Lecture notes for bio 106 essay



Reproductive roles Male's job is to get the sperm to the egg The sperm are specialized to deliver the male's genes to the egg Female's job is to produce a gamete (egg) containing the female's genes Egg is specialized to nourish the embryo Egg is large and contains nutrients Egg must be moved along Female must also nourish and protect the embryo and fetus This is the job of the uterus Male reproductive strategy: Produce millions of gametes and hope that one makes it to the egg Female reproductive strategy: Invest heavily in one gamete and nourish and protect it Egg Cell (Ovum; 1 copy each chromosome) Sperm Cell (1 copy of each chromosome) MEIOSIS (a type of cell division that begins with a cell with 2 copies of each chromosome and ends with 4 cells with 1 copy of each chromosome) FERTLIZATION (Fusion of Egg and Sperm nuclei; creates a cell with 2 copies of each chromosome) Zygote (cell formed by fusion of egg and sperm; has 2 copies of each chromosome; will develop into new individual) Ovary (in female) Testis (in male) 1 Gonads: a. Testes in male b. Ovaries in females 1. roduce gametes Male = sperm Female = ovum or egg join zygote; new individual 2. produce sex hormones testosterone estrogen + progesterone Male Reproductive System Testes located in scrotum why? sperm don't survive well at body temperature is 3-40 F cooler temp kept constant by reflex how? - pass through the inguinal canal before birth hole usually closed over with connective tissue possible problems 1. cryptorchidism failure of the testes to descend if not corrected, results in sterility corrected by surgery or by administering hormones 2. inquinal hernia inquinal canal does not completely close intestine may push into opening correct with surgery more common in men but may occur in women Inside Testes 1. eminiferous tubules about 1000 site where sperm are produced by a process called

spermatogenesis produce 100 million+ sperm each day from puberty until death spermatogenesis A. takes place in an orderly progression from the outside edge of seminiferous tubule to inside (lumen) B. involves changes in genetic information and changes in the shape and functioning of cell sperm carry father's genetic contribution to next offspring body cells have 2 copies of each chromosome (1 from Mom & 1 from Dad) gametes (egg or sperm) can have only 1 copy of each chromosome meiosis = the type of cell division that produces gametes 1 cell with 2 copies of each chromosome Meiosis spermatogenesis 4 cells with 1 copy of each chromosome Sperm Structure 2 designed to deliver male's genetic contribution to next generation 1.

Head – contains male's genetic contribution to next generation; almost all nucleus 2. Acrosome – a sac containing enzymes to will allow the sperm to digest the outer layers around egg so sperm nucleus can reach egg nucleus 3. Mitochondria – energy to fuel the trip to egg 4. Tail (or flagellum)—has contractile fibers for motility; allows the sperm to swim to egg 2. interstitial cells – produce male sex hormone – testosterone Still in testis – between seminiferous tubules Beginning at puberty these cells secrete testosterone They are stimulated to secrete testosterone by LH, a hormone produced by the anterior pituitary gland LH (from anterior pituitary) stimulates the release of testosterone

HORMONES chemical messengers produced by certain glands and released into the blood hormone reaches all cells only cells with a receptor for that hormone can respond a cell responds by doing what that cell does it might divide it might produce a chemical it might increase rate of certain chemical reactions so hormones have different effects on different cells cells without

receptors for that hormone cannot respond 3 Cell 1 (with receptor) Effect 1 Cell 2 (with receptor) Effect 2 Hormone Into blood supply throughout body Cell 3 (with receptor) Effect 3 Cell 4 (no receptor) No Effect Testosterone causes: development of male reproductive apparatus sperm maturation secondary sex characteristics sex drive (in part) Possible problems with testes Testicular cancer: most common in males 25-30 yrs. more common if testes did not descend after 6 yrs. may be hereditary usually does not cause pain Practice self exam! Feel for small lump Best done after a hot shower Sperm next enter a system of tubes to store and transport sperm 1. pididymis: tube about 20 feet long stores sperm sperm mature here, sperm change size and shape, metabolism changes, sperm become capable of moving but don't yet. sperm moved along by peristalsis (a wave of muscle contraction) 2. vas deferens: sperm duct conducts sperm from epididymis to urethra 3. urethra: conducts sperm to outside of body, also conducts urine but never at same time Accessory Glands: 1. Bulbourethral glands mucous secretion just before ejaculation lubricant? Buffers to adjust pH of urethra 2. Prostate gland secretes fluid, milky color alkaline activates sperm counteracts acidity of female reproductive tract Possible problems with prostate 1. enlarges in older men difficulty urinating & decreased bladder volume 2. rostate cancer grows slowly can spread detected by: rectal exam and blood test for PSA (prostate specific antigen) 4 3. Seminal vesicles make up most of the volume of semen secretion probably nourishes sperm (contains fructose, vitamin C, amino acids, prostaglandins) Result = semen Semen: secretions of accessory glands and sperm about 1 tsp. per ejaculation, about 20 % sperm Functions: transport sperm lubricate passageways nourish sperm decrease acidity of female reproductive tract

Penis: Functions: transfer sperm to female conducts sperm outside body tip is enlarged = glans penis (rich in sensory endings) Mechanism of erection 3 columns of spongy tissue arteries dilate? increase blood delivery veins close down blood accumulates

Erectile Dysfunction = Impotence inability to achieve or maintain an erection common problem many possible causes - both physical and emotional drugs now available to help a man have an erection when he is sexually stimulated (e. g. Viagra, Cialis, Levitra) These inhibit the breakdown of the neurotransmitter that causes the arteries in penis to dilate arteries are dilated longer increases and prolongs blood entering the penis? erection Female Reproductive System gonads = 2 ovaries 1. produce eggs or ova (singular = ovum) egg is specialized to provide nourishment for early embryo large cell full of nutrients 2. produce female hormones: estrogen progesterone Female also nourishes and protects the developing embryo and fetus = job of the uterus 5 Ovarian Cycle = series of events in the ovary that leads to production of egg, estrogen & progesterone 1. ollicle maturation primary follicle = an immature egg surrounded by a layer of follicle cells as follicle matures the immature egg gets larger follicle cells divide and form many layers around egg follicle cells secrete estrogen mature follicle egg completes first meiotic division layers of follicle cells splits forming a central cavity filled with fluid containing estrogen egg pushed to side with layer of follicle cells 2. ovulation = release of immature egg from ovary egg released with layer of follicle cells around it rest of follicle cells stay in ovary 3. corpus luteum forms from follicle cells remaining in ovary corpus luteum secretes estrogen and progesterone

