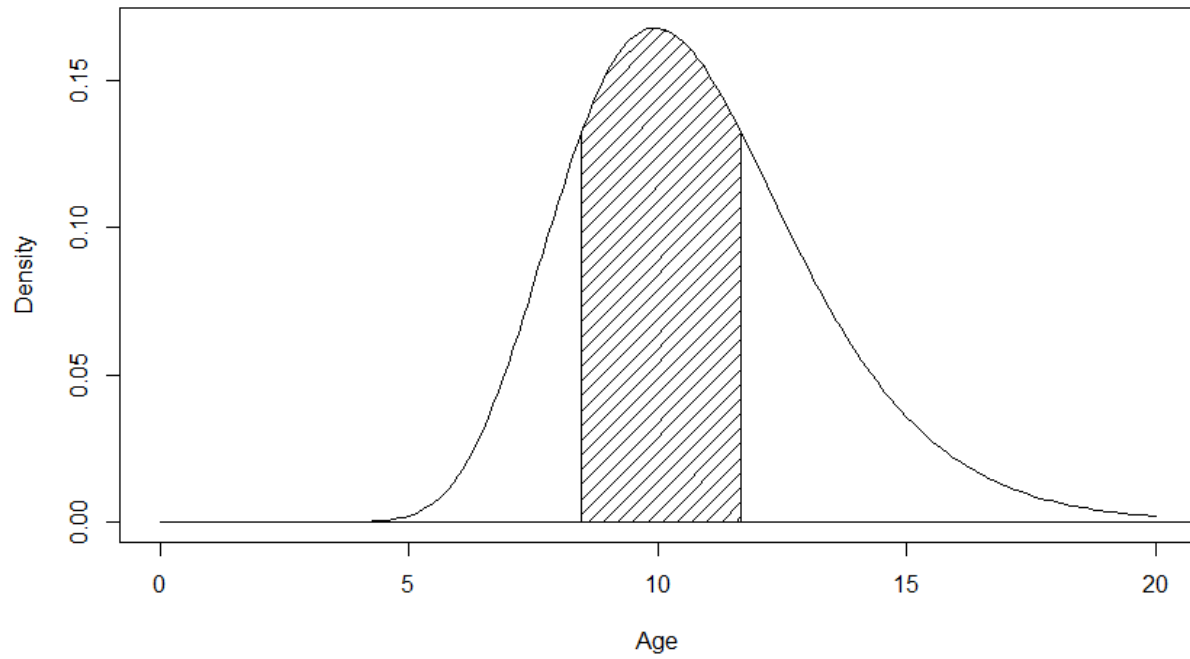
HPD male lower canine stage 10 (R 1/2), US Deaths 2008 prior



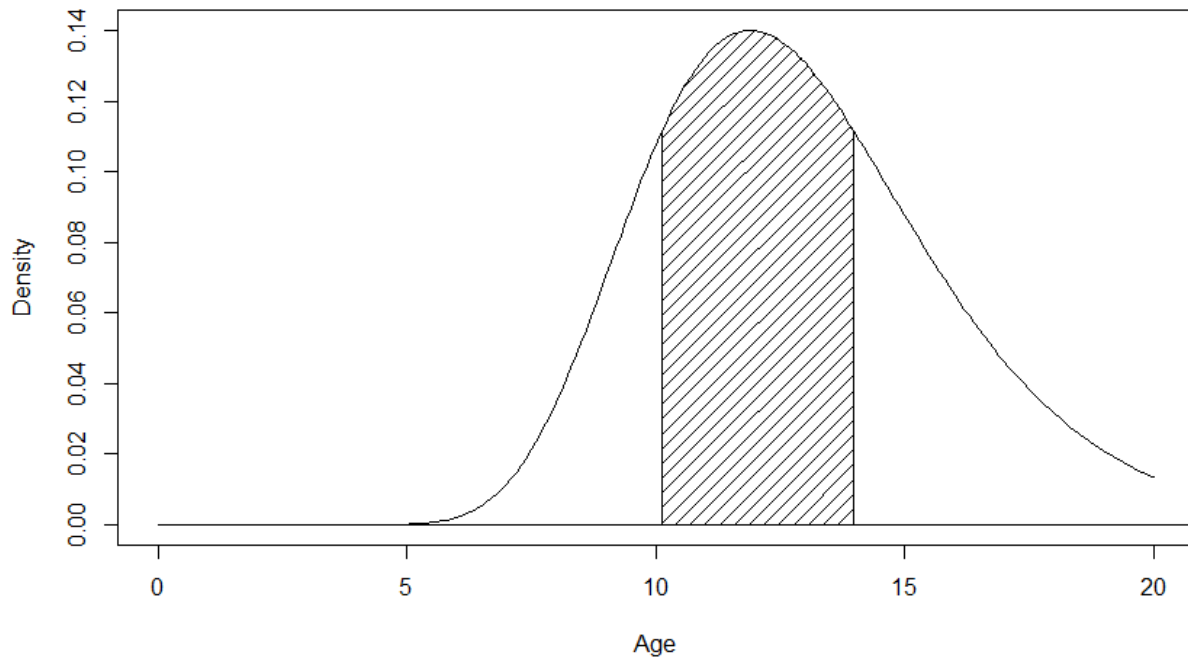
HPD male lower canine stage 11 (R 3/4), US Deaths 2008 prior



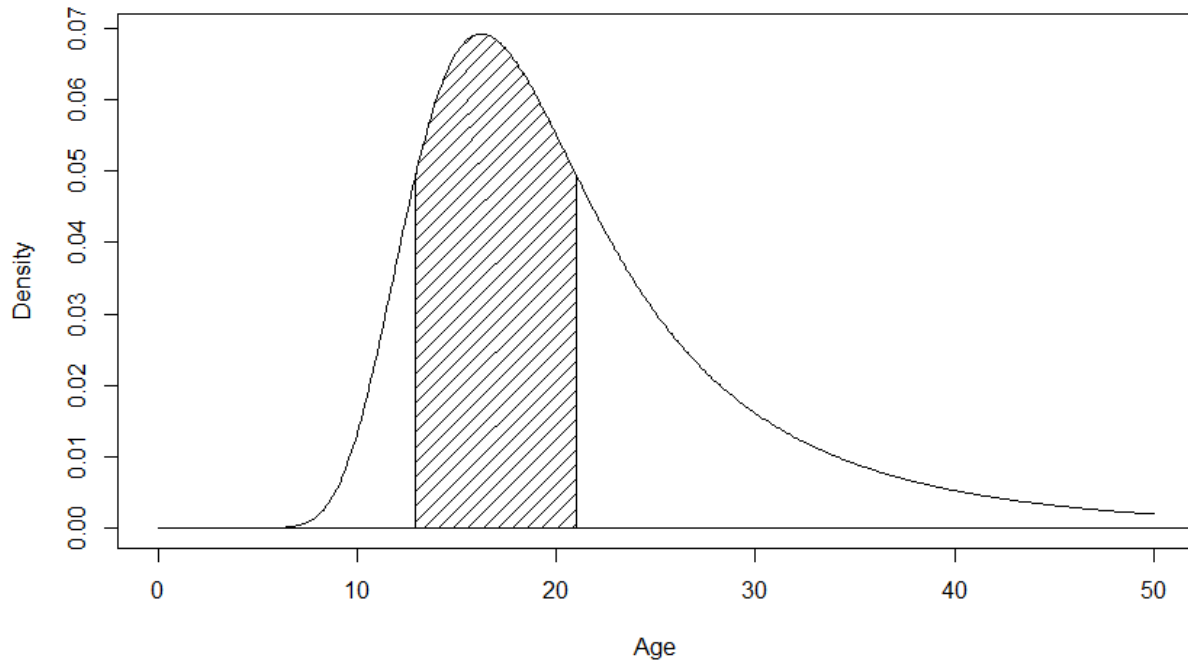
HPD male lower canine stage 12 (Rc), US Deaths 2008 prior



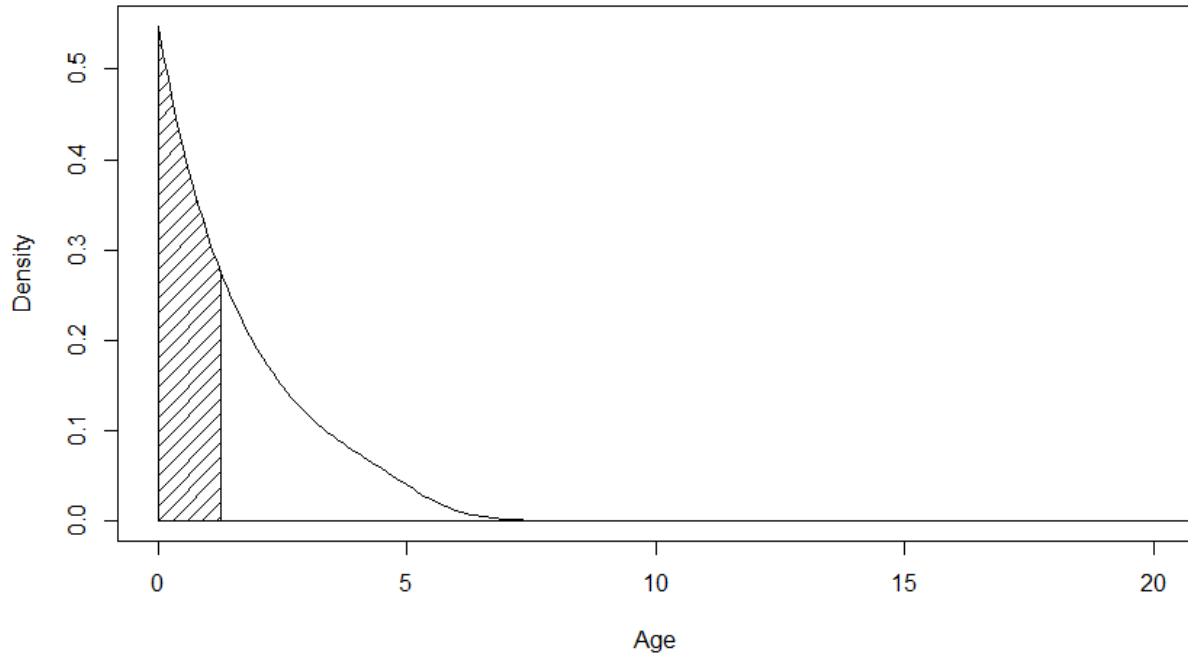
HPD male lower canine stage 13 (A 1/2), US Deaths 2008 prior



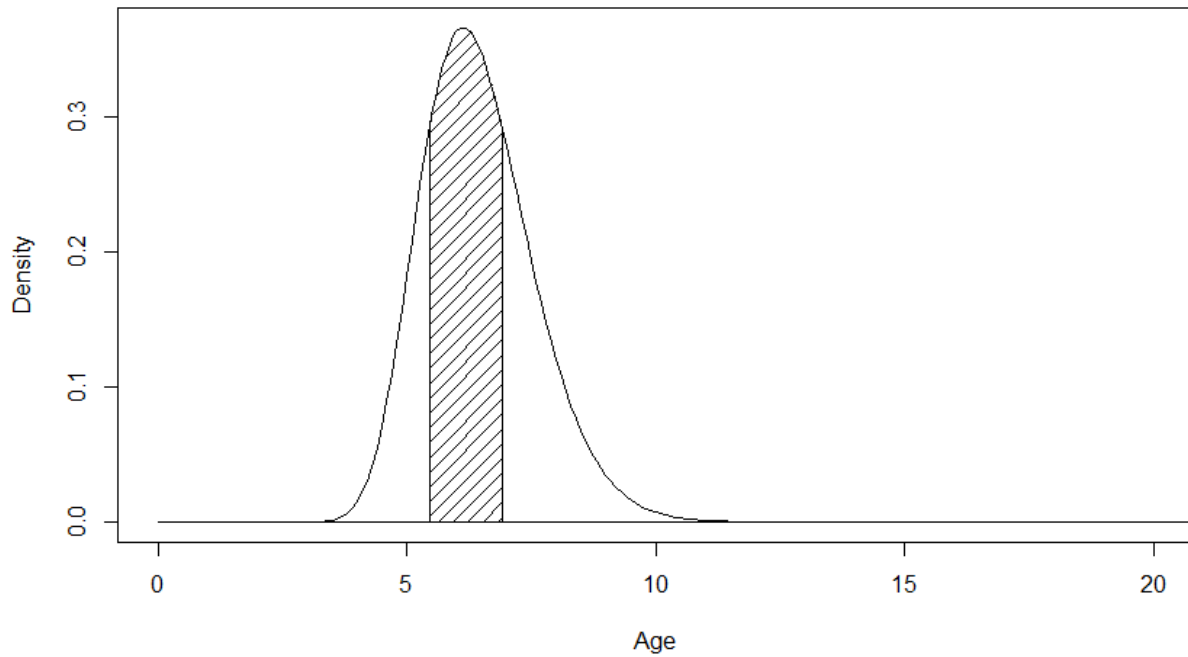
HPD male lower canine stage 14 (Ac), US Deaths 2008 prior



