(Directions 1-10: Read the following passa.ge carefully and answer the questions given below it.

For aslong as biologists have studied cancer,they have **nurtured** one dream. Their hope has been that, for all of the .many different cancers that can eat away at a body - from the gliobastomas that creep through the brain like kudzu through Georgia, to the lung and colon cancers that envelop vital organs like a death shroud - there will turn out to be a single step that determines whether cells turn cancerous. By targeting drugs at that step, physicians would be able to stop a cellfrom heading down the path towards cancerjust as a switchman stops a tra in from going down the wrong track. It turns out that a gene called p53 - the gene whose useless mutant form, luckless families passed from patient to child through the generations - may be that switch.If p53 is healthy and doing its job,it keepsthe cellon thetrack of normalcy. But if the p53 gene is absent, damaged or tied up by other molecules,the cell goes down the track toward cancer. cp53 fulfi s this long held hope',says Dr. Bert Vogelstein of the !Howard Hughes Medical Institute (HHMI) and the Johns Hopkins Oncology Centre, who found, in 1989, that some 80 per cent of colon cancers involve p52.cAlternations of the p53 gene appear to be involved,directly or indirectly,inthe majority of human cancers.1 All said and done,p53 has been implicated in more kinds of cancer than most people knew existed:52 and counting.

Not bad for a gene that first broke on the scene in a bad case of mistaken identity. In 1979, David Lane of the University of Dundee in Scotland and Arnold Levine of Princeton University independently discovered p53, the proteinthat the p53 gene makes.In 1982,biologists **isolated** the p53 gene.But it seemed to cause, rather than suppress, cancer. Few researchers were interested in yet another one of those. It wa s not until1989 that Levine and Vogelstein separately discovered p53's true colours:it was a tumour killer. With this realisation,some 2,000 biologists have now dropped the date they brought to the danc e-their previous research subject- and latched onto the pretty new arriva I. 'Our interest converged on p53 like no other molecule', recalls oncologist Richard Elledge of the University of Texa s Health Science Centre at San Antonio.Three years ago p53 won the scienceworld's Academy Award,earning cMolecule of the Year' honours from thejournal Science.Just last month researchers led by Nikola Pavletich of New York's Memorial Sloan Kettering Cancer Centre produced a stunning photo of p53 that shows how rogue molecules can prevent it from performing its good deeds.Today there are more than 5,200 published studies on p53, and biologists are convinced that p53 is bound for even greater glory. 'What I like so much about p53 is that it predicts ways to treat cancer',says Princeton's Levine.'For the first time,we can honestly translate from thelab to the clinic'.

P53 acts asthe cell's director of damage control. A healthy cell,usually, keeps a small number of p53 proteins around, continuous y degrading them and replenishing the supply. But if something- ionising radiation, a chemical carcinogen, and chemotherapy drugs- damages a cell's DNA in a way that threatens to set it on the path to cancer,the cellswitches into high alert.If everything isworking right, something signalsthe p53 to stop degrading 'and tells it that it's time to be active', says molecular biologist CarolPrives of Columbia University.The p53 supply bui ds up,p53 starts actinglike an office clerk who, discovering a typo in an originaldocument that is about to be copied,turns off the copier untilhe can fix the typo.

p53 turns off the cell's the cell's copying machinery untilhe cellcan repairits damaged DNA.To so this, p53 floats toward the cell's genes.It slips into a specific stretch of DNA.There,it **triggers** expression of genes nearby.These genes make proteinsthat directly inhibit growth of the cell.The tumour -to-be is stopped dead. Sometimes, though, p53 acts morelike the clerk so disgusted with the many typos that he just trashes the document: p53 activates the cell's suicide software.

Except whenit doesn't. Even good genes can go bad,andthe genethat makes p53 has several ways of fa iing.Those fa iures account, directly or indirectly, for an estimated 60 per cent of human cancers, including those of the breast, lung,liver, skin,prostate,bla dder,cervix and colon. Most often,the p53 gene goes bad by undergoing a mutation, typically a spelling mistake. One of the 2,362 chemical '11 etters' (designated A,T,G and C) that make up the p53 gene changes into

another letter. When the p53 gene contains such a spelling mistake,the p53 protein that the gene makes is garbled,too. And proteins are not very forgiving of errors.A single wrongletter in a crucial part of the p53 gene produces a protein with a wrong molecule;the protein is now no more ableto supress tumoursthan a cork is to cap a volcano.

The Li-Fraumenifamiies (the syndrome is named after its discoverers) inherit their p53 mutations. If the sperm or egg from which a baby grew held a mutant p53, then every single cell of the 30 trillion in her body will also harbour a mutant copy.In theory,inheriting only one mutant p53 gene,from one parent,should not be a problem aslong asthe chid inherits a healthy p53 gene from the other parent. The healthy copy should make enough p53 to keeptumours at bay.But p53 doesn't workthat way.