OVARY primary follicle (immature egg surrounded by follicle cells) mature follicle (many layers of follicle cells, fluid filled cavity, egg surrounded by follicle cells) after ovulation follicle cells remaining in ovary become corpus luteum estrogen progesterone egg 1. estrogen maturation of egg development and maintenance of reproductive structures cell division: thickens lining of uterus also occurs in breast tissue secondary sex characteristics pubic hair armpit hair broader pelvis breast development 2. progesterone prepares uterus for egg implantation maintains pregnancy 6 Oogenesis = the process by which an egg (ovum) is formed Meiosis: starts with a cell that has 2 copies of every chromosome ends with up to 4 cells with 1 copy of every chromosome 1 egg (ovum) and 3 non functional polar bodies IN FEMALES MEIOSIS IS NOT A CONTINUOUS PROCESS Preparations begin efore birth in all potential eggs Then development stops Beginning at puberty, 1 egg continues to the next stage of development The egg is ovulated (released from the ovary) Meiosis is completed ONLY if the egg is fertilized) Number of ova At puberty: potential for about 400, 000 eggs Usually 1 each month develops in each monthly cycle (if 2 form and both are fertilized get fraternal twins) total egg production ~ 450 eggs in lifetime menopause - rest of potential eggs have degenerated Oviducts (Fallopian tubes tubes that conduct the egg to the uterus - takes about 3 days open end enlarged and fringed - increased surface area for catching egg cilia line oviducts to help move egg along fertilization – usually in upper third of oviduct ectopic pregnancy - usually a tubal pregnancy early embryo implants and begins development at site other than uterus usually in Fallopian tube (oviduct) dangerous to mother – must be terminated Uterus provides nourishment & protects the developing embryo and fetus A. Cervix

- tubular portion the extends into vagina has opening through which sperm enter and baby exits B. Body - region in which fetus develops 1.

Endometrium – lining site where embryo implants built up each month cell division makes it thicker becomes more vascular (more blood vessels) glands develop that provide nutritious material then lost as menstrual fluid (woman gets her period) 2. muscle allows uterus to expand as fetus grows 60X bigger at full term pregnancy 7 provides force to push baby out Possible problems with uterus: 1. Pelvic Inflammatory Disease (PID): any bacterial infection of pelvic organs especially uterus, oviducts, ovaries; may spread (peritonitis) may be painful or chronic may have no symptoms often leaves oviduct scarred so that fertility is reduced and the risk of ectopic pregnancy is increased treated with antibiotics most commonly caused by sexually transmitted bacteria the bacteria that cause gonorrhea and chlamydia 2.

Cervical cancer: involves external surface of cervix detect with PAP test risk factors -intercourse at an early age -multiple sex partners associated with certain STDsparticularly the HPV (human papilloma virus) that causes genital warts use of condoms and/or diaphragm decreases risk Vagina ~3?? muscular passageway to uterus elastic – expands to allow baby through possible problem with vagina: vaginitis most commonly yeast (Candida albicans) not usually sexually transmitted not from poor hygiene bacteria in the vagina produce acid Anything that kills the bacteria or makes the vagina less acidic allows yeast to grow ? vaginitis Clitoris • • • Derived from same embryological structure as the glans penis Becomes engorged with blood during sexual excitement No known function other than pleasure 8

Menstrual or Uterine Cycle the endometrium (uterine lining) is built up to nourish the embryo and then it breaks down and is lost as menstrual flow it is a cycle caused by interplay of hormones want the uterus ready to receive embryo if there is one the ovarian cycle that produces the egg must be coordinated with the uterine cycle that prepares the uterus done by same hormones Hormones of the menstrual cycle OVARY Estrogen - from follicle cells in ovary and later from corpus luteum maturation of egg cell division in endometrium (uterine lining) cell division in breast tissue Progesterone from corpus luteum further development of endometrium maintains endometrium ANTERIOR PITUITARY FSH - follicle stimulating hormone: stimulates development of follicle LH - luteinizing hormone formation of corpus luteum from follicle cells remaining in ovary maintains corpus luteum Negative Feedback X? Y hormone X leads to an increase in hormone Y Y? X hormone Y causes decrease in hormone X when hormone X levels fall, less hormone Y produced less hormone Y means less inhibition of hormone X hormone X increases and stimulates release of hormone Y IN GENERAL FSH & LH stimulate release of estrogen and progesterone estrogen and progesterone inhibit release of FSH & LH BUT rapid rise in estrogen triggers LH release LH causes corpus luteum to form and secrete estrogen and progesterone corpus lutuem degenerates - estrogen and progesterone levels drop (removes inhibition of FSH) 9

Negative feedback in menstrual cycle Low levels of estrogen and progesterone stimulate the release of FSH (and LH) from anterior pituitary FSH stimulates estrogen release by follicle cells in ovary Estrogen (and progesterone) inhibit FSH (and LH) release FSH and LH levels drop Estrogen

and progesterone levels drop if the egg is fertilized corpus luteum is maintained by a hormone from the embryo called: human chorionic gonadotropic (HCG) hormone for about 5 months the corpus luteum secretes progesterone placenta eventually takes over progesterone secretion progesterone is needed to maintain endometrium if progesterone secretion stops – there is a miscarriage Menopause considered to have occurred when there is no period for 1 year follicles in ovary spontaneously degenerate eggs no longer produced ends child-bearing years estrogen & progesterone no longer produced in ovary occurs most often between 4555yrs. occurs gradually Perimenopause = time leading up to menopause Symptoms hot flashes & dizziness – dilation of arteries in top half of body may be: irritable headaches fatigue physical changes skin drier & less elastic ? wrinkles breasts decrease in size might be change in distribution of hair osteoporosis – decrease in bone density NEED NOT CHANGE SEXUAL DESIRE 10