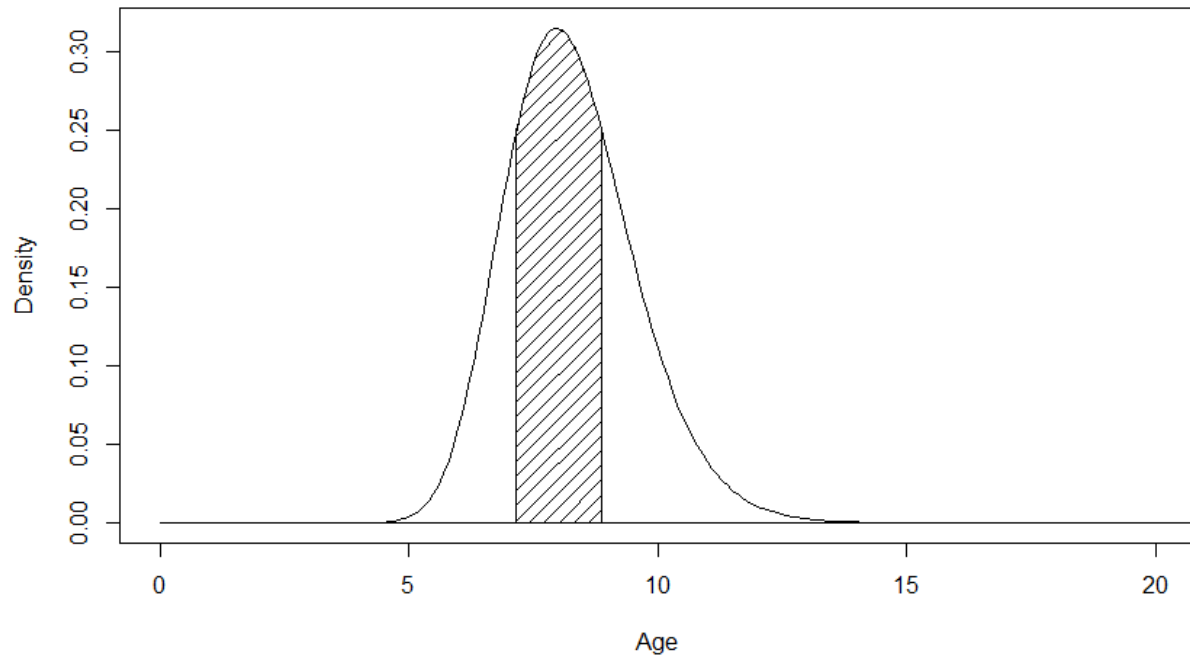
HPD male lower premolar 1 stage 6 (Crc), US Deaths 2008 prior



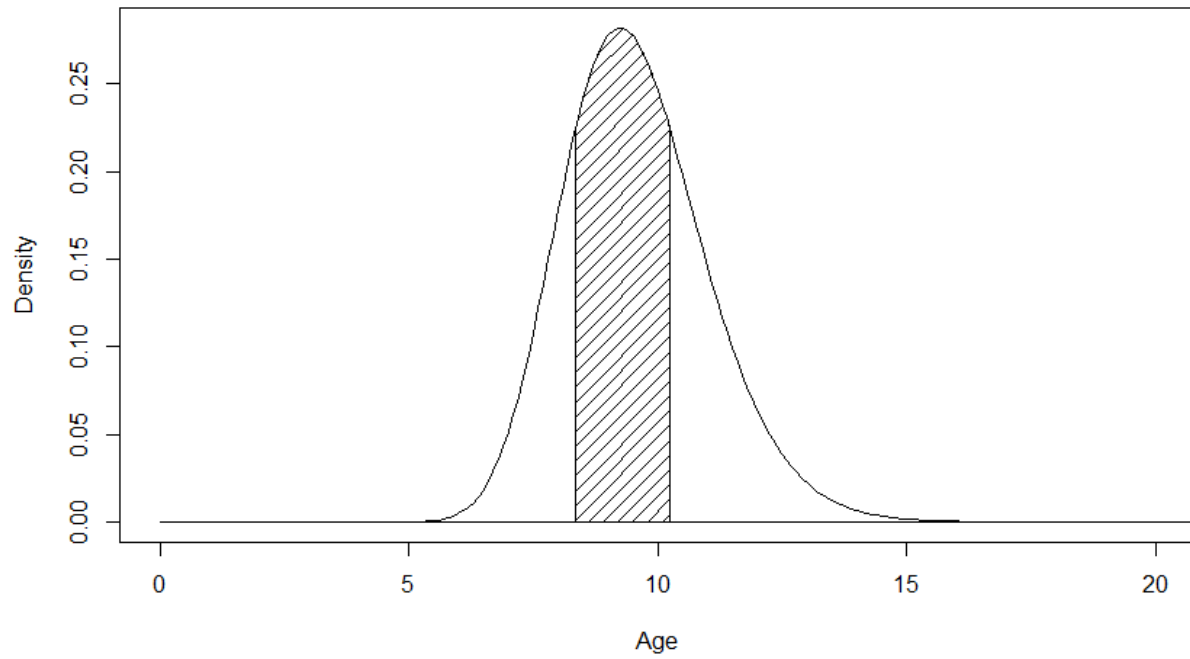
HPD male lower premolar 1 stage 7 (Ri), US Deaths 2008 prior



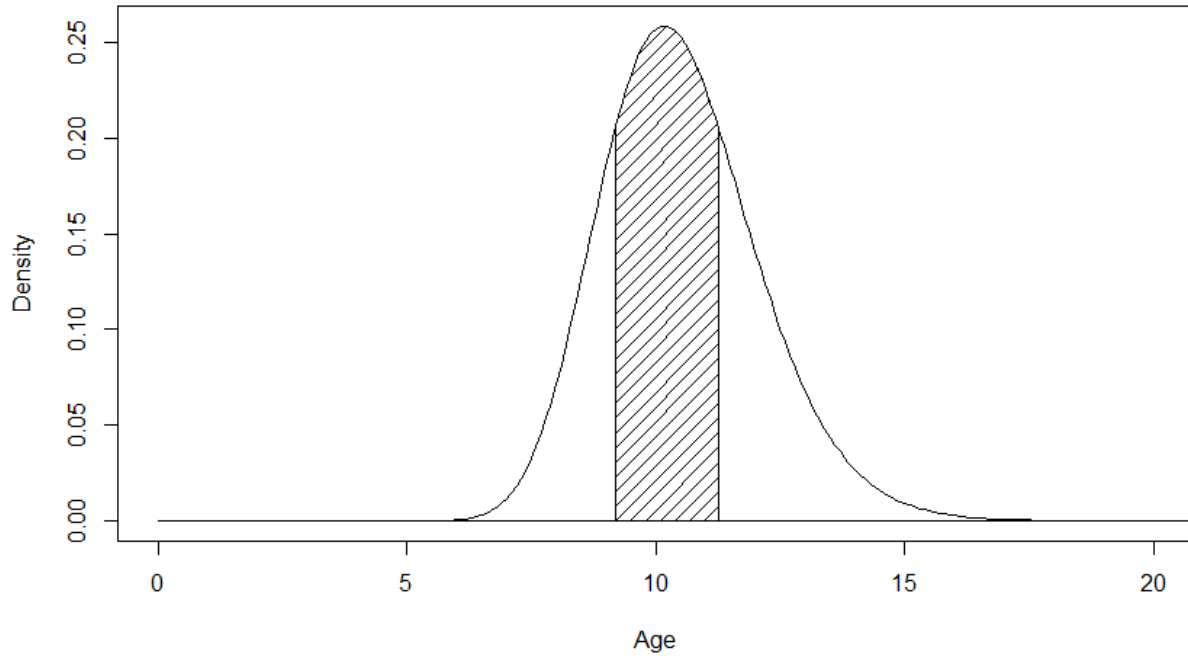
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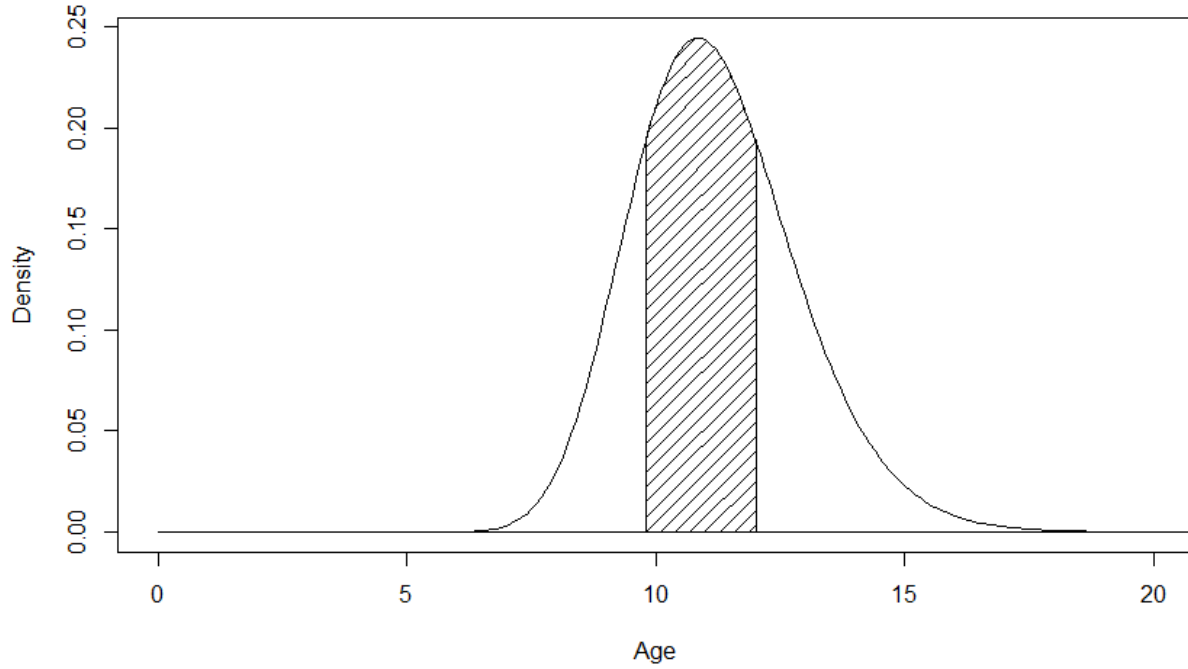
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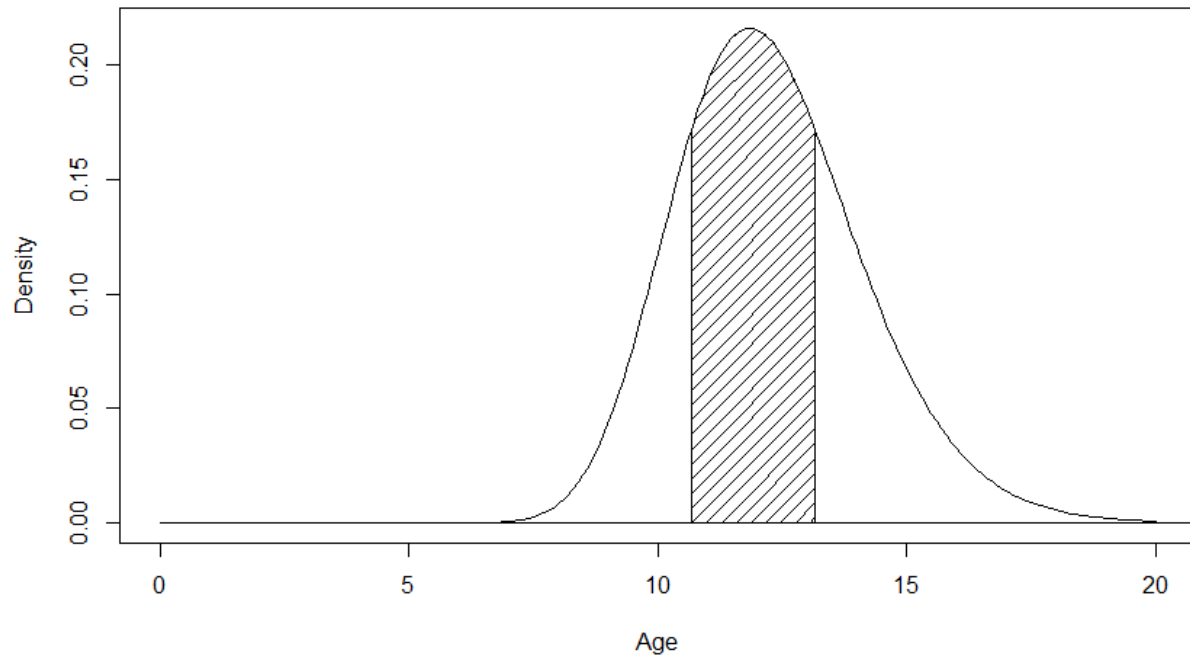
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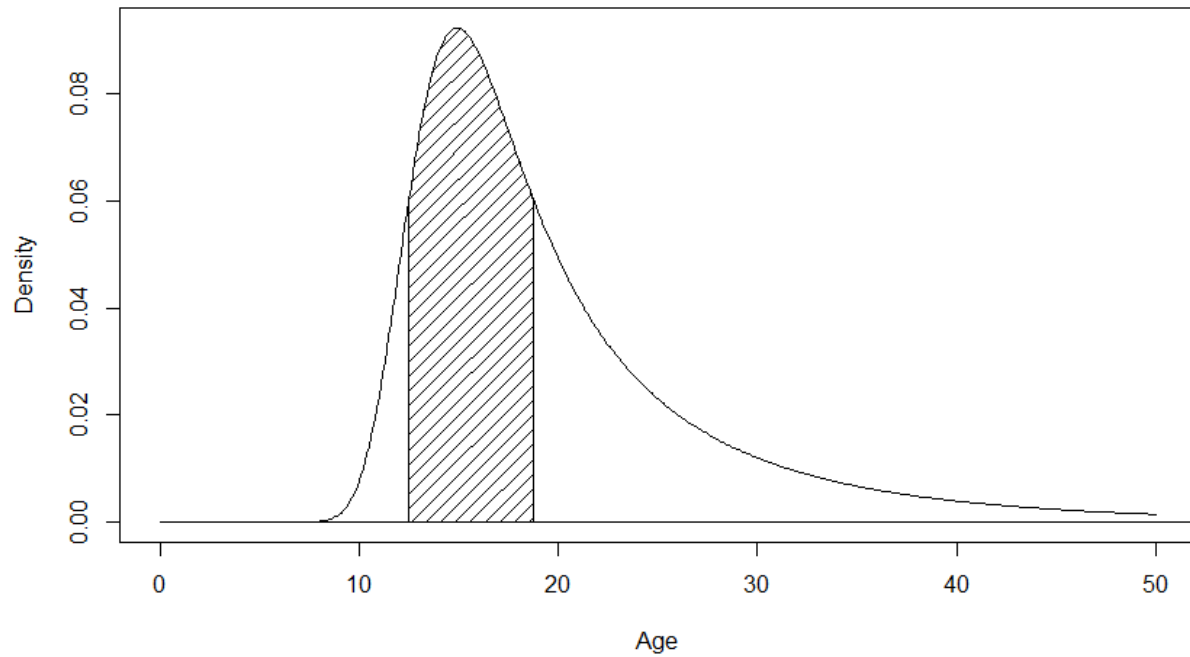
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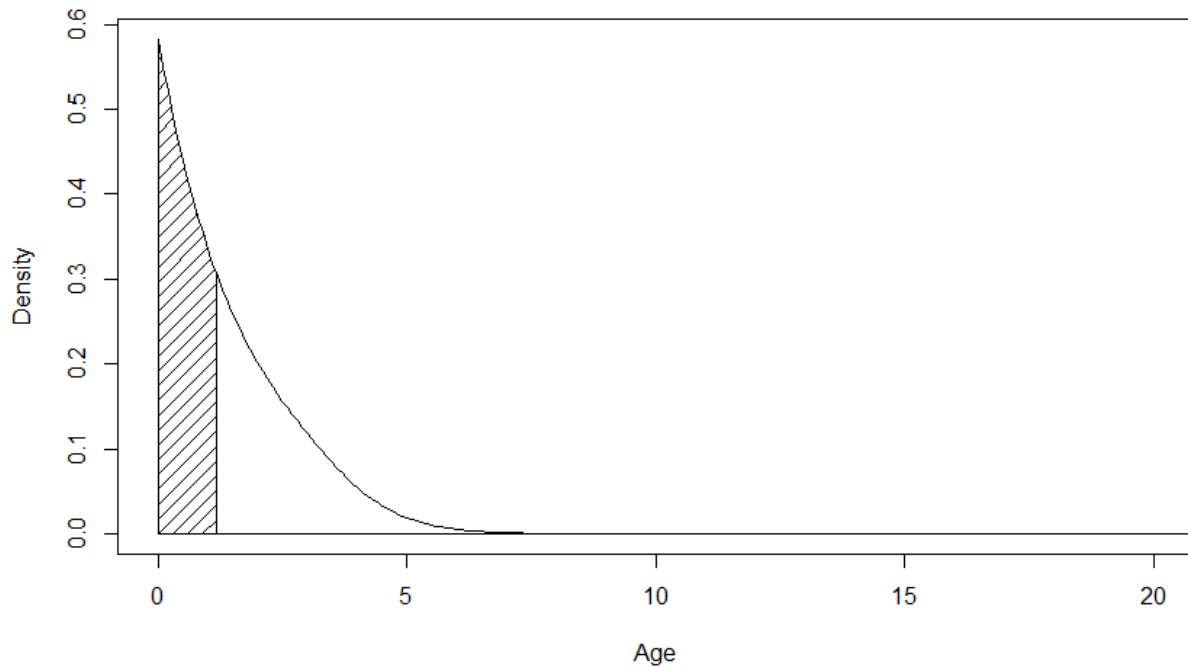
HPD male lower premolar 1 stage 13 (A 1/2), US Deaths 2008 prior