First of all,each cellwith one bad p53 gene is only 'one mutation away from completely lacking the function of this **critical** gene', explains p53 researcher Tyler Jacks of HHMI and the Massachusetts Institute of Technology. That mutation can occur when the cell- in the liver, lungs or any other organ - makes a spelling mistake as it copies its genes before dividing into two cells. Then the cellhaslost its primary defence against cancer.

But even a cellwhose healthy p53 gene stays that way can be trouble.The p53 proteinsmade by the genes,both good and bad,get together in groups of four to form a sinuous complex thatlooks like ribbon on a Christmas present. If the mutant gene is churning out mutant proteins, then each four-ribbon tangle likely has a mutant among its strands.That is enough to keep the p53 ribbon from binding to DNA and halting tumour growth.

Most p53 mutations, though, are not inherited. Instead, they arise from a copying error or an attack by a carcinogen. Bullky chemicals, such as benzopyrene in cigarette smoke, changes G to T and C to A,for instance.A single mutant gene is enough to leave a cellwith no healthy,tumour quashing p53.And just one out of controlcellcan give riseto deadly tumour.

1. The primary purpose of the passage is to propose :
   A. An alternative cause of cancer, the gene p53.
   B. The p53-if healthy-keeps cancer away, but if unhealthy or absent, could result in a cell becoming cancerous.
   C. An alternative cure of cancer, the gene p53.
   D. A new drug that will prevent a cell from becoming cancerous.
   E. None of these.
2. Which of the following statements, regarding p53, is true?
   (a) p53 probably has more preventive rather than curative powers.
   (b) p53, when it falls, accounts for about 90% of cancers.
   (c) The perception among biologists that p53 is the cause of cancer, was trashed in 1989.
   (d) Biologists opine that p53 does not really suggest any of treating cancer.
   (e) None of these.
3. According to Carol Prives, the p53 gene is akin to an office clerk, in that p53.
   (a) Passes on the job the p53 protein to perform damage control.
   (b) Stalls the cell's copying process until the cell can rectify its damaged DNA.
   (c) If the situation is hopeless, decides to given up, and goes into the mode of self-destruction.
   (d) Both (b) and (c)
   (e) None of these
4. All of the following statements are true, except.
   (a) A wrong mutation of the p53 gene results in the p53 protein being rendered ineffective in suppressing tumours.
   (b) Most p53 mutations are inherited.
   (c) A healthy p53 gene can be useless if faced by even a single mutatnt p53 gene.
   (d) The Li-Fraumeni families inherit their p53 mutations.
   (e) None of these
5. Which of the following is true regardin those who have Li-Fraumeni syndrome?
   (a) One single mutant p53 gene inherited either from the sperm or the egg does not imply increased chances of getting cancer.
   (b) Any cell with one bad p53 gene could lose its defence against cancer with just one wrong mutation, since the bad p53 gene could destroy the good p53 gene.
   (c) The good p53 gene cannot nullify the effects of a mutant p53 gene and it probably will be rendered impotent by the latter.
   (d) A mutatnt p53 gene need not necessarily produce mutatnt and faulty proteins.
   (e) None of these.
6. What repercussion does a faulty p53 mutation-'a speeling mistake'-have?
   (a) The mutated gene may or may not produce a p53 protein with a wrong molecule.

(b) The mutated gene prevents the good p53 gene from performing its function, i.e. halting tumour growth.
(c) It strips the cell of its defences against cancer.
(d) both (b) and (c)
(e) None of these

7. Which of the following is most similar in meaning to the word **nurtured** as used in the context of the passage?
(a) cherished (b) encouraged
(c) protected (d) cared
(e) All of the above

8. Which of the following is most similar in meaning to the word **isolated** as used in the context of the passage?
(a) secluded (b) withdrawn
(c) unapproachable (d) extracted
(e) All of the above

9. Which of the following is most opposite in meaning to the word **triggers** as used in the context of the passage?
(a) blocks (b) cheack
(c) halts (d) stops
(e) All of the above

10. Which fo the following is most opposite in meaning to the word **critical** as used in the context of the passage?
(a) negative (b) analytic
(c) unimportant (d) humble
(e) All of the above

Direction (11-15) : In each of the following sentences there are blank spaces. Below each sentence there are five pairs of words. Find out which pair of wordscan be used to fill up the blanks in the sentences to make it grammatically correct.