Estrogen DESIRABLE EFFECTS Brain regulates areas that prepare for reproduction maintains stable body temperature may protect memory Breast Breast programs glands promotes breast to produce milk cancer Liver & Heart helps regulate cholesterol production prevents atherosclerosi s Uterus Uterus programs uterus to promotes nourish fetus cancer of cell division in endometrium endometrium NEGATIVE EFFECTS Estrogen's Effect on Bone Bone maintains density causes calcium to be absorbed from gut promotes calcium deposit in bones Osteoporosis – decrease in bone density calcium salts make bone hard bones are constantly remodeled built up & broken down in response to stress (weight or pressure) until age 35 more build up than break down peak bone density influenced by: sex race size nutrition

exercise that puts weight on bones overall health Bone Formation Blood level

Of calcium Calcium in Bone 11

Bone Breakdown Diet influences level of calcium in blood good sources of calcium: milk and milk products (choose low fat) dark green vegetables nuts seeds Weight-bearing exercise stimulates bone formation in the bones that are stressed by the exercise walking jogging calcium levels are regulated by hormones calcitonin from thyroid gland causes calcium to be put into bones parathyroid hormone from parathyroid glands causes calcium to be removed from bone estrogen helps absorption of calcium from digestive system stimulates bone formation After menopause estrogen levels greatly decrease Bone Formation Calcitonin Estrogen Blood level Of calcium Calcium in Bone

The Breast Function To produce milk to nourish the young Structure The breast is composed almost entirely of fatty tissue and milk glands milk glands are called lobules each gland drains into a system of ducts these empty into a collecting chamber below nipple several ducts collect into one duct ducts drain through nipple Possible Problems 1. Premenstrual tenderness breast tissue is prepared each month along with egg and endometrium Estrogen causes cell division in breast tissue Progesterone causes increase in glandular activity Increase in blood supply to breast swelling and tenderness Parathyroid Hormone Bone Breakdown Bone Formation 12 2. fibrocystic breast disease (disorder) an exaggeration of monthly changes in breast tissue built up tissue is not completely reabsorbed and forms cysts feels like many lumps in breast 3. fibroadenoma – noncancerous lump in breast usually in upper & outer quadrant small moveable lump 4. reast cancer will return to this after discussion of cancer

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13 Cancer = uncontrolled cell division Cancer cells kill by: depriving other cells of nutrients preventing other cells from performing their duties blocking important pathways (air, blood, nerve) Cells divide Tumor Benign tumor: stays in one place; not cancer Malignant tumor: cancer, cells spread (metastasize) multiple tumors form in other parts of body usually spread via blood vessels or lymphatic system in healthy person cell division is regulated Cell Cycle = the orderly sequence of events in the life of a dividing cell G1 cell growth (G1 checkpoint - is the cell large enough to divide? S (synthesis) - genetic material (DNA of chromosomes) is duplicated G2 - growth and final preparations for cell division (G2 checkpoint - is the DNA replicated?) Cell division Mitosis - nucleus divides Cytoplasm divides produces two daughter cells Normal controls on cell growth regulate cell division to allow growth and replacement 14 Cancer cells escape controls Normal controls: 1. Genes regulate the cell cycle GENE carries the instructions for making a protein PROTEIN has a job in the cell it might form (part of) a structure it might be regulatory = determine whether a certain cellular process will occur mutation = change in the gene's information changes the instructions for the protein the new protein might not function or might function differently Normal Controls on Cell Division 1.

Genes regulate the cell cycle proto-oncogene – normal form of a gene that produces specific proteins that stimulate the cell cycle for growth and repair acts like accelerator about 60 known if other controls were faulty it would enhance the growth of a tumor tumor-suppressor gene – normal form of a gene that produces specific proteins that slow the cell cycle proteins stop cell cycle at one of the checkpoints acts like brakes Normal Genetic Controls

on Cell Division tumorprotosuppressor oncogene gene slows stimulates cell division mutations in these cell cycle genes can cause the cell to lose control over cell division Cancer oncogene = mutant protooncogene over stimulates cell division = stuck accelerator can help induce cancer dominant mutation only need 1 of the 2 copies to be mutant mutant tumor-suppressor gene impairs ability to slow cell cycle = broken brakes enhances tumor formation recessive mutation - need mutation in both copies of the gene to have an effect 15 p53 a tumor-suppressor gene detects damaged DNA 2. rogrammed cell death occurs when genes are damaged cancer cells have mutation in other genes that prevent the damaged cells from being destroyed 3. limited life span cell can only divide 50-60 times telomeres - protective pieces on tips of chromosomes end of telomeres shaved off with each cell division telomerase = enzyme that makes telomeres not present in normal cells is present in most cancer cells 4. Need for blood supply controls prevent new blood supply to tissue unless it is damaged cancer cells produce growth factors to attract new blood vessels blood vessels needed to bring nutrients and remove waste also provides route for cancer cells to spread tops cell division initiates DNA repair if too much DNA damage —> p53 triggers programmed cell death Development of cancer need several mutations in same cell before cancer starts leukemia – may be as few as 3 mutations colon cancer - may need as many as 9 mutations EXAMPLE ONLY: Development of colon cancer 1. Loss of tumor-suppressor gene from chromosome 5 a polyp forms on colon wall a benign, precancerous tumor grows 2. Activation of oncogene from chromosome 12 a class II adenoma (benign) forms) 3. Loss of tumor-suppressor gene from chromosome 18 a class III adenoma (benign) grows 4. loss of tumor-suppressor gene from

chromosome 17 a carcinoma (malignant tumor) forms 5. other changes ? cancer spreads to other tissues 16 5.