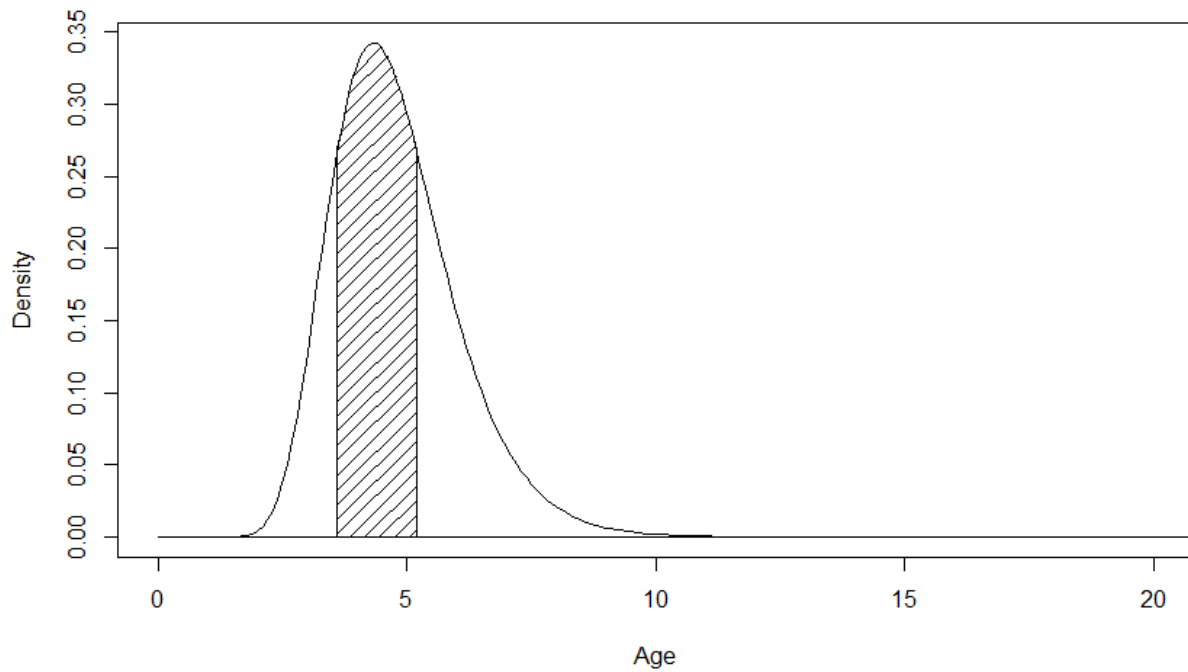
HPD male lower premolar 1 stage 14 (Ac), US Deaths 2008 prior



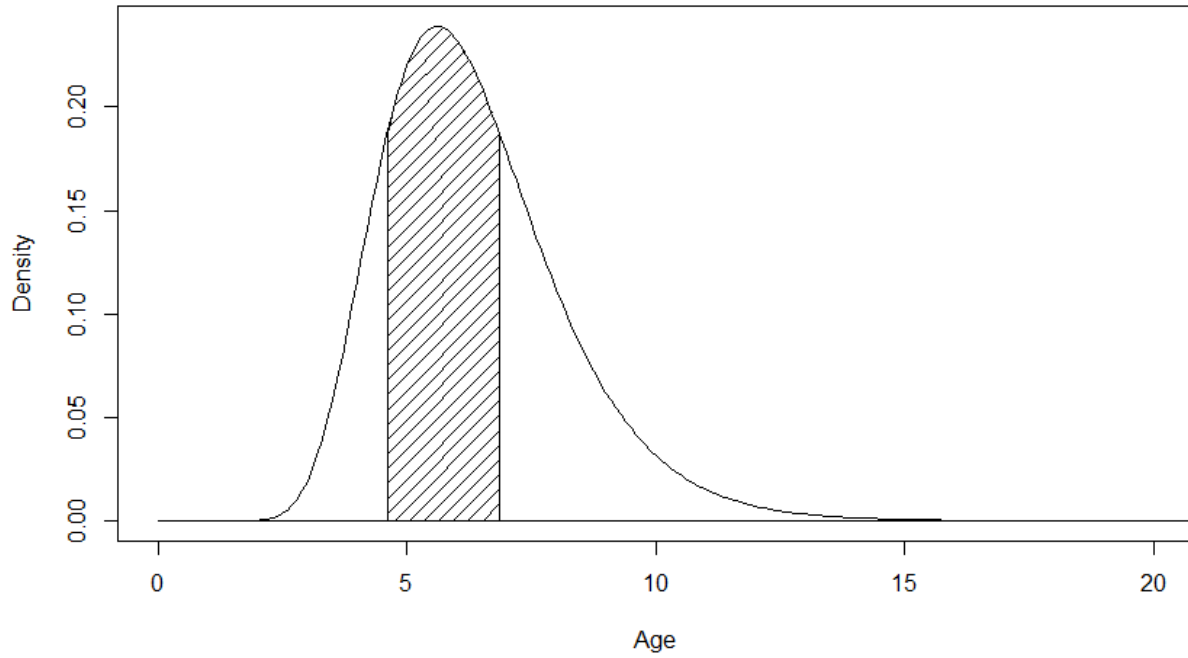
HPD male lower premolar 2 stage 5 (Cr 3/4), US Deaths 2008 prior



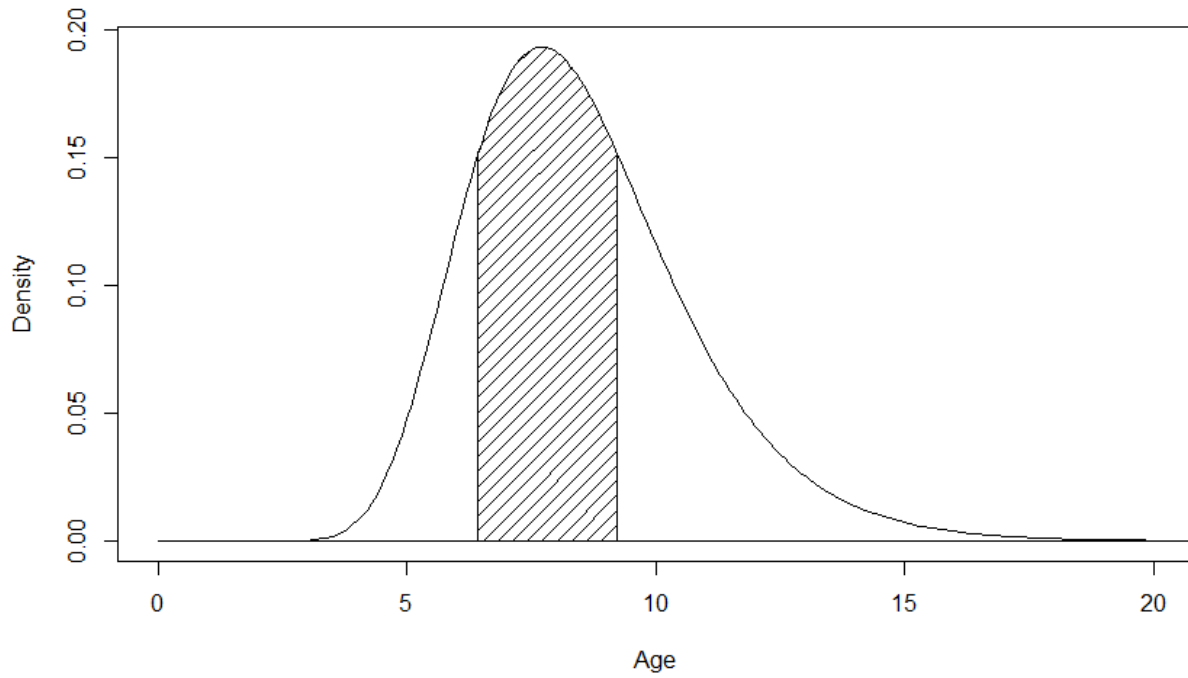
HPD male lower premolar 2 stage 6 (Crc), US Deaths 2008 prior