11. A major security ……. Came to the fore during Prime Minister Narendra Modi's visit as his ……. Was misled by a police van directing the way.
(a) forefront, direction (b) lapse, cavalcade
(c) threat, path (d) alarm, track
(e) gap, walkway

12. When Sri Lankan cricketers ……. out wearing pollution masks in the middle of a Test match a the Feroz Shah Kotla ground in Delhi, it ……. a new low for the city.
(a) trooped, heralded (b) filled, resulted
(c) cut, hovered (d) rolled, meanaced
(e) headed, claimed

13. Mr. Hegde's remarks are not …….. those of an elected representative who swears …….. to the constitutions.
(a) obliging, faith
(b) accentuating, dominance
(c) according, credence
(d) according, credence
(e) consenting, trust

14. Pakistan appears to have ……. low in its treatment of Kulbhushan Jadhav's family members by not only violating the letter and spirit of the understanding about this but also conducting the meeting in a ……. And intimidating atmosphere, there by defeating the very purpose of such a reunion.
(a) remainded, deserted (b) stooped, hostile
(c) faced, dangerous (d) repeated, safe
(e) complied, hazardous

15. The way in which the ……. Indian batting line-up ……. against genuine fast bowling is well known.
(a) luxurious, marches (b) opulent, pits
(c) prolific, takes (d) famed, collapses
(e) influential, goes

Direction (16-20) : In each of the questions given below a/an idiom/phrase is given in bold which is then followed by five options which then tries to decipher its meaning as used in the sentence. Choose the option which gives the meaning of the phrase most appropriately in context of the given sentence.

16. The authority asked for a few days time **to chew** the matter **over** before making a final decision.
(a) organize (b) think
(c) register (d) restrict
(e) ignore

17. The little kids were going **all agog** for Christmas.
(a) ignorant (b) angery
(c) restless (d) emotional
(e) alert

18. I am **dead on my feet** from standing at the bar all night.
(a) cold (b) jolted
(c) gloomy (d) somber
(e) exhausted

19. She is a **dead ringer** of one of my friends.
(a) identical (b) acquaintance
(c) enemy (d) neighbor
(e) nemesis

20. Just **hold your horses** till we have an official communication.
(a) snub (b) suffer
(c) tolerate (d) wait
(e) operate

Direction (21-30) : In each of the questions given below a sentence is given which is then divided into five parts out of which last part is correct. There is an error in three part of the sentence andonly one part is correct. You have to choose the correct part as your answer.

21. Severe sengue haemorrhagic fever cause (A)/ blood vessels to leak, which leads to a loss of blood (B)/pressure and if this isn't treat quickly (C)/ by replacing bodily fluids, the person can went (D) **into shock and can die. (e)**
(a) A (b) B (c) C (d) D
(e) All correct

22. In an desperate attempt to (a)/prevent herbivores like nilgai (blue bulls) and wild boar for (b)/destroying their crops, farmers often (c)/set up illegal high-voltage electrical fences around its (d)/fields (e)
(a) A (b) B (c) C (d) D
(e) All correct

23. Last week, I visited (A)/mine ancestral village (B)/and found my self listen to (C)/family stories told with my aunts (D)/**who are well into their 70s (E)**
(a) A (b) B (c) C (d) D
(e) All correct

24. When a clinical researcher or a (A)/pharmaceutical company wants to check (B)/whether a new medicine or treatment (C)/works, they choose a largenough (D)/**set of patients for the clinical trial. (E)**
(a) A (b) B (c) C (d) D
(e) All correct

25. Research have successfully attempted (A)/gene-editing inside the human body to correct a (B)/defect on the DNA that causes Hunter's syndrome (C)/a disorder where the body can't broke down sugar that (D)/**builds bones, skin, tendons and other tissue. (E)**
(a) A (b) B (c) C (d) D
(e) All correct

26. Choosing to use public transport (A)/over driving a private vehicle was a good (B)/way to make a immediate difference. (C)/that not only decreases emissions but also save money (D)/ **and encourages physical activity. (E)**
(a) A (b) B (c) C (d) D
(e) All correct

27. The freedom struggle of India produce (A)/innumerable brilliant personality of intellectual excellence (B)/and comprehensive perceptions of various (C)/issue concerned with the quality of (D)/**human life in the country (E)**
(a) A (b) B (c) C (d) D
(e) All correct

28. Pupils should always (A)/be given freedom to think (B)/ on academic and social problems (C)/and to find their solutions (D)/**independently. (E)**
(a) A (b) B (c) C (d) D
(e) All correct

29. The President and the Vice-President continues (A)/to get salaries less than top bureaucrats and (B)/the chiefs of the armed forces, because the laws are since (C)/to be amended to rectification an anomaly in the implementation (D)/ **of the Seventh Pay Commission recommendations. (E)**
(a) A (b) B (c) C (d) D
(e) All correct

30. The government is set to brought out (A)/a new policy to spur domestically electronics (B)/production by March 2018 on a bid (C)/to boost its flagship 'Make in India' programme (D)/**and curb the country's trade deficit. (E)**
(a) A (b) B (c) C (d) D
(e) All correct