Need for cell attachment normal cells must be anchored in place oncogenes produce proteins that break anchors but signal cell that it is anchored Immune System - The body's defense system cells of the immune system roam the body looking for cells they don't recognize as belonging cancer cells have changed and are not recognized as belonging if cells of the immune system encounter a cancer cell, they will be destroy it Factors that can cause cancer 1. carcinogens = chemicals that cause cancer cause mutations that lead to cancer many mutate p53 stimulate cell division (e.g. estrogen) inhibiting the immune system may be in environment, in food or drink, or inhaled at least 50 carcinogens some carcinogens are only carcinogenic after modification in the body 2. iruses consist of genetic information (usually DNA) inside a protein coat virus enters cell and uses host cell machinery to make new viruses viral DNA is inserted into host cell chromosome and is replicated with host cell DNA viral DNA is then a permanent part of host cell chromosome - it has transformed the cell into a cancer cell - all daughter cells will have the viral genes viruses have oncogenes that produce proteins that stimulate cell division viral protein produced may be hyperactive in stimulating cell division OR viral gene may direct human gene to produce too much of a protein that stimulates cell division result is a host cell that has been permanently changed by virus so that it contains an oncogene that stimulates cell division 3. radiation interacts with DNA and causes mutation ultraviolet (uv) radiation from sun causes skin cancer ionizing radiation natural sources (cosmic rays,

radioactive materials in earth's crust) medical sources (x-ray exams) 17 Risk Factors 1. revious breast or other form of cancer Breast Cancer most breast lumps are benign (not cancerous) fibrocystic breasts fibroadenoma death rate from breast cancer has been declining due to early detection practice monthly self exam breast cancer usually begins in the ducts (80%) or the glands begins as lump or tumor lump is usually in upper outer quadrant staged by size of lump and how far it has spread it can metastasize: break out of this site and spread to fatty tissue or other parts of body through lymphatic system or blood supply to determine whether cancer has spread they would look at sentinel nodes (first lymph nodes to which a tumor drains) 2. Gender females much more likely (men can get breast cancer) 3. Age chance of breast cancer goes up with age rises sharply after 40 4.

Family history risk is 2X if: first-degree relative (mother, sister, daughter) with breast cancer risk is 5X if: 2 first-degree relatives with breast cancer may have inherited genes e. g. BRCA 1 or BRCA 2 these greatly increase chance of breast cancer BRCA 1 = a tumorsuppressor gene turns off another gene that blocks cell cycle 18 5. Hormone history prolonged, uninterrupted exposure to estrogen increases risk estrogen stimulates cell division in breast if too much estrogen some believe it can lead to cancer some types of breast tumors are stimulated to grow by estrogen more menstrual cycles = more exposure to estrogen risk increased by: a. early puberty – before 11 b. late menopause – after 55 c. not having children or delaying first pregnancy – after 30 What about other sources of estrogen? irth control pills – probably not hormone replacement after menopause – slight environmental sources – unknown certain pollutants mimic estrogen (pesticides, ingredients in

plastics) electromagnetic fields can boost body's production of estrogen 6.

Obesity fat cells produce a substance that is converted to estrogen 7.

Alcohol alcohol increases level of estrogen interferes with use of folate, which protects against tumor growth 19 Birth Control Effectiveness rate = # of couples out of every 100 using that means of contraception for 1 year who do NOT become pregnant Failure Rate = # of couples out of every 100 using that means of contraception for 1 year who DO become pregnant Typical Use = average person, use may be improper or inconsistent Vs.

Perfect Use = proper and consistent use Effectiveness in Preventing STDs STDs spread by contact (direct contact is usually needed) Infected surface surface Uninfected Effectiveness in Preventing Pregnancy Highly Effective 1. Sterilization (tubal ligation or vasectomy) prevent sperm from reaching egg 2. Hormonal Contraception A. Estrogen and progesterone - prevents egg development and ovulation 1. oral - the pill 2. vaginal ring - NuvaRing woman inserts the ring so that it encircles the cervix worn 3 weeks, removed for the 4th 3. skin patch - OrthoEvra new patch once a week for 3 weeks 4th week no patch B. Progesterone-only contraception interferes with fertilization and implantation Types a. ral - mini pill (not used much in US) b. implants c. injections C. Emergency contraception -? morning after pills? 1. Preven estrogen and progesterone 2. Plan B - progesteroneonly first dose within 120 hours (5 days), second dose 12 hours later Decrease spread of STDs Certain means of contraception also prevent contact between body surfaces Condom - male or female Diaphragm or cervical cap (some protection to woman) Increase spread of STDs Pill may increase woman's risk of certain STDs Spermicides increase a woman's risk of getting an STD from an infected

partner; damages vaginal lining 20 3. IUD - interferes with fertilization and/or implantation; dislodges embryo 4.

Diaphragm, cervical cap, FemCap, or Lea's shield with spermicide – covers cervix and prevents sperm from reaching egg 5. Condom – prevents sperm from reaching egg 6. Vaginal sponge Moderately Effective 1. Spermicides – kill sperm Foams are best is used as only means of contraception May increase the risk of STD spread, particularly in women 2. Rhythm Method – abstinence on all days that could result in sperm meeting an egg Unreliable 1. Withdrawal (coitus interruptus) Does not work 1. Douching after intercourse 2. Intercourse standing up or in some other position 3. Intercourse during menstruation (during your period) 4. Intercourse while breastfeeding Reliable methods 1.