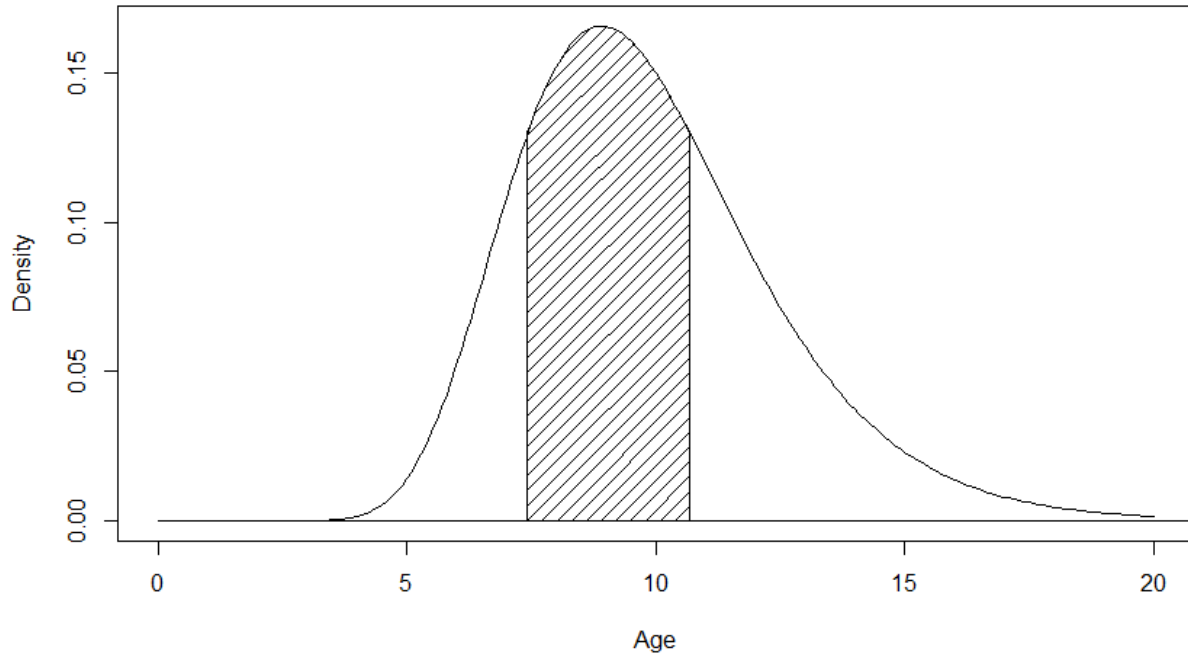
HPD male lower premolar 2 stage 7 (Ri), US Deaths 2008 prior



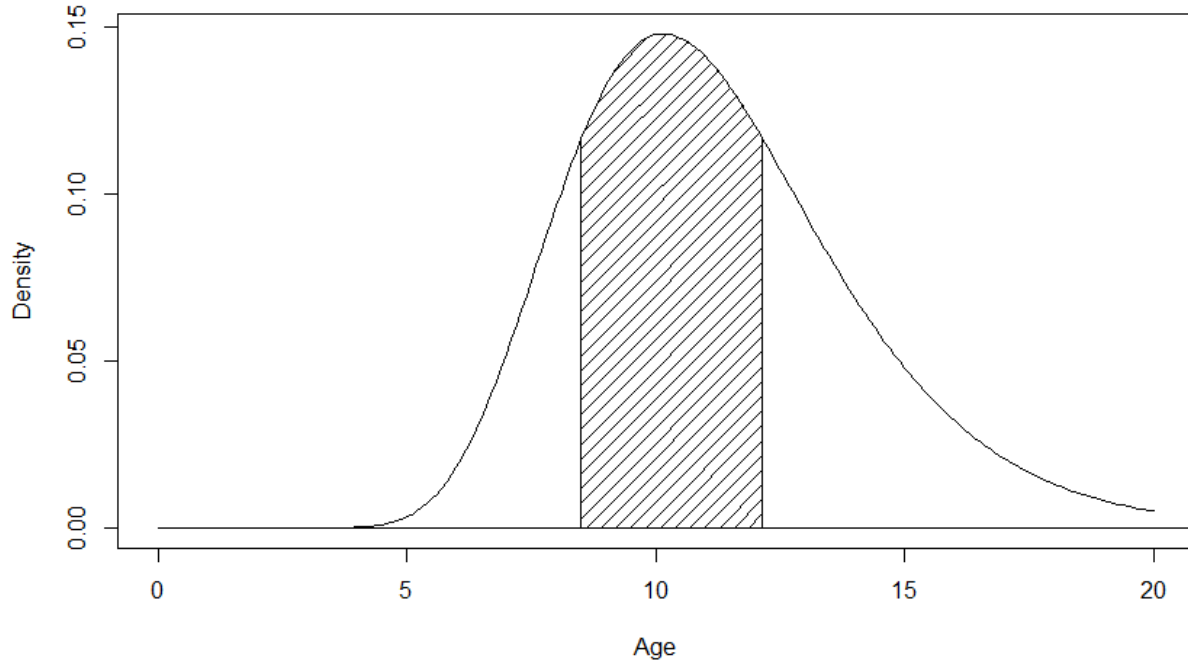
HPD male lower premolar 2 stage 9 (R 1/4), US Deaths 2008 prior



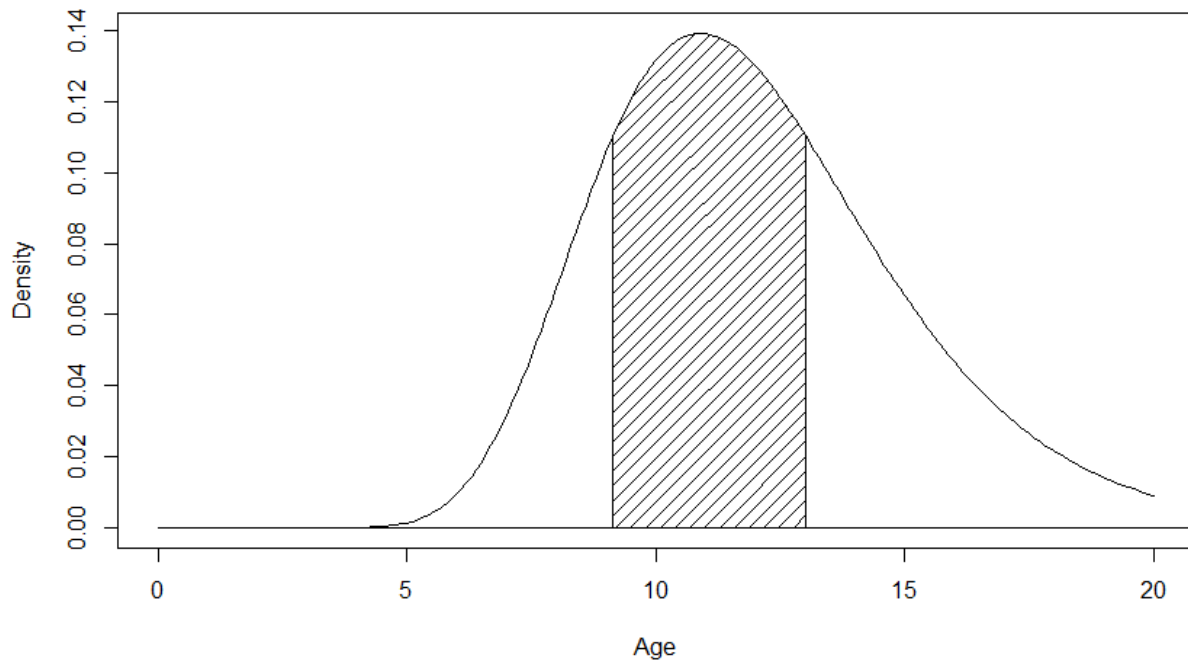
HPD male lower premolar 2 stage 10 (R 1/2), US Deaths 2008 prior



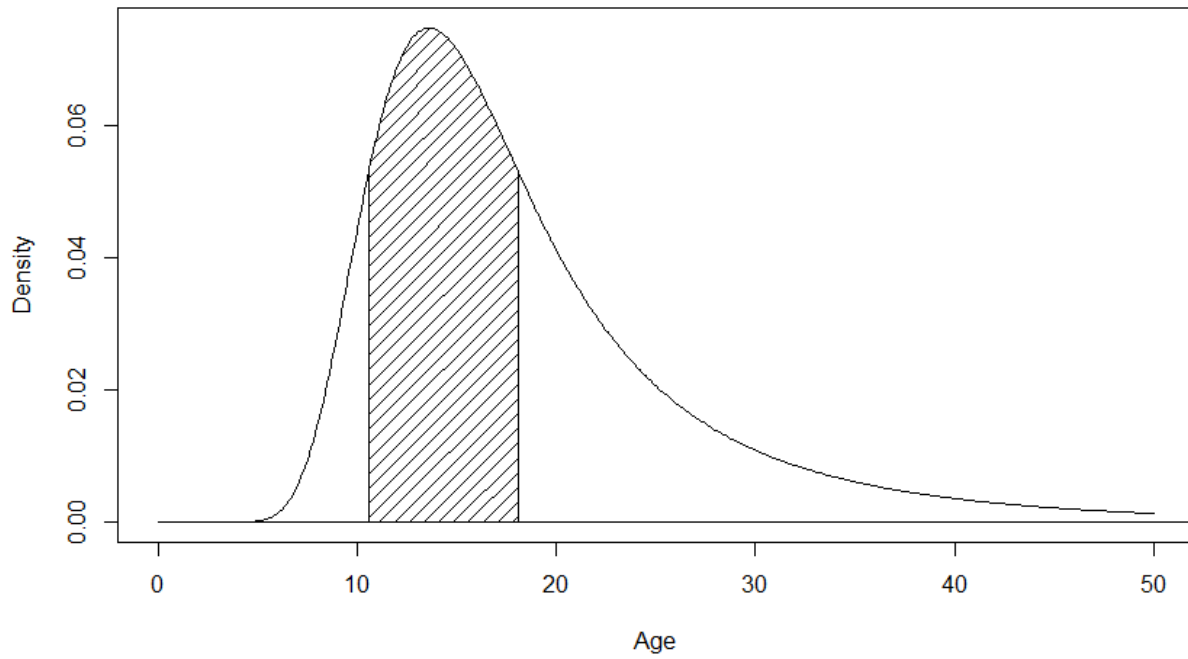
HPD male lower premolar 2 stage 11 (R 3/4), US Deaths 2008 prior