Sterilization — permanent birth control Tubal ligation or vasectomy prevent the sperm from meeting the egg Should NOT be used if you or your partner may change your mind For male = vasectomy Close off vas deferens so sperm can't leave the male's body Sperm reabsorbed Still ejaculate Simple operation No effect on masculinity No effect on sex life or sex drive Risks: minimal Less than 1% of time tubes grow back together Occasionally a little bleeding in scrotum For women = tubal ligation = have tubes tied Close off oviducts (fallopian tubes) More difficult procedure than a vasectomy because must enter the abdominal cavity Does not cause menopause Will still menstruate No effect on sex drive 21 2. Hormonal Contraception A. Estrogen and progesterone – prevents egg development and ovulation 1. oral – the pill 2. aginal ring – NuvaRing – woman inserts the ring so that it encircles the cervix; worn 3 weeks, removed for the 4th 3. skin patch – OrthoEvra new

patch once a week for 3 weeks; 4th week no patch expect same risks as pill, but know most about birth control pill Contain estrogen and progesterone Estrogen & progesterone inhibit FSH and LH Without FSH the egg doesn't develop Without LH ovulation cannot occur Almost 100% effective – if used properly If you miss more than one day, use another form of birth control Side Effects Headaches Breast tenderness Weight gain Vaginal Infections are more common Serious Risks caused primarily by estrogen Circulatory System Problems rare but can be fatal A.

Problems: (1) High blood pressure (2) Increased tendency to form blood clots (1) High blood pressure Increases with time on pill Increases with woman's age STOP SMOKING - cigarette smoking also causes high blood pressure Blood pressure = pressure exerted by blood on vessel walls Created by beating of heart Pressure must be great enough to move blood around the body If too great = high blood pressure Problems caused by high blood pressure 1. strains the heart and blood vessels 2. can lead to an aneurysm (weak spot in artery wall balloons out; can rupture; bleed to death internally 3. promotes atherosclerosis (fatty deposits in arteries) and arteriosclerosis (hardening of arteries) 4. amages kidneys; can lead to kidney failure 22 (2) Increased tendency to form blood clots Danger is that a blood clot can break free and lodge in a small blood vessel, blocking blood flow B. Consequences: (1) Increased risk of heart attack and stroke Heart attack = death of heart cells Stroke = death of nerve cells in brain Heart attack & stroke occur when blood flow to heart or brain is interrupted by: Burst vessel Fatty deposits (atherosclerosis) Blood clot (2) increased risk of pulmonary embolism 2. Increased risk of urinary tract infections 3. Increased susceptibility to

sexually transmitted diseases A. Change in pH of vagina – increased risk of chlamydia and gonorrhea B.

Change in cervical structure exposes vulnerable cells C. HPV (human papilloma virus that causes genital warts) infection is more likely to result in cervical cancer Progesterone seems to activate HPV in cervical cells grown is culture Non-contraceptive benefits of the pill Decreased risk of PID (pelvic inflammatory disease) Decreased risk of ovarian and endometrial cancer Decreased risk of ectopic pregnancy Decreased risk of iron deficiency anemia 1. 2. 3. 4. 2. Hormonal contraception (cont.) B. Progesterone-only contraception interferes with fertilization and implantation Types a. oral mini pill (not used much in US) b. implants -hormone containing rods implanted in upper arm c. njections - DepoProvera injection every 3 months 99% effective in preventing pregnancy no protection against STDs 23 Mechanism of action of progesterone only a. Thickens cervical mucus b. Interferes with movement of sperm c. makes implantation more difficult because endometrium thin d. Sometimes blocks ovulation e. Makes the corpus luteum degenerate too guickly (removes the source of progesterone that maintains the endometrium) Side effects a. Menstrual cycle disturbance Periods irregular More days of light bleeding Missed periods b. Weight gain c. breast tenderness d. bone density decreases 2. Hormonal contraception (cont.) C. Emergency contraception - "morning after pills" 1. Preven estrogen and progesterone 2.

Plan B – progesteroneonly first dose within 72 hours, second dose 12 hours later Emergency contraception is thought to work by inhibiting or delaying ovulation preventing fertilization altering the endometrium, making it an https://assignbuster.com/lecture-notes-for-bio-106-essay/

inhospitable place for implantation of the young embryo used after an act of unprotected intercourse if pregnancy not desired risk of pregnancy varies from 0 – 26 after a single act of intercourse – depending on day of cycle morning after pill decreases the risk of pregnancy by 75% (e. g. from 26% to 6. 5%) does not affect the embryo is it has already implanted Side effects: 1. nausea in 50-70% of women 22% vomit 2. menstrual cycle disturbance next period 2 – 3 days early or late 3.

IUD (intrauterine device) - interferes with fertilization and/or implantation; dislodges embryo Small device placed inside the uterus by physician remains effective for 1, 3 or 7 years, depending on the type 24 Effectiveness 97% with progesterone 99% with copper Mechanism of action of IUD- affects: Sperm - immobilizes sperm; interferes with their movement Ovum - speeds up movement to uterus Fertilization inhibited Endometrium - not properly developed for implantation FemCap - 3 sizes, latex free, removal strap Prevents the sperm from reaching the egg Must use with spermicidal cream or jelly Helps seal gaps Holds it in place Added chemical protection Must be fitted by a health care professional so seal is tight refit if weight changes by more than 10 lbs. Effectiveness 97-98% perfect use 81% typical use It is? at the time? rotection In place not more than 2 - 3 hr before intercourse Left in place at least 6 - 8 hrs. after intercourse Offers the woman some protection against STDs Risks with diaphragm - minimal 1. slight increase frequency of bladder infection 2. possible allergic reaction 3. toxic shock syndrome don't leave in place more than 24 hours or use when you have your period 1. 2. 3. 4. Disadvantages 1. if never had children, insertion is painful 2. may have heavier menstrual flow & more cramps 3. body may reject it - then not

protected against pregnancy Risks 1. pelvic inflammatory disease – primarily following insertion of the device can lead to sterility and increased risk of ectopic pregnancy 2. increased risk of ectopic pregnancy 4.