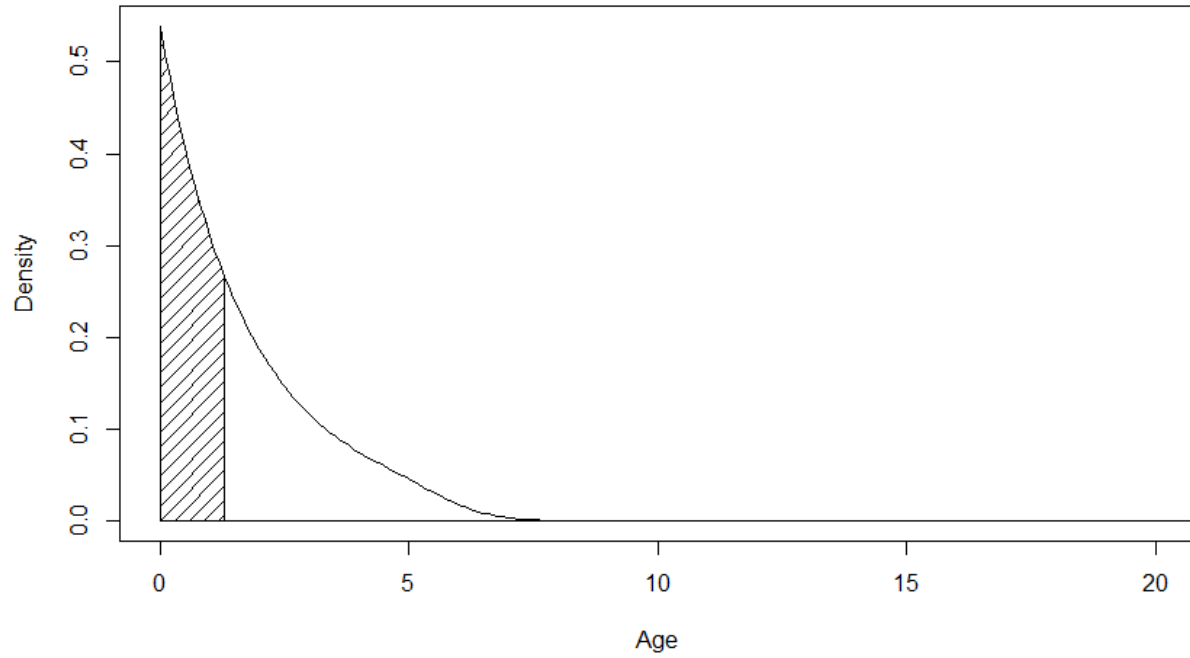
HPD male lower premolar 2 stage 12 (Rc), US Deaths 2008 prior



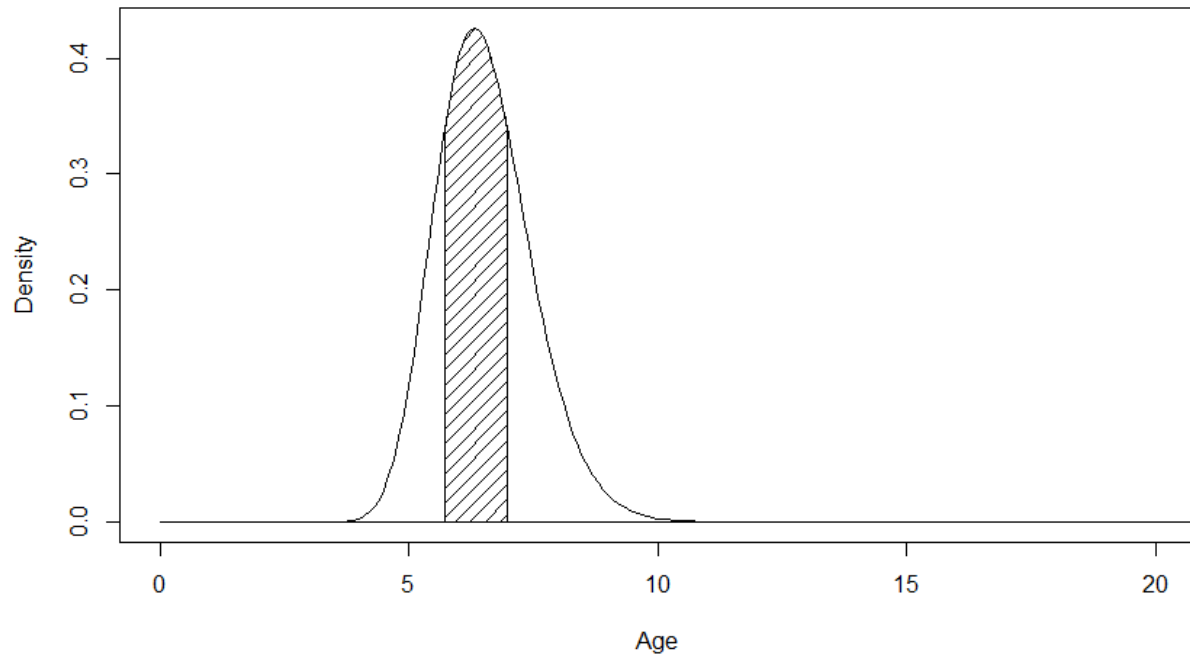
HPD male lower premolar 2 stage 13 (A 1/2), US Deaths 2008 prior



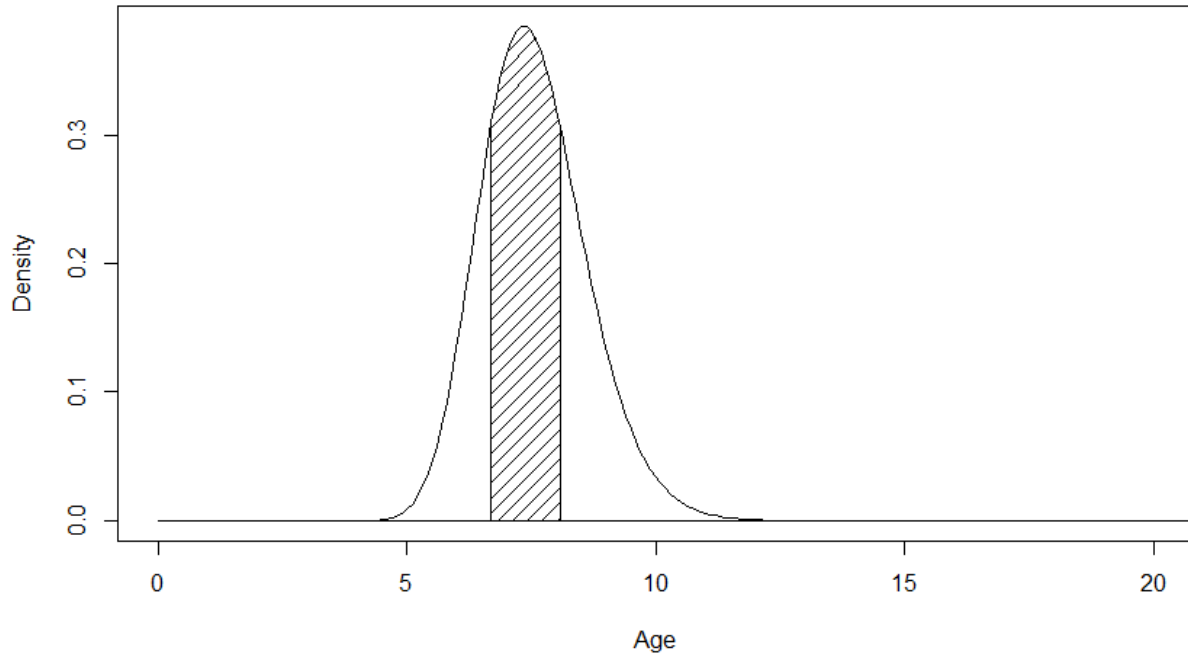
HPD male lower molar 1 stage 10 (R 1/2), US Deaths 2008 prior



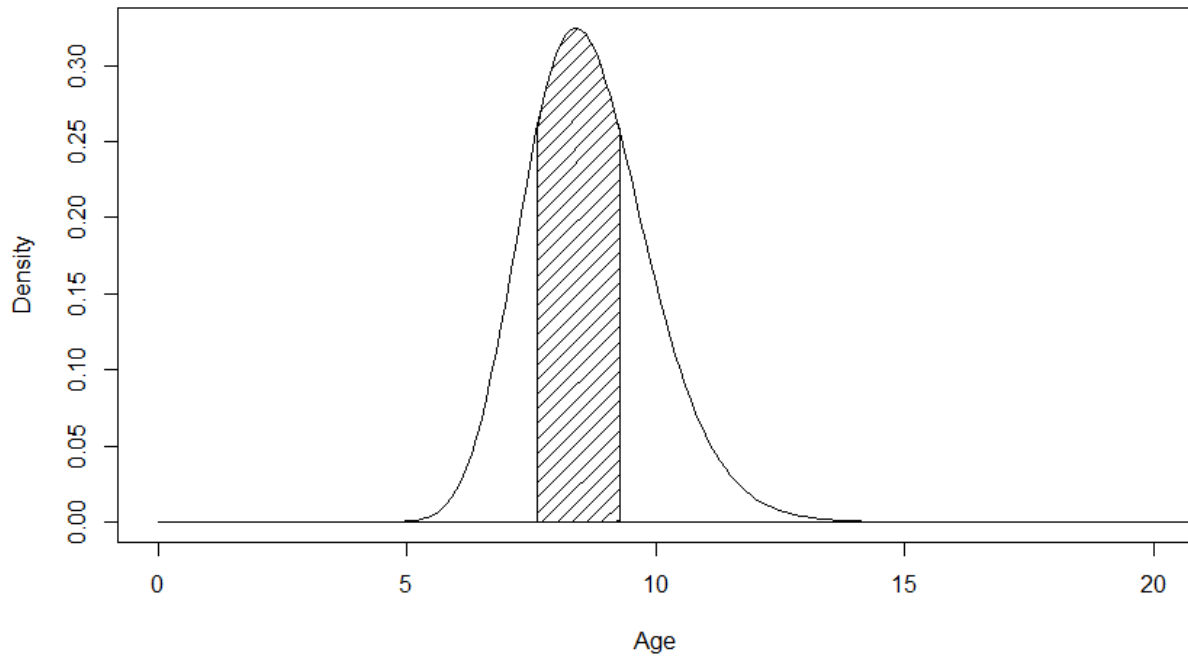
HPD male lower molar 1 stage 11 (R 3/4), US Deaths 2008 prior



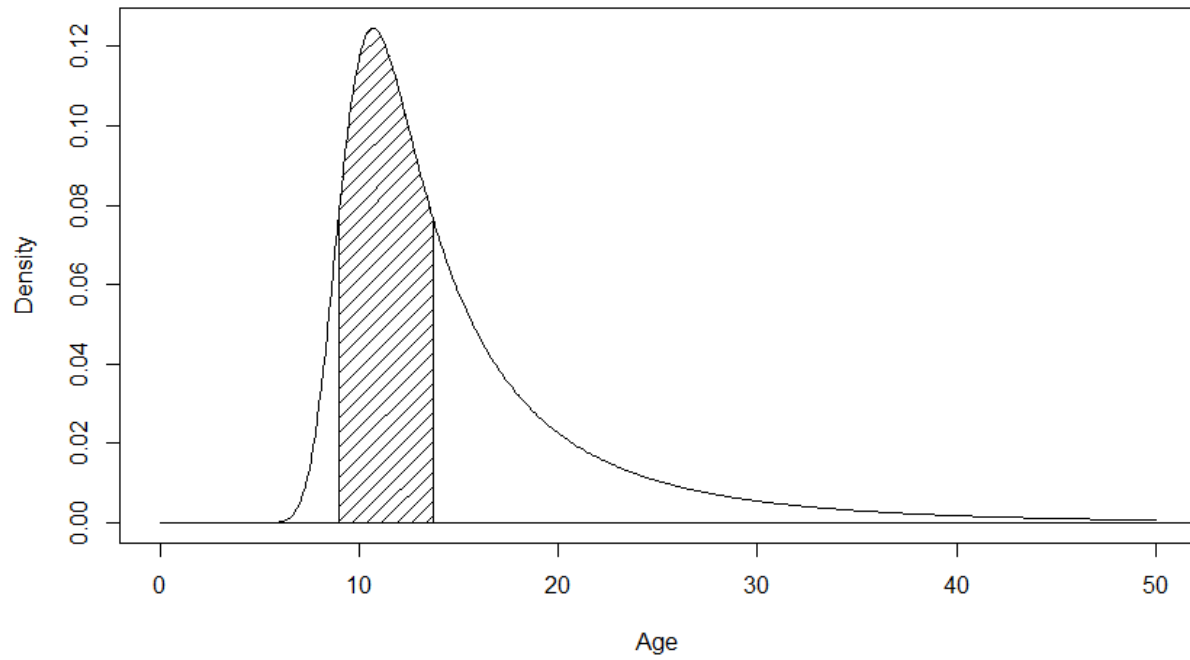
HPD male lower molar 1 stage 12 (Rc), US Deaths 2008 prior