Diaphragm or cervical cap with spermicide – covers cervix and prevents sperm from reaching egg Soft rubber cup on flexible ring that fits over the cervix Lea's shield one-size fits all reusable device 25 5. Condom – prevents sperm from reaching egg A. Male condom Thin strong latex sheath that covers the penis and prevents sperm from reaching the egg Disadvantages: Must be placed on an erect penis – before contact with vagina Decreased sensation Failures usually due to tearing if pulled on too tightly – leave ?? at tip if too little lubrication Penis should be withdrawn from vagina while still erect B. female condom Pouch of polyurethane with a flexible ring at each end Effectiveness in preventing pregnancy — 74% typical use Effectiveness against STDs Little known Does provide a barrier Male condom still better 6.

Vaginal sponge use: put in place before intercourse (moisten first) leave in place for at least 6 hours after intercourse effective for 24 hours works by: 1. creating a barrier to sperm 2. trapping sperm in sponge 3. spermicide to kill sperm effectiveness: about 83% less if you have had children Moderately Effective 1. Spermicides – kill sperm Foams are best is used as only means of contraception Effectiveness in preventing pregnancy – about 80% for 60 min. Increases a woman's risk of getting an STD from an infected partner; damages vaginal lining 26 2. Rhythm Method – abstinence on all days that could result in sperm meeting an egg Egg Can be fertilized for about24 hr. after ovulation Ovulation ccurs 14+/days before the onset of flow Sperm Can live for at least 2 days within woman's body Problem is predicting ovulation 2

days before it occurs Effectiveness about 75% Fertile period Subtract 14 days from cycle length Add 2 days on either side for uncertainty in time of ovulation Earliest fertile day = 2 days before earliest expected day of ovulation Latest fertile day = 1 day after the latest expected day of ovulation Works best if you avoid all days until at least 1 day after you know ovulation has occurred Ways of detecting ovulation 1. Body temperature Requires a special thermometer Must be done first thing in the morning When body temperature increases slightly and stays up ovulation has probably occurred 2. cervical mucus cervical secretion is more slippery and thinner at ovulation Unreliable 1. Withdrawal before ejaculation (coitus interruptus) Methods that DON'T work 1. Douching after intercourse 2. Intercourse standing up or in some other position 3. Intercourse during menstruation (during your period) 4. Intercourse while breastfeeding 27

Sexually Transmitted Disease (STD) and Sexually Transmitted Infection (STI) Extremely Common 2/3 of cases in people under age 25 More likely to affect women Women exposed greater surface area of mucous membrane during sexual contact Women less likely to know they are infected Infected area not easily seen Urethra less likely to be infected So less like to be pain Therefore, women more like to have serious consequences. Spread by contact (direct contact is usually needed) Infected? Uninfected surface surface Mucous membranes are most vulnerable linings of: Urethra Vagina, uterus, fallopian tubes Vulva (external genitalia of woman) Mouth and throat Rectum Eyes Many STDs can enter through break in skin

Bacteria A bacterium is a single cell A bacterium can divide very rapidly producing two daughter cells results in very rapid (exponantial) growth of https://assignbuster.com/lecture-notes-for-bio-106-essay/

the population *Bacteria produce harmful chemicals = toxins (poisons) of enzymes these toxins kills or damage body cells the damage to body cells causes the symptoms of the disease Bacteria ? Toxin or Enzyme ? Damages / Kills Body Cells Bacteria divide rapidly ? More Cells? More Toxin (or enzyme) ? More damage to body. * Sometimes the damage or symptom is caused by the body's defense mechanisms against the disease. Bacteria = cells with a slightly different structure than the cells found in your body Bacteria have a cell structures (called ribosomes) that have a sliightly different structure than human version Structural differences are important because they allow antibiotics to bactieria without killing host (your) cells. 28

Antibiotics kill bacteria by: preventing bacteria from making cell walls OR preventing bacteria from making complete proteins OR damaging the plasma membrane Bacteria can become resistant to antibiotics by: inactivating the antibiotic pumping the antibiotic out of cell devoloping the ability to function in spite of antibiotics Antibiotic – resistant bacteria are a major health threat Antibiotic Resistance Some bacteria are now resistant to every known antibiotic Bacteria get their resistance from genes that: Inactivate the antibiotic Pump the antibiotic out of the cell Allow them to function in spite of antibiotic How do bacteria get these resistance genes? 1. They can get their own genes through mutation and selection a. mutation rate is high because rate of cell division is high hen antibiotics are used that are not strong enough or are not used long enough, the most resistant survive each time antibiotic taken improperly, the more resistant bacteria survive resistance builds b. the "good bacteria? are killed by the antibiotic" the resistant bacteria can reproduce faster than normal, healthful bacteria

and cause illness 2. Bacteria can get resistance genes from other bacteria through plasmids that carry genes for resistance Plasmids – a small circular piece of DNA (genetic materal) that contains a few genes not necessary for bacteria to live, but bacteria with them often have an advantage can be inserted into bacterial chromosome and come out as circular piece again Plasmids can be copied and a copy gived to another bacterium through sex then both bacteria have the genes on the plasmid. 29

Certain plasmids (called R factors) have genes for resistance to antibiotics possible to have genes for 1 or 10 different antibiotics genes for resistance for one antibiotic can be added to a plasmid than has genes for resistance to other antibiotics leads to the development of bacteria that are resistance to many different antibiotics multi-drug resistance tends to happen in places where antibiotic use is heavy hospitals farm animals Resistance develops where antibiotics used most Hospitals Livestock Overuse and Misuse of antibiotics has led to resistance 1. Misuse for medical purposes Don't demand antibiotics for viral diseases – they don't work on viruses Take the full course of your prescription 2.

Widespead use in livestock and agriculture Used in livestock to promote growth Resistance genes can spread from the animals to the bacteria that harm humans Cook meat throroughly (be sure meat juices don't come in contact with other food) Wash fruits and vegetables Avoid raw eggs Chlamydia and Gonorrhea Caused by different bacteria but have similar symptoms Both primarily affect mucous membranes Most noticeable symptom – if it occurs – is pain during urination This occurs if urethra is infected Urethra is more likely to infected in a male Therefore males more

likely to have symptoms Often they don't cause symptoms Can still spread the cactiria to others Bacteria still damage reproductive structures 30

Chlamydia (Chlamydia trachomatis) Most common bacterial STD in US Highly contagious Symptoms – slow to appear, 3 weeks to months Men More likely to have symptoms than women Painful urination Discharge from urethra Women If urethra is infected Painful urination Discharge from urethra PID (chlamydia causes 50-90% of PID) Slight vaginal discharge Pain during intercourse Abdominal pain & fever Chlamydia is the STD the most likely to cause scar tissue to form in the tubes that gametes move through Because of scar tissue: Chlamydia is the STD most likely to cause sterility Chlamydia is the STD most likely to increase the risk of an ectopic pregnancy The bacteria that cause chlamydia Must live within a cell because they cannot generate their own ATP They use the ATP that the host cell produces Energy in food ATP Energy for cell activities Effects on Fetus in Utero can cause membranes to rupture can cause death of fetus contracted during birth blindness pneumonia infection of mouth, throat, rectum Diagnosis Urine test for DNA for Chalmydia Swab cervix (women) or urethra (males) and culture cells Pap test (women) Treatment: Antibiotics Gonorrhea Caused by diplococcus bacterium Neisseria gonorrheae Symptoms Often none Men More likely to have symptoms than women Painful urination Discharge from urethra 31

Women If urethra is infected Painful urination Discharge from urethra PID
Slight vaginal discharge Pain during intercourse Abdominal pain & fever
Acidity decreases if on pill or at menopause Effect on fetus: Contracted
during birth May cause blindness Diagnosis: Urine test form DNA Swab cervix

(women) or urethra (males) and culture cells Look for bacteria in cells

Treatment: Antibiotics New varieties are resistant to antibiotics Syphilis

Cause = bacterium (Treponema pallidum) Requires a warm, moist
environment Can invade any mucous membrane Usually in the genital area

Three Stages 1. Primary Stage 2-6 weeks after contact chancre forms may
be small swelling or deep lesion usually hard raised edges " crater-like"
painless at site of contact heals by itself in 4-6 weeks diagnosis at this pint is
by isolation of the bacterium from chancre 2.

Secondary Stage 2-10 weeks after chancre appears Symptoms Rash- doesn't itch, ulcerates Ulcers in mucous membranes In mouth, vulva, vagina, rectum Warly growths around anus and genital organs Headache Body ache May have: Sore throat Gastrointestinal upset Loss of hair Diagnosis at this stage: Blood test that looks for antibodies (VDRL) 32 3. Tertiary Stage 8-25 years after initial contact almost any organ can be infected and develop lesions called a gumma most common sites of gummas: 1. large arteries decrease diameter of artery aneurysm – weak spot in artery that balloons out 2. brain & spinal cord Blindness Deafness Paralysis Mental degeneration 3.

Skeleton Effect of fetus Transferred across placenta Can cause deformities

Can be fatal Treatment: Early stages curables with antibiotics Virus Viruses
have genetic material (usually DNA but some have RNA) and a protein coat
(capsid) Structure of a typical virus Steps in Viral Life Cycle 1. Attachement –
Viral protein binds to receptor on host cell 2. Penetration – virus enters host
cell 3. Biosynthesis – viral genetic material replicated by using host cell ?
machinery;? new coat proteins made 4. Assembly – newly synthesized viral
compnents put together to form new viruses 5. Release [called viral]

shedding or budding]; viruses leave cell with envelopes from host cell OR

Viruses genetic information can be integrated into host cell chromosome and

stay there in dormant form until it is reactivated Viruses can cause call

damage as they are released (shed) 1.

Rapid release – cell can rupture and die 2. slow release – cell damage and dearth occurs over long time period 3. Periodic release – viruses can remain in certain body cells (e. g. nerve cells) for life; they may be release from body cells periodically and enter new target cells. (herpes can remain in nerve cells; be released epriodically and enter new epithelial cells; damages the epithelial cells) 4. be integrated into host chromosome and stay there in dormant form until it is reactivated 33 Lytic Infection Persistent Infection Latent Infection Transformation to cancerous cell Rapid release of new viruses from infecded cell caused cell death.

The symptoms of the disease depend on which cells are killed Slow release of new viruses causes cell to remain alive and continue to produce new viruses for a prolonged period of time. Delay between infection and symptoms. Virus is present in the cell without harming the cell. Symptoms beging when the virus begins actively replication and causes cell death when new viruses exit the cell. Certain viruses insert their genetic information into host cell chromosomes. Some carry oncogenes (cancer – causing genes) that are active in the host cell. Some disrupt the functioning of the host cell's genes that regulate cell division, causing the cell to become cancerous. Viruses and Disease 1.

Can cause cell damage as they leave the host cell; the cell damage causes the symptoms 2. Can cause cancer when they insert themselves into host chromosome or by producing factors that affect the host genes that regulate cell division Genital Herpes Cause virus Herpes simplex -type 1 (HSV-1) usually associated with fever blisters + cold sores -type 2 (HSV-2) - usually causes similar sores in genital area Symptoms (if there are symptoms) first may have a tingling or itching sensation (called the prodrome) 2-20 days after contact blisters of fluid filled sores 1st attack lasts about 3 wks (1wk-4wks) can use ointments to relieve the pain will go away whether treated or not Also cause cancer hen they insert themselves into host chromosome or by producing factors that affect the host genes that regulate cell division Viruses and Disease 34 Genital Warts in about half (50%) of people with herpes: blisters reform periodically usually at times of stress because the virus moves the the sacral ganglia of the spinal cord not affected by the human immune system here can be reawakened + cause new sores -maybe 2x month or 1x in 10yrs -NO CURE Genital Herpes spread by contact of infected and uinfected surface no sex while blister present or during prodrome or for at least 10 days after blisters are gone use a condom at all timeseven when no blisters are present erpes can be spread to newborn if deliver vaginally while virus is present if infant's infection is in liver and central nervous system-can be fatal Diagnosis Examination of sores Culture fluid from sores Blood test for antibodies in women-Pap test Treatment -Syptoms only Acyclovir (Zovirax):-reduce severity of first outbreak and reduce frequency of recurrences Human Papilloma Virus (HPV) usually transmitted by sexual contact 50-70% of those who have sex with an infected person will get them more likely if on the pill or pregnant or