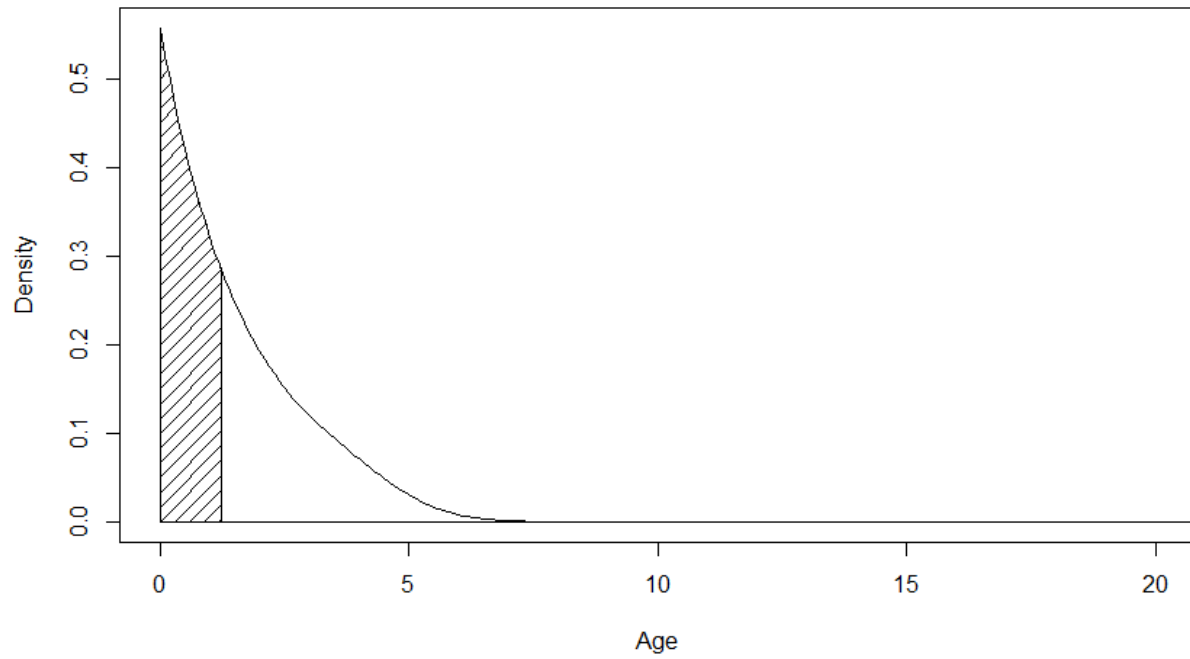
HPD male lower molar 1 stage 13 (A 1/2), US Deaths 2008 prior



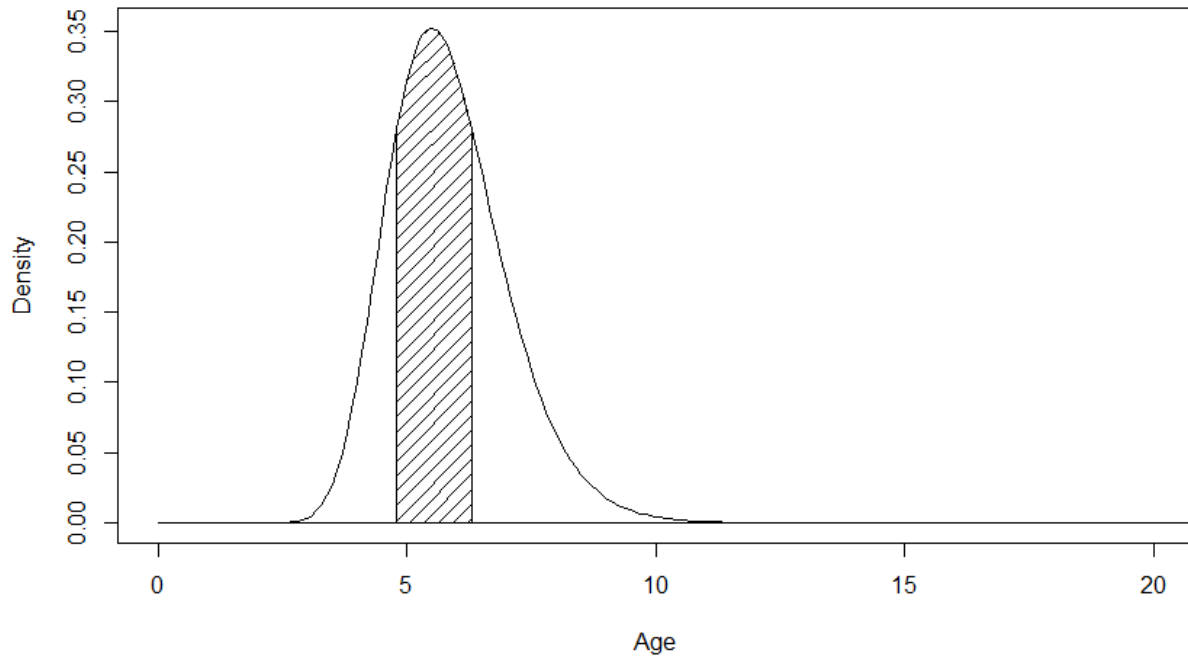
HPD male lower molar 1 stage 14 (Ac), US Deaths 2008 prior



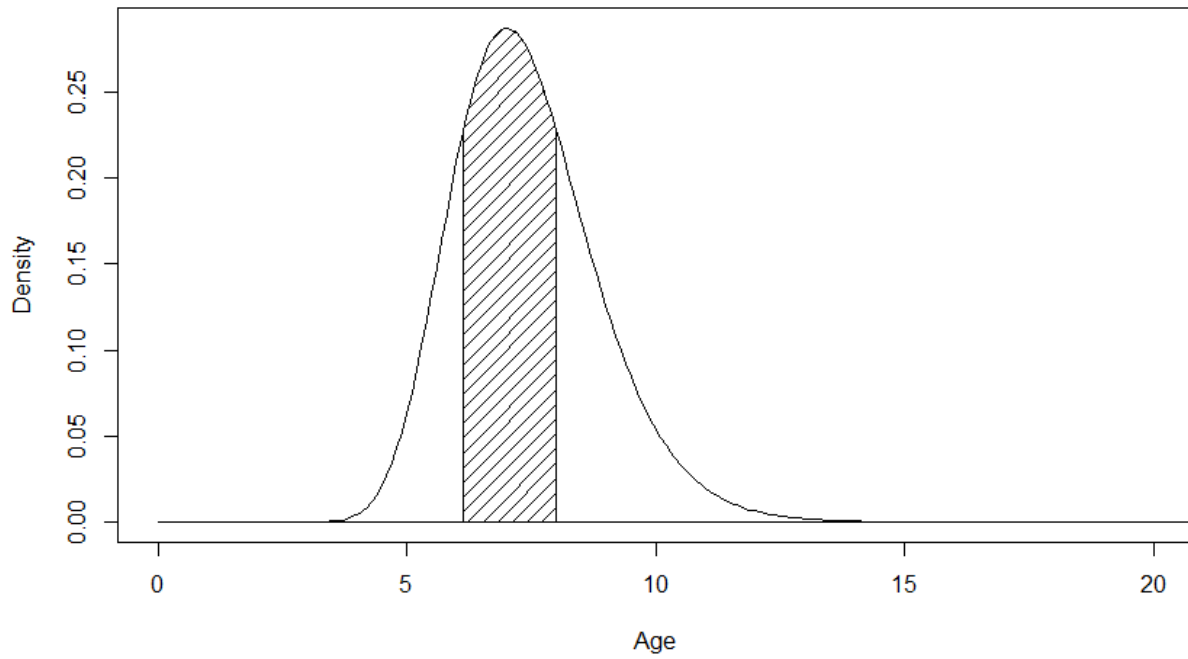
HPD male lower molar 2 stage 5 (Cr 3/4), US Deaths 2008 prior



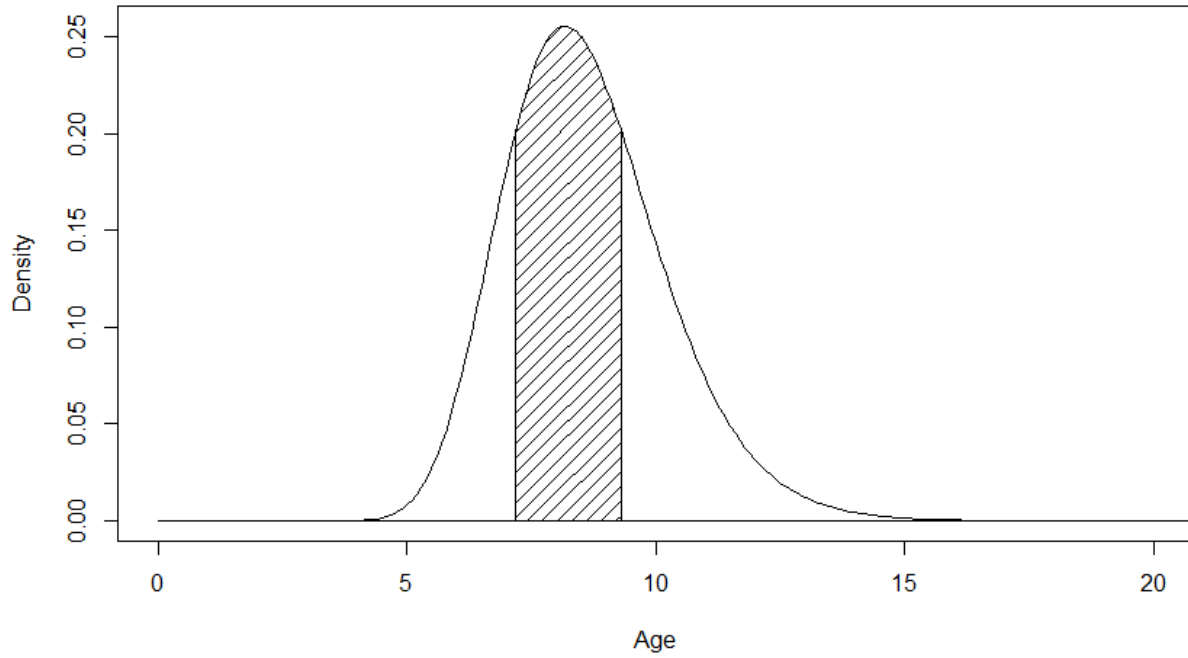
HPD male lower molar 2 stage 6 (Crc), US Deaths 2008 prior



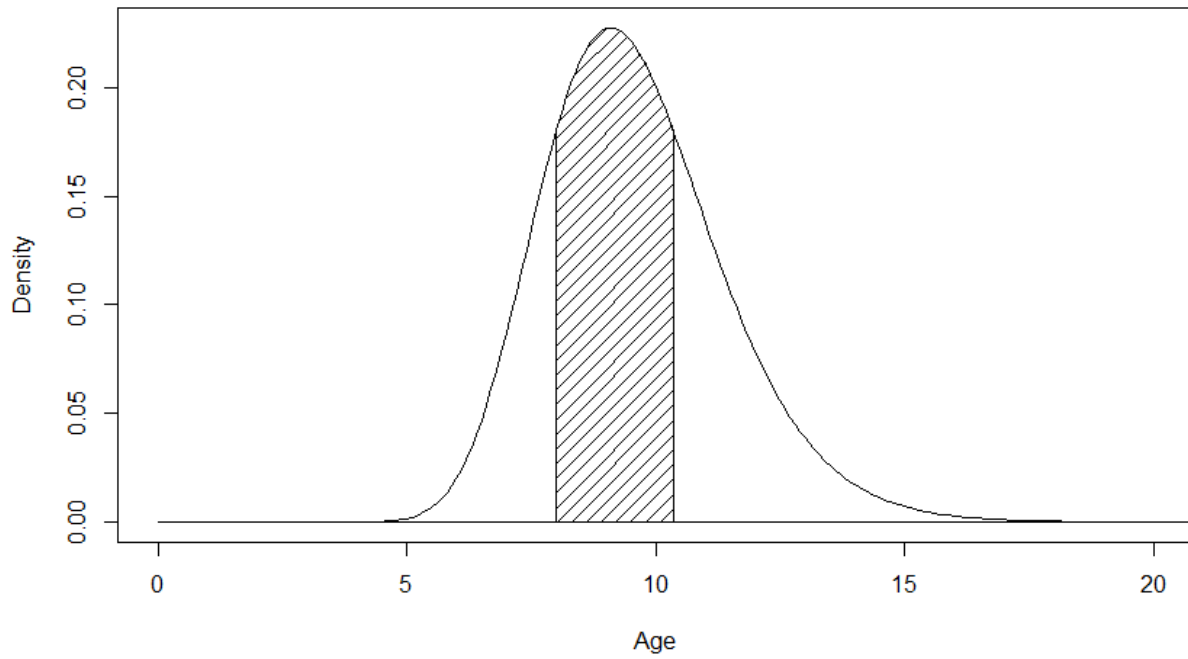
HPD male lower molar 2 stage 7 (Ri), US Deaths 2008 prior



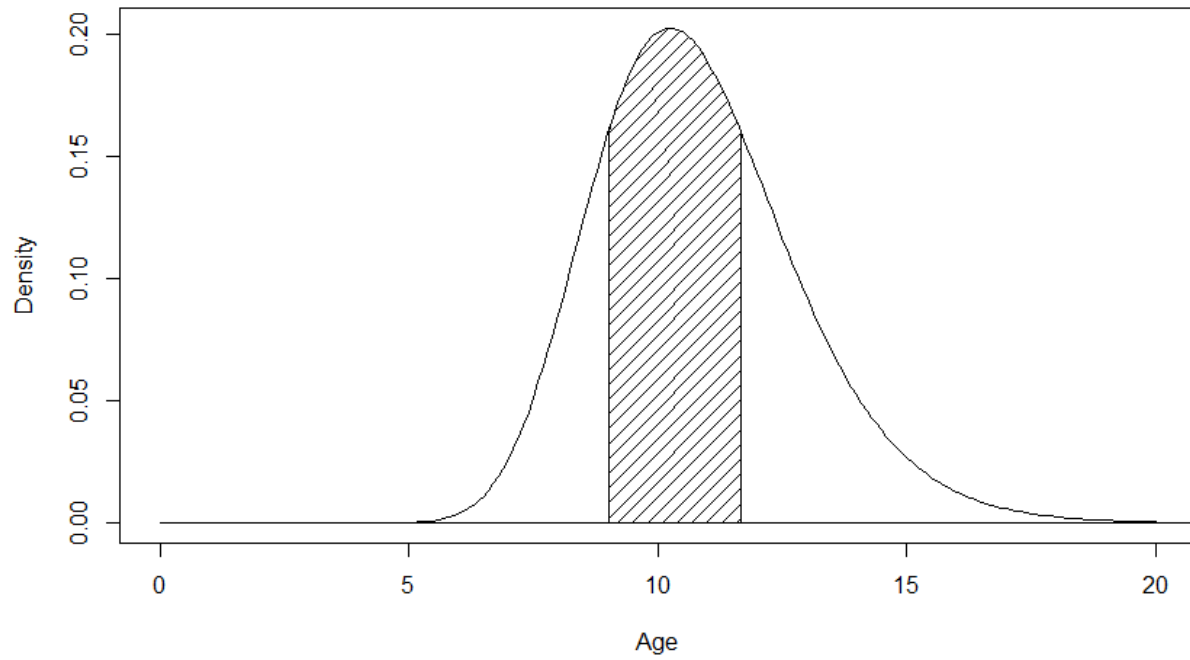
HPD male lower molar 2 stage 8 (C1i), US Deaths 2008 prior



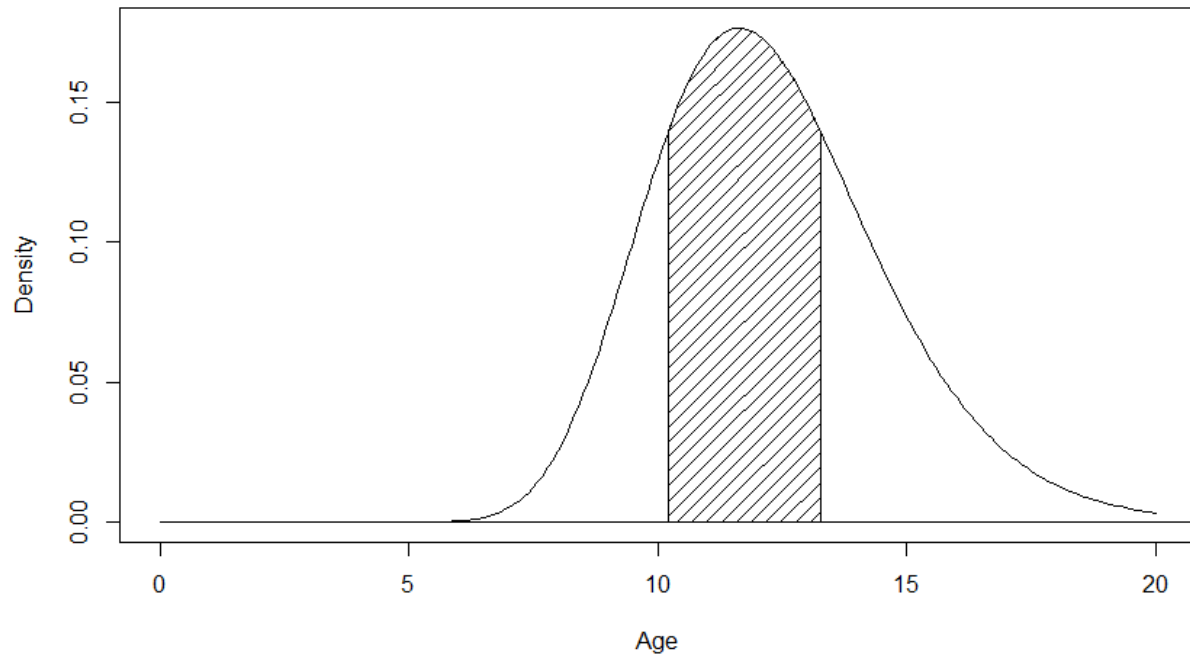
HPD male lower molar 2 stage 9 (R 1/4), US Deaths 2008 prior



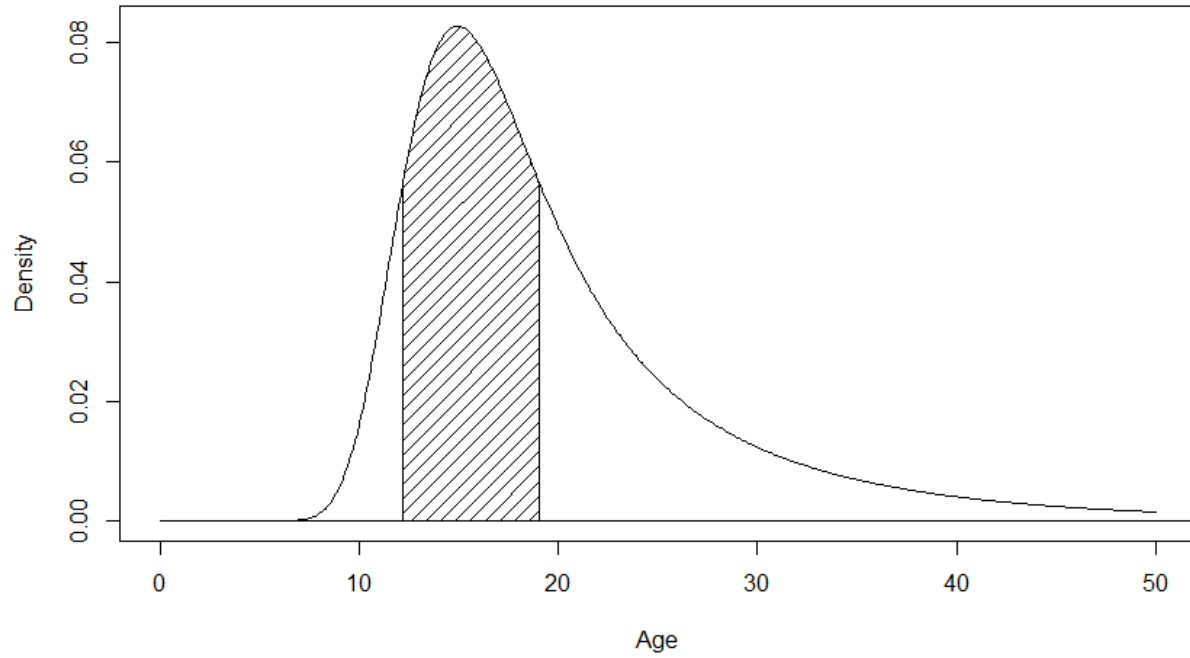
HPD male lower molar 2 stage 10 (R 1/2), US Deaths 2008 prior



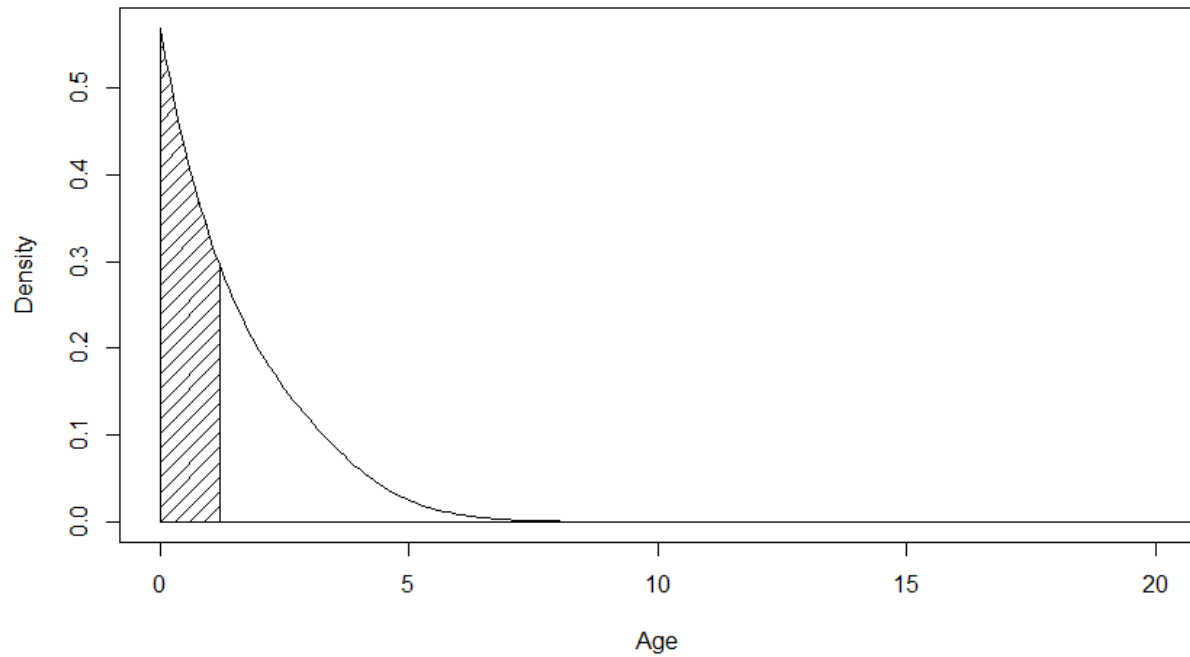
HPD male lower molar 2 stage 11 (R 3/4), US Deaths 2008 prior



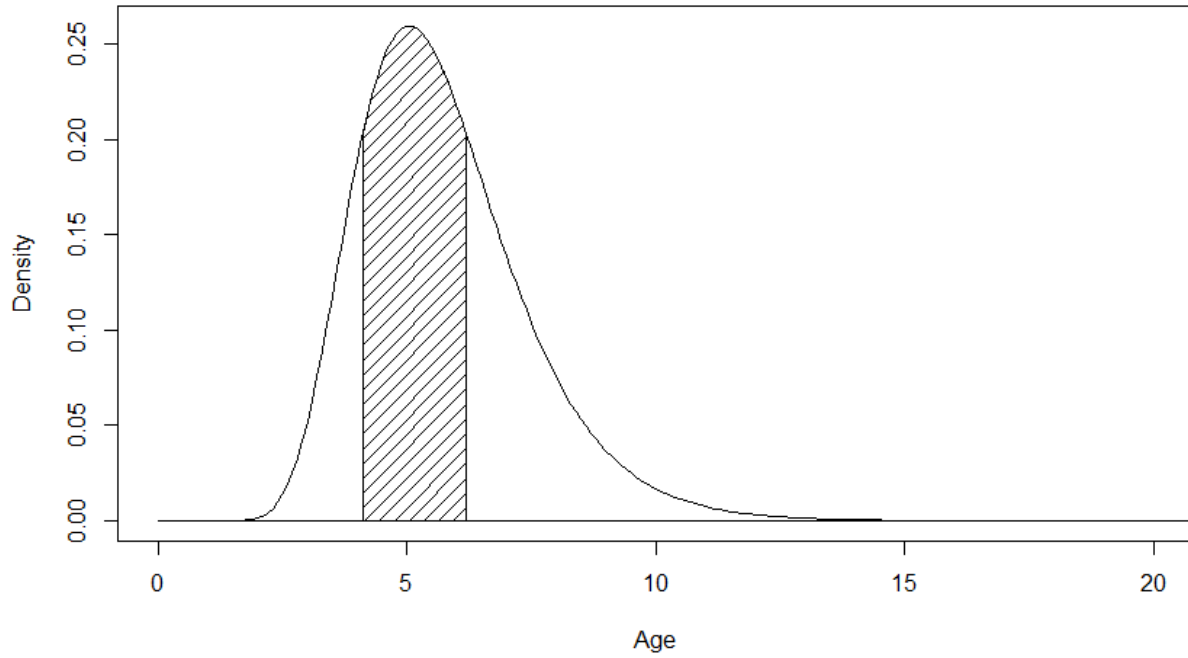
HPD male lower molar 2 stage 12 (Rc), US Deaths 2008 prior