uncircumsized appear 1-2 months after contact, maybe longer appearance of growth on dry areas- brownish on moist areas-pink they grow may cause foul-smelling discharge may itch -warts can be removed by: 1. freezing 2. burning 3. laser 4. treated with a chemical (podophyllin) that is painted onleft 4 hrs. washed away warts fall off * virus may remain Diagnosis appearance of wart in women-Pap test can look for DNA of HPV inside cells 35 Genital Warts *ASSOCIATED WITH INCREASED RISK OF CERVICAL CANCER -HPV found in 90% of women with cervical cancer There is now a vaccine against HPV and, therefore, against cervical cancer *ASSOCIATED WITH AND INCREASED RISK OF CANCER OF PENIS 36 BODY DEFENSES Innate Responses – Nonspecific Physical and Chemical Barriers Adaptive defense: Specific defenses (directed at specific target): The immune system: 1. Specific for particular "invader" (antigen) 2. Has memory for specific antigens previously encountered Lymphocyte encounters antigen Immune responses have 1.

Specificity: Specific for particular "invader" (antigen) Antigen = a large molecule (not recognized as belonging in the body) that triggers an immune response Ex: antigen can be on the surface of a bacterium or virus etc.; can be a bacterial toxin Your body cells have markers (molecules) that label them as belonging in your body Each lymphocyte has receptors on its surface that recognize a specific antigen When that antigen is present, it causes that lymphocyte to divide many times Effector cells Memory cells Effector cells attack specific target Memory cells remain and provide a quick response in subsequent exposure to same antigen Third line of defense: Specific defenses (directed at specific target): The immune system: Immune

system is 1. specific for a particular ? invader? antigen) 2. has memory for specific antigen previously encountered Creates an army of lymphocytes specialized to attack that antigen These are called Effector cells 2. Memory: have memory for specific antigen previously encountered Memory lymphocytes remain to cause a quick response the next time the same antigen is encountered 37 STEPS IN IMMUNE RESPONSE 1. Threat: foreign cell or molecule enters body 2. Detection Macrophage: detects invader engulfs invader digests invader 3. Alert: Macrophage places a piece of consumed antigen on its plasma membrane attached to a self marker presents the antigen to a helper T cell activates the helper T cell 4.

Alarm: Helper T Cell: after activation by a macrophage, it divides, forming effector helper T cells and memory helper T cells turns " on" both lines of immune response to fight that specific antigen by activating B cells and T cells 5. Build specific defense (clonal selection) Lymphocyte encounters antigen Effector cells attack specific target Memory cells remain and provide a quick response in subsequent exposure to same antigen 6. Defense A. Antibody-mediated immune response Effector B cell = Plasma cell Plasma cells secrete antibodies Targets = antigens outside of cell or on surface of cell B. Cell-mediated immune response Effector T cell = cytotoxic T cell Cytotoxic T cells kill foreign cells by causing them to burst Targets = cells bearing antigens (any cells recognized as foreign: e. g. nfected cell, bacteria, cancer cell) 7. Continued surveillance memory cells remain 8. Withdrawal of forces After antigen has been destroyed suppressor T cells shut down the immune response Effector cells cells Memory 38 A. Antibody-mediated immune response Effector B cell = Plasma cell Plasma cells secrete

antibodies Targets = antigens outside of cell or on surface of cell An antibody is a Y-shaped protein designed to recognize a specific antigen Antibodies help defend against a specific antigen Can only work against antigens that are free in blood Antibodies bind to the antigen Antibodies are secreted by plasma cells (effector B cells) Ways that Antibodies can Work 1.

Neutralization – bind to antigen prevent virus from being able to enter host cell inactivate toxin 2. Agglutination and precipitation -clumps " invaders" together makes it easier for other cells to engulf them 3. Activation of complement system complement (system) is a group of proteins that pokes holes causes " invader" to burst B. Cell-mediated immune response Effector T cell = cytotoxic T cell Cytotoxic T cells kill foreign cells by causing them to burst Targets = cells bearing antigens (any cells recognized as foreign: e. g. infected cell, bacteria, cancer cell) Cytotoxic T cells secrete proteins called perforins that poke holes in " invader" or foreign cells, causing them to burst 39

Cells Involved In the Immune System Macrophage: an antigen presenting cells engulfs and digests antigens places a piece of consumed antigen on its plasma membrane presents the antigen to a helper T cell activates the helper T cell B Cells: T Cells: Helper T Cell: the "on" switch for both lines of immune response after activation by a macrophage, it divides, forming effector helper T cells and memory helper T cells activate B cells and T cells Cytotoxic T cell: (effector T cell) responsible for cellmediated immune responses when activated by helper T cell, it divides to form effector cytotoxic T cells and memory cytotoxic T cells destroys cellular targets, such as virusinfected body cells, bacteria, fungi, arasites, and cancer cells

Suppressor T cell: the "off" switch for immune responses suppresses the activity of B cells and T cells after the foreign cell or molecule has been successfully destroyed involved in antibodymediated responses when activated by helper T cells, it divides to form plasma cells and memory cells Plasma Cell: effector in antibodymediated response secretes antibodies specific to the invader Memory Cells: responsible for memory of immune system generated by B cells or any type of T cell during an immune response enable quick and efficient response on subsequent exposures of the antigen may live for years 40 AIDS Acquired Immune Deficiency Syndrome Caused by HIV Human Immunodeficiency Virus HIV infects T cells T cells become HIV factories Organism enters body Macrophage detects it HIV kills helper T cells so THIS doesn't happen Activates a helper T cell Stimulates division of cytotoxic T cells (attack foreign cells) As helper T cell numbers drop, the body becomes increasingly susceptible to infection Stimulates B cells to form antibodies (destroys the infectious organism) 41