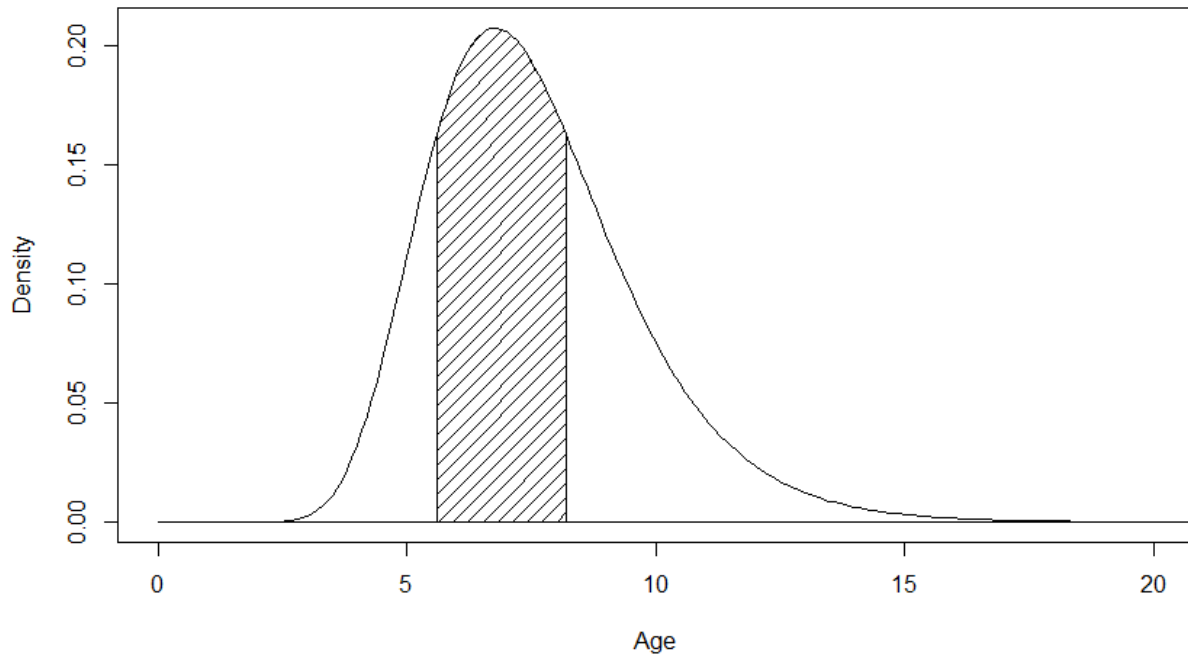
HPD male upper incisor 2 stage 9 (R 1/4), US Deaths 2008 prior



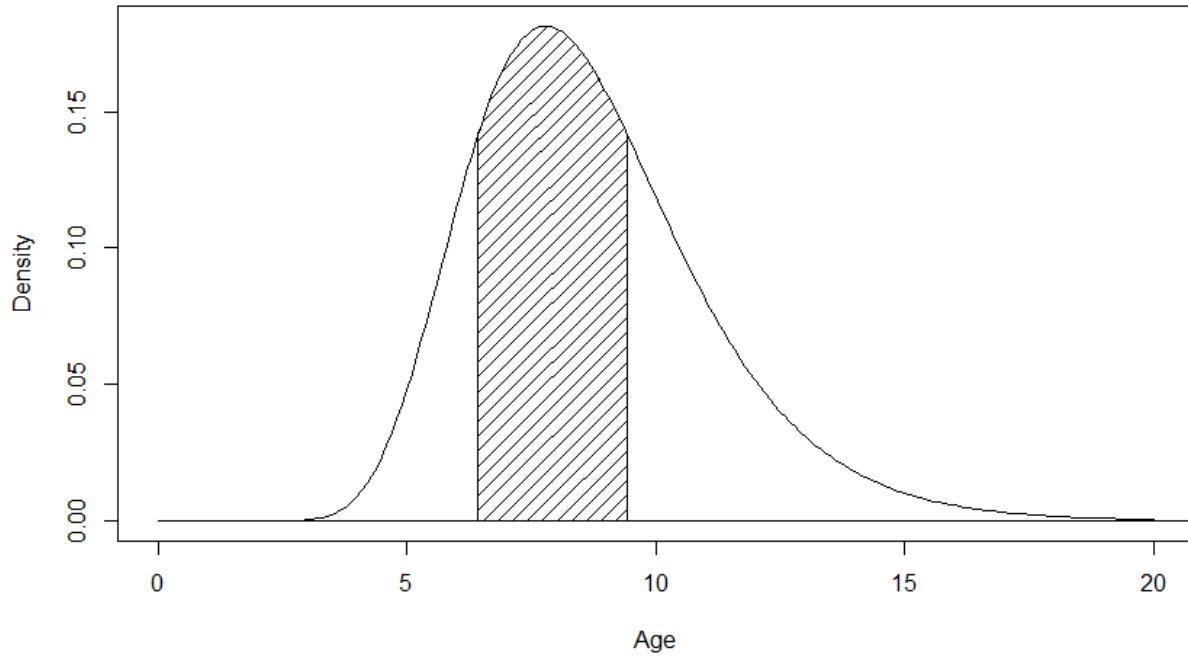
HPD male upper incisor 2 stage 10 (R 1/2), US Deaths 2008 prior



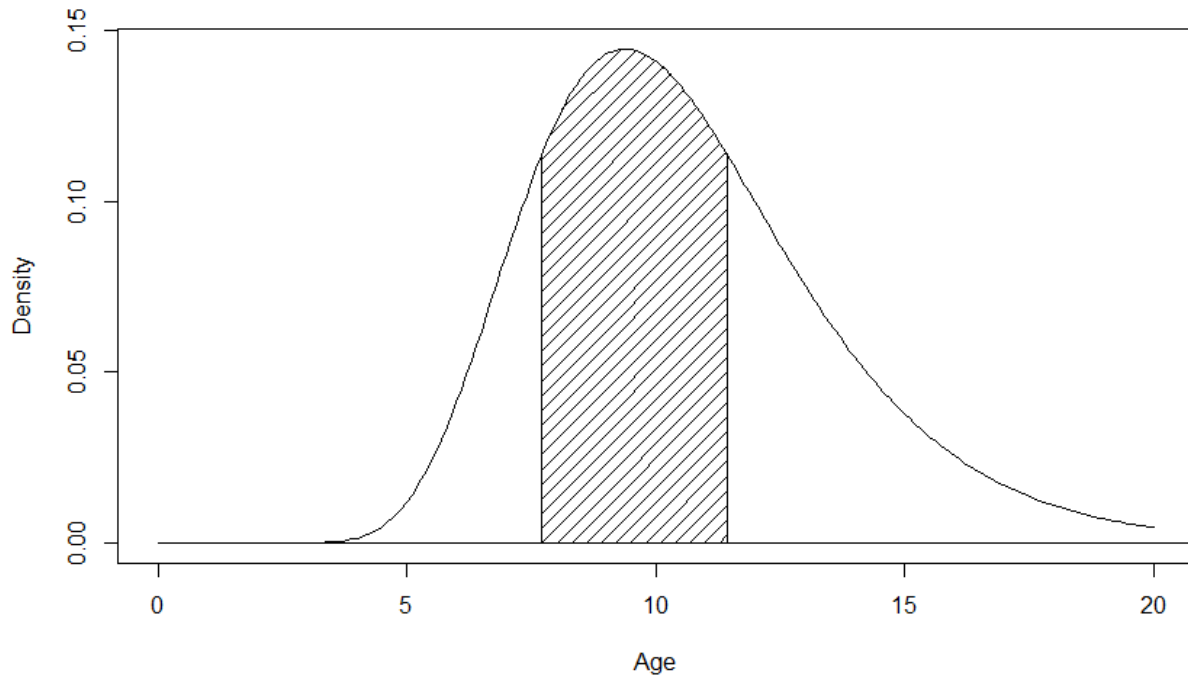
HPD male upper incisor 2 stage 11 (R 3/4), US Deaths 2008 prior



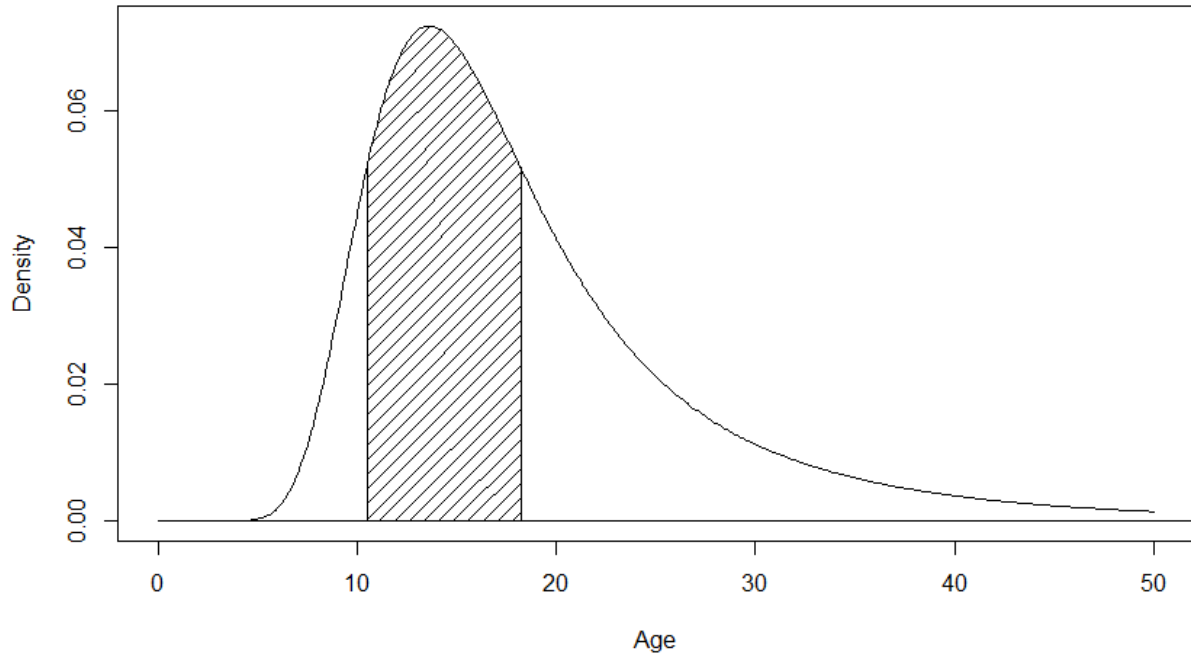
HPD male upper incisor 2 stage 12 (Rc), US Deaths 2008 prior



HPD male upper incisor 2 stage 13 (A 1/2), US Deaths 2008 prior



HPD male upper incisor 2 stage 14 (Ac), US Deaths 2008 prior



Appendix 6 – Letter of approval from the IRB



EAST CAROLINA UNIVERSITY

University & Medical Center Institutional Review Board Office

4N-70 Brody Medical Sciences Building- Mail Stop 682

600 Moyer Boulevard · Greenville, NC 27834

Office 252-744-2914 · Fax 252-744-2284 · www.ecu.edu/irb

Notification of Exempt Certification

From: Biomedical IRB
To: [Kelsey Roene](#)
CC: [Megan Perry](#)
Date: 9/10/2013
Re: [UMCIRB 13-001213](#)
A Bayesian Approach to Investigating Age-at-Death of Subadult Archaeological Samples

I am pleased to inform you that your research submission has been certified as exempt on 9/10/2013. This study is eligible for Exempt Certification under category #4.

It is your responsibility to ensure that this research is conducted in the manner reported in your application and/or protocol, as well as being consistent with the ethical principles of the Belmont Report and your profession.

This research study does not require any additional interaction with the UMCIRB unless there are proposed changes to this study. Any change, prior to implementing that change, must be submitted to the UMCIRB for review and approval. The UMCIRB will determine if the change impacts the eligibility of the research for exempt status. If more substantive review is required, you will be notified within five business days.

The UMCIRB office will hold your exemption application for a period of five years from the date of this letter. If you wish to continue this protocol beyond this period, you will need to submit an Exemption Certification request at least 30 days before the end of the five year period.

The Chairperson (or designee) does not have a potential for conflict of interest on this study.

